OCCUPATIONAL DISEASES

A Guide to Their Recognition

Revised Edition

June 1977

Editors —

Marcus M. Key, M.D.
Austin F. Henschel, Ph.D.
Jack Butler, M.D.
Robert N. Ligo, M.D.
Irving R. Tabershaw, M.D.
Manuscript Editor —
Lorice Ede, J.D.

U.S. DEPARTMENT OF HEALTH, EDUCATION, AND WELFARE
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Center for Disease Control
National Institute for Occupational Safety and Health

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Foreword

Although the history of occupational diseases extends back for centuries, many of them still go unrecognized today. At the present time, the potential sources of exposure are more numerous than ever. No matter how esoteric the causative agent, the diseases usually manifest themselves in relatively conventional forms. The problem is that the occupational origin is frequently overlooked.

It is hoped that the information contained in this guide will aid in the early detection of occupational diseases, thereby lessening an unnecessary burden on the nation's workforce as well as on our economy and productivity.

John F. Finklea, M.D.
Director, National Institute for
Occupational Safety and Health

Preface

The *problems* and *prospects* in the field of occupational safety and health have been epitomized by the *dangers,* acute and chronic, from the flood of new products our technology so freely gives us, and by the *support,* federal and state, implicit in the provisions of the Occupational Safety and Health Act of 1970, PL 91-596.

The Act is truly landmark legislation. It focused awareness on the actual and potential health problems inherent in the sudden accession of thousands of new chemical, physical, and biological combinations into the environment. The National Institute for Occupational Safety and Health was created by this Act. One of the Institute's primary functions is to assess the extent of, and means for preventing, health hazards in the workplace and to disseminate the information realized.

This guide is offered as one way of making available information necessary for timely recognition of symptoms of occupational diseases in furtherance of the national effort to assure "for every working man and woman in the nation safe and healthful working conditions."

Since 1918, when the earliest version of this guide was first published, there has been a recognized need and demand for guidance in diagnosing occupational diseases. With the enlarging scope and importance of occupational diseases and the continuing development of epidemiologic, clinical, and toxicologic information relating to their causation and diagnosis, a completely rewritten and enlarged edition of the text was published in 1964 with the title *Occupational Diseases: A Guide to Their Recognition.*

In the ensuing years from 1964 to 1977, changes have come about that warrant another complete revision and rewrite of the text. The stature of occupational safety and health dramatically changed with the passage of the Occupational Safety and Health Act of 1970. This field now has national recognition and the impetus of a national research and enforcement effort.

Although the content and organization of the book have changed, the purpose remains as stated in the 1964 edition: "to prevent and control the potential diseases of the occupational environment, thus leading to the fulfillment of the primary objective of optimal health for the working population."

This implies that the physician must be able to recognize work-related illnesses so as to take appropriate action, not only to institute proper treatment, but to assure that patient care is coordinated with management of the environment by those in control so that recurrence of such illnesses may be prevented.

Abstract

Occupational diseases are discussed in terms of occupational health hazards as a means to recognition of the disease. The text covers routes of entry and modes of action, chemical hazards, physical hazards, biological hazards, dermatoses, airway diseases, plant and wood hazards, chemical carcinogens, pesticides, sources of consultation, and a list of references.

Acknowledgments

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SECTION I

. . .the arts that men practice are various and diverse and from them may arise various diseases. Accordingly, I have tried to unearth in the shops of craftsmen, for these shops are schools whence one can depart with more precise knowledge, whatever may appeal to the taste of investigators, and, which is the main thing, to suggest medical precautions for the prevention and treatment of such diseases as usually affect the workers. . . .a doctor. . . should. . . question . . .carefully, . . .What occupation does he follow?

— Ramazzini

INTRODUCTION

The increase in the number and complexity of substances found in the workplace—substances that sometimes spill over into the community environment—makes imperative the dissemination, as efficiently and conveniently as possible, of certain basic information relating to occupational diseases.

This revised edition has been prepared as a ready reference in occupational diseases for physicians and nurses. Others who may find this reference helpful include consultants, industrial hygienists, and allied professional personnel who work with those engaged in business, industry, and agriculture. It is hoped that this book with its lists of references will be of use not only to the physician and occupational health nurse, but also to others responsible for planning and carrying out preventive occupational health programs.

DIAGNOSIS

Physicians are regularly consulted by workers with signs and symptoms of definite, as well as indefinite, character. The extent of the diagnostic problem is magnified by the introduction into the work environment of an ever-increasing number of substances whose potential for harm is not fully explored prior to their introduction for use.

The passage and implementation of the Toxic Substances Control Act of 1976 should, to a considerable extent, ameliorate the problem of awareness of toxic substances. Nevertheless, the physician must maintain a constant vigilance to lead him to suspect the occupational environment as a possible causative factor.

Occupational History

The physician must be mindful not only of the present occupation of the worker but of former ones as well since a patient suffering from certain ailments may no longer be exposed to the occupational environment responsible for his present condition. In addition, the physician must be alert to those situations where exposures to certain chemicals and other environmental hazards are only occasionally experienced by the worker.

By continued vigilance regarding the occupational history and the hazards encountered, the physician can use these occupational findings more effectively in forming judgments concerning disabilities and in the diagnosis and treatment of disease. With this knowledge and interest the physician can diagnose many previously puzzling and obscure cases. More important is the role the physician can play in preventing the recurrence of the illness by proper reporting and by coordinating concerns about the worker with management and responsible public officials. In this way, the physician may add not only to the knowledge of occupational diseases and disabilities, but also to the understanding of the possible part played by work factors in the development and aggravation of

diseases and disabilities not usually associated with the work environment.

Nonoccupational History

It must be pointed out that in evaluating signs and symptoms it is essential that the physician consider also the possible part played by the nonoccupational environment. For example, the worker may have taken a medicine which might account for the illness. On the other hand, the worker may engage in hobbies after work hours which involve the handling of an injurious agent.

Moreover, the physician in the study of the nonoccupational environment of the worker may find a factor possibly synergistic, or potentiating, in its effect on the hazards presented in the occupational environment. Questions concerning the nonoccupational environment should be routinely raised; in some cases, the information elicited is vital in establishing a diagnosis.

Bases for Diagnosis

Regardless of whether the environment concerned is occupational or nonoccupational, the diagnosis must be based on 1) a meticulously taken history, 2) knowledge of the nature and severity of the exposure, 3) signs and symptoms furnishing corroborative evidence as to its accuracy, and 4) supporting clinical and analytical laboratory tests indicating the extent of the exposure.

Using the Diagnosis

The primary purpose of the diagnosis is to mark the course of treatment necessary for the care of the worker to permit his return to health. But the physician will be remiss if the information coming to him from contact with his patient, as it relates to exposure at the workplace, is not transmitted to those who can best utilize such information in implementing preventive health programs for the protection of all workers.

Regulated Occupational Exposures

The physician and the nurse, as well as the industrial hygienist and other health professionals, must be aware of those occupations in which substances which present hazards to the worker have been covered by mandatory regulations as to extent of exposure permitted. Many of these regulations will provide for a minimum medical surveillance program and will specify certain tests and procedures necessary to the control of the exposure. All details of these surveillance programs have not been included in this text since the information is available in the Code of Federal Regulations, Title 29, Part 1910. Copies of Title 29 are available at most libraries and may be purchased from the U.S. Government Printing Office.

CONTENTS

Three major categories of hazard, chemical, physical, and biological, are presented. Numerous publications and the files of the National Institute for Occupational Safety and Health served as reference sources. The basic compilation from which the text was produced was prepared under Contract HSM-99-73-90 by Tabershaw-Cooper Associates.

In addition to the sections on chemical, physical, and biological hazards, because of their importance in occupational health, separate sections are presented on routes of entry and toxic mechanisms, plant and wood hazards, skin irritants and sensitizers, pneumoconioses, chemical carcinogens, and agricultural chemicals.

Diagnostic Tests

The special diagnostic tests suggested under the various hazards are intended as an aid to the physician with the hope that they will stimulate the use of more detailed textbook or reference material dealing with the test or disease in question. Reference to appropriate mandatory federal standards and recommended threshold limit values are included in some instances, but it must be recognized that the standard setting process under PL 91-596, the Occupational Safety and Health Act of 1970, is a continuing one. The most appropriate and best source for these standards is the previously referenced Title 29 of the U.S. Code of Federal Regulations.

Occupations

Occupations associated with different environmental agents appear in various sections under the heading Potential Occupational Exposures. The word potential is used because it is not to be assumed that the mere presence of an injurious agent will lead to an occupational disease or disability. Much depends upon such factors as severity and duration of exposure, individual susceptibility, and the health protection practices adopted by management and the worker.

When similar activities are performed in similiar or different industries, the same name is used for the occupation wherever possible. In general, the term worker includes both maker and user.

Sources

A section is included which lists sources of consultation on matters pertaining to industrial hygiene and occupational health.

A list of general references on occupational health comprises one section. Specific references are also subjoined to various sections, subsections, and the different chemical hazard items.

Exclusions

Material on treatment generally has not been included since such

information is readily available elsewhere. The prevention and control of health hazards has been given only minor attention because it was felt that this field is adequately covered in other publications, including *The Industrial Environment—Its Evaluation and Control* published by the National Institute for Occupational Safety and Health in 1973.

Special problems such as mental illness, alcoholism, and drug addiction, as well as related areas such as workers' compensation, have not been discussed since they are covered in other publications.

USE OF TEXT

Some of the sections will be followed by a reference list for material cited in the text; other sections will be followed by a bibliographical list for sources of supplementary information. These section lists are supplemented by Section XI.

Since this publication has been prepared primarily as a reference source for professional personnel interested in the prevention, diagnosis, and management of occupational diseases, it is probable that some readers will encounter some areas of little interest and will prefer to exercise their prerogative of judicious skipping. This is recommended. But it is also recommended that care be used to prevent misuse of the data presented. Nonprofessional interpretation of the clinical material must not become a substitute for competent medical consultation.

Inevitably in a compilation of this nature, there is a variety and divergence in style of writing, views, and priorities. These are evidenced in the various sections of the text, and they have been retained in the interest of clarity and emphasis.

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SECTION II

All substances are poisons; there is none which is not a poison. The right dose differentiates a poison and a remedy.

— Paracelsus

ROUTES OF ENTRY AND MODES OF ACTION

Herbert E. Stokinger, Ph.D.

Routes of Entry

There are at least three routes by which industrial substances can gain entry into the worker's body. In order of importance they are inhalation, skin contact, and ingestion. It is obvious that some substances may have multiple routes of entry. These will be noted in the appropriate section.

INHALATION

The adult human lung has an enormous gas-tissue interface (90 square meters total surface, 70 square meters alveolar surface). This large surface, together with the blood capillary network surface of 140 square meters, with its continuous blood flow, makes possible an extremely rapid rate of absorption of many substances from the air in the alveolar portion of the lungs into the blood stream.

Some highly water soluble substances, such as the soluble halogen salts (but not their acids) and soluble chromates (but not chromic acid), may pass through the lung so fast that none can be detected in this organ directly after cessation of their inhalation. On the other hand, there are many industrially important substances which, by reason of their extreme insolubility in body fluids, or their rapid reactivity with lung constituents, remain for extended periods in the lung. They resist complete clearance by phagocytic or other forms of clearance action and may result in irritation, inflammation, edema, emphysema, granulomatosis, fibrosis, malignancy, or allergic sensitization.

Some of the highly reactive industrial gases and vapors of low solubility can produce an immediate irritation and inflammation of the respiratory tract and pulmonary edema. Prolonged or continued exposure to these gases and vapors may lead to chronic inflammatory or neoplastic changes or to fibrosis of the lung. Fibrosis, as well as granulomatosis and malignancy, also may be produced by certain insoluble and relatively inert fibrous and nonfibrous solid particulates found in industry. Indeed, it is now thought that one of the prerequisites for particulate-induced bronchogenic carcinoma may be the insolubility of the particulate in the fluids and tissues of the respiratory tract, which thereby allows requisite residence time in the lung for tumor induction.

GASES, FUMES, VAPORS

The irritant acid gases as a group (the halogen acid gases and the basic oxide fume particulates, vanadium pentoxide fume and copper fume) are examples of direct, fast acting substances in the upper airway passages. Irritation of these passages occurs from these substances at

concentrations only slightly above the industrial air standard. Bis(chloromethyl) ether, on the other hand, is an example of a slow-acting irritant gas. It produces an esthioneuroepithelioma of the olfactory epithelium of the rat at extremely low concentrations (ca. 100 ppb) after several months of exposure. Many of the metal oxides of submicron particle size (fume) produce both immediate and long term effects; the latter may occur in organs and tissues remote from the site of entry. For example, cadmium oxide fume inhaled at concentrations well above the industrial air standard may produce immediate pulmonary edema that can be fatal; in addition, inhalation over many years of the fume at concentrations of a few multiples of the standard can result in eventual renal injury and pulmonary emphysema.

Very soluble gases, such as sulfur dioxide and ammonia at maximum tolerated concentrations (about 20 ppm for sulfur dioxide, up to 500 ppm for ammonia), seldom proceed much farther down the respiratory tract than the bifurcation of the trachea. Indeed these two gases are so soluble that for all practical purposes they are usually completely absorbed in the nasal passages, whereas the highly irritant but less soluble gases, such as nitrogen dioxide, phosgene, and ozone, reach the deeper recesses of the respiratory tract, affecting mainly the bronchioles and the adjacent alveolar spaces, where they may produce pulmonary edema within a few hours. If exposure is of sufficient concentration and duration, emphysema and fibrosis may ultimately develop.

Gases and vapors of low water solubility but high fat solubility, on the other hand, pass through the lung into the blood to be distributed to organ sites for which they have special affinity, provided they do not combine with blood components. Typical of those gases and vapors that exert their principal effects after absorption from the lung are such volatile liquids as carbon disulfide, volatile aliphatic hydrocarbons (the methane series), volatile aromatic hydrocarbons (the benzene series), the volatile halogenated hydrocarbons, and the aliphatic saturated ketones, such as methyl ethyl ketone, alcohols, and glycols.

It should be recognized and taken as a general rule, that each industrial chemical can affect a variety of bodily reactions, depending upon the nature and degree of exposure. For example, single exposures to carbon disulfide at levels several fold above the threshold limit value (TLV), can lead to narcosis and its sequelae; repeated daily exposures for many years at levels a few fold above the TLV can result in effects on the central nervous system (polyneuritis and psychosis) as well as on the cardiovascular system, liver, and kidney. Similarly, certain halogenated hydrocarbon solvents produce narcosis after brief exposures above the TLV; after long repeated daily exposures a few fold above the TLV, they may injure the liver or the kidney. Single, massive exposures to some of these substances can produce pulmonary edema.

ADSORPTION

The toxicologic action of some gases and vapors may be consider-

ably enhanced by adsorption on solid particles. The physicochemical theory for this is that those adsorbed gases which would normally never reach the deeper, more sensitive portion of the lung are carried there in very high concentrations when adsorbed on particles of small size.

There are now a number of instances that appear to confirm the above theory. A 27-fold increase in pulmonary airway resistance occurred from the inhalation of a mixture of the irritant gas, sulfur dioxide, and the inert particulate, sodium chloride, over that produced by sulfur dioxide alone at a concentration of 2.6 ppm. Other irritant gases in a sodium chloride admixture behave similarly. The serious health effects at Donora, Pa., are now attributed to the adsorption of sulfur dioxide on zinc ammonium sulfate. The Los Angeles eye irritation resulted from some unidentified vapors (aldehydes? peroxyacetyl nitrates?) adsorbed on smog particulates. Perhaps the most striking example of enhancement of toxicity from gaseous adsorption is that of radon; essentially no body retention of radon occurs if the inhaled air is dust-free.

BREATH ANALYSIS

If the inhaled gases and vapors are body-fat soluble and are not metabolized, they may be cleared from the body primarily via the respiratory system. Examples of these are some of the well-known industrial organic solvents; the volatile halogenated hydrocarbons; the volatile aliphatic, olefinic, and aromatic hydrocarbons (the methane and benzene series and certain olefinic homologues); some volatile aliphatic saturated ketones and ethers; aliphatic esters of low molecular weight; and certain other organic solvents such as carbon disulfide.

For those industrial solvents that continue clearing from the body in the exhaled breath for several hours following exposure, analysis of the rate of excretion in the breath of the exposed worker offers a laboratory test that may be very helpful in showing not only the nature of the substances to which the worker was exposed, but also the magnitude of the exposure and probable blood levels. By the use of gas chromatographic or infrared analysis of the breath samples, the identification of the substance is established, permitting comparison of the exposed workers' breath decay rate with published excretion curves (1). The physician can then estimate the magnitude of the original exposure. There is, however, considerable individual variation and it is not easy to set standard values.

PARTICULATES

The factors governing the sites of deposition, retention, distribution, and ultimate health effects of solid and liquid particulates obviously differ in most respects from those just mentioned for gases, fumes, and vapors. There are two exceptions, submicron particles ≤ 0.05 μm in diameter, which may act as gases adsorbed on particulates, and liquids adsorbed on particulates.

DEPOSITION

There are four major factors that influence the site of the ultimate toxicologic response to inhaled particulates: 1) the anatomic arrangement and physical dimensions of the respiratory system, 2) the physiologic character of breathing rate and depth, 3) the physical nature of the particle-size, surface area, "solubility," and hygroscopicity, and 4) the biochemical reactivity of the soluble components of the particle.

The knotty problem of handling particles of all sizes, shapes, and densities has been resolved by relating all particles to a median aerodynamic diameter. This is the diameter of a unit-density sphere with the same settling velocity as the particle of concern. The cut-off point for respirable size is conventionally taken as 5 μm expressed as an aerodynamic diameter.

The aerodynamic diameter of particulates determines which particles will or will not present exposure to the respiratory system and gives some indication of the degree of impaction in the various compartments of the respiratory system, and hence the site of particle deposition. Thus, a particle such as uranium dioxide with a high density of 10.9 and diameter of 0.5 μm will behave as a unit-density spherical particle of 1.65 μm, and can be expected to settle out of undisturbed air at the rate of a larger diameter particle, and impact more in the upper respiratory passages than its measured size would indicate. The sedimentation rate of fibers depends on their diameter and is independent of length. Fiber geometry is also important in relation to certain toxicologic properties, for example, in the induction of mesotheliomas.

To simplify calculations of the deposition pattern of the aerosol of concern, the manifold compartments of the respiratory system are reduced to three: the nasopharyngeal, tracheobronchial, and pulmonary (2). If the particulates are assumed to be present as log-normal distributions and three tidal volumes are used, a table can be developed showing the amount of particles deposited in each of the three compartments according to unit-density sizes, ranging from 0.01 to 10 μm. As might be expected, no particles less than 0.6 μm were deposited in the nasopharynx at any of the three breathing rates, whereas practically all of the particles greater than 6 μm were deposited at this site at all breathing rates. Thus, the major site of deposition of the smaller particles is the lung; deposition in the tracheobronchial compartment never exceeded 25 to 30% of the total particles inspired even at the smallest sizes (around 0.01 μm) and the slowest breathing rate (3). Although various degrees of mouth-breathing would upset the calculations of deposition in the upper respiratory tract, it is not believed to affect seriously pulmonary deposition.

Hygroscopicity, however, seriously affects deposition of smaller, highly water-soluble particles by increasing their size as they travel down the respiratory tract in its 95% humidity. Thus, in some instances, it is possible for a small size particle in an atmosphere of low humidity to

so increase its size in the respiratory tract as to alter considerably the deposition pattern characteristic of the entering particle size.

As is true with all generalizations, differences in deposition patterns can be expected, and indeed have been demonstrated, particularly for the lung where ventilation among the five lobes is normally variable.

Another factor which alters deposition patterns is electric charge, commonly associated with particle sizes less than 0.1 μm in diameter, i.e., newly generated fume. Such particles have enhanced nasopharyngeal deposition.

CLEARANCE AND RETENTION

In evaluating the health hazards from inhaled aerosols, four combined physio-chemical actions must be considered: 1) ciliary movement, 2) phagocytosis and lymphatic drainage, 3) direct intercellular penetration, and 4) solubilization or leaching.

Respiratory tract cilia do not extend beyond the terminal bronchioles. Particle clearance by upward ciliary movement takes place from the terminal bronchioles upward to the throat where the particles are transferred to the gastrointestinal tract by swallowing. Thus, exposure of the respiratory tract to particles may also involve exposure of the intestinal tract. With the exception of soluble particles impacting in the nasal passages and being absorbed there, clearance by solubilization from the tracheobronchial passages is not important.

Phagocytosis represents the major mechanism for clearing most particulates from the lung. Moreover, the presence of dusts stimulates the appearance of phagocytes at the site, so that repetitive exposures increase the rate of phagocytosis and hence the rate of clearance of dust from the lungs. Lymph drainage of the dust-filled phagocytes to the lymph nodes represents 2 to 10% of the clearance of the total pulmonary dust burden for certain insoluble oxide dusts.

Direct intercellular penetration offers another clearance mechanism of variable magnitude depending on the solubility, shape, and biologic activity of the dust. Thus, a particle that is not readily coated with serous protein, or other lung constituent, would penetrate the cell and then be cleared by this mechanism more readily than one that is coated.

Obviously solubilization represents the dominant clearance factor for particles readily soluble in respiratory tract fluids. Highly soluble dusts, such as the chromates of the alkali metals, pass through the lungs in a matter of minutes, and even grossly insoluble mineral dusts, such as certain types of asbestos, are subject to leaching of their metals and consequent clearance of these elements from the lung. A partial listing of inorganic compounds according to three pulmonary clearance classifications has been attempted for those compounds cleared in less than one day to 10 days, those requiring more than 10 days to 100 days for clearance, and those greater than 100 days, clearance time being expressed as biologic half-life (2).

While the site and degree of particle deposition is altered by a variety of factors and the magnitude of these factors is largely known, much less is known about the factors governing clearance. Mucociliary action, one of the major factors, varies with any factor or agent that affects ciliary beat or mucus production. They include temperature, humidity, industrial respiratory irritants, and cigarette smoking (4). The amount, site, and frequency of deposition also affect clearance rates indirectly, presumably by stimulating phagocytosis to varying degrees. Clearance rates differ appreciably among individuals (5).

SKIN CONTACT

Upon contact of a substance with the skin, four actions are possible: 1) the skin and its associated film of lipid can act as an effective barrier against penetration, injury, or other forms of disturbance; 2) the substance can react with the skin surface and cause primary irritation (dermatitis); 3) the substance can penetrate the skin and conjugate with tissue protein, resulting in skin sensitization; and 4) the substance can penetrate the skin, enter the blood stream, and act as a potential systemic poison.

Although one of the skin's principal physiologic functions is to serve as a protective barrier against entry of foreign substances into the body (6, 7), serious and even fatal poisonings have occurred from brief exposures of confined areas of the skin to highly toxic substances such as parathion and related organic phosphates, the organometallics, the alkyl leads and tins, and aniline, phenol, and hydrocyanic acid. Abrasions, lacerations, and cuts may greatly enhance the penetration of the skin.

How large a role the skin plays as a route of entry in occupational exposure can be seen by consideration of those substances in the American Conference of Industrial Hygienists' Threshold Limits list bearing the notation "Skin." Of the 579 items (*) in the 1976 Threshold Limit Value (8) booklet, 138 are listed under skin, indicating about one in four industrial substances presents an appreciable exposure via the skin. It should be noted, however, that appreciable exposure via the skin occurs generally from direct contact with undiluted substances, and for the most part exposure is not appreciable at or around the TLV. How important appreciable skin contact can be in industrial exposures is shown by substances, such as benzidine, which have negligible vapor pressure, but are readily absorbed through the skin. For such substances, the skin provides the major route of entry, and it is for this reason that no air standard has been set for these substances. Exposure is controlled by biologic monitoring or engineering and personal protective procedures.

(*) Items, not substances, for two reasons. Some listings refer to groups of substances, e.g., metals and insoluble compounds; others represent typical examples, e.g., substances in Appendices E and F.

ABSORPTION PATHWAYS

Although technically the two main routes of skin absorption are through epidermal cells (transepidermal) and through hair follicles and sebaceous glands (pilosebaceous), the transepidermal is the principal route because of the relatively small absorbing surface of the pilosebaceous units, even though these units offer greater permeability than the epidermal cell layers. Transepidermal absorption is influenced by the superficial barrier lying between the stratum corneum and the uppermost layer of living epidermis.

In general, percutaneous absorption of inorganic substances (electrolytes), including water, is negligible. Only those inorganic substances that are nonionic or ionize very slightly, such as boric acid, certain salts of mercury, and the halides of beryllium, are absorbed to any degree. On the other hand, fat-soluble substances (mainly organic compounds) are absorbed fairly rapidly.

Most substances that are both water- and fat-soluble, e.g., amines and nitriles, penetrate so rapidly that the rate of absorption is comparable to that of gastrointestinal or even pulmonary absorption. The numerous factors that affect absorption of hazardous substances through the skin can be grouped into physicochemical and physiologic.

PHYSICOCHEMICAL FACTORS

Chemical structure and its associated physical property, lipid/water solubility, are major determinants of absorption. Concentration and tissue reactivity, however, can take precedence over other physicochemical factors.

As a general rule, any substance having a caustic effect (such as phenol or cresols) or having a protein-coagulation effect (such as heavy or polyvalent metals), when contacted in high concentration, will be absorbed in relatively smaller amounts than when contacted at lower concentrations that do not cause protein coagulation. Moreover, prior contact of the skin with caustics or astringents decreases the absorption of other absorbable substances. Gases and vapors usually show increasing penetration with increasing concentration. However, skin absorption of gases, at or below the TLV contributes negligibly to the overall toxicity from air exposure. There appears to be a barrier, though relative, to even lipid and water-soluble substances; e.g., pentanediol and diethyleneglycol.

The reasons for the relative barrier effect are not known, but it is possible that chemical combination with skin constitutents may fix such substances *in situ*. Some substantiation for this view is the finding of mustard gas fixed in the epidermis and corium of human skin 24 hours after application (9).

Changes in pH have been shown to aid penetration of some of the few substances that have been measured; certain surfactants are absorbed most readily from buffered solutions with pH values greater than 10.5. On the other hand, certain polyvalent metals are absorbed more readily from solutions of low pH.

Prior application of any solvent that removes cellular lipid, such as benzene, alcohol, or chloroform, increases barrier-cell permeability. An increased penetration of iodine and dyes follows the use of saponin, a cholesterol precipitant. Conditions leading to minimal ionization at skin pH probably lead to greater absorption.

Vehicles, despite common impression otherwise, play a negligible role in increasing percutaneous absorption; substances incapable of penetrating the barrier are not "carried through" by a vehicle. Vehicles do, however, enhance absorption by transappendageal route (7). This is accomplished by diminishing the surface tension between the liquid and the follicular pore and by bringing the substance into more intimate contact with the follicular pore and the hair canal.

Temperature elevation may be expected to increase skin absorption by increasing vasodilatation and thus increasing the rate of transport away from the skin, and by increasing the rate of diffusion.

PHYSIOLOGIC FACTORS

The barrier effect on percutaneous absorption of both the lipid surface film on the skin and the horny layer has been overestimated. The horny layer has large pores that can be penetrated even by large molecular aggregates; the waxy lipid film is miscible with water because it contains cholesterol, its esters and waxes, all of which are emulsifying agents. Hence, the major barrier to absorption lies between the stratum corneum and the uppermost layer of living epidermis.

Human skin shows great differences in absorption at different anatomic regions (10). If the skin of the forearm is used as a frame of reference, the palm of the hand shows approximately the same penetration as the forearm for certain organic phosphates and carbamate insecticides. The dorsum of the hand and the skin of the abdomen have twice the penetration potential of that of the forearm, whereas follicle-rich sites, such as the scalp, forehead, angle of the jaw, and postauricular area have 4-fold greater penetration. The intertriginous axilla has a 4- to 7-fold increase; the skin of the scrotum allows almost total absorption.

The physiologic factors promoting absorption are chiefly due to elevated temperature effects on the skin resulting in hyperemia and sweating (hydration of skin). Hyperemia, which can be caused by some factors besides temperature elevation, promotes skin absorption by increasing the rate of removal of the penetrated substance from the corium by providing greater concentration gradients between skin surface and deeper tissues. Gases and vapors are the substances most affected when in aqueous solution. Hair, an excellent collector of fine dusts, materially increases transappendageal absorption.

SWEATING AND HYDRATION

It has been recognized clinically and observed in industry (11) that absorption of toxic substances is more likely to occur when the clothing that is worn keeps the skin wet than when it keeps the skin dry. Sweat-

ing may increase the skin lipids, suggesting that the absorption of lipid-soluble substances should increase. Sweat is also instrumental in eliciting certain allergic cutaneous hypersensitivities (e.g., from chromium or nickel). As these apply to the metals themselves, it is the acidity of the sweat that leaches small amounts from the metals in contact with the skin to induce the allergic hypersensitivity. Data on the relative magnitude of the increase in absorption from a wet skin are very limited; absorption of ethyl nicotinate was increased by a factor of 6 for a cold-soaked arm, 12 for a hot-soaked arm (10).

ABRADED SKIN

It is evident from skin-stripping experiments that abrasion of the skin dramatically increases percutaneous absorption as far as the substances histamine and Privine are concerned. Differences between intact and abraded skin ranging from 10,000 to 100,000 times were found for both substances (10).

INGESTION

Health hazards to the worker from ingestion of industrial substances in comparison with those from the inhalation and skin contact routes are generally of such a low order as to warrant only limited discussion.

First, the number of substances that can be ingested are fewer as it is virtually impossible to ingest a vapor or gas.* Second, the frequency and degree of contact are very limited; mouth contact with substances on hands, in food, in drink, and on cigarettes is far less frequent, of shorter duration, and lesser in amount during the work shift than that with other routes of entry.

Third, and most important, toxicity by mouth is generally of a lower order than that by inhalation. Reasons for this include: 1) poor absorption into the blood stream; 2) subjection to relatively high acidity (pH 1 to 2) in passing through the stomach; 3) subjection to the alkaline medium of the pancreatic juice on passing through the small intestine. Both these latter may act to reduce toxic organic substances through hydrolysis to less toxic substances. Moreover, the pancreatic enzymes begin to convert (metabolize) some substances to less toxic moieties well before the parent substance is absorbed.

Favoring low absorption also are the following: 1) Food and liquid mixed with the toxic substance not only provide detoxifying dilution, but also frequently reduce absorption because of the formation of less soluble substances resulting from complex interaction with ingested substances in the gastrointestinal tract. 2) There is a certain selectivity in absorption through the intestine that tends to limit absorption of "unnatural" substances. 3) After absorption into the blood stream, the

*Exceptions—mouth-breathing, gum and tobacco chewers can absorb appreciable amounts of gaseous substances during an 8-hour workshift.

toxic material goes directly to the liver which further metabolically alters, degrades, and detoxifies most substances.

It is worth noting, however, that the ingestion route contributes secondarily to the intake of particulates by inhalation. That portion of inhaled material that lodges in the upper parts of the respiratory tract is swept up the tract by ciliary action and is subsequently swallowed.

Although the foregoing statements on reduced toxicity by ingestion in comparison with that by inhalation are true in general, there are obviously striking exceptions. Notable among the exceptions are those highly toxic elements with slowly cumulative action such as arsenic, cadmium, lead, and mercury. Recognition of the potential of such elements to add to the body burden through ingestion has led to prohibiting eating, drinking, and smoking in areas where there are such exposures.

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Modes of Action

Toxic substances exert their effects by physical, chemical, or physiologic (enzymatic) means, or by a combination of these. This classification is consistent with a definition of toxicity which includes irritation, narcosis, tissue injury and disease, sensitization, carcinogenicity, mutagenicity, and teratogenicity. Depending upon the state of development of toxicologic knowledge, the mode of action for a particular substance or group of substances may be described at the level of an organ or tissue, at the cellular level, or at the subcellular, molecular, or free-radical level. In what follows, the lowest level of effect consistent with available knowledge will be used. In some cases, this could lead to erroneous classification; i.e., those mechanisms now regarded as physical may later be shown to be chemical or physiologic.

PHYSICAL

Harmful substances that have a solvent or emulsifying action can produce, after prolonged or repeated contact, a dry, scaly, and fissured dermatitis. This effect is commonly attributed to the physical removal of surface lipid, but may also be caused by denaturation of the keratin or injury to the water barrier layer of the skin. Acid or alkaline soluble gases, vapors, and liquids may dissolve in the aqueous protective film of the eye and mucous membranes of the nose and throat, or in sweat, causing irritation at these sites. Moreover, such insults may erode teeth and produce changes in hair structure.

Irritants

On the inner surfaces of the body, the lungs and gastrointestinal tract, physical contact with unphysiologic amounts of substances causes irritation. This may lead to inflammation, or produce contraction, as in the reflex constriction of the respiratory passages upon inhalation of an irritant gas with resultant coughing or choking. In the upper gastrointestinal tract, the effect may include vomiting and, further down in the tract, the irritation may result in abnormal peristalsis and defecation.

Inert Gases

Inert gases can exert serious and often fatal effects simply by physical displacement of oxygen, leading to asphyxia. Under pressure, inert gases such as nitrogen can produce compressed air illness by dissolving in unphysiologic amounts in the blood, lymph, and intercellular fluids, or may rupture delicate membranes such as the eardrum. Sudden, or too rapid, decrease in pressure results in decompression sickness. Less inert gases such as carbon dioxide and oxygen under greater than atmospheric pressure can lead to narcosis and other more serious effects, such as pulmonary hemorrhage and nerve and brain damage.

Adsorption

Physical adsorption of gases or vapors on solid or liquid particulates (aerosols), may, upon inhalation, lead to physiologic effects out of proportion to that anticipated from their inhaled concentration prior to adsorption. The action is known as synergism when the effect of gas and particulate exceeds the sum of the effects expected from either alone, or antagonism when the effect is less than expected. A physical theory has been developed to explain these abnormal actions. It is based on the molecular properties of gases, and accounts for the synergism by postulating that "adsorbed" layers of the gas on inhaled particulates will carry to the sensitive lung tissue enormously increased concentrations of the gas that become localized, point sources of contact. Synergism or potentiation results when a rapid rate of desorption of the gas from the particulate to the tissue occurs; antagonism, when the desorption rate is very slow or nonexistent, or when chemical combination has occurred.

Radioactivity

Radioactive particles can cause dislocation and breaking of chromosomal linkages, apparently from local energy release, commonly referred to as the "oxygen effect."

CHEMICAL

Few body reactions progress by purely chemical processes. Among these few are the production of acids and bases (chlorides, phosphates, and sodium) and water and the liberation of bicarbonate into the urine.

There are other industrially important types of poisoning which proceed through mechanisms that do not involve the intervention of enzyme action but for which the energy may be supplied by chemical action.

Direct Combination

Among the best known and understood mechanisms of poisoning is that of direct chemical combination of the toxic substance and a body constituent, as illustrated by carbon monoxide poisoning. In this instance, the gas combines rapidly and rather firmly with hemoglobin, forming a new compound, carboxyhemoglobin, which cannot perform the usual function of hemoglobin, transporting oxygen to the tissues.

Hydrogen sulfide likewise unites with hemoglobin to convert it to sulfhemoglobin, a nonoxygen carrying pigment, although this mechanism is not important in hydrogen sulfide poisoning.

Indirect Combination

A less well understood mechanism of injury, but on which there is nevertheless an enormous amount of indirect evidence, is the release by

toxic substances of natural body constituents, such as histamine, in abnormal amounts that lead to injury and even death. Instances of this mechanism are numerous and involve the intake into the body of such common substances as "hay fever" allergens or other biologic allergenic materials, raw cotton dust and subtilisins, for example. Prominent industrial chemical allergens are the organic isocyanates which act as haptenes with body proteins to become allergenic.

Intake of these substances results in release of histamine or histaminelike substances locally in large amounts with characteristic development of inflammation, edema, and other evidences of injury. Certain types of amines are capable of histamine release; in these instances the mechanism involved is believed to be one of displacement, whereby the tissue-bound histamine is displaced and liberated by the unnatural amine. Similarly, any type of simple cellular damage such as caused by respiratory irritants, e.g., ozone, results in the liberation of histaminelike substances.

There is accumulating evidence also that release of hormones from nerves may be the common mechanism by which a number of chemical substances exert their toxic action.

Chelation

A toxic mechanism that is increasingly being recognized to be one of the more common pathways of toxic action is chelation. Chelation is the term applied to the chemical combination of an organic substance and a metal whereby the metal is very firmly bound to the organic substance by both nonionic (organic) and ionic bonding. For example the therapeutic agent EDTA (ethylene-diamine tetraacetic acid) binds metals by chelation. Many drugs and antibiotics are now believed to act by chelation. By so acting, these substances exert their effects in a number of ways:

1) By removing biologically active metals that are normally bound in the cell or its components with resulting inactivation and cell damage. For example, treatment of lead poisoning with EDTA to remove lead may in addition remove other metals, such as zinc, that are required for important functions in certain kidney enzymes (e.g., carbonic anhydrase).

2) By reacting with fixed intracellular metals.

3) By chelating firmly with a fixed tissue constituent. This is believed to be the mechanism by which boron, as borate, exerts its toxic action. Borate is known to chelate with adjoining carbon atoms containing hydroxyl groups. If the structure prior to chelation happens to be a critical one in a metabolic chain, ordinary function ceases and injury occurs as a result of the altered chelated structure.

4) By increasing the absorption of a toxic agent. Instances are being recognized of toxicity resulting from abnormally increased amounts of absorption into the blood stream by a chelating compound. Iron, normally nontoxic when absorbed by the usual regulatory mechanism, may under unusual circumstances be absorbed in toxic amounts by the mechanism of chelation to form a soluble, easily absorbed substance.

TOXIC MECHANISMS

Of the three modes of action of toxic substances in the body—physical, chemical, and physiologic—the physiologic mode is most common.

Enzymes, the biologic catalysts of the body, estimated to total one million (1000 genes each governing an estimated 1000 enzymes), control the action of toxic substances. In what follows, an attempt is made to summarize the multitudinous ways that enzymes are involved in handling toxic substances following their entry into the body.

The toxicity concept can best and most simply be understood as the net result of the following two opposing reactions:

Reaction I — The toxic agent acts on the body.
Reaction II — The body acts on the toxic agent.

The net result of these two opposing reactions is the toxicity that is observed in any animal species. This is diagrammatically represented by Figure 1. The upward-pointing arrows are indicative of Reaction I; the downward-pointing arrows, Reaction II; arrows pointing in both directions are indicative of the known fact that in some instances, the homeostatic and adaptive Type II instead of providing beneficial reactions and reducing toxicity, actually result in harmful reactions and hence increased toxicity. In a typical example the "detoxication" of pyridine by methylation results in methyl pyridinium chloride which is eight times more acutely toxic than pyridine. See Figure 2.

Many toxic agents (insecticides, carcinogens, and drugs) stimulate liver enzyme activity, which in turn accelerates destruction of the toxic agent. Also, the production of immune bodies tends to counteract the action of a harmful substance (Reaction II) and hence reduces toxicity.

Chelation and combination represent a toxicity-reduction mechanism limited to metals. For example, the body's methallothionein chelates firmly with cadmium (and zinc), removing it to less sensitive sites and reducing its toxicity. The limiting factor in such toxicity reduction is the body's methallothionein reserves and capacity for methallothionein induction. Combinations of trace elements can be so effective a means of detoxication as to entirely antagonize the toxicity of highly toxic elements; e.g., an in vivo combination of mercury with selenium (and sulfur) can remove all traces of mercury toxicity.

The foregoing reactions result in an observed toxicity that is but a small fraction of the maximal toxic potential for those substances which react less than instantaneously. Obviously, Reaction II cannot occur with those substances that react on contact, e.g., unstable or highly reactive irritants such as ozone, nitrogen dioxide; or with high concentrations of those substances that immediately overwhelm these body defenses before they have time to come into play, such as high concentrations of the general asphyxiants, hydrogen sulfide, hydrogen cyanide, and carbon monoxide.

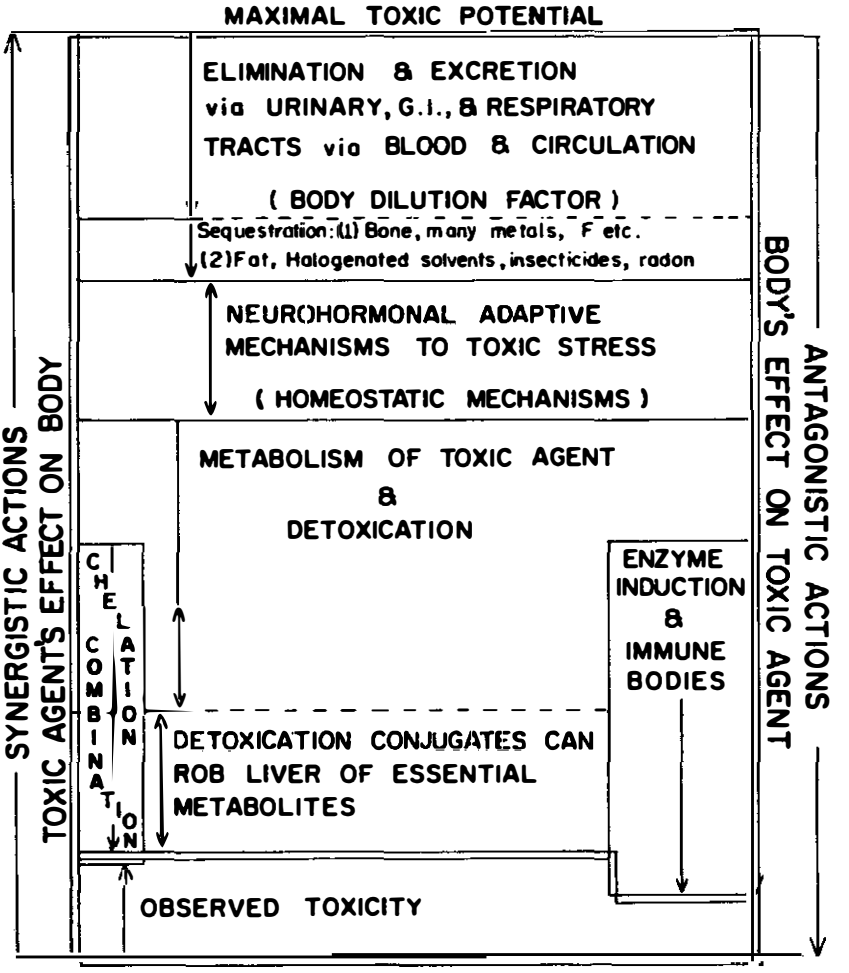


Figure 1. Diagrammatic concept of toxicity.

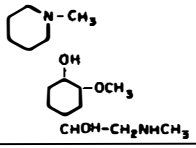
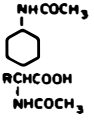
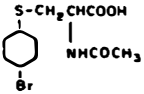
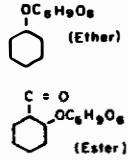
Type	Toxic substance	Detoxication product examples
Methylation $-CH_3$	Inorg. compounds of As, Te, Se. Ring N compounds Certain complex aromatic phenols	$(CH_3)_2$ Se 
Acetylation CH_3CO-	Aromatic amines Amino acids (Known exceptions: aromatic amine carcinogens, also aliphatic amines.)	 e.g., benzidene-hydroxylated aliphatic amines - aldehydes.
Ethereal sulfate $-OSO_3H$	Phenols (Cyclohexanol glucuronide)	
Acetyl Mercapturic acid $-SCH_2CHCOOH$ $NHCOCH_3$	Aromatic hydrocarbons Halogenated aromatic HC's Polycyclic HC's Sulfonated esters $C_2H_5SO_3-CH_3$ Nitroparaffins ($C_4H_9NO_2$)	 C_2H_5- acetyl cysteyl- C_4H_9- acetyl cysteyl-
Thiocyanate	Cyanide, inorganic Organic cyanides (Nitriles)	RCNS
Glycine $-NHCH_2COOH$	Aromatic acids Aromatic-aliphatic acids Furone carboxylic acids Thiophene " " Polycyclic " " (Bile acids)	
Glucuronoside	Aliphatic (1^0, 2^0, 3^0) and aromatic hydroxyl Aromatic carboxyl	 (Ether) (Ester)
Glucose hydrazone	Hydrazine Hydrazine derivatives ?	$NH_2N = CHC_5H_9O_5$

Figure 2. Major types of detoxication.

METABOLISM

Metabolism or biologic transformation of the toxic agent is one of the prime determinants for toxicity. These bio-transformations can be classified into three types of destructive transformation (oxidation, reduction and hydrolysis), synthetic transformation (conjugation) and enzyme induction.

OXIDATION

Oxidation is the most common form of bio-transformation reaction that occurs in response to a toxic substance. It includes the oxidation of alcohols and aldehydes to corresponding acids, the oxidation of alkyl groups to alcohols, the oxidation and hydroxylation of carbon ring compounds and ring-splitting, oxidation deamination of amines, oxidation of sulfur compounds to sulfoxides and sulfones, and dehydrohalogenation, all of which result in the body's excretion of highly oxygenated acids or acidic substances.

Although the intimate mechanism of oxidation is not precisely known in all instances, two general types are recognized: oxidation by direct addition of oxygen to the carbon, nitrogen, sulfur, or other bond; and oxidation by dehydrogenation. In either case, the energy required for the action is supplied by enzymes, resulting in free-radical reactions for the most part.

One of the most important examples in which oxidative mechanisms play a decisive role in the ultimate toxic response is the recently discovered metabolic formation of arene oxides. Arenes is the general term that applies to all aromatic nuclei including benzene and the polynucleated hydrocarbon carcinogens such as benzpyrene. The oxidation proceeds by the addition of an oxygen atom to two adjacent carbon atoms in the aromatic ring forming the so-called epoxide group. These epoxides are very unstable and highly reactive, and they, rather than the parent hydrocarbon, are believed to be the initiators of tumor production. In addition to subsequent isomerization to phenols, epoxides readily react with cellular macromolecules such as DNA, RNA, and protein, believed to be essential elements in tumorigenesis.

REDUCTION

Reduction of foreign substances is a less common body function than oxidation. It does occur, however, for those substances whose oxidation-reduction potential exceeds that of the body, such as nitro groups, certain aldehydes, certain oxidized forms of metal ions, and in certain reactions such as hydrogenation of carbon and nitrogen double bonds and reduction of disulfides to sulfhydryl derivatives.

In this connection, it must be noted that the entire body metabolism operates on an oxidation-reduction (O-R) system, poised at about the

potential of Vitamin C, which assumes the obvious fact that for each oxidation of a *natural body constituent,* there must be a reduction. Whether a toxic substance will be reduced depends upon its O-R potential relative to that of the body.

HYDROLYSIS

This reaction, which involves cleaving of a bond with the addition of water, is another means by which the body metabolizes and degrades toxic substances. If organic in nature, hydrolysis is performed by enzymes; if inorganic, by simple chemical action, as beryllium sulfate hydrolyzes to the (colloidal) hydroxide on entering the body.

Typical foreign substances that undergo hydrolysis are esters of all types; compounds of carbon, nitrogen, sulfur, and phosphorus, resulting in the formation of component acids and alcohols; amides which are hydrolyzed less well to the corresponding acid and ammonia; and ethers which are split by different enzymes, depending upon the nature of the alkyl group (the rate of splitting of aromatic ethers depends upon the nature and position of the substituents on the ring). Simple aliphatic ethers are excreted unchanged mainly via the respiratory tract; however, more complex ethers such as benadryl are split.

CONJUGATION

Synthetic mechanisms directly involved in the normal metabolic processes provide major pathways for disposing of toxic substances. These synthetic pathways involve conjugation of the toxic substance (any that the body cannot readily oxidize to carbon dioxide and water) with a limited number of defined body constituents. Figure 2 shows eight major types of conjugation and examples of the types of substances involved in each conjugation. These conjugation reactions, performed enzymatically, involve carbohydrate (glucoside formation), amino acids, methyl and acetyl groups, and sulfur derived indirectly from sulfur-containing amino acids.

These conjugates have two important properties essential for detoxication. They are, in general, considerably less toxic than the parent substances (with certain exceptions) and they are readily excreted in the urine. The well-known synthesis of phenylsulfate, which was one of the earliest synthetic mechanisms to be discovered (1876), converts highly toxic phenol to a substance which is practically nontoxic. Cyanide, both inorganic and organic forms, is synthesized to thiocyanate, a structure many times less toxic than cyanide. Certain toxic metal ions may react with sulfur of the body to be excreted as insoluble, and, thus nontoxic, metal sulfides.

It should be pointed out that these synthetic detoxifying mechanisms are not entirely free of injury to the body. In contributing some of its constituents, the body may deprive itself of vital amounts of these substances if synthesis is prolonged, and thus injure itself (Figure 1).

DETOXICATION AND BIOLOGIC INDICATOR

There are several important benefits to be derived from a knowledge of detoxication mechanisms. Such knowledge offers a biologic means, through analysis of appropriate body fluids or excreta, for positively identifying the agent to which the worker was exposed. If the analysis is performed quantitatively, it permits a reliable estimate of the degree of exposure. This capability permits, in turn, the development of biologic threshold limits for the control of worker exposure on an individual basis, because biologic estimates of exposure take into account absorption of the industrial substance by all routes, personal work habits, and hereditary characteristics. Furthermore, biologic analyses help evaluate the extent of the worker's exposure to a new product.

ENZYMES

The ultimate regulators of metabolism are enzymes and their associated factors of trace elements, vitamins, hormones, and antimetabolites. Substances that act chemically to produce injury to organs and tissues of the body usually do so by two basic means: either by depressing or by stimulating the activity of the enzyme systems. Severe, acute effects such as destruction of cell membrane integrity by corrosive agents or protein coagulants, etc., are obvious exceptions. A single substance may have more than one pathway and site of action. Multiple pathways of action may be invoked simply by differing doses of the toxic agent; low doses may stimulate enzyme action, high doses depress and inhibit the same or different enzyme systems. This is a characteristic action of most, if not all, toxic substances, including arsenic, benzene, chloroform, cobalt, fluoride and vanadium. A number of aspects of toxicity are shown in Figure 1.

1) Systemic toxicity is, by and large, a matter of the activity of enzyme systems, either by inhibition or overstimulation (removal of a natural inhibitor system), all accomplished at the free-radical level.

2) Substances display differing toxicities and have selective sites of action because different substances affect, to differing degrees, the various metabolic compartments and, thus, raise or lower the level of "observed toxicity." Different substances have differing chemical affinities for tissue sites.

3) Potcntiation and synergism, the enhanced toxicity of two or more simultaneously acting substances, can be explained by the action of one preventing the elimination or the metabolism of the other, wholly or in part, thus maintaining elevated systemic levels of the toxic agent, resulting in an observed toxicity greater than the addititive toxicity of the combined components (Figure 1).

4) Antagonistic action is explained by one component preventing, wholly or in part, the toxic action of another. This occurs when one component induces or supplies additional amounts of a critical enzyme system or factor that is being attacked by another component, the net result being to greatly reduce or even completely eliminate toxicity. A

similar mechanism appears to explain the antagonism of ethyl alcohol for methyl alcohol toxicity. In this case the liver alcohol dehydrogenase preferentially attacks ethyl alcohol, thus slowing down or preventing the oxidation of methyl alcohol to neurotoxic metabolites (Figure 1).

As mentioned previously, most of the metabolic activity of the body is a result of the activity of enzymes, which are biologic *catalysts* formed by living cells throughout the body. Consequently, it is reasonable that the bulk of all toxic mechanisms should involve interference in some way with normal enzyme activity.

All enzymes have a basic protein structure composed of 20 or more amino acids grouped in various chain arrangements in a three-dimensional structure. To perform the myriad of metabolic reactions of the body requires an estimated million diverse enzymes. This diversity of structure and function makes any simple classification inadequate. However, just as the major types of metabolism and detoxication were classified (Figure 2), so can the major metabolic reactions catalyzed by enzymes be classified.

CLASSES OF ENZYMES

The enzymes that perform oxidation-reduction reactions constitute one of the larger groups. The oxidases, which reduce the inhaled oxygen carried throughout the body by hemoglobin and myoglobin, reduce oxygen directly. One of the most important of these is cytochrome oxidase. Other important oxidases are xanthine oxidase with riboflavin as a prosthetic group, the polyphenol oxidases, with copper as prosthetic group, and tyrosinase responsible for the oxidation of tyrosine to the dark melanin pigment.

Closely related in action are the dehydrogenases, which catalyze the removal of hydrogen, and thus "oxidize" organic molecules. As body oxidations generally (respiration) proceed in this manner, there are several highly specific dehydrogenases. All cellular respiration involves three major classes of dehydrogenases: 1) pyridine-linked dehydrogenases, which require a dinucleotide as coenzyme, 2) flavin-linked dehydrogenases, which contain a flavin nucleotide, and 3) the cytochromes, which contain an iron-porphyrin ring system. More than 150 of the pyridine-linked dehydrogenases are known. One of these, glucose-6-phosphate dehydrogenase (G-6-PD), features prominently as the key system in rendering a worker hypersusceptible to hemolytic industrial chemicals. A genetic deficiency in G-6-PD can make a person susceptible to incurring a hemolytic crisis from exposure to such chemicals by either blocking the action of certain components of the G-6-PD system in the red cells or by the chemical's utilizing the hydrogen critically needed for cell respiration, resulting in loss of red cell integrity, and consequent cell lysis.

Another large and diversely acting group are the hydrolytic enzymes, chief among which are the phosphatases, which hydrolyze esters of phosphoric acid. These enzymes are involved in all catabolic (destruc-

tive) and anabolic (synthetic) reactions of the cells. Other representative hydrolytic enzymes are the esterases, such as liver esterases and pancreatic esterases. Others in this group are those that hydrolyze protein structures, proteolytic enzymes that break the common peptide bond of these structures. This group is further comprised of more specialized enzymes, the peptidases, the carboxy- and aminopeptidases, so named because of action on peptides with adjacent carboxyl (COOH) or amino (NH_2) groups; those that hydrolyze glycosidic linkages, the carbohydrases, which act on polysaccharides and glycosides.

The decarboxylases are a widespread group composed of keto-acid decarboxylase, which is responsible for the liberation of the end product of metabolism, carbon dioxide. Amino acid decarboxylases are responsible for the formation of amines by carbon dioxide liberation from amino acids. In the chain of metabolic end reactions, oxidative deaminating enzymes remove the amino group from these toxic amines, be they endogenous or of foreign origin, resulting in reduced toxicity, liberation of the end product, ammonia, and its excretion in the urine. Some of the ammonia, however, is transferred to other substrates by transferases. These transferases can also transfer other groups such as methyl, phosphate, and amino groups.

The above classes of enzymes, with other enzymes not classified, represent all the metabolic catalysts the body can muster to handle foreign chemical structures. As these structures may vary from closely similar to remotely related to the natural substrates of these enzymes, it is not difficult to see that destruction of a foreign toxic substance can range from nearly complete to scarcely perceptible.

ENZYMATIC ACTION

Enzymatic actions occur throughout the body without restriction to any particular organ site, although the liver cells perform a major proportion of the metabolic activity of the body. Similarly active, however, but less diversified, are the enzymes in the lung, kidney, intestine, brain and nervous tissue, and bone. From this, it may be inferred that enzymatic mechanisms may occur with the enzyme situated at cell surfaces or within the cell itself. *Although the activity of enzymes, in normal circumstances, occurs within or on cells which are inaccessible for measurement (except as biopsied tissue), toxic injury to cells may result in enzyme release in proportion to the injury into the blood and body fluids where they can be measured and serve as biologic indicators of exposure and/or response.*

In "metabolizing" a foreign substance, it is important to observe that the enzyme is merely performing a function that it normally performs in metabolizing natural foodstuffs; no special enzymes exist to metabolize toxic substances. Although "drug-metabolizing" enzymes are commonly mentioned, this does not mean that the body develops a new class of enzymes in response to the administration of a drug; drugs may, however, act to induce larger amounts of enzyme activity.

ENZYME CHARACTERISTICS

It is now recognized that certain enzymes, heretofore considered homogenous in composition and in action, may consist of several distinct components, each still acting, however, on the same substrate; these components are called isoenzymes, or isozymes. Isozyme components can differ in number and activity, depending on the tissue of origin, e.g., lactic acid dehydrogenase has as many as five different isozymes, depending upon whether originating from the heart, kidney, liver, or lung.

Many enzymes have additional specificity requirements, in that they require a metal or a vitamin, or both, as cofactor(s) or activator(s). For example, the enzyme cocarboxylase that splits carbon dioxide from certain organic acids, requires vitamin B_1 and magnesium ions as necessary constituents before it can function.

Because enzymes are proteins, they exhibit the physical and chemical properties of proteins. They undergo denaturation 1) by heat, as in burns; 2) by marked changes in acidity or alkalinity as effected, for example, by contact with corrosive agents; or 3) by chemical denaturing agents, such as urea in high concentrations. These agents alike cause structural and configurational changes in the protein, and the characteristic specificity is lost, and with it the catalytic activity of the enzyme.

Enzyme activity can be inhibited in a number of ways. For example, among the enzymes requiring a specific metal as activator, any agent that will displace or render inactive the metal will render the enzyme inactive to the degree that the metal was rendered inert or removed from the enzyme. Certain metals with similar spatial requirements for the specific metal required by the enzyme may do this. Certain poisonous metals such as beryllium are believed to act in this way. Cyanide may combine with the iron of an iron-dependent enzyme and inactivate or inhibit the enzyme.

Another common way an enzyme may become inhibited is from competition with a substance whose structure is sufficiently similar to the natural substrate, but does not quite fulfill the spatial requirements of the enzyme. This is probably the most common way in which toxic substances exert their effect on enzymes. Common examples are the competitive inhibitive actions of the various closely related organophosphate pesticides.

A third way by which enzyme activity is inhibited is by accumulation of the product of the enzyme's activity. This is one of the natural ways by which body enzyme activity is regulated and is known as metabolite inhibition.

The fundamental aspects of enzyme activity with respect to toxicity may be summarized as follows. Enzymes combine with the toxic substance. This combination may result in partial or complete inhibition of enzyme activity or the enzyme may act on the toxic substance more or less incompletely, possibly with the production of even more toxic substances, but generally with production of degraded, less toxic substances. If the enzyme whose activity is blocked is a critical one, there may be

slowing down of some vital function, resulting in alteration of cellular constituents in amount or type, even in cell death.

DIRECT COMBINATION

The simplest way by which a toxic substance can alter enzyme action is by direct combination of the substance with active groups on the enzyme structure. Such is believed to occur with metals such as mercury and arsenic which combine so tightly with the active group of the enzyme that further action is blocked. If the enzyme or enzymes represent critical systems for which there is no shunt mechanism, then cells may die or function subnormally resulting ultimately in injury to the cell, the organ, and the host. Similarly, nonmetallic substances such as cyanide can combine with and block the action of heavy metalbearing enzymes because of the production of an inactive metal-cyanide enzyme. The blocking of this enzyme system to a significant degree results in the well-known fatal cyanide poisoning.

Another mechanism of poisoning by direct combination is illustrated by substances such as ozone and nitrogen dioxide, and possibly iodine and fluorine, that destroy enzymes by oxidation of their functioning groups (Figure 3). In these cases, specific chemical groups such as -SH and -SS- on the enzyme are believed to be converted by oxidation to nonfunctioning groups; or the oxidants may break chemical bonds in the enzyme leading to denaturation and inactivation.

One of the more commonly encountered enzyme inhibition mechanisms of direct combination in occupational exposures is that of the inhibition of the action of cholinesterase (acetylcholine esterase), an enzyme that regulates nerve-muscle action by destroying the muscle excitor acetylcholine. This muscle excitor is a powerful pharmacologic substance, which, if not destroyed when it is free, can act as a poison. The destruction is accomplished by the hydrolysis of the potential poison into its components, an acetyl group and choline. A large number of pesticides, chiefly organic phosphates and carbamates, act in the body by blocking this enzyme action, thus allowing excessive amounts of the muscle stimulator to accumulate. The excessive stimulation results in paralysis of the host.

COMPETITIVE INHIBITION

A second, and one of the more usual toxic mechanisms involving enzymes, is that of competition of the toxic substance with normal metabolites, or the cofactor(s) essential for enzyme action, for the site of action on the enzyme. This form of competition is highly effective, and thus injurious, only when the chemical structure of the competing toxic substance resembles that of the constituent normally used by the enzyme; the closer the structural similarity, the more effective the competition.

The successful competition of an unnatural or foreign toxic substance for the enzyme sites of action blocks normal action by not permitting either significant amounts of normal substances to be metabolized, or by preventing combination of a cofactor necessary for enzyme action.

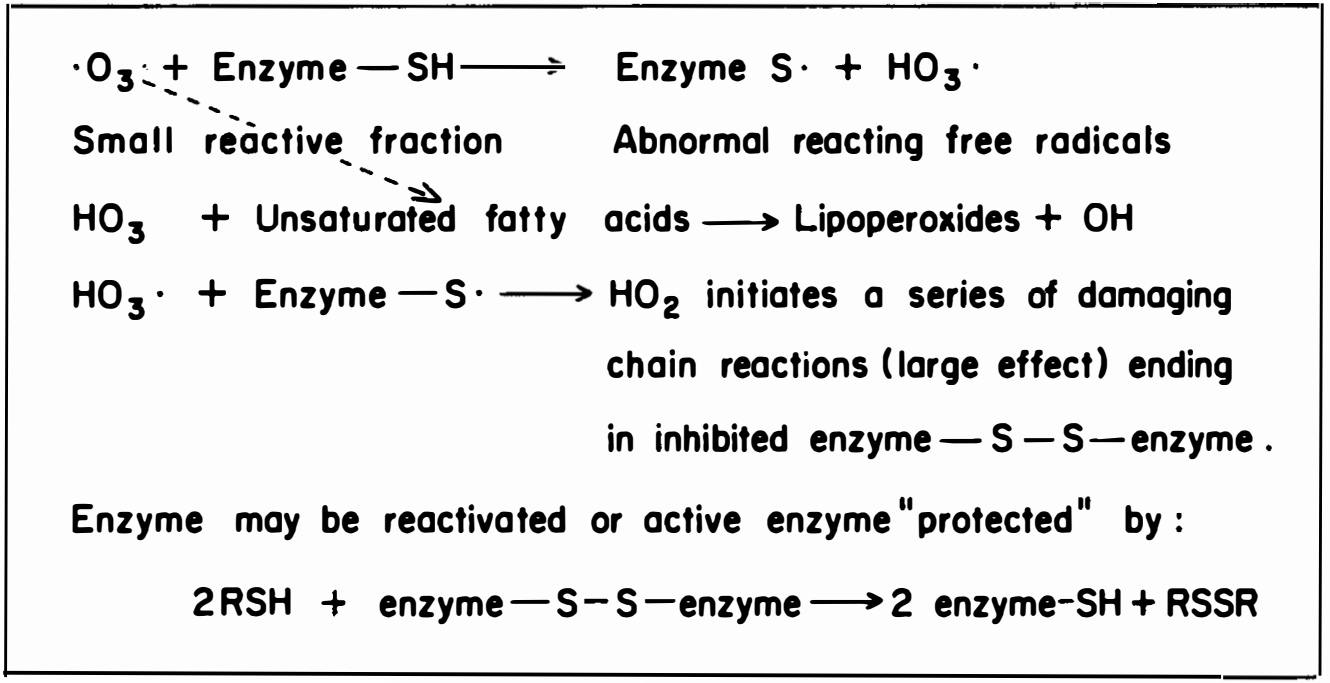


Figure 3. Ozone-initiated free radical chain reactions in respiratory tract.

The cofactor can be a metal or a highly complex specific organic substance such as a vitamin.

Competitive inhibition, first shown to be the action of sulfanilamide by reason of its close similarity to the B vitamin, para-aminobenzoic acid, has been demonstrated to function similarly in many other drug actions; it is also the basis of the mechanism of action of a number of anticancer drugs, many of which are appreciably toxic, for example, the fluoropyrimidines.

Toxic mechanisms may operate also by metal-to-metal competition. For example it is believed that the poisonous action of beryllium results from its capacity to compete effectively for the sites of combination of magnesium and manganese on critical body enzymes, by which action the enzyme is no longer able to function at its normal rate or may be inactivated completely. This competitive inhibition of foreign metals is a very general way by which metals exert their toxic action.

A highly interesting example of a competitive mechanism is that recently found to explain the increased toxicity sustained following simultaneous exposure to two structurally similar economic poisons, malathion and EPN. EPN is highly toxic while malathion has a far lower order of toxicity. When the two substances are present in the body together in sufficient quantity, however, EPN prevents the elimination of malathion, maintaining its concentration, and raising its toxicity, so that the summated toxicities of both are far beyond expectation.

Inasmuch as both substances have chemically similar structures, EPN effectively competes for the same enzyme that hydrolyzes and thus would otherwise reduce the toxicity of malathion. By inhibiting this enzyme action, the concentration of the toxic form of malathion is maintained at a high level in the body, and consequently the toxicity is enhanced.

This is not an isolated instance of such a competitive mechanism. A number of other combinations of economic poisons are believed to produce enhanced toxicities by similar mechanisms, for example, the combinations malathion and Dipterex , and Guthion and Dipterex .

LETHAL SYNTHESIS

Another means by which enzymes are involved in toxic mechanisms concerns the synthesis of a new toxic product by enzyme action on the toxic substance originally taken into the body. The newly synthesized product then exerts its toxic effect by interfering with normal metabolic processes.

A striking example of a substance involved in this type of mechanism is the rat poison 1080, sodium fluoroacetate. After it is absorbed into the body, an enzyme transfers the fluorine atom in fluoroacetate so as to form fluorocitrate from citric acid, an important intermediate in the cycle of terminal metabolism. This fiuorocitrate is unable to function to a significant degree in this important metabolic cycle and breaks the metabolic chain of activity, with the result that tissue respiration ceases, and death ensues.

TOXIC ENZYMES

A rather unusual type of toxic mechanism results when the toxic substance itself is an enzyme. Toxic enzymes are associated with the introduction into the body of substances such as snake and bee venoms and bacterial toxins. Although these substances exhibit a variety of toxic manifestations, the mechanisms of some of which are as yet unknown, the venoms of bees and certain snakes possess enzymes (phosphatidases) that lyse red blood cells, destroying the oxygen-carrying power of the blood, as well as enzymes (proteolytic) that destroy cells and inhibit blood coagulation. In addition, bee venom contains a substance that inhibits the dehydrogenases, enzymes important in the metabolism of many body functions.

INDUCIBLE ENZYMES

Thus far all of the mechanisms discussed have been depressant in action, but the response to toxic substances may under certain conditions stimulate metabolic activity. Inducible (adaptive) enzymes are those enzymes which permit the physiologic synthesis of additional amounts of the enzyme in response to the presence of an inducing agent. In this instance, the inducing agent may also be toxic.

Because inducible enzymes are difficult to demonstrate in the mammalian host (although a number have been so demonstrated in bacteria and yeasts), only few instances of industrial health interest are presently known in detail. High sucrose diets fortified with vitamins fed for 3 weeks to rats stimulate the enzymatic production of additional amounts of protein sulfhydryl groups in the kidney, which enables the rats to withstand otherwise lethal doses of mercury. The newly-formed sulfhydryl binds the mercury firmly, thus effectively reducing its toxic potential. High sucrose diets similarly protect against ozone lethality.

A mechanism exemplifying stimulation, probably mediated through inducible enzymes, is the increased production of serum alpha globulins by cobalt when absorbed into the body at relatively low levels of intake. At slightly higher levels of intake, cobalt stimulates the production of increased amounts of red blood cells (polycythemia production); associated with the polycythemia is increased production of hemoglobin. The exact mechanism of this stimulation is not known, but a hormone, erythropoietin, whose production is stimulated by cobalt, is believed involved. It appears also that the action of erythropoietin is not entirely restricted to stimulating bone marrow to increased production of red cells, but may include stimulation of other centers as well.

SECONDARY ENZYMATIC MECHANISMS

In this category are grouped those pathways of metabolism and mechanisms of injury that are not effected by the direct action of the toxic substance but develop either 1) as a result of metabolic alteration of the toxic substance following its entrance into the body or 2) as a consequence of an accumulation of toxic by-products from the initial,

direct action of the toxic substance. In the second instance, further injury occurs at a site in the body different from that of the original toxic action. Most, if not all, of the mechanisms considered here are performed by enzymes.

The body does not always act to its own advantage when handling a foreign, and sometimes toxic, substance. These peculiarly disadvantageous reactions result, however, merely because the body is equipped with certain definitive pathways of metabolism derived from the biochemical processes concerned with the utilization of food components or other environmental substances. These are its only resources when confronted with nonfood substances, and accordingly, these mechanisms are used insofar as they can act on foreign substances bearing chemical structures similar in some respects to food substances or other natural environmental materials. Whether this indiscriminate action by the body's enzymes results in an outcome favorable or unfavorable to the body depends only on the nature of the resultant modified foreign substance and not on any selective or guided action of enzymes.

METABOLISM

Mechanisms grouped here comprise all those metabolic activities that the body performs on a toxic substance in contradistinction to the actions that the toxic substance performs on the body. Broadly, the so-called "detoxication" mechanisms are those performed by the body, whether as a primary or secondary mechanism, in the process of attempting to eliminate the toxic substance, namely, oxidation, reduction, and synthesis. A few examples of each of these as a secondary mechanism will be given for well-known industrial substances of a toxic nature.

Oxidation

An example in which secondary oxidative mechanisms are believed to play a dominant role in the toxicity of an alcohol is that of methyl alcohol. Oxidation to formaldehyde, which subsequently interferes with oxidative enzyme synthesis, is believed to be the pathway by which methyl alcohol exerts its injurious effect on the optic nerve leading to blindness. Ethyl alcohol, and presumably other alcohols, proceed through this metabolic pathway of oxidation to the corresponding aldehyde, which is responsible, in part at least, for the toxic effects.

In this connection, it should be recognized that by no means do all metabolic alterations in the structure of toxic organic substances result in toxic by-products. A sizeable number of the metabolic products are detoxified in the process.

A striking example of the role of oxidative mechanisms in developing toxicity of organic substances is seen in the organic thiophosphate insecticides such as parathion. These substances, containing sulfur in the molecule, are relatively nontoxic until oxygen replaces the sulfur forming the "oxones" which are extremely toxic, completely inhibiting an important enzyme of nerve function, cholinesterase.

An example of oxidation among inorganic toxic substances is that

of uranium. The tetravalent form is unstable to the body's oxidation-reduction potential, and is oxidized to the more toxic hexavalent form. The hexavalent form then combines with active sites (phosphate groups) on the surface of cells, blocking normal metabolic processes necessary for cell survival.

Much, if not all, of the toxicity of the long-recognized poisoning action of aniline arises not from aniline itself, but from its various oxidation products formed in the body. The more important of these are para-aminophenol and, by further oxidation, the quinoneimine which is believed responsible for the methemoglobinemia that develops when aniline, or other aromatic amines, are absorbed into the body. The oxidized product of aniline oxidizes the ferrous iron of hemoglobin to the ferric form, resulting in methemoglobin, which is incapable of releasing oxygen.

Reduction

Although reduction is far less common a body function than oxidation, nevertheless several types of foreign organic substances are metabolized by this pathway to produce one or more substances that are more injurious than the parent substance. Among certain of the inorganic metal ions, reduction is also the pathway of metabolism. Organic nitro-groups are reduced by stages to amines. Some aldehydes are reduced to alcohols.

Unsaturated double bonds of carbon compounds may add hydrogen and thus become reduced. These types are not an exhaustive listing.

In general, however, reduction, contrary to oxidation, tends to result in products that are less toxic than the original substances, for example, reduction of aldehydes to alcohols, and are thus of lesser interest here. On the other hand, metabolism of nitrobenzene results in a number of products, one of which, para-aminophenol, is from 50-80 times more acutely toxic than the parent nitrobenzene.

Among inorganic ions, pentavalent arsenic is relatively inactive in the body until reduced to the trivalent state. The physiologically active form of manganese is trivalent. If manganese is taken into the body in the form of pyrolusite in which the manganese is tetravalent, reduction to the active form must occur, at least to that portion which is absorbed into the blood stream and later incorporated into active tissue components.

Synthesis

Synthesis is one of the more common means whereby the body contributes some tissue constituents in the conversion of the foreign substance to a new product, thereby disposing of the toxic agent. There are more than a dozen known synthetic mechanisms to accomplish this.

It should be pointed out, as noted previously, that these synthetic detoxifying mechanisms are not entirely free of injury to the body. In contributing some of its constituents, the body may deprive itself of vital amounts of these substances if synthesis is prolonged, and thus injure itself.

SECONDARY ORGAN INVOLVEMENT

A secondary mechanism of very general nature, and of considerable toxicologic importance, involves the indirect action of either the toxic agent or its metabolic by-products, or both. Once having injured a primary site, the substance may cause either the production or accumulation of deleterious products that in turn affect a secondary site.

A striking example of this secondary mechanism is the action of hexavalent uranium, which first injures the kidney in such a way as to prevent normal elimination of waste products such as urea, ammonia, and other substances. These products accumulate in the blood stream and injure the liver, resulting in fatty degeneration of this organ.

Similar indirect injury occurs when the lung, through direct injury by some toxic substance, restricts blood flow thus placing undue stress on the heart (cor pulmonale).

There are numerous other examples; in fact, the function of the body is so organized that there are few alterations of significant magnitude in an organ or tissue site that do not have repercussions in some other organ even at a remote site. The interlocking activities of the endocrine glands, with their respective hormones and their dependence on vitamins and minerals for normal function, is the basis for this entire group of secondary mechanisms.

An interesting example of the involvement of these highly sensitive interlocking endocrine systems is the simple inhalation of nonlethal concentrations of ozone, which produces alterations in the activities of the adrenal glands and disturbs the normal uptake of iodine by the thyroid gland, which in turn alters the activity of the thyroid-stimulating hormone of the pituitary body.

IMMUNE MECHANISMS

A mechanism whose toxic significance remains to be fully evaluated, but which nevertheless has been recognized for many years, is the stimulation of immune mechanisms as a result of the production of a new antigenic structure from the combination of a toxic substance with body constituents, usually protein. This mechanism is thought to be the basis of skin sensitivity resulting from contact with certain reactive organic substances, for example, the chloronitrobenzenes.

Another substance that illustrates this mechanism strikingly is toluene diisocyanate and related aromatic isocyanates. These substances, upon inhalation, have unusual avidity for combining with body protein with resultant allergic sensitization of the respiratory tract.

ENZYMOLOGY AND ITS USES

As the practice of industrial hygiene and occupational medicine are increasingly involved in the control of worker exposure and prevention of industrial disease, knowledge of how enzymatic mechanisms are involved in the toxic response becomes of increasing importance. Measurement of enzyme activity and identification of metabolic products have

been refined so that the clinical toxicologist can use these measurements as an aid in diagnosis of toxic injury and can identify early indicators of response, from the quantitative determination of which, biologic threshold limits can be developed.

These measurements fall into two broad categories: 1) those that measure *exposure,* as arsenic, fluorides, lead, mercury, and benzene, in blood, urine, hair, or breath; 2) those that measure *response* from exposure, as changes in amounts of a natural body constituent, a body metabolite, or changes in enzyme activity.

Some common examples of measurement of exposure: urinary arsenic, fluoride, lead, phenylsulfate from benzene exposure, dichlorodiphenylacetic acid (DDA) from dichlorodiphenyltrichloroethane (DDT). Among the better known measures of response are the measurement of inhibition of activity of plasma cholinesterase for effects from exposure to organic phosphate and carbamate insecticides, changes in urinary delta amino levulinic acid or its enzyme in the red blood cell as responses to overexposure to lead.

Measurement of changes in biochemical constituents as a result of exposure to toxic amounts of substances can be extended to many other industrial substances to which the worker may be significantly exposed, provided they are metabolized by the body. The following are exceptions: 1) Those substances which are constituents common to the body or which convert to same, e.g., sulfur dioxide, chloride, phosphate, or which create no alteration in body composition or function; 2) fast-acting substances such as (skin) irritants that decompose upon contact with body surfaces such as nitrogen dioxide, bis(chloromethyl) ether; 3) substances with predominantly sensitizing properties.

About 2,000 metabolites of substances of industrial interest are now recognized. Accordingly, to use them as biologic indicators, it is necessary to select the appropriate metabolite from this number, and to develop procedures for its quantitative determination, or in the case of a new substance without known metabolites, determine the readily measurable metabolite(s) and apply the procedure to the exposure situation.

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SECTION III

. . .that he may be able to look after his workmen, that they do not meet with those diseases to which they are more liable than workmen in other occupations, or if they do meet with them, that he himself may be able to heal them or may see that the doctors do so.

—Agricola

BIOLOGICAL HAZARDS

Tracy E. Barber, M.D., and E. Lee Husting, Ph.D.

Biological hazards include acute and chronic infections, parasitism, and toxic and allergic reactions to plant and animal agents. Infections may be caused by bacteria, viruses, rickettsia, chlamydia, or fungi. Parasitism may involve protozoa, helminths, or arthropods. Many of the occupational diseases are zoonoses and, consequently, agricultural or other workers associated with animals may be at risk. Some of the infectious or parasitic diseases are transmitted by parasitic arthropod species which act as an intermediate host or vector.

A spectrum of plants and animals produce irritating, toxic, or allergenic substances. Dusts may contain many kinds of allergenic materials, including insect scale, hairs, and fecal dust, sawdusts, plant pollens, and fungal spores.

Biological hazards which will not be discussed in detail include bites or attacks by domestic and wild animals. Divers occasionally encounter sharks or other dangerous fish, sea snakes, or other venomous sea animals. In some areas, risk of occupational exposure to bites from venomous snakes or insects is considerable.

Outdoor occupations that deal with plants or animals or their products, or with food and food processing are more likely to expose the worker to biological hazards. Laboratory and hospital personnel may also be exposed to biological hazards. Travel and work in new environments by previously unexposed and susceptible groups or individuals increase the risk of contracting endemic diseases.

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VIRAL DISEASES

Viral diseases likely to be encountered occupationally include animal respiratory viruses, poxviruses, enteroviruses, and arboviruses. Infections may be acquired from the vector or from the handling of animals or animal products in agriculture. Laboratory-acquired infections

may result from working with the agent, from accidents, from animals, from clinical or autopsy specimens, from aerosols, or from glassware. Viral transmission may occur among patients and staff of hospitals.

RABIES

POTENTIAL OCCUPATIONAL EXPOSURES

At special risk to this viral disease are veterinarians, wild animal handlers, and cave explorers; but the risk remains for farmers, ranchers, trappers, and those individuals who are involved with dogs and cats of unknown origin and for delivery personnel.

SYNONYMS

Hydrophobia

ROUTE OF ENTRY

This disease is transmitted to man by bites of rabid domestic or wild animals (1). In the U.S.A., the principal reservoir which presents a threat to man consists of four wild species: the skunk, the fox, the bat, and raccoon.

HARMFUL EFFECTS

Local—

None except secondary infection at the site of the bite wound.

Systemic—

This viral disease is an almost invariably fatal acute encephalitis. Characteristic prodromal signs and symptoms include headache, anorexia, nausea, and fever. Exaggerated sympathetic responses, drooling, and hydrophobia are typical in the later sensory stages. Death usually occurs following convulsions of the excitement phase, but occasionally occurs in a comatose paralytic stage.

PREVENTIVE MEASURES

Rabies is caused by a virus of the rhabdovirus group and requires an incubation period usually from a few weeks to several months, but occasionally up to one year. Prevention of the disease is accomplished by avoidance of animal bites and caves containing infected bats; vaccination of domestic pets and farm animals; post-exposure immunization; and, in certain high-risk individuals, pre-exposure immunization as appropriate (2,3,4).

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CAT-SCRATCH DISEASE

POTENTIAL OCCUPATIONAL EXPOSURES

Presumably this disease is a viral infection, but it has been suggested that the causative agent may be of the chlamydia type. Principal occupations at risk are workers in animal laboratories, cat and dog handlers, and veterinarians (1).

SYNONYMS

Cat-scratch fever, non-bacterial regional lymphadenitis, benign inoculation lymphoreticulosis.

ROUTE OF ENTRY

The route of entry is a break in the skin from a cat scratch or from sharp objects such as thorns or splinters.

HARMFUL EFFECTS

Local—

Papule or pustule at the primary site of inoculation and occasionally a transient macular or vesicular rash constitute the primary local effects.

Systemic—

Regional lymphadenitis, which occasionally suppurates, develops up to three weeks following inoculation. Other symptoms, headache, malaise, and fever, are typically mild. The disease itself is self-limiting and without sequelae, but can be confused with granulomatous or neoplastic disease. Diagnosis can be established by skin-testing and/or biopsy to rule out tumor.

PREVENTIVE MEASURES

Proper and immediate attention to scratches is a prerequisite to prevention.

REFERENCE

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ORF

POTENTIAL OCCUPATIONAL EXPOSURES

At high risk to this disease are shepherds, stockyard workers, shearers and veterinarians who come in contact with sheep and goats (1).

SYNONYMS

Ecthyma infectiosum, ecthyma contagiosum.

ROUTE OF ENTRY

Virus enters through small breaks in the skin usually on the hand or exposed parts of the body. The pustular lesion develops at the site of entry. Reservoir is sheep and goats.

HARMFUL EFFECTS

Local—

Apart from the pustular lesion at site of entry, there are no local effects.

Systemic—

The systemic effects of the disease are very mild, and usually consist of a mild fever and mild regional lymphangitis and lymphadenitis. The disease is self-limiting in man. Healing is spontaneous, but may take up to six weeks.

PREVENTIVE MEASURES

The virus is passed to the lamb during lambing season and causes "black lip" with lesions around the mouth, lip, and cornea of the animal. The virus can be contracted directly from the lesions of the nursing lamb or from wool. Control of the disease is dependent on pasture-control measures, since the virus may remain there in the form of dried crusts of lesions. Hygienic precautions should be observed by those who work with sheep.

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MILKER'S NODULES

POTENTIAL OCCUPATIONAL EXPOSURES

Milk producers, dairy farmers, veterinarians, and cattle breeders who are involved in direct handling of the infected teat of cows with mastitis are at the greatest potential risk (1).

SYNONYMS

Milker's nodes, pseudocowpox, paravaccinia.

ROUTE OF ENTRY

An animal pox virus related morphologically to the virus of contagious ecthyma and bovine papular stomatis enters through a break in the skin of the hands.

HARMFUL EFFECTS

Local—

Single or multiple nodules, usually on hands, occasionally on face or neck. Regional lymph nodes may be enlarged.

Systemic—

The disease is usually confined to the hands, but there may be mild systemic symptoms such as headache, malaise, and fever.

PREVENTIVE MEASURES

Prevention consists of proper management of mastitis in cows and hygienic measures by workers who are involved with these animals. Use of gloves, soap and water wash, and disinfectants is indicated.

REFERENCE

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NEWCASTLE DISEASE

POTENTIAL OCCUPATIONAL EXPOSURES

Exposure is limited to poultry handlers, veterinarians, and virologists since Newcastle disease is an infectious disease usually confined to birds, produced by *Myxovirus multiforme*, a paramyxovirus.

SYNONYMS

Avian pneumoencephalitis.

ROUTE OF ENTRY

Route of entry is via the upper respiratory tract.

HARMFUL EFFECTS

Local—

Lacrimation, conjunctivitis and edema of the eyelids.

Systemic—

Mild headache, fever, and respiratory involvement occasionally occur. The disease is self-limited. Diagnosis can be confirmed by isolation of the virus in embryonated eggs.

PREVENTIVE MEASURES

Prevention consists of proper care and handling of the infected birds.

VIRAL HEPATITIS

POTENTIAL OCCUPATIONAL EXPOSURES

Two forms of this disease are recognized, serum and infectious (1-4). Those with primary risk to serum hepatitis are health workers where it becomes an occupational disease. The frequency of hepatitis among oral surgeons appears to be quite high.

SYNONYMS

1) Viral hepatitis type A, epidemic hepatitis, catarrhal jaundice, infectious jaundice. 2) Viral hepatitis type B, serum hepatitis, homologous serum jaundice.

ROUTE OF ENTRY

Serum hepatitis is primarily limited to parenteral transmission, and infectious, to fecal-oral transmission. Parenteral transmission of infectious hepatitis can occur, but is rare.

HARMFUL EFFECTS

Local—

None.

Systemic—

Incubation period is from two to six weeks for infectious, seven to 23 weeks for serum hepatitis, followed by jaundice of varying degrees, anorexia, fever, liver enlargement and tenderness, and generalized debilitation. Clinically serum hepatitis and infectious are almost indistinguishable. Diagnosis is established in the laboratory.

PREVENTIVE MEASURES

Prevention of occupational viral hepatitis depends on group and personal measures (isolation of excreta, etc.), sterilization of instruments, the use of disposable instruments (especially needles and syringes), and an awareness of the hazards of transmission, both oral and parenteral, by workers in pediatric wards, hemodialysis units, and the laboratory. Prophylactic use of immune serum globulin will protect against clinical hepatitis in workers who have had accidental contact with positive blood or excreta, if given in the incubation period. Immune serum globulin will also give transient protection to those with potential exposure.

REFERENCES

1. Cassuto, J. 1973. Hepatitis. J. Occup. Med. 14:792.
2. Devenyi, R., and P. Jourdain. 1973. Viral hepatitis in health care personnel working with drug abusers. J. Occup. Med. 15:779.
3. Hayashi, S. J., R. M. Nakamura, and E. A. Giorgi. 1971. Problems of prevention and detection of hepatitis in personnel of hospital hemodialysis unit. J. Occup. Med. 13:388.
4. Redeker, A. G. 1975. Hepatitis B, Risk of infection from antigen-positive medical personnel and patients. J. Am. Med. Assoc. 233:1061.

RICKETTSIAL AND CHLAMYDIAL DISEASES

Rickettsiae formerly classified as viruses, but now considered to be small, true bacteria, multiply in arthropods which are the reservoir and which transmit these organisms to man. Except for Q fever, rickettsial diseases are typified by a characteristic rash and fever. Of occupational interest are Rocky Mountain spotted fever and Q fever.

Chlamydiae, once classified as viruses, and now regarded as bacteria, cause ornithosis (psittacosis) which is of occupational significance.

ROCKY MOUNTAIN SPOTTED FEVER

POTENTIAL OCCUPATIONAL EXPOSURES

Occupations at high risk are foresters, rangers, ranchers, farmers,

trappers, hunters, construction workers, resort operators and lumber jacks when they work in areas where ticks are present, and laboratory workers 1,2,3).

SYNONYMS

New world spotted fever, tick fever, and tickborne typhus fever.

ROUTE OF ENTRY

The reservoir of the causative *Rickettsia rickettsii* is the tick. The disease is transmitted to man by the bite of the infected tick, and by contamination of skin with tick tissue juices or feces. The disease is widespread in the United States.

HARMFUL EFFECTS

Local—

None other than local inflammation and pruritus from the bite.

Systemic—

This disease is characterized by a sudden onset, with persistent fever, headache, chills, myalgia, and conjunctival injection. A maculopapular rash on the extremities occurs about the third day and spreads rapidly to most of the body. Hemorrhages and petechiae are common. Fatality is about 20% in untreated cases; death is uncommon in cases treated promptly.

PREVENTIVE MEASURES

Prevention of the disease is accomplished by avoidance of the tick infested areas and early recognition and careful removal of the tick before attachment is accomplished. Use of repellants is helpful. Measures to reduce tick populations have generally proven impractical. Vaccination is generally limited to persons frequenting highly endemic areas and laboratory workers.

REFERENCES

1. Burdorfer, W. 1975. A review of Rocky Mountain spotted fever (tick-borne typhus), its agent, and its tick vectors in the United States. J. Med. Entomol. 12: 269.
2. Hattwick, M.A.W., R. J. O'Brien and B. F. Hanson. 1976. Rocky Mountain spotted fever: epidemiology of an increasing problem. Ann. Intern. Med. 84:732.
3. Torres, J., E. Humphreys, and A. L. Bisno. 1973. Rocky Mountain spotted fever in the Mid-South. Arch. Intern. Med. 132:340.

Q FEVER

POTENTIAL OCCUPATIONAL EXPOSURES

At highest risk to this disease are dairy farmers, ranchers, stockyard workers, slaughterhouse workers, hide and wool handlers, laboratory workers, and rendering plant workers (1).

SYNONYMS

Australian Q fever, Query fever.

ROUTE OF ENTRY

The reservoir for this rickettsial disease is the tick, wild animals, and domestic cattle, sheep, and goats where it causes no apparent disease. The rickettsiae *(Coxiella burnetti)* are shed by the infected animals in placental tissues and birth fluids. Rickettsiae exist in and around lambing pens and pastures in dried tissues for months. Man is inoculated by inhalation of contaminated dusts from these areas as well as from areas where infected animals are processed. The disease may also be transmitted by contact with the infected animals and contaminated materials such as soiled laundry of infected persons. The milk of infected cattle is also suspect in the transmission of the disease.

HARMFUL EFFECTS

Local—

None

Systemic—

The disease is flu-like in nature. Pneumonitis develops in most cases. Typical clinical manifestations of headache and fever develop after an incubation period lasting from 14 to 29 days. Anorexia and respiratory symptoms are delayed and occur usually on the fifth day after onset. Fatalities are rare, and chronic endocarditis is an infrequent complication. A protracted form of Q fever (without headache and respiratory symptoms) may also occur.

PREVENTIVE MEASURES

Prevention is dependent on avoidance of aerosolized products of conception from ruminant animals, vaccination of individuals at high risk, pasteurization of milk, and regulation of movement of infected animals. Outbreaks are generally of short duration, and the disease in man is controlled by antibiotic therapy.

REFERENCE

1. Brown, G. L. 1973. Clinical aspects of Q fever. Postgrad. Med. J. 49:539.

ORNITHOSIS

POTENTIAL OCCUPATIONAL EXPOSURES

Those individuals at high risk are pet shop owners, taxidermists, zoo attendants, and persons associated with the raising and processing of poultry. Laboratory and hospital personnel may also be occupationally exposed (1, 2).

SYNONYMS

Psittacosis and parrot fever.

ROUTE OF ENTRY

The infecting organism *Chlamydia psittaci* is present in the nasal discharge, droppings, tissues, and feathers of infected birds. Primarily, pigeons, lovebirds, parrots,and domestic fowl serve as a reservoir for the disease. Ornithosis may be contracted by man by inhalation of the dried discharges and droppings of the birds. Occasionally, transmission from human to human has been reported among health care personnel.

HARMFUL EFFECTS

Local—

None.

Systemic—

Following a 7- to 14-day incubation period, infection becomes apparent. Victims most often complain of headache and soon become febrile with a characteristically relatively slow pulse. Other symptoms include lethargy, insomnia, photophobia, nausea, vomiting, and diarrhea. Signs often include proteinuria, abnormal white blood cell count, and enlarged, nontender liver. Commonly, symptoms and chest X-rays indicate pneumonitis. In severe cases myalgia with stiffness and spasms, delirium and stupor, and rarely icterus develop. Diagnosis may be confirmed by a rising titer of complement-fixing antibodies in the blood. Isolation of the causative agent in the serum or sputum is also indicative. Isolation from bronchial secretion may continue for several months or years following an acute episode. Relapse is common.

PREVENTIVE MEASURES

Importation regulations and traffic control of imported birds are important preventive measures.

REFERENCES

1. Durfee, P. T., M. M. Pullen, R. W. Currier II, et al. 1975. Human psittacosis associated with commercial processing of turkeys. J. Am. Vet. Med. Assoc. 167:804.
2. Durfee, P. T. 1975. Psittacosis in humans in the United States, 1974. J. Infect. Dis. 132:604.

BACTERIAL DISEASES

Most frequently seen bacterial infections of an occupational nature are caused by neglected minor wounds, abrasions, and excoriated dermatitis where the integrity of the skin surface is broken. These infections are frequently caused by mixed bacterial infections, but chief among the offending organisms are staphylococci and streptococci. Most of these infections can be avoided by encouraging good personal hygiene habits, and the early reporting and proper care of the minor skin breaks, especially on the hand, forearm, and around the finger nails.

TETANUS

POTENTIAL OCCUPATIONAL EXPOSURES

Those individuals whose occupations include the hazard of traumatic injury, usually of a penetrating or crush type wound, are at highest risk; farmers and ranchers who work around domestic animals and soil are also at risk (1).

SYNONYMS

Lockjaw.

ROUTE OF ENTRY

The route of entry is generally through breaks in the skin from penetrating or crush wounds.

HARMFUL EFFECTS

Local—

None, other than the wound at entry.

Systemic—

This is an acute disease caused by the toxins produced in the body by the *Clostridium tetanii*. The disease is characterized by tonic spasms primarily of the masseter and neck muscles and secondarily of the muscles of the back. These spasms are extremely painful. When untreated, tetanus has a mortality rate greater than 70% in adults.

PREVENTIVE MEASURES

In the past, massive active immunization programs for the general public have been conducted. This is the best method of prevention of the disease. Once the wound has occurred and no prior immunization has taken place, human immune globulin or antitoxin (equine or bovine), administered soon after the injury, may be of use as a preventive measure. Human immune globulin is the preferred treatment as it obviates the risk of serum reaction so often encountered with the antitoxin.

REFERENCE

1. Bizot, W. H. 1963. Tetanus in industry. J. Occup. Med. 5:209.

ANTHRAX

POTENTIAL OCCUPATIONAL EXPOSURES

In the United States this disease is almost exclusively limited to agricultural workers and occupations handling imported goat hair, wool and hides (1).

SYNONYMS

Wool sorters disease, rag sorters disease, malignant pustule, milzbrand, and charbon.

ROUTE OF ENTRY

The anthrax bacillus originally gains entry through small breaks in the skin. Approximately three percent of the cases in the United States are pulmonary through inhaled spores (2-3).

HARMFUL EFFECTS

Local—

At the site of entry vesicles develop initially and progress to a depressed black eschar, at times surrounded by mild to moderate edema. Pain is unusual.

Systemic—

The disease spreads from the local area through the regional lymph nodes and blood stream, which may result in overwhelming septicemia and death in untreated cases. Inhalation of anthrax spores causes initial symptoms that are mild and nonspecific resembling a common upper respiratory infection. Respiratory distress, fever, and shock follow in three to five days, with death commonly 7 to 24 hours thereafter.

PREVENTIVE MEASURES

Certification of imported hides, hair, and wool as anthrax free by the exporting country has helped to reduce the incidence of anthrax. In the United Kingdom imported hair and wool are treated with warm formaldehyde solution. In the United States the chief preventive measure for high risk industrial workers is immunization. Improved personal hygiene of workers, protective clothing, ventilation and housekeeping controls in the plants are also valuable in control of the disease. Vaccination of animals in enzootic areas and strict adherence to laws regarding animals who have contracted or who have died of anthrax have helped reduce agricultural incidence.

REFERENCES

1. Kendall, C. E. 1959. Occupational anthrax in the United States. J. Occup. Med. 1:174.
2. Hughes, M. H. 1973. Anthrax. Br. Med. J. 1(5878):488.
3. Severn, M. 1976. A fatal case of pulmonary anthrax. Br. Med. J. 1(6012):748.

BRUCELLOSIS

POTENTIAL OCCUPATIONAL EXPOSURES

The acute form of this disease is now mainly confined to meat packing house employees and inspectors, livestock producers and marketers, and veterinarians (1,2).

SYNONYMS

Undulant fever, Malta fever, Bang's disease.

ROUTE OF ENTRY

Bacteria gain entry to the body through small cuts and scratches

which are contaminated with blood and fluids of infected animals. The inhalation route from dust around animal pens occurs, but is now quite rare.

HARMFUL EFFECTS

Local—

None.

Systemic—

Onset is with a flu-like syndrome of fever, headache, and myalgias (3). The fever is progressive with weight loss and weakness. The fever is quite characteristically nocturnal with a nearly normal daytime temperature. Chronic forms of brucellosis in the form of splenic abscesses, bone and joint disease with abscess formation, renal disease, and bladder disease are late complications usually occurring 10 - 20 years following an acute episode which was untreated or inadequately treated. Chronic forms of the disease which resemble neurasthenia remain an ill-defined entity both clinically and serologically, and actual existence of chronic brucellosis of this type is doubted. Diagnosis is established by blood cultures and rising agglutination titers.

PREVENTIVE MEASURES

Control of brucellosis in man is contingent upon control of the disease in animals, and the incidence in humans in a given locality is often the index of the disease control effectiveness in the domestic animal population of that locality. Awareness of the disease by the workmen and by doctors who see patients involved in occupations where exposure to infected animals is possible is very important. In the workplace hygienic practices and proper attention to minor cuts and scratches, especially on the hands and forearm can help control the disease.

Brucella canis infections in man are becoming a recognized problem in individuals with contact with dogs. The agglutination test which will identify antibodies in man as a result of infection with the other *Brucella* species is unreliable with *Brucella canis* unless the specific antigen is used.

REFERENCES

1. Busch, L. A., and R. L. Parker. 1972. Brucellosis in the United States. J. Infect. Dis. 125:289.
2. Report to Subcommittee on Public Health. 1970. Brucellosis in the United States. Arch. Environ. Health 25:66.
3. McDevitt, D. G. 1973. Symptomatology of chronic brucellosis. Br. J. Ind. Med. 30:385.

LEPTOSPIROSIS

POTENTIAL OCCUPATIONAL EXPOSURES

Those occupations at risk include farmers, field workers, sugarcane workers, livestock producers and marketers, packinghouse workers, sewer workers, miners, veterinarians, and military troops (1,2).

SYNONYMS

Weil's disease, swineherd's disease, canicola fever, or hemorrhagic jaundice.

ROUTE OF ENTRY

Infection generally results from bacterial penetration of the skin in scratched or abraded areas, and there is some evidence to suggest possibility of infection by ingestion. Leptospira may be found in farm animals, dogs, wild animals, and rats and other rodents. The organism is excreted in the urine, and the disease may be contracted by contact with the infected urine or tissue, and with water polluted with infected urine or tissue.

HARMFUL EFFECTS

Local—

None.

Systemic—

The incubation period is usually around ten days. The acute infection begins with fever, headache, and chills, followed by quite severe malaise, vomiting, muscular aches, and conjunctivitis. Frequently, meningeal irritation occurs and along with jaundice, renal insufficiency, hemolytic anemia, and hemorrhage into the skin and mucous membranes represent the characteristics of severe disease. Fatality rates in these instances may run as high as twenty per cent. Diagnosis is by agglutination and complement fixation tests and culture of leptospiras in blood during acute illness or in urine after the first week.

PREVENTIVE MEASURES

For those who work around suspect animals, protection should be provided by boots and gloves. Control of the disease is accomplished through rodent control, segregation of domestic animals, public health ordinances which prevent drainage from livestock feed lots into fresh water or recreational areas, and vaccination of farm animals and pets.

REFERENCES

1. U.S. Department of Health, Education, and Welfare, Public Health Service, Center for Disease Control. 1971. Leptospirosis Annual Summary. Center for Disease Control, Atlanta.
2. Heath, C. W. Jr., A. D. Alexander, and M. M. Galton. 1965. Leptospirosis in the United States. N. Engl. J. Med. 273:857.

PLAGUE

POTENTIAL OCCUPATIONAL EXPOSURES

Occupational exposure in the United States is primarily in sheepherding families, farmers, ranchers, hunters, and geologists working in sparsely populated areas of the Western states (1).

SYNONYMS

Black Death, Bubonic plague.

ROUTE OF ENTRY

The flea is critical in the maintenance of plague. In the United States, urban type plague has not been encountered since the San Francisco epidemic in 1907-1908. Sylvan plague occurs as a chronic epizootic among wild animals in the United States. Rodents and other wild mammals are the primary animal sources of the disease. The disease is transferred from the infected wild animal to man by the bite of the flea.

HARMFUL EFFECTS

Local—

The lymph nodes draining the site of the original infected bite become acutely inflamed and painful.

Systemic—

The infection spreads from the lymph nodes into the blood stream and produces localized infections in diverse parts of the body. Pneumonic involvement carries a very high mortality rate, and this form of plague is communicable from man to man.

PREVENTIVE MEASURES

Primarily, prevention consists of control of rat populations in urban areas, along with rat proofing of buildings, particularly rat control and rat proofing of harbor and dock areas. Active immunization of persons travelling in endemic plague areas and of laboratory workers is important. Health education efforts should be promoted, especially in the rural areas of the western part of the United States among the native population.

REFERENCE

1. Ree, W. P., D. L. Palmer, R. C. Williams, Jr., and A. L. Kisch. 1970 Bubonic plague in the Southwestern United States—A review of recent experience. Medicine (Baltimore) 49:465.

FOOD POISONING

POTENTIAL OCCUPATIONAL EXPOSURES

Rarely is food poisoning an occupational disease, but in many instances the worker acts as the contaminating agent of otherwise pure food. The three primary types of contamination are by bacteria of the Salmonella group, *Clostridium perfringens*, and *Staphylococcus aureus*. At highest risk are those individuals subjected to mass feeding techniques such as in the military, in prisons, and in certain instances in cafeterias in the workplace.

SYNONYMS

Gastroenteritis, acute G.I.'s.

ROUTE OF ENTRY

In the case of salmonella, entry is by the fecal-oral route. In the case of staphylococcus, the enterotoxin develops entirely within the food and enters orally. *Clostridium perfringens* also enters orally.

HARMFUL EFFECTS

Local—

None.

Systemic—

Symptoms of Salmonellosis usually appear 12 to 24 hours after ingestion of contaminated food, which usually will distinguish it from staphylococcal food poisoning.

It is a febrile disease and can range from a trivial diarrhea to an extremely severe disease with enteric fever, septicemia, dysenteric syndromes, and such focal manifestations as pneumonia, meningitis, and arthritis.

Staphylococcal food poisoning is produced by an enterotoxin which develops as the staphylococcus grows in the food product. Onset of the symptoms usually occurs after three hours, but can vary from one to six depending on the quantity of the toxin ingested. Mild cases cause increased salivation, nausea and vomiting with retching, abdominal pain and cramps, watery diarrhea. Severe cases go on to show bloody vomitus and stool and marked dehydration. Generally speaking there is complete recovery within 24 hours.

Clostridium perfingens is often present in animal tissue which if held at incubating temperatures for several hours prior to consumption can result in proliferation of the organisms and subsequent human illness. The disease is characterized by a sudden onset of abdominal colic which is followed by diarrhea. Nausea is common, but vomiting is rare. The disease is mild and recovery usually occurs within 24 hours.

There is no treatment except symptomatic treatment for uncomplicated food poisoning. Salmonellosis is treated by antibiotic therapy.

PREVENTIVE MEASURES

Primarily prevention consists of proper handling of food products, with clean hands and garments, and with freedom from skin lesions. Refrigeration of the food products at a proper level will prevent the growth of the bacteria. Adequate cooking of foods will kill the salmonella organisms. Once the enterotoxin of the staphylococcus is produced in the food, no amount of cooking, freezing, or any other known method will remove it from the food.

TUBERCULOSIS

POTENTIAL OCCUPATIONAL EXPOSURES

Those individuals who are at greatest occupational risk are health professionals in hospitals and sanitoriums caring for individuals afflicted with this disease.

SYNONYMS

Consumption.

ROUTE OF ENTRY

Entry of the causative agent is by inhalation and by fomites.

HARMFUL EFFECTS

Local—

None.

Systemic—

The majority of cases now involve pulmonary tuberculosis. This is an infectious disease characterized by the formation of tubercles in the tissue of the lung. General symptoms are fever, night sweats, emaciation, and cough with expectoration.

PREVENTIVE MEASURES

Preventive measures include isolation control of patients with an active disease, proper waste handling, repeated Mantoux testing of those individuals involved with the care and treatment of patients with this disease, and periodic X-ray examination of tuberculin-positive personnel.

MYCOBACTERIAL INFECTIONS

POTENTIAL OCCUPATIONAL EXPOSURES

Health care,pathologists,and other laboratory personnel exposed to the tubercule bacilla are at risk of developing innoculation-type cutaneous tuberculosis. Gulf Coast fishermen and operators of tropical fish stores (1,2) are at risk of developing cutaneous granulomas.

SYNONYMS

1) Verruca necrogenica, prospectors' wart, butchers' tubercle; 2) tuberculous paronychia; 3) swimming-pool granuloma, fish-tank granuloma.

ROUTE OF ENTRY

In the case of verruca necrogenica and tuberculous paronychia, entry is generally through a puncture wound or break in the skin, such as a hang-nail, and contamination with *Mycobacterium tuberculosis*. In the case of the granulomatous lesion, route of entry is an abrasion or a puncture wound commonly inflicted by a fish fin or bone. The wound in this instance is contaminated with *Mycobacterium marinum (balnei)*.

HARMFUL EFFECTS

Local—

A warty nodule develops at a puncture site or a paronychia develops usually from a hang-nail and neither respond to local treatment, surgical drainage, or antibiotics. They do, however, respond to systemic antituberculosis chemotherapy. Granulomatous lesions are usually confined to the hands and forearms. These also fail to respond to the usual treatment. After diagnosis is made by culture, they do respond to broad

surgical excision of the lesion. Systemic antituberculosis chemotherapy is apparently ineffective. Infections of this type have been acquired in salt water baths in the brackish water of the Chesapeake Bay, and in brackish water of the Alabama and Louisiana coasts.

Systemic—

None reported.

PREVENTIVE MEASURES

Use of gloves is required to protect the hands; prompt and proper care should be given to puncture wounds to prevent infection if a break in skin occurs (1).

REFERENCES

1. Miller, W. C. 1973. Mycobacterium marinum in Gulf fishermen. Arch. Env. Health 27:8.
2. O'Donnel, T. F. Jr., P. F. Furgenson, and N. F. Weyerich. 1971. An occupational hazard—tuberculous paronychia. Arch. Surg. 103-757.

ERYSIPELOID

POTENTIAL OCCUPATIONAL EXPOSURES

This disease is commonly seen in butchers and poultry and fish handlers where puncture wounds from bone spicules and fin spines are frequent (1).

SYNONYMS

None.

ROUTE OF ENTRY

Route of entry is generally through breaks in the skin of the hands caused by scratches, abrasions, and puncture wounds.

HARMFUL EFFECTS

Local—

Infection begins as erythema around the site of the wound with fairly rapid peripheral spread. The spreading edge tends to be painful and there tends to be central clearing as the spreading progresses.

Systemic—

Frequently this disease is associated with painful localized lymphadenitis. Occasionally septicemia can develop, but this can be controlled by the expedient use of antibiotics. Wound cultures of erysipeloid show *Erysipelothrix rhusiopathiae (insidiosa),* but generally the cultures reveal a mixture of organisms.

PREVENTIVE MEASURES

Protective gloves are especially necessary for workers in the poultry industry. There must be prompt and proper attention to bone scratches, puncture wounds, and any other wounds on the hands of employees working around poultry, meat, and fish.

REFERENCE

1. Proctor, D. M., and I. W. Richardson. 1954. A report of 235 cases of erysipeloid in Aberdeen. Brit. J. Ind. Med. 11:175.

TULAREMIA

POTENTIAL OCCUPATIONAL EXPOSURES

Those occupations at highest risk are forestry workers, butchers and locker plant operators, hunters and cooks, farmers, veterinarians, and laboratory workers (1).

SYNONYMS

Deer fly fever, rabbit fever.

ROUTE OF ENTRY

This is a disease of rodents, resembling plague, which is transmitted by bites of flies, fleas, ticks, and lice and may be acquired by man through handling of infected animals. It is caused by *Francisella tularensis*. Generally speaking the route of entry is through small cuts and scratches on the hands from the lesions of the infected animals. This is particularly true of hunters who dress wild rabbits and women who handle these wild rabbits in cooking. Inoculation of the conjunctival sac occurs when infectious fluids are splashed in the eye or from wiping eyes with contaminated fingers. Man may become infected from the bites of infected insects; from bites of animals carrying the organism in their mouth as a result of feeding on infected carcasses; by eating insufficiently cooked infected meat; or by drinking contaminated water.

HARMFUL EFFECTS

Local—

In man, an ulcer forms at the site of the inoculation, followed by inflammation of the regional lymph nodes.

Systemic—

Severe constitutional symptoms usually appear following the development of the ulcer. These consist of headache, myalgia, chills, and rapid rise in temperature.

PREVENTIVE MEASURES

Control of the disease depends on the use of rubber gloves in handling carcasses of wild animals or safety hoods in laboratory experimentation; avoidance of arthropods, flies, mosquitos, and wood ticks when working in endemic area; avoidance of drinking raw water in endemic areas; and thorough cooking of wild game.

REFERENCE

1. Boyce, J. M. 1975. Recent trends in the epidemiology of tularemia in the United States. J. Infect. Dis. 131:197.

FUNGAL DISEASES

The incidence of fungal disease of an occupational nature is not great and is mainly confined to farmers, outdoor workers, and animal raisers. Diagnosis in fungal diseases is made by microscopic identification of the fungus with cultural confirmation.

Fungal diseases may be roughly classified by systemic, subcutaneous superficial, or hypersensitivity effects. Occasionally, the subcutaneous fungal diseases may spread systemically. Systemic effects may be due to opportunistic fungal disease such as candidiasis or aspergillosis which mainly disseminate in individuals with lowered resistance or increased susceptibility. Histoplasmosis and coccidioidomycosis are not opportunistic in the sense that they occur systemically in healthy individuals. Subcutaneous infections, usually introduced through a penetration of the skin, include mycetoma, sporotrichosis, and chromoblastomycosis. The dermatophytoses, including the *Tinea* groups, are superficial infections caused by three genera of fungi. Finally, there may be hypersensitivity reactions due to fungal antigens inhaled with dusts during agricultural or other activities. These usually involve pneumonitis with asthmalike symptoms.

CANDIDIASIS

POTENTIAL OCCUPATIONAL EXPOSURES

Those occupations at high risk are dishwashers, bartenders, cooks, bakers, poultry processors, packinghouse workers, cannery workers, and certain health workers.

SYNONYMS

Moniliasis, thrush.

ROUTE OF ENTRY

Candida albicans is still considered the major cause of candidiasis, although other species of the same genus have been found to produce similar signs and symptoms. Candida species are ubiquitous in nature and are considered part of the normal human flora. It is a mild and opportunistic infection involving the skin and mucous membranes.

HARMFUL EFFECTS

Local—

Candidiasis is manifested by cutaneous, oral, and vaginal lesions. These can become chronic and spread to other mucosal surfaces or intertriginous areas in the groin, anti-cubital fossa, interdigital folds, the inframammary areas, the umbilicus, and the axilla.

Systemic—

Systemic effects are rare and are usually found in persons having surgery, diabetes mellitus, debilitating diseases, and immunosuppressive

and antibiotic therapy. Aspiration pneumonia is probably the chief form of visceral candidiasis. Endocarditis, meningitis, and ulcers of the digestive tract are occasionally seen. Frequently these systemic complications are fatal.

PREVENTIVE MEASURES

In those occupations predisposed to working in water, proper care and protection of the hands is essential. Protective cream and/or gloves are indicated.

ASPERGILLOSIS (Allergic)

POTENTIAL OCCUPATIONAL EXPOSURES

The occupational instance of this disease is limited by the opportunistic nature of the *aspergillus* species (1,2). *Aspergillus* is a group of fungi of low pathogenicity for man unless resistance is overcome by an overwhelming inoculum or debilitating illness. Farmers and grain mill workers are at high risk because the plant and animal matter in their work environment provide excellent growth media for fungal spores. Bird handlers and raisers are also at high risk.

The syndrome of allergic aspergillosis occurs in asthmatics who develop hypersensitivity to aspergillus antigens. Allergic reactions occur in workers in rope factories (hemp disease) and are a frequent result of aspergillosis exposure in Great Britain.

SYNONYMS

Hemp disease.

ROUTE OF ENTRY

Route of entry is generally by inhalation of the spores.

HARMFUL EFFECTS

Local—

Aspergillus fumigatus and other species of aspergillus are the cause of aspergillosis. The fungus is usually found in large colonies in moist, decaying vegetation heated by bacterial fermentation and in warm, cereal grain storages. The disease may become disseminated or localized to the lung, ear, orbit, or parinasal sinuses.

Systemic—

Secondary invasion by the hyphae into the blood stream may disseminate the fungus to other parts of the body. Formation of abscesses or granulomas in the brain, heart, kidney, and spleen may occur.

PREVENTIVE MEASURES

Farmers and grain mill workers working where the presence of fungi is suspected should be provided with protective masks.

REFERENCES

1. de Haller, R. and F. Suter, eds. 1974. Aspergillosis and Farmer's Lung in Man and Animal. Huber, Bern.
2. Jordan, M. C., C. W. Bierman, and P. P. VanArsdale, 1976. Allergic bronchopulmonary aspergillosis. Arch. Intern. Med. 128:576.

COCCIDIOIDOMYCOSIS

POTENTIAL OCCUPATIONAL EXPOSURES

This disease is endemic in the arid and semiarid areas of the Southwestern United States and parts of northern Mexico and Argentina. It is caused by the inhalation of the spores of *Coccidioides immitis.* The infections generally occur during the dry season and following dust storms. At high risk to coccidioidomycosis are migrant workers, farmers, construction workers, military personnel, bulldozer operators, and excavation workers in these endemic areas. Also at risk are cotton mill workers and laboratory personnel who frequently handle the organisms in the laboratory (1).

SYNONYMS

Valley fever, coccidioidal granuloma.

ROUTE OF ENTRY

The fungus gains entry by inhalation of dust.

HARMFUL EFFECTS

Local—

None.

Systemic—

Inhalation of the dust causes a respiratory infection, which is usually symptomatic. The clinical picture presented is one of acute bronchitis or pneumonia; chills, fever, cough, and poorly localized chest pain. Recovery normally follows in two or three weeks leaving some pulmonary scarring and, in severe cases, calcification or cavity formation. In some individuals who have had prior exposure to *Coccidioides immitis,* hypersensitivity results and is manifested in two forms: 1) erythema multiforme and erythema nodosum—sterile, self-limiting skin lesions of the lower extremities (Valley fever) and 2) pleural effusion—a result of antigenic material introduced into the pleural cavity. Coccidioidal granuloma is a disseminated form of coccidioidomycosis which is often fatal. Abscess formation in the lungs and the rest of the body, including the central nervous system, may occur. Immunologic resistance develops in individuals who recover. There are occasions of unusual syndromes (2).

PREVENTIVE MEASURES

Laboratory workers should be especially careful when handling this organism. Workers from nonendemic areas should not be recruited for dusty operations in endemic areas. Dust in areas around camps and

construction sites can be controlled by planting grass or by watering or oiling exposed soils, roads, and airstrips. Where the fungus is known to exist and the worker cannot otherwise be protected, he should be supplied with a mask.

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HISTOPLASMOSIS

POTENTIAL OCCUPATIONAL EXPOSURES

This disease is an opportunistic primary pulmonary infection which occasionally disseminates. The occurrence is worldwide (1). The causative agent, *Histoplasma capsulatum,* is a dimorphic fungus growing on soils enriched by bat, chicken, and other bird excrement. Occupations that involve employment around old barnyards, chicken houses, or caves are likely to have a high incidence of the disease. Farmers who spread fertilizers or soil containing chicken droppings or large quantities of organic matter may also be exposed. Occasionally cases occur among construction workers resulting from exposure when a building which pigeons have adopted as a nesting and roosting site is demolished or when ground under the roost of wild birds is disturbed.

SYNONYMS

Darling's disease, reticuloendotheliosis.

ROUTE OF ENTRY

In most cases the portal of entry is the lung. Infection may occur through the gastrointestinal tract, but this is thought to be secondary to primary lesions of the mouth and pharynx.

HARMFUL EFFECTS

Local—

Chronic localized histoplasmosis takes two primary clinical forms: a) pulmonary; this type of infection resembles pulmonary tuberculosis in all respects; b) mucocutaneous ulcers of the mouth, tongue, pharynx, gums, larynx, penis, or bladder; these are rare lesions found only in adults.

Systemic—

Pulmonary histoplasmosis ranges from a slight self-limited infection to fatal disseminated disease. Least resistance to histoplasmosis is found in young infants and in adults after the fifth decade.

Adult histoplasmosis shows a marked predilection for men. Histoplasmosis of the lips, nose, mouth, and larynx occurs almost exclusively

in adults, and is the initial manifestation in about one third of all fatal cases. Adult complications of the disease include subacute vegetative endocarditis, massive lymphadenopathy resembling tuberculosis or lymphoma, various forms of pneumonia, cerebral histoplasmoma, and meningitis.

In infants there is fever, emaciation, anemia, leukopenia, and evidence of the widespread involvement of the viscera including liver, spleen, lung, lymph nodes, adrenals, skin, kidney, brain, eyes, and endocardium.

PREVENTIVE MEASURES

The incidence of histoplasmosis is limited by the control of exposures and by dust control methods (watering or oiling of soil surfaces). Disinfectant solutions may also be sprayed on the soil. Use of masks is recommended in areas which are known to be contaminated when exposure cannot be prevented.

REFERENCE

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MYCETOMA

POTENTIAL OCCUPATIONAL EXPOSURES

At special risk are farmers and individuals exposed to puncture wounds.

SYNONYMS

Maduromycosis.

ROUTE OF ENTRY

Route of entry is penetration through skin usually by puncture wounds and usually following trauma. Reservoir for the fungi is in soil and decayed vegetation.

HARMFUL EFFECTS

Local—

The condition can be caused by true fungi or by actinomycetes producing a lesion. Sinus tracts develop presenting colonies of fungus (granules) in the exudate. Isolation of the fungus in culture and study of the granules are necessary for identification of the microorganisms.

Systemic—

The causative organism rarely disseminates, and systemic effects are minimal.

PREVENTIVE MEASURES

Prevention of disease is dependent on the prevention of puncture wounds (1). Treatment is difficult and frequently ineffective and amputation may become necessary.

REFERENCES

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SPOROTRICHOSIS

POTENTIAL OCCUPATIONAL EXPOSURES

Sporotrichosis is a subcutaneous mycotic infection. The agent, *Sporothrix (Sporotrichum) schenkii,* is a saprophyte on plants and may be present on thorns or in sphagnum moss (1). Those at highest risk are farmers and gardeners, horticulturists, florists, nursery workers, and those using sphagnum moss. It has also been reported in miners in South Africa who came in contact with heavily infected timber shorings.

SYNONYMS

None.

ROUTE OF ENTRY

The principal route of entry is by contaminated splinters or soil penetrating the skin through cuts and scratches.

HARMFUL EFFECTS

Local—

The fungus causes a series of hard, red, nodular lesions as it spreads up the extremities, following the lymphatic system. The nodules are granulomas which undergo necrosis and become ulcerated.

Systemic—

Rarely does this disease become systemic, but when it does it involves muscles, mucus membranes, the viscera, the skeletal system, and, even more rarely, the lungs (2).

PREVENTIVE MEASURES

Prevention consists of protection of the hands and forearms especially against splinters, thorns, and contaminated soil. This is especially important for people working with sphagnum moss and can be afforded by long gloves and arm protectors.

REFERENCES

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2. Evers, R. H., and R. R. Whereatt. 1974. Pulmonary sporotrichosis. Chest. 66:91.

CHROMOBLASTOMYCOSES

POTENTIAL OCCUPATIONAL EXPOSURES

Chromoblastomycoses are subcutaneous mycoses caused by a group of fungi which are slow growing saprophytic fungi (1). Those at highest risk are farm workers and other individuals subjected to scratches on the feet and legs, particularly those who live in tropical areas.

SYNONYMS

Verrucous dermatitis.

ROUTE OF ENTRY

The route of entry is any break in the continuity of the skin of the feet and the legs.

HARMFUL EFFECTS

Local—

At the site of the wound a progressive cauliflower-like lesion is produced on the skin. The disease itself is characterized by dermal microabscesses containing fungi, by epidermal hyperplasis and hyperkeratosis, and by extensive fibrosis which may obstruct the lymph channels.

Systemic—

Usually none, unless the lymphatic scarring results in elephantiasis. Hematogenous spread to the groin has been reported.

PREVENTIVE MEASURES

Preventive measures include the wearing of shoes and the covering of the legs of workmen to prevent the cuts and scratches necessary to introduce the fungi.

REFERENCES

1. Carrion, A. L. 1975. Chromoblastomycosis and related infections: New concepts, differential diagnosis and nomenclatorial implications. Int. J. Dermatol. 14:27.

DERMATOPHYTOSES

POTENTIAL OCCUPATIONAL EXPOSURES

These infections are caused by three genera of fungi. At high risk are farmers, animal handlers, pet and hide handlers, wool sorters, cattle ranchers, athletes, lifeguards, gymnasium employees, and animal laboratory workers.

SYNONYMS

Ringworm, athlete's foot, jock strap itch.

ROUTE OF ENTRY

The main reservoir of these superficial fungi is in man and animals. Ringworm of the feet or athlete's foot develops in areas of maceration between the toes. Ringworm of the nails is generally due to spread from ringworm on the feet or hands. Ringworm of the groin occurs in the folds of the upper inner thigh where chafing and irritation are common. Ringworm of the body or ringworm of the hand are usually due to contact with animals or other human beings with similar conditions. Ringworm of the scalp can be due to contact with animals, but most generally it is transmitted from man to man.

HARMFUL EFFECTS

Local—

In all of these superficial ringworm infections, it is necessary to make sure of the diagnosis by cultural methods if possible because they mimic too closely other diseases.

The lesion of the skin is characteristically flat, spreading, ring shaped, with reddish, vesicular, scaly or crusted periphery, and a central clearing area. The lesion of nails and of hair demonstrates a keratolytic property of the ringworm fungus, which can cause the nail to disintegrate; hair to dissolve; and the scaffolding of the stratum corneum, the keratinized cell, to be demolished. Living epidermis is not affected.

Ringworm of the feet is the most common type of ringworm infection. The intertriginous involvement may remain chronic and localized or may develop acute exacerbations, with extensive formation of vesicles and bullae over the feet and occasionally vesicular lesions elsewhere on the body, particularly the hands ("id" reaction).

Systemic—

None

PREVENTIVE MEASURES

Prevention depends on recognition of the disease in animals and proper handling techniques with these animals. Also necessary are sterilization and proper laundering of towels and general cleanliness in showers and dressing rooms of gymnasiums and swimming pools. Education for personal hygiene is also effective control.

MISCELLANEOUS FUNGAL DISEASES

POTENTIAL OCCUPATIONAL EXPOSURES

There is a group of conditions related to inhalation of fungus and actinomycetes spores which are hypersensitivity diseases (1-6). Repeated exposure sensitizes the individual to the spores (protein sensitization), and the disease state recurs on subsequent exposure. At high risk are farmers who handle hay in confined areas, saw mill operators, mushroom workers, sugarcane workers, cork workers, workers exposed to redwood processing, and workers handling other agricultural products on an industrial basis such as seeds, textile fibers, wood, and gum.

SYNONYMS

Farmer's lung, maple-bark disease, mushroom worker's lung, bagassosis, suberosis (cork mold), sequoiosis (redwood mold).

ROUTE OF ENTRY

Inhalation of the spores is the primary route of entry.

HARMFUL EFFECTS

Local—

None.

Systemic—

High exposure to the spores as in "farmer's lung" may cause an acute respiratory infection and the offending fungi may be cultured from the sputum. Repeated exposure sensitizes these individuals to the spores and these workers may develop acute asthmatic attacks even when the dust is very dilute in the atmosphere.

PREVENTIVE MEASURES

Prevention is possible through proper handling of products contaminated with molds, proper ventilation, and use of dust respirators able to retain the smallest spores. Once the hypersensitivity reaction is recognized, an individual should not be subjected to further exposure.

REFERENCES

1. Wenzel, F. J., and D. A. Emanuel. 1967. The epidemiology of maple bark disease. Arch. Env. Hlth. 14:385.
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3. Kaltreider, B. F. 1973. Hypersensitivity pneumonitis: Immunologically mediated lung disease resulting from the inhalation of organic antigens. J. Occup. Med. 15:949.
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5. Lacy, J. 1973. The air spora of a portuguese cork factory. Ann. Occup. Hyg. 16:223.
6. Nicholson, D. P. 1972. Extrensic allergic pneumonitis (editorial) Am. J. Med. 53:131.

PARASITIC DISEASES

Parasitic infections of occupational significance are caused by protozoa, helminths, and arthropods. Diseases caused by protozoa include malaria, amebiasis, leishmaniasis, trypanosomiasis, and a variety of less common blood and gastrointestinal infections. Helminthic diseases include schistosomiasis, creeping eruption, and hookworm. Arthropods such as mites and chiggers, may cause dermatoses and may act as vectors or hosts for other nonarthropod parasites.

Many parasitic diseases are zoonoses; that is, they are transmissible under natural conditions between vertebrate animals and man. Parasitic diseases may have reservoirs in infected wild or domestic animals, or in infected persons. Recent increases in the amount and speed of international travel have resulted in dissemination of parasitic diseases to areas where they have been unknown or uncommon. The physician should be alert to the possibility that diseases usually considered exotic or tropical may appear in urban areas in nontropical regions, and may even be transmitted under seemingly improbable circumstances.

Certain occupational groups are at great risk of contracting a parasitic disease because of their exposure to vectors carrying a parasitic disease, their direct contact with the infective form of a parasite, and indirectly their presence in areas where conditions are crowded or sanitation and hygiene are inadequate.

SWIMMER'S ITCH

POTENTIAL OCCUPATIONAL EXPOSURES

Individuals at risk include those required to work in and around fresh lakes, ponds, and swamps, e.g., skin divers, dock workers, watermen, and lifeguards.

SYNONYMS

Schistosome dermatitis, cercarial dermatitis, clam digger's itch, swamp itch.

ROUTE OF ENTRY

Route of entry is by penetration of certain species of freshwater schistosome cercariae through the wetted skin.

HARMFUL EFFECTS

Local—

Itching during the drying of exposed wetted skin surfaces is followed initially by localized redness and edema and later by pruritic macules and papules. Occasionally areas covered by clothing will also be affected.

Systemic—

None.

PREVENTIVE MEASURES

Exposed wetted skin should be thoroughly dried immediately. Cercariae in small bodies of water can be controlled by addition of a mixture of copper salts or by spraying with formaldehyde.

CREEPING ERUPTION

POTENTIAL OCCUPATIONAL EXPOSURES

Those occupations at highest exposure are ditch diggers, masons, gardeners, utility workers, laborers, plumbers, and lifeguards. It is prevalent in areas where hookworm eggs in cat or dog feces are deposited on warm sandy soil and subsequently develop into infective larvae.

SYNONYMS

Cutaneous larva migrans.

ROUTE OF ENTRY

Penetration of the skin is the route of entry. The eggs discharged in the feces develop into a filariform stage and then are capable of penetrating the skin. Transmission to man requires environmental temperature and humidity appropriate for development of the egg to the infective filariform larval stage. Beaches and other moist sandy areas are hazardous, because animals choose such areas for defecation.

HARMFUL EFFECTS

Local—

Larvae burrow into the skin, producing a serpiginous track of erythema, induration, and pruritis.

Systemic—

None.

PREVENTIVE MEASURES

Recreational areas should be kept free of dogs and cats. Individuals whose occupations require they work in moist, warm, sandy soil, particularly in the southern parts of the United States, should be supplied with rubber boots and gloves.

HOOKWORM DISEASE

POTENTIAL OCCUPATIONAL EXPOSURES

This disease prevails in tropical countries where defecation on soil by infected persons permits development of the larvae, and subsequent infection by others contacting the larvae. Warm climate, sandy soil, and moisture favor continuation of the cycle. Individuals whose occupations bring them into direct contact with the soil, for instance, barefoot farmers in the South, ditch diggers, sewer workers, and recreation workers and lifeguards at recreational beaches, are at high risk of exposure.

SYNONYMS

Ancylostomiasis, uncinariasis.

ROUTE OF ENTRY

The larvae invade the exposed skin.

HARMFUL EFFECTS

Local—

At the site where the larvae invade the skin, there is erythema and edema, with severe pruritis. The most frequent area of these penetrations is the feet, especially the interdigital area, and this circumstance has been called "ground itch" in those localities.

Systemic—

The hookworm migrates from the skin via the circulatory system through the lungs, to the gastrointestinal tract. The hookworm attaches to the wall of the small intestine. Nonspecific gastrointestinal symptoms occur. The major finding in these cases is an iron-deficiency anemia as a consequence of chronic intestinal blood loss caused by the parasites.

PREVENTIVE MEASURES

Education of rural populations and field workers to use sanitary facilities for the proper disposal of feces is necessary. Elimination of soil pollution is necessary in order to control this disease. There should

be mandatory wearing of shoes and/or rubber boots and the protection of hands by gloves to protect individual workers.

ASCARIASIS

POTENTIAL OCCUPATIONAL EXPOSURES

Ascariasis is a nematode infection which may be occupationally important where human feces are deposited on the soil rather than in sanitary facilities. Those occupations at high risk are the same as for hookworm.

SYNONYMS

None.

ROUTE OF ENTRY

The route of entry is the oral route, generally by the ingestion of embryonated eggs contained in contaminated food, or by the introduction of the eggs into the mouth by the hands after contact with contaminated soil.

HARMFUL EFFECTS

Local—

None.

Systemic—

After the eggs are ingested, the larvae are liberated in the small intestine. They migrate through the intestinal wall and ultimately reach the lung. After a short period of time, the larvae pass through the bronchioles, bronchi, trachea, and epiglottis, are swallowed, and develop into male and female adults in the small intestine. Clinical manifestations are usually diverse; severe bronchopneumonia can develop with heavy infections, as can abdominal pain in partial or complete intestinal obstruction.

PREVENTIVE MEASURES

As with hookworm this is a matter of education of those people in exposed areas to the proper disposal of feces and strict attention to personal hygiene (1,2).

REFERENCES

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2. Barrett-Connor, E. 1972. Human intestinal nematodiasis in the United States. Calif. Med. 117:8.

MITES, CHIGGERS, AND TICKS

POTENTIAL OCCUPATIONAL EXPOSURES

These are mainly ectoparasites, and may transmit disease. Eggs

are deposited in soil or on a host, and hatch into parasitic larvae which feed on blood. The chigger is commonly encountered in grassy areas in large areas of the United States but predominantly in the South. The larvae crawl up the legs of man or animals and attach to the skin. Those at highest risk are construction workers, linemen, farmers, pipeline workers, and surveyors.

The fowl or chicken mite which parasitizes both wild and domestic fowl is sometimes a parasite of man. Poultry workers are likely to be affected.

Mites of the family Pyemotidae attack man and cause a dermatitis called "straw itch" or "grain itch." Persons at risk include farmers, gardeners, potters, broommakers, and others handling wheat, barley, or straw. These mites exist in grain elevators, straw mattresses, and dust from farm machinery.

Mites belonging to the family Acaridae infest cereals, grains, copra, vanilla pods, and cheeses. Persons handling these products can become occupationally exposed.

The wood tick, the dog tick, the Lone Star tick, and the Gulf Coast tick have all been incriminated in the transmission of disease to man by their bite. Those individuals most likely to be subjected to tick bite are individuals whose occupations require them to be in contact with nature, such as forest rangers, woodsmen, employees of recreational areas, hunters, trappers, ranchers, and farmers.

SYNONYMS

None

ROUTE OF ENTRY

In all instances route of entry is by the bite of the parasite through the skin of man.

HARMFUL EFFECTS

Local—

Chigger bites appear on the lower legs and elsewhere at sites of clothing restriction. Itching and swelling surround the bite for one or two days, after which the surrounding area may become edematous, indurated, or dusty red in color. Itching may cause loss of sleep and fever and secondary infection may be present.

The fowl or chicken mite causes itching and dermatitis. This mite has been demonstrated to carry viruses which cause St. Louis encephalitis, Newcastle disease, and western equine encephalitis. Food mites produce pruritic papular eruptions on exposed areas, usually shoulders and chest. A tick bite is usually quite innocuous, and it is only the presence of the tick that draws attention to the bite. Removal of the tick is important in that the tick should not be crushed and care must be taken so that the head is also removed.

Systemic—

In most cases, none. Secondary infection occurs. In the case of the

tick, if the head is not completely removed or if the tick is allowed to remain for days feeding on human blood, a condition known as tick paralysis can develop, which is due to a neurotoxin which the tick apparently injects while engorging. This acts upon the spinal cord and bulbar nuclei causing incoordination, weakness, and paralysis.

PREVENTIVE MEASURES

Preventive measures include use of protective clothing, insecticides, and repellants and paying close attention to personal hygiene (1).

REFERENCE

1. Gouck, H. K. 1966. Protection from ticks, fleas, chiggers, and leaches. Arch. Dermatol. 93:112.

SECTION IV

Figure 4. Occupational contact dermatitis.

DERMATOSES

Donald J. Birmingham, M.D.

Any abnormality of the skin induced or aggravated by the work environment is termed an "occupational dermatosis." A dermatosis, therefore, can represent one or more alterations in the skin ranging from the mildest erythema and scaling to a complicated eczematous, acneform, pigmentary, neoplastic, granulomatous, or ulcerative disorder. Commonly used terms as "industrial dermatitis" or "occupational contact dermatitis" actually designate an inflammatory process of eczematous nature; whereas descriptive titles as "cutting oil acne," "tar melanosis," "pitch cancer," "silica granuloma," "chrome ulcer," among others, are used to identify cause and effect.

Occupational diseases of the skin are common for at least two reasons: first, the skin has a large surface area available for contact exposure; and, second, the work environment contains innumerable natural and artificial materials capable of exerting chemical, mechanical, physical, biological, or photoreactive insults to the skin. Causal agents, therefore, are diverse; but chemicals are by far the most frequent offenders.

Organic and inorganic chemicals are everywhere in modern industry, on the farm, and even in the household. In acting as primary irritants or allergic sensitizers or as photosensitizers, the usual clinical effect is contact eczematous dermatitis. This type of cutaneous response characterizes about 75 to 80 per cent of the occupational dermatoses observed and about four-fifths of these are due to contact with primary irritant chemicals. See Figure 4.

NATURAL PROTECTION

Anyone who works is a potential candidate for developing an occupational dermatosis; however, many workers are not affected with these disorders. Several intrinsic factors influence the behavior of the worker's skin, the most important of these is its own natural defense. Anatomically, the skin acts as a body envelope whose connective tissue, including the elastic fibers, provides flexibility which buffers moderate blunt mechanical trauma. The outermost layer of the skin, thickest on the palms and soles, is a complex protein called "keratin." Though composed of dead cells, the keratin layer is a most important line of cutaneous defense. It protects against the rapid entrance of water and water-soluble chemicals and, similarly, prevents mass loss of water from the body. In a limited way, it offers resistance to mild acids and water; but its chemical nature does not furnish satisfactory defense against alkalis, strong acids, solvents, or prolonged immersion. These agents disrupt the chemical nature and the cohesiveness of the keratin cells, thereby, weakening their barrier effect. Protection also results from the increased thickening of the keratin layer which follows repetitive trauma or the action of ultraviolet rays.

Keratin cells are covered by a film of sweat and sebaceous gland material (sebum) mixed with the by-products of the keratinizing epidermis. All of these materials make up the acid surface film, sometimes called the "acid mantle." This film may provide some impedance to the rapid entrance of water and water-soluble chemicals; but its ready removal by water, soap, detergents, and solvents obviously limits its effectiveness in defense.

Residing below the keratin layer are the living epidermal cells which stem from the basal cells located in the lowermost area of the epidermal layer. Basal cells or germinative cells supply the epidermal cells, which in turn, become keratin cells. Located among the basal cells are the pigment-forming elements, the "melanocytes," which manufacture melanin. This material, the product of an enzyme reaction, is picked up by the epidermal cells and migrates upward with them, at the same time furnishing protection against sunlight and ultraviolet rays.

Blood vessels, nerves, sweat glands, and sebaceous glands are located within the dermis. The major defense against heat is furnished by the interaction of blood vessels, nerves, and sweat glands. Located within the upper dermis are numerous nerve receptors and effectors which delineate sensory perception and, thereby, afford protection against certain external stimuli. An antimicrobial capacity appears to be present in the skin and probably is influenced by the skin's secretions and excretions, products arising from the cellular turnover of the epidermal cells, and the inherent dryness which the skin possesses.

PRIMARY IRRITANTS

Most occupational dermatitis is caused by primary irritant chemicals. These agents will cause dermatitis by direct action on the normal skin at the site of contact if they are permitted to act in sufficient intensity or quantity for a sufficient length of time. Thus, any normal skin will almost always react to a primary irritant if the above requirements are met. Strong or absolute irritants as sulfuric acid, sodium hydroxide, or methyl bromide produce an observable effect within a few minutes, depending upon their concentration. Conversely, weak or marginal irritants as acetone, soap and water, and mineral oil may require several days before a clinically recognizable change occurs.

The precise mechanism of how primary irritants disturb the skin is far from understood, but clinical effect generally varies with the degree of injury. Strong acids react vigorously with skin to form acid albuminates. Clinically, an acid burn resembles a thermal burn. Strong alkalis combine with fats and also dissolve protein, including the cytoplasm of keratin cells. Marginal alkaline irritants produce less destruction of tissue, but subtly cause dehydration of the keratin by inducing loss of cell cohesion, cracking, and loss of continuity.

Lipid solvents remove the surface film, disturb the water-holding quality of the keratin cells, and injure the membranous structure of epidermal cells. Metallic salts of arsenic, mercury, zinc, and chromium

precipitate skin protein and if the concentration of these materials is sufficient, ulceration of the skin occurs. Otherwise, contact dermatitis results. Oxidizing and reducing agents also injure keratin and epidermal cells. The events which occur in the various layers following exposure to most of the irritant chemicals are not well understood.

ALLERGIC SENSITIZERS

It is generally estimated that 20 per cent of occupational contact dermatitis is caused by allergenic materials. Naturally, this will vary in any industrial plant, depending upon the materials being handled. Practically any chemical can act as a sensitizer, but certain ones are more conspicuous because of this capacity. Some examples are: poison ivy, poison oak, epoxy monomers and their amine hardeners, potassium dichromate, nickel, formaldehyde, tetramethylthiuram disulfide, and mercaptobenzothiozole.

Cutaneous sensitizers differ from primary irritants in their mode of action and in the effects they create within the skin. Most sensitizers do not produce demonstrable cutaneous changes on first contact or perhaps for many days or weeks. However, the sensitizer induces certain specific cellular changes in the skin so that after a period of incubation (five to seven days or more) further contact with the same or a closely related agent on the same or other parts of the body results in an acute dermatitic reaction.

The essential differences between primary irritation and allergenic sensitization, therefore, are time and a different mechanism of action. Another difference which may be noted is that an irritant usually affects a number of workers whereas a sensitizer generally affects a few. This, of course, does not apply to potent sensitizers as poison ivy or epoxy resin systems. In spite of these differences, it can be extremely difficult to distinguish between a marginal irritant and a cutaneous sensitizer because the marginal irritant also requires prolonged or repeated contact before a dermatitis appears.

PHOTOSENSITIZERS

Photosensitivity is the capacity of an organ or organism or certain chemicals and plants to be stimulated to activity by light or to react to light. Photosensitizers are divided into "phototoxic" and "photoallergic" materials. Several derivatives of coal tar, e.g., anthracene, phenanthrene, and pitch, possess this activity.

Photobiologic effects as melanosis or photodermatitis are associated with specific chlorinated hydrocarbons, particularly the chlorobenzols, the diphenyls, and the triphenyls; dyes as acridine, eosin, and rose bengal; certain essential oils as bergamot; and a number of plants as limes, wild parsnip, fennel, and "pink-rot" celery.

Phototoxicity, like primary irritation, can affect anyone; however, heavily pigmented skin is more resistant. Similarly, photoallergens, like allergic sensitizers, affect far fewer people.

DIAGNOSIS

Recognizing and diagnosing an occupational dermatosis, like any cutaneous disease, depends upon satisfying several basic criteria. These fundamental tenets deal with the morphology or appearance of the eruption, its location, the history and course of the disease, and laboratory data.

The eruption should look like a contact dermatitis or one of the other clinical types classified as an occupational dermatosis. Lesions should be located on sites of greatest exposure, notably the backs of the hands, the volar surfaces of the wrists, the forearms, and the cubital areas—particularly when dusts and liquids are the contactants. Involvement of the face, eyelids, ears, and "V" of the neck usually indicates exposure to fumes, vapors, or mists. Excessively dusty exposures can affect large areas of the skin by direct contact or by manual transmission.

When the disease is suspected of being occupational, the history should reveal that the rash occurred after beginning work and that periods of remission and recurrence correlate with work exposure. A thorough history reveals the substances being handled at work and at home, including medications applied or ingested; the manner in which contact has been made; cleansing habits with soaps or solvents; and the use of protective equipment, including barrier creams, gloves, etc.

Most workers with an active occupational dermatosis relate that the eruption diminishes, but rarely disappears completely, over the weekend. Additionally, the eruption almost always worsens upon return to work after the weekend. When the eruption fails to clear after six to eight weeks of sick leave or after complete withdrawal from the suspected contact agent through a job change, other reasons for the dermatitis should be explored.

Actually, the prolonged or recurrent eruption may represent an underlying disorder as atopic dermatitis, psoriasis, nonoccupational contact dermatitis, nummular eczema, among other nonoccupational diseases. Certain laboratory procedures may be indicated and may further define the disease process. These may include skin scrapings and culture for detecting the presence of a superficial fungus, anaerobic or aerobic cultures for bacteria, histopathologic diagnosis following skin biopsy, diagnostic patch tests or photopatch tests to detect an allergen. Additional analytical tests for porphyrins in urine and for metals in urine or blood, skin or hair may also be indicated.

CLINICAL CLASSIFICATION

Occupational dermatoses have considerable morphologic variety. Their appearance and pattern rarely indicate the provoking substance, but may provide a clue as to a class of materials being encountered. Diagnosis depends upon appearance and location, but mostly upon history. Supervening infections or undesirable therapeutic effects make the diagnosis more difficult.

Despite the numerous clinical reactions the skin can display, the

following groupings comprise the majority of the occupational dermatoses:

(1) Acute contact eczematous dermatitis characterized by erythema, edema, papules, vesicles or bullae, crust, scale, and finally, desquamation. These are the signs of an inflammatory eczematous dermatitis caused by contact with a primary irritant or a sensitizer or a photosensitizer.

(2) Chronic eczematous dermatitis characterized by erythema, lichenification, scaling, dryness, and fissuring resulting from contact with substances which dehydrate the skin as alkali, liquids and dusts, solvents, soaps and detergents.

(3) Folliculitis and acneform dermatoses including chloracne characterized by plugged sebaceous follicles and nodular and suppurative lesions. Chloracne also shows multiple cystic lesions which contain straw-colored material. These dermatoses are caused by contact with insoluble oils, greases, tars, waxes, and certain chlorinated hydrocarbons as the chloronapthalenes.

(4) Neoplastic (benign and malignant) types as keratoses, papillomata, epitheliomas, and carcinomas of the exposed areas. These usually are caused by certain petroleum products, coal tar and certain derivatives, sunlight, and ionizing radiation.

(5) Pigmentary disturbances characterized by an increase or decrease of pigment in the epidermis. Increased pigmentation can result from contact with coal tar compounds, certain petroleum oils, vegetables, fruits, sunlight, and trauma. Decreased or absent pigmentation may result from burns; forceful trauma; chronic dermatitis; monobenzyl ether of hydroquinone; and certain phenolics as tertiary butyl catechol, tertiary amyl phenol, and tertiary butyl phenol.

(6) Granulomatous dermatoses characterized by chronic indolent focal inflammations which tend to heal with scar. These lesions can result from bacterial, viral, fungal or inanimate agents as asbestos, beryllium, and silica.

(7) Ulcerative lesions characterized by a loss of tissue on a cutaneous or mucous membrane surface leading to necrosis. Ulcerations can be caused by arsenic trioxide, calcium compounds, cement and concrete, chromic acid, burns and trauma. They may also result from purposeful or unconscious manipulation.

(8) Miscellaneous lesions. Some occupational dermatoses, because of their unusual nature, do not fit into the above classifications. Among such miscellaneous lesions are:

(a) alopecia induced by chloroprene;

(b) acro-osteolysis, with or without Raynaud's;

(c) sclerodermoid changes believed due to vinyl chloride polymerization;

(d) discolorations of the hair, skin, and nails due to various chemicals;

(e) porphyria cutanea tarda caused by a certain chlorinated hydrocarbon intermediate.

OCCUPATIONS AND AGENTS

The following is a list of occupations each accompanied by certain agents frequently associated with that occupation and capable of producing a dermatosis. Additional agents for the occupations listed as well as additional occupations will be found in other sections, principally the one on chemical hazards.

Abrasive Wheel Makers
- carborundum
- emery
- resin glues

Agricultural Workers
- See Farmers

Aircraft Workers
- adhesives (resins)
- alkalis
- bichromates
- chromates
- chromic acid
- cutting fluids
- cyanides
- epoxy resins
- flame retardants
- glass fibers
- hydraulic fluids
- hydrofluoric acid
- lubricants
- nitric acid
- oils
- paints
- plastics
- rubber
- solvents
- thinners
- ultraviolet light
- vibrating tools
- X-rays

Animal Handlers
- antibiotics
- bacteria
- cleaners & detergents
- deodorants
- feeds
- fungi
- germicides
- insecticides
- medicaments
- parasites
- pesticides
- viruses

Artists (Painters)
- acrylics
- epoxies
- paint removers
- pigments
- plasticizers
- solvents

Artists (Sculptors)
- dusts
- plaster of Paris
- pneumatic tools
- polishes

Athletes
- adhesives
- antibiotics
- bacteria
- lime
- medications
- protective gear
- soaps

Automobile Workers (Assembly)
- adhesives
- asbestos
- antifreeze
- brake fluids
- brake linings
- flame retardants
- gasoline
- hydraulic fluids
- oils
- rubber
- solvents

Automobile Workers (Body)
- abrasives
- adhesives
- alkalis
- lead
- paints
- rubber compounds
- solder
- solvents

Automobile Workers (Mechanic)
- acids
- adhesives
- alkalis
- antifreeze
- brake fluids
- brake linings
- cleansers
- epoxy resins
- gasoline
- hydraulic fluids
- lubricants
- rubber
- solvents
- thinners

Bakers
- benzoyl peroxide
- cinnamon
- dough
- dusts
- flavors (oils)
- flour
- fungi
- heat
- moisture
- spices
- sugar

Barbers
- ammonium thioglycolate
- antiseptics
- bacteria
- cosmetics
- depilatories
- detergents
- dyes
- fungi
- hair conditioners
- hair sprays
- hair straighteners
- hair tonics
- perfumes
- shampoos
- shaving creams
- ultraviolet
- vibrating machines
- wave solutions

Bartenders
- citrus fruits
- detergents
- disinfectants
- flavors
- moisture
- soaps

Bath Attendants
- deodorants
- fungi
- liniments
- lotions
- oils
- soaps
- ultraviolet

Battery Makers
- alkali
- cobalt
- epoxy sealer
- fiber glass plates
- mercury
- moisture
- nickel
- pitch
- plastics
- solvents
- sulfuric acid
- zinc chloride

Bleachers
- borax
- chlorine compounds
- hydrochloric acid
- hydrogen peroxide
- oxalic acid

Bleachers (cont'd)
- per-salts
- potassium hydroxide
- sodium hydroxide
- solvents

Bookbinders
- formalin
- glues (natural)
- glues (resin)
- inks
- shellac
- solvents

Brick Masons
- cement
- chromates
- cold
- epoxy resins
- lime
- moisture
- sunlight

Briquette Makers
- coal tar pitch

Bronzers
- acetone
- ammonia
- ammonium sulfide
- amyl acetate
- antimony sulfide
- arsenic
- arsine
- benzine
- benzol
- cyanides
- heat
- hydrochloric acid
- lacquers
- mercury
- methyl alcohol
- petroleum hydrocarbons
- phosphorus
- resins
- sodium hydroxide
- sulfur dioxide
- turpentine
- varnishes

Broom Makers
- bacteria
- bleaches
- dust, vegetable
- dyes
- fungi
- glues - natural & resin
- parasites
- pitch
- plastics
- rubber
- shellac
- solvents
- tar
- varnish
- woods

Brush Makers
- See Broom Makers

Butchers
- antibiotics
- bacteria
- brine
- cold
- detergents
- enzymes
- fungi
- moisture
- parasites

Button Makers
- bacteria
- dusts - animal, vegetable, mineral
- dyes
- hydrogen peroxide
- plastics

Cabinet Makers
- bleaches
- glues - resin & casein
- insulation agents
- oils
- polishes
- rosin
- shellac
- solvents

Cabinet Makers (cont'd)
- stains
- woods

Cable Splicers
- chlorinated diphenyls
- chlorinated naphthalenes
- dyes
- epoxy resins
- solvents

Cable Workers
- See Cable Splicers

Candle Makers
- ammonium salts
- borax
- boric acid
- chlorine
- chromates
- hydrochloric acid
- potassium nitrate
- sodium hydroxide
- stearic acid
- waxes

Candy Makers
- chocolate
- citric acid
- dyes - food
- essential oils (flavors)
- fruits
- pineapple juice
- spices
- sugar
- tartaric acid

Canners
- bacteria
- citrus oil
- dyes
- enzymes
- fruit acids & sugars
- fungi
- moisture
- parasites
- resins
- salt
- vegetable juices

Carpenters
- See Cabinet Makers

Carpet Makers
- alizarine dye
- aniline dyes
- anthrax bacillus
- bleaches
- chlorine
- fungicides
- glues
- insecticides
- jute
- loom oils
- solvents

Carroters - Felt Hat
- acids
- anthrax bacillus
- quinones

Case Hardeners
- heat
- oils (quench)
- sodium carbonate
- sodium cyanide
- sodium dichromate
- sodium nitrite

Cellulose Workers
- acids
- alkalis
- bleaches
- carbon disulfide
- finishing oils

Cement Workers
- cement
- chromates
- cobalt
- epoxy resins
- lime
- moisture
- pitch
- resins

Chemical Workers
- See Section VII

Chrome Platers
- chromium compounds
- degreasers (solvents)
- metal cleaners (alkali)
- sulfuric acid

Clerks
- adhesives
- carbon paper
- copy paper
- duplicating fluids
- duplicating materials
- indelible pencils
- ink removers
- inks
- rubber
- solvents
- type cleaner
- typewriter ribbons

Cloth Preparers
- acids
- alkalis
- amino resins
- detergents, synthetic
- dyes
- flame retardants
- formaldehyde
- fungicides
- moisture
- potassium salts
- soaps
- sodium metasilicate
- sodium salts
- sodium silicate

Coal Tar Workers
- anthracene oil
- benzol
- coal tar
- creosote
- cresol
- naphtha
- pitch
- solvents
- sunlight

Compositors
- alkalis
- inks
- solvents

Construction Workers
- adhesives, resin
- cement
- concrete
- cold
- creosote
- gasoline
- glass fiber
- oils
- paints
- pitch
- poisonous plants
- sealers
- solvents
- sunlight
- ultraviolet light
- wood preservatives
- woods

Cooks
- fruit acids
- heat
- moisture
- monilia
- spices
- sugars
- vegetable juices

Cotton Sizers
- acids
- aluminum salts
- arsenic salts
- calcium salts
- dicyandiamide formaldehyde
- fungicides
- magnesium salts
- melamine formaldehyde
- sodium hydroxide
- starch
- urea formaldehyde
- zinc chloride

Dairy Workers
- antibiotics
- bacteria

Dairy Workers (cont'd)
- deodorants
- detergents
- fungi
- mites
- viruses

Degreasers
- alkalis
- chlorinated hydrocarbon solvents
- petroleum solvents
- ultrasonic devices

Dentists
- anesthetics, local
- antibiotics
- bacteria
- disinfectants
- eugenol
- ionizing radiation
- mercury & metallic amalgams
- oil of clove
- resins
- soaps
- waxes

Dishwashers
- bacteria
- detergents, synthetic
- grease
- moisture
- monilia
- soaps
- water softeners

Disinfectant Makers
- carbolic acid
- chloride of lime
- chlorinated phenols
- chlorine
- cresol
- formaldehyde
- iodine
- mercurials
- quarternary ammonium compounds
- surfactants
- zinc chloride

Dock Workers
- bacteria
- castor bean pomace
- chemicals
- cold
- fumigants
- fungi
- grains
- heat
- insecticides
- insects
- irritating cargoes
- mites
- moisture
- petroleum
- sunlight
- tar

Druggists
- acids
- alkalis
- antibiotics
- bleaching powder
- detergents, synthetic
- drugs
- iodoform
- soaps
- sugar

Dry Cleaners
- acetic acid
- ammonia
- amyl acetate
- benzine
- carbon tetrachloride
- dusts
- methanol
- nitrobenzene
- perchloroethylene
- sizing chemicals
- Stoddard solvent
- trichloroethylene
- turpentine
- waterproofing chemicals

Dye Makers
- acids
- alkalis

Dye Makers (cont'd)
- antimony compounds
- benzine
- calcium salts
- coal tar products
- cresol
- dextrins
- dye intermediates
- ferrocyanides
- formaldehyde
- gums
- hydroquinone
- lead salts
- potassium chlorate

Dyers
- acids
- alkalis
- bleaches
- detergents, synthetic
- dyes
- mercurial salts
- moisture
- solvents
- zinc chloride

Electric Apparatus Makers
- acids
- asbestos
- chlorinated diphenyls
- chlorinated naphthalenes
- enamels
- epoxy resins
- ionizing radiation
- phenolic resins
- pitch
- rubber
- solder fluxes
- solvents
- synthetic waxes
- varnishes

Electricians
- chlorinated diphenyls
- chlorinated naphthalenes
- electricity
- epoxy resins
- solder fluxes
- solvents
- waxes, synthetic

Electroplaters
- acids
- alkalis
- benzine
- chromic acid
- heat
- lime
- moisture
- nickel
- potassium cyanide
- soaps
- waxes, chlorinated
- zinc chloride
- zinc cyanide

Embalmers
- bacteria
- formaldehyde
- fungi
- ionizing radiation
- mercury
- oil of cinnamon
- oil of clove
- phenol
- thymol
- zinc chloride

Enamelers
- acids
- alkalis
- antimony
- arsenic
- chromium
- cobalt
- nickel

Engravers
- acids
- alkalis
- chromic acid
- ferric chloride
- potassium cyanide
- solvents
- tropical woods

Etchers
- acids
- alkalis

Explosive Workers
- ammonium salts
- mercury compounds
- nitroglycerin
- PETN
- picric acid
- tetryl
- TNT

Farmers
- antibiotics
- bacteria
- cold
- detergents, synthetic
- disinfectants
- feeds
- fertilizers
- fruits
- fungi
- fungicides
- heat
- lubricants
- oils
- paints
- parasites
- pesticides
- poison ivy, oak, sumac
- ragweed
- solvents
- sunlight
- tar
- vegetables
- wood preservatives

Felt Hat Makers
- acids
- bacteria
- dyes
- Glauber's salt
- hydrogen peroxide
- mercuric nitrate (if used)
- sodium carbonate

Fertilizer Makers
- acids
- ammonium compounds
- calcium cyanamide
- castor bean pomace
- fluorides
- lime
- manure
- nitrates
- pesticides
- phosphates
- potassium salts

Fish Dressers
- bacteria
- brine
- cold
- moisture
- redfeed
- sunlight
- trauma

Florists
- bacteria
- bulbs
- fertilizers
- fungi
- herbicides
- parasites
- poisonous plants

Flour Mill Workers
- chemical bleaches
- dusts
- parasites
- pesticides

Food Preservers
- bleaches
- brine
- ionizing radiation
- moisture
- monilia
- resins
- spices
- sugar
- vinegar
- waxes

Foundry Workers
- acids
- heat
- lime
- resin binder systems
- solvents
- ultraviolet light

Fur Processors
- acids
- alkalis
- alum
- bacteria
- bleaches
- chromates
- dyes
- formaldehyde
- fungi
- lime
- oils
- salt

Furnace Workers
- heat
- ultraviolet light

Furniture Polishers
- acids
- alkalis
- benzine
- essential oils in polish
- methyl alcohol
- naphtha
- pyridine
- rosin
- soaps
- solvents
- stains
- turpentine
- waxes

Galvanizers
- acids
- alkalis
- zinc chloride

Garage Workers
- air guns (grease)
- antifreeze
- detergents, synthetic
- epoxy resins
- gasoline
- gasoline additives
- glass fiber
- greases
- moisture
- oils
- paint removers
- paints
- solvents

Gardeners
- bacteria
- fertilizers
- fungi
- fungicides
- herbicides
- insecticides
- insects
- plants
- poison ivy
- poison oak
- sunlight

Glass Workers
- arsenic
- borax
- boric acid
- glass fiber
- glass wool
- heat
- hyrdofluoric acid
- lead compounds
- lime
- metallic oxides
- petroleum oils
- resins
- soda ash
- ultraviolet light

Hairdressers
- See Barbers

Highway Workers
- See Road Workers

Histology Technicians
- alcohol
- aniline
- benzol
- epoxy resins
- formaldehyde
- mercury bichloride
- osmium tetroxide
- potassium dichromate
- stains
- toluene
- waxes
- xylene

Ink Makers
- anti-skinning agents (antioxidants)
- chrome pigments
- cobalt compounds (driers)
- detergents, synthetic
- dyes
- mercurial pigments
- resins
- soaps
- solvents
- turpentine
- varnishes

Insecticide Makers
- aldrin
- allethrin
- arsenic trioxide
- calcium arsenate
- chlordane
- DDT
- dieldrin
- lindane
- malathion
- methoxychlor
- parathion
- piperonyl compounds
- pyrethrin
- strobane
- See also Pesticides section

Janitors
- bacteria
- detergents, synthetic
- disinfectants
- house plants
- polishes (essential oils)
- soaps
- solvents
- waxes

Jewelers
- acids
- adhesives, resin
- chromium
- cyanides
- mercury
- mercury solvents
- nickel
- rouge
- solder flux

Laboratory Workers, Chemical
- acids
- alkalis
- chromates
- detergents, synthetic
- moisture
- organic chemicals
- soaps
- solvents

Laundry Workers
- alkalis
- bactericides
- bleaches
- chemical dusts
- detergents, synthetic
- enzymes
- fiber glass
- fungicides
- heat
- moisture
- optical brighteners
- soaps

Linoleum Makers
- asphalt
- dyes
- oils
- pigments
- resins
- solvents

Longshoremen
See Dock Workers

Machinists
antioxidants
aqueous cutting fluids, synthetic
chlorinated cutting oils
chromates
germicides
greases
insoluble cutting oils
lubricants
rust inhibitors
soluble cutting fluids
solvents

Masons
See Brick Masons

Match Factory Workers
ammonium phosphate
chromates
dextrins
dyes
formaldehyde
glues
gums
phosphorus sesquisulfide
potassium chlorate
red phosphorus
waxes

Meat Packers
See Butchers

Mechanics
See Aircraft Workers, Automobile Workers, & Garage Workers

Mercerizers
acids
alkalis
heat
moisture

Metal Polishers
abrasives
acids
alkalis
ammonia
naphtha
pine oil
potassium cyanide
soaps
soluble oils
solvents
triethanolamine
waxes
wood (pine)

Mirror Makers
acids
ammonia
cyanides
formaldehyde
lacquers
silver nitrate
solvents
tartaric acid
varnishes

Mordanters
acids
alkalis
aluminum salts
antimony compounds
arsenates
chromates
copper salts
iron salts
lead salts
phosphates
silicates
tin salts
zinc chloride

Nickel Platers
acids
alkalis
degreasers
detergents, synthetic
heat
moisture
nickel sulfate
zinc chloride

Nitroglycerin Makers
- ethylene glycol dinitrate
- nitric acid
- nitroglycerin
- sodium carbonate
- sulfuric acid

Nurses
- anesthetics, local
- antibiotics
- antiseptics
- bacteria
- detergents, synthetic
- disinfectants
- drugs
- ethylene oxide
- fungi
- ionizing radiation
- moisture
- rubber gloves
- soaps
- tranquilizers
- viruses

Oil Field Workers
- acids
- alkalis
- brine
- crude petroleum
- explosives
- ionizing radiation
- lubricating oils
- sunlight

Optical Workers
- alkalis
- grinding fluids
- oils
- turpentine

Packinghouse Workers
- antibiotics
- bacteria
- bone fragments
- brine
- cold
- detergents, synthetic
- enzymes
- fungi
- parasites
- spices

Paint Makers
- anti-mildew agents
- chromates
- coal tar distillates
- driers
- fish oils
- latex
- oil, vegetable
- petroleum solvents
- pigments
- plasticizers
- resins
- thinners
- turpentine
- zinc chloride

Painters
- acetone
- acids
- alkalis
- benzine
- chlorinated hydrocarbons
- chromates
- driers
- paint strippers
- paints, oil base
- paints, resin
- pigments
- solvents
- thinners
- turpentine

Paper Box Makers
- anti-flame agents
- dyes
- glues, natural & resin
- mildew proofers
- waxes

Paraffin Workers
- paraffin
- paraffin distillates
- solvents

Pencil Makers
- aniline dyes
- chromium pigments
- glues
- gums
- lacquer
- lacquer thinners
- methyl violet
- pyridine
- red cedar wood
- resins
- solvents
- waxes

Petroleum Refinery Workers
- acids
- alkalis
- aluminum chloride
- gas oil
- gasoline
- hydrofluoric acid
- kerosene
- paraffin
- paraffin distillates
- petroleum
- petroleum solvents
- tar
- waxes

Photoengravers
- ammonium bichromate
- etching acids
- inks
- photo developers
- solvents
- ultraviolet light

Photographers
- acids
- alkalis
- chromates
- hydroquinone
- methyl para-aminophenol sulfate
- para-aminophenol
- paraformaldehyde
- paraphenylenediamines
- pyrogallic acid
- sodium hypochlorite
- sodium sulfide
- turpentine

Physicians
- adhesives
- anesthetics, local
- antibiotics
- antiseptics
- bacteria
- detergents, synthetic
- drugs
- fungi
- ionizing radiation
- soaps
- tranquilizers
- viruses

Pipeline Layers
- burns
- fluxes, welding
- ionizing radiation
- parasites
- poisonous plants
- solvents
- sunlight
- tar
- ultraviolet, welding

Pitch Workers
- heat
- pitch
- solvents
- sunlight
- tar

Plasterers
- lime
- moisture

Plastics and Resin Makers
See Section VII

Plumbers
- adhesives
- caulking compound
- cold

Plumbers (cont'd)
- fluxes, solder
- hydrochloric acid
- parasites
- solvents
- tar
- zinc chloride

Printers
- alkalis
- aniline
- chromates
- glues
- gums
- inks
- roller wash
- solvents

Railroad Shop Workers
- alkalis
- antiseptics
- chlorinated hydrocarbons
- chromate (antioxidants)
- cutting fluids
- detergents, synthetic
- diesel fuel oil
- greases
- insecticides
- lacquers
- lubes
- Magnaflux (fluorescein)
- paint
- paint strippers
- paint thinners
- solvents
- ultraviolet light

Railroad Track Workers
- cold
- creosote
- fungicides
- heat
- herbicides
- pitch
- poisonous plants
- sunlight
- tar

Rayon Workers
- acetic anhydride
- acids
- alkalis
- ammonium sulfide
- bleaches
- calcium bisulfite
- carbon disulfide
- coning oils
- sodium cyanide
- sodium sulfide
- sodium sulfite
- solvents

Refrigeration Workers
- ammonia
- brine
- chromates
- cold
- dry ice
- ethyl bromide
- ethyl chloride
- glass fiber
- methyl chloride
- sulfur dioxide

Road Workers
- asphalt
- cement
- cold
- concrete
- epoxy resins
- herbicides
- paint
- parasites
- pitch
- poisonous plants
- sunlight
- tar

Rocket Fuel Handlers
- aniline
- boron hydrides
- chlorine trifluoride
- dimethylhydrazine
- ethyl oxide
- fuming nitric acid
- gasoline

Rocket Fuel Handlers (cont'd)
- hydrazine
- hydrogen fluoride
- hydrogen peroxide
- kerosene
- liquid oxygen

Rope Makers
- alkalis
- bleaches
- dusts
- dyes
- oils
- pitch
- soaps
- tar

Rubber Workers
- accelerators
- acids
- activators
- adhesive removers
- alkalis
- antimony
- antioxidants
- benzol
- chloroprene dimers
- chromium pigments
- formaldehyde
- heat
- oils
- plasticizers
- resins
- retarders
- soaps
- solvents
- tar
- turpentine
- zinc chloride

Shipyard Workers
- burns (welding)
- chlorinated diphenyls
- chlorinated naphthalenes
- chromates
- cold
- fungicides
- glass fiber
- paint removers
- paint thinners
- paints
- resins
- solvents
- tar
- ultraviolet light
- wood preservatives

Shoemakers (Manufacturers)
- adhesives
- ammonia
- amyl acetate
- amyl alcohol
- aniline dyes
- benzine
- benzol
- fungicides
- hexane
- naphtha
- resins
- rubbers
- shoe polishes
- tanning agents
- waxes

Slaughterhouse Workers
- See Packinghouse Workers

Soap Makers
- alkalis
- bacteriostats
- detergents, synthetic
- oils, vegetable
- perfumes

Solderers
- acids
- cyanides
- fluxes
- heat
- hydrazine salts
- rosin
- zinc chloride

Stevedores
- See Dock Workers

Stockyard Workers
- bacteria
- fungi
- insecticides
- parasites

Stone Workers
- cement
- cold
- dusts
- heat
- lime
- vibrating tools

Sugar Refiners
- acids
- burlap
- fungi
- heat
- jute
- lime
- sugar

Tannery Workers
- acetic acid
- alum
- ammonium chloride
- arsenic salts
- bacteria
- benzol
- brine
- calcium hydrosulfide
- chromium compounds
- dimethylamine
- dyes, mineral
- dyes, vegetable
- formaldehyde
- lime
- oils
- pancreatic extract
- sodium hydroxide
- sodium sulfide
- solvents
- sulfuric acid
- tannin

Tar Workers
- heat
- pitch
- solvents
- sunlight
- tar

Taxidermists
- anthrax bacillus
- arsenic salts
- bacteria
- calcined alum
- fungi
- mercuric chloride
- parasites
- solvents
- tannin
- zinc chloride

Temperers
- oils
- sodium carbonate
- sodium cyanide
- sodium dichromate
- sodium nitrite

Tinners
- paint
- pitch
- sunlight
- zinc chloride

Typists
- See Clerks

Undertakers
- See Embalmers

Upholsterers
- bacteria
- flame retardants
- fungi
- glues
- lacquer
- lacquer solvents
- methyl alcohol
- parasites

Veterinarians
- anesthetics, local

Veterinarians (cont'd)
- antibiotics
- bacteria
- deodorants
- drugs
- fungi
- mercuric chloride
- parasites
- pesticides
- soaps & detergents
- viruses

Watchmakers
- acids
- chromates
- metal polishes
- nickel
- potassium cyanide
- rouge
- solvents

Waterproofers
- aluminum sulfate
- melamine formaldehyde resins
- oils
- paraffin
- pitch
- resin paints
- rubber
- solvents
- tar
- waxes

Welders
- fluxes
- heat
- ultraviolet light

Wire Drawers
- alkalis
- drawing oils
- lime
- soaps
- sulfuric acid

Wood Preservers
- chlorophenols
- chromates
- copper compounds
- creosote
- cresols
- mercuric chloride
- phenylmercuric compounds
- resins
- tar
- zinc chloride
- zinc sulfate

Wood Workers
- acid bleaches
- amino resin glues
- epoxy glues
- fillers
- formaldehyde
- lacquers
- oil stains
- paints
- phenolic resin glues
- rosin
- solvents
- varnishes
- woods

See also Cabinet Makers

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SECTION V

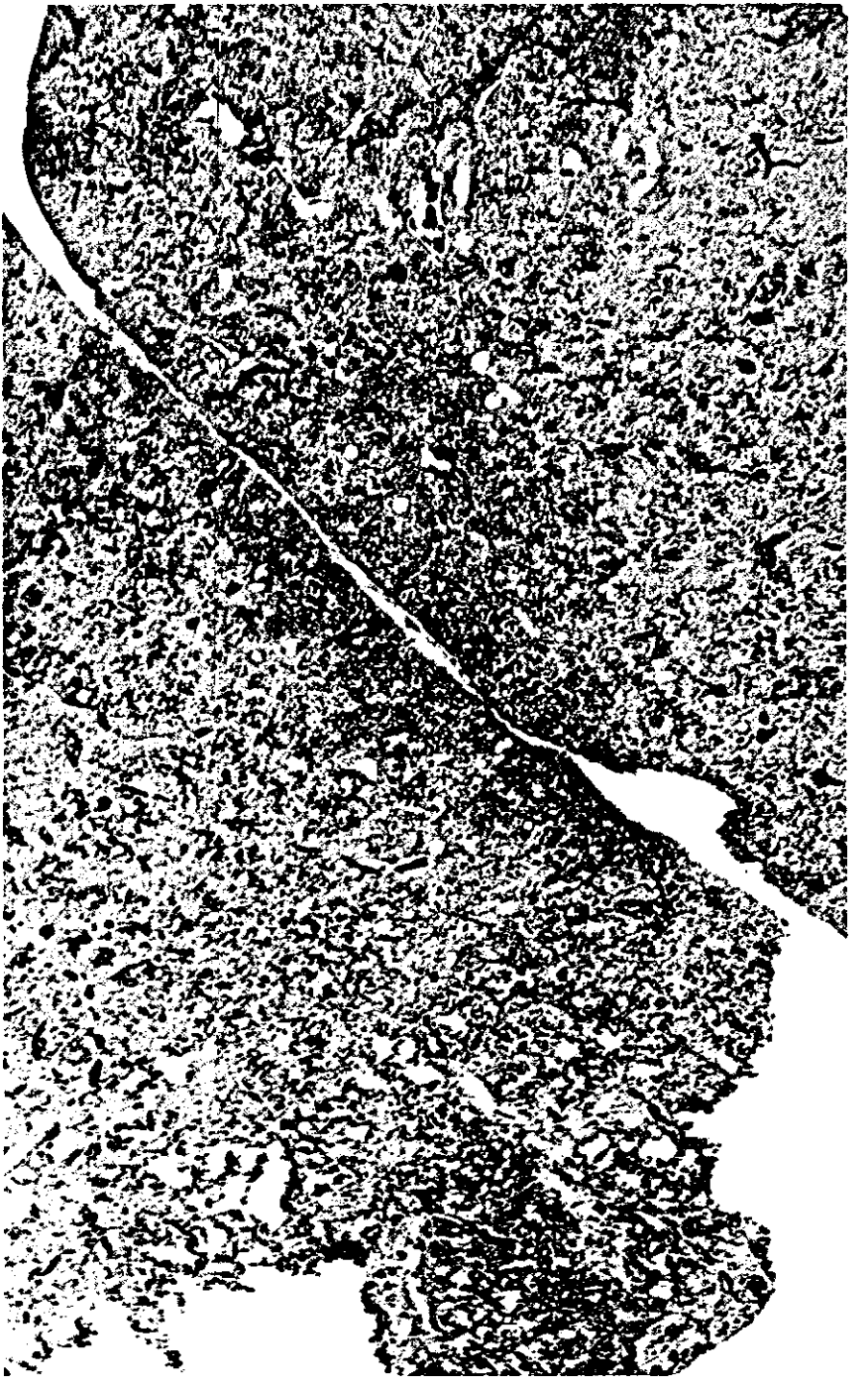

Figure 5. Coal worker's pneumoconiosis — Gough section.

DISEASES OF THE AIRWAYS AND LUNGS

W. Keith C. Morgan, M.D., and N. LeRoy Lapp, M.D.

AIRWAYS MECHANISMS AND RESPONSES

Gas exchange takes place in the acini of the lung parenchyma; that is, in those portions of the lung from the first order of respiratory bronchioles down to the alveoli. These respiratory bronchioles originate from the terminal bronchioles which are the smallest airways not concerned with gas exchange. Before inspired air can participate in gas exchange it must travel through a series of conducting tubes (the bronchial tree) until it finally reaches the first order respiratory bronchioles. The conducting system of airways does not participate in gas exchange and is, therefore, often known as the dead space. Inhaled particles may be deposited either in the lung parenchyma (the respiratory bronchioles, atrial sacs, and alveoli) or in the dead space. Some of the inhaled particulates are breathed in and out of the respiratory tract without deposition.

The site of deposition of an individual particle is governed by three factors: first, the aerodynamic properties of the particle, viz. the size, shape, speed, and density; secondly, the circumference and shape of the airway; and thirdly, the breathing pattern. Particles between 0.5 and 5 microns in diameter tend to be deposited in the alveoli and respiratory bronchioles and may, under certain conditions, cause a group of diseases known as the pneumoconioses to develop. Larger particles are in the main deposited in the conducting system of the lungs. The effect of an inhaled dust is, therefore, dependent partly on its site of deposition and partly on its toxic and antigenic properties. See Figure 5.

AIRWAYS RESISTANCE

Before considering the various occupational insults that may be inflicted upon the conducting system of the lung, certain basic anatomical and physiological considerations should be borne in mind. The resistance to air flow in the human airways can be partitioned into central and peripheral components (1). The central component comprises the resistance that is located in the upper airways, trachea, and main and segmental bronchi down to the airways that have diameters 2 mm or greater. The distal component is located in those airways whose diameter is less than 2 mm, including the gas exchanging units of the lung. Macklem and Mead have demonstrated that, of the total airways resistance, no less than 80 to 90 percent is located in the larger airways and only around 10 percent resides in the smaller airways (1). Thus, changes in the resistance to flow in the smaller airways have little influence on total airways resistance. Similarly, those indices of ventilatory capacity that are derived from the forced expiratory volume maneuver are little affected by an increase in the resistance of the small airways because these

indices for the main part reflect changes in the larger airways during dynamic compression.

Although some of the smaller air passages of the respiratory conducting system, e.g., the terminal bronchioles, are included in the peripheral airways, the remainder of the conducting system is comprised of the central airways. The respiratory symptoms and respiratory impairment that are associated with the deposition of particles in the larger airways are both more obvious and more easily demonstrated than are those associated with particulate deposition in the smaller airways (2).

RESPONSES TO DUST DEPOSITION

The deposition of inhaled dust in the central airways of the lung may induce one or more of the following four basic responses:

1. Immunologically induced airways constriction. This includes both Type I and Type III Reactions (3) and is best termed occupational asthma.
2. Pharmacologically induced airways constriction.
3. Acute irritation and reflex broncho-constriction.
4. Non-specific response to dust, viz. chronic bronchitis. This type of response is not related to the toxic properties of the dust or to its propensity to generate an immunologic reaction. Each of these responses are dealt with in turn.

IMMUNOLOGICALLY INDUCED (Asthma)

Bronchial constriction or occupational asthma may be induced by either a Type I or Type III immunological reaction (3). Type I reactions are immediate and are mediated by a specific immunoglobulin IgE. The concentration of IgE in the blood may be increased in subjects with extrinsic asthma. When a susceptible subject, viz. an atopic individual, is exposed to an antigen, there is an increase in the IgE specific to that antigen. The specific IgE binds to the mast cells present in the bronchial wall and as a result histamine and a slow reacting substance (SRS-A) are liberated. The Type III responses, which are related to the Arthus phenomenon and are associated with the presence of precipitins in the blood, occur several hours after the challenge. They are due to immunoglobulin, IgG.

Individuals with occupational asthma complain of wheeziness and shortness of breath. Initially, these symptoms occur only while the individual is at work, but later they may persist at home and on weekends. Workers who are atopic are more prone to develop occupational asthma and may do so with a relatively short exposure. Nevertheless, normal individuals may be affected although their symptoms often do not appear for several years; that is, until they have become sensitized. *Rhinitis* and *conjunctivitis* are common accompaniments of occupational asthma.

The diagnosis is made from the occupational and medical history and, if necessary, by appropriate challenge tests. When the subject is

exposed to the appropriate antigen, a decline in ventilatory capacity is usually induced. If it is a Type I reaction, the reduction in forced expiratory volume in 1 second (FEV_1) and in forced vital capacity (FVC) is usually evident within 10 to 15 minutes. If it is a Type III response, the decline is often delayed for 3 to 4 hours. Skin testing for immediate flare and wheal response is useful; however, cutaneous and bronchial responses do not necessarily correlate well with each other.

Potential exposure resulting in occupational asthma is commonly found in the following occupations: (4-17)

Grain and cereal workers, including bakers.

Woodworkers including carpenters, joiners, and sawmill operators. Western red cedar, mahogany, oak, and iroko have all been incriminated as potent sensitizing agents.

Printing. In this case gum arabic is responsible for the sensitization.

Manufacturers of detergent enzymes. These enzymes are manufactured from the products produced by fermentation of *Bacillus subtilis.* The enzyme responsible is also known as alcalase.

Soldering. This is usually due to the flux which contains aminoethylethanolamine.

Isocyanate workers. Isocyanates are used to manufacture polyurethane foams. Two compounds have been incriminated as causes of occupational asthma. These are toluene diisocyanate (TDI) and diisocyanatodiphenyl methane (MDI).

Electroplaters, photographers, and persons exposed to platinum.

Less commonly, asthma may be associated with occupational exposures to the following: nickel, chromium, the Mexican bean weevil, locusts, silkworms, coffee, castor beans, and tungsten carbide.

PHARMACOLOGICALLY INDUCED (Byssinosis)

Airways constriction may be induced by the deposition of certain dusts in the airways in the absence of an immunological reaction (18). Thus, when certain dusts settle on the bronchial walls, the liberation of naturally occurring broncho-constrictors such as histamine and possibly serotonin may take place. Since this does not involve any immunological mechanism, the liberation of such substances can be said to be a pharmacological response to an extrinsic agent.

Although there is still some doubt, the broncho-constriction seen in byssinosis may be of this type. There is good evidence to suggest that the cotton bract contains an agent which, when it comes into contact with the bronchial mucosa, leads to the liberation of excess histamine. Byssinosis is seen in cotton, hemp, and flax workers and a similar condition possibly occurs in workers who are exposed to sisal.

The usual history of byssinosis is that the worker develops chest tightness and wheezing on return to the mill on a Monday morning. In the early course of the disease the symptoms disappear by Tuesday or Wednesday. However, with continued exposure, the tightness and shortness of breath begin to persist for longer periods until it is present all

the time. In established long standing byssinosis, the worker ends up continually short of breath and with over-distended lungs. The diagnosis is established by measuring the patient's ventilatory capacity before he starts work on Monday and again after he has finished.

IRRITANTS

If the bronchi are insulted by irritant gases or fumes, they constrict in a reflex fashion (19). Such constriction is usually accompanied by coughing, and both the coughing and bronchial constriction are mediated through vagal reflexes. Irritant gases such as chlorine, ammonia, ozone, sulfur dioxide, and the oxides of nitrogen may all produce an acute *tracheitis* and *bronchitis* which are associated with reflex broncho-constriction and coughing. With large and prolonged exposures, the lung parenchyma may also be affected. The solubility of the inhaled gas will determine whether there is a predominant proximal or distal involvement of the airways.

Aside from the above gases to which workers may be exposed as a result of industrial mishaps, certain occupational groups may be routinely exposed to other noxious fumes and aerosols in their working environment. These include: beryllium, boron hydrides (volatile), cadmium, chromium compounds (hexavalent), hydrofluoric acid, zinc chloride, manganese, mercury, osmium, and vanadium pentoxide.

DUST

Prolonged exposure to dust may lead to *industrial bronchitis* (20). The heavier the dust exposure, the more likely is the development of industrial bronchitis. The characteristic features of this condition are cough and sputum in the absence of localized destructive disease of the lungs.

The symptoms of industrial bronchitis differ in no way from those seen in chronic bronchitis due to cigarette smoking. Both conditions are characterized by production of excess mucus. The mucus is secreted by the goblet cells and more particularly the mucus glands of the bronchial tree. Airways obstruction is seen less often in industrial bronchitis than it is in the naturally occurring form of chronic bronchitis due to cigarette smoking.

Coal miners and steel workers are particularly prone to this form of airways disease. The symptoms tend to regress when dust exposure is reduced.

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EXTRINSIC ALLERGIC ALVEOLITIDES

The inhalation of organic dusts may lead to two distinct pulmonary responses. First, and more common, is that mediated by reaginic antibody and usually referred to as the Type I. It occurs most commonly in atopic subjects and is characterized by changes in the airflow resistance in the conducting system of the lungs. This type of response has been described in the preceding discussion of diseases of the airways. The second and less common type of reaction affects the lung parenchyma, viz., the respiratory bronchioles and alveoli and does not appear to be related to atopy. Pepys coined the term extrinsic allergic alveolitis (hypersensitivity pneumonitides) to describe this response (1), which is also known as hypersensitivity pneumonitis.

CAUSATION

Although a large number of organic dusts have been identified as causes of extrinsic alveolitis, the pathophysiological effects are similar no matter which dust is responsible. The demonstration of specific precipitins in the serum of subjects affected by allergic alveolitis led Pepys to hypothesize that the basic mechanism in extrinsic allergic alveolitis is an Arthus or Type III reaction (1). Since most of the antigenic dusts

that produce extrinsic allergic alveolitis are organic, and since the serum of afflicted subjects often contain specific precipitins, there is an obvious resemblance to serum sickness. In the latter condition, the antigen is introduced into the body by injection, while in contrast, in extrinsic allergic alveolitis the antigen is inhaled.

Although not everybody who is repeatedly exposed to the antigen develops extrinsic allergic alveolitis, a small percentage does. Similarly, while there is good evidence that a substantial proportion of the subjects who are exposed to the antigen develop antibodies, the presence of antibodies alone is not necessarily an indication that the patient has or is likely to suffer from hypersensitivity allergic pneumonitis. Most subjects who develop allergic pneumonitis do so as a consequence of occupational exposure, but in some instances the person's avocation is more likely to be responsible.

The main differences between Type I and Type III pulmonary responses are shown in Table 1. It is important to add that since Pepys first called attention to the syndrome of extrinsic allergic alveolitis, its recognized causes have doubled or tripled (2).

CLINICAL FEATURES

Whatever the antigen, the clinical features of the condition are relatively similar. The acute form of the disease is more easily recognized and usually presents the symptoms of a sudden onset of fever, chills, shortness of breath, and a dry cough which appear between 4 to 8 hours following exposure. The patient may be severely distressed and on physical examination shows the presence of cyanosis, marked tachypnea, often of around 35 to 40 per minute, and diffusely scattered crepitations at both lung bases.

During the acute phase, pulmonary function tests show desaturated blood, reduced arterial PCO_2, and a mild to moderate respiratory alkalosis. The lung volumes are greatly reduced, especially the vital capacity; however, there is no evidence of increased resistance to flow in the airways.

Measurements of the mechanical properties of the lungs indicate that the lungs are stiffer than normal and that their compliance is greatly reduced. The radiographic appearances are those of a diffuse acinous filling process predominantly affecting the mid and lower zones. The appearances in an air bronchogram are somewhat suggestive of pulmonary edema; however, there is no cardiac enlargement. Symptoms and signs gradually regress over a period of a week to ten days.

Besides the acute form of hypersensitivity pneumonitis, a more chronic form exists. This occurs with repeated lesser exposures, and although on the first two or three occasions there may be mild fever and chills, the continued lesser insults are not so obviously related to occupational exposure. In the chronic form which appears over a period of several months, the afflicted subject notices the onset of dyspnea, sometimes with occasional mild fever. This is usually accompanied by loss of weight, and general lethargy.

Table 1. Pulmonary response differences (2).

	Extrinsic allergic asthma (Type I)	Extrinsic allergic alveolitis (Type III)
Predisposing factors	Atopy	None known
Region affected	Conducting system of the lungs: bronchi to terminal bronchioles	Acini, respiratory bronchioles, and alveoli
Histology	Mucous plugging, bronchial edema, and eosinophilic infiltration	Granulomatous pneumonitis, occasionally undergoing organization and leading to interstitial fibrosis.
Onset of symptoms	Immediate	4 to 6 hours
Systemic reaction	None	Usual and accompanied by fever
Signs	Wheezes (rhonchi)	Crackles (crepitations)
Radiographic signs	Overdistension	Acinous filling pattern, often coexisting with some reticulonodulation in more chronic forms of the syndrome
Serological findings	Elevated IgE	Precipitins present (90% of cases)
Pulmonary impairment	Increased air flow resistance	Restrictive pattern
Eosinophilia	Common	Transient and uncommon
Skin tests	Immediate and urticarial	Edematous reaction appearing in 4 to 6 hours

Physical examination may reveal some cyanosis, clubbing may be present, and there may be diffuse scattered crepitations in both lower zones. The radiographic appearances are more suggestive of chronic interstitial fibrosis than of extrinsic allergic alveolitis. Pulmonary function tests in the subacute and chronic forms of the syndrome show restrictive disease with small lungs.

As the disease progresses, the lungs become smaller, the dyspnea worsens, and the end result resembles fibrosing alveolitis. In the established chronic case, the histological appearance cannot be distinguished from that seen in chronic interstitial fibrosis of unknown etiology. Both farmer's lung and pituitary snuff allergic alveolitis have been known to present a chronic interstitial fibrosis appearance.

PREVENTION

Elimination of personal exposure to the antigen can prevent the development or recurrence of the disease. This necessitates either environmental controls or personal protection. Environmental controls may include the elimination of conditions conducive to bacterial and fungal growth, process changes preventing the production of the antigen, or ventilation and particulate controls that eliminate contact of the antigen with the worker.

Personal protection can best be provided by the use of respirators to prevent inhalation of the antigen. The appropriate respirator should be selected on the basis of the characteristics of the dust or spores, the situation involved, and individual acceptance. When protection is inadequate for an individual with the hypersensitivity, removal of that individual from the offending environment is indicated.

PATHOLOGY

In the acute phase of the disease, the histological appearances of the lung show that the alveolo-capillary membrane is thickened, and that there is histiocytic, lymphocytic, and plasma cell infiltration. There may also be an edema-like fluid present in the alveoli. Numerous epithelioid tubercles may be seen but caseation necrosis is absent, and tubercle bacilli and fungi are not seen. The one exception to this is that occasionally in maple bark disease, *Cryptostroma corticale* spores may be seen in the lung parenchyma. Even so, maple bark disease is not a true fungal infection, but an allergy to the spores of this organism. The general appearance of extrinsic allergic alveolitis is that of a granulomatous interstitial pneumonia, with the granulomata bearing a resemblance to those seen in sarcoidosis.

Early in the disease, there is often an increase in the number of reticulin fibers but later on collagenous fibrosis predominates. A bronchiolitis affecting the respiratory bronchioles may also be present. Pathological changes found in chronic farmer's lung and other extrinsic allergic alveolitis are those of an interstitial fibrosis with collagenous thickening of the septa and lymphocytic infiltration. The fibrosis is often worse in the upper lobes and there may be frequent pigment laden macrophages present in the alveoli. Later on the intima of the pulmonary arteries is thickened and when this occurs pulmonary hypertension supervenes. In the terminal stages, cystic areas with honeycombing may be present in the lungs.

IMMUNOLOGY

The abrupt onset within three to four hours of exposure argues against this syndrome being an infective process. In addition, inhalation of aqueous extracts of mouldy hay will reproduce the clinical features of farmer's lung as will extracts of *Micropolyspora faeni.* The reaction develops several hours after the challenge and is associated with a

Table 2. Common clinical conditions (2).

Clinical condition	Source of offending agent	Precipitins against
Farmer's lung	Mouldy hay	*Micropolyspora faeni* *Thermoactinomyces vulgaris*
Baggassosis	Mouldy bagasse	*Thermoactinomyces vulgaris*
Mushroom worker's lung	Mushroom compost	*Micropolyspora faeni* *Thermoactinomyces vulgaris*
Suberosis	Cork dust	Cork dust
Maple bark disease	Maple bark	*Cryptostroma corticale*
Sequoiosis	Redwood sawdust	*Graphium* *Pullaria*
Papuan lung (New Guinea lung)	Mouldy thatch dust	Thatch of huts
Wood pulp worker's disease	Wood pulp	*Alternaria*
Malt worker's lung	Mouldy barley	*Aspergillus clavatus* *Aspergillus fumigatus*
Dog house disease	Mouldy straw	*Aspergillus versicolor*
Bird fancier's lung (Pigeon breeder's lung)	Pigeon, parrot and other bird droppings	Sera, protein, and droppings
Pituitary snuff taker's lung	Bovine and porcine pituitary snuff	Pituitary antigens
Wheat weevil disease	Wheat flour	*Sitophilus granarius*
Furrier's lung	Animal hairs	
Coffee worker's lung	Coffee bean	Coffee bean dust
Paprika splitter's lung	Paprika	
Lycoperdonosis	Puffball Lycoperdon Pyriform	

decline in ventilatory and diffusing capacities. In addition, there is a rise in the temperature of the patient which is usually accompanied by marked hyperventilation.

During the acute stage, precipitins are nearly always present in the serum of the affected subject, but with convalescence the titer often drops and may become negative if further exposure does not occur. Nevertheless, the presence of precipitins does not confirm the diagnosis of extrinsic allergic alveolitis; neither does their absence exclude it.

This syndrome is produced by a large number of different antigens, of which some of the more common ones are shown in Table 2. If a subject presents the clinical features of the syndrome, a detailed occupational history should be taken to see whether an antigen of organic nature in the patient's working environment might be responsible. It is also necessary, however, to stress that certain conditions develop as a result of the patient's avocation, e.g., pigeon fanciers' disease, pituitary snuff hypersensitivity pneumonitis. Therefore, if extrinsic allergic alveolitis is suspected, the patient's serum should be examined for precipitins against the offending antigens, and if present, the patient may be challenged with the aerosolized antigen and his ventilatory capacity or preferably his diffusing capacity assessed at intervals for four to eight hours following the challenge. If either falls, then the diagnosis of extrinsic allergic alveolitis can be made.

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PNEUMOCONIOSES

The word "pneumoconiosis" literally means dust in the lungs. Not all dusts that can be deposited in the lungs cause recognizable disease, so that the most widely accepted definition of pneumoconiosis is that of the International Labour Organization which states that "Pneumoconiosis is the accumulation of dust in the lungs and the tissue reaction to its presence. For the purpose of this definition, 'dust' is meant to be an aerosol composed of solid inanimate particles (1)."

DEPOSITION OF PARTICLES

Inhaled particles closely follow the movement of the air in which they are suspended as they are carried into the lungs during the inspiratory phase of respiration. They are, however, acted upon by certain forces which tend to promote their deposition within certain anatomic regions of the lung (2). The first of these, gravitational sedimentation, refers to the fact that the falling speed or terminal velocity of an airborne particle under the influence of gravity is proportional to its density and the square of its diameter. Thus, larger and more dense particles settle out more rapidly than smaller, less dense particles. This mechanism is responsible for most of the deposition of particles during breathing.

The second factor promoting deposition of particles in the lungs is inertial impaction and refers to the fact that a particle tends to maintain its original direction of travel despite a change in direction of the airstream in which it was suspended. This mechanism is largely responsible for the deposition of particles in the nose and at bifurcations of the lower airways.

The third factor promoting deposition of particles in the lungs is termed diffusion or, preferably, Brownian motion. All airborne particles are moving at random owing to their constant bombardment by gas molecules. In general, only the particles smaller than about 0.1 micron in diameter acquire sufficient Brownian motion to become deposited as a result of this mechanism alone.

Aerodynamic behavior refers to the mobility of particles regardless of their apparent size and shape. Thus, a relatively large, loosely aggregated clump of particles may behave aerodynamically in the same fashion as a much smaller dense particle. A fiber nearly 100 microns long but with a 3-micron diameter may behave in much the same fashion as a spherical particle about 3 microns in diameter as regards its ability to penetrate into the deeper regions of the lungs.

Other factors that appear to be important in determining the deposition of airborne particles are the pattern of breathing; namely, nose versus mouth breathing, and possibly individual variation in the filtration efficiency owing to differences in size and shape of the airways.

The International Commission on Radiological Protection has calculated the probabilities for the regional deposition of aerosols as a function of particle size based upon certain breathing patterns (3). In general, these curves show that the majority of particles larger than 15 microns will be deposited in the nose. However, as the particle size decreases to about 10 microns, an increasingly larger percentage will be deposited in the tracheobronchial tree; between about 5 microns and about 0.5 microns, the particles will be deposited in the alveoli and respiratory bronchioles. It is the deposition of particles of approximately 0.5 to 5 microns in the alveoli and respiratory bronchioles that gives rise to the group of diseases known as the pneumoconioses.

CLEARANCE OF PARTICLES

The conducting airways down to the terminal bronchioles are lined by a ciliated epithelium covered by a layer of mucus secreted by glands located in their walls and by goblet cells interspersed at intervals in the epithelium. The mucous layer is continuously propelled upward toward the mouth by the motion of the tiny hair-like cilia. Particles deposited in this layer are cleared usually within 24 hours to the oropharynx where they are swallowed along with the mucus. Wide variations in bronchial clearance are known to exist in man, but the reasons for these are not entirely clear (4).

Particles that penetrate to and are deposited in the alveolar areas of the lungs are cleared much more slowly than those deposited in the conducting airways. Evidence indicates that the particles are engulfed by alveolar macrophages and are somehow carried to the mucus escalator system to be cleared. The rate of clearance of an alveolar dust load probably depends to a large extent upon the availability of alveolar macrophages (5). The rate of alveolar clearance is also in part determined by the relative biological toxicity of the dust particles to the alveolar macrophages and, in part, by the total amount of dust already

present within the lungs (2). Nonetheless, both bronchial and alveolar clearance of dust particles are relatively efficient since abundant evidence exists that only a small fraction of the total dust load deposited in the lungs is retained in even the most advanced cases of pneumoconiosis.

PATHOLOGY

The different forms of pneumoconiosis are difficult to classify. One useful classification is that of Nagelschmidt in which four types of reaction appear to be distinguishable (6):

1. Hyaline-nodular fibrosis (classical silicosis)
2. Simple pneumoconiosis of coal miners
3. Mixed dust pneumoconiosis
4. Diffuse interstitial fibrosis

SILICOSIS

The characteristic lesion of silicosis is the silicotic nodule (7). In its simplest form the silicotic nodule consists of a central core of hyalinized reticulin fibers arranged in concentric layers which, towards the periphery, blend with coarser fibers of collagen to form a relatively distinct capsule. Early in the course of silicosis these nodules occur adjacent to, or in the walls of, the respiratory bronchioles. These nodules are thought to be formed by the death of macrophages laden with fine silica and the fibrosis resulting from the release of intracellular enzymes along with the ingested particles. The silica particles are ingested by new macrophages which are in turn killed, thereby releasing their potent intracellular enzymes to promote further fibrosis and, thus, the process becomes progressive (8).

The upper lobes and hilar lymph nodes are often more severely affected than the lung bases. In early stages, the nodules may remain isolated, but as the disease progresses the nodules crowd closer together until they appear to form a continuous mass of fibrous tissue. However, on close inspection, discreet nodules can usually be distinguished and what appeared to be a diffuse fibrosis is in reality many compressed nodules.

Silicosis not only favors the growth of tubercle bacilli, but may suppress the usual features of epitheloid cell proliferation, giant cell formation, and lymphocytic reaction to the extent that caseous necrosis in the center of a silicotic nodule may be the only indication of coexistent tuberculosis infection (9). Silicosis is also associated with pulmonary hypertension and cor pulmonale, probably partially as a consequence of damage by adjacent nodules to the walls of blood vessels which produces mechanical obstruction, and partially as a result of abnormal blood gas tensions leading to vasoconstriction.

COALWORKERS' PNEUMOCONIOSIS

The characteristic lesion in coalworkers' pneumoconiosis is the coal macule which consists of an accumulation of dust-laden macrophages

around respiratory bronchioles surrounded by a halo of dilated airspaces (10). In addition to the accumulations of coal in the macule, there is a slight increase in reticulin fibers and, to a lesser extent, collagen fibers. The presence of coal macules around the walls of the respiratory bronchioles may lead to atrophy or even to disappearance of the smooth muscle, this leading to a permanent dilatation of these small airways commonly called focal emphysema.

In about 1 to 2 percent of miners with simple dust accumulation, large, solid, black masses develop which represent accumulations of coal dust within macrophages and between reticulin and collagen fibers. These lesions are commonly formed in the upper lobes and differ from silicotic conglomerate masses in that the masses are not composed of discreet compressed nodules. The cause of the large lesions ("progressive massive fibrosis") in coal workers is not known. They are probably not due to coexisting tuberculous infection, but may represent an immunological reaction to the accumulated dust load. See Figure 5.

Caplan described the appearance of multiple rounded nodules in the lungs of coal miners with rheumatoid arthritis that subsequently proved to be necrobiotic nodules resembling those seen in rheumatoid arthritis (11). Microscopically, these lesions demonstrate a pale, necrotic center surrounded by granulomatous tissue having a typically "palisaded" appearance at the periphery of the nodule. Typical Caplan nodules have subsequently been reported in other occupations than coal mining, suggesting that they are not specifically related to coal dust exposure.

MIXED DUST PNEUMOCONIOSIS

In the mixed dust pneumoconioses the pathology depends to a large extent upon the relative proportion of free silica or quartz present in the airborne dust. Those with a quartz content of less than about 0.1 percent tend to develop small nodular areas in the lungs in almost direct proportion to the total amount of dust deposited, but little in the way of reticulin or collagen fibrosis, and very little emphysema. The pathological lesions more nearly resemble those found in coal miners.

On the other hand, dusts in which the quartz content ranges from about 2 percent to about 18 or 20 percent of the total dust tend to produce lesions that more nearly resemble those seen in classical silicosis.

Some examples of dusts that contain almost no free quartz are kaolin, talc, iron oxide associated with welding, coal, and coke used in making carbon electrodes.

DIFFUSE INTERSTITIAL FIBROSIS

There are a number of pneumoconioses that tend to produce diffuse interstitial fibrosis as their characteristic pathological lesion (6). Among these are berylliosis, aluminosis, Shaver's disease, and asbestosis. It appears likely that certain slowly dissolving constituents in the dusts give

rise to a peculiar disseminated interstitial fibrosis rather than to the focal or nodular types seen in coal miners or silicosis. As a general rule, the amount of dust found in the lungs in this type of pneumoconiosis is small and the fibrotic reaction that occurs is out of proportion to the amount of dust deposited.

RADIOGRAPHIC DIAGNOSIS

Unfortunately, the ability to diagnose the presence of pneumoconiosis during life is not as precise and clear-cut as the pathological responses described above. There are two general patterns of radiographic response recognizable. Both classical silicosis and the mixed dust pneumoconioses, including that seen in coal workers, tend to produce nodular opacities or a combination of reticular-nodular opacities on the chest roentgenogram. These are basically rounded shadows and are classified by type, profusion, and extent under the ILO U/C Classification of the pneumoconioses (12).

The pneumoconioses that produce the diffuse interstitial pathology are generally manifested as reticular and linear opacities on the chest roentgenograms. These likewise are generally classified by type, profusion, and extent as irregular opacities under the ILO U/C Classification of pneumoconiosis. Both rounded and irregular opacities may be present as the background upon which complicated pneumoconiosis or progressive massive fibrosis develops.

CLASSIFICATION

The ILO U/C Classification is intended to provide a simple reproducible means of systematically recording the radiographic changes associated with the inhalation of all types of mineral dusts. It is likely to be most useful in relating the radiographic features to indices of dust exposure and changes in lung function, particularly in epidemiological studies. It should also make possible comparison of data obtained in studies from other countries.

The system basically classifies the radiographic features by small (less than 1 cm) and large (greater than 1 cm) opacities; thus, simple pneumoconiosis is diagnosed when none of the opacities exceed 1 cm in diameter, and complicated pneumoconiosis is diagnosed when one or more of the opacities exceed 1 cm in diameter. Within the small opacity category, one recognizes the type, profusion, and extent of involvement of the lungs by the opacities.

The simple pneumoconiosis is further subdivided into small rounded opacities and small irregular opacities. The small rounded opacities are classified into types p, q, r, according to the approximate diameter of the predominant opacities. The p type includes rounded opacities up to about 1.5 mm in diameter; the q (m) type includes rounded opacities exceeding about 1.5 mm up to about 3 mm in diameter; the r (n) type includes rounded opacities exceeding about 3 mm and up to about 10 mm in diameter.

CATEGORY

Profusion refers to the number of small opacities per unit area. Thus, the lung fields are divided into three zones on each side, and the number of opacities within each zone is graded. Standard radiographs are available for comparison which divide the profusion into categories 0, 1, 2, and 3. Category 0 refers to the absence of opacities or the presence of less profuse opacities than in category 1; category 1 shows small rounded opacities present, but few in number, and the normal lung markings are usually visible; category 2 shows numerous small rounded opacities, and the normal lung markings are still visible; category 3 shows very numerous small rounded opacities, and the normal lung markings are partly or totally obscured.

Actually, there is a continuum of changes from normality to the most advanced category and, to recognize this, the British National Coal Board developed a 12-point scale (13). This scale permits subdivisions of profusion into finer grades and is useful in epidemiological studies where progression of pneumoconiosis is important. The radiograph is classified into one of the four categories in the usual way by comparison with the standard midcategory films. If, during the process, the category above or below was considered as a serious alternative, this is also recorded. Thus, if a category ½ is recorded, it means that on comparison with standard radiographs the radiograph most nearly matched the category 1, but category 2 was seriously considered as an alternative.

The extent of pneumoconiosis is recorded by noting which of the lung zones are involved. Each lung is divided into three roughly equal zones by imaginary lines drawn at approximately one-third and two-thirds of the vertical distance between the apex of the lung and the dome of the diaphragm. Thus, each lung is divided into upper, middle, and lower zones for the purposes of recording the extent of pneumoconiosis.

IRREGULAR OPACITIES

Small irregular opacities are classified in much the same way as the small regular opacities, by type, profusion, and extent. Irregular opacities characteristically occur in asbestosis, but also occasionally in the other pneumoconioses. The variability, however, of these opacities in shape and width makes it virtually impossible to provide quantitative dimensions as is done in the rounded opacities; therefore, the types are divided on the basis of thickness. The s type refers to fine irregular, or linear, opacities; the t type refers to medium irregular opacities, and the u type refers to coarse (blotchy) irregular opacities. Standard radiographs of the three types of irregular opacities are available for comparison. Profusion of irregular opacities is graded in exactly the same way as is done in the rounded small opacities.

PLEURAL CHANGES

Certain pleural changes have recently become recognized as accompaniments to the parenchymal changes referred to above as part of

some pneumoconioses. Therefore, the ILO U/C Classification records pleural thickening by site (costophrenic angles, chest wall, diaphragm), width, and extent.

Pleural calcification is also classified by site and extent. When the cardiac outline and diaphragm are ill-defined, this is also recorded. A number of obligatory and optional symbols are also included in the classification for the benefit of a more complete description of the radiographic findings. The reader is referred to the complete ILO U/C Classification for details regarding these features which are beyond the scope of this chapter (12).

PHYSIOLOGICAL RESPONSES

The lungs have relatively few ways of responding to the dust burdens presented to them. Physiologically, two major patterns of response can be identified: an obstructive impairment and a restrictive impairment.

The obstructive pattern is characterized by a reduction in expiratory air flow, usually associated with either an increased airway resistance or a loss of lung recoil, or both. Increased airway resistance most commonly results from intrinsic narrowing of the airways owing to spasm of the smooth muscle in the walls (such as occurs in asthma), or to edema, inflammation, and mucus plugs (such as occurs in chronic bronchitis). These aspects have been more fully discussed in the chapter on airways mechanisms and responses.

OBSTRUCTIVE IMPAIRMENT

The obstructive pattern of physiological impairment in the pneumoconioses is more likely the result of localized or diffuse abnormalities in the lung recoil, owing to destructive changes in and around the small airways (less than 2 mm diameter) caused by the dust deposits. In simple pneumoconiosis these changes may be severe enough to cause alterations in the distribution of the inspired air and minor degrees of mismatching of ventilation and blood flow detectable only by using very sensitive techniques. These physiological impairments could prove disabling for heavy physical activities, but not for ordinary activities or at rest.

Spirometric tests of ventilatory capacity are usually within normal limits, or very nearly normal, in simple silicosis, simple coalworkers' pneumoconiosis, and the simple mixed dust pneumoconioses, unless asthma, chronic bronchitis, or emphysema, coexist. In the complicated form of the pneumoconioses (PMF), ventilatory capacity as measured by spirometry is often abnormal and consists of elements of both obstruction and restriction. Here, in addition to the loss of lung recoil owing to focal emphysema, some of the obstructive impairment may be attributable to distortion and kinking of airways by the large conglomerate masses. These large masses also generally interfere with gas exchange by reducing the surface area available for diffusion and the obliteration of the capillary bed.

Table 3. Agent, pathology, and impairment associated with pneumoconioses.

Agent	Type of Pathology	Type of Respiratory Impairment
1. Silica		
Simple	Nodular fibrosis	Restrictive, diffusion
Complicated	Conglomerate nodular fibrosis	Restrictive, obstructive, diffusion
2. Hematite	Nodular fibrosis	Restrictive, diffusion
3. Mixed dusts		
Iron and silica	Nodular fibrosis (Rarely conglomerate nodular fibrosis)	Restrictive, diffusion
4. Silicates	Nodular fibrosis	Restrictive, obstructive
Talc	(Rarely conglomerate nodular fibrosis)	
Kaolin		
Bentonite		
Diatomite		
Tripoli		
Fuller's earth		
Mica		
Sillimanite		
Cement	Nonspecific bronchitis	Obstructive
5. Coal		
Simple	Peribronchiolar macules, focal emphysema	Obstructive (small airways)
Complicated	Conglomerate nodular fibrosis	Obstructive, restrictive, diffusion
6. Graphite	Peribronchiolar macules, focal emphysema	Obstructive (small airways)
7. Aluminum	Interstitial fibrosis	Restrictive, diffusion
8. Asbestos	Interstitial fibrosis	Restrictive, diffusion
9. Beryllium	Interstitial fibrosis (granulomata)	Restrictive, diffusion
10. Tungsten carbide	Interstitial fibrosis	Restrictive, diffusion
11. Barium	Simple dust accumulation	None known
12. Cerium	Simple dust accumulation	None known
13. Iron	Simple dust accumulation	None known
14. Tin	Simple dust accumulation	None known
15. Titanium	Simple dust accumulation	None known

The simple forms of classical silicosis, coal workers' pneumoconiosis, and the mixed dust pneumoconiosis, generally demonstrate mild obstructive impairment; whereas, the complicated forms (PMF) usually present mixtures of obstruction, restriction, and abnormalities of gas exchange.

RESTRICTIVE IMPAIRMENT

The restrictive pattern of physiological response is characterized by a reduction in lung volumes and ventilatory capacity, usually unaccompanied by an increased air flow resistance or hyperinflation. The restrictive pattern is also associated with an increased lung recoil, reduction in surface area for gas exchange and/or thickening of the air blood interface of the lungs.

The pneumoconioses that lead to diffuse interstitial fibrosis usually present the restrictive pattern of physiological impairment. In these, the earliest impairments are those involving gas exchange and diffusing capacity, and may be detectable only during exercise. In the later stages, gas exchange and diffusion abnormalities are detectable also at rest and are associated with a reduction in lung volumes, such as the vital capacity, total lung capacity, and the inspiratory reserve volume. Again, in cases where pneumoconiosis coexists with asthma or chronic bronchitis, this restrictive pattern may be associated with some element of obstructive impairment.

Asbestosis, berylliosis, aluminosis, and Shaver's disease are examples of pneumoconioses that are characteristically associated with the restrictive pattern of physiological impairment.

A summary of agent and type of pathology and respiratory impairment is given in Table 3.

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SECTION VI

What is a weed? A plant whose virtues have not been discovered.
— Ralph Waldo Emerson

PLANT AND WOOD HAZARDS

Tracy E. Barber, M.D., and E. Lee Husting, Ph.D.

Both local and systemic manifestations occur from occupational exposure to plants, plant products, and woods. Dermatitis is frequently observed; other effects include asthma, hay fever, irritations, toxic effects, and allergenic responses.

PLANTS AND PLANT PRODUCTS

Dermatitis due to plants of the genus *Rhus* is the most frequently seen allergic contact dermatitis in the United States. Plants in the genus *Rhus* include poison ivy, poison oak, and poison sumac. The contact dermatitis caused by these plants is identifiable by characteristic linear and bullous lesions. At least one of the species of *Rhus* is found in every part of the continental United States. Poison ivy occurs in every state but California where it is poison oak that has been the main cause of occupational skin disease. The oakleaf form of poison ivy and poison sumac are found mainly in the south and east, from Texas to New Jersey. Western poison oak occurs in Washington, Oregon, and California.

The genus *Rhus* belongs to the family Anacardiacae which includes the cashew nut tree, the Japanese lac tree, the Indian marking nut tree, and the mango. A phenolic liquid is extracted from cashew nut shells, and this liquid, used to form resins for varnishes and brake shoe linings, is an irritant as well as a sensitizing agent. The fruit of the mango may cause contact dermatitis in *Rhus*-sensitive persons.

Dermatitis results from contact with the milky sap found in the roots, stems, leaves, and fruit of *Rhus* plants. In a dry atmosphere, the sap may retain its potency for months or perhaps years. The sap may be transmitted on soot particles when the plant is burned, or may be carried by animals, equipment, or apparel.

Allergic contact dermatitis may also be caused by the bastard feverfew (a common southeastern weed), English ivy, and castor bean plants. Allergic dermatitis may be caused by contact with certain flowers (such as primrose, chrysanthemum, poinsettia) and bulbs of hyacinth, narcissus, and tulips. The lipid fraction of ragweed pollen may cause eczematous dermatitis, while a water-soluble fraction may cause asthma or hay fever.

Contact dermatitis has occurred from handling fruits and vegetables, including carrots, asparagus, and some citrus fruits. Fruit and vegetable handlers may also suffer contact dermatitis due to insecticides and fungicides. Indirect effects of handling fruit and vegetables include chapping and moniliasis from exposure to moisture, photosenitization dermatitis from sunlight, and parasitism by mites.

Photosenitization is the delayed development of erythema, edema, vesicles, and bullae after contact with plant juices and exposure to sunlight. This accentuated localized sunburn is a phototoxic, rather than a

photoallergenic effect, and may result in either hyperpigmentation or depigmentation. Plants which cause photosenitization include fig, rue, lime, bergamot, parsnips, parsley, carrots, fennel, dill, and pink rot celery.

Hay fever, asthma, and urticaria frequently occur in castor bean processors, resulting from a potent allergen found in the dried pomace remaining after castor oil extraction. Castor bean workers, dock workers handling the pomace, or farmers using the pomace for fertilizer may be affected.

Historically, paprika sorter's disease was frequent in women splitting paprika fruit who inhaled spores and mycelia of a mold growing in the fruit. This exposure has been eliminated since the entire fruits are now ground mechanically.

Exposure to grain dusts may result in coughing, wheezing, breathlessness, dermatitis, and grain fever. The incidence of these symptoms is higher in individuals with a history of past allergy, suggesting that allergy may be party responsible for the response to grain dusts.

Tobacco cropper's or green-tobacco sickness, characterized by weakness, nausea, and vomiting, has been observed in persons pulling tobacco leaves from the plants during cropping. It is believed that a noxious material in green tobacco gum, most likely nicotine, is absorbed through the skin.

POTENTIAL OCCUPATIONAL EXPOSURES

- Agricultural workers
- Botanists
- Bulb handlers, plant
- Camp workers
- Canners
- Castor bean workers
- Construction workers
- Dock workers
- Field laborers
- Flower cutters
- Flower packers
- Foresters
- Fruit pickers
- Fruit processors
- Gardeners
- Grain elevator workers
- Highway workers
- Hop pickers
- Horticulturists
- Loggers
- Pipeline workers
- Road builders
- Surveyors
- Telephone linemen
- Tobacco croppers
- Utility workers
- Vegetable harvesters
- Vegetable processors

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WOODS

Woods, wood dusts, and substances from woods may be toxic, irritant, allergenic, or carcinogenic.

Toxic woods, such as East Indian satinwood, South African boxwood, and ipe, contain substances which cause systemic signs and symptoms when absorbed, inhaled, or ingested. Wood toxins are often alkaloids. Effects may include headache, anorexia, nausea, vomiting, bradycardia, dyspnea, or somnolence.

Irritant woods cause injury to mucous membranes upon contact, and severe irritants may affect intact skin, causing dermatitis. Examples of irritant woods are mansonia, dahoma, and cocobolo.

Allergenic woods such as certain members of the birch, pine, dogwood, beech, mahogany, mulberry, and myrtle families may cause allergic manifestations including asthma and contact dermatitis in sensitized individuals.

It is believed that the inhalation of fine dusts from wood, especially hard wood dust, causes nasal cancer. Many woodworkers in the furniture industry develop squamous metaplasia in the nasal mucous membrane.

Furniture workers frequently exhibit an allergic response to western red cedar. The response occurs after contact with the sawdust of this wood, and symptoms are intensified by contact with the wood. Symptoms include asthma, rhinitis, urticaria, dermatitis, and conjunctivitis. Asthma and rhinitis are frequent in carpenters, while conjunctivitis occurs more often in sawmill workers.

POTENTIAL OCCUPATIONAL EXPOSURES

- Cabinet makers
- Carpenters
- Furniture makers
- Lumbermen
- Musicians
- Sawmill workers
- Violin makers
- Wood workers

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SECTION VII

. . .New industrial materials will produce new hazards requiring careful detective work before the exact agent or mode of action is incriminated, and to which the classical approach can be applied, but the principles are known, and the work can proceed along what are now fairly conventional lines.

—The Committee on Environmental Health Problems - 1962.
PHS Publication No. 908

CHEMICAL HAZARDS

Irving R. Tabershaw, M.D., H.M.D. Utidjian, M.D., D.I.H. and Barbara L. Kawahara, M.P.H.

Raw materials from many sources are converted by the chemical industry into the substances used by the industry itself to produce cosmetics, detergents and soaps, drugs, dyes, pigments, explosives, fertilizers, petrochemicals, inks, paints, pesticides, plastics, synthetic fibers, and many other products. Other industries use the substances in the production of durable and nondurable goods.

This section deals with the harmful health effects of some of these substances. Items discussed include the capacity of the substance to produce local and/or systemic effects, special diagnostic tests that may aid diagnosis of the illness by identification of the agent, potential occupational exposures, protection methods, and references.

HARMFUL EFFECTS. Under the heading Harmful Effects are given only the chief or dominant effects that characterize the usual responses to the toxic agent. Because of the lack of information on the mutagenic, teratogenic, or carcinogenic effects of many chemicals, consideration is given only in specific instances to these effects.

Local and systemic effects are given in an effort to categorize the effects of the toxic agent. It was arbitrarily decided to limit local effects to the skin and eyes and to the mucous membranes of the upper respiratory tract. Systemic effects include the manifestations elicited by the absorption of the toxic agent into the body and its distribution to the internal organs and particularly the effects of the agent on the tissues of the lower respiratory tract.

ROUTE OF ENTRY. The route of entry section is intended to supply information on the avenue by which the toxic agent is most likely to gain entrance into the body when encountered in the industrial environment.

SPECIAL DIAGNOSTIC TESTS. Ordinary tests such as complete blood counts, urinalyses, and chest roentgenograms are not included under the heading Special Diagnostic Tests. Similarly, liver and kidney function tests and cutaneous patch tests have not been included, even though they may be of considerable diagnostic importance. It is felt that the physician need not be reminded of the methods for determining abnormalities in the target organs which are mentioned under Systemic Effects.

It should be pointed out that many of these special diagnostic tests are difficult to carry out and should be performed only by qualified laboratories. In addition, the fact should be kept in mind that normal values may vary somewhat, even those from competent analytical laboratories.

Because of the absence of significant, interpretable information, no reference is made to behavioral patterns of response to toxic agents.

PERMISSIBLE EXPOSURE LIMITS. In the section on Permissible Exposure Limits, the standard as currently promulgated by the Secretary of the U. S. Department of Labor under the Occupational Safety and Health Act of 1970 (PL 91-596) is given. These standards contain the permissible exposure limit as found in 29 CFR 1910.1000 as of January 1, 1977. Where NIOSH has published a recommended revision to the OSHA regulation, the NIOSH recommended level is also noted. In addition in some instances the assigned Threshold Limit Value (TLV) appearing in the current (1976) list published by the American Conference of Governmental Industrial Hygienists (ACGIH) is given.

The Department of Labor promulgates standards that are mandatory in the United States; the TLVs issued by the ACGIH are guidelines and are to be considered as practical guides in the control of health hazards and should not be regarded as fine lines between safe and dangerous exposure levels. In 1971, most of the 1968 ACGIH TLV list was included in the standard for air contaminants (29 CFR 1910.93 amended to 1910.1000). United States standards are first published in the Federal Register (FR), which is available by subscription from the U. S. Government Printing Office. These regulations are then codified in the Code of Federal Regulations (CFR), generally in Title 29. This publication is also available from the Superintendent of Documents, U.S. Government Printing Office, Washington, D. C. Since these regulations are mandatory and often stipulate medical requirements as to diagnostic tests, it would be wise to check this reference if there is any question about an illness resulting from occupational exposure.

POTENTIAL OCCUPATIONAL EXPOSURES. The list of occupations appended to a particular chemical substance lists occupations in which workers are potentially exposed to the toxic agent. Whether the exposure to the toxic agent constitutes a hazard depends upon such factors as concentration of the agent, how the agent is handled and used, duration of exposure, susceptibility of the worker to the agent, and the health protection practices that may be adopted by management. Thus, all hazardous situations imply an exposure, but all exposures are not hazardous.

SYMPTOMS AND EXPOSURE. In resolving the problem of the relationship between the signs and symptoms presented by the worker and the potential toxic exposure in his occupation, it is important to understand the ways by which a toxic chemical enters the body and to secure factual information as to the physical and chemical characteristics of the work environment and the personal hygiene of the worker. At the same time, it is essential to recognize that 1) chemical formulas offer, at most, only rough guides to the prediction of toxic response and 2) the forms of acute and chronic toxicity are so often dissimilar that prediction cannot be made of the nature of chronic toxicity from the acute manifestations.

SELECTION OF CHEMICALS. Most of the known occupational disease-producing chemicals are given; certain chemicals, however, are excluded because of insufficient data.

The chemicals given are arranged by chemical groups; for example, aliphatic hydrocarbons, alcohols, glycols. Some chemicals are discussed in the section on pesticides.

REFERENCES. A number of excellent secondary references are available on chemical hazards. These include the series of NIOSH Criteria Documents, the AIHA Hygienic Guides, and the ACGIH Documentation of the Threshold Limit Values for Substances in Workroom Air. An additional source is the Medical Surveillance Guidelines from the OSHA-NIOSH Standards Completion Program. These provide detailed information on toxicology, signs and symptoms, special tests, treatments, surveillance, and prevention. Specific references cited in a section are listed at the end of that section. Supplemental references covering some chemical groups are provided in bibliographies for those groups.

General texts of interest are listed in the following bibliography.

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ALIPHATIC HYDROCARBONS

Aliphatic hydrocarbons are saturated or unsaturated, branched or unbranched open carbon chains. Within this group there are three subgroups: alkanes (saturated hydrocarbons), alkenes (unsaturated hydrocarbons with one or more double bonds), and alkynes (unsaturated hydrocarbons with one or more triple bonds). Synonyms are paraffins, olefins, and acetylenes, respectively. Compounds of lower molecular weight containing fewer than 4 carbons are usually gases at room temperature, whereas larger molecules, containing from 5 to 16 carbons, are liquids, and those having more than 16 carbons are usually solids.

Aliphatic hydrocarbons are derived from petroleum by the cracking, distillation, and fractionation of crude oil. Most of these compounds are used industrially in mixtures, such as natural gas, petroleum naphtha, gasoline, kerosene, and mineral spirits. Aliphatic hydrocarbons are used principally as fuels, refrigerants, propellants, dry cleaning agents, lubricants, solvents, and chemical intermediates.

Aliphatic hydrocarbons are asphyxiants and central nervous system depressants. Lower members of the series, methane and ethane, are pharmacologically less active than higher members of the series, their main hazards resulting from the simple displacement of oxygen and from fire and from explosion. Higher members of the series cause narcosis. At least one member (hexane) has neurotoxic properties. Another common effect is irritation of the skin and mucous membranes of the upper respiratory tract. Repeated and prolonged skin contact may result in dermatitis, due to the defatting of skin. Due to its low viscosity, aspiration of liquid may result in diffuse chemical pneumonitis, pulmonary edema, and hemorrhage. Contamination of aliphatic hydrocarbons by benzene significantly increases the hazard. Therefore, it is important that benzene content, if suspected, be determined.

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ACETYLENE

DESCRIPTION

HC≡CH, acetylene, is a colorless gas with a faint ethereal odor.

SYNONYMS

Ethine, ethyne, narcylene.

POTENTIAL OCCUPATIONAL EXPOSURES

Acetylene can be burned in air or oxygen and is used for brazing, welding, cutting, metallizing, hardening, flame scarfing, and local heating in metallurgy. The flame is also used in the glass industry. Chemically, acetylene is used in the manufacture of vinyl chloride, acrylonitrile, synthetic rubber, vinyl acetate, trichloroethylene, acrylate, butyrolactone, 1,4-butanediol, vinyl alkyl ethers, pyrrolidone, and other substances.

A partial list of occupations in which exposure may occur includes:

- Acetaldehyde makers
- Acetone makers
- Alcohol makers
- Braziers
- Carbon black makers
- Ceramic makers
- Copper purifiers
- Descalers
- Drug makers
- Dye makers
- Foundry workers
- Gougers
- Hardeners
- Heat treaters
- Lead burners
- Rubber makers
- Scarfers

PERMISSIBLE EXPOSURE LIMITS

No Federal standard has been established. NIOSH has recommended a ceiling limit of 2,500 ppm.

ROUTE OF ENTRY

Inhalation of gas.

HARMFUL EFFECTS

Local—

Acetylene is nonirritating to skin or mucous membranes.

Systemic—

At high concentrations pure acetylene may act as a mild narcotic and asphyxiant. Most accounted cases of illness or death can be attributed to acetylene containing impurities of arsine, hydrogen sulfide, phosphine, carbon disulfide, or carbon monoxide.

Initial signs and symptoms of exposure to harmful concentrations of impure acetylene are rapid respiration, air hunger, followed by im-

paired mental alertness and muscular incoordination. Other manifestations include cyanosis, weak and irregular pulse, nausea, vomiting, prostration, impairment of judgment and sensation, loss of consciousness, convulsions, and death. Low order sensitization of myocardium to epinephrine resulting in ventricular fibrillation may be possible.

MEDICAL SURVEILLANCE

No specific considerations are needed.

SPECIAL TESTS

None in common use.

PERSONAL PROTECTIVE METHODS

Acetylene poisoning can quite easily be prevented if 1) there is adequate ventilation and 2) impurities are removed when acetylene is used in poorly ventilated areas. General industrial hygiene practices for welding, brazing, and other metallurgical processes should also be observed.

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ALICYCLIC HYDROCARBONS

DESCRIPTION

Cyclopropane: C_3H_6
Cyclohexane: C_6H_{12}
Cyclohexene: C_6H_{10}
Methylcyclohexane: C_7H_{14}

Alicyclic hydrocarbons are saturated or unsaturated molecules in which three or more carbon atoms are joined to form a ring structure. The saturated compounds are called cycloalkanes, cycloparaffins, or naphthenes. The cyclic hydrocarbons with one or more double bonds are called cycloalkenes or cyclo-olefins. These compounds are colorless liquids.

SYNONYMS

None.

POTENTIAL OCCUPATIONAL EXPOSURES

Uses vary with compounds. Cyclopropane is used as an anesthetic. Cyclohexane is used as a chemical intermediate, as a solvent for fats, oils, waxes, resins, and certain synthetic rubbers, and as an extractant of essential oils in the perfume industry. Cyclohexene is used in the manufacture of adipic, maleic, and cyclohexane carboxylic acid. Methylcyclohexane is used as a solvent for cellulose ethers and in the production of organic synthetics.

A partial list of occupations in which exposure may occur includes:

Adipic acid makers
Benzene makers
Fat processors
Fungicide makers
Lacquerers
Nylon makers
Oil processors
Paint removers
Plastic molders
Resin makers
Rubber makers
Varnish removers
Wax makers

PERMISSIBLE EXPOSURE LIMITS

The Federal standards are:

Cyclohexane 300 ppm (1050 mg/m^3);
Cyclohexene 300 ppm (1015 mg/m^3);
Methylcyclohexane 500 ppm (2000 mg/m^3).

Presently there is no standard for cyclopropane.

(Note: The 1976 ACGIH lists a TLV of 400 ppm (1,600 mg/m^3) for methylcyclohexane.)

ROUTE OF ENTRY

Inhalation of gas or vapor.

HARMFUL EFFECTS

Local—

Repeated and prolonged contact with liquid may cause defatting of the skin and a dry, scaly, fissured dermatitis. Mild conjunctivitis may result from acute vapor exposure.

Systemic—

Alicyclic hydrocarbons are central nervous system depressants, although their acute toxicity is low. Symptoms of acute exposure are excitement, loss of equilibrium, stupor, coma, and, rarely, death as a result of respiratory failure. The concentration of cyclopropane required to produce surgical anesthesia is low, and there is a wide margin between anesthetic and toxic concentrations. The myocardium may become more sensitive to epinephrine during narcosis with cyclopropane. Severe diarrhea and vascular collapse resulting in heart, lung, liver, and brain degeneration have been reported in oral administration of alicyclic hydrocarbons to animals.

The danger of chronic poisoning is relatively slight because these compounds are almost completely eliminated from the body. Metabolism of cyclohexane, for example, results in cyclohexanone and cyclohexanol entering the bloodstream and does not include the metabolites of phenol, as with benzene. Damage to the hematopoietic system does not occur except when exposure is compounded with benzene, which may be a contaminant. Alicyclic hydrocarbons are excreted in the urine as sulfates or glucuronides, the particular content of each varying. Small quantities of these compounds are not metabolized and may be found in blood, urine, and expired breath.

MEDICAL SURVEILLANCE

Consider possible irritant effects to the skin and respiratory tract in any preplacement or periodic examination, as well as any renal or liver complications.

SPECIAL TESTS

None in common use. Some metabolites have been found in blood and urine.

PERSONAL PROTECTIVE METHODS

Skin protection with barrier creams or gloves.

Workers exposed to high concentrations of gas or vapor may need masks.

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Fabre, R., R. Truhaut, and S. Laham. 1959. Etude de metabolisme du cyclohexane chez lapin. C. R. Acad. Sci., Ser. D. 248:1081.

1,3-BUTADIENE

DESCRIPTION

$H_2C=CH-CH=CH_2$, 1,3-butadiene, is a colorless, flammable gas with a pungent, aromatic odor. Because of its low flash point, 1,3-butadiene's fire and explosion hazard may be more serious than its health hazard.

SYNONYMS

Biethylene, bivinyl, butadiene monomer, divinyl, erythrene, methylallene, pyrrolylene, vinylethylene.

POTENTIAL OCCUPATIONAL EXPOSURES

1,3-Butadiene is used chiefly as the principal monomer in the manufacture of many types of synthetic rubber. Presently, butadiene is finding increasing usage in the formation of rocket fuels, plastics, and resins.

A partial list of occupations in which exposure may occur includes:

- Organic chemical synthesizers
- Resin makers
- Rocket fuel handlers
- Rocket fuel makers
- Rubber makers

PERMISSIBLE EXPOSURE LIMITS

The Federal standard for 1,3-butadiene is 1,000 ppm (2,200 mg/m^3).

ROUTE OF ENTRY

Inhalation of gas or vapor.

HARMFUL EFFECTS

Local—

Butadiene gas is slightly irritating to the eyes, nose, and throat. Dermatitis and frostbite may result from exposure to liquid and evaporating gas.

Systemic—

In high concentrations, 1,3-butadiene gas can act as an irritant, producing cough, and as a narcotic, producing fatigue, drowsiness, headache, vertigo, loss of consciousness, respiratory paralysis, and death. One report states that chronic exposure may result in central nervous system disorders, diseases of the liver and biliary system, and tendencies toward hypotension, leukopenia, increase in ESR, and decreased hemoglobin content in the blood. These changes have not been seen by most observers in humans.

MEDICAL SURVEILLANCE

No specific considerations are needed.

SPECIAL TESTS

None in common use.

PERSONAL PROTECTIVE METHODS

Masks are recommended in contaminated areas with high concentrations of the gas.

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Carpenter, C. P., C. B. Shaffer, and H. F. Smyth, Jr. 1944. Studies on the inhalation of 1,3-butadiene; with a comparison of its narcotic effect with benzol, toluol, and styrene, and a note on the elimination of styrene by the human. J. Ind. Hyg. Toxicol. 26:69.

GASOLINE

DESCRIPTION

Gasoline is a highly flammable, mobile liquid with a characteristic odor.

SYNONYMS

Petrol, motor spirits, benzin.

POTENTIAL OCCUPATIONAL EXPOSURES

Gasoline is used as a fuel, diluent, and solvent throughout industry. A partial list of occupations in which exposure may occur includes:

- Filling station attendants
- Garage mechanics
- Gasoline engine operators
- Motor transport drivers
- Pipeline workers
- Refinery workers
- Tank car cleaning crews

PERMISSIBLE EXPOSURE LIMITS

Presently, the composition of gasoline is so varied that a single Federal standard for all types of gasoline is not applicable. It is recommended, however, that atmospheric concentrations should be limited by the aromatic hydrocarbon content.

ROUTE OF ENTRY

Most cases of poisoning reported have resulted from inhalation of vapor and ingestion. It is not known whether gasoline poisoning may be compounded by percutaneous absorption.

HARMFUL EFFECTS

Local—

Gasoline is irritating to skin, conjunctiva, and mucous membranes. Dermatitis may result from repeated and prolonged contact with the liquid, which may defat the skin. Certain individuals may develop hypersensitivity.

Systemic—

Gasoline vapor acts as a central nervous system depressant. Exposure to low concentrations may produce flushing of the face, staggering gait, slurred speech, and mental confusion. In high concentrations, gasoline vapor may cause unconsciousness, coma, and possibly death resulting from respiratory failure.

Other signs also may develop following acute exposure. These signs are early acute hemorrhage of the pancreas, centrilobular cloudy swelling and fatty degeneration of the liver, fatty degeneration of the proximal convoluted tubules and glomeruli of the kidneys, and passive congestion of the spleen.

Ingestion and aspiration of the liquid gasoline usually occurs during siphoning.

Chemical pneumonitis, pulmonary edema, and hemorrhage may follow. Aromatic hydrocarbon content may also cause hematopoietic changes. Absorption of alkyl lead antiknock agents contained in many gasolines poses an additional problem especially where there is prolonged skin contact. The existence of chronic poisoning has not been established.

MEDICAL SURVEILLANCE

No special considerations are necessary.

SPECIAL TESTS

None in common use.

PERSONAL PROTECTIVE METHODS

Barrier creams and impervious gloves, protective clothing.
Masks in heavy exposure to vapors.

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n-HEPTANE

DESCRIPTION

$CH_3(CH_2)_5CH_3$, n-heptane, is a clear liquid which is highly flammable and volatile.

SYNONYMS

None.

POTENTIAL OCCUPATIONAL EXPOSURES

n-Heptane is used as a solvent and as a standard in testing knock of gasoline engines.

A partial list of occupations in which exposure may occur includes:

Process workers (where heptane is used as the solvent)
Refinery laboratory workers
Refinery workers

PERMISSIBLE EXPOSURE LIMITS

The Federal standard for n-heptane is 500 ppm (2000 mg/m^3). (Note: The 1976 ACGIH lists a TLV of 400 ppm (1,600 mg/m^3).

ROUTE OF ENTRY

Inhalation of the vapor.

HARMFUL EFFECTS

Local—

n-Heptane can cause dermatitis and mucous membrane irritation. Aspiration of the liquid may result in chemical pneumonitis, pulmonary edema, and hemorrhage.

Systemic—

Systemic effects may arise without complaints of mucous membrane irritation. Exposure to high concentrations causes narcosis producing vertigo, incoordination, intoxication characterized by hilarity, slight nausea, loss of appetite, and a persisting gasoline taste in the mouth. These effects may be first noticed on entering a contaminated area. n-Heptane may cause low order sensitization of the myocardium to epinephrine.

MEDICAL SURVEILLANCE

Preplacement examinations should evaluate the skin and general health, including respiratory, liver, and kidney function.

SPECIAL TESTS

None have been commonly used.

PERSONAL PROTECTIVE METHODS

Barrier creams and gloves. Masks where exposed to vapor.

n-HEXANE

DESCRIPTION

$CH_3(CH_2)_4CH_3$, n-hexane, is a colorless, volatile liquid and is highly flammable.

SYNONYMS

None.

POTENTIAL OCCUPATIONAL EXPOSURES

n-Hexane is used as a solvent, particularly in the extraction of edible fats and oils, as a laboratory reagent, and as the liquid in low temperature thermometers. Technical and commercial grades consist of 45 to 85% n-hexane, as well as cyclopentanes, isohexane, and from 1 to 6% benzene.

A partial list of occupations in which exposure may occur includes:

Fat processors
Oil processors
Thermometer makers

PERMISSIBLE EXPOSURE LIMITS

The Federal standard is 500 ppm (1800 mg/m^3) for workroom exposure to n-hexane. (Note: The 1976 ACGIH lists a TLV of 100 ppm (360 mg/m^3).)

ROUTE OF ENTRY

Inhalation of vapor.

HARMFUL EFFECTS

Local—

Dermatitis and irritation of mucous membranes of the upper respiratory tract.

Systemic—

Asphyxia may be produced by high concentrations. Acute exposure may cause narcosis resulting in slight nausea, headache, and dizziness. Myocardial sensitization to epinephrine may occur but is of low order. Peripheral neuropathy has been reported resulting from exposure to n-hexane.

MEDICAL SURVEILLANCE

Consider the skin, respiratory system, central and peripheral nervous system, and general health in preplacement and periodic examinations.

SPECIAL TESTS

None in use.

PERSONAL PROTECTIVE METHODS

Barrier creams and gloves are recommended, as are masks where workers are exposed to vapors.

BIBLIOGRAPHY

Paulson, G. W., and G. W. Waylonis. 1976. Polyneuropathy due to n-hexane. Arch. Intern. Med. 136:880.

KEROSENE

DESCRIPTION

Kerosene is a pale yellow or clear, mobile liquid, composed of a mixture of petroleum distillates, having a characteristic odor. Chemically, it is composed of aliphatic hydrocarbons with 10 to 16 carbons per molecule and benzene and naphthalene derivatives.

SYNONYMS

Kerosine, coal-oil, range-oil.

POTENTIAL OCCUPATIONAL EXPOSURES

Kerosene is used as a fuel for lamps, stoves, jets, and rockets. It is also used for degreasing and cleaning metals and as a vehicle for insecticides.

A partial list of occupations in which exposure may occur includes:

- Farmers
- Garage workers
- Grease removers
- Heating fuel handlers
- Insecticide workers
- Jet fuel handlers
- Metal cleaners
- Petroleum refinery workers

PERMISSIBLE EXPOSURE LIMITS

Presently there is no Federal standard for kerosene vapor in workroom air.

ROUTE OF ENTRY

Inhalation of vapor.

HARMFUL EFFECTS

Local—

The liquid may produce primary skin irritation as a result of de-

fatting. Aspiration of liquid may cause extensive pulmonary injury. Because of its low surface tension, kerosene may spread over a large area, causing pulmonary hemorrhage and chemical pneumonitis. Kerosene mist may also cause mucous membrane irritation.

Systemic—

Inhalation of high concentrations may cause headache, nausea, confusion, drowsiness, convulsions, and coma. When kerosene is ingested, it may cause nausea, vomiting, and, in severe cases, drowsiness progressing to coma, and death by hemorrhagic pulmonary edema and renal involvement.

MEDICAL SURVEILLANCE

No specific considerations are needed.

SPECIAL TESTS

None in use.

PERSONAL PROTECTIVE METHODS

Barrier creams, gloves, and protective clothing are recommended. Where workers are exposed to vapors, masks are recommended.

BIBLIOGRAPHY

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Richardson, J. A., and H. R. Pratt-Thomas. 1951. Toxic effects of varying doses of kerosene administered by different routes. Am. J. Med. Sci. 221:531.

NAPHTHA

DESCRIPTION

Naphthas derived from both petroleum and coal tar are included in this group. Petroleum naphthas composed principally of aliphatic hydrocarbons are termed "close-cut" fractions. "Medium-range" and "wide-range" fractions are made up of 40 to 80 per cent aliphatic hydrocarbons, 25 to 50 per cent naphthenic hydrocarbons, 0 to 10 per cent benzene, and 0 to 20 per cent other aromatic hydrocarbons.

Coal tar naphtha is a mixture of aromatic hydrocarbons, principally toluene, xylene, and cumene. Benzene, however, is present in appreciable amounts in those coal tar naphthas with low boiling points.

SYNONYMS

Petroleum naphtha: ligroin, benzine, petroleum ether, petroleum benzine.

POTENTIAL OCCUPATIONAL EXPOSURES

Naphthas are used as organic solvents for dissolving or softening rubber, oils, greases, bituminous paints, varnishes, and plastics. The less flammable fractions are used in dry cleaning, the heavy naphthas serving as bases for insecticides.

A partial list of occupations in which exposure may occur includes:

Chemical laboratory workers	Petroleum refinery workers
Detergent makers	Rubber coaters
Dry cleaners	Solvent workers
Fat processors	Stainers
Insecticide workers	Varnish makers
Metal degreasers	Wax makers
Oil processors	Wool processors
Painters	Xylene makers

PERMISSIBLE EXPOSURE LIMITS

The Federal standard for petroleum naphtha is 500 ppm (2,000 mg/m^3); for coal tar naphtha it is 100 ppm (400 mg/m^3).

ROUTE OF ENTRY

Inhalation of vapor. Percutaneous absorption of liquid is probably not important in development of systemic effects unless benzene is present.

HARMFUL EFFECTS

Local—

The naphthas are irritating to the skin, conjunctiva, and the mucous membranes of the upper respiratory tract. Skin "chapping" and photosensitivity may develop after repeated contact with the liquid. If confined against skin by clothing, the naphthas may cause skin burn.

Systemic—

Petroleum naphtha has a lower order of toxicity than that derived from coal tar, where the major hazard is brought about by the aromatic hydrocarbon content. Sufficient quantities of both naphthas cause central nervous system depression. Symptoms include inebriation, followed by headache and nausea. In severe cases, dizziness, convulsions, and unconsciousness occasionally result. Symptoms of anorexia and nervousness have been reported to presist for several months following an acute overexposure, but this appears to be rare. One fraction, hexane, has been reported to have been associated with peripheral neuropathy. (See Hexane.) If benzene is present, coal tar naphthas may produce blood changes such as leukopenia, aplastic anemia, or leukemia. The kidneys and spleen have also been affected in animal experiments. (See Benzene.)

MEDICAL SURVEILLANCE

Preplacement and periodic medical examinations should include the central nervous system. If benzene exposure is present, workers should have a periodic complete blood count (CBC) including hematocrit, hemoglobin, white blood cell count and differential count, mean corpusclar volume and platelet count, reticulocyte count, serum bilirubin determination, and urinary phenol in the preplacement examination and

at 3-month intervals. There are no specific diagnostic tests for naphtha exposure but urinary phenols may indicate exposure to aromatic hydrocarbons. It should be noted that benzene content of vapor may be higher than predicted by content in the liquid.

SPECIAL TESTS

None in common use.

PERSONAL PROTECTIVE METHODS

Workers should use barrier creams, protective clothing, gloves and masks where exposure to the vapor is likely.

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NATURAL GAS

DESCRIPTION

Natural gas consists primarily of methane (85%) with lesser amounts of ethane (9%), propane (3%), nitrogen (2%), and butane (1%). Methane is a colorless, odorless, flammable gas.

SYNONYMS

Marsh gas.

POTENTIAL OCCUPATIONAL EXPOSURES

Natural gas is used principally as a heating fuel. It is transported as a liquid under pressure. It is also used in the manufacture of various chemicals including acetaldehyde, acetylene, ammonia, carbon black, ethyl alcohol, formaldehyde, hydrocarbon fuels, hydrogenated oils, methyl alcohol, nitric acids, synthesis gas, and vinyl chloride. Helium can be extracted from certain types of natural gas.

A partial list of occupations in which exposure may occur includes:

- Coal miners
- Electric power plant workers
- Gas fuel users
- Helium extractors
- Hydrogen makers
- Nitric acid makers
- Organic chemical synthesizers
- Petroleum refinery workers
- Synthetic gas makers
- Vinyl chloride makers

PERMISSIBLE EXPOSURE LIMITS

There is no Federal standard for natural gas, methane, nitrogen, or butane. The Federal standard for propane is 1000 ppm (1,800 mg/m^3). (Note: the 1974 ACGIH lists a TLV of 600 ppm (145 mg/m^3) for butane as an intended change.)

ROUTE OF ENTRY

Inhalation of gas.

HARMFUL EFFECTS

Local—

Upon escape from pressurized tanks, natural gas may cause frostbite.

Systemic—

Natural gas is a simple asphyxiant. Displacement of air by the gas may lead to shortness of breath, unconsciousness, and death from hypoxemia. Incomplete combustion may produce carbon monoxide.

MEDICAL SURVEILLANCE

No specific considerations are needed.

SPECIAL TESTS

None are in use.

PERSONAL PROTECTIVE METHOD

Adequate ventilation should quite easily prevent any potential hazard.

PARAFFIN

DESCRIPTION

Paraffin is a white, somewhat translucent solid and consists of a mixture of solid aliphatic hydrocarbons. It may be obtained from petroleum.

SYNONYMS

Paraffin wax, hard paraffin.

POTENTIAL OCCUPATIONAL EXPOSURES

Paraffin is used in the manufacture of paraffin paper, candles, food package materials, varnishes, floor polishes, and cosmetics. It is also used in waterproofing and extracting of essential oils from flowers for perfume.

A partial list of occupations in which exposure may occur includes:

Candle makers	Polish makers
Cosmetic makers	Varnish makers
Perfume makers	Waxpaper makers

PERMISSIBLE EXPOSURE LIMITS

Paraffin wax fume has no established Federal standard; however, in 1975 the ACGIH recommended a TLV of 0.2 mg/m^3 for paraffin wax fume.

ROUTE OF ENTRY

Inhalation of fumes.

HARMFUL EFFECTS

Local—

Occasionally sensitivity reactions have been reported.

Chronic exposure can produce chronic dermatitis, wax boils, folliculitis, comedones, melanoderma, papules, and hyperkeratoses.

Systemic—

Carcinoma of the scrotum in wax pressmen exposed to crude petroleum wax has been documented. Other malignant lesions of an exposed area in employees working with finished paraffin are less well documented. Carcinoma of the scrotum, occurring in workmen exposed 10 years or more, began as a hyperkeratotic nevus-like lesion and developed into a squamous cell carcinoma. The lesions can metastasize to regional inguinal and pelvic lymph nodes. Paraffinoma has been reported from use of paraffin for cosmetic purposes.

MEDICAL SURVEILLANCE

Medical examinations should be concerned especially with the skin. Surveillance should be continued indefinitely.

SPECIAL TESTS

None appear useful.

PERSONAL PROTECTIVE METHODS

Strict personal hygienic measures and protective clothing form the basis of a protective program.

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TURPENTINE

DESCRIPTION

Turpentine is the oleoresin from species of Pinus Pinacae trees. The crude oleoresin (gum turpentine) is a yellowish, sticky, opaque mass and the distillate (oil of turpentine) is a colorless, volatile liquid. Chemically, it contains alpha pinene, beta pinene, camphene, monocyclic terpenes, and terpene alcohols.

SYNONYMS

Gum turpentine, oil of turpentine, spirit of turpentine, gum spirit, gum [derived from pine resin], wood turpentine [derived from pine stumps], sulfate wood pulp waste.

POTENTIAL OCCUPATIONAL EXPOSURES

Turpentines have found wide use as chemical feedstock for the manufacture of floor, furniture, shoe, and automobile polishes, camphor, cleaning materials, inks, putty, mastics, cutting and grinding fluids, paint thinners, resins, and degreasing solutions. Recently, alpha and beta pinenes, which can be extracted, have found use as volatile bases for various compounds.

A partial list of occupations in which exposure may occur includes:

- Art glass workers
- Belt dressing makers
- Camphor makers
- Drug makers
- Furniture polish makers
- Ink makers
- Insecticide makers
- Lacquer makers
- Lithographers
- Oil additive makers
- Paint workers
- Pine oil makers
- Resin makers
- Rubber workers
- Solvent workers
- Stain makers
- Varnish workers
- Wax makers

PERMISSIBLE EXPOSURE LIMITS

The Federal standard for turpentine is 100 ppm (560 mg/m^3).

ROUTE OF ENTRY

Inhalation of vapor and percutaneous absorption of liquid are the usual paths of occupational exposure. However, symptoms have been reported to develop from percutaneous absorption alone.

HARMFUL EFFECTS

Local—

High vapor concentrations are irritating to the eyes, nose, and bronchi. Aspiration of liquid may cause direct lung irritation resulting in pulmonary edema and hemorrhage. Turpentine liquid may produce contact dermatitis. Eczema from turpentine is quite common and has been attributed to the auto-oxidation products of the terpenes (formic acid, formaldehyde, and phenols). This hypersensitivity usually develops in a small portion of the working population. Liquid turpentine splashed in the eyes may cause corneal burns and demands emergency treatment.

Systemic—

Turpentine vapor in acute concentrations may cause central nervous system depression. Symptoms include headache, anorexia, anxiety, excitement, mental confusion, and tinnitus. Convulsions, coma, and death have been reported in animal experiments.

Turpentine vapor also produces kidney and bladder damage. Chronic nephritis with albuminuria and hematuria has been reported as a result of repeated exposures to high concentrations. Predisposition to pneumonia may also occur from such exposures. Recovery usually takes from a few days to a few weeks. Several animal experiments of chronic

low level exposure have produced no ill effects to the central nervous system, kidneys, bladder, or blood.

MEDICAL SURVEILLANCE

Consideration should be given to skin disease or skin allergies in any preplacement or periodic examinations. Liver, renal, and respiratory disease should also be considered.

SPECIAL TESTS

None in common use.

PERSONAL PROTECTIVE METHODS

Rubber gloves, protective clothing, masks for high concentrations.

ALCOHOLS

The alcohols are hydrocarbons in which one or more hydrogen atoms are substituted by hydroxyl (-OH) groups. Compounds with one hydroxyl group are referred to as alcohol. Glycols have two, and glycerols have three, substituted hydroxyl groups. They are widely used as industrial solvents in a variety of products.

In general, alcohols are irritating to mucous membranes. Their toxicity varies, but usually they produce some narcotic effect. They have some disinfectant action and, because of their lipid solubility, most are absorbed to some extent through the skin.

ALLYL ALCOHOL

DESCRIPTION

$H_2C=CHCH_2OH$, allyl alcohol, is a colorless liquid with a pungent odor.

SYNONYMS

Vinyl carbinol, propenyl alcohol, 2-propenol-1, propenol-3.

POTENTIAL OCCUPATIONAL EXPOSURES

Allyl alcohol is primarily used in the production of allyl esters. These compounds are used as monomers and prepolymers in the manufacture of resins and plastics. Allyl alcohol is also used in the preparation of pharmaceuticals, in organic syntheses, and as a fungicide and herbicide.

A partial list of occupations in which exposure may occur includes:

- Acrolein makers
- Allyl ester makers
- Drug makers
- Fungicide makers
- Glycerine makers
- Herbicide makers
- Organic chemical synthesizers
- Plasticizer makers
- Resin makers

PERMISSIBLE EXPOSURE LIMITS

The Federal standard is 2 ppm (5 mg/m^3).

ROUTE OF ENTRY

Inhalation of vapor; percutaneous absorption of liquid.

HARMFUL EFFECTS

Local—

Liquid and vapor are highly irritating to eyes and upper respiratory tract. Skin irritation and burns have occurred from contact with liquid but are usually delayed in onset and may be prolonged.

Systemic—

Local muscle spasms occur at sites of percutaneous absorption. Pulmonary edema, liver and kidney damage, diarrhea, delirium, convulsions, and death have been observed in laboratory animals, but have not been reported in man.

MEDICAL SURVEILLANCE

Preplacement and periodic examinations should include the eyes, skin, respiratory tract, and liver and kidney function.

SPECIAL TESTS

No specific test is available.

PERSONAL PROTECTIVE METHODS

Protective clothing to prevent skin contact should be made of neoprene; these must be discarded at the first sign of deterioration. The odor and irritant properties of allyl alcohol should be sufficient warning to prevent serious injury.

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AMYL ALCOHOL

DESCRIPTION

$C_5H_{11}OH$, amyl alcohol, has eight isomers. All are colorless liquids, except the isomer 2-dimethyl-1-propanol, which is a crystalline solid.

Amyl alcohols are obtained from fusel oil which forms during the fermentation of grain, potatoes, or beets for ethyl alcohol. The fusel oil is a mixture of amyl alcohol isomers, and the composition is determined somewhat by the sugar source. Amyl alcohols may be prepared by acid hydrolysis of a petroleum fraction.

SYNONYMS

Pentanols, pentyl alcohols, fusel oil, grain oil, potato spirit, potato oil.

POTENTIAL OCCUPATIONAL EXPOSURES

Amyl alcohols are used in the manufacture of lacquers, paints, varnishes, paint removers, shoe cements, perfumes, pharmaceuticals, chemicals, rubber, plastics, fruit essences, explosives, hydraulic fluids, ore-flotation agents, in the preparation of other amyl derivatives, in the extraction of fats, and in the textile and petroleum refining industries.

A partial list of occupations in which exposure may occur includes:

- Amyl acetate makers
- Amyl nitrite makers
- Explosive makers
- Fat processors
- Flotation workers
- Lacquer makers
- Mordanters
- Oil processors
- Painters
- Perfume makers
- Petroleum refiners
- Photographic chemical makers
- Plastic makers
- Rubber makers
- Shoe finishers
- Textile workers
- Wax processors

PERMISSIBLE EXPOSURE LIMITS

The Federal standard for 3-methyl-1-butanol (isomyl alcohol) is 100 ppm (360 mg/m^3). There are no standards for the other isomers.

ROUTE OF ENTRY

Inhalation of vapor, percutaneous absorption.

HARMFUL EFFECTS

Local—

The liquid and vapor are mild irritants to the membranes of the eyes and upper respiratory tract and skin.

Systemic—

In low concentrations, amyl alcohol may cause irritation of nose and throat, nausea, vomiting, flushing, headache, diplopia, vertigo, and muscular weakness. In higher dosage, it is a narcotic.

MEDICAL SURVEILLANCE

Consider possible irritant effects on skin and respiratory tract in any preplacement or periodic examinations.

SPECIAL TESTS

None in common use. Amyl alcohol can be determined in blood.

PERSONAL PROTECTIVE METHODS

Barrier creams and personal protective clothing should be used to prevent skin contact.

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n-BUTYL ALCOHOL

DESCRIPTION

$CH_3(CH_2)_3OH$, n-butyl alcohol, is a colorless volatile liquid with a pungent odor.

SYNONYMS

1-Butanol, butyl hydroxide, propylcarbinol, butyric alcohol, hydroxybutane, n-butanol, n-propylcarbinol.

POTENTIAL OCCUPATIONAL EXPOSURES

n-Butyl alcohol is used as a solvent for paints, lacquers, varnishes, natural and synthetic resins, gums, vegetable oils, dyes, camphor, and alkaloids. It is also used as an intermediate in the manufacture of pharmaceuticals and chemicals and in the manufacture of artificial leather, safety glass, rubber and plastic cements, shellac, raincoats, photographic films, perfumes, and in plastic fabrication.

A partial list of occupations in which exposure may occur includes:

- Alkaloid makers
- Detergent makers
- Drug makers
- Dye makers
- Lacquerers
- Photographic film makers
- Plasticizer makers
- Stainers
- Urea-formaldehyde resin makers
- Varnish makers

PERMISSIBLE EXPOSURE LIMITS

The Federal standard is 100 ppm (300 mg/m^3). (Note: the 1976 ACGIH lists a TLV of 50 ppm (150 mg/m^3).)

ROUTE OF ENTRY

Inhalation of vapor and percutaneous absorption.

HARMFUL EFFECTS

Local—

The liquid is a primary skin irritant. The vapor is an irritant to the conjunctiva and mucous membranes of the nose and throat. A mild keratitis characterized by corneal vacuoles has been noted at vapor concentrations over 200 ppm.

Systemic—

Inhalation of high concentrations, in addition to the local effects, have produced transitory and persistent dizziness with Meniere's syndrome. Slight headache and drowsiness may also occur.

MEDICAL SURVEILLANCE

Consider irritant effects on eyes, respiratory tract, and skin in any preplacement or periodic examinations.

SPECIAL TESTS

None have been used. Blood levels can be determined.

PERSONAL PROTECTIVE METHODS

Barrier creams and protective clothing should be used where skin contact may occur.

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ETHYL ALCOHOL

DESCRIPTION

CH_3CH_2OH, ethyl alcohol, is a colorless, volatile, flammable liquid. Ethyl alcohol is produced by fermentation and distillation or by synthesis.

SYNONYMS

Ethanol, grain alcohol, spirit of wine, cologne spirit, ethyl hydroxide, ethyl hydrate.

POTENTIAL OCCUPATIONAL EXPOSURES

Ethyl alcohol is used in the chemical synthesis of a wide variety of compounds such as acetaldehyde, ethyl ether, ethyl chloride, and butadiene. It is a solvent or processing agent in the manufacture of pharmaceuticals, plastics, lacquers, polishes, plasticizers, perfumes, cosmetics, rubber accelerators, explosives, synthetic resins, nitrocellulose, adhesives, inks, and preservatives. It is also used as an antifreeze and as a fuel.

A partial list of occupations in which exposure may occur includes:

- Acetaldehyde makers
- Acetic anhydride makers
- Adhesive makers
- Beverage makers
- Detergent makers
- Distillers
- Dye makers
- Ethyl ether makers
- Histology technicians
- Ink makers
- Lacquer makers
- Motor fuel blenders
- Organic chemical synthesizers
- Rubber makers
- Shellac processors
- Solvent workers
- Stainers

PERMISSIBLE EXPOSURE LIMITS

The Federal standard is 1,000 ppm (1,900 mg/m^3).

ROUTE OF ENTRY

Inhalation of vapor and percutaneous absorption.

HARMFUL EFFECTS

Local—

Mild irritation of eye and nose occurs at very high concentrations.

The liquid can defat the skin, producing a dermatitis characterized by drying and fissuring.

Systemic—

Prolonged inhalation of high concentrations, besides the local effect on the eyes and upper respiratory tract, may produce headache, drowsiness, tremors, and fatigue. Tolerance may be a factor in individual response to a given air concentration.

Bizarre symptoms (other than typical manifestations of intoxication) may result from the denaturants often present in industrial ethyl alcohol. Ethyl alcohol may act as an adjuvant, increasing the toxicity of other inhaled, absorbed, or ingested chemical agents. An exception is methanol where ethyl alcohol counteracts methanol toxicity.

MEDICAL SURVEILLANCE

No special considerations needed.

SPECIAL TESTS

Ethyl alcohol can readily be determined in blood, urine, and expired air.

PERSONAL PROTECTIVE METHODS

Personal protective equipment is recommended where skin contact may occur.

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ETHYLENE CHLOROHYDRIN

DESCRIPTION

CH_2ClCH_2OH, ethylene chlorohydrin, is a colorless liquid with an ether-like odor.

SYNONYMS

Glycol chlorohydrin, 2-chloroethanol, β-chloroethyl alcohol.

POTENTIAL OCCUPATIONAL EXPOSURES

Ethylene chlorohydrin is used in the synthesis of ethylene glycol, ethylene oxide, amines, carbitols, indigo, malonic acid, novocaine, and in other reactions where the hydroxyethyl group ($-CH_2CH_2OH$) is introduced into organic compounds, for the separation of butadiene from hydrocarbon mixtures, in dewaxing and removing cycloalkanes from mineral oil, in the refining of rosin, in the manufacture of certain pesti-

cides, and in the extraction of pine lignin. In the lacquer industry, it is used as a solvent for cellulose acetate, cellulose esters, resins and waxes, and in the dyeing and cleaning industry, it is used to remove tar spots, as a cleaning agent for machines, and as a solvent in fabric dyeing. It has also found use in agriculture in speeding up sprouting of potatoes and in treating seeds to inhibit biological activity.

A partial list of occupations in which exposure may occur includes:

- Cellulose acetate makers
- Drug makers
- Dye makers
- Ethyl cellulose workers
- Indigo makers
- Lacquer makers
- Novocaine makers
- Organic chemical synthesizers
- Potato growers
- Procaine makers
- Resin workers
- Textile dyers and printers

PERMISSIBLE EXPOSURE LIMITS

The Federal standard is 5 ppm (16 mg/m^3).

ROUTE OF ENTRY

Inhalation of vapor; percutaneous absorption of liquid.

HARMFUL EFFECTS

Local—

High vapor concentrations are irritating to the eyes, nose, throat, and skin.

Systemic—

Ethylene chlorohydrin is extremely toxic and in addition to local irritation of eyes, respiratory tract, and skin, inhalation of the vapor may produce nausea, vomiting, dizziness, headache, thirst, delirium, low blood pressure, collapse, and unconsciousness. The urine may show red cells, albumin, and casts. Death may occur in high concentrations with damage to the lung and brain. There is little margin of safety between early reversible symptoms and fatal intoxication. The toxic effects may be related to its metabolites, chloroacetaldehyde and chloroacetic acid.

MEDICAL SURVEILLANCE

Preplacement examination, including a complete history and physical should be performed. Examination of the respiratory system, liver, kidneys, and central nervous system should be stressed. The skin should be examined. A chest X-ray should be taken and pulmonary function tests performed (FVC-FEV).

The above procedures should be repeated on an annual basis, except that the X-ray is needed only when indicated by pulmonary function testing.

SPECIAL TESTS

None commonly used. Presence in blood can probably be determined by appropriate gas chromatographic methods.

PERSONAL PROTECTIVE METHODS

The liquid readily penetrates rubber. Protective clothing should be discarded at first sign of deterioration. Barrier creams may be used and scrupulous personal hygiene should be practiced.

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METHYL ALCOHOL

DESCRIPTION

CH_3OH, methyl alcohol, is a colorless, volatile liquid with a mild odor.

SYNONYMS

Methanol, carbinol, wood alcohol, wood spirit.

POTENTIAL OCCUPATIONAL EXPOSURES

Methyl alcohol is used as a starting material in organic synthesis of chemicals such as formaldehyde, methacrylates, methyl amines, methyl halides, and ethylene glycol, and as an industrial solvent for inks, resins, adhesives, and dyes for straw hats. It is an ingredient in paint and varnish removers, cleaning and dewaxing preparations, spirit duplicating fluids, embalming fluids, antifreeze mixtures, and enamels and is used in the manufacture of photographic film, plastics, celluloid, textile soaps, wood stains, coated fabrics, shatterproof glass, paper coating, waterproofing formulations, artificial leather, and synthetic indigo and other dyes. It has also found use as an extractant in many processes, an antidetonant fuel-injection fluid for aircraft, a rubber accelerator, and a denaturant for ethyl alcohol.

A partial list of occupations in which exposures may occur includes:

Acetic acid makers
Art glass workers
Bookbinders
Bronzers
Dyers
Enamel makers
Ester makers
Feather workers
Felt hat makers
Foundry workers
Gilders
Ink makers
Lasters
Leather workers
Millinery workers
Painters
Photoengravers

PERMISSIBLE EXPOSURE LIMITS

The Federal standard is 200 ppm (260 mg/m^3).

ROUTE OF ENTRY

Inhalation of vapor; percutaneous absorption of liquid.

HARMFUL EFFECTS

Local—

Contact with liquid can produce defatting and a mild dermatitis. Methyl alcohol is virtually nonirritating to the eyes or upper respiratory tract below 2,000 ppm, and it is difficult to detect by odor at less than this level.

Systemic—

Methyl alcohol may cause optic nerve damage and blindness. Its toxic effect is thought to be mediated through metabolic oxidation products, such as formaldehyde or formic acid, and may result in blurring of vision, pain in eyes, loss of central vision, or blindness. Other central nervous system effects result from narcosis and include headache, nausea, giddiness, and loss of consciousness. Formic acid, may produce acidosis. These symptoms occur principally after oral ingestion and are very rare after inhalation.

MEDICAL SURVEILLANCE

Consider eye disease and visual acuity in any periodic or placement examinations, as well as skin and liver and kidney functions.

SPECIAL TESTS

Determination of methyl alcohol in blood, and methyl alcohol and formic acid in urine. Estimation of alkali reserve which may be impaired because of acidosis following accidental ingestion.

PERSONAL PROTECTIVE METHODS

Barrier creams and protective clothing.

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Keeney, A. H., and S. M. Mellingkoff. 1951. Methyl alcohol poisoning. Ann. Intern. Med. 34:331.

PROPYL ALCOHOL

DESCRIPTION

There are two isomers of propyl alcohol, n-propyl alcohol (CH_3-CH_2CH_2OH) and isopropyl alcohol ($CH_3CHOHCH_3$). Both are colorless, volatile liquids.

SYNONYMS

n-Propyl alcohol: 1-Propanol, propylic alcohol. Isopropyl alcohol: Isopropanol, 2-propanol, secondary propyl alcohol, dimethylcarbinol.

POTENTIAL OCCUPATIONAL EXPOSURES

Isopropyl alcohol is the more widely used of the two isomers. In the pharmaceutical industry, it has replaced ethyl alcohol in liniments,

skin lotions, cosmetics, permanent wave preparations, hair tonics, mouth washes, and skin disinfectants and is widely used as a rubbing alcohol. Isopropyl alcohol is used in the manufacture of acetone, isopropyl derivatives, and safety glass, as a solvent in perfumes, resins and plastics, dye solutions, nitrocellulose lacquers, and in many extraction processes and is an ingredient of antifreezes, deicing agents, liquid soaps, and window cleaners. Further applications include use as a preservative and dehydrating agent, a coupling agent in oil emulsions, an extracting agent for sulfonic acids from petroleum oils, and for coatings in textiles. n-Propyl alcohol is used in lacquers, dopes, cosmetics, dental lotions, cleaners, polishes, and pharmaceuticals and as a surgical antiseptic. It is a solvent for vegetable oils, natural gums and resins, rosin, shellac, certain synthetic resins, ethyl cellulose, and butyral.

A partial list of occupations in which exposure may occur includes:

Disinfectant makers
Drug makers
Gum processors
Metal degreasers
Nurses
Perfume makers
Polish makers
Resin makers
Soap makers
Stainers
Vegetable oil processors
Wax makers
Window cleaning fluid makers

PERMISSIBLE EXPOSURE LIMITS

The Federal standard for n-propyl alcohol is 200 ppm (500 mg/m^3) and for isopropyl alcohol, 400 ppm (980 mg/m^3).

ROUTE OF ENTRY

Isopropyl alcohol: Inhalation of vapor.
n-Propyl alcohol: Inhalation of vapor, percutaneous absorption.

HARMFUL EFFECTS

Local—

The two isomers are similar in physical and in most physiological properties. The vapors are mildly irritating to the conjunctiva and mucous membranes of the upper respiratory tract.

Systemic—

No cases of poisoning from industrial exposure have been recorded for either isomer. n-Propyl alcohol can produce mild central nervous system depression; isopropyl alcohol is potentially narcotic in high concentrations.

MEDICAL SURVEILLANCE

No specific considerations are needed.

SPECIAL TESTS

Isopropyl alcohol and its metabolite, acetone, may be detected in blood, urine, and body tissues.

PERSONAL PROTECTIVE METHODS

Clothing and barrier creams are recommended.

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Von Oettingen, W. F. 1943. The aliphatic alcohols: their toxicity and potential dangers in relations to their chemical constitution and their fate in metabolism. Public Health Bulletin No. 281. U.S. Public Health Service, p. 112.

GLYCOLS AND DERIVATIVES

Glycols are dihydric alcohols which are colorless, viscous liquids. Because these compounds are soluble in alcohol and water and have high boiling points and low freezing points, they are used as solvents and antifreeze. Ethylene glycol, like ethyl alcohol, is often called by the class name, i.e., glycol. These compounds have relatively low toxicity, and the major hazard appears when the liquids are heated during processing.

ETHYLENE GLYCOL

DESCRIPTION

$HOCH_2CH_2OH$, ethylene glycol, is a colorless, odorless, viscous liquid with a sweetish taste.

SYNONYMS

1, 2-Ethanediol, glycol alcohol, glycol, EG.

POTENTIAL OCCUPATIONAL EXPOSURES

Because of ethylene glycol's physical properties, it is used in antifreeze, hydraulic fluids, electrolytic condensors, and heat exchangers. It is also used as a solvent and as a chemical intermediate for ethylene glycol dinitrate, glycol esters, and resins.

A partial list of occupations in which exposure may occur includes:

- Antifreeze makers
- Brake fluid makers
- Explosive makers
- Glue makers
- Hydraulic fluid makers
- Ink makers
- Metal cleaners
- Metal polishers
- Paint makers
- Resin makers
- Textile makers
- Tobacco workers
- Wax makers

PERMISSIBLE EXPOSURE LIMITS

There is no Federal standard; however, ACGIH in 1975 recommended a TLV of 10 mg/m^3 for particulate ethylene glycol and 100 ppm (260 mg/m^3) for the vapor form.

ROUTE OF ENTRY

Inhalation of particulate or vapor. Percutaneous absorption may also contribute to intoxication.

HARMFUL EFFECTS

Local—

None.

Systemic—

Ethylene glycol's vapor pressure is such that at room temperature toxic concentrations are unlikely to occur. Poisoning resulting from vapor usually occurs only if ethylene glycol liquid is heated; therefore, occupational exposure is rare. Chronic symptoms and signs include: anorexia, oliguria, nystagmus, lymphocytosis, and loss of consciousness. Inhalation seems to primarily result in central nervous system depression and hematopoietic dysfunction, whereas, ingestion may result in depression followed by respiratory and cardiac failure, renal and brain damage.

MEDICAL SURVEILLANCE

No special considerations are needed.

SPECIAL TESTS

Urinalysis for oxalic acid, an ethylene glycol metabolite, may be useful in diagnosis of poisoning by oral ingestion.

PERSONAL PROTECTIVE METHODS

Masks should be worn in areas of vapor concentration.

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Troisi, F. M. 1950. Chronic intoxication by ethylene glycol vapor. Br. J. Ind. Med. 7:65.

ETHYLENE GLYCOL ETHERS AND DERIVATIVES

DESCRIPTION

Ethylene glycol monoethyl ether: $CH_3CH_2OCH_2CH_2OH$.

Ethylene glycol monoethyl ether acetate: $CH_3CH_2OCH_2CH_2OOCCH_3$.

Ethylene glycol monomethyl ether: $CH_3OCH_2CH_2OH$.

Ethylene glycol monomethyl ether acetate: $CH_3OCH_2CH_2OOCCH_3$.

Ethylene glycol monobutyl ether: $CH_3CH_2CH_2CH_2OCH_2CH_2OH$.

These substances are colorless liquids with a slight odor.

SYNONYMS

Ethylene glycol monoethyl ether: cellosolve, 2-ethoxyethanol.

Ethylene glycol monoethyl ether acetate: cellosolve acetate, 2-ethoxyethyl acetate.

Ethylene glycol monomethyl ether: methyl cellosolve, 2-methoxyethanol.

Ethylene glycol monomethyl ether acetate: methyl cellosolve acetate, 2-methoxyethyl acetate.

Ethylene glycol monobutyl ether: butyl cellosolve, 2-butoxyethanol.

POTENTIAL OCCUPATIONAL EXPOSURES

Ethylene glycol ethers are used as solvents for resins, lacquers, paints, varnishes, gum, perfume, dyes and inks, and as a constituent of painting pastes, cleaning compounds, liquid soaps, cosmetics, nitrocellulose, and hydraulic fluids. Acetate derivatives are used as solvents for oils, greases and ink, in the preparation of lacquers, enamels, and adhesives, and to dissolve resins and plastics.

A partial list of occupations in which exposure may occur includes:

- Cellophane sealers
- Cleaning solution makers
- Dry cleaners,
- Film makers
- Hydraulic fluid makers
- Ink makers
- Lacquer makers
- Nail polish makers
- Oil processors
- Plastic makers
- Printers
- Stainers
- Textile dyers
- Wax processors

PERMISSIBLE EXPOSURE LIMITS

The Federal standards for these compounds are:

Ethylene glycol monoethyl ether	200 ppm	740 mg/m^3
Ethylene glycol monoethyl ether acetate	100 ppm	540 mg/m^3
Ethylene glycol monomethyl ether	25 ppm	80 mg/m^3
Ethylene glycol monomethyl ether acetate	25 ppm	[1]20 mg/m^3
Ethylene glycol monobutyl ether	50 ppm	240 mg/m^3

ACGIH in 1975 recommended a TLV of 25 ppm (120 mg/m^3) for ethylene glycol monomethyl ether acetate.

ROUTE OF ENTRY

Inhalation of vapor and percutaneous absorption of liquid.

HARMFUL EFFECTS

Local—

Ethylene glycol ethers are only mildly irritating to the skin. Vapor may cause conjunctivitis and upper respiratory tract irritation. Temporary corneal clouding may also result and may last several hours. Acetate derivatives cause greater eye irritation than the parent compounds. The butyl and methyl ethers may penetrate skin readily.

Systemic—

Acute exposure to these compounds results in narcosis, pulmonary

edema, and severe kidney and liver damage. Symptoms from repeated overexposure to vapors are fatigue and lethargy, headache, nausea, anorexia, and tremor. Anemia and encephalopathy have been reported with ethylene glycol monomethyl ether. Rats show increased hemolysis of erythrocytes from inhalation of ethylene glycol monobutyl ether. This has not been shown in man. Acute poisoning by ingestion resembles ethylene glycol toxicity, with death from renal failure.

MEDICAL SURVEILLANCE

Preplacement and periodic examinations should evaluate blood, central nervous system, renal and liver functions, as well as the skin and respiratory tract.

SPECIAL TESTS

None currently used.

PERSONAL PROTECTIVE METHODS

Use glasses and protective clothing to prevent skin absorption. Respiratory protection may be needed if ventilation is poor or if compounds are heated or atomized.

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Zavon, M. R. 1963. Methyl cellosolve intoxication. Am. Ind. Hyg. Assoc. J. 24:36.

ETHERS AND EPOXY COMPOUNDS

Ethers are organic molecules which contain a carbon-oxygen-carbon linkage. Colorless, volatile liquids, these compounds are generally used industrially as solvents and chemical feedstock for organic synthetics. Ethyl ether, the simplest ether, has been used as a general anesthetic and has been historically known as "ether." Occupationally, exposure to chlorinated ethers is much more significant. Two compounds, bis-(chloromethyl) ether and chloromethyl methyl ether, have produced carcinoma and, therefore, have received much attention recently. Dioxane, although it is not chlorinated, has also shown potential tumorigenic hazard. Skin, eye, and mucous membrane irritation is common to all the chemicals covered. Pulmonary edema, with the added hazard of delayed appearance, may also occur as a result of particular ether exposures.

Epoxy compounds are cyclic ethers with the structure -C-O-C-. The most important industrially are the alpha-epoxy compounds in which the epoxy group is in the 1-2 position. These are the most reactive and are used as chemical intermediates in the manufacture of surface-active agents, plasticizers, synthetic resins, solvents, etc.

BIS(CHLOROMETHYL) ETHER

DESCRIPTION

$ClCH_2OCH_2Cl$, bis(chloromethyl) ether, is a colorless, volatile liquid with a suffocating odor. This substance may form spontaneously in warm moist air by the combination of formaldehyde and hydrogen chloride.

SYNONYMS

BCME, sym-dichloromethyl ether.

POTENTIAL OCCUPATIONAL EXPOSURES

Exposure to bis(chloromethyl) ether may occur in industry and in the laboratory. This compound is used as an alkylating agent in the manufacture of polymers, as a solvent for polymerization reactions, in the preparation of ion exchange resins, and as an intermediate for organic synthesis.

A partial list of occupations in which exposure may occur includes:

Ion exchange resin makers	Organic chemical synthesizers
Laboratory workers	Polymer makers

PERMISSIBLE EXPOSURE LIMITS

Bis(chloromethyl) ether is included in the Federal standard for carcinogens; all contact with it should be avoided.

ROUTE OF ENTRY

Inhalation of vapor and perhaps, but to a lesser extent, percutaneous absorption.

HARMFUL EFFECTS

Local—

Vapor is severely irritating to the skin and mucous membranes and may cause corneal damage which may heal slowly.

Systemic—

Bis(chloromethyl) ether has an extremely suffocating odor even in minimal concentration so that experience with acute poisoning is not available. It is not considered a respiratory irritant at concentrations of 10 ppm. Bis(chloromethyl) ether is a known human carcinogen. Animal experiments have shown increases in lung adenoma incidence; olfactory esthesioneuroepitheliomas which invaded the sinuses, cranial vault, and brain; skin papillomas and carcinomas; and subcutaneous fibrosarcomas. There have been several reports of increased incidence of human lung carcinomas (primarily small cell undifferentiated) among ether workers exposed to bis(chloromethyl) ether as an impurity. The latency period is relatively short — 10 to 15 years. Smokers as well as non-smokers may be affected.

MEDICAL SURVEILLANCE

Preplacement and periodic medical examinations should include an examination of the skin and respiratory tract, including chest X-ray. Sputum cytology has been suggested as helpful in detecting early malignant changes, and in this connection a smoking history is of importance. Possible effects on the fetus should be considered.

SPECIAL TESTS

None have been suggested.

PERSONAL PROTECTIVE METHODS

These are designed to supplement engineering controls and should be appropriate for protection of all skin or respiratory contact. Full body protective clothing and gloves should be used on entering areas of potential exposure. Those employed in handling operations should be provided with full face, supplied air respirators of continuous flow or pressure demand type. On exit from a regulated area, employees should remove and leave protective clothing and equipment at the point of exit, to be placed in impervious containers at the end of the work shift for decontamination or disposal. Showers should be taken before dressing in street clothes.

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CHLOROMETHYL METHYL ETHER

DESCRIPTION

$ClCH_2OCH_3$, chloromethyl methyl ether, is a volatile, corrosive liquid. Commercial chloromethyl methyl ether contains from 1 to 7 per cent bis(chloromethyl) ether, a known carcinogen.

SYNONYMS

CMME, methyl chloromethyl ether, monochloromethyl ether, chloromethoxymethane.

POTENTIAL OCCUPATIONAL EXPOSURES

Chloromethyl methyl ether is a highly reactive methylating agent and is used in the chemical industry for synthesis of organic chemicals. Most industrial operations are carried out in closed process vessels so that exposure is minimized.

A partial list of occupations in which exposure may occur includes: Organic chemical synthesizers

PERMISSIBLE EXPOSURE LIMITS

Chloromethyl methyl ether is included in the Federal standard for carcinogens; all contact with it should be avoided.

ROUTE OF ENTRY

Inhalation of vapor and possibly percutaneous absorption.

HARMFUL EFFECTS

Local—

Vapor exposure results in severe irritation of the skin, eyes, and nose. Rabbit skin tests using undiluted material resulted in skin necrosis.

Systemic—

Chloromethyl methyl ether is only moderately toxic given orally. Acute exposure to chloromethyl methyl ether vapor may result in pulmonary edema and pneumonia.

Several studies of workers with CMME manufacturing exposure have shown an excess of bronchiogenic cancer predominately of the small cell-undifferentiated type with relatively short latency period (typically 10-15 years). Therefore, commercial grade chloromethyl methyl ether must be considered a carcinogen. At present it is not known whether or not chloromethyl methyl ether's carcinogenic activity is due to bis(chloromethyl) ether (BCME) contamination, but this may be a moot question inasmuch as two of the hydrolysis products of CMME can combine to form BCME. Animal experiments to determine chloromethyl methyl ether's ability to produce skin cancer indicated marginal carcinogenic activity; highly pure CMME was used. Inhalation studies, using technical grade CMME showed only one bronchiogenic cancer and one esthesioneuroepithelioma out of 79 animals exposed.

MEDICAL SURVEILLANCE

Preplacement and periodic medical examinations should include an examination of the skin and respiratory tract, including a chest X-ray. Sputum cytology has been suggested as helpful in detecting early malignant changes, and in this connection a detailed smoking history is of importance. Possible effects on the fetus should be considered.

SPECIAL TESTS

None have been suggested.

PERSONAL PROTECTIVE METHODS

These are designed to supplement engineering controls and to prevent all skin or respiratory contact. Full body protective clothing and gloves should be used on entering areas of partial exposure. Those employed in handling operations should be provided with fullface, sup-

plied air respirators of continuous flow or pressure demand type. On exit from a regulated area, employees should be required to remove and leave protective clothing and equipment at the point of exit, to be placed in impervious containers at the end of the work-shift for decontamination or disposal. Showers should be taken prior to dressing in street clothes.

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DICHLOROETHYL ETHER

DESCRIPTION

$ClCH_2CH_2OCH_2CH_2Cl$, dichloroethyl ether, is a clear, colorless liquid with a pungent, fruity odor.

SYNONYMS

Dichloroether, dichloroethyl oxide, sym-dichloroethyl ether, bis-(2-chloroethyl) ether, 2, 2-dichloroethyl ether.

POTENTIAL OCCUPATIONAL EXPOSURES

Dichloroethyl ether is used in the manufacture of paint, varnish, lacquer, soap, and finish remover. It is also used as a solvent for cellulose esters, naphthalenes, oils, fats, greases, pectin, tar, and gum; in drycleaning; in textile scouring; and in soil fumigation.

A partial list of occupations in which exposure may occur includes:

- Cellulose ester makers
- Degreasers
- Drycleaners
- Ethyl cellulose processors
- Fat processors
- Gum processors
- Lacquer makers
- Oil processors
- Paint makers
- Soap makers
- Tar processors
- Textile scourers
- Varnish workers

PERMISSIBLE EXPOSURE LIMITS

The Federal standard for dichloroethyl ether is 15 ppm (90 mg/m^3); however, the ACGIH recommended TLV in 1975 was 5 ppm (30 mg/m^3).

ROUTE OF ENTRY

Inhalation of vapor, percutaneous absorption.

HARMFUL EFFECTS

Local—

Irritation of the conjunctiva of the eyes with profuse lacrimation, irritation to mucous membranes of upper respiratory tract, coughing, and nausea may result from exposure to vapor. The liquid when placed in animal eyes has produced damage. Vapors in minimal concentrations (3 ppm) are distinctly irritating and serve as a warning property.

Systemic—

In animal experiments dichloroethyl ether has caused severe irritation of the respiratory tract and pulmonary edema. Animal experiments have also shown dichloroethyl ether to be capable of causing drowsiness, dizziness, and unconsciousness at high concentrations. Except for accidental inhalation of high concentrations, the chief hazard in industrial practice is a mild bronchitis which may be caused by repeated exposure to low concentrations.

MEDICAL SURVEILLANCE

Consideration should be given to the skin, eyes, and respiratory tract, and to the central nervous system in placement or periodic examinations.

SPECIAL TESTS

None have been proposed.

PERSONAL PROTECTIVE METHODS

In cases of vapor concentrations, protective clothing with full-face respirator with air supply should be worn. Skin protection (gloves, protective clothing) is needed to prevent skin absorption. Goggles should be used to prevent eye burns.

BIBLIOGRAPHY

Schrenk, H. H. 1933. Acute response of guinea pigs to vapors of some new commercial organic compounds. VII. Dichloroethyl ether. Public Health Reports 48:1389.

DIOXANE

DESCRIPTION

$OCH_2CH_2OCH_2CH_2$, dioxane, is a volatile, colorless liquid that may form explosive peroxides during storage.

SYNONYMS

1,4-Diethylene dioxide, diethylethene ether, 1,4-dioxane, para-dioxane.

POTENTIAL OCCUPATIONAL EXPOSURES

Dioxane finds its primary use as a solvent for cellulose acetate,

dyes, fats, greases, lacquers, mineral oil, paints, polyvinyl polymers, resins, varnishes, and waxes. It finds particular usage in paint and varnish strippers, as a wetting agent and dispersing agent in textile processing, dye baths, stain and printing composition, and in the preparation of histological slides.

A partial list of occupations in which exposure may occur includes:

Adhesive workers
Cellulose acetate workers
Cement workers
Degreasers
Deodorant makers
Detergent workers
Emulsion makers
Fat processors
Glue makers
Histology technicians
Lacquer makers
Metal cleaners
Oil processors
Paint makers
Polish makers
Shoe cream makers
Varnish remover makers
Wax makers

PERMISSIBLE EXPOSURE LIMITS

The Federal standard is 100 ppm (360 mg/m^3); however, the ACGIH 1975 recommended TLV was 50 ppm (180 mg/m^3) of technical grade.

ROUTE OF ENTRY

Inhalation of vapor as well as percutaneous absorption.

HARMFUL EFFECTS

Local—

Liquid and vapor may be irritating to eyes, nose, and throat.

Systemic—

Exposure to dioxane vapor may cause drowsiness, dizziness, loss of appetite, headache, nausea, vomiting, stomach pain, and liver and kidney damage. Prolonged skin exposure to the liquid may cause drying and cracking.

MEDICAL SURVEILLANCE

Preplacement and periodic examinations should be directed to symptoms of headache and dizziness, as well as nausea and other gastrointestinal disturbances. The condition of the skin and of renal and liver function should be considered.

SPECIAL TESTS

No specific bio-monitoring tests are available.

PERSONAL PROTECTIVE METHODS

In areas of vapor concentration, protective clothing, barrier creams, gloves, and masks should be used.

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Hoch-Ligeti, C., M. F. Argus, and J. C. Arcos. 1970. Induction of carcinomas in the nasal cavity of rats by dioxane. Br. J. Cancer 24: 164.

Johnstone, R. T. 1959. Death due to dioxane? Arch Ind. Health 20:445.

EPICHLOROHYDRIN

DESCRIPTION

CH_2OCHCH_2Cl, epichlorohydrin, is a colorless liquid with a chloroform-like odor.

SYNONYMS

Epi, chloropropylene oxide, 1-chloro-2,3-epoxypropane, chloromethyloxidrane, 2-epichlorohydrin.

POTENTIAL OCCUPATIONAL EXPOSURES

Epichlorohydrin is used in the manufacture of many glycerol and glycidol derivatives and epoxy resins, as a stabilizer in chlorine-containing materials, as an intermediate in the preparation of cellulose esters and ethers, paints, varnishes, nail enamels, and lacquers, and as a cement for celluloid.

A partial list of occupations in which exposure may occur includes:

- Cellulose ether workers
- Epoxy resin makers
- Glycerol derivative makers
- Glycerophosphoric acid makers
- Glycidol derivative makers
- Gum processors
- Lacquerers
- Lacquer makers
- Nail enamel makers
- Organic chemical synthesizers
- Paint makers
- Resin makers
- Solvent workers
- Varnish makers

PERMISSIBLE EXPOSURE LIMITS

The Federal standard is 5 ppm (19 mg/m^3). NIOSH has recommended a time-weighted average limit of 2 mg/m^3 with a ceiling concentration of 19 mg/m^3 based on a 15-minute sampling period.

ROUTE OF ENTRY

Inhalation of vapor, percutaneous absorption of liquid.

HARMFUL EFFECTS

Local—

Epichlorohydrin is highly irritating to eyes, skin, and respiratory tract. Skin contact may result in delayed blistering and deep-seated pain. Allergic eczematous contact dermatitis occurs occasionally.

Systemic—

The earliest symptoms of intoxication may be referable to the gastrointestinal tract (nausea, vomiting, abdominal discomfort) or pain in the region of the liver. Labored breathing, cough, and cyanoses may be

evident and the onset of chemical pneumonitis may occur several hours after exposure. Animals exposed repeatedly to this chemical have developed lung, kidney, and liver injury.

MEDICAL SURVEILLANCE

Consider possible effects on the skin, eyes, lungs, liver, and kidney in preplacement or periodic examinations.

SPECIAL TESTS

None currently used.

PERSONAL PROTECTIVE METHODS

Goggles and rubber, protective clothing should be worn. Epichlorohydrin slowly penetrates rubber, so all contaminated clothing should be thoroughly washed. Respirators are required in areas of vapor concentrations.

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Shell Chemical Company. 1972. Epichlorohydrin — Industrial Hygiene Bulletin. Shell Chemical Company, New York.

ETHYLENE OXIDE

DESCRIPTION

H_2COCH_2, ethylene oxide, is a colorless gas with a sweetish odor.

SYNONYMS

1,2-Epoxyethane, oxirane, dimethylene oxide, anprolene.

POTENTIAL OCCUPATIONAL EXPOSURES

Ethylene oxide is used as an intermediate in organic synthesis for ethylene glycol, polyglycols, glycol ethers esters, ethanolamines, acrylonitrile, plastics, and surface-active agents. It is also used as a fumigant for foodstuffs and textiles, an agricultural fungicide, and for sterilization, especially for surgical instruments.

A partial list of occupations in which exposure may occur includes:

- Acrylonitrile makers
- Butyl cellosolve makers
- Detergent makers
- Disinfectant makers
- Ethanolamine makers
- Ethylene glycol makers
- Exterminators
- Foodstuff fumigators
- Fumigant makers
- Fungicide workers
- Gasoline sweeteners
- Grain elevator workers
- Organic chemical synthesizers
- Polyglycol makers
- Polyoxirane makers
- Rocket fuel handlers
- Surfactant makers
- Textile fumigators

PERMISSIBLE EXPOSURE LIMITS

The Federal standard is 50 ppm (90 mg/m^3).

ROUTE OF ENTRY

Inhalation of gas.

HARMFUL EFFECTS

Local—

Aqueous solutions of ethylene oxide or solutions formed when the anhydrous compound comes in contact with moist skin are irritating and may lead to a severe dermatitis with blisters, blebs, and burns. It is also absorbed by leather and rubber and may produce burns or irritation. Allergic eczematous dermatitis has also been reported. Exposure to the vapor in high concentrations leads to irritation of the eyes. Severe eye damage may result if the liquid is splashed in the eyes. Large amounts of ethylene oxide evaporating from the skin may cause frostbite.

Systemic—

Breathing high concentrations of ethylene oxide may cause nausea, vomiting, irritation of the nose, throat, and lungs. Pulmonary edema may occur. In addition, drowsiness and unconsciousness may occur. Ethylene oxide has been found to cause cancer in female mice exposed to it for prolonged periods.

Ethylene oxide is a well-known mutagen in commercial use in plants. No mutagenic effect has been demonstrated in man or animals.

MEDICAL SURVEILLANCE

Preplacement and periodic examinations should consider the skin and eyes, allergic history, the respiratory tract, blood, liver, and kidney function.

SPECIAL TESTS

None in common use.

PERSONAL PROTECTIVE METHODS

Eyes and skin should be protected and protective clothing changed when it is contaminated. In areas of high vapor concentration, respirators should be supplied to cover the face, including eyes. Shoes contaminated by this chemical should be discarded.

BIBLIOGRAPHY

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Jacobson, K. H., E. B. Hackley, and L. Feinsilver. 1956. The toxicity of inhaled ethylene oxide and propylene oxide vapors. AMA Arch. Ind. Health 13:237.

Sexton, R. J., and E. V. Henson. 1949. Dermatological injuries by ethylene oxide. J. Ind. Hyg. Toxicol. 31:297.

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ETHYL ETHER

DESCRIPTION

$CH_3CH_2OCH_2CH_3$, ethyl ether, is a colorless, mobile, highly flammable, volatile liquid with a characteristic pungent odor.

SYNONYMS

Anesthetic ether, diethyl ether, diethyl oxide, ether, ethoxyethane, ethyl oxide, sulfuric ether.

POTENTIAL OCCUPATIONAL EXPOSURES

Ethyl ether is used as a solvent for waxes, fats, oils, perfumes, alkaloids, dyes, gums, resins, nitrocellulose, hydrocarbons, raw rubber, and smokeless powder. It is also used as an inhalation anesthetic, a refrigerant, in diesel fuels, in dry cleaning, as an extractant, and as a chemical reagent for various organic reactions.

A partial list of occupations in which exposure may occur includes:

- Acetic acid makers
- Alcohol denaturers
- Collodion makers
- Diesel fuel blenders
- Drug makers
- Explosive makers
- Fat processors
- Gasoline engine primers
- Dye makers
- Gum processors
- Motor fuel makers
- Nitrocellulose makers
- Oil processors
- Perfume makers
- Rayon makers
- Rubber workers
- Smokeless powder makers
- Wax makers

PERMISSIBLE EXPOSURE LIMITS

The Federal standard is 400 ppm (1,200 mg/m^3).

ROUTE OF ENTRY

Inhalation of vapor.

HARMFUL EFFECTS

Local—

Ethyl ether vapor is mildly irritating to the eyes, nose, and throat. Contact with liquid may produce a dry, scaly, fissured dermatitis.

Systemic—

Ethyl ether has predominantly narcotic properties. Overexposed individuals may experience drowsiness, vomiting, and unconsciousness. Death may result from severe overexposure. Chronic exposure results in some persons in anorexia, exhaustion, headache, drowsiness, dizziness, excitation, and psychic disturbances. Albuminuria has been reported. Chronic exposure may cause an increased susceptibility to alcohol.

MEDICAL SURVEILLANCE

Preplacement or periodic examinations should evaluate the skin and respiratory tract, liver, and kidney function. Persons with a past history of alcoholism may be at some increased risk due to possibility of ethyl ether addiction (known as "ether habit").

SPECIAL TESTS

Tests for exposure may include expired breath for unmetabolized ethyl ether and blood for ethyl ether content by oxidation with chromate solution or by gas chromatographic methods.

PERSONAL PROTECTIVE METHODS

Barrier creams, gloves, protective clothing, and, in areas of vapor concentration, fullface respirator should be used.

BIBLIOGRAPHY

Hamilton, A., and G. R. Minot. 1920. Ether poisoning in the manufacture of smokeless powder. J. Ind. Hyg. 2:41.

Von Oettingen, W. F. 1958. Poisoning, A Guide to Clinical Diagnosis and Treatment, 2nd ed. W. B. Saunders Co., Philadelphia.

ESTERS

Esters are organic compounds with the structure R-COOR′. They are generally the result of the reaction between an organic or inorganic acid and alcohol with the elimination of water; however, other reactions, such as between an alcohol and an acid halide, will also form esters. The organic acid may be aliphatic or aromatic and may contain other substituents. Mono-, di-, or tricarboxylic esters may be formed.

Esters are an industrially important group of compounds. They are used in plastics and resins, as plasticizers, in lacquer solvents, in flavors and perfumes, in pharmaceuticals, and in industries such as automotive, aircraft, food processing, chemical, pharmaceutical, soap, cosmetic, surface coating, textiles, and leather.

There are four basic types of physiological effects of esters, and these can generally be related to structure. 1) Anesthesia and primary irritation are characteristic of most simple aliphatic esters. 2) Lacrimation, vesication, and lung irritation are due to the halogen atom in halogenated esters. 3) Cumulative organic damage to the nervous system or neuropathy can be caused by some, but not all, phosphate esters. 4) Most aliphatic and aromatic esters used as plasticizers are physiologically inert.

ACETATES

DESCRIPTION

Methyl acetate: CH_3COOCH_3.
Ethyl acetate: $CH_3COOC_2H_5$.
n-Propyl acetate: $CH_3COOC_3H_7$.
Isopropyl acetate: $CH_3COOCH(CH_3)_2$.
n-Butyl acetate: $CH_3COOC_4H_9$.
Amyl acetate: $CH_3COOC_5H_{11}$.

The acetates are colorless, volatile, flammable liquids.

SYNONYMS

Methyl acetate: none.
Ethyl acetate: acetic ether, vinegar naphtha.
n-Propyl acetate: acetic acid-propyl ester.
Isopropyl acetate: none.
n-Butyl acetate: butyl ethanoate, acetic acid butyl ester.
Amyl acetate: isoamyl acetate, pear oil, banana oil, amyl acetate ester, pentyl acetate.

POTENTIAL OCCUPATIONAL EXPOSURES

The acetates are a group of solvents for cellulose nitrate, cellulose acetate, ethyl cellulose, resins, rosin, cumar, elemi, phenolics, oils, fats, and celluloid. They are also used in the manufacture of lacquers, paints, varnishes, enamel, perfumes, dyes, dopes, plastic and synthetic finishes (e.g., artificial leather), smokeless powder, photographic film, footwear, pharmaceuticals, food preservatives, artificial glass, artificial silk, furniture polish, odorants, and other organic syntheses.

A partial list of occupations in which exposure may occur includes:

- Cellulose acetate makers
- Cumar makers
- Dope makers
- Dye makers
- Elemi makers
- Lacquer makers
- Nitrocellulose makers
- Paint makers
- Perfume makers
- Resin makers
- Rosin makers
- Varnish makers

PERMISSIBLE EXPOSURE LIMITS

The Federal standards are:

Methyl acetate	200 ppm	610 mg/m^3
Ethyl acetate	400 ppm	1,400 mg/m^3
n-Propyl acetate	200 ppm	840 mg/m^3
Isopropyl acetate	250 ppm	950 mg/m^3
n-Butyl acetate	150 ppm	710 mg/m^3
Isoamyl acetate	100 ppm	525 mg/m^3
n-Amyl acetate	100 ppm	525 mg/m^3
sec-Amyl acetate	125 ppm	650 mg/m^3
sec-Butyl acetate	200 ppm	950 mg/m^3
tert-Butyl acetate	200 ppm	950 mg/m^3

ROUTE OF ENTRY

Inhalation and ingestion.

HARMFUL EFFECTS

Local—

In higher concentrations, acetates are irritants to the mucous membranes. All irritate eyes and nasal passages in varying degrees. Prolonged exposure can cause irritation of the intact skin. These local effects are the primary risk in industry.

Systemic—

All acetates may cause headache, drowsiness, and unconsciousness if concentrations are high enough. Those effects are relatively slow and gradual in onset and slow in recovery after exposure.

MEDICAL SURVEILLANCE

Consider initial effects on skin and respiratory tract in any preplacement or periodical examinations, as well as liver and kidney function.

SPECIAL TESTS

None in common use.

PERSONAL PROTECTIVE METHODS

Barrier creams and protective clothing with gloves should be used, as well as fullface masks in areas of vapor concentration.

BIBLIOGRAPHY

Von Oettingen, W. F. 1960. The aliphatic acids and their esters: toxicity and potential dangers. The saturated monobasic acids and their esters: aliphatic acids with three to eighteen carbons and their esters. AMA Arch. Ind. Health 21:100.

ETHYL SILICATE

DESCRIPTION

$(CH_2H_5O)_4Si$, ethyl silicate, is a colorless, flammable liquid with a sharp odor detectable at 85 ppm.

SYNONYMS

Tetraethyl orthosilicate, tetraethoxy silane.

POTENTIAL OCCUPATIONAL EXPOSURES

Ethyl silicate is used in production of cases and molds for casting of metals and as a hardener for water and weather-resistant concrete.

A partial list of occupations in which exposure may occur includes:

- Acidproof cement makers
- Adhesive makers
- Brick preserver makers
- Building coaters
- Cement preserver makers
- Heat resistant paint makers
- Lacquer makers
- Metal casters
- Plaster preserver makers
- Silicate paint makers

PERMISSIBLE EXPOSURE LIMITS

There is no Federal standard; however, the ACGIH recommended TLV is 100 ppm (approximately 850 mg/m^3) determined as a time-weighted average. This TLV has not been confirmed in human exposure. At 3,000 ppm, ethyl silicate vapors are intolerable.

ROUTE OF ENTRY

Inhalation of vapor.

HARMFUL EFFECTS

Local—

Ethyl silicate is a primary irritant to the eyes and the nose.

Systemic—

Damage to the lungs, liver, and kidneys, and anemia have been observed in animal experiments but have not been reported for human exposure.

MEDICAL SURVEILLANCE

Placement or periodic examinations should include the skin, eyes, respiratory tract, as well as liver and kidney functions.

SPECIAL TESTS

None currently used.

PERSONAL PROTECTIVE METHODS

Fullface masks in areas of vapor concentration.

BIBLIOGRAPHY

Pozzini, U. C., and C. P. Carpenter. 1951. Response of rodents to repeated inhalation of vapors of tetraethyl orthosilicate. Arch. Ind. Hyg. Occup. Med. 4:465.

FORMATES

DESCRIPTION

Methyl formate: $HCOOCH_3$.
Ethyl formate: $HCOOC_2H_5$.
These are colorless, mobile, flammable liquids with agreeable odors.

SYNONYMS

Methyl formate: Methyl methanoate.
Ethyl formate: None.

POTENTIAL OCCUPATIONAL EXPOSURES

Formates are solvents for cellulose nitrate, oils, greases, fats, cellulose acetate, fatty acids, acetylcellulose, collodion, and celluloid. They are also used as larvicides, fumigants, flavoring agents in the production of lemonade, rum, arrack, and essences, and they are used in chemical synthesis.

A partial list of occupations in which exposure may occur includes:

- Cellulose acetate workers
- Flavoring makers
- Fumigant makers
- Fumigators
- Grain fumigators
- Nitrocellulose workers
- Organic chemical synthesizers
- Pesticide workers
- Tobacco fumigators

PERMISSIBLE EXPOSURE LIMITS

The Federal standards are:

Methyl formate	100 ppm	250 mg/m^3
Ethyl formate	100 ppm	300 mg/m^3

ROUTE OF ENTRY

Inhalation, ingestion, and skin absorption.

HARMFUL EFFECTS

Local—

Methyl formate is a mild irritant to mucous membranes, especially eyes and respiratory system. Ethyl formate may be irritating to skin and mucous membranes in high concentrations.

Systemic—

Methyl formate has an irritant and narcotic effect, and in high concentrations may cause drowsiness and unconsciousness. Systemic intoxication in industry is rare.

MEDICAL SURVEILLANCE

Consider eye and respiratory disease or symptoms in any placement or follow-up examinations.

SPECIAL TESTS

None in common use.

PERSONAL PROTECTIVE METHODS

Barrier creams should be used to protect the skin, and masks should be used in areas of vapor concentration.

BIBLIOGRAPHY

Von Oettingen, W. F. 1959. The aliphatic acids and their esters — toxicity and potential dangers. The saturated monobasic aliphatic acids and their esters. AMA Arch. Ind. Health 20:517.

CARBOXYLIC ACIDS AND ANHYDRIDES

The carboxylic acids and acid anhydrides have similar properties because of their acid characteristics. Carboxylic acids, and those compounds with the COOH moiety, may be aliphatic or aromatic and may have more than one carboxyl group. The acid anhydrides are derivatives of carboxylic acids.

These compounds have a primary irritant effect, the degree determined in part by acid dissociation and water solubility. Some may cause severe tissue damage similar to that seen with strong mineral acids. Sensitization may also occur, but is more common with the anhydrides than the acids.

ACETIC ACID

DESCRIPTION

CH_3 COOH, acetic acid, is a colorless liquid with a pungent vinegar-like odor. Glacial acetic acid contains 99% acid.

SYNONYMS

Ethanoic acid, ethylic acid, methane carboxylic acid, pyroligneous acid, vinegar acid.

POTENTIAL OCCUPATIONAL EXPOSURES

Acetic acid is widely used as a chemical feedstock for the production of vinyl plastics, acetic anhydride, acetone, acetanilide, acetyl chloride, ethyl alcohol, ketene, methyl ethyl ketone, acetate esters, and cellulose acetates. It is also used alone in the dye, rubber, pharmaceutical, food preserving, textile, and laundry industries. It is utilized, too, in the manufacture of Paris green, white lead, tint rinse, photographic chemicals, stain removers, insecticides, and plastics.

A partial list of occupations in which exposure may occur inludes:

- Acetate ester makers
- Acetate fiber makers
- Aspirin makers
- Dye makers
- Food preservers
- Insecticide makers
- Laundry workers
- Photographic chemical makers
- Plastic makers
- Resin makers
- Rubber makers
- Stain removers
- Textile printers
- Tint rinse makers
- White lead makers

PERMISSIBLE EXPOSURE LIMITS

The Federal standard is 10 ppm (25 mg/m^3).

ROUTE OF ENTRY

Inhalation of vapor.

HARMFUL EFFECTS

Local—

Acetic acid vapor may produce irritation of the eyes, nose, throat, and lungs. Inhalation of concentrated vapors may cause serious damage to the lining membranes of the nose, throat, and lungs. Contact with concentrated acetic acid may cause severe damage to the skin and severe eye damage, which may result in loss of sight. Repeated or prolonged exposure to acetic acid may cause darkening, irritation of the skin, erosion of the exposed front teeth, and chronic inflammation of the nose, throat, and bronchi.

Systemic—

Bronchopneumonia and pulmonary edema may develop following acute overexposure. Chronic exposure may result in pharyngitis and catarrhal bronchitis. Ingestion, though not likely to occur in industry, may result in penetration of the esophagus, bloody vomiting, diarrhea, shock, hemolysis, and hemoglobinuria which is followed by anuria.

MEDICAL SURVEILLANCE

Consideration should be given to the skin, eyes, teeth, and respiratory tract in placement or periodic examinations.

SPECIAL TESTS

None in common use.

PERSONAL PROTECTIVE METHODS

When working with glacial acetic acid, personal protective equipment, protective clothing, gloves, and goggles should be worn. Eye fountains and showers should be available in areas of potential exposure.

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Capellini, A., and E. Sartorelli. 1967. Epidodio di intossicizione collettiva da anidride acetica ed acido acetica. Med. Lav. 58:108.

Henson, E. V. 1959. Toxicology of the fatty acids. J. Occup. Med. 1:339.

Von Oettingen, W. F. 1960. The aliphatic acids and their esters: toxicity and potential dangers. The saturated monobasic acids and their esters: aliphatic acids with three to eighteen carbons and their esters. AMA Arch. Ind. Health 21:100.

ACETIC ANHYDRIDE

DESCRIPTION

$CH_3COOCOCH_3$, acetic anhydride, is a colorless, strongly refractive liquid which has a strongly irritating odor.

SYNONYMS

Acetic oxide, acetyl oxide, ethanoic anhydride.

POTENTIAL OCCUPATIONAL EXPOSURES

Acetic anhydride is used as an acetylating agent or as a solvent in the manufacture of cellulose acetate, acetanilide, synthetic fibers, plastics, explosives, resins, pharmaceuticals, perfumes, and flavorings; and it is used in the textile dyeing industry.

A partial list of occupations in which exposure may occur includes:

- Acetate fiber makers
- Acetic acid makers
- Aspirin makers
- Cellulose acetate fiber makers
- Drug makers
- Dye makers
- Explosive makers
- Flavoring makers
- Perfume makers
- Photographic film makers
- Plastic makers
- Resin makers
- Textile makers

PERMISSIBLE EXPOSURE LIMITS

The Federal standard is 5 ppm (20 mg/m^3).

ROUTE OF ENTRY

Inhalation of vapor.

HARMFUL EFFECTS

Local—

In high concentrations, vapor may cause conjunctivitis, photophobia, lacrimation, and severe irritation of the nose and throat. Liquid acetic anhydride does not cause a severe burning sensation when it comes in contact with the skin. If it is not removed, the skin may become white and wrinkled, and delayed severe burns may occur. Both liquid and vapor may cause conjunctival edema and corneal burns, which may develop into temporary or permanent interstitial keratitis with cor-

neal opacity due to progression of the infiltration. Contact and, occasionally, hypersensivitity dermatitis may develop.

Systemic—

Immediate complaints following concentrated vapor exposure include conjunctival and nasopharyngeal irritation, cough, and dyspnea. Necrotic areas of mucous membranes may be present following acute exposure.

MEDICAL SURVEILLANCE

Consideration should be given to the skin, eyes, and respiratory tract in any placement or periodic examinations.

SPECIAL TESTS

None currently used.

PERSONAL PROTECTIVE METHODS

Personal protective equipment (protective clothing, gloves, and goggles) should be used. Eye fountains and showers should be made available in areas where contact might occur.

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FORMIC ACID

DESCRIPTION

HCOOH, formic acid, is a colorless, flammable, fuming liquid, with a pungent odor.

SYNONYMS

Methanoic acid, formylic acid, hydrogen carboxylic acid.

POTENTIAL OCCUPATIONAL EXPOSURES

Formic acid is a strong reducing agent and is used as a decalcifier. It is used in dyeing color fast wool, electroplating, coagulating latex rubber, regenerating old rubber, and dehairing, plumping, and tanning leather. It is also used in the manufacture of acetic acid, airplane dope, allyl alcohol, cellulose formate, phenolic resins, and oxalate; and it is used in the laundry, textile, insecticide, refrigeration, and paper industries.

A partial list of occupations in which exposure may occur includes:

Airplane dope makers
Allyl alcohol makers
Dyers
Electroplaters
Insecticide makers
Lacquer makers
Laundry workers
Leather makers
Paper makers
Perfume makers
Rubber workers
Textile makers
Wine makers

PERMISSIBLE EXPOSURE LIMITS

The Federal standard is 5 ppm (9 mg/m^3).

ROUTE OF ENTRY

Inhalation of vapor and percutaneous absorption.

HARMFUL EFFECTS

Local—

The primary hazard of formic acid results from severe irritation of the skin, eyes, and mucous membranes. Lacrimation, increased nasal discharge, cough, throat discomfort, erythema, and blistering may occur depending upon solution concentrations.

Systemic—

These have not been reported from inhalation exposure and are unlikely due to its good warning properties.

Swallowing formic acid has caused a number of cases of severe poisoning and death. The symptoms found in this type of poisoning include salivation, vomiting, burning sensation in the mouth, bloody vomiting, diarrhea, and pain. In severe poisoning, shock may occur. Later, breathing difficulties may develop. Kidney damage may also be present.

MEDICAL SURVEILLANCE

Consideration should be given to possible irritant effects on the skin, eyes, and lungs in any placement or periodic examinations.

SPECIAL TESTS

None currently used.

PERSONAL PROTECTIVE METHODS

Workers should be supplied with protective clothing, gloves, and goggles. Respiratory protection will be needed in areas of high vapor exposure.

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OXALIC ACID

DESCRIPTION

HOOCCOOH. $2H_2O$, oxalic acid in solution, is a colorless liquid. Anhydrous oxalic acid is monoclinic in form and is produced by careful drying of the crystalline dihydrate.

SYNONYMS

Dicarboxylic acid, ethane-di-acid, ethanedioic acid.

POTENTIAL OCCUPATIONAL EXPOSURES

Oxalic acid is used as an analytic reagent and in the manufacture of dyes, inks, bleaches, paint removers, varnishes, wood and metal cleansers, dextrin, cream of tartar, celluloid, oxalates, tartaric acid, purified methyl alcohol, glycerol, and stable hydrogen cyanide. It is also used in the photographic, ceramic, metallurgic, rubber, leather, engraving, pharmaceutical, paper, and lithographic industries.

A partial list of occupations in which exposure may occur includes:

- Bleach makers
- Celluloid makers
- Ceramic makers
- Cream of tartar makers
- Dye makers
- Glycerine makers
- Ink makers
- Laundry workers
- Paper makers
- Rubber makers
- Tannery workers
- Textile dyers
- Printers
- Wood bleachers

PERMISSIBLE EXPOSURE LIMITS

The Federal standard is 1 mg/m^3.

ROUTE OF ENTRY

Inhalation of mist and, occasionally, dust.

HARMFUL EFFECTS

Local—

Liquid has a corrosive action on the skin, eyes, and mucous membranes, which may result in ulceration. Local prolonged contact with extremities may result in localized pain, cyanosis, and even gangrenous changes probably resulting from localized vascular damage.

Systemic—

Chronic exposure to mist or dust has been reported to cause chronic inflammation of the upper respiratory tract. Ingestion is of lesser importance occupationally. Symptoms appear rapidly and include shock, collapse, and convulsive seizures. Such cases may also have marked kidney damage with deposition of calcium oxalate in the lumen of the renal tubules.

MEDICAL SURVEILLANCE

Evaluate skin, respiratory tract, and renal functions in placement or periodic examinations.

SPECIAL TESTS

The presence of increased urinary oxalate crystals may be helpful in evaluating oral poisoning. Determination of blood calcium and oxalate levels may also be used for this purpose.

PERSONAL PROTECTIVE METHODS

Protective clothing and goggles should be worn when working in areas where direct contact is possible. Respiratory protection from mist or dust may be needed.

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PHTHALIC ANHYDRIDE

DESCRIPTION

$C_8H_4O_3$, phthalic anhydride, is moderately flammable, white, lustrous, solid, with needle-like crystals.

SYNONYMS

Phthalic acid anhydride, benzene-o-dicarboxylic acid anhydride, phthalandione.

POTENTIAL OCCUPATIONAL EXPOSURES

Phthalic anhydride is used in the manufacture of phthaleins, benzoic acid, alkyd and polyester resins, synthetic indigo, and phthalic acid, which is used as a plasticizer for vinyl resins. To a lesser extent, it is used in the production of alizarin dye, anthranilic acid, anthraquinone, diethyl phthalate, dimethyl phthalate, erythrosin, isophthalic acid, methyl aniline, phenolphthalein, phthalamide, sulfathalidine, and terephthalic acid. It has also found use in pesticides and herbicides, as well as perfumes.

A partial list of occupations in which exposure may occur includes:

- Alizarin dye makers
- Alkyd resin makers
- Automobile finish makers
- Cellulose acetate plasticizer makers
- Dacron fiber makers
- Erythrosin makers
- Insecticide makers
- Mylar plastic makers
- Organic chemical synthesizers
- Phthalein makers
- Resin makers
- Vat dye makers
- Vinyl plasticizer makers

PERMISSIBLE EXPOSURE LIMITS

The Federal standard is 2 ppm (12 mg/m^3).

ROUTE OF ENTRY

Inhalation of dust, fume, or vapor.

HARMFUL EFFECTS

Local—

Phthalic anhydride, in the form of a dust, fume, or vapor, is a potent irritant of the eyes, skin, and respiratory tract. The irritant effects are worse on moist surfaces. Conjunctivitis and skin erythema, burning, and contact dermatitis may occur. If the chemical is held in contact with the skin, as under clothes or shoes, skin burns may develop. Hypersensitivity may develop in some individuals. Inhalation of the dust or vapors may cause coughing, sneezing, and a bloody nasal discharge. Impurities, naphthoquinone, as well as maleic anhydride, may also contribute to eye, skin, and pulmonary irritation.

Systemic—

Repeated exposure may result in bronchitis, emphysema, allergic asthma, urticaria, and chronic eye irritation.

MEDICAL SURVEILLANCE

Emphasis should be given to a history of skin or pulmonary allergy, and preplacement and periodic examinations should evaluate the skin, eye, and lungs, as well as liver and kidney functions. The hydrolysis product, phthalic acid, is rapidly excreted in the urine, although this has not been used in biological monitoring. Diagnostic patch testing may be helpful in evaluating skin allergy.

SPECIAL TESTS

None in common use.

PERSONAL PROTECTIVE METHODS

Proper ventilation, rubber gloves, protective clothing, head coverings, and goggles are recommended when repeated or prolonged contact is possible. Respiratory protection may be needed in dusty areas or where fumes or vapors are present.

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ALDEHYDES AND KETONES

Aldehydes and ketones are aliphatic or aromatic organic compounds which contain the carbonyl group, C=O.

The aldehydes, R−CH=O, are used primarily as chemical feedstock because of their relatively high reactivity. They are volatile, colorless liquids, with the exception of formaldehyde, which is a gas, and can exhibit additional hazard due to its flammability. Typically, these compounds are strongly irritating to the skin, eyes, and respiratory tract. Acute exposure may result in pulmonary injuries such as edema, bronchitis, and bronchopneumonia. Skin and pulmonary sensitization may develop in some individuals and result in contact dermatitis and, more rarely, asthmatic attacks. After hypersensitivity develops, individuals may develop symptoms due to other aldehydes. For this reason, medical surveillance and industrial hygiene practices are of importance.

Ketones are characterized by the structure R-O-R. They are similar in their chemical and toxicological properties, and all are flammable, colorless liquids with a pungent odor similar to acetone. They are used as industrial solvents and raw materials or as intermediates in chemical synthesis. Prolonged exposure is usually precluded by the intense irritation of the eyes and respiratory tract.

ACETALDEHYDE

DESCRIPTION

CH_3CHO, acetaldehyde, is a flammable, volatile, colorless liquid with a characteristic odor. It is produced by oxidation of alcohol with a metallic catalyst, by hydration of acetylene, or, usually, by direct oxidation of ethylene.

SYNONYMS

Acetic aldehyde, aldehyde, ethanol, ethyl aldehyde.

POTENTIAL OCCUPATIONAL EXPOSURES

Acetaldehyde can be reduced or oxidized to form acetic acid, acetic anhydride, acrolein, aldol, buranol, chloral, 2-methyl-5-ethyl pyridine, paraldehyde, and pentaerythritol. It is also used in the manufacture of disinfectants, drugs, dyes, explosives, flavorings, lacquers, mirrors (silvering), perfume, photographic chemicals, phenolic and urea resins, rubber accelerators and antioxidants, varnishes, vinegar, and yeast.

A partial list of occupations in which exposure may occur includes:

- Acetic acid makers
- Antioxidant makers
- Disinfectant makers
- Explosives workers
- Flavoring makers
- Mirror silverers
- Paraldehyde makers
- Urea resin makers
- Rubber makers
- Vinegar makers
- Yeast makers

PERMISSIBLE EXPOSURE LIMITS

The Federal standard is 200 ppm (360 mg/m^3); however, the ACGIH 1976 recommended TLV is 100 ppm (180 mg/m^3).

ROUTE OF ENTRY

Inhalation of vapor.

HARMFUL EFFECTS

Local—

The liquid and the fairly low levels of the vapor are irritating to the eyes, skin, upper respiratory passages, and bronchi. Repeated exposure may result in dermatitis, rarely, and skin sensitization.

Systemic—

Acute involuntary exposure to high levels of acetaldehyde vapors may result in pulmonary edema, preceded by excitement, followed by narcosis. It has been postulated that these symptoms may have been similar to those of alcohol, which is converted to acetaldehyde and acetic acid. Chronic effects have not been documented, and seem unlikely, since voluntary inhalation of toxicologically significant levels of acetaldehyde are precluded by its irritant properties at levels as low as 200 ppm (360 mg/m^3 of air).

MEDICAL SURVEILLANCE

Consideration should be given to skin, eyes, and respiratory tract in any preplacement or periodic examinations.

SPECIAL TESTS

None appear needed, but effects of exposure can be determined from blood results by gas chromatographic methods, if desired.

PERSONAL PROTECTIVE METHODS

Protective clothing, gloves, goggles, and respiratory protective equipment where high concentrations of the gas or vapor are expected.

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James, T. N., and E. S. Bear. 1967. Effects of ethanol and acetaldehyde on the heart. Am. Heart J. 74:243.

ACROLEIN

DESCRIPTION

$H_2C=CHCHO$, acrolein, is a clear, yellowish liquid which is a petroleum byproduct. It is commercially produced by the oxidation of propylene.

SYNONYMS

Acraldehyde, acrylic aldehyde, allyl aldehyde, propenal.

POTENTIAL OCCUPATIONAL EXPOSURES

Acrolein is the feedstock for several types of plastics, plasticizers, acrylates, textile finishes, synthetic fibers, and methionines. It is also used in the manufacture of colloidal forms of metals, in perfumes, and, due to its pungent odor, as a warning agent in methyl chloride refrigerants. Other potential exposures may arise when acrolein vapor is given off when oils and fats containing glycerol are heated.

A partial list of occupations in which exposure may occur includes:

- Acrylate makers
- Fat processors
- Methionine makers
- Perfume makers
- Plastic makers
- Refrigerant makers
- Renderers
- Rubber makers
- Textile resin makers

PERMISSIBLE EXPOSURE LIMITS

The Federal standard for exposure to acrolein is 0.1 ppm (0.25 mg/m^3).

ROUTE OF ENTRY

Inhalation of vapor and percutaneous absorption.

HARMFUL EFFECTS

Local—

In the liquid or pungent vapor form, acrolein produces intense irritation to the eye and mucous membranes of the respiratory tract. Skin burns and dermatitis may result from prolonged or repeated exposure. Sensitization in a few individuals may also occur.

Systemic—

Because of acrolein's pungent, offensive odor and the intense irritation of the conjunctiva and upper respiratory tract, severe toxic effects from acute exposure are rare, as workmen will not tolerate the vapor even in minimal concentrations. Acute exposure to acrolein may cause bronchial inflammation, resulting in bronchitis or pulmonary edema.

MEDICAL SURVEILLANCE

Preplacement and periodic medical examinations should consider respiratory, skin, and eye disease. Pulmonary function tests may be helpful.

SPECIAL TESTS

None in common use.

PERSONAL PROTECTIVE METHODS

Protection in handling and transporting is advocated. Suitable ventilation and protective clothing should be provided for employees working in areas of possible exposure. Protective respiratory equipment in areas of vapor concentration.

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Gusev, M. I., A. I. Schechnikova, I. S. Dronov, M. D. Grebenskova, and A. I. Golovina. 1966. Determination of the daily average maximum permissible concentration of acrolein in the atmosphere. Hyg. Sanit. 31(1):8.

FORMALDEHYDE

DESCRIPTION

HCHO, formaldehyde, is a colorless, pungent gas. It is produced commercially by the catalytic oxidation of methyl alcohol and sold in aqueous solution containing 30-50% formaldehyde and from 0-15% methanol, which is added to prevent polymerization. Formaldehyde solution is called formalin, formol, or morbicid.

SYNONYMS

Oxomethane, oxymethylene, methylene oxide, formic aldehyde, methyl aldehyde.

POTENTIAL OCCUPATIONAL EXPOSURES

Formaldehyde has found wide industrial usage as a fungicide, germicide, and in disinfectants and embalming fluids. It is also used in the manufacture of artificial silk and textiles, latex, phenol, urea, thiourea and melamine resins, dyes, and inks, cellulose esters and other organic molecules, mirrors, and explosives. It is also used in the paper, photographic, and furniture industries.

A partial list of occupations in which exposure may occur includes:

- Anatomists
- Biologists
- Deodorant makers
- Disinfectant makers
- Embalming fluid makers
- Formaldehyde resin makers
- Hide preservers
- Ink makers
- Latex makers
- Photographic film makers
- Textile printers
- Wood preservers

PERMISSIBLE EXPOSURE LIMITS

The Federal standard is 3 ppm determined as a TWA. The acceptable ceiling concentration is 5 ppm with an acceptable maximum peak above this value of 10 ppm for a maximum duration of 30 minutes. ACGIH in 1975 recommended a TLV of 2 ppm (3 mg/m^3) as a ceiling value. NIOSH has recommended a ceiling of 1 ppm (1.2 mg/m^3) for any 30-minute sampling period.

ROUTE OF ENTRY

Inhalation of gas.

HARMFUL EFFECTS

Local—

Formaldehyde gas may cause severe irritation to the mucous membranes of the respiratory tract and eyes. The aqueous solution splashed in the eyes may cause eye burns. Urticaria has been reported following inhalation of gas. Repeated exposure to formaldehyde may cause dermatitis either from irritation or allergy.

Systemic—

Systemic intoxication is unlikely to occur since intense irritation of upper respiratory passages compels workers to leave areas of exposure. If workers do inhale high concentrations of formaldehyde, coughing, difficulty in breathing, and pulmonary edema may occur. Ingestion, though usually not occurring in industrial experience, may cause severe irritation of the mouth, throat, and stomach.

MEDICAL SURVEILLANCE

Consider the skin, eyes, and respiratory tract in any preplacement or periodic examination, especially if the patient has a history of allergies.

SPECIAL TESTS

None in common use.

PERSONAL PROTECTIVE METHODS

Prevention of intoxication may be easily accomplished by supplying adequate ventilation and protective clothing. Barrier creams may also be helpful. In areas of high vapor concentrations, full protective face masks with air supply is needed, as well as protective clothing.

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FURFURAL

DESCRIPTION

$C_5H_4O_2$, furfural, is an aromatic heterocyclic aldehyde with an amber color and aromatic odor. This liquid is obtained from cereal straws and brans containing pentosans by hydrolysis and dehydration with sulfuric acid.

SYNONYMS

Furfurol (a misnomer), furfuraldahyde, artificial ant oil, pyromucic aldehyde, furol, 2-furaldehyde.

POTENTIAL OCCUPATIONAL EXPOSURES

Furfural is used as a solvent for wood resin, nitrated cotton, cellulose acetate, and gums. It is used in the production of phenolic plastics, thermosetting resins, refined petroleum oils, dyes, and varnishes. It is also utilized in the manufacture of pyromucic acid, vulcanized rubber, insecticides, fungicides, herbicides, germicides, furan derivatives, polymers, and other organic chemicals.

A partial list of occupations in which exposure may occur includes:

- Adipic acid makers
- Butadiene refiners
- Cellulose acetate makers
- Disinfectant workers
- Dye makers
- Herbicide makers
- Lysine makers
- Metal refiners
- Nylon makers
- Organic chemical synthesizers
- Pyromucic acid makers
- Road builders

PERMISSIBLE EXPOSURE LIMITS

The Federal standard is 5 ppm (20 mg/m^3).

ROUTES OF ENTRY

Inhalation of vapor, percutaneous absorption.

HARMFUL EFFECTS

Local—

Liquid and concentrated vapor are irritating to the eyes, skin, and mucous membranes of the upper respiratory tract. Eczematous dermatitis as well as skin sensitization, resulting in allergic contact dermatitis and photosensitivity, may develop following repeated exposure.

Systemic—

Workers chronically exposed to the vapor have had complaints of headache, fatigue, itching of the throat, lacrimation, loss of the sense of taste, numbness of the tongue, and tremor. Occupational overexposure is relatively rare due to the liquid's low vapor pressure, and symptoms usually disappear rapidly after removal from exposure.

MEDICAL SURVEILLANCE

Consider skin irritation and skin allergies (especially to aldehydes) in preplacement or periodic examinations. Also consider possible respiratory irritant effects.

SPECIAL TESTS

None commonly used.

PERSONAL PROTECTIVE METHODS

Protective clothing and adequate ventilation should be provided in areas where toxic exposure may occur. In areas of vapor concentrations, fullface masks may be required.

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KETONES

DESCRIPTION

The ketone family includes:

Acetone: CH_3COCH_3
Diacetone: $(CH_3)_2COHCH_2COCH_3$
Methyl ethyl ketone: $CH_3COCH_2CH_3$
Methyl n-propyl ketone: $CH_3CH_2CH_2COCH_3$
Methyl n-butyl ketone: $CH_3COCH_2CH_2CH_2CH_3$
Methyl isobutyl ketone: $(CH_3)_2CHCH_2COCH_3$

SYNONYMS

Acetone: 2-Propanone, dimethyl ketone, beta-ketopropane, pyroacetic ether.

Diacetone: Diacetone alcohol, diacetonyl alcohol, dimethylacetonyl

carbinol, 4-hydroxy-4 methyl-2-pentanone, 2-methyl-2-pentanol-4-one.

Methyl ethyl ketone: Butanone, 2-butanone, MEK, ethyl methyl ketone,

Methyl n-propyl ketone: Ethyl acetone, 2-pentanone, MPK.

Methyl n-butyl ketone: n-Butyl methyl ketone, propyl acetone, 2-hexanone, MBK.

Methyl isobutyl ketone: Hexone, isopropylacetone, 4-methyl-2-pentanone, MIBK.

POTENTIAL OCCUPATIONAL EXPOSURES

This group of ketone solvents has many uses in common. They are low-cost solvents for resins, lacquers, oils, fats, collodion, cotton, cellulose acetate, nitrocellulose, cellulose esters, epoxy resins, gums, pigments, dyes, vinyl polymers, and copolymers. They are used as chemical intermediates, in the manufacture of smokeless powder and explosives, and in the paint, lacquer, varnish, plastics, dyeing, celluloid, photographic, cement, rubber, artificial silk and leather, synthetic rubber, and lubricating oil industries. They are also used in hydraulic fluids, metal cleaning compounds, quick drying inks, airplane dopes, compositions for paper and textiles, pharmaceuticals, cosmetics, and as paint removers and dewaxers.

A partial list of occupations in which exposure may occur includes:

- Adhesive makers
- Celluloid makers
- Dope workers
- Dye makers
- Explosive makers
- Garage mechanics
- Lacquer and oil processors
- Shoe makers
- Solvent workers
- Varnish and stain makers
- Wax makers

PERMISSIBLE EXPOSURE LIMITS

The Federal standards are:

Acetone	1,000 ppm	2,400 mg/m^3
Diacetone	50 ppm	240 mg/m^3
Methyl ethyl ketone	200 ppm	590 mg/m^3
Methyl n-propyl ketone	200 ppm	700 mg/m^3
Methyl n-butyl ketone	100 ppm	410 mg/m^3
Methyl isobutyl ketone	100 ppm	410 mg/m^3

ROUTES OF ENTRY

Inhalation of vapor, percutaneous absorption.

HARMFUL EFFECTS

Local—

These solvents may produce a dry, scaly, and fissured dermatitis after repeated exposure. High vapor concentrations may irritate the conjunctiva and mucous membranes of the nose and throat, producing eye and throat symptoms.

Systemic—

In high concentrations, narcosis is produced, with symptoms of headache, nausea, light headedness, vomiting, dizziness, incoordination, and unconsciousness.

Recent reports indicate that exposure of workers to methyl n-butyl ketone has been associated with the development of peripheral neuropathy. Rat experiments have also shown nerve changes characteristic of peripheral neuropathy after exposure to 1,300 ppm.

MEDICAL SURVEILLANCE

Preplacement examinations should evaluate skin and respiratory conditions. In the case of methyl n-butyl ketone, special attention should be given to the central and peripheral nervous systems.

SPECIAL TESTS

Acetone can be determined in the blood, urine, and expired air, and has been used as an index of exposure. A neurotoxic metabolite of methyl n-butyl ketone has been found in the rat. This is not present with methyl isobutyl ketone.

PERSONAL PROTECTIVE METHODS

Contact with skin and eyes should be avoided by the use of protective clothing. In areas of high vapor concentration, masks should be used.

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ALIPHATIC HALOGENATED HYDROCARBONS

Halogenated hydrocarbons typically are colorless, volatile liquids with excellent organic solvent properties. Chemically, they consist of saturated or unsaturated carbon chains in which one hydrogen atom or more have been replaced by one or more halogens (fluorine, chlorine, bromine, or iodine). Hydrocarbons having only one or two halogens are usually flammable and less toxic than similar hydrocarbons with complete halogen substitution. Hydrocarbons containing fluorine tend to be less toxic, while hydrocarbons containing bromine or iodine are generally more toxic than hydrocarbons containing chlorine. The latter, chlorinated hydrocarbons, are widely used because of their low cost.

Halogenated hydrocarbons have found wide use as solvents in degreasing, dewaxing, drycleaning, and extracting processes. Other uses are as aerosol propellants, fumigants, insecticides, refrigerants, and

chemical intermediates for drugs, plastics, and synthetic rubber. Some of these compounds have been used medically as anesthetics and anthelmintics. The toxicologic effects of halogenated hydrocarbons vary from one compound to another but, generally, most cause central nervous system depression. Also common to most is the defatting of the skin which may lead to dermatitis. Upon inhalation of high concentrations of vapor, liver or kidney injury may occur, but it should be noted that while some compounds may have no effect, others may affect only one of these two organs, and still others may affect both. Pulmonary irritation and damage to the hematopoietic system may also occur after exposure to certain compounds.

Medical surveillance of workers exposed to halogenated hydrocarbons should include periodic examinations, including urinalysis to check for renal damage and appropriate tests for liver dysfunction. Evidence of acute exposure to chlorinated compounds may be obtained in some cases by analysis of expired air obtained soon after exposure. If acute poisoning is suspected, epinephrine should not be used in treatment as it has been noted that epinephrine injected during narcosis caused by exposure to certain halogenated solvents may lead to cardiac arrhythmia.

CARBON TETRACHLORIDE

DESCRIPTION

CCl_4, carbon tetrachloride, is a colorless, nonflammable liquid with a characteristic odor. Oxidative decomposition by flame causes phosgene and hydrogen chloride to form.

SYNONYMS

Tetrachloromethane, perchloromethane.

POTENTIAL OCCUPATIONAL EXPOSURES

Carbon tetrachloride is used as a solvent for oils, fats, lacquers, varnishes, rubber, waxes, and resins. Fluorocarbons are chemically synthesized from it. It is also used as an azeotropic drying agent for spark plugs, a dry cleaning agent, a fire extinguishing agent, a fumigant, and an anthelmintic agent. The use of this solvent is widespread, and substitution of less toxic solvents when technically possible is recommended.

A partial list of occupations in which exposure may occur includes:

- Chemists
- Degreasers
- Fat processors
- Firemen
- Fluorocarbon makers
- Grain fumigators
- Ink makers
- Insecticide makers
- Lacquer makers
- Metal cleaners
- Propellant makers
- Refrigerant makers
- Rubber makers
- Solvent workers
- Wax makers

PERMISSIBLE EXPOSURE LIMITS

The Federal standard is 10 ppm (65 mg/m^3) as an 8-hour TWA

with an acceptable ceiling concentration of 25 ppm; acceptable maximum peaks above the ceiling of 200 ppm are allowed for one 5-minute duration in any 4-hour period. NIOSH has recommended a ceiling limit of 2 ppm based on a 1-hour sampling period at a rate of 750 ml/min.

ROUTES OF ENTRY

Inhalation of vapor. Percutaneous absorption has been demonstrated in animals.

HARMFUL EFFECTS

Local—

Carbon tetrachloride solvent removes the natural lipid cover of the skin. Repeated contact may lead to a dry, scaly, fissured dermatitis. Eye contact is slightly irritating, but this condition is transient.

Systemic—

Excessive exposure may result in central nervous system depression, and gastrointestinal symptoms may also occur. Following acute exposure, signs and symptoms of liver and kidney damage may develop. Nausea, vomiting, abdominal pain, diarrhea, enlarged and tender liver, and jaundice result from toxic hepatitis. Diminished urinary volume, red and white blood cells in the urine, albuminuria, coma, and death may be consequences of acute renal failure. The hazard of systemic effects is increased when carbon tetrachloride is used in conjunction with ingested alcohol.

MEDICAL SURVEILLANCE

Preplacement and periodic examinations should include an evaluation of alcohol intake and appropriate tests for liver and kidney functions. Special attention should be given to the central and peripheral nervous system, the skin, and blood.

SPECIAL TESTS

Expired air and blood levels may be useful as indicators of exposure.

PERSONAL PROTECTIVE METHODS

Barrier creams, gloves, protective clothing, and masks should be used as appropriate where exposure occurs.

BIBLIOGRAPHY

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Luse, S. A., and W. G. Wood. 1967. The brain in fatal carbon tetrachloride poisoning. Arch. Neurol. 17:304.

Nielson, V. K., and J. Larsen. 1965. Acute renal failure due to carbon tetrachloride poisoning. Acta. Med. Scand. p. 178.

CHLOROFORM

DESCRIPTION

$CHCl_3$, chloroform, is a clear, colorless liquid with a characteristic odor. Though nonflammable, chloroform decomposes to form hydrochloric acid, phosgene, and chlorine upon contact with a flame.

SYNONYMS

Trichloromethane, methenyl chloride.

POTENTIAL OCCUPATIONAL EXPOSURES

Chloroform was one of the earliest general anesthetics, but its use for this purpose has been abandoned because of toxic effects. Chloroform is widely used as a solvent (especially in the lacquer industry); in the extraction and purification of penicillin and other pharmaceuticals; in the manufacture of artificial silk, plastics, floor polishes, and fluorocarbons; and in sterilization of catgut.

A partial list of occupations in which exposure may occur includes:

- Chemists
- Drug makers
- Fluorocarbon makers
- Lacquer workers
- Polish makers
- Silk synthesizers
- Solvent workers

PERMISSIBLE EXPOSURE LIMITS

The Federal standard is 50 ppm (240 mg/m^3). The ACGIH recommended 1976 TLV is 25 ppm. NIOSH's recommended limit is a ceiling of 2 ppm based on a 1-hour sample collected at 750 l/min.

ROUTE OF ENTRY

Inhalation of vapors.

HARMFUL EFFECTS

Local—

Chloroform may produce burns if left in contact with the skin.

Systemic—

Chloroform is a relatively potent anesthetic at high concentrations. Death from its use as an anesthetic has resulted from liver damage and from cardiac arrest. Exposure may cause lassitude, digestive disturbance, dizziness, mental dullness, and coma. Chronic overexposure has been shown to cause enlargement of the liver and kidney damage. Alcoholics seem to be affected sooner and more severely from chloroform exposure. Disturbance of the liver is more characteristic of exposure than central nervous system depression or renal injury. There is some animal experimental evidence that suggests chloroform may be a carcinogen.

MEDICAL SURVEILLANCE

Preplacement and periodic examinations should include appropriate

tests for liver and kidney functions, and special attention should be given to the nervous system, the skin, and to the history of alcoholism.

SPECIAL TESTS

Expired air and blood levels may be useful in estimating levels of acute exposure.

PERSONAL PROTECTIVE METHODS

Protective clothing and gloves should be worn to protect the skin, and masks should be worn in areas of high vapor concentration.

BIBLIOGRAPHY

Challen, P. J. R., D. E. Hickish, and J. R. Bedford. 1958. Chronic chloroform intoxication. Br. J. Ind. Med. 15:243.

CHLOROPRENE

DESCRIPTION

$H_2C=CC_1-CH=CH_2$, chloroprene, is a colorless, flammable liquid posessing a pungent odor.

SYNONYMS

2-chloro-1,3-butadiene.

POTENTIAL OCCUPATIONAL EXPOSURES

The only major use of chloroprene is in the production of artificial rubber (neoprene, duprene).

A partial list of occupations in which exposure may occur includes:

Duprene makers
Neoprene makers
Rubber makers

PERMISSIBLE EXPOSURE LIMITS

The Federal standard is 25 ppm (90 mg/m^3). Because of suspected carcinogenic and/or mutagenic potential, the limit may be lowered.

ROUTES OF ENTRY

Inhalation of vapor and skin absorption.

HARMFUL EFFECTS

Local—

Chloroprene acts as a primary irritant on contact with skin, conjunctiva, and mucous membranes and may result in dermatitis, conjunctivitis, and circumscribed necrosis of the cornea. Temporary hair loss has been reported during the manufacture of polymers.

Systemic—

Inhalation of high concentrations may result in anesthesia and respiratory paralysis. Chronic exposure may produce damage to the lungs,

nervous system, liver, kidneys, spleen, and myocardium. Russian studies suggest that chloroprene exposure is associated with an increased incidence of cancer of the skin and lungs, but this has not been confirmed. Fetal effects have been noted in rodents.

MEDICAL SURVEILLANCE

Preplacement and periodic examinations should include an evaluation of the skin, eyes, respiratory tract, and central nervous system. Liver and kidney function should be evaluated.

SPECIAL TESTS

None commonly used.

PERSONAL PROTECTIVE METHODS

Protective clothing, chemical safety goggles, air-supplied or self-contained respirators, and safety harnesses should be worn where there is exposure to the liquid or high concentrations of the vapor.

BIBLIOGRAPHY

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1,2-DIBROMOETHANE

DESCRIPTION

$BrCH_2CH_2Br$, 1,2-dibromoethane, is a colorless nonflammable liquid with a chloroformlike odor.

SYNONYMS

Ethylene dibromide, ethylene bromide, sym-dibromoethane.

POTENTIAL OCCUPATIONAL EXPOSURES

1,2-Dibromoethane is used principally as a fumigant for ground pest control and as a constituent of ethyl gasoline. It is also used in fire extinguishers, gauge fluids, and waterproofing preparations; and it is used as a solvent for celluloid, fats, oils, and waxes.

A partial list of occupations in which exposure may occur includes:

- Antiknock compound makers
- Cabbage growers
- Drug makers
- Farmers
- Fat processors
- Fire extinguisher makers
- Fumigant workers
- Gum processors
- Lead scavenger makers
- Resin makers
- Termite controllers
- Tetraethyl lead makers
- Wool reclaimers

PERMISSIBLE EXPOSURE LIMITS

The Federal standard is 20 ppm (145 mg/m^3) as an 8-hour TWA with an acceptable ceiling concentration of 300 ppm; acceptable maximum peaks above the ceiling of 50 ppm are allowed for 5 minutes duration.

ROUTES OF ENTRY

Inhalation of the vapor and absorption through the skin.

HARMFUL EFFECTS

Local—

Prolonged contact of the liquid with the skin may cause erythema, blistering, and skin ulcers. These reactions may be delayed 24-48 hours. Dermal sensitization to the liquid may develop. The vapor is irritating to the eyes and to the mucous membranes of the respiratory tract.

Systemic—

Inhalation of the vapor may result in severe acute respiratory injury, central nervous system depression, and severe vomiting. Animal experiments have produced injury to the liver and kidneys.

MEDICAL SURVEILLANCE

Preemployment and periodic examinations should evaluate the skin and eyes, respiratory tract, and liver and kidney functions.

SPECIAL TESTS

None commonly used.

PERSONAL PROTECTIVE METHODS

1,2-Dibromoethane can penetrate most types of rubber and leather protective clothing, and, therefore, protective clothing made from other materials should be provided. Masks must be worn in areas with excessive vapor concentrations.

BIBLIOGRAPHY

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Rowe, V. K., H. D. Spencer, D. D. McCollister, R. L. Hollingsworth, and E. M. Adams. 1952. Toxicity of ethylene dibromide determined on experimental animals. AMA Arch. Indust. Health p. 158.

1,2-DICHLOROETHANE

DESCRIPTION

$Cl_1CH_2CH_2Cl$, 1,2-dichloroethane, is a colorless, flammable liquid which has a pleasant odor, sweetish taste.

SYNONYMS

Ethylene dichloride, sym-dichloroethane, ethylene chloride, glycol dichloride, β-dichloroethane.

POTENTIAL OCCUPATIONAL EXPOSURES

In recent years, 1,2-dichloroethane has found wide use in the manufacture of ethyl glycol, diaminoethylene, chloroecholine chloride, polyvinyl chloride, nylon, viscose rayon, styrene-butadiene rubber, and various plastics. It is a solvent for resins, asphalt, bitumen, rubber, cellulose acetate, cellulose ester, and paint; a degreaser in the engineering, textile, and petroleum industries; and an extracting agent for soya bean oil and caffeine. It is also used as an antiknock agent in gasoline, a pickling agent, a fumigant, and a drycleaning agent. It has found use in photography, xerography, water softening, and in the production of adhesives, cosmetics, pharmaceuticals, and varnishes.

A partial list of occupations in which exposure may occur includes:

- Adhesive makers
- Bakelite processors
- Camphor workers
- Drycleaners
- Exterminators
- Gasoline blenders
- Insecticide makers
- Metal degreasers
- Ore upgraders
- Solvent workers
- Textile cleaners
- Vinyl chloride makers

PERMISSIBLE EXPOSURE LIMITS

The Federal standard is 50 ppm (200 mg/m^3) as an 8-hour TWA with an acceptable ceiling concentration of 100 ppm. Acceptable maximum peaks above the ceiling of 200 ppm are allowed for 5 minutes duration in any 3-hour period.

ROUTES OF ENTRY

Inhalation of vapor and skin absorption of liquid.

HARMFUL EFFECTS

Local—

Repeated contact with liquid can produce a dry, scaly, fissured dermatitis. Liquid and vapor may also cause eye damage.

Systemic—

Inhalation of high concentrations may cause nausea, vomiting, mental confusion, dizziness, and pulmonary edema. Chronic exposure has been associated with liver and kidney damage.

MEDICAL SURVEILLANCE

Preplacement and periodic examinations should include an evaluation of the skin and liver and kidney functions.

SPECIAL TESTS

None commonly used; can be determined in expired air.

PERSONAL PROTECTIVE METHODS

Protective clothing, goggles, and gloves are to be worn. In high vapor concentrations,use fullface masks and supplied air respirators.

BIBLIOGRAPHY

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Yodiken, R. E., and J. R. Babcock, Jr., 1973. 1,2-Dichloroethane poisoning. Arch. Environ. Health 26:281.

1,2-DICHLOROETHYLENE

DESCRIPTION

ClCH=CHCl, 1,2-dichloroethylene, exists in two isomers, cis 60% and trans 40%. There are variations in toxicity between these two forms. At room temperature, it is a liquid with a slight acrid, ethereal odor. Gradual decomposition results in hydrochloric acid formation in the presence of ultraviolet light or upon contact wth hot metal.

SYNONYMS

Acetylene dichloride, sym-dichloroethylene, 1,2-dichloroethene.

POTENTIAL OCCUPATIONAL EXPOSURES

1,2-Dichloroethylene is used as a solvent for waxes, resins, and acetyl cellulose. It is also used in the extraction of rubber, as a refrigerant, in the manufacture of pharmaceuticals and artificial pearls, and in the extraction of oils and fats from fish and meat.

A partial list of occupations in which exposure may occur includes:

Carbolic acid processors
Drug makers
Drycleaners
Solvent workers
Wax makers

PERMISSIBLE EXPOSURE LIMITS

The Federal standard is 200 ppm (790 mg/m^3).

ROUTE OF ENTRY

Inhalation of the vapor.

HARMFUL EFFECTS

Local—

This liquid can act as a primary irritant producing dermatitis and irritation of mucous membranes.

Systemic—

1,2-Dichloroethylene acts principally as a narcotic, causing central nervous system depression. Symptoms of acute exposure include dizzi-

ness, nausea and frequent vomiting, and central nervous system intoxication similar to that caused by alcohol. Renal effects, when they do occur, are transient.

MEDICAL SURVEILLANCE

Consider possible irritant effects on skin or respiratory tract as well as liver and renal function in preplacement or periodic examinations.

SPECIAL TESTS

None commonly used; expired air analyses may be useful in detecting exposure.

PERSONAL PROTECTIVE METHODS

Barrier creams and gloves are needed to protect the skin. In high vapor concentrations, masks and protective clothing are required.

BIBLIOGRAPHY

McBirney, R. S. 1954. Trichloroethylene and dichloroethylene poisoning. AMA Arch. Ind. Hyg. Occup. Med. 10:130.

ETHYL CHLORIDE

DESCRIPTION

CH_3CH_2Cl, ethyl chloride, is a flammable gas with an ethereal odor, burning taste. It is flammable, and the products of combustion include phosgene and hydrogen chloride.

SYNONYMS

Monochloroethane, hydrochloric ether, chloroethane, chlorethyl.

POTENTIAL OCCUPATIONAL EXPOSURES

Ethyl chloride is used as an ethylating agent in the manufacture of tetraethyl lead, dyes, drugs, and ethyl cellulose. It can be used as a refrigerant and as a local anesthetic (freezing).

A partial list of occupations in which exposure may occur includes:

- Anesthetists
- Dentists
- Drug makers
- Ethylation workers
- Fat and oil processors
- Nurses
- Perfume makers
- Phosphorus and sulfur processors
- Physicians
- Refrigeration workers
- Resin makers
- Sulfur processors
- Tetraethyl lead makers
- Wax makers

PERMISSIBLE EXPOSURE LIMITS

The Federal standard is 1000 ppm (2600 mg/m^3).

ROUTES OF ENTRY

Inhalation of gas and slight percutaneous absorption.

HARMFUL EFFECTS

Local—

The liquid form of ethyl chloride is mildly irritating to skin and eyes. Frostbite can occur due to rapid liquid evaporation.

Systemic—

Ethyl chloride exposure may produce headache, dizziness, incoordination, stomach cramps, and eventual loss of consciousness. In high concentrations, it is a respiratory tract irritant, and death due to cardiac arrest has been recorded. Renal damage has been reported in animals. Effects from chronic exposure have not been reported.

MEDICAL SURVEILLANCE

Consider possible acute cardiac effects in any preplacement or periodic examination.

SPECIAL TESTS

None commonly used. Ethyl chloride is excreted in expired air.

PERSONAL PROTECTIVE METHODS

In high vapor concentrations, use mask with full face protection, gloves, and protective clothing.

BIBLIOGRAPHY

Von Oettingen, W. F. 1955. The Halogenated Aliphatic, Olefinic, Cyclic, Aromatic, and Aliphatic-aromatic Hydrocarbons Including the Halogenated Insecticides, Their Toxicity and Potential Dangers. Public Health Service Publication No. 414. U. S. Public Health Service.

FLUOROCARBONS

DESCRIPTION

Chemically, fluorocarbons are hydrocarbons containing fluorine and include compounds that may contain other halogens in addition to fluorine. Generally, these compounds are colorless, nonflammable gases, though a few are liquids at room temperature. Decomposition of chlorine-containing fluoromethanes caused by contact with an open flame or hot metal produces hydrogen chloride, hydrogen fluoride, phosgene, carbon dioxide, and chlorine.

SYNONYMS

See listing of economically important fluorocarbons under "Permissible Exposure Limits." Also, Freon® is a trademark for a number of fluorocarbons used particularly in refrigeration products and equipment.

POTENTIAL OCCUPATIONAL EXPOSURES

The fluorocarbons are used primarily as refrigerants and polymer intermediates. They are also used as aerosol propellants, anesthetics, fire extinguishers, foam blowing agents, in drycleaning, and in degreasing of electronic equipment. The fluorocarbons have found wide use due to their relatively low toxicity.

A partial list of occupations in which exposure may occur includes:

- Aerosol bomb workers
- Ceramic mold makers
- Drycleaners
- Drug makers
- Fire extinguisher workers
- Heat transfer workers
- Metal conditioners
- Plastic makers
- Pressurized food makers
- Refrigeration makers
- Rocket fuel makers
- Solvent workers

PERMISSIBLE EXPOSURE LIMITS

Federal standards for selected fluorocarbons of economic importance are listed as follows:

Compound	Federal Standards	
	ppm	mg/m³
Bromotrifluoromethane Fluorocarbon 13B1	1,000	6,100
Dibromodifluoromethane Fluorocarbon 12B2	100	860
Dichlorodifluoromethane Fluorocarbon 12	1,000	4,950
Dichloromonofluoromethane Fluorocarbon 21	1,000	4,200
Dichlorotetrafluoroethane Fluorocarbon 114	1,000	7,000
Fluorotrichloromethane Fluorocarbon 11	1,000	5,600
1,1,1,2-Tetrachloro-2,2-difluoroethane Fluorocarbon 112	500	4,170
1,1,2,2-Tetrachloro-1,2-difluoroethane Fluorocarbon 112	500	4,170
1,1,2-Trichloro-1,2,2-trifluoroethane Fluorocarbon 113	1,000	7,600
Bromochlorotrifluoroethane Fluorocarbon 123B1	No Standard	
Chlorodifluoromethane Fluorocarbon 22	No Standard (ACGIH TLV of 1,000 ppm)	
Chloropentafluoroethane Fluorocarbon 115	No Standard	
Chlorotrifluoroethylene Fluorocarbon 1113	No Standard	
Chlorotrifluoromethane Fluorocarbon 13	No Standard	
Difluoroethylene Fluorocarbon 1131	No Standard	
Fluoroethylene Fluorocarbon 1141	No Standard	
Hexafluoropropylene Fluorocarbon 1216	No Standard	
Octafluorocyclobutane Fluorocarbon C-318	No Standard	
Tetrafluoroethylene Fluorocarbon 1114	No Standard	

ROUTE OF ENTRY

Inhalation of vapor or gas.

HARMFUL EFFECTS

Local—

These compounds may produce mild irritation to the upper respiratory tract. Dermatitis occurs only rarely. Decomposition products may also be the cause of these effects.

Systemic—

Mild central nervous system depression may occur in cases of exposure to very high concentrations of fluorocarbons. Symptoms from acute exposure may manifest themselves in occasional tremor and incoordination. It has been reported that dizziness has resulted from an exposure to 5% dichlorodifluoromethane and unconsciousness from exposure to 15%. Cardiac arrhythmias, with sudden death, have occurred from breathing some of these chemicals. Typically, fluorocarbons have very low levels of toxicity, and their predominant hazard is from simple asphyxia.

Fluoroalkenes are more toxic than fluoroalkanes. Liver and kidney damage has been reported to occur from chronic exposure to fluoroalkenes, whereas no chronic effects have been reported from fluoroalkanes.

MEDICAL SURVEILLANCE

Though these compounds are of a low level of toxicity, they should not be considered inert. There are no specific diagnostic tests for the toxic effects occurring at very high concentrations. Preplacement and periodic examinations should consider possible cardiac effects from acute exposure.

SPECIAL TESTS

None commonly used. Compounds are usually excreted rapidly in expired air.

PERSONAL PROTECTIVE METHODS

Simple ventilation can avert acute poisoning. Masks are rarely needed.

BIBLIOGRAPHY

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METHYL AND ETHYL BROMIDE

DESCRIPTION

CH_3Br, methyl bromide, is a colorless, nearly odorless gas. It is synthesized from sodium bromide, methyl alcohol, and sulfuric acid.

C_2H_5Br, ethyl bromide, is a colorless, volatile, flammable liquid possessing an etherlike odor, burning taste. It becomes yellowish on exposure to air. It is produced from potassium bromide, ethyl alcohol, and sulfuric acid.

SYNONYMS

Methyl bromide: bromomethane.

Ethyl bromide: bromoethane, monobromoethane, monobromomethane.

POTENTIAL OCCUPATIONAL EXPOSURES

Methyl Bromide: The primary use of methyl bromide is as an insect fumigant for soil, grain, warehouses, mills, ships, etc. It is also used as a chemical intermediate and a methylating agent, a refrigerant, a herbicide, a fire extinguishing agent, a low-boiling solvent in aniline dye manufacture; for degreasing wool; for extracting oils from nuts, seeds, and flowers; and in ionization chambers.

Ethyl bromide: This chemical is used as an ethylating agent in organic synthesis and gasoline, as a refrigerant, and as an extraction solvent. It has limited use as a local anesthetic.

A partial list of occupations in which exposure may occur includes:

Anesthetists	Grain fumigators
Color makers	Refrigerant makers
Dye makers	Soil fumigators
Fire extinguisher workers	Solvent workers
Fruit fumigators	Wool degreasers

PERMISSIBLE EXPOSURE LIMITS

The Federal standard for methyl bromide is 20 ppm (80 mg/m^3) as a ceiling value. The Federal standard for ethyl bromide is 200 ppm (890 mg/m^3) as a TWA. The ACGIH recommended TLV for methyl bromide is 15 ppm (60 mg/m^3) as a TWA.

ROUTES OF ENTRY

Inhalation and percutaneous absorption.

HARMFUL EFFECTS

Local—

Methyl bromide is irritating to the eyes, skin, and mucous membranes of the upper respiratory tract. In cases of moderate skin exposure, there may be an itching dermatitis, and in severe cases, vesicles and second-degree burns. Methyl bromide may be absorbed by leather, resulting in prolonged skin contact. Repeated or prolonged skin contact with ethyl bromide may cause irritation.

Systemic—

High concentrations of either methyl or ethyl bromide may cause lung irritation which may result in pulmonary edema and death. Acute exposure to methyl bromide may produce delayed effects. Onset of symptoms is usually delayed from 30 minutes to 6 hours; the first to appear are malaise, visual disturbances, headaches, nausea, vomiting, somnolence, vertigo, and tremor in the hands. The tremor may become more severe and widespread, developing into epileptiform type convul-

sions followed by coma and death due to pulmonary or circulatory failure or both. A period of delirium and mania may precede convulsions, but convulsions have been reported without any other warning symptoms. Kidney damage may occur; permanent brain damage may result.

In chronic poisoning, the effects of methyl bromide are usually limited to the central nervous system: Lethargy; muscular pains; visual, speech, and sensory disturbances; and mental confusion being the most prominent complaints. Ethyl bromide exposure has not been associated with chronic effects other than skin irritation.

MEDICAL SURVEILLANCE

Methyl bromide: Evaluate the central nervous system, respiratory tract, and skin in preplacement and periodic examinations.

Ethyl bromide: No specific considerations needed.

SPECIAL TESTS

None in common use. Blood bromide levels have been measured in cases of methyl bromide intoxication, but their value in routine monitoring of exposure has not been established.

PERSONAL PROTECTIVE METHODS

Rubber, not leather, protective clothing should be utilized. When masks are worn, they should provide full face protection.

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METHYL CHLORIDE

DESCRIPTION

CH_3Cl, methyl chloride, is a colorless gas possessing a faint, sweet odor.

SYNONYMS

Monochloromethane, chloromethane.

POTENTIAL OCCUPATIONAL EXPOSURES

Methyl chloride is used as a methylating and chlorinating agent in organic chemistry. In petroleum refineries it is used as an extractant for greases, oils, and resins. Methyl chloride is also used as a solvent in the synthetic rubber industry, as a refrigerant, and as a propellant in polystyrene foam production. In the past it has been used as a local anesthetic (freezing).

A partial list of occupations in which exposure may occur includes:

Aerosol packagers	Methyl cellulose makers
Drug makers	Polystyrene foam makers
Flavor extractors	Refrigeration workers
Low temperature solvent workers	Rubber makers
Methylation workers	Vapor pressure thermometer makers

PERMISSIBLE EXPOSURE LIMITS

The Federal standard is 100 ppm (210 mg/m^3) as an 8-hour TWA, with an acceptable ceiling concentration of 200 ppm; acceptable maximum peaks above the ceiling of 300 ppm are allowed for 5 minutes duration in a 3-hour period.

ROUTE OF ENTRY

Percutaneous absorption.

HARMFUL EFFECTS

Local—

Skin contact with the discharge from the pressurized gas may cause frostbite. The liquid may damage eyes.

Systemic—

Signs and symptoms of chronic exposure include staggering gait, difficulty in speech, nausea, headache, dizziness, and blurred vision. Vomiting has also occurred in some cases. These effects may be observed following a latency period of several hours.

Acute exposure is much like chronic except that the latency period is shorter and the effects more severe. Coma or convulsive seizures may occur. Acute poisoning predominantly depresses the central nervous system, but renal and hepatic damage may also occur. Recently noted in these cases is the depression of bone marrow activity. Recovery from severe exposure may take as long as 2 weeks.

MEDICAL SURVEILLANCE

Preplacement and periodic examinations should give careful consideration to a previous history of the central nervous system, and to renal or hepatic disorders.

SPECIAL TESTS

None in common use.

PERSONAL PROTECTIVE METHODS

Masks should be used in areas of high vapor concentrations.

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Scharnweber, H. C., G. N. Spears, and S. R. Cowles. 1974. Chronic methyl chloride intoxication in six industrial workers. J. Occup. Med. 16:112.

METHYLENE CHLORIDE

DESCRIPTION

$CHCl_2$, methylene chloride, is a nonflammable, colorless liquid with a pleasant aromatic odor noticeable at 300 ppm (this, however, should not be relied upon as an adequate warning of unsafe concentrations).

SYNONYMS

Dichloromethane, methylene dichloride, methylene bichloride.

POTENTIAL OCCUPATIONAL EXPOSURES

Methylene chloride is used mainly as a low temperature extractant of substances which are adversely affected by high temperature. It can be used as a solvent for oil, fats, waxes, bitumen, cellulose acetate, and esters. It is also used as a paint remover and as a degreaser.

A partial list of occupations in which exposure may occur includes:

- Aerosol packagers
- Anesthetic makers
- Bitumen makers
- Degreasers
- Fat extractors
- Flavoring makers
- Leather finish workers
- Oil processors
- Paint remover makers
- Resin makers
- Solvent workers
- Stain removers

PERMISSIBLE EXPOSURE LIMITS

The Federal standard is 500 ppm (1,740 mg/m^3) as an 8-hour TWA with an acceptable ceiling concentration of 1,000 ppm; acceptable maximum peaks above the ceiling of 2,000 ppm are allowed for 5 minutes duration in a 2-hour period.

Note: The 1976 ACGIH TLV is 200 ppm (720 mg/m^3.)

NIOSH has recommended a time-weighted average limit of 75 ppm in the absence of occupational exposure to carbon monoxide greater than 9 ppm (TWA). For carbon monoxide exposures greater than 9 ppm, a formula is recommended. NIOSH also recommended a ceiling limit of 500 ppm as determined by a 15-minute sampling period.

ROUTES OF ENTRY

Inhalation of vapors and percutaneous absorption of liqiud.

HARMFUL EFFECTS

Local—

Repeated contact with methylene chloride may cause a dry, scaly, and fissured dermatitis. The liquid and vapor are irritating to the eyes and upper respiratory tract at higher concentrations. If the liquid is held in contact with the skin, it may cause skin burns.

Systemic—

Methylene chloride is a mild narcotic. Effects from intoxication include headache, giddiness, stupor, irritability, numbness, and tingling

in the limbs. Irritation to the eyes and upper respiratory passages occurs at higher dosages. In severe cases, observers have noted toxic encephalopathy with hallucinations, pulmonary edema, coma, and death. Cardiac arrythmias have been produced in animals but have not been common in human experiences. Exposure to this agent may cause elevated carboxyhemoglobin levels which may be significant in smokers, or workers with anemia or heart disease, and those exposed to CO.

MEDICAL SURVEILLANCE

Changes in liver, respiratory tract, and central nervous system should be considered during preplacement or periodic medical examinations. Smoking history should be known; anemias or cardiovascular disease may increase the hazard.

SPECIAL TESTS

The metabolism and excretion of methylene chloride has been thoroughly studied. Blood and expired air analyses are useful indicators of exposure. Carboxyhemoglobin levels may be useful indicators of excessive exposure, especially in nonsmokers.

PERSONAL PROTECTIVE METHODS

Protective clothing, gloves to prevent skin contact, and, in areas of high concentration, fullface masks.

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PROPYLENE DICHLORIDE

DESCRIPTION

$CH_3CHClCH_2Cl$, propylene dichloride, is a colorless liquid with a characteristic unpleasant odor.

SYNONYMS

1,2-Dichloropropane, propylene chloride.

POTENTIAL OCCUPATIONAL EXPOSURES

Propylene dichloride is widely used for degreasing and drycleaning; it is also used as a soil fumigant and in the manufacture of cellulose

plastics, rubber, waxes, scouring compounds and other manufacture of organic synthetics.

A partial list of occupations in which exposure may occur includes:

- Cellulose plastic makers
- Drycleaners
- Fat processors
- Fumigant workers
- Gum processors
- Metal degreasers
- Oil processors
- Organic chemical synthesizers
- Rubber makers
- Scouring compound makers
- Solvent workers
- Stain removers
- Wax makers

PERMISSIBLE EXPOSURE LIMITS

The Federal standard is 75 ppm (350 mg/m^3).

ROUTE OF ENTRY

Inhalation of vapor.

HARMFUL EFFECTS

Local—

Propylene dichloride may cause dermatitis by defatting the skin. More severe irritation may occur if it is confined against the skin by clothing. Undiluted, it is moderately irritating to the eyes, but does not cause permanent injury.

Systemic—

In animal experiments, acute exposure to propylene dichloride produced central nervous system narcosis, fatty degeneration of the liver and kidneys.

MEDICAL SURVEILLANCE

Evaluate the skin and liver and renal function on a periodic basis, as well as cardiac and respiratory status and general health.

SPECIAL TESTS

None in common use. Propylene dichloride can be determined in expired air.

PERSONAL PROTECTIVE METHODS

Barrier creams or gloves and protective clothing. Masks in areas of vapor concentration.

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TETRACHLOROETHANE

DESCRIPTION

$CHCl_2CHCl_2$, tetrachloroethane, is a heavy, volatile liquid which is nonflammable and has a sweetish, chloroform-like odor. Oxidative

decomposition of tetrachloroethane by ultraviolet radiation or by contact with hot metal results in the formation of small quantities of phosgene, hydrochloric acid, carbon monoxide, carbon dioxide, or dichloroacetyl chloride.

SYNONYMS

1,1,2,2-Tetrachloroethane, sym-tetrachloroethane, acetylene tetrachloride, ethanetetrachloride.

POTENTIAL OCCUPATIONAL EXPOSURES

Tetrachloroethane is used as a drycleaning agent, as a fumigant, in cement, and in lacquers. It is used in the manufacture of tetrachloroethylene, artificial silk, artificial leather, and artificial pearls. Recently, its use as a solvent has declined due to replacement by less toxic compounds. It is also used in the estimation of water content in tobacco and many drugs, and as a solvent for chromium chloride impregnation of furs.

A partial list of occupations in which exposure may occur includes:

- Biologists
- Drycleaners
- Fat processors
- Fumigators
- Gasket makers
- Herbicide workers
- Insecticide workers
- Lacquer workers
- Metal cleaners
- Mineralogists
- Oil processors
- Paint makers
- Phosphorus processors
- Resin makers
- Soil treaters
- Solvent workers
- Varnish workers
- Waxers

PERMISSIBLE EXPOSURE LIMITS

The Federal standard is 5 ppm (35 mg/m^3). NIOSH has recommended a time-weighted average limit of 1 ppm.

ROUTES OF ENTRY

Inhalation of vapor and absorption of liquid through the skin. There is some evidence that tetrachloroethane absorbed through the skin affects the central nervous system only.

HARMFUL EFFECTS

Local—

Repeated or prolonged contact with this chemical can produce a scaly and fissured dermatitis.

Systemic—

Early effects brought on by tetrachloroethane narcotic action include tremors, headache, a prickling sensation and numbness of limbs, loss of kneejerk, and excessive sweating. Paralysis of the interossei muscles of the hands and feet and disappearance of ocular and pharyngeal reflexes have also occurred due to peripheral neuritis which may de-

velop later. Blood changes include increases in mononuclear leukocytes, progressive anemia, and a slight thrombocytosis.

Clinical symptoms following these changes are fatigue, headache, constipation, insomnia, irritability, anorexia, and nausea. Later on, liver dysfunction may result in complaints of general malaise, drowsiness, loss of appetite, nausea, an unpleasant taste in the mouth, and abdominal discomfort. This may be followed by jaundice, mental confusion, stupor or delirium, hematemesis, convulsions, and purpuric rashes.

Pulmonary edema ascribed to capillary injury has been noted in severe cases, along with renal damage, though it is not known to what extent this contributes to the total toxic picture. Nephritis may develop, and the urine may contain albumin and casts. Fatty degeneration of the myocardium has been reported only in animal experiments.

MEDICAL SURVEILLANCE

Preplacement and periodic examination should be comprehensive because of the possible involvement of many systems. Special attention should be given to liver, kidney, and bone marrow function, as well as to the central and peripheral nervous systems. Alcoholism may be a predisposing factor.

SPECIAL TESTS

None commonly used. Blood or breath analyses may be useful.

PERSONAL PROTECTIVE METHODS

Gloves and protective clothing should be worn, and appropriate respirators or masks should be used in areas of elevated vapor concentration.

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TETRACHLOROETHYLENE

DESCRIPTION

$Cl_2C=CCl_2$, tetrachloroethylene, is a clear, colorless, nonflammable liquid with a characteristic odor. The odor is noticeable at 50 ppm, though after a short period it may become inconspicuous, thereby becoming an unreliable warning signal.

SYNONYMS

Perchloroethylene, carbon dichloride, ethylene tetrachloride.

POTENTIAL OCCUPATIONAL EXPOSURES

Tetrachloroethylene is a widely used solvent with particular use as a drycleaning agent, a degreaser, a chemical intermediate, a fumigant, and medically as an anthelmintic.

A partial list of occupations in which exposure may occur includes:

Cellulose ester processors	Metal degreasers
Degreasers	Printers
Dope processors	Rubber workers
Drug makers (anthelmintics)	Soap workers
Drycleaners	Solvent workers
Electroplaters	Tar processors
Ether processors	Vacuum tube makers
Fumigant workers	Wax makers
Gum processors	Wool scourers

PERMISSIBLE EXPOSURE LIMITS

The Federal standard is 100 ppm (670 mg/m^3), as an 8-hour TWA with an acceptable ceiling concentration of 200 ppm; acceptable maximum peaks above the ceiling of 300 ppm are allowed for 5 minutes duration in a 3-hour period. NIOSH has recommended a time-weighted average limit of 50 ppm and a ceiling limit of 100 ppm determined by 15-minute samples, twice daily.

ROUTES OF ENTRY

Inhalation of vapor and percutaneous absorption of liquid.

HARMFUL EFFECTS

Local—

Repeated contact may cause a dry, scaly, and fissured dermatitis. High concentrations may produce eye and nose irritation.

Systemic—

Acute exposure to tetrachloroethylene may cause central nervous system depression, hepatic injury, and anesthetic death. Cardiac arrhythmias and renal injury have been produced in animal experiments. Signs and symptoms of overexposure include malaise, dizziness, headache, increased perspiration, fatigue, staggering gait, and slowing of mental ability. These usually subside quickly upon removal into the open air.

MEDICAL SURVEILLANCE

Evaluate skin, and liver and kidney function, as well as central nervous system. Alcoholism may be a predisposing factor.

SPECIAL TESTS

Breath analyses may be helpful in evaluating exposures.

Workers with preemployment histories of liver, kidney, or nervous disorders should be advised as to possible increased risk.

PERSONAL PROTECTIVE METHODS

Skin protection in the form of barrier creams, gloves, and protective clothing should be used. In areas of vapor concentration, fullface masks should be worn.

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1,1,1-TRICHLOROETHANE

DESCRIPTION

CH_3CCl_8, 1,1,1-trichloroethane, is a colorless, nonflammable liquid with an odor similar to chloroform. Upon contact with hot metal or exposure to ultraviolet radiation, it will decompose to form the irritant gases hydrochloric acid, phosgene, and dichloroacetylene.

SYNONYMS

Methyl chloroform.

POTENTIAL OCCUPATIONAL EXPOSURES

In recent years, 1,1,1-trichloroethane has found wide use as a substitute for carbon tetrachloride. In liquid form it is used as a degreaser and for cold cleaning, dip-cleaning, and bucket cleaning of metals. Other industrial applications of 1,1,1-trichloroethane's solvent properties include its use as a drycleaning agent, a vapor degreasing agent, and a propellant.

A partial list of occupations in which exposure may occur includes:

Degreasers	Metal degreasers
Drycleaners	Propellant makers
Machinery cleaners	Stain removers

PERMISSIBLE EXPOSURE LIMITS

The Federal standard is 350 ppm (1,900 mg/m^3). NIOSH has recommended a 350 ppm ceiling as determined by a 15-minute sampling period.

ROUTES OF ENTRY

Inhalation of vapor and moderate skin absorption.

HARMFUL EFFECTS

Local—

Liquid and vapor are irritating to eyes on contact. This effect is usually noted first in acute exposure cases. Mild conjunctivitis may develop but recovery is usually rapid. Repeated skin contact may produce a dry, scaly, and fissured dermatitis, due to the solvent's defatting properties.

Systemic—

1,1,1-trichloroethane acts as a narcotic and depresses the central nervous system. Acute exposure symptoms include dizziness, incoordination, drowsiness, increased reaction time, unconsciousness, and death.

MEDICAL SURVEILLANCE

Consider the skin, liver function, cardiac status, especially arrythmias, in preplacement or periodic examinations.

SPECIAL TESTS

Expired air analyses may be useful in monitoring exposure.

PERSONAL PROTECTIVE METHODS

1,1,1-Trichloroethane attacks natural rubber; therefore, protective clothing of leather, polyvinyl alcohol, or neoprene is recommended. In areas of high concentrations, fullface masks should be worn.

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1,1,2-TRICHLOROETHANE

DESCRIPTION

$CH_2ClCHCl_2$, 1,1,2-trichloroethane, is a colorless, nonflammable liquid. It is an isomer of 1,1,1-trichloroethane but should not be confused with it toxicologically. 1,1,2-Trichloroethane is comparable to carbon tetrachloride and tetrachloroethane in toxicity.

SYNONYMS

Vinyl trichloride.

POTENTIAL OCCUPATIONAL EXPOSURES

1,1,2-Trichloroethane is used as a chemical intermediate and as a solvent, but is not as widely used as its isomer 1,1,1-trichloroethane.

A partial list of occupations in which exposure may occur includes:

Organic chemical synthesizers
Solvent makers

PERMISSIBLE EXPOSURE LIMITS

The Federal standard is 10 ppm (45 mg/m^3) as a TWA.

ROUTES OF ENTRY

Inhalation of vapor and absorption through the skin.

HARMFUL EFFECTS

Local—

Irritation to eyes and nose, and infection of the conjunctiva have been shown in animals.

Systemic—

Little is known of the toxicity of 1,1,2-trichloroethane since no human toxic effects have been reported. Animal experiments show 1,1,2-trichloroethane to be a potent central nervous system depressant. The injection of anesthetic doses in animals was associated with both liver and renal neurosis.

MEDICAL SURVEILLANCE

Consider the skin, central nervous system, and liver and kidney function. Alcoholism may be a synergistic factor.

SPECIAL TESTS

None commonly used, but expired air analyses may be useful in monitoring exposure.

PERSONAL PROTECTIVE METHODS

Protective clothing and gloves should be worn. Respirators should be used in areas of high vapor concentration.

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TRICHLOROETHYLENE

DESCRIPTION

$ClCH=CCl_2$, trichloroethylene, a colorless, nonflammable, noncorrosive liquid has the "sweet" odor characteristic of some chlorinated hydrocarbons. Decomposition of trichloroethylene, due to contact with hot metal or ultraviolet radiation, forms products including chlorine gas, hydrogen chloride, and phosgene. Dichloroacetylene may be formed from the reaction of alkali with trichloroethylene.

SYNONYMS

Ethylene trichloride, ethinyl trichloride, trichloroethene.

POTENTIAL OCCUPATIONAL EXPOSURES

Trichloroethylene is primarily used as a solvent in vapor degreasing. It is also used for extracting caffeine from coffee, as a drycleaning agent,

and as a chemical intermediate in the production of pesticides, waxes, gums, resins, tars, paints, varnishes, and specific chemicals such as chloroacetic acid.

A partial list of occupations in which exposure may occur includes:

Anesthetic makers	Metal cleaners
Caffeine processors	Oil processors
Cleaners	Perfume makers
Disinfectant makers	Printers
Degreasers	Resin workers
Drug makers	Rubber cementers
Drycleaners	Shoe makers
Dye makers	Soap makers
Electronic equipment cleaners	Solvent workers
Fat processors	Textile cleaners
Glass cleaners	Tobacco denicotinizers
Mechanics	Varnish workers

PERMISSIBLE EXPOSURE LIMITS

The Federal standard is 100 ppm (535 mg/m^3) as an 8-hour TWA with an acceptable ceiling concentration of 200 ppm; acceptable maximum peaks above the ceiling of 300 ppm are allowed for 5 minutes duration in a 2-hour period. The NIOSH Criteria for a Recommended Standard recommends limits of 100 ppm as a TWA and a peak of 150 ppm determined by a sampling time of 10 minutes.

ROUTES OF ENTRY

Inhalation and percutaneous absorption.

HARMFUL EFFECTS

Local—

Exposure to trichloroethylene vapor may cause irritation of the eyes, nose, and throat. The liquid, if splashed in the eyes, may cause burning irritation and damage. Repeated or prolonged skin contact with the liquid may cause dermatitis.

Systemic—

Acute exposure to trichloroethylene depresses the central nervous system exhibiting such symptoms as headache, dizziness, vertigo, tremors, nausea and vomiting, irregular heart beat, sleepiness, fatigue, blurred vision, and intoxication similar to that of alcohol. Unconsciousness and death have been reported. Alcohol may make the symptoms of trichloroethylene overexposure worse. If alcohol has been consumed, the overexposed worker may become flushed. Trichloroethylene addiction and peripheral neuropathy have been reported. Recent reports indicate that exposure to trichloroethylene may induce liver tumors in mice.

MEDICAL SURVEILLANCE

Preplacement and periodic examinations should include the skin,

respiratory, cardiac, central, and peripheral nervous systems, as well as liver and kidney function. Alcohol intake should be evaluated.

SPECIAL TESTS

Expired air analysis and urinary metabolites have been used to monitor exposure.

PERSONAL PROTECTIVE METHODS

Gloves and protective clothing should be worn, and fullface mask should be used in areas of excessive vapor concentrations.

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VINYL CHLORIDE

DESCRIPTION

$CH_2=CHCl$, vinyl chloride, is a flammable gas at room temperature and is usually encountered as a cooled liquid. The colorless liquid forms a vapor which has a pleasant ethereal odor.

SYNONYMS

Chloroethylene, chloroethene, monochloroethylene.

POTENTIAL OCCUPATIONAL EXPOSURES

Vinyl chloride is used as a vinyl monomer in the manufacture of polyvinyl chloride and other resins. It is also used as a chemical intermediate and as a solvent.

A partial list of occupations in which exposure may occur includes:

Polyvinyl resin makers
Organic chemical synthesizers
Rubber makers

PERMISSIBLE EXPOSURE LIMITS

The Federal standard for exposure to vinyl chloride sets a limit of 1 ppm over an 8-hour period, and a ceiling of 5 ppm averaged over any period not exceeding 15 minutes.

ROUTES OF ENTRY

Vinyl chloride gas is absorbed by inhalation. Skin absorption has been suggested but experimental evidence is presently lacking.

HARMFUL EFFECTS

Local—

Vinyl chloride is a skin irritant, and contact with the liquid may cause frostbite upon evaporation. The eyes may be immediately and severely irritated.

Systemic—

Vinyl chloride depresses the central nervous system causing symptoms which resemble mild alcohol intoxication. Lightheadedness, some nausea, and dulling of visual and auditory responses may develop in acute exposures. Death from severe vinyl chloride exposure has been reported.

Chronic exposure of workers involved in reactor vessel entry and hand cleaning may result in the triad of acro-osteolysis, Raynaud's phenomenon, and sclerodermatus skin changes. Chronic exposure may also cause hepatic damage.

Vinyl chloride is regarded as a human carcinogen, and a causal agent of angiosarcoma of the liver. Excess cancer of the lung and the lymphatic and nervous systems has also been reported. Experimental evidence of tumor induction in a variety of organs, including liver, lung, brain, and kidney, as well as nonmalignant alterations, such as fibrosis and connective tissue deterioration, indicate the multisystem oncogenic and toxicologic effects of vinyl chloride.

MEDICAL SURVEILLANCE

Preplacement and periodic examinations should emphasize liver function and palpation. Liver scans and grey-scale ultrasonography have been useful in detecting liver tumors. Medical histories should include alcoholic intake; past hepatitis; exposure to hepatotoxic agents, drugs and chemicals; past blood transfusions; past hospitalizations. Radiographic examinations of the hands may be helpful if acroosteolysis is suspected. Long term followup of exposed persons is essential as in the case of other carcinogens.

SPECIAL TESTS

None in common use. Metabolism is being studied.

PERSONAL PROTECTIVE METHODS

Where vinyl chloride levels cannot meet the standard, workers should be required to wear respiratory protection, either air supplied respirator or, if the level does not exceed 25 ppm, a chemical cartridge or cannister type gas mask. In hazard areas, proper protective clothing to prevent skin contact with the vinyl chloride or polyvinyl chloride residue should be worn.

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ALIPHATIC AMINES

The aliphatic amines are derivatives of ammonia (NH_3) in which one or more hydrogen atoms are replaced by alkyl or alkanol radicals. They tend to have a characteristic fishlike ammonia odor in the free base form.

These compounds are generally prepared by alkylation of ammonia or hydrogenation of the appropriate nitrite. They are widely used in industry, particularly as chemical intermediates.

The amines are basic compounds and may form strongly alkaline solutions which can be highly irritating and cause damage on contact with eyes and skin. Skin absorption may be significant as many are capable of cutaneous sensitization. Some members of this series may have physiologic or pharmacologic effects—e.g., histamine liberation and vasodilation, but, in general, local effects predominate in industrial exposures.

Because of the strong irritant properties of aliphatic amines, eyes, skin, and respiratory tract should be protected from exposure to them.

N-BUTYLAMINE

DESCRIPTION

CH_3-CH_2-CH_2-CH_2-NH_2, n-butylamine, is a flammable colorless liquid with an ammoniacal odor.

SYNONYMS

1-aminobutane.

POTENTIAL OCCUPATIONAL EXPOSURES

n-Butylamine is used in pharmaceuticals, dyestuffs, rubber, chemicals, emulsifying agents, photography, desizing agents for textiles, pesticides, and synthetic agents.

A partial list of occupations in which exposure may occur includes:

- Butylaminophenol makers
- Chemists
- Drug makers
- Dye makers
- Emulsifier makers
- Insecticide makers
- Petroleum dewaxers
- Rubber makers
- Tanning chemical makers

PERMISSIBLE EXPOSURE LIMITS

The Federal standard is 5 ppm (15 mg/m^3) as a ceiling value.

ROUTES OF ENTRY

Inhalation and percutaneous absorption.

HARMFUL EFFECTS

Local—

Butylamine vapor is irritating to the nose, throat, and eyes. Contact with the liquid may produce severe eye damage and skin burns.

Systemic—

Inhalation of concentrations at or above the threshold limit may produce mild headaches and flushing of the skin and face.

Butylamine vapor has produced pulmonary edema in animal experiments.

MEDICAL SURVEILLANCE

Evaluate risks of eye or skin injury and respiratory irritation in periodic or placement examinations.

SPECIAL TESTS

None have been developed.

PERSONAL PROTECTIVE METHODS

Protective clothing and goggles should be worn where possibility of skin or eye contact with the liquid exists. In areas of elevated vapor concentration, fullface masks with organic vapor canister or supplied air respirators and protective clothing should be worn. The odor and irritation of the mucous membranes cannot be relied upon for exposure control.

ETHANOLAMINES

DESCRIPTION

Monoethanolamine: $H_2NCH_2CH_2OH$, Diethanolamine: $HN(CH_2CH_2OH)$, Triethanolamine: $N(CH_2CH_2,(OH)_3$. All three compounds are water soluble liquids. Monoethanolamine has a low vapor pressure while the vapor pressure of the other ethanolamines is very low. Monoethanolamine and diethanolamine have ammonia odors while triethanolamine has only a faint non-ammonia odor. The acid salts have less odor and are of low volatility. Ethanolamines can be detected by odor as low as 2-3 ppm.

SYNONYMS

Monoethanolamine: Ethanolamine, 2-aminoethanol, colamine
Diethanolamine: 2,2'-Iminodiethanol
Triethanolamine: 2,2',2"-Nitrilotriethanol

POTENTIAL OCCUPATIONAL EXPOSURES

Monoethanolamine is widely used in industry to remove carbon dioxide and hydrogen from natural gas, to remove hydrogen sulfide and carbonyl sulfide, as an alkaline conditioning agent, and as an intermediate for soaps, detergents, dyes, and textile agents.

Diethanolamine is an absorbent for gases, a solubilizer for 2,4-dichlorophenoxyacetic acid (2,4-D), and a softener and emulsifier intermediate for detergents. It also finds use in the dye and textile industry.

Triethanolamine is used as a plasticizer, neutralizer for alkaline dispersions, lubricant additive, corrosion inhibitor, and in the manufacture of soaps, detergents, shampoos, shaving preparations, face and hand creams, cements, cutting oils, insecticides, surface active agents, waxes, polishes, and herbicides.

A partial list of occupations in which exposure may occur includes:

- Cement makers
- Detergent makers
- Dye makers
- Emulsifier makers
- Herbicide makers
- Insecticide makers
- Natural gas workers
- Plastic workers
- Polish makers
- Soap makers
- Surfactant makers
- Textile workers
- 2,4-D makers

PERMISSIBLE EXPOSURE LIMITS

The Federal standard for monoethanolamine is 3 ppm (6 mg/m^3). There are no standards for the other compounds.

ROUTES OF ENTRY

Inhalation of vapor and percutaneous absorption.

HARMFUL EFFECTS

Local—

Ethanolamine has had wide use in industry, yet reports of injury

in man are lacking. Ethanolamine in animal experiments was highly irritating to the skin, eyes, and respiratory tract. Diethanolamine and triethanolamine produced much less irritation. In human experiments, ethanolamine produced only redness of the skin.

Systemic—

No specific published data on human exposure are available. Animal experiments indicate that it is a central nervous system depressant. Acute high level exposures produced pulmonary damage and non-specific hepatic and renal lesions in animals.

MEDICAL SURVEILLANCE

Evaluate possible irritant effects on skin and eyes.

SPECIAL TESTS

None in common use.

PERSONAL PROTECTIVE METHODS

Protective clothing should be worn, and in areas of elevated vapor concentrations, fullface masks should be supplied.

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ETHYLENEDIAMINE

DESCRIPTION

$H_2N\text{-}CH_2\text{-}CH_2\text{-}NH_2$, ethylenediamine, is a strongly alkaline, colorless, clear, thick, liquid with an ammonia odor.

SYNONYMS

Ethanediamine, 1,2-diaminoethane.

POTENTIAL OCCUPATIONAL EXPOSURES

Ethylenediamine is used as a solvent, an emulsifier for casein and shellac solutions, a stabilizer in rubber latex, a chemical intermediate in the manufacture of dyes, corrosion inhibitors, synthetic waxes, fungicides, resins, insecticides, asphalt wetting agents, and pharmaceuticals, and also in controlling acidity or alkalinity.

A partial list of occupations in which exposure may occur includes:

- Albumin processors
- Casein processors
- Drug makers
- Dye makers
- Emulsion workers
- Ethylenediamine tetraacetic acid (EDTA) makers
- Fungicide makers
- Insecticide makers
- Oil neutralizers
- Resin makers
- Rubber makers
- Shellac processors
- Surfactant makers

PERMISSIBLE EXPOSURE LIMITS

The Federal standard is 10 ppm (25 mg/m³).

ROUTES OF ENTRY

Inhalation of vapor and percutaneous absorption.

HARMFUL EFFECTS

Local—

Ethylenediamine vapor may cause irritation of the nose and tingling of the face. Cutaneous sensitivity has been reported.

In animal experiments, the liquid has produced severe irritation of the eyes and corneal damage. It has also produced severe irritation and necrosis.

Systemic—

In animal experiments, high concentrations of ethylenediamine vapor have produced damage to liver, lungs, and kidneys.

MEDICAL SURVEILLANCE

Consider possible irritant effects on skin, eyes and respiratory system. History of allergic redness of skin or asthmatic symptoms may be important in placement and periodic examinations.

SPECIAL TESTS

None have been developed.

PERSONAL PROTECTIVE METHODS

Protective clothing, gloves, and goggles should be worn to protect the skin and eyes. Fullface masks with organic vapor canisters must be used in areas of high vapor concentrations. Recent reports indicate that a non-occupational allergic contact dermatitis may develop after use of pharmaceuticals containing ethylenediamine.

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CYANIDES AND NITRILES

This class of compounds contains the —CN group and includes, in addition to cyanides and nitriles, related chemicals such as cyanogens, isocyanates, and cyanamides.

Hydrogen cyanide and its soluble salts are rapidly acting poisons. The cyanide ion when released in the body is capable of inhibiting many enzymes, the most sensitive being cytochrome oxidase. Deaths from acute exposure are due to chemical asphyxia at the cellular level.

In high concentrations, nitriles (R-CN) can cause similar symptoms, but the onset is slower. It is believed that the CN ion is released from

the nitriles, the rate of release determining the toxicity. In addition, nitriles may be primary irritants.

Other —CN derivatives differ in their toxic properties and may not have the same mechanism of action.

There seems to be a detoxification mechanism for cyanide ion; the enzyme rhodanese (transulfurase) is the catalyst for the reaction

$$S_2O_3^{=} + CN^{-} \rightarrow SO_3^{=} + SCN^{-}$$

in which cyanide ion is converted to thiocyanate ion. Theoretically, this endogenously produced thiocyanate may have lesser toxic effects, similar to those seen in thiocyanate therapy, at high doses. It is unlikely that this is of practical significance, however, because of the minute quantities of CN^- and SCN^- ions involved in poisoning cases.

There are several tests of biological tissues available which are suitable for diagnostic purposes of acute intoxication but not for routine medical surveillance. Thiocyanate is excreted in the urine and may be present in the serum; smokers have a higher thiocyanate level than nonsmokers. Cyanide ion may also be found in blood and tissues.

ACETONITRILE

DESCRIPTION

CH_3-CN, acetonitrile, is a colorless liquid with an ether-like odor.

SYNONYMS

Methyl cyanide, ethanenitrile, cyanomethane.

POTENTIAL OCCUPATIONAL EXPOSURES

Acetonitrile is used as an extractant for animal and vegetable oils, as a solvent, particularly in the pharmaceutical industry, and as a chemical intermediate.

A partial list of occupations in which exposure may occur includes:

Animal oil processors
Organic chemical synthesizers
Vegetable oil processors

PERMISSIBLE EXPOSURE LIMITS

The Federal standard is 40 ppm (70 mg/m^3).

ROUTES OF ENTRY

Inhalation and percutaneous absorption.

HARMFUL EFFECTS

Local—

At high concentrations, nose and throat irritation have been reported. Splashes of the liquid in the eyes may cause irritation. Acetonitrile may cause slight flushing of the face and a feeling of chest tightness.

Systemic—

Acetonitrile has a relatively low acute toxicity, but there have been reports of severe and fatal poisonings in man after inhalation of high concentrations. Signs and symptoms may include nausea, vomiting, respiratory depression, weakness, chest or abdominal pain, hematemesis, convulsions, shock, unconsciousness, and death. In most cases there is a latent period of several hours between exposure and onset of symptoms. It has been thought that acetonitrile itself has relatively little toxic effect and that the delayed response is due to the slow release of cyanide. No chronic disease has been reported.

MEDICAL SURVEILLANCE

Consider the skin, respiratory tract, heart, central nervous system, renal and liver function in placement and periodic examinations. A history of fainting spells or convulsive disorders might present an added risk to persons working with toxic nitriles.

SPECIAL TESTS

None commonly used. Blood CN can be determined but may be of little help in evaluating low level exposures.

PERSONAL PROTECTIVE METHODS

Protective clothing should be worn, and in areas of high concentration, air supplied respirators and complete skin protection are necessary. Workers in these areas must be educated to the nature of acetonitrile hazard. They should also be trained in artificial respiration and in the use of amyl nitrite antidote in emergency situations.

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ACRYLONITRILE

DESCRIPTION

$CH_2=CH-CN$, acrylonitrile, is a colorless liquid with a faint acrid odor. It is both flammable and explosive.

SYNONYMS

Vinyl cyanide, cyanoethylene, propene nitrile.

POTENTIAL OCCUPATIONAL EXPOSURES

Acrylonitrile is used in the manufacture of synthetic fibers, acrylostyrene plastics, acrylonitrile-butadiene styrene plastics, nitrile rubbers, chemicals, and adhesives. It is also used as a pesticide.

A partial list of occupations in which exposure may occur includes:

Acrylic resin makers	Rubber makers
Organic chemical synthesizers	Synthetic fiber makers
Pesticide workers	Textile finish makers

PERMISSIBLE EXPOSURE LIMITS

The Federal standard is 20 ppm (45 mg/m^3).

ROUTES OF ENTRY

Inhalation and percutaneous absorption. It may be absorbed from contaminated rubber or leather.

HARMFUL EFFECTS

Local—

Acrylonitrile may cause irritation of the eyes. Repeated and prolonged exposure may produce skin irritation. When acrylonitrile is held in contact with the skin (e.g., after being absorbed into shoe leather or clothing), it may produce blistering after several hours of no apparent effect. Unless the contaminated clothing is removed promptly and the area washed off, blistering will occur.

Systemic—

Acrylonitrile exposure may produce nausea, vomiting, headache, sneezing, weakness, and light-headedness. Exposure to high concentrations may produce profound weakness, asphyxia, and death.

MEDICAL SURVEILLANCE

Consider the skin, respiratory tract, heart, central nervous system, renal and liver function in placement and periodic examinations. A history of fainting spells or convulsive disorders might present an added risk to persons working with toxic nitriles.

SPECIAL TESTS

None commonly used.

PERSONAL PROTECTIVE METHODS

Leather should not be used in protective clothing since it is readily penetrated by acrylonitrile. Rubber clothing should be frequently washed and inspected because it will soften and swell. Acrylonitrile should be handled with all of the same precautions as taken for hydrogen cyanide, and workers' education should be identical. Liquid splashed on skin should be immediately washed off. Eyes should be protected from splash (goggles), and, in areas of vapor concentration, special cyanide masks or air supplied masks should be provided. Workers should be trained in artificial respiration and in the use of amyl nitrite antidote in emergency situations.

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CALCIUM CYANAMIDE

DESCRIPTION

NCN=Ca, calcium cyanamide, is a blackish-grey, shiny powder.

SYNONYMS

Nitrolim, calcium carbimide, cyanamide.

POTENTIAL OCCUPATIONAL EXPOSURES

Calcium cyanamide is used in agriculture as a fertilizer, herbicide, defoliant for cotton plants, and pesticide. It is also used in the manufacture of dicyanidamide and calcium cyanide, as a desulfurizer in the iron and steel industry, and in steel hardening.

A partial list of occupations in which exposure may occur includes:

- Ammonia makers
- Cotton defoliant workers
- Cyanamide makers
- Fertilizer workers
- Herbicide workers
- Nitrogen compound makers
- Organic chemical synthesizers
- Steel workers

PERMISSIBLE EXPOSURE LIMITS

There is no Federal standard for calcium cyanamide. (Note: The 1976 ACGIH TLV was 0.5 mg/m^3.)

ROUTE OF ENTRY

Inhalation of dust.

HARMFUL EFFECTS

Local—

Calcium cyanamide is a primary irritant of the mucous membranes of the respiratory tract, eyes, and skin. Inhalation may result in rhinitis, pharyngitis, laryngitis, and bronchitis. Conjunctivitis, keratitis, and corneal ulceration may occur. An itchy erythematous dermatitis has been reported and continued skin contact leads to the formation of slowly healing ulcerations on the palms and between the fingers. Sensitization occasionally develops. Chronic rhinitis and perforation of the nasal septum have been reported after long exposures. All local effects appear to be due to the caustic nature of cyanamide.

Systemic—

Calcium cyanamide causes a characteristic vasomotor reaction. There is erythema of the upper portions of the body, face, and arms,

accompanied by nausea, fatigue, headache, dyspnea, vomiting, oppression in the chest, and shivering. Circulatory collapse may follow in the more serious cases. The vasomotor response may be triggered or intensified by alcohol ingestion. Pneumonia or lung edema may develop. Cyanide ion is not released in the body, and the mechanism of toxic action is unknown.

MEDICAL SURVEILLANCE

Evaluate skin, respiratory tract, and history of alcohol intake in placement or periodic examinations.

SPECIAL TESTS

None commonly used.

PERSONAL PROTECTIVE METHODS

In addition to personal protective equipment, waterproof barrier creams may be used to provide additional face and skin protection. Personal hygiene measures are to be encouraged, such as showering after work and a complete change of clothing. In areas of heavy dust concentrations, fullface dust masks are recommended.

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o-CHLOROBENZYLIDENE MALONITRILE

DESCRIPTION

$ClC_6H_4CH=C(CN)_2$ o-chlorobenzylidene malonitrile (OCBM), is a white crystalline solid.

SYNONYMS

OCBM, CS.

POTENTIAL OCCUPATIONAL EXPOSURES

OCBM is used as a riot control agent.

A partial list of occupations in which exposure may occur includes:

Riot controllers

PERMISSIBLE EXPOSURE LIMITS

The Federal standard is 0.05 ppm (0.4 mg/m^3).

ROUTE OF ENTRY

Inhalation.

HARMFUL EFFECTS

Local—

OCBM is extremely irritating and acts on exposed sensory nerve endings (primarily in the eyes and upper respiratory tract). The signs

and symptoms from exposure to the vapor are conjunctivitis and pain in the eyes, lacrimation, erythema of the eyelids, blepharospasms, irritation and running of the nose, burning in the throat, coughing and constricted feeling in the chest, and excessive salivation. Vomiting may occur if saliva is swallowed. Most of the symptoms subside after exposure ceases. Burning on the exposed skin is increased by moisture. With heavy exposure, vesiculation and erythema occur. Photophobia has been reported.

Systemic—

Animal experiments indicate that OCBM has a relatively low toxicity. The systemic changes observed in human experiments are nonspecific reactions to stress. OCBM is capable of sensitizing guinea pigs; there also appears to be a cross-reaction in guinea pigs previously sensitized to 1-chloroacetophenone (CN)

MEDICAL SURVEILLANCE

Consideration should be given to the eyes, skin, and respiratory tract in any placement or periodic evaluations.

SPECIAL TESTS

None have been proposed.

PERSONAL PROTECTIVE METHODS

Because of its extremely irritant properties, those using OCBM in high concentrations should wear respirators and eye protection.

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Punte, C. L., E. J. Owens, and P. J. Gutentag. 1963. Exposures to orthochlorobenzylidene malonitrile. Arch. Environ. Health 6:366.

HYDROGEN CYANIDE

DESCRIPTION

Hydrogen cyanide, a colorless gas or liquid, intensely poisonous, with the odor of bitter almonds, is highly flammable and explosive and is a very weak acid. Hydrogen cyanide, HCN (together with its soluble salts), owes its toxicity to the $-CN$ moiety and not to its acid properties. HCN vapor is released when cyanide salts come in contact with any acid.

SYNONYMS

Hydrocyanic acid, prussic acid.

POTENTIAL OCCUPATIONAL EXPOSURES

Hydrogen cyanide is used as a fumigant, in electroplating, and in chemical synthesis of acrylates and nitriles, particularly acrylonitrile. It may be generated in blast furnaces, gas works, and coke ovens. Cyanide salts have a wide variety of uses, including electroplating, steel hardening, fumigating, gold and silver extraction from ores, and chemical synthesis.

A partial list of occupations in which exposure may occur includes:

- Acid dippers
- Acrylate makers
- Ammonium salt makers
- Blast furnace workers
- Cellulose product treaters
- Coke oven workers
- Cyanogen makers
- Electroplaters
- Fumigant workers
- Gas workers
- Gold extractors
- Jewelers
- Organic chemical synthesizers
- Polish makers
- Silver extractors
- Steel workers

PERMISSIBLE EXPOSURE LIMITS

The Federal standard for hydrogen cyanide is 10 ppm (11 mg/m^3). NIOSH has recommended 5 mg/m^3 expressed as cyanide and determined as a ceiling concentration based on a 10-minute sampling period.

ROUTES OF ENTRY

Inhalation of vapor and percutaneous absorption of liquid and concentrated vapor.

HARMFUL EFFECTS

Local—

Hydrogen cyanide is a mild upper respiratory irritant and may cause slight irritation of the nose and throat. There may also be irritation from skin and eye contact with the liquid. Hydrogen cyanide liquid may cause eye irritation.

Systemic—

Hydrogen cyanide is an asphyxiant. It inactivates certain enzyme systems, the most important being cytochrome oxidase, which occupies a fundamental position in the respiratory process and is involved in the ultimate electron transfer to molecular oxygen. Inhalation, ingestion, or skin absorption of hydrogen cyanide may be rapidly fatal. Larger doses may cause loss of consciousness, cessation of respiration, and death. Lower levels of exposure may cause weakness, headache, confusion, nausea, and vomiting. These symptoms may be followed by unconsciousness and death.

MEDICAL SURVEILLANCE

Preplacement and periodic examinations should include the cardiovascular and central nervous systems, liver and kidney function, blood, history of fainting or dizzy spells.

SPECIAL TESTS

Blood CN levels may be useful during acute intoxication. Urinary thiocyanate levels have been used but are nonspecific and are elevated in smokers.

PERSONAL PROTECTIVE METHODS

If personal protective equipment is necessary, air supplied or self-contained gas masks specific for hydrogen cyanide, and clothing impervious to HCN vapor should be worn. Eye protection can be provided by fullface respirators or goggles. All personnel working with processes involving cyanides should be specially trained so that they fully understand the hazard, and so they will faithfully follow all rules laid down for safe handling.

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ISOCYANATES

DESCRIPTION

Both toluene diisocyanate (TDI) and methylene bisphenyl isocyanate (MDI) are liquids and may exist in different isomers: 2,4-toluene diisocyanate and methylene bisphenyl 4,4'-diisocyanate. Other less commonly used isocyanates are hexamethylene diisocyanate (HDI) and 1,5-naphthalene diisocyanate (NDI).

SYNONYMS

Toluene diisocyanate: TDI, tolylene diisocyanate, diisocyanatotoluene. Methylene bisphenyl isocyanate: MDI, diphenylmethane diisocyanate, methane diisocyanate.

POTENTIAL OCCUPATIONAL EXPOSURES

TDI is more widely used than MDI. Polyurethanes are formed by the reaction of isocyanates with polyhydroxy compounds. Since the reaction proceeds rapidly at room temperature, the reactants must be mixed in pots or spray guns just before use. These resins can be produced with various physical properties, e.g., hard, flexible, semirigid foams, and have found many uses, e.g., upholstery padding, thermal insulation, molds, surface coatings, shoe innersoles, and in rubbers, adhesives, paints, and textile finishes. Because of TDI's high volatility, exposure can occur in all phases of its manufacture and use. MDI has a much lower volatility, and problems generally arise only in spray applications.

A partial list of occupations in which exposure may occur includes:

- Adhesive workers
- Insulation workers
- Isocyanate resin workers
- Lacquer workers
- Organic chemical synthesizers
- Paint sprayers
- Polyurethane makers
- Rubber workers
- Ship burners
- Textile processors
- Wire coating workers

PERMISSIBLE EXPOSURE LIMITS

The Federal standard for MDI is 0.02 ppm (0.2 mg/m^3) as a ceiling value. The Federal standard for the 2,4 isomer of TDI is also 0.02 ppm (0.14 mg/mg^3) as a ceiling value. However, the standard recommended in the NIOSH Criteria Document for TDI is 0.005 ppm (0.036 mg/m^3) as a TWA and 0.02 for any 20-minute period.

ROUTE OF ENTRY

Inhalation of vapor.

HARMFUL EFFECTS

Local—

TDI and MDI may cause irritation of the eyes, respiratory tract, and skin. The irritation may be severe enough to produce bronchitis and pulmonary edema. Nausea, vomiting, and abdominal pain may occur. If liquid TDI is allowed to remain in contact with the skin, it may produce redness, swelling, and blistering. Contact of liquid TDI with the eyes may cause severe irritation, which may result in permanent damage if untreated. Swallowing TDI may cause burns of the mouth and stomach.

Systemic—

Sensitization to TDI and MDI may occur, which may cause an asthmatic reaction with wheezing, dyspnea, and cough. These symptoms may first occur during the night following exposure to these chemicals. Some decrease in lung function in the absence of symptoms has been observed in some workers exposed to TDI for long periods of time.

MEDICAL SURVEILLANCE

Preplacement and periodic medical examinations should include chest roentgenograph, pulmonary function tests, and an evaluation of any respiratory disease or history of allergy. Periodic pulmonary function tests may be useful in detecting the onset of pulmonary sensitization.

SPECIAL TESTS

None in common use.

PERSONAL PROTECTIVE METHODS

Protective clothing and goggles should be worn if there is a possibility of contact with the liquids. In areas of vapor concentration, full-

face masks with organic vapor canisters or respirators with supplied air and fullface pieces should be worn.

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AROMATIC HYDROCARBONS

Aromatic hydrocarbons are characterized by the presence of the aromatic nucleus. The basic aromatic nucleus is benzene, C_6H_6. In benzene, the carbon atoms are arranged as a regular hexagon, with a hydrogen atom attached to each of the carbon atoms. The bond between each of the carbon atoms is neither a single bond nor a double bond, but an intermediate form of higher stability. (The electronic character of the benzene nucleus is usually referred to as "resonance.") The fact that the bonds are intermediate between single and double results in all of the carbon atoms being equivalent. The hydrogen atoms on the aromatic nucleus may be replaced by other univalent elements or groups. Aromatic hydrocarbons encompass compounds that include only carbon and hydrogen.

Aromatic hydrocarbons have enjoyed wide usage as solvents and as chemical intermediates. Benzene, the typical aromatic hydrocarbon, has been replaced as a commercial solvent by toluene and other less toxic compounds. These chemicals are also used as feedstock for many organic compounds and are used in the manufacture of fuels, dyes, pharmaceuticals, plastics, resins, and polyesters.

Typically, the vapor of aromatic hydrocarbons causes central nervous system depression or other effects, and, depending on the compound, hepatic, renal, or bone marrow disorders. Vapor is absorped through the lungs, and the liquid may be absorbed through the skin. Repeated and prolonged skin contact may cause defatting of the skin, which leads to dermatitis.

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BENZENE

DESCRIPTION

C_6H_6, benzene, is a clear, volatile, colorless, highly flammable liquid with a charactersitic odor. The most common commercial grade

contains 50-100% benzene, the remainder consisting of toluene, xylene, and other constituents which distill below 120 C.

SYNONYMS

Benzol, phenyl hydride, coal naphtha, phene, benxole, cyclohexatriene.

POTENTIAL OCCUPATIONAL EXPOSURES

Benzene is used as a constituent in motor fuels, as a solvent for fats, inks, oils, paints, plastics, and rubber, in the extraction of oils from seeds and nuts, and in photogravure printing. It is also used as a chemical intermediate. By alkylation, chlorination, nitration, and sulfonation, chemicals such as styrene, phenols, and maleic anhydride are produced. Benzene is also used in the manufacture of detergents, explosives, pharmaceuticals, and dyestuffs.

A partial list of occupations in which exposure may occur includes:

- Adhesive makers
- Asbestos product impregnators
- Dry-battery makers
- Chemists
- Benzene hexachloride makers
- Burnishers
- Carbolic acid makers
- Chlorinated benzene workers
- Detergent makers
- Dye makers
- Furniture finishers
- Glue makers
- Linoleum makers
- Maleic acid makers
- Nitrobenzene makers
- Petrochemical workers
- Putty makers
- Rubber makers
- Styrene makers
- Welders

PERMISSIBLE EXPOSURE LIMITS

The Federal emergency standard for benzene effective May 21, 1977, is 1 ppm for an 8-hour TWA, with 5 ppm as a maximum peak above the acceptable ceiling for a maximum duration of 15 minutes.

ROUTES OF ENTRY

Inhalation of vapor which may be supplemented by percutaneous absorption although benzene is poorly absorbed through intact skin.

HARMFUL EFFECTS

Local—

Exposure to liquid and vapor may produce primary irritation to skin, eyes, and upper respiratory tract. If the liquid is aspirated into the lung, it may cause pulmonary edema and hemorrhage. Erythema, vesiculation, and dry, scaly dermatitis may also develop from defatting of the skin.

Systemic—

Acute exposure to benzene results in central nervous system depression. Headache, dizziness, nausea, convulsions, coma, and death

may result. Death has occurred from large acute exposure as a result of ventricular fibrillation, probably caused by myocardial sensitization to endogenous epinephrine. Early reported autopsies revealed hemorrhages (non-pathognomonic) in the brain, pericardium, urinary tract, mucous membranes, and skin.

Chronic exposure to benzene is well documented to cause blood changes. Benzene is basically a myelotoxic agent. Erythrocyte, leukocyte, and thrombocyte counts may first increase, and then aplastic anemia may develop with anemia, leukopenia, and thrombocytopenia. The bone marrow may become hypo- or hyper-active and may not always correlate with peripheral blood.

Recent epidemiologic studies along with case reports of benzene related blood dyscrasias and chromosomal aberrations have led NIOSH to conclude that benzene is leukemogenic. The evidence is most convincing for acute myelogenous leukemia and for acute erythroleukemia, but a connection with chronic leukemia has been noted by a few investigators.

Recent work has shown increases in the rate of chromosomal aberations associated with benzene myelotoxicity. These changes in the bone marrow are stable or unstable and may occur several years after exposure has ceased. "Stable" changes may give rise to leukemic clones and seem to involve chromosomes of the G group.

MEDICAL SURVEILLANCE

Preplacement and periodic examinations should be concerned especially with effects on the blood and bone marrow and with a history of exposure to other myelotoxic agents or drugs or of other diseases of the blood. Preplacement laboratory exams should include: (a) complete blood count (hematocrit, hemoglobin, mean corpuscular volume, white blood count, differential count, and platelet estimation), (b) reticulocyte count, (c) serum bilirubin, and (d) urinary phenol.

The type and frequency of periodic hematologic studies should be related to the data obtained from biologic monitoring and industrial hygiene studies, as well as any symptoms or signs of hematologic effects. Recommendations for proposed examinations have been made in the criteria for a recommended standard. Examinations should also be concerned with other possible effects such as those on the skin, central nervous system, and liver and kidney functions.

SPECIAL TESTS

Biologic monitoring should be provided to all workers subject to benzene exposure. It consists of sampling and analysis of urine for total phenol content. The objective of such monitoring is to be certain that no worker absorbs an unacceptable amount of benzene. Unacceptable absorption of benzene, posing a risk of benzene poisoning, is considered to occur at levels of 75 mg phenol per liter of urine (with urine specific gravity corrected to 1.024), when determined by methods specified in the NIOSH "Criteria for Recommended Standard - Benzene." Alter-

native methods shown to be equivalent in accuracy and precision may also be useful. Biological monitoring should be done at quarterly intervals. If environmental sampling and analysis are equal to or exceed accepted safe limits, the urinary phenol analysis should be conducted every two weeks. This increased monitoring frequency should continue for at least 2 months after the high environmental level has been demonstrated.

Two follow-up urines should be obtained within one week after receipt of the original results, one at the beginning and the other at the end of the work week. If original elevated findings are confirmed, immediate steps should be taken to reduce the worker's absorption of benzene by improvement in environmental control, personal protection, personal hygiene, and administrative control.

PERSONAL PROTECTIVE METHODS

Protective clothing should be worn at all times; benzene-wetted clothing should be changed at once. Impervious clothing and gloves to cover exposed areas of body should be worn where exposure is continuous. In areas where there is likelihood of spill or splash, face shields or goggles should be provided. In areas of elevated vapor concentration, organic vapor cartridge masks or supplied air or self-contained breathing apparatus may be required.

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DIPHENYL

DESCRIPTION

$C_6H_5C_6H_5$, diphenyl, is a colorless to light yellow, leaflet solid with a potent characteristic odor.

SYNONYMS

Biphenyl, phenylbenzene.

POTENTIAL OCCUPATIONAL EXPOSURES

Diphenyl is a fungistat for oranges which is applied to the inside of shipping containers and wrappers. It is also used as a heat transfer agent and as an intermediate in organic synthesis. Diphenyl is produced by thermal dehydration of benzene.

A partial list of occupations in which exposure may occur includes:

Orange packers
Fungicide workers
Organic chemical synthesizers

PERMISSIBLE EXPOSURE LIMITS

The Federal standard is 0.2 ppm (1 mg/m^3).

ROUTES OF ENTRY

Inhalation of vapor or dust; percutaneous absorption.

HARMFUL EFFECTS

Local—

Repeated exposure to dust may result in irritation of skin and respiratory tract. The vapor may cause moderate eye irritation. Repeated skin contact may produce a sensitization dermatitis.

Systemic—

In acute exposure, diphenyl exerts a toxic action on the central nervous system, on the peripheral nervous system, and on the liver. Symptoms of poisoning are headache, diffuse gastrointestinal pain, nausea, indigestion, numbness and aching of limbs, and general fatigue. Liver function tests may show abnormalities. Chronic exposure is characterized mostly by central nervous system symptoms, fatigue, headache, tremor, insomnia, sensory impairment, and mood changes. Such symptoms are rare, however.

MEDICAL SURVEILLANCE

Consider skin, eye, liver function and respiratory tract irritation in any preplacement or periodic examination.

SPECIAL TESTS

None in common use.

PERSONAL PROTECTIVE METHODS

Because of its low vapor pressure and low order of toxicity, it does not usually present a major problem in industry. Protective creams, gloves, and masks with organic vapor canisters for use in areas of elevated vapor concentrations should suffice. Elevated temperature may increase the requirement for protective methods or ventilation.

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NAPHTHALENE

DESCRIPTION

$C_{10}H_8$, naphthalene, is a white crystalline solid with a characteristic "moth ball" odor.

SYNONYMS

Naphthalin, moth flake, tar camphor, white tar.

POTENTIAL OCCUPATIONAL EXPOSURES

Naphthalene is used as a chemical intermediate or feedstock for synthesis of phthalic, anthranilic, hydroxyl (naphthols), amino (naphthylamines), and sulfonic compounds which are used in the manufacture of various dyes. Naphthalene is also used in the manufacture of hydronaphthalenes, synthetic resins, lampblack, smokeless powder, and celluloid. Naphthalene has been used as a moth repellent.

A partial list of occupations in which exposure may occur includes:

- Beta naphthol makers
- Celluloid makers
- Coal tar workers
- Dye chemical makers
- Fungicide makers
- Hydronaphthalene makers
- Lampblack makers
- Moth repellent workers
- Phthalic anhydride makers
- Smokeless powder makers
- Tannery workers
- Textile chemical makers

PERMISSIBLE EXPOSURE LIMITS

The Federal standard is 10 ppm (50 mg/m^3).

ROUTE OF ENTRY

Inhalation of vapor or dust.

HARMFUL EFFECTS

Local—

Naphthalene is a primary irritant and causes erythema and dermatitis upon repeated contact. It is also an allergen and may produce dermatitis in hypersensitive individuals. Direct eye contact with the dust has produced irritation and cataracts.

Systemic—

Inhaling high concentrations of naphthalene vapor or ingesting may cause intravascular hemolysis and its consequences. Initial symptoms include eye irritation, headache, confusion, excitement, malaise, profuse sweating, nausea, vomiting, abdominal pain, and irritation of the bladder. There may be progressive jaundice, hematuria, hemoglobinuria, renal tubular blockade, and acute renal shutdown. Hematologic features include red cell fragmentation, icterus, severe anemia with nucleated red cells, leukocytosis, and dramatic decreases in hemoglobin, hematocrit, and red cell count. Individuals with a deficiency of glucose-6-phosphate dehydrogenase in erythrocytes are more susceptible to hemolysis by naphthalene.

MEDICAL SURVEILLANCE

Consider eyes, skin, blood, liver, and renal function in placement and follow-up examinations. Low erythrocyte glucose 6-phosphate dehydrogenase increases risk.

SPECIAL TESTS

None in common use.

PERSONAL PROTECTIVE METHODS

As used in industry, they are rarely necessary. In dusty areas and areas of high vapor concentration, dust type or organic vapor canister masks should be supplied. Skin protection with gloves, barrier creams, or protective clothing may be useful.

STYRENE/ETHYL BENZENE

DESCRIPTION

$C_6H_5CH=CH_2$, styrene, is a colorless to yellowish, very refractive, oily liquid with a penetrating odor.

$C_6H_5C_2H_5$, ethyl benzene, is a colorless flammable liquid with a pungent odor.

SYNONYMS

Styrene: Cinnamene, cinnemenol, cinnamol, phenethylene, phenylethylene, styrene monomer, styrol, styrolene, vinyl benzene.

Ethyl benzene: Ethylbenzol, phenylethane, EB.

POTENTIAL OCCUPATIONAL EXPOSURES

Upon heating to 200 C, styrene polymerizes to form polystyrene, a plastic. It is also used in combination with 1,3-butadiene or acrylonitrile to form copolymer elastomers, butadiene-styrene rubber, and acrylonitrile-butadienestyrene (ABS). It is also used in the manufacture of resins, polyesters, and insulators.

Ethyl benzene is used in the manufacture of cellulose acetate, styrene, and synthetic rubber. It is also used as a solvent or diluent and as a component of automotive and aviation gasoline.

A partial list of occupations in which exposure may occur includes:

- Adhesive makers
- Aviation fuel blenders
- Emulsifier agent makers
- Fibrous glass moulders
- Insulator makers
- Lacquer workers
- Organic chemical synthesizers
- Petroleum refinery workers
- Polyester resin laminators
- Polystyrene makers
- Potting compound workers
- Protective coating workers
- Resin makers
- Rubber makers
- Solvent workers
- Varnish makers.

PERMISSIBLE EXPOSURE LIMITS

The Federal standard for styrene for an 8-hour TWA is 100 ppm (420 mg/m^3). The acceptable ceiling concentration is 200 ppm with an acceptable maximum peak of 600 ppm for a maximum duration of 5 minutes in any 3 hours. The Federal standard for ethyl benzene is 100 ppm (435 mg/m^3).

ROUTES OF ENTRY

Inhalation of vapor; percutaneous absorption.

HARMFUL EFFECTS

Local—

Liquid and vapor are irritating to the eyes, nose, throat, and skin. The liquids are low-grade cutaneous irritants, and repeated contact may produce a dry, scaly, and fissured dermatitis.

Systemic—

Acute exposure to high concentrations may produce irritation of the mucous membranes of the upper respiratory tract, nose, and mouth, followed by symptoms of narcosis, cramps, and death due to respiratory center paralysis. Effects of short-term exposure to styrene under laboratory conditions include prolonged reaction time and decreased manual dexterity.

MEDICAL SURVEILLANCE

Consider possible irritant effects on the skin, eyes, and respiratory tract in any preplacement or periodic examinations, as well as blood, liver, and kidney function.

SPECIAL TESTS

None in common use. Mandelic acid in urine has been used as a measure of the intensity of styrene exposure.

PERSONAL PROTECTIVE METHODS

Barrier creams or gloves and protective clothing may be all that are needed where the vapor concentrations do not exceed existing standards. Where vapor concentration exists above allowable standards, masks with organic vapor canisters and face plates or respirators with air supply are recommended. Clothing saturated with styrene or ethylbenzene should be changed at once. Personal hygiene is encouraged with frequent changes of work clothes.

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TOLUENE

DESCRIPTION

$C_6H_5CH_3$, toluene, is a clear, colorless, noncorrosive liquid with a sweet, pungent, benzene-like odor.

SYNONYMS

Toluol, methylbenzene, phenylmethane, methylbenzol.

POTENTIAL OCCUPATIONAL EXPOSURES

Toluene may be encountered in the manufacture of benzene. It is

also used as a chemical feed for toluene diisocyanate, phenol, benzyl and benzyl derivatives, benzoic acid, toluene sulfonates, nitrotoluenes, vinyl toluene, and saccharin; as a solvent for paints and coatings; or as a component of automobile and aviation fuels.

A partial list of occupations in which exposure may occur includes:

Aviation fuel blenders,
Benzene makers
Chemical laboratory workers
Coke oven workers
Gasoline blenders
Lacquer workers
Paint thinner makers
Perfume makers
Petrochemical workers
Rubber cement makers
Saccharin makers
Solvent workers
Toluene diisocyanate makers
Vinyl toluene makers

PERMISSIBLE EXPOSURE LIMITS

The Federal standard is 200 ppm as an 8-hour TWA with an acceptable ceiling concentration of 300 ppm; acceptable maximum peaks above the ceiling of 500 ppm are allowed for 10 minutes duration. NIOSH has recommended a limit of 100 ppm (TWA) with a ceiling of 200 ppm for a ten minute sampling period.

ROUTES OF ENTRY

Inhalation of vapor and percutaneous absorption of liquid.

HARMFUL EFFECTS

Local—

Toluene may cause irritation of the eyes, respiratory tract, and skin. Repeated or prolonged contact with liquid may cause removal of natural lipids from the skin, resulting in dry, fissured dermatitis. The liquid splashed in the eyes may cause irritation and reversible damage.

Systemic—

Acute exposure to toluene predominantly results in central nervous system depression. Symptoms and signs include headache, dizziness, fatigue, muscular weakness, drowsiness, incoordination with staggering gait, skin paresthesias, collapse, and coma.

MEDICAL SURVEILLANCE

Preplacement and periodic examinations should evaluate possible effect on skin, central nervous system, as well as liver and kidney function. Hematologic studies should also be done if there is significant contamination of the solvent with benzene.

SPECIAL TESTS

Hippuric acid levels above 5 g/liter of urine may result from exposure greater than 200 ppm determined as a TWA. Blood levels can also be determined for toluene.

PERSONAL PROTECTIVE METHODS

Where vapor concentration exists above allowable standards, em-

ployees should be provided with respirators (air supplied) or gas masks with organic vapor canister and fullface plate. Impervious clothing, gloves, or other coverings to protect potentially exposed areas of the body should be supplied to employees in operations requiring continued exposure to liquid toluene. Toluene-wet clothing should be immediately removed unless impervious, and work clothing changed at least twice a week. Safety glasses or goggles should be worn in areas where splash or spill is likely.

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U.S. Department of Health, Education, and Welfare, Public Health Service, Center for Disease Control, National Institute for Occupational Safety and Health. 1973. Criteria for a Recommended Standard .... Occupational Exposure to Toluene. U.S. Government Printing Office, Washington, D.C.

XYLENE

DESCRIPTION

$C_6H_4(CH_3)_2$, xylene, exists in three isomeric forms, ortho-, meta- and para-xylene. Commercial xylene is a mixture of these three isomers and may also contain ethyl benzene as well as small amounts of toluene, trimethyl benzene, phenol, thiophene, pyridine, and other non-aromatic hydrocarbons. Metaxylene is predominant in commercial xylene and shares physical properties with ortho-xylene in that both are mobile, colorless, flammable liquids. Para-xylene, at low temperature (13-14 C), forms colorless plates or prisms.

SYNONYMS

Xylol, dimethylbenzene.

POTENTIAL OCCUPATIONAL EXPOSURES

Xylene is used as a solvent; as a constituent of paint, lacquers, varnishes, inks, dyes, adhesives, cements, cleaning fluids and aviation fuels; and as a chemical feedstock for xylidines, benzoic acid, phthalic anhydride, isophthalic, and terephthalic acids, as well as their esters (which are specifically used in the manufacture of plastic materials and synthetic textile fabrics). Xylene is also used in the manufacture of quartz crystal oscillators, hydrogen peroxide, perfumes, insect repellants, epoxy resins, pharmaceuticals, and in the leather industry.

A partial list of occupations in which exposure may occur includes:

- Adhesive workers
- Aviation gasoline workers
- Benzoic acid makers
- Cleaning fluid makers
- Histology technicians
- Lacquer workers
- Leather workers
- Paint workers
- Phthalic anhydride makers
- Polyethylene terephthalate film makers
- Quartz crystal oscillator makers
- Solvent workers
- Synthetic textile makers
- Terephthalic acid makers
- Varnish makers

PERMISSIBLE EXPOSURE LIMITS

The Federal standard is 100 ppm (435 mg/m^3).

ROUTES OF ENTRY

Inhalation of vapor and, to a small extent, percutaneous absorption of liquid.

HARMFUL EFFECTS

Local—

Xylene vapor may cause irritation of the eyes, nose, and throat. Repeated or prolonged skin contact with xylene may cause drying and defatting of the skin which may lead to dermatitis. Liquid xylene is irritating to the eyes and mucous membranes, and aspiration of few milliliters may cause chemical pneumonitis, pulmonary edema, and hemorrhage. Repeated exposure of the eyes to high concentrations of xylene vapor may cause reversible eye damage.

Systemic—

Acute exposure to xylene vapor may cause central nervous system depression and minor reversible effects upon liver and kidneys. At high concentrations xylene vapor may cause dizziness, staggering, drowsiness, and unconsciousness. Also at very high concentrations, breathing xylene vapors may cause pulmonary edema, anorexia, nausea, vomiting, and abdominal pain.

MEDICAL SURVEILLANCE

Preplacement and periodic examinations should evaluate possible effects on the skin and central nervous system, as well as liver and kidney functions. Hematalogic studies should be done if there is any significant contamination of the solvent with benzene.

SPECIAL TESTS

Although metabolites are known, biologic monitoring has not been widely used. Hippuric acid or the ether glucuronide of ortho-toluic acid may be useful in diagnosis of meta-, para- and ortho-xylene exposure, respectively.

PERSONAL PROTECTIVE METHODS

When vapor concentrations exceed allowable standards, fullface masks with organic vapor canisters or air supplied respirators should be furnished. Impervious protective clothing and gloves should be worn to cover exposed portions of the body of employees exposed to liquid xylene. Xylene-wet clothing should be changed quickly. Personal hygiene, as well as appropriate changes of work clothes, is necessary. Goggles or safety glasses in areas of spill or splash, or in areas where vapors concentrate, are advised. Barrier creams may be useful.

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PHENOLS AND PHENOLIC COMPOUNDS

This group of compounds is characterized by the substitution of one or more hydrogens in a benzene ring by hydroxyl (−OH) groups. Phenol (C_6H_5OH) is the simplest of the compounds. Additional substitutions are possible. Quinone ($C_6H_4O_2$) is included in this group because it is derived from hydroquinone although its physical, chemical, and toxic properties are quite different. These substances are widely distributed in industry and some (e.g., phenolcresol) find use in pharmaceuticals because of their disinfectant action.

These materials generally enter the body by inhalation and percutaneous absorption. Their toxicity varies, but some are highly irritating to the skin, mucous membranes of the upper respiratory tract, and eyes. Some are corrosive for all tissue; cresote, a complex mixture of phenolic and aromatic compounds, may cause skin cancer. Systemic effects usually involve the central nervous or cardio-vascular systems or both; this may be accompanied by renal and hepatic damage.

Appropriate engineering controls and personal protective devices should be used to prevent absorption by either the respiratory or percutaneous route, and eye protection should be utilized where necessary.

CRESOL

DESCRIPTION

$CH_3C_6H_4OH$, cresol, is a mixture of the three isomeric cresols, ortho-,meta-, and para-cresol, and is a colorless, yellowish, brownish-yellow, or pinkish liquid with a phenolic odor. Creosols are soluble in alcohol, glycol, and dilute alkalis. Also they may be combustible.

SYNONYMS

Cresylic acid, cresylol, hydroxytoluene, methyl phenol, oxytoluene, tricresol.

POTENTIAL OCCUPATIONAL EXPOSURES

Cresol is used as a disinfectant, as an ore flotation agent, and as an intermediate in the manufacture of chemicals, dyes, plastics, and antioxidants. A mixture of isomers is generally used; the concentrations of the components are determined by the source of the cresol.

A partial list of occupations in which exposure may occur includes:

Antioxidant makers	Paint remover workers
Chemical disinfectant workers	Pitch workers
Dye makers	Plastic makers
Flotation agent makers	Resin makers
Foundry workers	Stain workers
Insulation enamel workers	Wool scourers

PERMISSIBLE EXPOSURE LIMITS

The Federal standard is 5 ppm (22 mg/m^3).

ROUTES OF ENTRY

Inhalation or percutaneous absorption of liquid or vapor.

HARMFUL EFFECTS

Local—

Cresol is very corrosive to all tissues. It may cause burns if it is not removed promptly and completely and in case of extensive exposure, if it is not removed completely from contaminated areas of the body very quickly, death may result. When it contacts the skin, it may not produce any sensation immediately. After a few moments, prickling and intense burning occur. This is followed by loss of feeling. The affected skin shows wrinkling, white discoloration, and softening. Later gangrene may occur. If the chemical contacts the eyes, it may cause extensive damage and blindness. A skin rash may result from repeated or prolonged exposure of the skin to low concentrations of cresol. Discoloration of the skin may also occur from this type of exposure.

Systemic—

When cresol is absorbed into the body either through the lungs, through the skin, or mucous membranes, or by swallowing, it may cause systemic poisoning. The signs and symptoms of systemic poisoning may develop in 20 or 30 minutes. These toxic effects include: weakness of the muscles, headache, dizziness, dimness of vision, ringing of the ears, rapid breathing, mental confusion, loss of consciousness, and sometimes death.

Prolonged or repeated absorption of low concentrations of cresol through the skin, mucous membranes, or respiratory tract may cause chronic systemic poisoning. Symptoms and signs of chronic poisoning include vomiting, difficulty in swallowing, salivation, diarrhea, loss of appetite, headache, fainting, dizziness, mental disturbances, and skin rash. Death may result if there has been severe damage to the liver and kidneys.

MEDICAL SURVEILLANCE

Consider the skin, eyes, respiratory system, and liver and kidney function in placement or periodic examinations.

SPECIAL TESTS

Can be determined in urine, but because large amounts are normally present, a urine test is of little value as a procedure for evaluating exposure.

PERSONAL PROTECTIVE METHODS

Protective goggles and clothing should be worn to prevent direct contact with cresol. Masks with organic vapor canisters are advisable in areas of vapor concentration.

BIBLIOGRAPHY

American Industrial Hygiene Association. 1969. Community air quality guides. Phenol and cresol. Am. Ind. Hyg. Assoc. J. 30:425.

CREOSOTE

DESCRIPTION

Creosote is a flammable, heavy, oily liquid with a characteristic sharp, smoky smell, and caustic burning taste. In pure form it is colorless, but the industrial product is usually brownish. It is produced by the destructive distillation of wood or coal tar at temperatures above 200 C. The chemical composition is determined by the source and may contain guaiacol, creosols, phenol, cresols, pyridine, and numerous other aromatic compounds.

SYNONYMS

Creosotum, cresote oil, brick oil.

POTENTIAL OCCUPATIONAL EXPOSURES

Creosote is used primarily as a wood preservative, and those working with the treated wood may be exposed. It is also used as a waterproofing agent, an animal dip, a constituent in fuel oil, a lubricant for die molds, as pitch for roofing, and in the manufacture of chemicals and lampblack. In the pharmaceutical industry, it is used as an antiseptic, disinfectant, antipyretic, astringent, styptic, germicide, and expectorant.

A partial list of occupations in which exposure may occur includes:

- Coal tar workers
- Fuel oil blenders
- Lampblack makers
- Organic chemical synthesizers
- Pitch workers
- Water proofers
- Wood preservers

PERMISSIBLE EXPOSURE LIMITS

There is no Federal standard for creosote.

ROUTE OF ENTRY

Skin absorption.

HARMFUL EFFECTS

Local—

The liquid and vapors are strong irritants producing local erythema,

burning, itching, pigmentation (grayish yellow to bronze), vesiculation, ulceration, and gangrene. Eye injuries include keratitis, conjunctivitis, and permanent corneal scars. Contact dermatitis is reported in industry. Photosensitization has been reported. Skin cancer may occur.

Systemic—

Symptoms of systemic illness include salivation, vomiting, vertigo, headache, loss of pupillary reflexes, hypothermia, cyanosis, convulsions, thready pulse, respiratory difficulties, and death.

MEDICAL SURVEILLANCE

Consider the skin, eyes, respiratory tract, and central nervous system in placement and periodic examination.

SPECIAL TESTS

None commonly used.

PERSONAL PROTECTIVE METHODS

Protective clothing should be worn where employees are exposed to the liquid or high vapor concentration. Masks with fullface protection and organic vapor canisters should be worn. Gloves and goggles are advisable in any area where spill or splash might occur.

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HYDROQUINONE

DESCRIPTION

$C_6H_4(OH)_2$, hydroquinone, exists as colorless, hexagonal prisms.

SYNONYMS

Quinol, hydroquinol, p-diphenol, hydrochinone, dihydroxybenzene, p-dihydroxybenzene, p-hydroxyphenol, 1,4-benzenediol.

POTENTIAL OCCUPATIONAL EXPOSURES

Hydroquinone is a reducing agent and is used as a photographic developer and as an antioxidant or stabilizer for certain materials which polymerize in the presence of oxidizing agents. Many of its derivatives are used as bacteriostatic agents, and others, particularly 2,5-bis(ethyleneimino) hydroquinone, have been reported to be good antimitotic and tumor-inhibiting agents.

A partial list of occupations in which exposure may occur includes:

- Antioxidant makers
- Bacteriostatic agent makers
- Drug makers
- Fur processors
- Motor fuel blenders
- Paint makers
- Organic chemical synthesizers
- Photographic developer makers
- Plastic stabilizer workers
- Stone coating workers
- Styrene monomer workers

PERMISSIBLE EXPOSURE LIMITS

The Federal standard is 2 mg/m³.

ROUTE OF ENTRY

Inhalation of dust.

HARMFUL EFFECTS

Local—

The dust is a mild primary irritant. Skin sensitization to the dry solid is very rare but does occur on occasion from contact with its alkaline solutions. The skin may be depigmented by repeated applications of ointments of hydroquinone, but this virtually never occurs from contact with dust or dilute water solutions. Following prolonged exposure to elevated dust levels, brownish conjunctiva stains may appear. These may be followed by corneal opacities and structural changes in the cornea which may lead to loss of visual acuity. The early pigmentary stains are reversible, while the corneal changes tend to be progressive.

Systemic—

Oral ingestion of large quantities of hydroquinone may produce blurred speech, tinnitus, tremors, sense of suffocation, vomiting, muscular twitching, headache, convulsions, dyspnea and cyanosis from methemoglobinemia, and coma and collapse from respiratory failure. The urine is usually green or brownish green. No systemic symptoms have been found following inhalation of hydroquinone dust.

MEDICAL SURVEILLANCE

Careful examination of the eyes, including visual acuity and slit lamp examinations, should be carried out in preplacement and periodic examinations. Also examine skin.

SPECIAL TESTS

Hydroquinone is excreted in the urine as a sulfate ester. This has not been helpful in following worker exposure to dust.

PERSONAL PROTECTIVE METHODS

The eyes should be protected by goggles or dust masks with full-face shield. Protective clothing is recommended along with good hygiene practice, clothes changing after each shift, and showering prior to dressing in street clothes. Oxidation of hydroquinone may produce quinone vapor which is highly irritating.

BIBLIOGRAPHY

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PHENOL

DESCRIPTION

C_6H_5OH, phenol, is a white crystalline substance with a distinct aromatic, acrid odor.

SYNONYMS

Carbolic acid, phenic acid, phenylic acid, phenyl hydrate, hydroxybenzene, monohydroxybenzene.

POTENTIAL OCCUPATIONAL EXPOSURES

Phenol is used in the production or manufacture of explosives, fertilizer, coke, illuminating gas, lampblack, paints, paint removers, rubber, asbestos goods, wood preservatives, synthetic resins, textiles, drugs, pharmaceutical preparations, perfumes, bakelite, and other plastics (phenol-formaldehyde resins). Phenol also finds wide use as a disinfectant in the petroleum, leather, paper, soap, toy, tanning, dye, and agricultural industries.

A partial list of occupations in which exposure may occur includes:

- Coal tar workers
- Disinfectant makers
- Dye workers
- Explosive workers
- Fertilizer makers
- Illuminating gas workers
- Lampblack makers
- Organic chemical synthesizers
- Paint and paint remover workers
- Paper makers
- Rubber reclaimers
- Soap workers
- Tannery workers
- Weed killer users
- Wood preservers

PERMISSIBLE EXPOSURE LIMITS

The Federal standard is 5 ppm (19 mg/m³).

ROUTES OF ENTRY

Inhalation of mist or vapor; percutaneous absorption of mist, vapor, or liquid.

HARMFUL EFFECTS

Local—

Phenol has a marked corrosive effect on any tissue. When it comes in contact with the eyes it may cause severe damage and blindness. On contact with the skin, it does not cause pain but causes a whitening of the exposed area. If the chemical is not removed promptly, it may cause a severe burn or systemic poisoning.

Systemic—

Systemic effects may occur from any route of exposure. These include paleness, weakness, sweating, headache, ringing of the ears, shock, cyanosis, excitement, frothing of the nose and mouth, dark colored urine, and death. If death does not occur, kidney damage may appear.

Repeated or prolonged exposure to phenol may cause chronic phenol poisoning. This condition is very rarely reported. The symptoms of chronic poisoning include vomiting, difficulty in swallowing, diarrhea, lack of appetite, headache, fainting, dizziness, dark urine, mental disturbances, and possibly, skin rash. Liver and kidney damage and discoloration of the skin may occur.

MEDICAL SURVEILLANCE

Consider the skin, eye, liver, and renal function as part of any preplacement or periodic examination.

SPECIAL TESTS

Phenol can be determined in blood or urine.

PERSONAL PROTECTIVE METHODS

In areas where there is likelihood of a liquid spill or splash, impervious protective clothing and goggles should be worn. In areas of heavy vapor concentrations, fullface mask with forced air supply should be used, as well as protective clothing, gloves, rubber boots, and apron.

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QUINONE

DESCRIPTION

$C_6H_4O_2$, quinone, exists as large yellow, monoclinic prisms; the vapors have a pungent, irritating odor.

SYNONYMS

Benzoquinone, chinone, p-benzoquinone, 1,4-benzoquinone

POTENTIAL OCCUPATIONAL EXPOSURES

Because of its ability to react with certain nitrogen compounds to form colored substances, quinone is widely used in the dye, textile, chemical, tanning, and cosmetic industries. It is used as an intermediate in chemical synthesis for hydroquinone and other chemicals.

A partial list of occupations in which exposure may occur includes:

- Chemical laboratory workers
- Cosmetic makers
- Dye makers
- Gelatin makers
- Hydrogen peroxide makers
- Organic chemical synthesizers
- Photographic film developers
- Protein fiber makers
- Tannery workers
- Textile workers

PERMISSIBLE EXPOSURE LIMITS

The Federal standard is 0.1 ppm (0.4 mg/m^3).

ROUTE OF ENTRY

Inhalation of vapor.

HARMFUL EFFECTS

Local—

Solid quinone in contact with skin or the lining of the nose and throat may produce discoloration, severe irritation, swelling, and the formation of papules and vesicles. Prolonged contact with the skin may cause ulceration. Quinone vapor is highly irritating to the eyes. Following prolonged exposure to vapor, brownish conjunctival stains may appear. These may be followed by corneal opacities and structural changes in the cornea and loss of visual acuity. The early pigmentary stains are reversible, while the corneal dystrophy tends to be progressive.

Systemic—

No systemic effects have been found in workers exposed to quinone vapor over many years.

MEDICAL SURVEILLANCE

Careful examination of the eyes, including visual acuity and slit lamp examinations, should be done during placement and periodic examinations. Also evaluate skin.

SPECIAL TESTS

No useful laboratory tests for monitoring exposure have been developed.

PERSONAL PROTECTIVE METHODS

In areas of high vapor concentrations, protection must be aimed at the eyes and respiratory tract. Fullface mask with organic vapor canisters or respirators with forced air afford protection. The skin can be damaged by contact with solid quinone, solutions, or vapor condensing on the skin, so protective clothing, gloves, and boots are indicated. Personal hygiene is encouraged, with clothes being changed after each shift or after becoming damp from contact with the liquid. Workers should shower before changing to street clothes.

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AROMATIC HALOGENATED HYDROCARBONS

Aromatic compounds having a halogen bearing side chain are extensively used in the manufacture of basic and acid colors, pharmaceuticals, pesticides, resins, and as chemical intermediates. The vapor and

liquid of some of these compounds are highly irritating to all mucous membranes and skin, and some are powerful lacrimators.

The chlorinated naphthalenes and diphenyls produce a severe and disfiguring acne on skin contact. Percutaneous absorption and inhalation of vapor may lead to severe liver damage in certain instances.

With exception of the chlorinated benzenes, the more highly chlorinated the compound, the greater the toxicity.

BENZYL CHLORIDE

DESCRIPTION

$C_6H_5CH_2Cl$, benzyl chloride is a colorless liquid with an unpleasant, irritating odor.

SYNONYMS

Alpha-chlorotoluene.

POTENTIAL OCCUPATIONAL EXPOSURES

Benzyl chloride is used in production of benzal chloride, benzyl alcohol, and benzaldehyde. Industrial usage includes the manufacture of plastics, dyes, synthetic tannins, perfumes, resins, and pharmaceuticals.

A partial list of occupations in which exposure may occur includes:

- Drug makers
- Dye makers
- Gasoline additive makers
- Germicide makers
- Perfume makers
- Photographic developer makers
- Plastic makers
- Resin makers
- Rubber makers
- Tannin makers
- Wetting agent makers

PERMISSIBLE EXPOSURE LIMITS

The Federal standard is 1 ppm (5 mg/m^3).

ROUTE OF ENTRY

Inhalation of vapor.

HARMFUL EFFECTS

Local—

Benzyl chloride is a severe irritant to the eyes and respiratory tract. At 160 mg/m^3 it is unbearably irritating to the eyes and nose. Liquid contact with the eyes produces severe irritation and may cause corneal injury. Skin contact may cause dermatitis.

Systemic—

Benzyl chloride is regarded as a potential cause of pulmonary edema. One author has reported disturbances of liver functions and mild leakopenia in some workers, but this has not been confirmed. Sarcomas have been produced in rats which were injected with benzyl chloride.

MEDICAL SURVEILLANCE

Preplacement and periodic examinations should include the skin, eyes, and an evaluation of the liver, kidney, respiratory tract, and blood.

SPECIAL TESTS

None in common use.

PERSONAL PROTECTIVE METHODS

Personal protective equipment should include industrial filter respirators with goggles, and protective clothing for face, hands, and arms.

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CHLORODIPHENYLS AND DERIVATIVES

DESCRIPTION

$C_{12}H_{10-x}Cl_x$, Chlorodiphenyls, are diphenyl rings in which one or more hydrogen atoms are replaced by a chlorine atom. Most widely used are chlorodiphenyl (42% chlorine), containing 3 chlorine atoms in unassigned positions, and chlorodiphenyl (54% chlorine) containing 5 chlorine atoms in unassigned positions. These compounds are light, straw-colored liquids with typical chlorinated aromatic odors; 42% chlorodiphenyl is a mobile liquid and 54% chlorodiphenyl is a viscous liquid.

Chlorinated diphenyl oxides are ethers of chlorodiphenyls and are included in this group. They range from clear, oily liquids to white to yellowish waxy solids, depending on the degree of chlorination.

SYNONYMS

Chlorobiphenyls, polychlorinated diphenyl, PCB.

POTENTIAL OCCUPATIONAL EXPOSURES

Chlorinated diphenyls are used alone and in combination with chlorinated naphthalenes. They are stable, thermoplastic, and non-flammable, and find chief use in insulation for electric cables and wires in the production of electric condensers, as additives for extreme pressure lubricants, and as a coating in foundry use.

A partial list of occupations in which exposure may occur includes:

- Cable coaters
- Dye makers
- Electric equipment makers
- Herbicide workers
- Lacquer makers
- Paper treaters
- Plasticizer makers
- Resin makers
- Rubber workers
- Textile flameproofers
- Transformer workers
- Wood preservers

PERMISSIBLE EXPOSURE LIMITS

The Federal standards for dichlorophenyl (42%) and dichloro-

diphenyl (54%) are 1 mg/m³ and 0.5 mg/m³ respectively.

ROUTES OF ENTRY

Inhalation of fume or vapor and percutaneous absorption of liquid.

HARMFUL EFFECTS

Local—

Prolonged skin contact with its fumes or cold wax may cause the formation of comedones, sebaceous cysts, and pustules, known as chloracne. Irritation to eyes, nose, and throat may also occur. The above standards are considered low enough to prevent systemic effects, but it is not known whether or not these levels will prevent local effects.

Systemic—

Generally, toxic effects are dependent upon the degree of chlorination; the higher the degree of substitution, the stronger the effects. Acute and chronic exposure can cause liver damage. Signs and symptoms include edema, jaundice, vomiting, anorexia, nausea, abdominal pains, and fatigue.

Studies of accidental oral intake indicate that chlorinated diphenyls are embryotoxic, causing stillbirth, a characteristic grey-brown skin, and increased eye discharge in infants born to women exposed during pregnancy.

MEDICAL SURVEILLANCE

Placement and periodic examinations should include an evaluation of the skin, lung, and liver function. Possible effects on the fetus should be considered.

SPECIAL TESTS

None in common use.

PERSONAL PROTECTIVE METHODS

Protection of exposed skin should be encouraged, since the above standards may not be low enough to prevent chloracne. Barrier creams, protective clothing, and good personal hygiene are good protective measures. Respirators should be used in areas of vapor concentration.

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CHLORINATED BENZENES

DESCRIPTION

Chlorinated benzenes are aromatic rings with one or more chlorines

substituted for a hydrogen. Included in this group are:

Chlorobenzene: phenyl chloride, monochlorobenzene, chlorobenzol.
o-dichlorobenzene: 1,2-dichlorobenzene
m-dichlorobenzene: 1,3-dichlorobenzene.
p-dichlorobenzene: 1,4-dichlorobenzene.
1,2,3-trichlorobenzene: None.
1,2,4-trichlorobenzene: None.
1,3,5-trichlorobenzene: None.
1,2,4,5-tetrachlorobenzene: None.
Hexachlorobenzene: perchlorobenzene.

Compounds with only a few chlorines are usually colorless liquids at room temperature and have an aromatic odor. The more highly substituted compounds are crystals (typically monoclinic).

SYNONYMS

None.

POTENTIAL OCCUPATIONAL EXPOSURES

Chlorobenzene is used as a solvent and as an intermediate in dyestuffs. o-Dichlorobenzene is used as a solvent, fumigant, insecticide, and chemical intermediate. p-Dichlorobenzene finds use as an insecticide, chemical intermediate, disinfectant and moth preventative. Other chlorinated benzenes are not as widely used in industry but find use as chemical intermediates, and to an even lesser extent, as insecticides and solvents.

A partial list of occupations in which exposure may occur includes:

Cellulose acetate workers	Insecticide makers and workers
Deodorant makers	Lacquer workers
Disinfectant workers	Organic chemical synthesizers
Dyers	Paint workers
Dye makers	Resin makers
Fumigant workers	Seed disinfectors

PERMISSIBLE EXPOSURE LIMITS

The Federal standards are:

chlorobenzene	75 ppm	350 mg/m^3
o-dichlorobenzene	50 ppm	300 mg/m^3
p-dichlorobenzene	75 ppm	450 mg/m^3

Threshold limit values for the other compounds have not as yet been established.

ROUTES OF ENTRY

Inhalation of vapor, percutaneous absorption of the liquid.

HARMFUL EFFECTS

Local—

Chlorinated benzenes are irritating to the skin, conjunctiva, and

mucous membranes of the upper respiratory tract. Prolonged or repeated contact with liquid chlorinated benzenes may cause skin burns.

Systemic—

In contrast to aliphatic halogenated hydrocarbons, the toxicity of chlorinated benzenes generally decreases as the number of substituted chlorine atoms increases. Basically, acute exposure to these compounds may cause drowsiness, incoordination, and unconsciousness. Animal exposures have produced liver damage.

Chronic exposure may result in liver, kidney, and lung damage as indicated by animal experiments.

MEDICAL SURVEILLANCE

Preplacement and periodic examinations should consider skin, liver, lung, and kidney.

SPECIAL TESTS

None commonly used. Urinary excretion of 2,5-dichlorophenol may be useful as an index of exposure.

PERSONAL PROTECTIVE METHODS

Barrier creams, protective clothing, and good personal hygiene are good preventive measures. Respirators in areas of vapor concentrations are advised.

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CHLORINATED NAPHTHALENES

DESCRIPTION

$C_{10}H_{8-x}Cl$, the chlorinated naphthalenes, are naphthalenes in which one or more hydrogen atoms have been replaced by chlorine to form wax-like substances, beginning with monochloronaphthalene and going on to the octochlor derivatives. Their physical states vary from mobile liquids to waxy-solids depending on the degree of chlorination.

SYNONYMS

Chloronaphthalenes

POTENTIAL OCCUPATIONAL EXPOSURES

Industrial exposure from individual chlorinated naphthalenes is rarely encountered; rather it usually occurs from mixtures of two or more chlorinated naphthalenes. Due to their stability, thermoplasticity, and nonflammability, these compounds enjoy wide industrial application. These compounds are used in the production of electric condensers, in the insulation of electric cables and wires, as additives to extreme pressure lubricants, as supports for storage batteries, and as a coating in foundry use.

A partial list of occupations in which exposure may occur includes:

Cable coaters	Rubber workers
Condenser impregnators	Solvent workers
Electric equipment makers	Transformer workers
Insecticide workers	Wire coaters
Petroleum refinery workers	Wood preservers
Plasticizer makers	

PERMISSIBLE EXPOSURE LIMITS

The Federal standards are:

Trichloronaphthalene	5.0 mg/m^3	Set I
Tetrachloronaphthalene	2 mg/m^3	Set I
Pentachloronaphthalene	0.5 mg/m^3	Set G
Hexachloronaphthalene	0.2 mg/m^3	Set H

ROUTES OF ENTRY

Inhalation of fumes and percutaneous absorption of liquid.

HARMFUL EFFECTS

Local—

Chronic exposure to chlorinated naphthalenes can cause chloracne, which consists of simple erythematous eruptions with pustules, papules, and comedones. Cysts may develop due to plugging of the sebaceous gland orifices.

Systemic—

Cases of systemic poisoning are few in number and they may occur without the development of chloracne.

It is believed that chloracne develops from skin contact and inhalation of fumes, while systemic effects result primarily from inhalation of fumes. Symptoms of poisoning may include headaches, fatigue, vertigo, and anorexia. Jaundice may occur from liver damage. Highly chlorinated naphthalenes seem to be more toxic than those chlorinated naphthalenes with a lower degree of substitution.

MEDICAL SURVEILLANCE

Preplacement and periodic examinations should be concerned particularly with skin lesions such as chloracne and with liver function.

SPECIAL TESTS

None are in common use.

PERSONAL PROTECTIVE METHODS

Skin contact should be avoided whenever possible. Barrier creams, protective clothing, and good personal hygiene are all good preventive measures. Use of respirators in areas of vapor concentration is advised.

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AROMATIC AMINES

The aromatic amines are aromatic hydrocarbons in which at least one hydrogen atom has been replaced by an amino ($-NH_2$) group. The hydrogen atoms in the amino group may be replaced by aryl or alkyl groups, giving rise to secondary and tertiary amino compounds. The aromatic amines are infrequently formed in nature although they do occur, e.g., anthranilic acid esters in grapes. They are generally synthesized by nitration of the aromatic hydrocarbon with subsequent reduction to the amine; an alternate method is by reaction of ammonia and a chloro- or hydroxy-hydrocarbon. Their most important uses are as intermediates in the manufacture of dyestuffs and pigments; however, they are also used in the chemical, textile, rubber, dyeing, paper, and other industries.

Most of the aromatic amines in the free base form are readily absorbed through the skin in addition to the respiratory route. The amino salts have a lower lipid solubility and, therefore, a lower amount of skin absorption. The two major toxic effects of these compounds are methemoglobinemia and cancer of the urinary tract. Other effects may be hematuria, cystitis, anemia, and skin sensitization.

Several of the aromatic amines have been shown to be carcinogenic in humans or animals or both. Occupational tumors of the bladder were recognized in the dyestuff industry as early as 1895. The most common site of cancer is the bladder, but cancer of the pelvis, ureter, kidney, and urethra do occur. It is thought that bladder cancer results from the presence of an active metabolite(s) of the amino compound in the urine, which acts on the bladder epithelium. Several of these metabolites have been identified and have been shown to have carcinogenic properties by implantation in mouse bladders. Man and the dog seem to be more sus-

ceptible to bladder tumors, suggesting a similarity in metabolism of the aromatic amino compounds.

The minimum exposure which produces cancer is not known. There are documented cases of tumors with exposures of less than one year; however, the latent period from first exposure to the development of tumors is usually long and ranges from 4 to over 40 years, with a mean of about 20 years. Bladder tumors are also relatively common in unexposed populations, and the incidence is considerably increased in heavy smokers. They are more common in older age males. It is unknown whether smoking plus exposure to a bladder carcinogen would be synergistic, but this seems possible, e.g., asbestos and smoking.

Clinically, occupationally induced bladder tumors are indistinguishable from those found in the general population; however, they generally occur at an earlier age than usual. These tumors may range from the extremes of benign papillomas to infiltrating carcinomas. Severe or fatal complications which may arise from papillomas are local spreading tumors, severe hemorrhage, and infection of the bladder and kidney.

Hematuria often does not appear until the tumor(s) is inoperable. Micro-examination of the urine is not specific, but routine cystoscopy is a reliable indicator of tumors at an early stage. Exfoliative cytology of urinary sediment using the stained smear method of Papanicolau permits early differentiation of malignant neoplasms and benign papillomas from normal tissue. Those individuals who give a positive test should be examined by cystoscopy and followed indefinitely. Renal pelvis, ureteric, and urethral tumors can also be detected by cytodiagnosis.

Because there may be significant skin absorption of the aromatic amines, protective clothing and polyvinyl chloride or rubber gloves should be worn, and there should be adequate wash and change facilities. Workers exposed to carcinogens should have a complete change of work clothes in addition to protective clothing. The recommended means of control of carcinogenic compounds is by engineering methods aimed at zero exposure levels and a program of periodic medical surveillance.

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2-ACETYLAMINOFLUORENE

DESCRIPTION

$CCH_2C_6H_4C_6H_3NHCOCH_3$, 2-Acetylaminofluorene, is a tan crystalline solid.

SYNONYMS

2-acetaminofluorene, N-acetylaminophenathrene, N-2-fluorenylacetamide.

POTENTIAL OCCUPATIONAL EXPOSURES

Very little 2-acetylaminofluorene is produced. It is used primarily for cancer research purposes. It was patented as a pesticide, but was never used for this purpose. Thus, occupations in which exposure may occur are those in areas of research.

PERMISSIBLE EXPOSURE LIMITS

2-Acetylaminofluorene is included in the Federal standard for carcinogens; all contact with it should be avoided.

ROUTES OF ENTRY

Probably by inhalation and percutaneous absorption.

HARMFUL EFFECTS

Local—

Unknown.

Systemic—

2-Acetylaminofluorene's carcinogenic activity was first discovered in rats in which it produced nodular hyperplasia and cancer consistently in the bladder, kidney, pelvis, liver, and pancreas by ingestion. Later feeding experiments in dogs demonstrated bladder and liver tumors. Guinea pigs appear resistant to its carcinogenic effects. No human effects have been reported.

MEDICAL SURVEILLANCE

Preplacement and periodic examinations should include history of other exposure to carcinogens, smoking history, family history, alcohol, and medications. The skin, respiratory tract, kidney, bladder, and liver should be evaluated for possible effects. Sputum and bladder cytology should be performed. Fetal effects may occur.

The scope and frequency of medical surveillance examinations can be related to the hazard, which probably is greater among research chemists or those involved in animal inhalation studies.

SPECIAL TESTS

None in common use, although urinary metabolites are known.

PERSONAL PROTECTIVE METHODS

Personal protective methods are designed to supplement engineering controls and to prevent all skin or inhalation exposure.

Full body protective clothing and gloves may be required. Those employed in handling operations should be provided with fullface, supplied air respirators of continuous flow or pressure demand type. On exit from a regulated area, employees should shower and change into

street clothes; leaving their protective clothing and equipment at the point of exit to be placed in impervious containers at the end of the work shift for decontamination or disposal. Effective methods should be used for decontamination and changing of clothes and gloves.

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AMINODIPHENYL

DESCRIPTION

$C_6H_5H_6H_4NH_2NH_2$, 4-aminodiphenyl, is a yellowish brown crystal.

SYNONYMS

Biphenyline, p-phenylaniline, xenylamine, 4-aminobiphenyl, 4-biphenylamine, p-aminobiphenyl, p-aminodiphenyl, p-biphenylamine.

POTENTIAL OCCUPATIONAL EXPOSURES

It is no longer manufactured commercially and is only used for research purposes. 4-Aminodiphenyl was formerly used as a rubber antioxidant and as a dye intermediate.

A partial list of occupations in which exposure may occur includes:

Diphenylamine workers
Research workers

PERMISSIBLE EXPOSURE LIMITS

4-Aminodiphenyl is included in the Federal standards for carcinogens; all contact with it should be avoided.

ROUTES OF ENTRY

Inhalation and percutaneous absorption.

HARMFUL EFFECTS

Local—

None reported.

Systemic—

4-Aminodiphenyl is a known human bladder carcinogen. An exposure of only 133 days has been reported to have ultimately resulted in a bladder tumor. The latent period is generally from 15 to 35 years. Acute exposure produces headaches, lethargy, cyanosis, urinary burning, and hematuria. Cystoscopy reveals diffuse hyperemia, edema, and frank slough.

MEDICAL SURVEILLANCE

Placement and periodic examinations should include an evaluation of exposure to other carcinogens; use of alcohol, smoking, and medications; and family history. Special attention should be given on a regular basis to urine sediment and cytology. If red cells or positive smears are seen, cystoscopy should be done at once. The general health of exposed persons should also be evaluated in periodic examinations.

SPECIAL TESTS

None commonly used. One urinary metabolite is 3-amino-4-hydroxydiphenyl.

PERSONAL PROTECTIVE METHODS

These are designed to supplement engineering controls and to prevent all skin or respiratory contact. Full body protective clothing and gloves should be used by those employed in handling operations. Full-face, supplied air respirators of continuous flow or pressure demand type should also be used. On exit from a regulated area, employees should shower and change into street clothes, leaving their clothing and equipment at the point of exit to be placed in impervious containers at the end of the work shift for decontamination or disposal. Effective methods should be used to clean and decontaminate gloves and clothing.

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ANILINE

DESCRIPTION

$C_6H_5NH_2$, aniline, is a clear, colorless, oily liquid with a characteristic odor.

SYNONYMS

Aminobenzene, phenylamine, aniline oil, aminophen, arylamine.

POTENTIAL OCCUPATIONAL EXPOSURES

Aniline is widely used as an intermediate in the synthesis of dyestuffs. It is also used in the manufacture of rubber accelerators and antioxidants, pharmaceuticals, marking inks, tetryl, optical whitening agents, photographic developers, resins, varnishes, perfumes, shoe polishes, and many organic chemicals.

A partial list of occupations in which exposure may occur includes:

Acetanilide workers	Perfume makers
Bromide makers	Photographic chemical makers
Coal tar workers	Plastic workers
Disinfectant makers	Printers
Dye workers	Rocket fuel makers
Ink makers	Rubber workers
Leather workers	Tetryl makers
Lithographers	Varnish workers
Nitraniline workers	

PERMISSIBLE EXPOSURE LIMITS

The Federal standard is 5 ppm (19 mg/m^3).

ROUTES OF ENTRY

Inhalation of vapors; percutaneous absorption of liquid and vapor.

HARMFUL EFFECTS

Local—

Liquid aniline is mildly irritating to the eyes and may cause corneal damage.

Systemic—

Absorption of aniline, whether from inhalation of the vapor or from skin absorption of the liquid, causes anoxia due to the formation of methemoglobin. Moderate exposure may cause only cyanosis. As oxygen deficiency increases, the cyanosis may be associated with headache, weakness, irritability, drowsiness, dyspnea, and unconsciousness. If treatment is not given promptly, death can occur. The development of intravascular hemolysis and anemia due to aniline-induced methemoglobinemia has been postulated, but neither is observed often in industrial practice, despite careful study of numerous cases.

MEDICAL SURVEILLANCE

Preplacement and periodic physical examinations should be performed on all employees working in aniline exposure areas. These should include a work history to elicit information on all past exposures to aniline, other aromatic amines, and nitro compounds known to cause chemical cyanosis, and the clinical history of any occurrence of chemical cyanosis; a personal history to elicit alcohol drinking habits; and general physical examination with particular reference to the cardiovascular system. Persons with impaired cardiovascular status may be at greater risk from the consequences of chemical cyanosis. A preplacement complete blood count and methemoglobin estimation should be performed as baseline levels, also follow-up studies including periodic blood counts and hematocrits.

SPECIAL TESTS

Methemoglobin levels, and other abnormal hemoglobins, and/or

urine para-aminophenols, and other aniline metabolites,have been used for biologic monitoring for occupational aniline exposure.

PERSONAL PROTECTIVE METHODS

In areas of vapor concentration, the use of respirators alone is not sufficient; skin protection by protective clothing should be provided even though there is no skin contact with liquid aniline. Butyl rubber protective clothing is reportedly superior to other materials. In severe exposure situations, complete body protection has been employed, consisting of air-conditioned suit with air supplied helmet and cape. Personal hygiene practices including prompt removal of clothing which has absorbed aniline, thorough showering after work and before changing to street clothes, and clean working clothes daily are essential.

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BENZIDINE AND ITS SALTS

DESCRIPTION

$NH_2C_6H_4C_6H_4NH_2$, benzidine, is a crystalline solid with a significant vapor pressure. The salts are less volatile, but tend to be dusty.

SYNONYMS

4,4'-Biphenyldiamine, para-diaminodiphenyl, 4,4'-diaminobiphenyl, 4,4'-diphenylenediamine, benzidine base.

POTENTIAL OCCUPATIONAL EXPOSURES

Benzidine is used primarily in the manufacture of azo dyestuffs; there are over 250 of these produced. Other uses, including some which may have been discontinued, are in the rubber industry as a hardener, in the manufacture of plastic films, for detection of occult blood in feces, urine, and body fluids, in the detection of H_2O_2 in milk, in the production of security paper, and as a laboratory reagent in determining HCN, sulfate, nicotine, and certain sugars. No substitute has been found for its use in dyes.

A partial list of occupations in which exposure may occur includes:

- Biochemists
- Dye workers
- Medical laboratory workers
- Organic chemical synthesizers
- Plastic workers
- Rubber workers
- Wood chemists

PERMISSIBLE EXPOSURE LIMITS

Benzidine and its salts are included in a Federal standard for carcinogens; all contact with them should be avoided.

ROUTES OF ENTRY

Inhalation, percutaneous absorption, and ingestion of dust.

HARMFUL EFFECTS

Local—

Contact dermatitis due to primary irritation or sensitization has been reported.

Systemic—

Benzidine is a known human urinary tract carcinogen with an average latent period of 16 years. The first symptoms of bladder cancer usually are hematuria, frequency of urination, or pain.

MEDICAL SURVEILLANCE

Placement and periodic examinations should include an evaluation of exposure to other carcinogens; use of alcohol, smoking, and medications; and family history. Special attention should be given on a regular basis to urine sediment and cytology. If red cells or positive smears are seen, cystoscopy should be done at once. The general health of exposed persons should also be evaluated in periodic examinations.

SPECIAL TESTS

None in common use although several metabolites are known.

PERSONAL PROTECTIVE METHODS

These are designed to supplement engineering controls and to prevent all skin or respiratory contact. Full body protective clothing and gloves should also be used. On exit from a regulated area employees should shower and change into street clothes, leaving their protective clothing and equipment at the point of exit to be placed in impervious containers at the end of the work shift for decontamination or disposal. Effective methods should be used to clean and decontaminate gloves and clothing.

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3,3'-DICHLOROBENZIDINE AND ITS SALTS

DESCRIPTION

$C_6H_3ClNH_2C_6H_3ClNH_2$, 3,3'-dichlorobenzidine, is a gray or purple crystalline solid.

SYNONYMS

4,4'-Diamino-3,3'-dichlorobiphenyl, 3,3'-dichlorobiphenyl-4,4'-diamine, 3,3'-dichloro-4,4-biphenyldiamine.

POTENTIAL OCCUPATIONAL EXPOSURES

The major uses of dichlorobenzidine are in the manufacture of pigments for printing ink, textiles, plastics, and crayons and as a curing agent for solid urethane plastics. There are no substitutes for many of its uses.

A partial list of occupations in which exposure may occur includes:

Pigment makers
Polyurethane workers

PERMISSIBLE EXPOSURE LIMITS

3,3'-Dichlorobenzidine and its salts are included in a Federal standard for carcinogens; all contact with it should be avoided.

ROUTES OF ENTRY

Inhalation and probably percutaneous absorption.

HARMFUL EFFECTS

Local—

May cause allergic skin reactions.

Systemic—

3,3'-Dichlorobenzidine was shown to be a potent carcinogen in rats and mice in feeding and injection experiments, but no bladder tumors were produced. However, no cases of human tumors have been observed in epidemiologic studies of exposure to the pure compound.

MEDICAL SURVEILLANCE

Preplacement and periodic examinations should include history of exposure to other carcinogens, smoking, alcohol, medication, and family history. The skin, lung, kidney, bladder, and liver should be evaluated; sputum or urinary cytology may be helpful.

SPECIAL TESTS

None in common use.

PERSONAL PROTECTIVE METHODS

These are designed to supplement engineering controls and to prevent all skin or respiratory contact. Full body protective clothing and gloves should be used by those employed in handling operations. Fullface supplied air respirators of continuous flow or pressure demand type should also be used. On exit from a regulated area, employees should shower and change into street clothes, leaving their protective clothing and equipment at the point of exit to be placed in impervious containers at the end of the work shift for decontamination or disposal. Effective methods should be used to clean and decontaminate gloves and clothing.

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4-DIMETHYLAMINOAZOBENZENE

DESCRIPTION

$C_6H_5NNC_6H_4N(CH_3)_2$, 4-dimethylaminoazobenzene, is a flaky yellow crystal.

SYNONYMS

Aniline-N,N-dimethyl-p(phenylazo), benzeneazo dimethylaniline, fat yellow, oil yellow, butter yellow, methyl yellow.

POTENTIAL OCCUPATIONAL EXPOSURES

4-Dimethylaminoazobenzene is only used for research purposes. It was formerly used as a dye,but has been substituted by diethylaminoazobenzene. It was also formerly used for coloring margarine and butter.

A partial list of occupations in which exposure may occur includes:

Research workers

PERMISSIBLE EXPOSURE LIMITS

4-Dimethylaminoazobenzene is included in the Federal standard for carcinogens; all contact with it should be avoided.

ROUTES OF ENTRY

Probably inhalation and percutaneous absorption.

HARMFUL EFFECTS

Local—

Unknown.

Systemic—

Cancer of the liver has been produced in rats and mice in feeding experiments. No human effects have been reported.

MEDICAL SURVEILLANCE

Preplacement and periodic examinations should include a history of exposure to other carcinogens; use of alcohol, smoking, and medications; and family history. Special attention should be given to liver size and liver function tests.

SPECIAL TESTS

None commonly used.

PERSONAL PROTECTIVE METHODS

These are designed to supplement engineering controls and to prevent all contact with skin and the respiratory tract. Protective clothing and gloves should be provided, and also appropriate type dust or supplied air respirators. On exit from a regulated area, employees should shower and change into street clothes, leaving their clothes at the point of exit, to be placed in impervious containers at the end of the work shift for decontamination or disposal.

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4,4'-METHYLENEBIS(2-CHLOROANILINE)

DESCRIPTION

$CH_2(C_6H_4ClNH_2)_2$, 4,4'-methylenebis (2-chloroaniline) or moca, is a yellow to light gray-tan pellet and is also available in liquid form.

SYNONYMS

Moca, 4,4'-diamino-3,3'-dichlorodiphenylmethane, 4,4'-methylene-2,2-dichloroaniline.

POTENTIAL OCCUPATIONAL EXPOSURES

Moca is primarily used in the production of solid elastomeric parts. Other uses are as a curing agent for epoxy resins and in the manufacture of cross-linked urethane foams used in automobile seats and safety padded dashboards; it is also used in the manufacture of gun mounts, jet engine turbine blades, radar systems, and components in home appliances.

A partial list of occupations in which exposure may occur includes:

Elastomer makers
Epoxy resin workers
Polyurethane foam workers

PERMISSIBLE EXPOSURE LIMITS

Moca is included in the Federal standard for carcinogens; all contact with it should be avoided.

ROUTES OF ENTRY

Inhalation; percutaneous absorption.

HARMFUL EFFECTS

Local—

None reported.

Systemic—

Feeding experiments with rats produced liver and lung cancer. No tumors were found in experiments with dogs. No tumors or other ill-

ness have been reported from chronic exposure in man except a mild cystitis which subsided within a week.

MEDICAL SURVEILLANCE

Preplacement and periodic examinations should include a history of exposure to other carcinogens, alcohol and smoking habits, use of medications, and family history. Special attention should be given to liver size and function and to any changes in lung symptoms or X-rays.

SPECIAL TESTS

None commonly used.

PERSONAL PROTECTIVE METHODS

These are designed to supplement engineering controls and to prevent all contact with skin and the respiratory tract. Protective clothing and gloves should be provided, and also appropriate type dust or supplied air respirators. On exit from a regulated area, employees should shower and change into street clothes, leaving the protective clothing and equipment at the point of exit, to be placed in impervious containers at the end of the work shift for decontamination or disposal.

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alpha-NAPHTHYLAMINE

DESCRIPTION

$C_{10}H_7NH_2$, alpha-naphthylamine, exists as white needlelike crystals which turn red on exposure to air.

SYNONYMS

1-Aminonaphthalene, naphthalidam, naphthalidine.

POTENTIAL OCCUPATIONAL EXPOSURES

alpha-Naphthylamine is used in the manufacture of dyes, condensation colors, and rubber, and in the synthesis of many chemicals such as alpha-naphthol, sodium naphthionate o-naphthionic acid, Nevile's acid, Winther's acid, sulfonated naphthylamines, alpha-naphthylthiouria (a rodenticide), and N-phenyl-alpha-naphthylamine.

A partial list of occupations in which exposure may occur includes:

Dye makers
Chemical synthesizers
Rubber workers

PERMISSIBLE EXPOSURE LIMITS

alpha-Naphthylamine is included in the Federal standard for carcinogens; all contact with it should be avoided.

ROUTES OF ENTRY

Inhalation and percutaneous absorption.

HARMFUL EFFECTS

Local—

None reported.

Systemic—

It has not been established whether alpha-naphthylamine is a human carcinogen per se or is associated with an excess of bladder cancer due to its beta-naphthylamine content. Workers exposed to alpha-naphthylamine developed bladder tumors. The mean latent period was 22 years compared to 16 years for beta-naphthylamine. One animal experiment demonstrated papillomata, but these results have never been confirmed.

MEDICAL SURVEILLANCE

Placement and periodic examinations should include an evaluation of exposure to other carcinogens; use of alcohol, smoking, and medications; and family history. Special attention should be given on a regular basis to urine sediment and cytology. If red cells or positive smears are seen, cystoscopy should be done at once. The general health of exposed persons should also be evaluated in periodic examinations.

SPECIAL TESTS

None commonly used. Some metabolites are known.

PERSONAL PROTECTIVE METHODS

These are designed to supplement engineering controls and to prevent all skin or respiratory contact. Full body protective clothing and gloves should be used by those employed in handling operations. Full-face, supplied air respirators of continuous flow or pressure demand type should also be used. On exit from a regulated area, employees should shower and change into street clothes, leaving their protective clothing and equipment at the point of exit to be placed in impervious containers at the end of the work shift for decontamination or disposal. Effective methods should be used to clean and decontaminate gloves and clothing. Showers should be taken prior to dressing in street clothes.

beta-NAPHTHYLAMINE

DESCRIPTION

$C_{10}H_7NH_2$, beta-Naphthylamine, is a white to reddish crystal.

SYNONYMS

2-Naphthylamine, 2-aminonaphthalene.

POTENTIAL OCCUPATIONAL EXPOSURES

beta-Naphthylamine is presently used only for research purposes. It is present as an impurity in alpha-naphthylamine. It was widely used in the manufacture of dyestuffs, as an antioxidant for rubber, and in rubber coated cables.

A partial list of occupations in which exposure may occur includes:
beta-Naphthylamine workers
Research workers

PERMISSIBLE EXPOSURE LIMITS

beta-Naphthylamine is included in the Federal standard for carcinogens; all contact with it should be avoided.

ROUTES OF ENTRY

Inhalation and percutaneous absorption.

HARMFUL EFFECTS

Local—

beta-Naphthylamine is mildly irritating to the skin and has produced contact dermatitis.

Systemic—

beta-Naphthylamine is a known human bladder carcinogen with a latent period of about 16 years. The symptoms are frequent urination, dysuria, and hematuria. Acute poisoning leads to methemoglobinemia or acute hemorrhagic cystitis.

MEDICAL SURVEILLANCE

Preplacement and periodic examinations should include an evaluation of exposure to other carcinogens; use of alcohol, smoking, and medications; and family history. Special attention should be given on a regular basis to urine sediment and cytology. If red cells or positive smears are seen, cystoscopy should be done at once. The general health of exposed persons should also be evaluated in periodic examinations.

SPECIAL TESTS

None in common use; some metabolites are known.

PERSONAL PROTECTIVE METHODS

These are designed to supplement engineering controls and to prevent all skin or respiratory contact. Full body protective clothing and gloves should be used by those employed in handling operations. Full-face, supplied air respirators of continuous flow or pressure demand type should also be used. On exit from a regulated area, employees should shower and change into street clothes, leaving their clothing and equipment at the point of exit to be placed in impervious containers at the end of the work shift for decontamination or disposal. Effective methods should be used to clean and decontaminate gloves and clothing. Showers should be taken prior to dressing in street clothes.

NITRO COMPOUNDS

The aliphatic nitro compounds are characterized by the $-C-NO_2$ structure. Closely related chemicals are the alkyl nitrites ($-C-O-NO$), alkyl nitrates ($-C-O-NO_2$), and chloronitroparaffins (e.g., CCl_2NO_2). All differ significantly in their chemical and toxicological characteristics.

The aromatic nitro compounds, in which a nitro group is substituted directly on a benzene ring, are a more homogeneous group. Most of them can be produced by nitration of the aromatic. They are widely used, especially in explosive and dyestuff manufacture. Aromatic nitro compounds rapidly penetrate the skin, and this may be the major route of absorption. In acute exposures, they produce cyanosis and in chronic exposures, anemia. Local irritation and liver damage are also common. A portion of the absorbed dose is excreted in the urine unchanged; however the major portion is first metabolized to aminophenol derivatives before excretion. Many colorimetric tests are available for detecting the parent compounds or metabolites in the urine.

Other clinical tests which may be of value are urinalysis, blood chemistry, and blood analysis for anemia, methemoglobin, and Heinz bodies. Physical examinations are an important aspect of prevention. Individuals with cardiovascular, renal, hepatic, or respiratory diseases, blood dyscrasia, allergies, or chronic alcoholism may be at increased risk from exposure to aromatic nitro compounds.

Work practices should include protective clothing made of butyl rubber and emphasis on personal hygiene.

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DINITROBENZENE

DESCRIPTION

$C_6H_4(NO_2)_2$, dinitrobenzene, may exist in three isomers; the meta-form is the most widely used.

SYNONYMS

Dinitrobenzol.

POTENTIAL OCCUPATIONAL EXPOSURES

Dinitrobenzene is used in the synthesis of dyestuffs, dyestuff intermediates, and explosives and in celluloid production.

A partial list of occupations in which exposure may occur includes:

- Celluloid makers
- Dye makers
- Explosive workers
- Organic chemical synthesizers

PERMISSIBLE EXPOSURE LIMITS

The Federal standard for all isomers of dinitrobenzene is 1 mg/m³.

ROUTES OF ENTRY

Inhalation and percutaneous absorption of liquid.

HARMFUL EFFECTS

Local—

Exposure to dinitrobenzene may produce yellowish coloration of the skin, eyes, and hair.

Systemic—

Exposure to any isomer of dinitrobenzene may produce methemoglobinemia, symptoms of which are headache, irritability, dizziness, weakness, nausea, vomiting, dyspnea, drowsiness, and unconsciousness. If treatment is not given promptly, death may occur. Consuming alcohol, exposure to sunlight, or hot baths may make symptoms worse. Dinitrobenzene may also cause a bitter almond taste or burning sensation in the mouth, dry throat, and thirst. Reduced vision may occur. In addition liver damage, hearing loss, and ringing of the ears may be produced. Repeated or prolonged exposure may cause anemia.

MEDICAL SURVEILLANCE

Preemployment and periodic examinations should be concerned particularly with a history of blood dyscrasias, reactions to medications, alcohol intake, eye disease, and skin and cardiovascular status. Liver and renal functions should be evaluated periodically as well as blood and general health.

SPECIAL TESTS

Methemoglobin levels should be followed until normal in all cases of suspected cyanosis. Dinitrobenzene can be determined in the urine; levels greater than 25 mg/liter may indicate significant absorption.

PERSONAL PROTECTIVE METHODS

Dinitrobenzene is readily absorbed through intact skin and its vapors are highly toxic. Protective clothing impervious to the liquid should be worn in areas where the likelihood of splash or spill exists. When splash or spill occurs on ordinary work clothes, they should be removed immediately and the area washed thoroughly. In areas of elevated vapor concentrations fullface masks with organic vapor canisters or air supplied respirators with fullface piece should be used. Daily changes of work clothing and mandatory showering at the end of each shift before changing to street clothes should be enforced.

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DINITRO-O-CRESOL

DESCRIPTION

$CH_3C_6H_2(NO_2)_2OH$, dinitro-o-cresol, exists in 9 isomeric forms of which 3,5-dinitro-o-cresol is the most important commercially. It is a yellow crystalline solid.

SYNONYMS

DNOC; 4,6-Dinitro-o-cresol is also known as 3,5-dinitro-o-cresol, 2-methyl-4,6-dinitrophenol, 3,5-dinitro-2-hydroxytoluene.

POTENTIAL OCCUPATIONAL EXPOSURES

DNOC is widely used in agriculture as a herbicide and pesticide; it is also used in the dyestuff industry.

A partial list of occupations in which exposure may occur includes:

Dye makers
Herbicide workers
Pesticide workers

PERMISSIBLE EXPOSURE LIMITS

The Federal standard for all isomers of DNOC is 0.2 mg/m³.

ROUTES OF ENTRY

Inhalation and percutaneous absorption.

HARMFUL EFFECTS

Local—

None reported except for staining of skin and hair.

Systemic—

DNOC blocks the formation of high energy phosphate compounds, and the energy from oxidative metabolism is liberated as heat. Early symptoms of intoxication by inhalation or skin absorption are elevation of the basal metabolic rate and rise in temperature accompanied by fatigue, excessive sweating, unusual thirst, and loss of weight. The clinical picture resembles in part a thyroid crisis. Weakness, fatigue, increased respiratory rate, tachycardia, and fever may lead to rapid deterioration and death. Bilateral cataracts have been seen following oral ingestion for therapeutic purposes.

These have not been seen during industrial or agricultural use.

MEDICAL SURVEILLANCE

Consider eyes, thyroid, and cardiovascular system, as well as general health.

SPECIAL TESTS

None commonly used.

PERSONAL PROTECTIVE METHODS

Since dinitro-o-cresol is used extensively in agriculture as well as

industry, worker education to the toxic properties of the chemical are necessary. Where there is a possibility of skin contamination or vapor inhalation, full protection should be provided. Impervious protective clothing and fullface masks with organic vapor canisters or air supplied respirators are advised. A clean set of work clothes daily, and showers following each shift before change to street clothes are essential.

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DINITROPHENOL

DESCRIPTION

There are six isomers of dinitrophenol of which 2,4-dinitrophenol is the most important industrially. It is an explosive, yellow crystalline solid.

SYNONYMS

DNP.

POTENTIAL OCCUPATIONAL EXPOSURES

2,4-DNP is used in the manufacturing of dyestuff intermediates, wood preservatives, pesticides, herbicides, explosives, chemical indicators, photograph developers, and also in chemical synthesis.

A partial list of occupations in which exposure may occur includes:

- Chemical indicator makers
- Dye makers
- Explosive workers
- Herbicide workers
- Organic chemical synthesizers
- Photographic developer makers
- Wood preservative workers

PERMISSIBLE EXPOSURE LIMITS

There is no Federal standard for DNP. A useful guideline of 0.2 mg/m^3 is based on data for dinitro-o-cresol.

ROUTES OF ENTRY

Percutaneous absorption and inhalation of dust and vapors.

HARMFUL EFFECTS

Local—

DNP causes yellow staining of exposed skin. Dermatitis may be due to either primary irritation or allergic sensitivity.

Systemic—

The isomers differ in their toxic effects. In general, DNP disrupts oxidative phosphorylation (as in the case of DNOC) which results in increased metabolism, oxygen consumption, and heat production. Acute intoxication is characterized by sudden onset of fatigue, thirst, sweating, and oppression of the chest. There is rapid respiration, tachycardia, and a rise in body temperature. In less severe poisoning, the symptoms are nausea, vomiting, anorexia, weakness, dizziness, vertigo, headache, and sweating. The liver may be sensitive to pressure, and there may also be jaundice. DNP poisoning is more severe in warm environments. If not fatal, the effects are rapidly and completely reversible. Chronic exposure results in kidney and liver damage and cataract formation. Occasional hypersensitivity reactions, e.g., neutropenia, skin rashes, peripheral neuritis, have been seen after oral use.

MEDICAL SURVEILLANCE

Consider skin, eyes, thyroid, blood, central nervous system, liver and kidney function, as well as general health in preplacement and periodic examinations.

SPECIAL TESTS

Can be measured in urine as such or as an aminophenol derivative.

PERSONAL PROTECTIVE METHODS

Because of its wide use in agriculture, lumbering, photography, as well as in the petrochemical industry, worker education to the toxic properties of dinitrophenol are important. Impervious protective clothing, fullface masks with organic vapor canisters or air supplied respirators are necessary in areas of high concentration of dust or vapor. Spills and splashes that contaminate clothing require the worker to immediately change clothes and wash the area thoroughly. Workers should have clean work clothes on every shift and should be required to shower prior to changing to street clothing.

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Gosselin, R. E., H. C. Hodge, R. P. Smith, and M. N. Gleason. 1976. Clinical Toxicology of Commercial Products, 4th ed. Williams and Wilkins Co. Baltimore.

DINITROTOLUENE

DESCRIPTION

Six isomers of DNT exist, the most important being 2,4-dinitro-1-toluene.

SYNONYMS

Dinitrotoluol, DNT.

POTENTIAL OCCUPATIONAL EXPOSURES

DNT is used in the manufacture of explosives and dyes in organic synthesis, e.g., trinitrotoluene.

A partial list of occupations in which exposure may occur includes:

Dye makers
Explosive workers
Organic chemical synthesizers

PERMISSIBLE EXPOSURE LIMITS

The Federal standard is 1.5 mg/m^3.

ROUTES OF ENTRY

Inhalation of vapor and percutaneous absorption of liquid.

HARMFUL EFFECTS

Local—

None.

Systemic—

The effects from exposure to dinitrotoluene are caused by its capacity to produce anoxia due to the formation of methemoglobin. Cyanosis may occur with headache, irritability, dizziness, weakness, nausea, vomiting, dyspnea, drowsiness, and unconsciousness. If treatment is not given promptly, death may occur. The onset of symptoms may be delayed. The ingestion of alcohol may cause increased susceptibility. Repeated or prolonged exposure may cause anemia.

MEDICAL SURVEILLANCE

Preemployment and periodic examinations should be concerned particularly with a history of blood dyscrasias, reactions to medications, alcohol intake, eye disease, skin, and cardiovascular status. Liver and renal functions should be evaluated periodically as well as blood and general health.

SPECIAL TESTS

None commonly used. Forms a blue color with alcoholic NaOH.

PERSONAL PROTECTIVE METHODS

Liquid soaked clothing should be immediately removed and the skin area washed thoroughly. Impervious protective clothing should be provided if skin exposure to liquid is anticipated. In areas of elevated vapor concentration, fullface masks with organic vapor canisters or air-supplied respirators should be required.

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NITROBENZENE

DESCRIPTION

$C_6H_5NO_2$, nitrobenzene, is a pale yellow liquid whose odor resembles bitter almonds.

SYNONYMS

Nitrobenzol, oil of mirbane, oil of bitter almonds.

POTENTIAL OCCUPATIONAL EXPOSURES

Nitrobenzene is used in the manufacture of explosives and aniline dyes and as a solvent and intermediate. It is also used in shoe and floor polishes, leather dressings, and paint solvents, and to mask other unpleasant odors. Substitution reactions with nitrobenzene are used to form meta-derivatives.

A partial list of occupations in which exposure may occur includes:

- Aniline dye makers
- Explosive makers
- Organic chemical synthesizers
- Paint makers
- Polish makers

PERMISSIBLE EXPOSURE LIMITS

The Federal standard is 1 ppm (5 mg/m^3).

ROUTES OF ENTRY

Inhalation and percutaneous absorption of liquid.

HARMFUL EFFECTS

Local—

Nitrobenzene may cause irritation of the eyes.

Systemic—

There is a latent period of 1-4 hours before signs and symptoms appear. Nitrobenzene affects the central nervous system producing fatigue, headache, vertigo, vomiting, general weakness, and in some cases severe depression, unconsciousness, and coma. Nitrobenzene is a powerful methemoglobin former; cyanosis appears when methemoglobin reaches 15%. Sulfhemoglobin formation may also contribute to nitrobenzene toxicity. Chronic exposure may lead to spleen and liver damage, jaundice, liver impairments, and hemolytic icterus. Anemia and Heinz bodies in the red blood cells have also been observed. Alcohol ingestion may increase the toxic effects.

MEDICAL SURVEILLANCE

Preemployment and periodic examinations should be concerned particularly with a history of dyscrasias, reactions to medications, alcohol intake, eye disease, skin, and cardiovascular status. Liver and renal functions should be evaluated periodically, as well as blood and general health.

SPECIAL TESTS

Follow methemoglobin levels until normal in all cases of suspected cyanosis. The metabolites in urine, p-nitro and p-amino phenol, can be used as an evidence of exposure.

PERSONAL PROTECTIVE METHODS

Impervious protective clothing should be worn in areas where risk of splash or spill exists. When splashed or spilled on ordinary work clothes, the clothes should be removed at once and the skin area washed thoroughly. In areas of vapor concentration fullface masks with organic vapor canisters or air supplied respirators should be used. Clean work clothing should be supplied daily, and showering made mandatory after each shift before workers change to street clothes.

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Salmowa, J., J. Piotrowski, and U. Neuhorn. 1963. Evaluation of exposure to nitrobenzene. Absorption of nitrobenzene vapor through lungs and excretion of p-nitrophenol in urine. Brit. J. Ind. Med. 20:41.

4-NITROBIPHENYL

DESCRIPTION

$C_6H_5C_6H_4NO_2$,4-nitrobiphenyl, exists as yellow plates or needles.

SYNONYMS

4-Nitrodiphenyl, p-nitrobiphenyl, p-nitrodiphenyl, PNB.

POTENTIAL OCCUPATIONAL EXPOSURES

4-Nitrobiphenyl was formerly used in the synthesis of 4-aminodiphenyl. It is presently used only for research purposes; there are no commercial uses.

A partial list of occupations in which exposure may occur includes:
Research workers

PERMISSIBLE EXPOSURE LIMITS

4-Nitrobiphenyl was included in the Federal standard for carcinogens; all contact with it should be avoided.

ROUTES OF ENTRY

Inhalation and percutaneous absorption.

HARMFUL EFFECTS

Local—

None reported.

Systemic—

4-Nitrobiphenyl is considered to be a human carcinogen. This is based on the evidence that it will induce bladder tumors in dogs and that human cases of bladder cancer were reported from a mixed exposure to 4-aminodiphenyl and 4-nitrobiphenyl. These human cases were attributed to 4-aminodiphenyl because the information available at the time showed that it produced bladder tumors in dogs. 4-Amino biphenyl may be a metabolite.

MEDICAL SURVEILLANCE

Placement and periodic examinations should include an evaluation of exposure to other carcinogens, as well as an evaluation of smoking, of use of alcohol and medications, and of family history. Special attention should be given on a regular basis to urine sediment and cytology. If red cells or positive smears are seen, cystoscopy should be done at once. The general health of exposed persons should also be evaluated in periodic examinations.

SPECIAL TESTS

None commonly used. Can probably be determined in the urine as a metabolite.

PERSONAL PROTECTIVE METHODS

These are designed to supplement engineering controls and to prevent all skin or respiratory contact. Full body protective clothing and gloves should be used by those employed in handling operations. Full-face, supplied air respirators of continuous flow or pressure demand type should also be used. On exit from a regulated area, employees should shower and change into street clothes, leaving their protective clothing and equipment at the point of exit to be placed in impervious containers at the end of the work-shift for decontamination or disposal. Effective methods should be used to clean and decontaminate gloves and clothing.

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NITROGLYCERIN and ETHYLENE GLYCOL DINITRATE

DESCRIPTION

$C_3H_5(ONO_2)_3$, nitroglycerin.

$O_2NOCH_2OCH_2ONO_2$, ethylene glycol dinitrate.

Both are oily, yellow liquids and are highly explosive. They may be detonated by mechanical shock, heat, or spontaneous chemical reaction.

SYNONYMS

Nitroglycerin: nitroglycerol, glyceryl trinitrate, trinitroglycerol, glonoin, trinitrin.

Ethylene glycol dinitrate: nitroglycol, glycol dinitrate, ethylene dinitrate, EGDN.

POTENTIAL OCCUPATIONAL EXPOSURES

Although ethylene glycol dinitrate is an explosive in itself, it is primarily used to lower the freezing point of nitroglycerin; together these compounds are the major constituents of commercial dynamite, cordite, and blasting gelatin. Occupational exposure generally involves a mixture of the two compounds. Ethylene glycol dinitrate is 160 times more volatile than nitroglycerin. Nitroglycerin is also used as a pharmaceutical.

A partial list of occupations in which exposure may occur includes:

Drug makers
Explosive makers

PERMISSIBLE EXPOSURE LIMITS

The Federal standard for nitroglycerin is 0.2 ppm (2 mg/m^3). The standard for ethylene glycol dinitrate and/or nitroglycerin is 0.2 ppm (1 mg/m^3) as a ceiling value, and, at concentrations greater than 0.02 ppm, personal protection may be necessary to avoid headache. These levels should be reduced when the substance is also absorbed percutaneously.

ROUTES OF ENTRY

Inhalation of dust or vapor; ingestion of dust; percutaneous absorption.

HARMFUL EFFECTS

Local—

None reported.

Systemic—

Exposure to small amounts of ethylene glycol dinitrate and/or nitroglycerin by skin exposure, inhalation, or swallowing may cause severe throbbing headaches. With larger exposure, nausea, vomiting, cyanosis, palpitations of the heart, coma, cessation of breathing, and death may occur. A temporary tolerance to the headache may develop, but this is lost after a few days without exposure. On some occasions a worker may have anginal pains a few days after discontinuing repeated daily exposure.

MEDICAL SURVEILLANCE

Placement and periodic examinations should be concerned with central nervous system, blood, glaucoma, and especially history of alcoholism.

SPECIAL TESTS

None commonly used, but urinary and blood ethylene glycol dinitrate may be determined by gas chromatography.

PERSONAL PROTECTIVE METHODS

Both compounds are readily absorbed through the skin, lungs, and mucous membranes. It is, therefore, essential that adequate skin protection be provided for each worker: impervious clothing where liquids are likely to contaminate and full body clothing where dust creates the problem. All clothing should be discarded at the end of the shift and clean work clothing provided each day. Showers should be taken at the end of each shift and prior to changing to street clothing. In case of spill or splash that contaminates work clothing, the clothes should be changed at once and the skin area washed thoroughly. Masks of the dust type or organic vapor canister type may be necessary in areas of concentration of dust or vapors.

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NITROPARAFFINS

DESCRIPTION

Nitroparaffiins are characterized by a $-C-NO_2$ group and may be either mono- or poly-substituted. Only certain mononitroparaffins are included in this section: nitromethane (CH_3NO_2), nitroethane ($C_2H_5-NO_2$), 1-nitropropane ($C_3H_7-NO_2$), and 2-nitropropane ($CH_3-CH(NO_2)-CH_3$. All of these are colorless liquids. Other mononitroparaffins are not commonly used, and use of the polynitroparaffins is limited almost entirely to fuels and fuel additives.

SYNONYMS

None.

POTENTIAL OCCUPATIONAL EXPOSURES

Nitroparaffins are used as solvents for cellulose esters, vinyl copolymer, and other resins, oils, fats, waxes, and dyes. They are also used in various coating materials such as shellac, synthetic and processed rubber, paint and varnish removers, alkyl resins, and other high polymer coatings, and also in organic synthesis.

A partial list of occupations in which exposure may occur includes:

- Cellulose workers
- Dye makers
- Fat processors
- Organic chemical synthesizers
- Plastic makers
- Resin makers
- Rubber makers
- Stainers
- Wax makers

PERMISSIBLE EXPOSURE LIMITS

The Federal standards for these substances are: nitromethane 100 ppm (250 mg/m^3), nitroethane 100 ppm (310 mg/m^3), 1-nitropropane 25 ppm (90 mg/m^3), and 2-nitropropane 25 ppm (90 mg/m^3).

ROUTE OF ENTRY

Inhalation of vapor.

HARMFUL EFFECTS

Local—

The nitroparaffins are irritants to the eyes and upper respiratory tract. There may be slight skin irritation due to solvent drying of skin.

Systemic—

Only one report of occupational illness from nitroparaffins has been reported. The workers were exposed to 20-45 ppm of 2-nitropropane and complained of anorexia, nausea, vomiting diarrhea, and occipital headache. Animal experiments indicate that high concentrations of nitroparaffins may produce light narcosis and central nervous system irritation. The lethal dose is generally lower than that producing significant narcosis. Liver and kidney damage have been observed in animals at lethal concentrations. Nitroparaffins release nitrate in vivo; however, methemoglobinemia and Heinz bodies have only been observed with 2-nitropropane. Experimental evidence also indicates that the toxicity of nitroparaffins increases with the size of the molecule.

MEDICAL SURVEILLANCE

Based on animal data, preplacement and periodic examination should consider respiratory and central nervous system effects as well as liver and kidney function.

SPECIAL TESTS

None commonly used. In the case of 2-nitropropane, Heinz bodies and methemoglobin levels would be of interest.

PERSONAL PROTECTIVE METHODS

Barrier creams or gloves to protect exposed skin and, where vapor concentrations are excessive, fullface mask with organic vapor canister or air supplied respirators are advised.

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NITROPHENOL

DESCRIPTION

There are three isomers of nitrophenol $NO_2C_6H_4OH$. The meta-

form is produced from m-nitroaniline, and the ortho-and para-isomers are produced by nitration of phenol. They are colorless to slightly yellowish crystals with an aromatic to sweetish odor.

SYNONYMS

None.

POTENTIAL OCCUPATIONAL EXPOSURES

Nitrophenols are used in the synthesis of dyestuffs and other intermediates and as a chemical indicator.

A partial list of occupations in which exposure may occur includes:

Chemical indicator makers
Organic chemical synthesizers

PERMISSIBLE EXPOSURE LIMITS

There is no Federal standard for nitrophenol.

ROUTES OF ENTRY

Inhalation and percutaneous absorption of liquid.

HARMFUL EFFECTS

Local—

Unknown.

Systemic—

There is very little information available on the toxicity for humans of nitrophenols. Animal experiments have shown central and peripheral vagus stimulation, CNS depression, methemoglobinemia, and dyspnea. The p-isomer is the most toxic.

MEDICAL SURVEILLANCE

Based on animal studies, individuals with cardiovascular, renal, or pulmonary disease and those with anemia are probably more subject to poisoning by nitrophenol. Liver and renal function and blood should be evaluated in placement or periodic examinations.

SPECIAL TESTS

None commonly used. Nitrophenol is excreted rapidly in the urine as a conjugate. It may also be present as a metabolite of parathion.

PERSONAL PROTECTIVE METHODS

Nitrophenols are readily absorbed through intact skin and by inhalation; full body protective clothing and appropriate type organic vapor canisters in areas of concentrations of dust or vapors should be provided. Spills on work clothing necessitate immediate clothing change and thorough washing of the skin area. Clean work clothes should be supplied daily; showers should be taken at the end of each shift prior to changing to street clothes.

PICRIC ACID

DESCRIPTION

$C_6H_2(NO_2)_3OH$, picric acid, is a pale yellow, odorless, intensely bitter crystal which is explosive upon rapid heating or mechanical shock.

SYNONYMS

Picronitric acid, trinitrophenol, nitroxanthic acid, carbazotic acid, phenol trinitrate.

POTENTIAL OCCUPATIONAL EXPOSURES

Picric acid is used in the manufacture of explosives, rocket fuels, fireworks, colored glass, matches, electric batteries, and disinfectants. It is also used in the pharmaceutical and leather industries, and in dyes, copper and steel etching, forensic chemistry, histology, textile printing, and photographic emulsions.

A partial list of occupations in which exposure may occur includes:

- Battery makers
- Colored glass makers
- Copper etchers
- Disinfectant makers
- Drug makers
- Dye makers
- Explosive makers
- Forsenic chemists
- Histology technicians
- Matchmakers
- Photographic chemical workers
- Tannery workers

PERMISSIBLE EXPOSURE LIMITS

The Federal standard for picric acid is 0.1 mg/m³.

ROUTES OF ENTRY

Inhalation and ingestion of dust; percutaneous absorption.

HARMFUL EFFECTS

Local—

Picric acid dust or solutions are potent skin sensitizers. In solid form, picric acid is a skin irritant, but in aqueous solution it irritates only hypersensitive skin. The cutaneous lesions which appear usually on exposed areas of the upper extremities consist of dermatitis with erythema, papular, and vesicular eruptions. Desquamation may occur following repeated or prolonged contact. Skin usually turns yellow upon contact, and areas around nose and mouth as well as the hair are most often affected. Dust or fume may cause eye irritation which may be aggravated by sensitization. Corneal injury may occur from exposure to picric acid dust and solutions.

Systemic—

Inhalation of high concentrations of dust by one worker caused temporary coma followed by weakness, myalgia, anuria, and later polyuria. Following ingestion of picric acid, there may be headache, vertigo, nausea, vomiting, diarrhea, yellow coloration of the skin, hema-

turia, and albuminuria. High doses may cause destruction of erythrocytes, hemorrhagic nephritis, and hepatitis. High doses which cause systemic intoxication will color all tissues yellow, including the conjunctiva and aqueous humor, and cause yellow vision.

MEDICAL SURVEILLANCE

Preplacement and periodic medical examinations should focus on skin disorders such as hypersensitivity atopic dermatitis, and liver and kidney function.

SPECIAL TESTS

None commonly used. It is probably excreted as picric and picramic acid in the urine.

PERSONAL PROTECTIVE METHODS

Skin protection by clothing and barrier creams can avoid the irritant and sensitizing action of picric acid. Masks of the dust type will prevent absorption by inhalation. Fullface masks are advisable or combination of chemical goggles with halfmask. Daily change of clean work clothes and showering after each shift before changing to street clothes are mandatory.

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TETRYL

DESCRIPTION

Tetryl is a yellow solid.

SYNONYMS

Trinitrophenylmethylnitramine, nitramine, tetranitromethylaniline, pyrenite, picrylmethylnitramine, picrylnitromethylamine, N-methyl-N-2,4, 6-tetranitroaniline, tetralite.

POTENTIAL OCCUPATIONAL EXPOSURES

Tetryl is used in explosives as an intermediary detonating agent and as a booster charge; it is also used as a chemical indicator.

A partial list of occupations in which exposure may occur includes:

Chemical indicator makers
Explosive makers

PERMISSIBLE EXPOSURE LIMITS

The Federal standard is 1.5 mg/m^3.

ROUTES OF ENTRY

Inhalation and skin absorption.

HARMFUL EFFECTS

Local—

Tetryl is a potent sensitizer, and allergic dermatitis is common. Dermatitis first appears on exposed skin areas, but can spread to other parts of the body in fair skinned individuals or those with poor personal hygiene. The severest forms show massive generalized edema with partial obstruction of the trachea due to swelling of the tongue, and these cases require hospitalization. Contact may stain skin and hair yellow or orange. Tetryl is acutely irritating to the mucous membranes of the respiratory tract and the eyes, causing coughing, sneezing, epistaxis, conjunctivitis, and palpebral and periorbital edema.

Systemic—

Tetryl exposure may cause irritability, easy fatigability, malaise, headaches, lassitude, insomnia, nausea, and vomiting. Anemia either of the marrow depression or deficiency type has been observed among tetryl workers. Tetryl exposure has produced liver and kidney damage in animals.

MEDICAL SURVEILLANCE

Preplacement physical examination should give special attention to those individuals with a history of allergy, blood dyscrasias, or skin, liver, or kidney disease. Periodic examinations should be directed primarily to the control of dermatitis and allergic reactions, plus any effects on the respiratory tract, eyes, central nervous system, blood, liver, or kidneys.

SPECIAL TESTS

None in common use.

PERSONAL PROTECTIVE METHODS

Skin protection is necessary by means of protective clothing and gloves. Where significant air concentration of dusts or vapors exist, masks to prevent inhalation are necessary. Daily change to clean work clothes is strongly advised, with showers after each shift mandatory, before dressing in street clothes.

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TRINITROTOLUENE

DESCRIPTION

TNT exists in 5 isomers; 2,4,6-trinitrotoluene is the most commonly

used. All are crystalline solids in pure form. TNT is a relatively stable high explosive.

SYNONYMS

TNT, sym-trinitrotoluol, methyltrinitrobenzene.

POTENTIAL OCCUPATIONAL EXPOSURES

TNT is used as an explosive, i.e., as a bursting charge in shells, bombs, and mines.

A partial list of occupations in which exposure may occur includes:
Explosives workers

PERMISSIBLE EXPOSURE LIMITS

The Federal standard is 1.5 mg/m^3.

ROUTES OF ENTRY

Inhalation of dust, fume, or vapor; ingestion of dust; percutaneous absorption from dust.

HARMFUL EFFECTS

Local—

Exposure to trinitrotoluene may cause irritation of the eyes, nose, and throat with sneezing, cough, and sore throat. It may cause dermatitis and may stain the skin, hair, and nails a yellowish color.

Systemic—

Numerous fatalities have occurred in workers exposed to TNT from toxic hepatitis or aplastic anemia. TNT exposure may also cause methemoglobinemia with cyanosis, weakness, drowsiness, dyspnea, and unconsciousness. In addition it may cause muscular pains, heart irregularities, renal irritation, cataracts, menstrual irregularities, and peripheral neuritis.

MEDICAL SURVEILLANCE

Placement or periodic examinations should give special considerations to history of allergic reactions, blood dyscrasias, reactions to medications, and alcohol intake. The skin, eye, blood, and liver and kidney function should be followed.

SPECIAL TESTS

Urine may be examined for TNT by the Webster test or for the urinary metabolite 2,6-dinitro-4-aminotoluene; however, both may be negative if there is liver injury.

PERSONAL PROTECTIVE METHODS

Protective clothing should be worn. The Webster skin test (colorimetric test with alcoholic sodium hydroxide) or indicator soap should be used to make sure workers have washed all TNT off their skins. Daily change of clean work clothes should be provided, and showers

made compulsory at the end of each shift prior to changing to street clothes.

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MISCELLANEOUS ORGANIC NITROGEN COMPOUNDS

This group of organic nitrogen compounds includes examples of heterocyclic compounds, hydrazines, substituted amides, an imine, and a nitrosoamine.

Heterocyclic nitrogen compounds contain one or more nitrogen atoms in the ring structure and are widely distributed in nature as well as in industrial use. The ring may be three, five, or six membered, and there may be other hetero atoms in addition to nitrogen.

The hydrazine compounds are characteristized by their structure. Amides are derivatives of acids, and some have wide usage as solvents. Imines are highly reactive substances of the general structure, e.g., $R_2C=NH$. Many of them appear to be biological alkylating agents and to have radiomimetic properties. They are somewhat similar in these respects to epoxy compounds, with the nitrogen group in place of an oxygen in a ring structure.

The nitroso group, e.g., $-N=O$, forms another reactive class of nitrogen compounds widely used in synthetic chemical reactions. When combined with a carbon atom, e.g., $C-N=O$, they often show skin irritant or sensitizing properties, and some are methemoglobin formers. When attached to the nitrogen of certain aliphatic amines, however, e.g., $(CH_3)_2-N-N=O$ (N-nitroso dimethyl amine), they sometimes become potent experimental animal carcinogens.

ACRIDINE

DESCRIPTION

$C_{13}H_9N$, acridine, is a colorless or light yellow crystal, very soluble in boiling water.

SYNONYMS

Dibenzopyridine, 10-azaanthracene.

POTENTIAL OCCUPATIONAL EXPOSURES

Acridine and its derivatives are widely used in the production of dyestuffs such as acriflavine, benzoflavine, and chrysaniline, and in the synthesis of pharmaceuticals such as aurinacrine, proflavine, and rivanol.

A partial list of occupations in which exposure may occur includes:

Chemical laboratory workers	Drug makers
Coal tar workers	Dye makers
Disinfectant makers	Organic chemical synthesizers

PERMISSIBLE EXPOSURE LIMITS

There is no Federal standard for acridine.

ROUTE OF ENTRY

Inhalation of vapor.

HARMFUL EFFECTS

Local—

Acridine is a severe irritant to the conjunctiva of the eyes, the mucous membranes of the respiratory tract, and the skin. It is a powerful photosensitizer of the skin. Acridine causes sneezing on inhalation.

Systemic—

Yellowish discoloration of sclera and conjunctiva may occur. Mutational properties have been ascribed to acridine, but its effect on humans is not known.

MEDICAL SURVEILLANCE

Evaluate the skin, eyes, and respiratory tract in the course of any placement or periodic examinations.

SPECIAL TESTS

None commonly used. Can be detected in blood or urine.

PERSONAL PROTECTIVE METHODS

Prevent skin, eye, or respiratory contact with protective clothing, gloves, goggles, and appropriate dust respirators. In case of spills or splashes, the skin area should be thoroughly washed and the contaminated clothing changed. Clean work clothing should be supplied on a daily basis, and the worker should shower prior to changing to street clothes.

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N,N-DIMETHYLFORMAMIDE

DESCRIPTION

$HCON(CH_3)_2$, dimethylformamide, is a colorless liquid which at

25 C is soluble in water and organic solvents. It has a fishy, unpleasant odor at relatively low concentrations, but the odor has no warning property.

SYNONYMS

DMF, the "universal organic solvent," DMFA.

POTENTIAL OCCUPATIONAL EXPOSURES

Dimethylformamide has powerful solvent properties for a wide range of organic compounds. Because of dimethyl formamide's physical properties, it has been used when solvents with a slow rate of evaporation are required.

It finds particular usage in the manufacture of polyacrylic fibers, butadiene, purified acetylene, pharmaceuticals, dyes, petroleum products, and other organic chemicals.

A partial list of occupations in which exposure may occur includes:

- Acetylene purifiers
- Butadiene makers
- Drug makers
- Dye makers
- Organic chemical synthesizers
- Petroleum refinery workers
- Resin makers
- Solvent workers
- Synthetic fiber makers

PERMISSIBLE EXPOSURE LIMITS

The Federal standard is 10 ppm (30 mg/m^3).

ROUTES OF ENTRY

Inhalation of vapor, and it is readily absorbed through intact skin.

HARMFUL EFFECTS

Local—

Dimethylformamide exposure may cause dermatitis.

Systemic—

Inhalation of dimethylformamide or skin contact with this chemical may cause colicky abdominal pain, anorexia, nausea, vomiting, constipation, diarrhea, facial flushing (especially after drinking alcohol), elevated blood pressures, hepatomegaly, and other signs of liver damage. This chemical has produced kidney damage in animals.

MEDICAL SURVEILLANCE

Preplacement and periodic examinations should be concerned particularly with liver and kidney function and with possible effects on the skin.

SPECIAL TESTS

None in common use.

PERSONAL PROTECTIVE METHODS

Organic vapor masks or air supplied respirators may be required in elevated vapor concentrations. Percutaneous absorption should be pre-

vented by gloves and other protective clothing. Goggles should be used to prevent eye splashes. In cases of spills or splashes, the wet clothing should be immediately removed and the skin area thoroughly cleaned. Clean clothing should be issued to workers on a daily basis and showers taken before changing to street clothes.

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ETHYLENEIMINE

DESCRIPTION

H_2CNHCH_2, ethyleneimine, is a colorless volatile liquid with an ammoniacal odor.

SYNONYMS

Azacyclopropane, aziridine, dimethyleneimine, ethylenimine, vinylamine, azirane, dihydroazirine, EI.

POTENTIAL OCCUPATIONAL EXPOSURES

Ethyleneimine is a highly reactive compound and is used in many organic synthesesis. The polymerization products, polyethyleneimines, are used as auxiliaries in the paper industry and as flocculation aids in the clarification of effluents. It is also used in the textile industry for increasing wet strength, flameproofing, shrinkproofing, stiffening, and waterproofing.

A partial list of occupations in which exposure may occur includes:

- Effluent treaters
- Paper makers
- Polyethyleneimine makers
- Organic chemical synthesizers
- Textile makers

PERMISSIBLE EXPOSURE LIMITS

Ethyleneimine was included in the Federal standard for carcinogens; all contact with it should be avoided.

ROUTES OF ENTRY

Inhalation and percutaneous absorption.

HARMFUL EFFECTS

Local—

The vapor is strongly irritating to the conjunctiva and cornea, the mucous membranes of the nose, throat, and upper respiratory tract, and the skin. The liquid is a severe irritant and vesicant in humans, and

severe eye burns have followed contact with the cornea. Skin sensitization has occurred.

Systemic—

Acute exposures in humans have caused nausea, vomiting, headaches, dizziness, and pulmonary edema. In mice acute lethal exposures to vapor produced pulmonary edema, renal damage, and hematuria. Compounds with the aziridine structure have some of the properties of alkylating agents. Ethyleneimine has been reported to induce mutagenic effects in *in vitro* cultures, microorganisms, plants, and animals.

In repeated exposures rodents have developed pancytopenia and gonadal effects. Rats given twice weekly subcutaneous injections of ethyleneimine in oil for about 33 weeks developed sarcoma at the injection site and one case of transitional cell carcinoma in the kidney was observed. Feeding experiments with mice at 13 ppm in the diet for 74 weeks produced hepatomas and pulmonary tumors. These effects have not been reported in humans.

MEDICAL SURVEILLANCE

Based partly on animal experimental data, examinations should include history of exposure to other carcinogens, smoking, alcohol, medications, and family history. The skin, eye, lung, liver, and kidney should be evaluated. Sputum or urine cytology may be helpful.

SPECIAL TESTS

None in common use. Chromosomal studies have been made, but are probably not useful for routine surveillance.

PERSONAL PROTECTIVE METHODS

These are designed to supplement engineering control and prevent all skin or respiratory exposure. Full body protective clothing and gloves should be used. Fullface supplied air respirators with continuous flow or pressure demand type should also be used. Eyes should be protected at all times. On exit from a regulated area employees should shower and change to street clothes, leaving their protective clothing and equipment at the point of exit, to be placed in impervious containers at the end of the work shift for decontamination or disposal.

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HEXAMETHYLENETETRAMINE

DESCRIPTION

$(CH_2)_6N_4$, hexamethylenetetramine, is an odorless, crystalline solid.

SYNONYMS

Methenamine, hexamine, formamine, ammonioformaldehyde.

POTENTIAL OCCUPATIONAL EXPOSURES

Hexamethylenetetramine is used as an accelerator in the rubber industry, as a curing agent in thermosetting plastics, as a fuel pellet for camp stoves, and in the manufacture of resins, pharmaceuticals, and explosives.

A partial list of occupations in which exposure may occur includes:

- Drug makers
- Explosive makers
- Fuel tablet makers
- Phenol-formaldehyde resin workers
- Resin makers
- Rubber makers
- Textile makers
- Urea-formaldehyde resin workers

PERMISSIBLE EXPOSURE LIMITS

There is no Federal standard for hexamethylenetetramine.

ROUTES OF ENTRY

Ingestion and skin contact.

HARMFUL EFFECTS

Local—

Very mild skin irritant.

Systemic—

None. Side effects from ingestion are urinary tract irritation, skin rash, and digestive disturbances. Large oral doses can cause severe nephritis which may be fatal.

MEDICAL SURVEILLANCE

No specific considerations are necessary.

SPECIAL TESTS

None.

PERSONAL PROTECTIVE METHODS

If repeated or prolonged skin exposure is likely, gloves or protective clothing may be needed.

HYDRAZINE and DERIVATIVES

DESCRIPTION

Hydrazine (H_2N-NH_2) is a colorless, oily liquid with an ammoniacal odor. Phenylhydrazine ($C_6H_5NHNH_2$) is an oily, colorless liquid

or a crystalline solid. Dimethylhydrazine, UDMH, ($(CH_3)_2-N-NH_2$) is a hygroscopic mobile liquid. Hydrazine and UDMH are soluble in water and alcohol. Phenylhydrazine is slightly soluble in water.

SYNONYMS

Hydrazine: Hydrazine base, diamine.

Phenylhydrazine: Hydrazinobenzene.

Dimethylhydrazine: UDMH, 1,1-dimethylhydrazine, asymmetrical dimethylhydrazine.

POTENTIAL OCCUPATIONAL EXPOSURES

Both UDMH and hydrazine are used in liquid rocket fuels. Because of its strong reducing capabilities, hydrazine is used as an intermediate in chemical synthesis and in photography and metallurgy. It is also used in the preparation of anticorrosives, textile agents, and pesticides, and as a scavenging agent for oxygen in boiler water. Hydrazine salts find use as fluxes in soft soldering and aluminum soldering. Phenylhydrazine is very reactive with carbonyl compounds and is a widely used reagent in conjunction with sugars, aldehydes, and ketones, in addition to its use in the synthesis of dyes, pharmaceuticals such as antipyrin, cryogenin, and pyramidone, and other organic chemicals. The hydrochloride salt is used in the treatment of polycythemia vera.

A partial list of occupations in which exposure may occur includes:

Acrylic and vinyl textile dyers
Agricultural chemical makers
Anticorrosion additive makers
Antioxidant workers
Boiler operators
Chemists
Drug makers
Insecticide makers
Jet fuel workers
Oxygen scavenger makers
Photographic developer makers
Rocket fuel workers
Solder flux makers
Water treaters

PERMISSIBLE EXPOSURE LIMITS

The Federal standard compounds are:

Hydrazine 1 ppm (1.3 mg/m³)
Phenylhydrazine 5 ppm (22 mg/m³)
Dimethylhydrazine 0.5 ppm (1 mg/m³)

ROUTES OF ENTRY

Inhalation and percutaneous absorption.

HARMFUL EFFECTS

Local—

All three compounds have similar toxic local effects due to their irritant properties. The vapor is highly irritating to the eyes, upper respiratory tract, and skin, and causes delayed eye irritation. Severe exposure may produce temporary blindness. The liquid is corrosive, producing penetrating burns and severe dermatitis. Permanent corneal lesions may occur if the liquid is splashed in the eyes. A sensitization dermatitis may be produced.

Systemic—

Inhalation of hydrazine may cause dizziness and nausea. In animals hydrazine has caused liver and kidney damage and pulmonary edema. It has also been reported to cause adenocarcinoma of the lung and liver in animals.

MEDICAL SURVEILLANCE

Based partly on experimental animal data, placement should include a history of exposure to other carcinogens, smoking, alcohol, medications, and family history. The skin, eye, lungs, liver, kidney, blood, and central nervous system should be evaluated. Sputum or urine cytology may give useful information.

SPECIAL TESTS

Hydrazine may be detected in the blood; UDMH has been measured in blood and urine. Some phenylhydrazine metabolites are known. None of these are in common use, however.

PERSONAL PROTECTIVE METHODS

Protective clothing, gloves, and goggles should be worn to reduce any skin or eye contact. Fullface supplied air masks and full protective clothing may be required if vapor concentrations are significant. Clean work clothes should be supplied on a daily basis, and workers should shower prior to change to street clothes.

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N-NITROSODIMETHYLAMINE

DESCRIPTION

$(CH_3)_2NN=O$, n-nitrosodimethylamine (DMN), is a yellow liquid of low viscosity, soluble in water, alcohol, and ether.

SYNONYMS

Dimethylnitrosamine, DMN.

POTENTIAL OCCUPATIONAL EXPOSURES

DMN is used in the manufacture of dimethylhydrazine. It has also been used as an industrial solvent and a nematocide. There are patents

for its use as a solvent in the fiber and plastics industry, as an antioxidant, as a softener for copolymers, as an additive for lubricants, and in condensers to increase the dielectric constant.

A partial list of occupations in which exposure may occur includes:

Dimethylhydrazine makers
Nematocide makers
Solvent workers

PERMISSIBLE EXPOSURE LIMITS

DMN is included in the Federal standard for carcinogens; all contact with it should be avoided.

ROUTES OF ENTRY

Inhalation of vapor and possibly percutaneous absorption.

HARMFUL EFFECTS

Local—

The liquid and vapor are not especially irritating to the skin or eyes, and warning properties are poor.

Systemic—

DMN is a highly toxic substance in most species, including man. Systemic effects are characterized by onset in a few hours of nausea and vomiting, abdominal cramps, and diarrhea. Also headache, fever, weakness, enlargement of the liver, and jaundice may occur. Chronic exposures may lead to liver damage (central necrosis), with jaundice and ascites. There have been a number of reported cases, including severe liver injury in man and one death. Autopsy revealed an acute diffuse centrolobular necrosis. Recovery occurred in other cases.

In rats, guinea pigs, and other experimental animals, DMN is a highly potent carcinogen, producing malignant tumors, primarily of the liver and kidney, but also in the lung. Both ingestion and inhalation routes have produced tumors. These have not been reported in man, but in view of its potency in various other species, the material has been presumed to be carcinogenic in man also.

MEDICAL SURVEILLANCE

Based on human experience and on animal studies, preplacement and periodic examinations should include a history of exposure to other carcinogens, alcohol and smoking habits, medications, and family history. Special attention should be given to liver size and function, and to any changes in lung symptoms or X-rays. Renal function should be followed. Sputum and urine cytology may be useful.

SPECIAL TESTS

None commonly used.

PERSONAL PROTECTIVE METHODS

These are designed to supplement engineering controls and to prevent all contact with the skin, eyes, or respiratory tract. Full body pro-

tective clothing and gloves should be provided and also appropriate type fullface supplied air respirators of continuous flow or pressure demand type. On exit from a regulated area, employees should be required to shower before changing into street clothes, leaving their protective clothing and equipment at the point of exit, to be placed in impervious containers at the end of the work shift for decontamination or disposal.

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PYRIDINE

DESCRIPTION

C_5H_5N, pyridine, is a colorless liquid with an unpleasant odor. It is both flammable and explosive when exposed to a flame and decomposes on heating to release cyanide fumes. Pyridine is soluble in water, alcohol, and ether. The odor can be detected well below 1 ppm.

SYNONYMS

Azine.

POTENTIAL OCCUPATIONAL EXPOSURES

Pyridine is used as a solvent in the chemical industry and as a denaturant for ethyl alcohol. It is used in the manufacture of paints, explosives, dyestuffs, rubber, vitamins, sulfa drugs, and disinfectants.

A partial list of occupations in which exposure may occur includes:

- Alcohol denaturant makers
- Alcohol denaturers
- Drug makers
- Dye makers
- Explosive workers
- Organic chemical synthesizers
- Paint makers
- Rubber workers
- Resin workers
- Solvent workers
- Vitamin makers

PERMISSIBLE EXPOSURE LIMITS

The Federal standard is 5 ppm (15 mg/m^3).

ROUTES OF ENTRY

Inhalation of vapor and percutaneous absorption of liquids.

HARMFUL EFFECTS

Local—

Irritation of the conjunctiva of the eye and cornea and mucous membranes of the upper respiratory tract and skin may occur. It oc-

casionally causes skin sensitization, and photosensitization has been reported.

Systemic—

Very high concentrations may cause narcosis. Repeated, intermittent, or continuous low level exposure may lead to transient effects on the central nervous system and gastrointestinal tract. The symptoms include headache, dizziness, insomnia, nervousness, anorexia, nausea, vomiting, and diarrhea. Low back pain and urinary frequency with no changes in urine sediment or liver or renal function and complete recovery have been reported to follow exposures to about 100 ppm. Liver and kidney injury have been reported from its use as an oral medication.

MEDICAL SURVEILLANCE

Placement and periodic examinations should consider possible effects on skin, central nervous system, and liver and kidney function.

SPECIAL TESTS

None in common use. Metabolites are known and can be determined in blood and urine.

PERSONAL PROTECTIVE METHODS

Rubber and plastic gloves should not be relied upon to prevent contact with pyridine as its salts penetrate the material. The odor is detectable at less than 1 ppm but cannot be relied upon as a preventive. In areas of elevated vapor concentration, workers should be supplied with fullface supplied air masks and protective clothing. Clothing that is contaminated by spills or splashes should be immediately changed and discarded and the area of involved skin thoroughly washed. Clean work clothes should be supplied daily with the worker showering after his shift before changing to street clothes.

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N,N-DIMETHYLACETAMIDE

DESCRIPTION

$CH_3CON(CH_3)_2$, dimethylacetamide, is a colorless, nonvolatile liquid.

SYNONYMS

Acetic acid dimethylamide, DMA, DMAC, acetyl dimethylamide.

POTENTIAL OCCUPATIONAL EXPOSURES

Dimethylacetamide is used commercially as a solvent in various industries.

A partial list of occupations in which exposure may occur includes:

Solvent workers

PERMISSIBLE EXPOSURE LIMITS

The Federal standard is 10 ppm (35 mg/m³), skin.

ROUTES OF ENTRY

Inhalation of vapor and absorption through intact skin.

HARMFUL EFFECTS

Local—

None known.

Systemic—

Jaundice has been noted in workers exposed chronically to dimethylacetamide vapor although skin absorption may also have occurred. Liver injury consists of cord-cell degeneration, but recovery is usually rapid. Other symptoms from large oral doses as an anticancer drug include depression, lethargy, and visual and auditory hallucinations.

MEDICAL SURVEILLANCE

Preplacement and periodic medical examinations should give special attention to skin, central nervous system, and liver function or disease.

SPECIAL TESTS

None commonly used.

PERSONAL PROTECTIVE METHODS

Organic vapor masks or air supplied respirators may be required in elevated vapor concentrations. Percutaneous absorption should be prevented by gloves and other protective clothing. Goggles should be used to prevent eye splashes. In cases of spills or splashes, the wet clothing should be immediately removed and the involved skin area thoroughly cleaned. Clean clothing should be issued to workers on a daily basis and showers taken before changing to street clothes.

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MISCELLANEOUS ORGANIC CHEMICALS

BETA-PROPIOLACTONE

DESCRIPTION

OCH_2CH_2CO, beta-propiolactone, is a colorless liquid which slowly hydrolyzes to hydracrylic acid and must be cooled to remain stable.

SYNONYMS

2-Oxetanone, propiolactone, BPL, 3-hydroxy-beta-lactone-propanoic acid.

POTENTIAL OCCUPATIONAL EXPOSURES

beta-Propiolactone is used as a chemical intermediate in synthesis of acrylate plastics and as a vapor sterilizing agent, disinfectant, and a viricidal agent.

A partial list of occupations in which exposure may occur includes:

Acrylate plastic makers	Plastic makers
Chemists	Resin makers
Disinfectant workers	Viricidal agent makers

PERMISSIBLE EXPOSURE LIMITS

beta-Propiolactone is included in the Federal standards for carcinogens; all contact with it should be avoided.

ROUTES OF ENTRY

Inhalation of vapor and percutaneous absorption.

HARMFUL EFFECTS

Local—

Repeated or prolonged contact with liquid may cause erythema, vesication of the skin, and, as reported in animals, hair loss and scarring. In rodents, beta-propiolactone has also produced skin papilloma and sarcoma by skin painting, subcutaneous injection, and oral administration. Tumors of the connective tissue are also suspected. Direct eye contact with concentrated liquid may result in permanent corneal opacification. Skin cancer has not been reported in man.

Systemic—

The systemic effect of beta-propiolactone in humans is unknown due to lack of reported cases. Acute exposure in animals has caused liver necrosis and renal tubular damage. Death has occurred following rapid development of spasms, dyspnea, convulsions, and collapse at relatively low levels (less than 5 ml/kg.) Beta propiolactone has been implicated as a carcinogen by a number of animal studies which produced a variety of skin tumors, stomach tumors, and hepatoma depending on the route of administration.

MEDICAL SURVEILLANCE

Based on its high toxicity and carcinogenic effects in animals, preplacement and periodic examinations should include a history of exposure to other carcinogens, alcohol and smoking habits, medication and family history. The skin, eye, lung, liver, and kidney should be evaluated. Sputum cytology, may be helpful in evaluating the presence or absence of carcinogenic effects.

SPECIAL TESTS

None in common use.

PERSONAL PROTECTIVE METHODS

These are designed to supplement engineering controls and to pre-

vent all contact with skin or respiratory tract. Full body protective clothing and gloves should be provided as well as fullface supplied air respirators of continuous flow or pressure demand type. Employees should remove and leave protective clothing and equipment at the point of exit, to be placed in impervious containers at the end of work shift for decontamination or disposal. Showers should be taken before dressing in street clothes.

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TRICRESYL PHOSPHATES

DESCRIPTION

Tricresyl phosphates are available as the ortho-isomer (TOCP), the meta-isomer (TMCP), and the para-isomer (TPCP). The ortho-isomer is the most toxic of the three; the meta- and para-isomers are relatively inactive. The commercial product may contain the ortho-isomer as a contaminant unless special precautions are taken during manufacture. Pure tri-para-cresylphosphate is a solid, and ortho- and meta- are colorless, oily, odorless liquids.

SYNONYMS

Tritolyl phosphate, TCP.

POTENTIAL OCCUPATIONAL EXPOSURES

Tricresyl phosphate is used as a plasticizer for chlorinated rubber, vinyl plastics, polystyrene, polyacrylic, and polymethacrylic esters, as an adjuvant in milling of pigment pastes, as a solvent and as a binder in nitrocellulose and various natural resins, and as an additive to synthetic lubricants and gasoline. It is also used as hydraulic fluid, fire retardant and in the recovery of phenol in coke-oven waste waters.

A partial list of occupations in which exposure may occur includes:

- Gasoline additive makers
- Gasoline blenders
- Hydraulic fluid workers
- Lead scavenger makers
- Nitrocellulose workers
- Plasticizer workers
- Polystyrene makers
- Polyvinyl chloride makers
- Solvent workers
- Surgical instrument sterilizers
- Waterproofing makers

PERMISSIBLE EXPOSURE LIMITS

The Federal standard for tri-ortho-cresyl phosphate is 0.1 mg/m^3; there is no standard for the meta- and para-isomers.

ROUTES OF ENTRY

Inhalation of ortho-isomer vapor or mist, especially when heated;

ingestion and percutaneous absorption of liquids. The widespread epidemics of poisoning that have occurred have been due to ingested ortho-isomer as a contaminant of foodstuff. There have been relatively few reports of neurological symptoms in workers handling these substances. Experimental human studies with labeled phosphorus derivatives show only 0.4% of the applied dose was absorbed.

HARMFUL EFFECTS

Local—

None reported.

Systemic—

The major effects from inhaling, swallowing, or absorbing tricresyl phosphate through the skin are on the spinal cord and peripheral nervous system; the poison attacking the anterior horn cells and pyramidal tract as well as the peripheral nerves. Gastrointestinal symptoms on acute exposure (nausea, vomiting, diarrhea, and abdominal pain) are followed by a latent period of 3 to 30 days with the progressive development of muscle soreness and numbness of fingers, calf muscles, and toes, with foot and wrist drop. In chronic intoxication, the g.i. symptoms pass unnoticed, and after a long latent period, flaccid paralysis of limb and leg muscles appear. There are minor sensory changes and no loss of sphincter control.

MEDICAL SURVEILLANCE

Preplacement and periodic examinations should include evaluation of spinal cord and neuromuscular function, especially in the extremities, and a history of exposure to other organo-phosphate esters, pesticides, or neurotoxic agents. Periodic cholinesterase determination may relate to exposure, but not necessarily to neuromuscular effect.

SPECIAL TESTS

None used except for determination of serum or red cell choline or acetylcholine esterases.

PERSONAL PROTECTIVE METHODS

Protective clothing should be worn to prevent skin absorption and, where dust or vapor concentrates, masks should be supplied to employees.

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CARBON DISULFIDE

DESCRIPTION

CS_2, carbon disulfide, is a highly refractive, flammable liquid which in pure form has a sweet odor and in commercial and reagent grades has a foul smell. It can be detected by odor at about 1 ppm but the sense of smell fatigues rapidly and, therefore, odor does not serve as a good warning property. It is slightly soluble in water, but more soluble in organic solvents.

SYNONYMS

Carbon bisulfide, dithiocarbonic anhydride.

POTENTIAL OCCUPATIONAL EXPOSURES

Carbon disulfide is used in the manufacture of viscose rayon, ammonium salt, carbon tetrachloride, carbanilide, xanthogenates, flotation agents, soil disinfectants, dyes, electronic vacuum tubes, optical glass, paints, enamels, paint removers, varnishes, varnish removers, tallow, textiles, explosives, rocket fuel, putty, preservatives, and rubber cement; as a solvent for phosphorus, sulfur, selenium, bromine, iodine, alkali cellulose, fats, waxes, lacquers, camphor, resins, and cold vulcanized rubber. It is also used in degreasing, chemical analysis, electroplating, grain fumigation, oil extraction, and drycleaning.

A partial list of occupations in which exposure may occur includes:

- Ammonium salt makers
- Bromine processors
- Carbon tetrachloride makers
- Degreasers
- Drycleaners
- Electroplaters
- Fat processors
- Flotation agent makers
- Iodine processors
- Oil processors
- Paint workers
- Preservative makers
- Putty makers
- Rayon makers
- Resin makers
- Rocket fuel makers
- Rubber cement makers
- Rubber workers
- Sulfur processors
- Tallow makers
- Textile makers
- Vacuum tube makers
- Varnish makers
- Wax processors

PERMISSIBLE EXPOSURE LIMITS

The Federal standard is 20 ppm (60 mg/m^3) determined as an 8-hour TWA. The acceptable ceiling concentration is 30 ppm (90 mg/m^3) with a maximum peak above this for an 8-hour workshift of 100 ppm (300 mg/m^3) for a maximum duration of 30 minutes.

ROUTES OF ENTRY

Inhalation of vapor which may be compounded by percutaneous absorption of liquid or vapor.

HARMFUL EFFECTS

Local—

Carbon disulfide vapor in sufficient quantities is severely irritating to

eyes, skin, and mucous membranes. Contact with liquid may cause blistering with second and third degree burns. Skin sensitization may occur. Skin absorption may result in localized degeneration of peripheral nerves which is most often noted in the hands. Respiratory irritation may result in bronchitis and emphysema, though these effects may be overshadowed by systemic effects.

Systemic—

Intoxication from carbon disulfide is primarily manifested by psychological, neurological, and cardiovascular disorders. Recent evidence indicates that once biochemical alterations are initiated they may remain latent; clinical signs and symptoms then occur following subsequent exposure.

Following repeated carbon disulfide exposure, subjective psychological as well as behavioral disorders have been observed. Acute exposures may result in extreme irritability, uncontrollable anger, suicidal tendencies, and a toxic manic depressive psychosis. Chronic exposures have resulted in insomnia, nightmares, defective memory, and impotency. Less dramatic changes include headache, dizziness, and diminished mental and motor ability, with staggering gait and loss of coordination.

Neurological changes result in polyneuritis. Animal experimentation has revealed pyramidal and extrapyramidal tract lesions and generalized degeneration of the myelin sheaths of peripheral nerves. Chronic exposure signs and symptoms include retrobulbar and optic neuritis, loss of sense of smell, tremors, paresthesias, weakness, and, most typically, loss of lower extremity reflexes.

Atherosclerosis and coronary heart disease have been significantly linked to exposure to carbon disulfide. Atherosclerosis develops most notably in the blood vessels of the brain, glomeruli, and myocardium. Abnormal electroencephalograms and retinal hypertension typically occur before renal involvement is noted. Any of the above three areas may be affected by chronic exposure, but most often only one aspect can be observed. A significant increase in coronary heart disease mortality has been observed in carbon disulfide workers. Studies also reveal higher frequency of angina pectoris and hypertension. Abnormal electrocardiograms may also occur and are also suggestive of carbon disulfide's role in the etiology of coronary disease.

Other specific effects include chronic gastritis with the possible development of gastric and duodenal ulcers; impairment of endocrine activity, specifically adrenal and testicular; abnormal erythrocytic development with hypochromic anemia; and possible liver dysfunction with abnormal serum cholesterol. Also in women, chronic menstrual disorders may occur. These effects usually occur following chronic exposure and are subordinate to the other symptoms.

Recently human experience and animal experimentation have indicated several possible biochemical changes. Carbon disulfide and its metabolites (i.e., dithiocarbamic acids and isothiocyanates) show amino acid interference, cerebral monoamine oxidase inhibition, endocrine dis-

orders, lipoprotein metabolism interference, blood protein, and zinc level abnormalities, and inorganic metabolism interference due to chelating of polyvalent ions. The direct relationship between these biochemical changes and clinical manifestations is only suggestive.

MEDICAL SURVEILLANCE

Preplacement and periodic medical examinations should be concerned especially with skin, eyes, central and peripheral nervous system, cardiovascular disease, as well as liver and kidney function. Electrocardiograms should be taken.

SPECIAL TESTS

CS_2 can be determined in expired air, blood, and urine. The iodine-azide test is most useful although non-specific, and it may indicate other sulfur compounds.

PERSONAL PROTECTIVE METHODS

Local exhaust, general ventilation, and personal protective equipment should be utilized. In modern manufacture, CS_2 fumes are generally controlled by closed operations. Where fumes are present in unacceptable concentrations, vapor gas mask with fullface or used air respirators should be used. In all areas where there is likelihood of spill or splash on any skin area, protection should be afforded by protective clothing, goggles, face shields, aprons, and coats.

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DIMETHYL SULFATE

DESCRIPTION

$(CH_3)_2SO_4$, dimethyl sulfate, is an oily, colorless liquid slightly soluble in water, but more soluble in organic solvents.

SYNONYMS

Sulfuric acid dimethyl ester.

POTENTIAL OCCUPATIONAL EXPOSURES

Industrial use of dimethyl sulfate is based upon its methylating properties. It is used in the manufacture of methyl esters, ethers and amines, in dyes, drugs, perfume, phenol derivatives, and other organic chemicals. It is also used as a solvent in the separation of mineral oils.

A partial list of occupations in which exposure may occur includes:

- Amine makers
- Drug makers
- Dye makers
- Methylation workers
- Organic chemical synthesizers
- Perfume makers
- Phenol derivative makers

PERMISSIBLE EXPOSURE LIMITS

The Federal standard is 1 ppm (5 mg/m^3).

ROUTES OF ENTRY

Inhalation of vapor; percutaneous absorption of liquid.

HARMFUL EFFECTS

Local—

Liquid is highly irritating and causes skin vesiculation and analgesia. Lesions are typically slow-healing and may result in scar tissue while analgesia may last several months. Liquid and vapor are irritating to the mucous membranes, and exposure produces lacrimation, rhinitis, edema of the mucosa of the mouth and throat, dysphagia, sore throat, and hoarseness. Irritation of the skin and mucous membranes may be delayed in appearance. Eye irritation may result in conjunctivitis, keratitis, and photophobia. In severe cases corneal opacities, perforation of the nasal septum and permanent or persistent visual disorders have been reported.

Systemic—

The toxicity of dimethyl sulfate is based upon its alkylating properties and its hydrolysis to sulfuric acid and methyl alcohol. Acute exposure may cause respiratory dysfunctions such as pulmonary edema, bronchitis, and pneumonitis following a latent period of 6 to 24 hours. Cerebral edema and other central nervous system effects such as drowsiness, temporary blindness, tachycardia or bradycardia may be linked to dimethyl sulfate's effect on nerve endings. Secondary pulmonary effects such as susceptibility to infection, as well as, more pronounced effects in those persons with preexisting respiratory disorders, are also noteworthy. Chronic poisoning occurs only rarely and is usually limited to ocular and respiratory disabilities. It has been reported to be carcinogenic in rats, but this has not been verified in man.

MEDICAL SURVEILLANCE

Preplacement and periodic medical examinations should give special consideration to the skin, eyes, central nervous system, lung. Chest X-rays should be taken and lung, liver, and kidney functions evaluated.

Sputum and urinary cytology may be useful in detecting the presence or absence of carcinogenic effects.

SPECIAL TESTS

None in common use.

PERSONAL PROTECTIVE METHODS

These are designed to supplement engineering controls and to reduce skin, eye, or respiratory contact to a negligible level. The liquid and the vapor of dimethyl sulfate are extremely irritating so that the skin, eyes, as well as the respiratory tract should be protected at all times. Protective clothing, gloves, goggles, face shields, aprons, and boots should be used in areas where there is danger of splash or spill. Fullface vapor masks or supplied air respirators may be necessary in areas of vapor build up or leaks. Attention should be given to personal hygiene with a change of work clothes daily and shower before change to street clothes.

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MERCAPTANS

DESCRIPTION

Methyl mercaptan: CH_3SH; ethyl mercaptan: CH_3CH_2SH; n-butyl mercaptan: $CH_3(CH_2)_2CHSH$; and perchloromethyl mercaptan: CCl_3SCl.

These compounds are typically flammable liquids except methyl mercaptan which is a gas. Perchloromethyl mercaptan is yellow; the rest are colorless. A strong unpleasant odor is the most characteristic property of mercaptans and may be detected at very low levels, i.e. less than 0.5 ppm. Perchloromethyl mercaptan is insoluble in water, but others are slightly soluble.

SYNONYMS

Methyl mercaptans: Methanethiol, mercaptomethane, thiomethyl alcohol, methyl sulfhydrate.

Ethyl mercaptan: Ethanethiol, mercaptoethane, ethyl sulfhydrate, thioethyl alcohol.

n-Butyl mercaptan: 1-Butanethiol, n-butyl thioalcohol, thiobutyl alcohol.

POTENTIAL OCCUPATIONAL EXPOSURES

In general, mercaptans find use as intermediates in the manufacture of pesticides, fumigants, dyes, pharmaceuticals, and other chemicals, and as gas odorants, i.e., to serve as a warning property for hazardous odor-

less gases. Particular usages for methyl mercaptan include the synthesis of methionine and the manufacture of fungicides and jet fuels. Ethyl mercaptan is used as an adhesive stabilizer and butyl mercaptan may be used as a solvent.

A partial list of occupations in which exposure may occur includes:

- Drug makers
- Dye makers
- Fumigant makers
- Fumigators
- Jet fuel blenders
- Methionine makers
- Organic chemical synthesizers
- Pesticide makers
- Warning agent workers

PERMISSIBLE EXPOSURE LIMITS

The Federal standard for each mercaptan is:

Methyl mercaptan	10 ppm	20 mg/m^3
Ethyl mercaptan	10 ppm	25 mg/m^3
Butyl mercaptan	10 ppm	35 mg/m^3
Perchloromethyl mercaptan	0.1 ppm	0.8 mg/m^3

The above standards are determined as TWAs except ethyl and methyl mercaptan which are ceiling values. ACGIH has lowered the TLVs of all but perchloromethyl mercaptan: Methyl mercaptan, 0.5 ppm (1 mg/m^3); ethyl mercaptan, 0.5 ppm (1.0 mg/m^3); butyl mercaptan, 0.5 ppm (1.5 mg/m^3); all TWAs.

ROUTE OF ENTRY

Inhalation of gas or vapor.

HARMFUL EFFECTS

Local—

Mercaptans have an intensely disagreeable odor and are irritating to skin, eyes, and mucous membranes of the upper respiratory tract. Liquid may cause contact dermatitis and vapor may cause irritation to nose and throat. Perchloromethyl mercaptan is stronger in its irritant ability than the other mercaptans which cause only slight to moderate irritation.

Systemic—

Methyl mercaptan acts toxicologically like hydrogen sulfide and may depress the central nervous system resulting in respiratory paralysis and death. Victims who survive severe exposures may suffer from headache, dizziness, staggering gait, nausea, and vomiting. Respiratory tract irritation may lead to pulmonary edema and possibly renal and hepatic damage. The above effects are based primarily on animal experimentation. In a recent case of acute methyl mercaptan exposure, a worker developed acute anemia and methemoglobinemia 24 hours following coma.

MEDICAL SURVEILLANCE

Preplacement and periodic medical examinations should consider skin, eyes, lung and central nervous system as well as liver and kidney functions. Blood studies may be helpful in following acute intoxication from methyl mercaptan.

SPECIAL TESTS

None commonly used.

PERSONAL PROTECTIVE METHODS

In areas where liquid mercaptan is likely to be spilled or splashed on the skin, impervious clothing, gloves, gauntlets, aprons, and boots should be supplied. Otherwise protective methods are as for sulfur dioxide. (See Sulfur Dioxide.)

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TETRAMETHYLTHIURAM DISULFIDE

DESCRIPTION

$C_6H_{12}N_2S_4$, tetramethylthiuram disulfide, is a white or yellow crystal insoluble in water, but soluble in organic solvents.

SYNONYMS

Thiram, bis-(dimethylthiocarbamyl) disulfide, TMTD, thirad, thiuram.

POTENTIAL OCCUPATIONAL EXPOSURES

Tetramethylthiuram disulfide is used as a rubber accelerator and vulcanizer; a seed, nut, fruit, and mushroom disinfectant; a bacteriostat for edible oils and fats; and as an ingredient in sun-tan and antiseptic sprays and soaps. It is also used as a fungicide, rodent repellent, wood preservative, and may be used in the blending of lubricant oils.

A partial list of occupations in which exposure may occur includes:

- Food disinfectant makers
- Fungicide workers
- Lubricating oil blenders
- Rat repellent makers
- Rubber makers
- Soap makers
- Wood preservative makers

PERMISSIBLE EXPOSURE LIMITS

The Federal standard for thiram (tetramethylthiuram disulfide) is 5 mg/m³.

ROUTE OF ENTRY

Inhalation of dust, spray, or mist.

HARMFUL EFFECTS

Local—

Irritation of mucous membranes conjunctivitis, rhinitis, sneezing, and cough may result from excessive exposures. Skin irritation with erythema and urticaria may also occur. Allergic contact dermatitis has been reported in workers who wore rubber gloves containing tetramethylthiuram disulfide.

Systemic—

Systemic effects have not been reported in the U.S. literature. Bronchitis was mentioned in one European report in workers exposed to thiram or other products during synthesis. Intolerance to alcohol has been observed in workers exposed to thiram, manifested by flushing of face, palpitation, rapid pulse, dizziness, and hypotension. These effects are thought to be due to the blocking of the oxidation of acetaldehyde. It should be noted in this connection that the diethyl homologue of this compound, tetraethylthiuram disulfide, is marketed as the drug "Antabuse" and that severe and disagreeable symptoms ensue immediately in subjects who ingest the smallest amount of ethyl alcohol after they have been "premedicated" with the drug.

MEDICAL SURVEILLANCE

Preplacement and periodic medical examinations should give special attention to history of skin allergy, eye irritation, and significant respiratory, liver, or kidney disease. Workers should be aware of the potentiating action of alcoholic beverages when working with tetramethylthiuram disulfide.

SPECIAL TESTS

None in common use.

PERSONAL PROTECTIVE METHODS

Skin and eye protection should be provided by protective clothing, gloves, and goggles. Employees should be encouraged to shower following each shift and to change to clean work clothes at the start of each shift. In areas where dust, spray, or mist are excessive, respiratory protection by dust masks or gas mask respirators with proper canister or supplied air respirators should be provided.

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HALOGENS

BROMINE/HYDROGEN BROMIDE

DESCRIPTION

Br, bromide, is a dark reddish-brown, fuming, volatile liquid with a suffocating odor. Bromine is soluble in water and alcohol. HBr, hydrogen bromide, is a corrosive colorless gas.

SYNONYMS

Bromine: none.

Hydrogen bromide: anhydrous hydrobromic acid.

POTENTIAL OCCUPATIONAL EXPOSURES

Bromine is primarily used in the manufacture of gasoline antiknock compounds (1,2-dibromoethane). Other uses are for gold extraction, in brominating hydrocarbons, in bleaching fibers and silk, in the manufacture of pharmaceuticals, military gas, dyestuffs, and as an oxidizing agent.

Hydrogen bromide and its aqueous solutions are used in the manufacture of organic and inorganic bromides, as a reducing agent and catalyst in controlled oxidations, in the alkylation of aromatic compounds, and in the isomerization of conjugated diolefins.

A partial list of occupations in which exposure may occur includes:

Drug makers	Organic chemical synthesizers
Dye makers	Petroleum refinery workers
Gasoline additive makers	Photographic chemical makers
Gold extractors	Silk and fiber bleachers

PERMISSIBLE EXPOSURE LIMITS

The Federal standards are: bromine 0.1 ppm (0.7 mg/m^3); and hydrogen bromide 3 ppm (10 mg/m^3).

ROUTES OF ENTRY

Inhalation of vapor or gas. Bromine may be absorbed through the skin.

HARMFUL EFFECTS

Local—

Bromine and hydrogen bromide and its aqueous solutions are extremely irritating to eyes, skin, and mucous membranes of the upper respiratory tract. Severe burns of the eye may result from liquid or concentrated vapor exposure. Liquid bromine splashed on skin may cause vesicles, blisters, and slow healing ulcers. Continued exposure to low concentrations may result in acne-like skin lesions. These are more common in the oral use of sodium bromide as a sedation.

Systemic—

Inhalation of bromine is corrosive to the mucous membranes of

the nasopharynx and upper respiratory tract, producing brownish discoloration of tongue and buccal mucosa, a characteristic odor of the breath, edema and spasm of the glottis, asthmatic bronchitis, and possibly pulmonary edema which may be delayed until several hours following exposure. A measles-like skin rash may occur. Exposure to high concentrations of bromine can lead to rapid death due to choking caused by edema of the glottis and pulmonary edema.

Bromine has cumulative properties and is deposited in tissues as bromides, displacing other halogens. Exposures to low concentrations result in cough, copious mucous secretions, nose bleeds, respiratory difficulty, vertigo, and headache. Usually these symptoms are followed by nausea, diarrhea, abdominal distress, hoarseness, and asthmatic type respiratory difficulty.

Other effects from chronic exposure have been reported in Soviet literature, e.g., loss of corneal reflexes, joint pains, vegetative disorders, thyroid dysfunction, and depression of the bone marrow. These have not been noted in the U.S. literature.

Hydrogen bromide (hydrobromic acid) is less toxic than bromine, but is an irritant to the mucous membranes of the upper respiratory tract. Long term exposures can cause chronic nasal and bronchial discharge and dyspepsia. Skin contact may cause burns.

MEDICAL SURVEILLANCE

The skin, eyes, and respiratory tract should be given special emphasis during preplacement and periodic examinations. Chest X-rays as well as general health, blood, liver, and kidney function should be considered. Exposure to other irritants or bromine compounds in medications may be important.

SPECIAL TESTS

None commonly used. Blood bromides can be determined but are probably not helpful in following exposures.

PERSONAL PROTECTIVE METHODS

Respiratory protection with gas masks with acceptable canister or supplied air respirators is essential in areas of excessive vapor concentration. Where aqueous solutions or liquids are used, or high vapor concentrations are present, skin and eyes should be protected against spills or splashes by impervious clothing, gloves, aprons, and face shields or goggles.

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CHLORINATED LIME

DESCRIPTION

Chlorinated lime is a white or grayish-white hygroscopic powder with a chlorine odor. It is a relatively unstable chlorine carrier in solid form and is a complex compound of indefinite composition. Chemically, it consists of varying proportions of calcium hypochlorite, calcium chlorite, calcium oxychloride, calcium chloride, free calcium hydroxides, and water. The commercial product generally contains 24-37% available chlorine. On exposure to moisture, chlorine is released.

SYNONYMS

Chloride of lime, bleaching powder.

POTENTIAL OCCUPATIONAL EXPOSURES

Chlorinated lime is a bleaching agent, i.e., it has the ability to chemically remove dyes or pigments from materials. It is used in the bleaching of wood pulp, linen, cotton, straw, oils, and soaps, and in laundering, as an oxidizer in calico printing, a chlorinating agent, a disinfectant, particularly for drinking water and sewage, a decontaminant for mustard gas, and as a pesticide for caterpillars.

A partial list of occupations in which exposure may occur includes:

- Disinfectant makers
- Dyers
- Laundry workers
- Oil bleachers
- Sewage treaters
- Soap bleachers
- Straw bleachers
- Textile bleachers
- Textile printers
- Water treaters
- Wood pulp bleachers

PERMISSIBLE EXPOSURE LIMITS

There is no Federal standard for chlorinated lime. (See Chlorine.)

ROUTES OF ENTRY

Inhalation of dust. Inhalation of vapor and ingestion.

HARMFUL EFFECTS

Local—

The toxic effects of chlorinated lime are due to its chlorine content. The powder and its solutions have corrosive action on skin, eyes, and mucous membranes, can produce conjunctivitis, blepharitis, corneal ulceration, gingivitis, contact dermatitis, and may damage the teeth.

Systemic—

The dust is irritating to the respiratory tract and can produce laryn-

gitis and pulmonary edema. Chlorinated lime is extremely hygroscopic and with the addition of water evolves free chlorine. Inhalation of the vapor is extremely irritating and toxic. (See Chlorine.) Ingestion of chlorinated lime causes severe oral, esophageal, and gastric irritation.

MEDICAL SURVEILLANCE

Consider possible effects on skin, teeth, eyes, or respiratory tract. There are no specific diagnostic tests.

SPECIAL TESTS

None commonly used.

PERSONAL PROTECTIVE METHODS

In dusty areas, the worker should be protected by appropriate respirators. Simple dust masks should not be used since the moisture present in expired air will release the chlorine. Skin effects can be minimized with protective clothing. Most important is the fact that free chlorine is liberated when chlorinated lime comes in contact with water. All precautions should be followed to protect the worker under these circumstances. (See Chlorine.)

CHLORINE

DESCRIPTION

Cl, chlorine, is a greenish-yellow gas with a pungent odor. It is slightly soluble in water and is soluble in alkalis. It is the commonest of the four halogens which are among the most chemically reactive of all the elements.

SYNONYMS

None.

POTENTIAL OCCUPATIONAL EXPOSURES

Gaseous chlorine is a bleaching agent in the paper and pulp and textile industries for bleaching cellulose for artificial fibers. It is used in the manufacture of chlorinated lime, inorganic and organic compounds such as metallic chlorides, chlorinated solvents, refrigerants, pesticides, and polymers, e.g. synthetic rubber and plastics; it is used as a disinfectant, particularly for water and refuse, and in detinning and dezincing iron.

A partial list of occupations in which exposure may occur includes:

- Aerosol propellant makers
- Bleachers
- Chlorinated solvent makers
- Disinfectant makers
- Dye makers
- Flour bleachers
- Iron workers
- Laundry workers
- Paper bleachers
- Pesticide makers
- Plastic makers
- Rayon makers
- Refrigerant makers
- Silver extractors
- Swimming pool maintenance workers
- Tin recovery workers

PERMISSIBLE EXPOSURE LIMITS

The Federal standard is 1 ppm (3 mg/m³). NIOSH has recommended a ceiling limit of 0.5 ppm for a 15-minute sampling period.

ROUTE OF ENTRY

Inhalation of gas.

HARMFUL EFFECTS

Local—

Chlorine reacts with body moisture to form acids. It is itself extremely irritating to skin, eyes, and mucous membranes, and it may cause corrosion of teeth. Prolonged exposure to low concentrations may produce chloracne.

Systemic—

Chlorine in high concentrations acts as an asphyxiant by causing cramps in the muscles of the larynx (choking), swelling of the mucous membranes, nausea, vomiting, anxiety, and syncope. Acute respiratory distress including cough, hemoptysis, chest pain, dyspnea, and cyanosis develop, and later tracheobronchitis, pulmonary edema, and pneumonia may supervene.

MEDICAL SURVEILLANCE

Special emphasis should be given to the skin, eye, teeth, cardiovascular status in placement and periodic examinations. Chest X-rays should be taken and pulmonary function followed.

SPECIAL TESTS

None in common use.

PERSONAL PROTECTIVE METHODS

Whenever there is likelihood of excessive gas levels, workers should use respiratory protection in the form of fullface gas mask with proper canister or supplied air respirator. The skin effects of chlorine can generally be controlled by good personal hygiene practices. Where very high gas concentrations or liquid chlorine may be present, full protective clothing, gloves, and eye protection should be used. Changing work clothes daily and showering following each shift where exposures exist are recommended.

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FLUORINE and COMPOUNDS

DESCRIPTION

F, elemental fluorine, is a yellow gas. Sulfuric acid reacts with fluorspar producing hydrofluoric acid (HF) which is starting material for synthesis of most fluorine compounds. Fluorine forms fluorides but not fluorates or perfluorates.

SYNONYMS

Fluorine: none.

Hydrogen fluoride: hydrofluoric acid gas, fluohydric acid gas, anhydrous hydrofluoric acid.

Fluorides: none.

POTENTIAL OCCUPATIONAL EXPOSURES

Elemental fluorine is used in the conversion of uranium tetrafluoride to uranium hexafluoride, in the synthesis of organic and inorganic fluorine compounds, and as an oxidizer in rocket fuel.

Hydrogen fluoride, its aqueous solution hydrofluoric acid, and its salts are used in production of organic and inorganic fluorine compounds such as fluorides and plastics; as a catalyst, particularly in paraffin alkylation in the petroleum industry; as an insecticide; and to arrest the fermentation in brewing. It is utilized in the fluorination processes, especially in the aluminum industry, in separating uranium isotopes, in cleaning cast iron, copper, and brass, in removing effloresence from brick and stone, in removing sand from metallic castings, in frosting and etching glass and enamel, in polishing crystal, in decomposing cellulose, in enameling and galvanizing iron, in working silk, in dye and analytical chemistry, and to increase the porosity of ceramics.

Fluorides are used as an electrolyte in aluminum manufacture, a flux in smelting nickel, copper, gold, and silver, as a catalyst for organic reactions, a wood preservative, a fluoridation agent for drinking water, a bleaching agent for cane seats, in pesticides, rodenticides, and as a fermentation inhibitor. They are utilized in the manufacture of steel, iron, glass, ceramics, pottery, enamels, in the coagulation of latex, in coatings for welding rods, and in cleaning graphite, metals, windows, and glassware. Exposure to fluorides may also occur during preparation of fertilizer from phosphate rock by addition of sulfuric acid.

A partial list of occupations in which exposure may occur includes:

- Aluminum fluoride makers
- Aluminum makers
- Bleachers
- Brass cleaners
- Casting cleaners
- Ceramic workers
- Copper cleaners
- Crystal glass polishers
- Fermentation workers
- Fluorochemical workers
- Glass etchers
- Incandescent lamp frosters
- Insecticide makers
- Ore dissolvers
- Stone cleaners
- Uranium refiners
- Yeast makers

PERMISSIBLE EXPOSURE LIMITS

The applicable Federal standards are: fluorine 0.1 ppm (0.2 mg/m^3), fluoride as dust (2.5 mg/m^3), hydrogen fluoride 3 ppm, ceiling 5 ppm, and peak 10 ppm for 30 minutes. For hydrogen fluoride NIOSH has recommended 2.5 mg/m^3 (fluoride ion) TWA with a ceiling of 5 mg/m^3 (fluoride ion) for a 15-minute sampling period.

ROUTES OF ENTRY

Inhalation of gas, mist, dust, or fume; ingestion of dust.

HARMFUL EFFECTS

Local—

Fluorine and some of its compounds are primary irritants of skin, eyes, mucous membranes, and lungs. Thermal or chemical burns may result from contact; the chemical burns cause deep tissue destruction and may not become symptomatic until several hours after contact, depending on dilution. Nosebleeds and sinus trouble may develop on chronic exposure to low concentration of fluoride or fluorine in air. Accidental fluoride burns, even when they involve small body areas (less than 3%), can cause systemic effects of fluoride poisoning by absorption of the fluoride through the skin.

Systemic—

Inhalation of excessive concentration of elemental fluorine or of hydrogen fluoride can produce bronchospasm, laryngospasm, and pulmonary edema. Gastrointestinal symptoms may be present. A brief exposure to 25 ppm has caused sore throat and chest pain, irreparable damage to the lungs, and death.

Most cases of acute fluoride intoxication result from ingestion of fluoride compounds. The severity of systemic effects is directly proportional to the irritating properties and the amount of the compound that has been ingested. Gastrointestinal symptoms of nausea, vomiting, diffuse abdominal cramps, and diarrhea can be expected. Large doses produce central nervous system involvement with twitching of muscle groups, tonic and clonic convulsions, and coma.

The systemic effects of prolonged absorption of fluorides from either dusts or vapors have long been a source of some uncertainty. Fluorides are retained preferentially in bone, and excessive intake may result in an osteosclerosis that is recognizable by X-ray. The first signs of changes in density appear in the lumbar spine and pelvis. Usually some ossification of ligaments occurs. Recent investigations suggest that rather severe skeletal fluorosis can exist in workers without any untoward physiological effects, detrimental effects on their general health, or physical impairment.

Fluorides occur in nature and enter the human body through inhalation or ingestion (natural dusts and water). In children, mottling of the dental enamel may occur from increased water concentrations. These exposures are usually minimal and occur over extended periods.

Residential districts which adjoin manufacturing areas can be subjected to continual exposures at minimal levels, or to heavy exposure in the event of accident or plant failure, as in the case of the Meuse Valley disaster.

MEDICAL SURVEILLANCE

Preemployment and periodic examinations should consider possible effects on the skin, eyes, teeth, respiratory tract, and kidneys. Chest X-rays and pulmonary function should be followed. Kidney function should be evaluated. If exposures have been heavy and skeletal fluorosis is suspected, pelvic X-rays may be helpful. Intake of fluoride from natural sources in food or water should be known.

SPECIAL TESTS

In the case of exposure to fluoride dusts, periodic urinary fluoride excretion levels have been very useful in evaluating industrial exposures and environmental dietary sources.

PERSONAL PROTECTIVE METHODS

In areas with execssive gas or dust levels for any type of fluorine, worker protection should be provided. Respiratory protection by dust masks or gas masks with an appropriate canister or supplied air respirator should be provided. Goggles or fullface masks should be used. In areas where there is likelihood of splash or spill, acid resistant clothing including gloves, gauntlets, aprons, boots, and goggles or face shield should be provided to the worker. Personal hygiene should be encouraged, with showering following each shift and before change to street clothes. Work clothes should be changed following each shift, especially in dusty areas. Attention should be given promptly to any burns from fluorine compounds due to absorption of the fluorine at the burn site and the possibility of developing systemic symptoms from absorption from burn sites.

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HYDROGEN CHLORIDE

DESCRIPTION

HCl, hydrogen chloride, is a colorless, nonflammable gas, soluble in water. The aqueous solution is known as hydrochloric acid or muriatic acid and may contain as much as 38% HCl.

SYNONYMS

Anhydrous hydrochloric acid, chlorohydric acid.

POTENTIAL OCCUPATIONAL EXPOSURES

Hydrogen chloride itself is used in the manufacture of pharmaceutical hydrochlorides, chlorine, vinyl chloride from acetylene, alkyl chlorides from olefins, arsenic trichloride from arsenic trioxide; in the chlorination of rubber; as a gaseous flux for babbitting operations; and in organic synthesis involving isomerization, polymerization, alkylation, and nitration reactions.

The acid is used in the production of fertilizers, dyes, dyestuffs, artificial silk, and paint pigments; in refining edible oils and fats; in electroplating, leather tanning, ore refining, soap refining, petroleum extraction, pickling of metals, and in the photographic, textile, and rubber industries.

A partial list of occupations in which exposure may occur includes:

- Battery makers
- Bleachers
- Chemical synthesizers
- Dye makers
- Electroplaters
- Fertilizer makers
- Food processors
- Galvanizers
- Glue makers
- Metal cleaners
- Oil well workers
- Organic chemical synthesizers
- Photoengravers
- Plastic workers
- Rubber makers
- Soap makers
- Tannery workers
- Textile workers
- Tantalum ore refiners
- Tin ore refiners
- Wire annealers

PERMISSIBLE EXPOSURE LIMITS

The Federal standard for hydrogen chloride is 5 ppm (7mg/m^3) as a ceiling value.

ROUTE OF ENTRY

Inhalation of gas or mist.

HARMFUL EFFECTS

Local—

Hydrochloric acid and high concentrations of hydrogen chloride gas are highly corrosive to eyes, skin, and mucous membranes. The acid may produce burns, ulceration, and scarring on skin and mucous membranes, and it may produce dermatitis on repeated exposure. Eye con-

tact may result in reduced vision or blindness. Dental discoloration and erosion of exposed incisors occur on prolonged exposure to low concentrations. Ingestion may produce fatal effects from esophageal or gastric necrosis.

Systemic—

The irritant effect of vapors on the respiratory tract may produce laryngitis, glottal edema, bronchitis, pulmonary edema, and death.

MEDICAL SURVEILLANCE

Special consideration should be given to the skin, eyes, teeth, and respiratory system. Pulmonary function studies and chest X-rays may be helpful in following recovery from acute overexposure.

SPECIAL TESTS

None in common use.

PERSONAL PROTECTIVE METHODS

Appropriate gas masks with canister or supplied air respirators should be provided when vapor concentrations are excessive. Acid resistant clothing including gloves, gauntlets, aprons, boots, and goggles or face shield should be provided in all areas where there is likelihood of splash or spill of liquid. Personal hygiene and showering after each shift should be encouraged.

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METALLIC COMPOUNDS

ALUMINUM AND COMPOUNDS

DESCRIPTION

Al, aluminum, is a light, silvery-white, soft, ductile, malleable amphoteric metal, soluble in acids or alkali, insoluble in water. The primary sources are the ores cryolite and bauxite; aluminum is never found in the elemental state.

SYNONYMS

None.

POTENTIAL OCCUPATIONAL EXPOSURES

Most hazardous exposures to aluminum occur in smelting and refining processes. Aluminum is mostly produced by electrolysis of Al_2O_3 dissolved in molten cryolite (Na_3AlF_6). Aluminum is alloyed with

copper, zinc, silicone, magnesium, manganese, and nickel; special additives may include chromium, lead, bismuth, titanium, zirconium, and vanadium. Aluminum and its alloys can be extruded or processed in rolling mills, wireworks, forges, or foundries, and are used in the shipbuilding, electrical, building, aircraft, automobile, light engineering, and jewelry industries. Aluminum foil is widely used in packaging. Powdered aluminum is used in the paints and pyrotechnic industries. Alumina (aluminum oxide, Al_2O_3) has been utilized as abrasives, refractories, and catalysts, and in the past in the first firing of china and pottery. Aluminum chloride ($AlCl_3$) is used in petroleum processing and in the rubber industry. Alkyl aluminum compounds find use as catalysts in the production of polyethylene.

A partial list of occupations in which exposure may occur includes:

- Aluminum alloy grinders
- Aluminum workers
- Ammunition makers
- Fireworks makers
- Foundry workers
- Petroleum refinery workers
- Plastic makers
- Rubber makers

PERMISSIBLE EXPOSURE LIMITS

There is no Federal standard specifically for metallic aluminum. It may be considered as a nuisance dust, the applicable standards being: respirable fraction, 15 mppcf or 5 mg/m³; total dust, 50 mppcf or 15 mg/m³.

ROUTE OF ENTRY

Inhalation of dust or fume.

HARMFUL EFFECTS

Local—

Particles of aluminum deposited in the eye may cause necrosis of the cornea. Salts of aluminum may cause dermatoses, eczema, conjunctivitis, and irritation of the mucous membranes of the upper respiratory system by the acid liberated by hydrolysis.

Systemic—

The effects on the human body caused by the inhalation of aluminum dust and fumes are not known with certainty at this time. Present data suggest that pneumoconiosis might be a possible outcome. In the majority of cases investigated, however, it was found that exposure was not to aluminum dust alone, but to a mixture of aluminum, silica fume, iron dusts, and other materials.

MEDICAL SURVEILLANCE

Preemployment and periodic physical examinations should give special consideration to the skin, eyes, and lungs. Lung function should be followed.

SPECIAL TESTS

None commonly used.

PERSONAL PROTECTIVE METHODS

Workers in electrolysis manufacturing plants should be provided with respirators for protection from fluoride fumes. Dust masks are recommended in areas exceeding the nuisance levels. Aluminum workers generally should receive training in the proper use of personal protective equipment. Workers involved with salts of aluminum may require protective clothing, barrier creams, and where heavy concentrations exist, fullface air supplied respirators may be indicated.

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ARSENIC

DESCRIPTION

As, elemental arsenic, occurs to a limited extent in nature as a steel gray metal that is insoluble in water. Arsenic in this discussion includes the element and any of its inorganic compounds excluding arsine. Arsenic trioxide (As_2O_x), the principal form in which the element is used, is frequently designated as arsenic, white arsenic, or arsenous oxide. Arsenic is present as an impurity in many other metal ores and is generally produced as arsenic trioxide as a by-product in the smelting of these ores, particularly copper. Most other arsenic compounds are produced from the trioxide.

SYNONYMS

None.

POTENTIAL OCCUPATIONAL EXPOSURES

Arsenic compounds have a variety of uses. Arsenates and arsenites are used in agriculture as insecticides, herbicides, larvicides, and pesticides. Arsenic trichloride is used primarily in the manufacture of pharmaceuticals. Other arsenic compounds are used in pigment production, the manufacture of glass as a bronzing or decolorizing agent, the manufacture of opal glass and enamels, textile printing, tanning, taxidermy, and antifouling paints. They are also used to control sludge formation in lubricating oils. Metallic arsenic is used as an alloying agent to harden lead shot and in lead-base bearing materials. It is also alloyed with copper to improve its toughness and corrosion resistance.

A partial list of occupations in which exposure may occur includes:

Alloy makers
Aniline color makers
Arsenic workers
Babbitt metal workers
Brass makers
Bronze makers
Ceramic enamel makers
Ceramic makers
Copper smelters
Drug makers
Dye makers
Enamelers
Fireworks makers
Gold refiners
Herbicide makers
Hide preservers
Insecticide makers
Lead shot makers
Lead smelters
Leather workers
Painters
Paint makers
Petroleum refinery workers
Pigment makers
Printing ink workers
Rodenticide makers
Semiconductor compound makers
Silver refiners
Taxidermists
Textile printers
Tree sprayers
Type metal workers
Water weed controllers
Weed sprayers

PERMISSIBLE EXPOSURE LIMITS

The Federal standard for arsenic and its compounds is 0.5 mg/m^3 of air as As. NIOSH has recommended 0.002 mg/m^3 of air as As based on its carcinogenic effects.

ROUTES OF ENTRY

Inhalation and ingestion of dust and fumes.

HARMFUL EFFECTS

Local—

Trivalent arsenic compounds are corrosive to the skin. Brief contact has no effect, but prolonged contact results in a local hyperemia and later vesicular or pustular eruption. The moist mucous membranes are most sensitive to the irritant action. Conjunctiva, moist and macerated areas of skin, the eyelids, the angles of the ears, nose, mouth, and respiratory mucosa are also vulnerable to the irritant effects. The wrists are common sites of dermatitis, as are the genitalia if personal hygiene is poor. Perforations of the nasal septum may occur. Arsenic trioxide and pentoxide are capable of producing skin sensitization and contact dermatitis. Arsenic is also capable of producing keratoses, especially of the palms and soles. Arsenic has been cited as a cause of skin cancer, but the incidence is low.

Systemic—

The acute toxic effects of arsenic are generally seen following ingestion of inorganic arsenical compounds. This rarely occurs in an industrial setting. Symptoms develop within ½ to 4 hours following ingestion and are usually characterized by constriction of the throat followed by dysphagia, epigastric pain, vomiting, and watery diarrhea. Blood may appear in vomitus and stools. If the amount ingested is suf-

ficiently high, shock may develop due to severe fluid loss, and death may ensue in 24 hours. If the acute effects are survived, exfoliative dermatitis and peripheral neuritis may develop.

Cases of acute arsenical poisoning due to inhalation are exceedingly rare in industry. When it does occur, respiratory tract symptoms—cough, chest pain, dyspnea—giddiness, headache, and extreme general weakness precede gastrointestinal symptoms. The acute toxic symptoms of trivalent arsenical poisoning are due to severe inflammation of the mucous membranes and greatly increased permeability of the blood capillaries.

Chronic arsenical poisoning due to ingestion is rare and generally confined to patients taking prescribed medications. However, it can be a concomitant of inhaled inorganic arsenic from swallowed sputum and improper eating habits. Symptoms are weight loss, nausea and diarrhea alternating with constipation, pigmentation and eruption of the skin, loss of hair, and peripheral neuritis. Chronic hepatitis and cirrhosis have been described. Polyneuritis may be the salient feature, but more frequently there are numbness and parasthenias of "glove and stocking" distribution. The skin lesions are usually melanotic and keratotic and may occasionally take the form of an intradermal cancer of the squamous cell type, but without infiltrative properties. Horizontal white lines (striations) on the fingernails and toenails are commonly seen in chronic arsenical poisoning and are considered to be a diagnostic accompaniment of arsenical polyneuritis.

Inhalation of inorganic arsenic compounds is the most common cause of chronic poisoning in the industrial situation. This condition is divided into three phases based on signs and symptoms.

First Phase: The worker complains of weakness, loss of appetite, some nausea, occasional vomiting, a sense of heaviness in the stomach, and some diarrhea.

Second Phase: The worker complains of conjunctivitis, a catarrhal state of the mucous membranes of the nose, larynx, and respiratory passage. Coryza, hoarseness, and mild tracheobronchitis may occur. Perforation of the nasal septum is common, and is probably the most typical lesion of the upper respiratory tract in occupational exposure to arsenical dust. Skin lesions, eczematoid and allergic in type, are common.

Third Phase: The worker complains of symptoms of peripheral neuritis, initially of hands and feet, which is essentially sensory. In more severe cases, motor paralyses occur; the first muscles affected are usually the toe extensors and the peronei. In only the most severe cases will paralysis of flexor muscles of the feet or of the extensor muscles of hands occur.

Liver damage from chronic arsenical poisoning is still debated, and as yet the question is unanswered. In cases of chronic and acute arsenical poisoning, toxic effects to the myocardium have been reported based on EKG changes. These findings, however, are now largely discounted and the EKG changes are ascribed to electrolyte disturbances concom-

itant with arsenicalism. Inhalation of arsenic trioxide and other inorganic arsenical dusts does not give rise to radiological evidence of pneumoconiosis. Arsenic does have a depressant effect upon the bone marrow, with disturbances of both erythropoiesis and myelopoiesis. Evidence is now available incriminating arsenic compounds as a cause of lung cancer as well as skin cancer.

MEDICAL SURVEILLANCE

In preemployment physical examinations, particular attention should be given to allergic and chronic skin lesions, eye disease, psoriasis, chronic eczematous dermatitis, hyperpigmentation of skin, keratosis and warts, baseline weight, baseline blood and hemoglobin count, and baseline urinary arsenic determinations. In annual examinations, the worker's general health, weight, and skin condition should be checked, and the worker observed for any evidence of excessive exposure or absorption of arsenic.

SPECIAL TESTS

Chest X-rays and lung function should be evaluated; analysis of urine, hair, or nails for arsenic should be made every 60 days as long as exposure continues.

PERSONAL PROTECTIVE METHODS

Workers should be trained in personal hygiene and sanitation, the use of personal protective equipment, and early recognition of symptoms of absorption, skin contact irritation, and sensitivity. With the exception of arsine and arsenic trichloride, the compounds of arsenic do not have odor or warning qualities. In case of emergency or areas of high dust or spray mist, workers should wear respirators that are supplied-air or self-contained positive-pressure type with fullface mask. Where concentrations are less than 100 x standard, workers may be able to use halfmask respirators with replaceable dust or fume filters. Protective clothing, gloves and goggles, a hood for head and neck should be provided. When liquids are processed, impervious clothing should be supplied. Clean work clothes should be supplied daily and the workers should shower prior to changing to street clothes.

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ARSINE

DESCRIPTION

AsH_3, arsine, is a colorless gas with a slight garlic-like odor which cannot be considered a suitable warning property in concentrations below 1 ppm. Arsine's solubility is 20 ml. in 100 ml. of water at 20 C.

SYNONYMS

Hydrogen arsenide, arseniuretted hydrogen.

POTENTIAL OCCUPATIONAL EXPOSURES

Arsine is not used in any industrial process but this gas is generated by side reactions or unexpectedly; e.g., it may be generated in metal pickling operations, metal drossing operations, or when inorganic arsenic compounds contact sources of nascent hydrogen. It has been known to occur as an impurity in acetylene. Most occupational exposure occurs in chemical, smelting, and refining industry. Cases of exposure have come from workers dealing with zinc, tin, cadmium, galvanized coated aluminum, and silicon steel metals.

A partial list of occupations in which exposure may occur includes:

Acid dippers	Jewelers
Aniline workers	Lead burners
Bronzers	Paper makers
Dye makers	Plumbers
Etchers	Solderers
Fertilizer makers	Submarine workers
Galvanizers	Tinners

PERMISSIBLE EXPOSURE LIMITS

The Federal standard for arsine is 0.05 ppm. NIOSH has recommended that arsine be controlled to the same concentration as other forms of inorganic arsenic (0.002 mg/m^3).

ROUTE OF ENTRY

Inhalation of gas.

HARMFUL EFFECTS

Local—

High concentrations of arsine gas will cause damage to the eyes. Most experts agree, however, that before this occurs systemic effects can be expected.

Systemic—

Arsine is an extremely toxic gas that can be fatal if inhaled in sufficient quantities. Acute poisoning is marked by a triad of main effects caused by massive intravascular hemolysis of the circulating red cells. Early effects may occur within an hour or two and are commonly characterized by general malaise, apprehension, giddiness, headache, shivering, thirst, and abdominal pain with vomiting. In severe acute cases

the vomitus may be blood stained and diarrhea ensues as with inorganic arsenical poisoning. Pulmonary edema has occurred in severe acute poisoning.

Invariably, the first sign observed in arsine poisoning is hemoglobinuria, appearing with discoloration of the urine up to port wine hue (first of the triad). Jaundice (second of triad) sets in on the second or third day and may be intense, coloring the entire body surface a deep bronze hue. Coincident with these effects is a severe haemolytic-type anemia. Severe renal damage may occur with oliguria or complete suppression of urinary function (third of triad), leading to uremia and death. Severe hepatic damage may also occur, along with cardiac damage and EKG changes. Where death does not occur, recovery is prolonged.

In cases where the amount of inhaled arsine is insufficient to produce acute effects, or where small quantities are inhaled over prolonged periods, the hemoglobin liberated by the destruction of red cells may be degraded by the reticuloendothelial system and the iron moiety taken up by the liver, without producing permanent damage. Some hemoglobin may be excreted unchanged by the kidneys. The only symptoms noted may be general tiredness, pallor, breathlessness on exertion, and palpitations as would be expected with severe secondary anemia.

MEDICAL SURVEILLANCE

In preemployment physical examinations, special attention should be given to past or present kidney disease, liver disease, and anemia. Periodic physical examinations should include tests to determine arsenic levels in the blood and urine. The general condition of the blood and the renal and liver functions should also be evaluated. Since arsine gas is a by-product of certain production processes, workers should be trained to recognize the symptoms of exposure and to use appropriate personal protective equipment.

SPECIAL TESTS

None in common use.

PERSONAL PROTECTIVE METHODS

In most cases, arsine poisoning cannot be anticipated except through knowledge of the production processes. Where arsine is suspected in concentrations above the acceptable standard, the worker should be supplied with a supplied air fullface respirator or a self-contained positive pressure respirator with full facepiece.

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ANTIMONY AND COMPOUNDS

DESCRIPTION

Sb, antimony, is a silvery-white, soft metal insoluble in water and organic solvents. The ores most often found are stibnite, valentinite, kermesite, and senarmontite.

SYNONYMS

None.

POTENTIAL OCCUPATIONAL EXPOSURES

Exposure to antimony may occur during mining, smelting or refining, alloy and abrasive manufacture, and typesetting in printing. Antimony is widely used in the production of alloys, imparting increased hardness, mechanical strength, corrosion resistance, and a low coefficient of friction. Some of the important alloys are babbitt, pewter, white metal, Britannia metal and bearing metal (which are used in bearing shells), printing-type metal, storage battery plates, cable sheathing, solder, ornamental castings, and ammunition. Pure antimony compounds are used as abrasives, pigments, flameproofing compounds, plasticizers, and catalysts in organic synthesis; they are also used in the manufacture of tartar emetic, paints, lacquers, glass, pottery, enamels, glazes, pharmaceuticals, pyrotechnics, matches, explosives. In addition they are used in dyeing, for blueing steel, and in coloring aluminum, pewter, and zinc. A highly toxic gas, stibine, may be released from the metal under certain conditions.

A partial list of occupations in which exposure may occur includes:

- Bronzers
- Ceramic makers
- Drug makers
- Fireworks makers
- Leather mordanters
- Miners
- Paint makers
- Pewter workers
- Rubber makers
- Textile workers
- Typesetters

PERMISSIBLE EXPOSURE LIMITS

The Federal standard for antimony and its compounds is 0.5 mg/m^3, expressed as Sb (see also Stibine).

ROUTE OF ENTRY

Ingestion or inhalation of dust or fume; percutaneous absorption.

HARMFUL EFFECTS

Local—

Antimony and its compounds are generally regarded as primary skin irritants. Lesions generally appear on exposed, moist areas of the body, but rarely on the face. The dust and fumes are also irritants to the eyes, nose, and throat, and may be associated with gingivitis, anemia, and ulceration of the nasal septum and larynx. Antimony trioxide causes a dermatitis known as "antimony spots." This form of dermatitis results in intense itching followed by skin eruptions. A diffuse erythema may occur, but usually the early lesions are small erythematous papules. They may enlarge, however, and become pustular. Lesions occur in hot weather and are due to dust accumulating on exposed areas that are moist due to sweating. No evidence of eczematous reaction is present, nor an allergic mechanism.

Systemic—

Systemic intoxication is uncommon from occupational exposure. However, miners of antimony may encounter dust containing free silica; cases of pneumoconiosis in miners have been termed "silico-antimoniosis." Antimony pneumoconiosis, per se, appears to be a benign process.

Antimony metal dust and fumes are absorbed from the lungs into the blood stream. Principal organs attacked include certain enzyme systems (protein and carbohydrate metabolism), heart, lungs, and the mucous membrane of the respiratory tract. Symptoms of acute oral poisoning include violent irritation of the nose, mouth, stomach, and intestines, vomiting, bloody stools, slow shallow respiration, pulmonary congestion, coma, and sometimes death due to circulatory or respiratory failure. Chronic oral poisoning presents symptoms of dry throat, nausea, headache, sleeplessness, loss of appetite, and dizziness. Liver and kidney degenerative changes are late manifestations.

Antimony compounds are generally less toxic than antimony. Antimony trisulfide, however, has been reported to cause myocardial changes in man and experimental animals. Antimony trichloride and pentachloride are highly toxic and can irritate and corrode the skin. Antimony fluoride is extremely toxic, particularly to pulmonary tissue and skin.

MEDICAL SURVEILLANCE

Preemployment and periodic examinations should give special attention to lung disease, skin disease, disease of the nervous system, heart and gastrointestinal tract. Lung function, EKG's, blood, and urine should be evaluated periodically.

SPECIAL TESTS

Blood and urine antimony levels have been suggested, but are not in common use.

PERSONAL PROTECTIVE METHODS

A combination of protective clothing, barrier creams, gloves, and personal hygiene will protect the skin. Washing and showering facilities should be available, and eating should not be permitted in exposed areas. Dust masks and supplied air respirators should be available in all areas where the Federal standard is exceeded.

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BARIUM AND COMPOUNDS

DESCRIPTION

Ba, barium, a silver white metal, is produced by reduction of barium oxide. The primary sources are the minerals barite ($BaSO_4$) and witherite ($BaCO_3$). Barium may ignite spontaneously in air in the presence of moisture, evolving hydrogen. Barium is insoluble in water but soluble in alcohol. Most of the barium compounds are soluble in water. The peroxide, nitrate, and chlorate are reactive and may present fire hazards in storage and use.

SYNONYMS

None.

POTENTIAL OCCUPATIONAL EXPOSURES

Metallic barium is used for removal of residual gas in vacuum tubes and in alloys with nickel, lead, calcium, mangnesium, sodium, and lithium.

Barium compounds are used in the manufacture of lithopone (a white pigment in paints), chlorine, sodium hydroxide, valves, and green flares; in synthetic rubber vulcanization, X-ray diagnostic work, glassmaking, papermaking, beet-sugar purification, animal, and vegetable oil refining. They are used in the brick and tile, pyrotechnics, and electronics industries. They are found in lubricants, pesticides, glazes, textile dyes and finishes, pharmaceuticals, and in cements which will be exposed to salt water; and barium is used as a rodenticide, a flux for magnesium alloys, a stabilizer and mold lubricant in the rubber and plastics industries, an extender in paints, a loader for paper, soap, rub-

ber, and linoleum, and as a fire extinguisher for uranium or plutonium fires.

A partial list of occupations in which exposure may occur includes:

Animal oil refiners	Paint makers
Brick makers	Plastic makers
Ceramic makers	Soap makers
Glass makers	Textile workers
Ink makers	Tile makers
Linoleum makers	Wax processors

PERMISSIBLE EXPOSURE LIMITS

The Federal standard for soluble barium compounds is 0.5 mg/m^3.

ROUTES OF ENTRY

Ingestion or inhalation of dust or fume.

HARMFUL EFFECTS

Local—

Alkaline barium compounds, such as the hydroxide and carbonate, may cause local irritation to the eyes, nose, throat, and skin.

Systemic—

Barium poisoning is virtually unknown in industry, although the potential exists when the soluble forms are used. When ingested or given orally, the soluble, ionized barium compounds exert a profound effect on all muscles and especially smooth muscle, markedly increasing their contractility. The heart rate is slowed and may stop in systole. Other effects are increased intestinal peristalsis, vascular constriction, bladder contraction, and increased voluntary muscle tension.

The inhalation of the dust of barium sulfate may lead to deposition in the lungs in sufficient quantities to produce "baritosis"—a benign pneumoconiosis. This produces a radiologic picture in the absence of symptoms and abnormal physical signs. X-rays, however, will show disseminated nodular opacities throughout the lung fields, which are discrete, but sometimes overlap.

MEDICAL SURVEILLANCE

Consideration should be given to the skin, eye, heart, and lung in any placement or periodic examination.

SPECIAL TESTS

None have been used.

PERSONAL PROTECTIVE METHODS

Employees should receive instruction in personal hygiene and the importance of not eating in work areas. Good housekeeping and adequate ventilation are essential. Dust masks, respirators, or goggles may be needed where amounts of significant soluble or alkaline forms are encountered, as well as protective clothing.

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BERYLLIUM AND COMPOUNDS

DESCRIPTION

Be, beryllium, is a grey-metal which combines the properties of light weight and high tensile strength. Beryllium is slightly soluble in hot water and in dilute acids and alkalis. All beryllium compounds are soluble to some degree in water. Beryl ore is the primary source of beryllium, although there are numerous other sources.

SYNONYMS

None

POTENTIAL OCCUPATIONAL EXPOSURES

Beryllium metal is widely used in the atomic energy field as a moderator for fission reactions, as a reflector to reduce leakage of neutrons from the reactor core, and, in a mixture with uranium, as a neutron source. Beryllium foil is the window material for X-ray tubes. Beryllium may be alloyed with a number of metals to increase hardness. Beryllium-copper alloy is the most common and is used in parts subjected to abnormal wear, extreme vibration, or shock loading such as in bushings, current-carrying springs, electric contacts and switches, and radio and radar components; it is also used in non-sparking tools. Beryllium-nickel alloy has high tensile strength, increased hardness, and age-hardening characteristics which make it useful in diamond drill-bit matrices, watch-balance wheels, and certain airplane parts. Beryllium bronzes are used in non-spark tools, electrical switch parts, watch springs, diaphragms, shims, cams, and bushings. Other alloys may be formed with zinc, magnesium, iron, aluminum, gold, silver, platinum, nickel, and steel. Beryllium also has potential for use in the aircraft and aerospace industry.

Beryllium compounds are utilized in the manufacture of ceramics and refractories, as chemical reagents and gas mantle hardeners, and in atomic energy reactions. The use of phosphors produced from beryllium oxide in fluorescent lamps has been discontinued.

Hazardous exposure to beryllium is generally associated with the milling and use of beryllium and not the mining and handling of beryl ore.

A partial list of occupations in which exposure may occur includes:

Beryllium alloy workers	Gas mantle makers
Cathode ray tube makers	Missile technicians
Ceramic makers	Nuclear reactor workers
Electric equipment makers	Refractory material makers

PERMISSIBLE EXPOSURE LIMITS

The present Federal standard for beryllium and beryllium compounds is $2\mu g/m^3$ as an 8-hour TWA with an acceptable ceiling con-

centration of 5 $\mu g/m^3$. The acceptable maximum peak is 25 $\mu g/m^3$ for a maximum duration of 30 minutes. The standard recommended in the NIOSH Criteria Document is 2 μg Be/m^3 as an 8-hour TWA with a peak value of 25 μg Be/m^3 as determined by a minimum sampling time of 30 minutes.

ROUTE OF ENTRY

Inhalation of fume or dust.

HARMFUL EFFECTS

Local—

The soluble beryllium salts are cutaneous sensitizers as well as primary irritants. Contact dermatitis of exposed parts of the body are caused by acid salts of beryllium. Onset is generally delayed about two weeks from the time of first exposure. Complete recovery occurs following cessation of exposure. Eye irritation and conjunctivitis can occur. Accidental implantation of beryllium metal or crystals of soluble beryllium compound in areas of broken or abraded skin may cause granulomatous lesions. These are hard lesions with a central nonhealing area. Surgical excision of the lesion is necessary. Exposure to soluble beryllium compounds may cause nasopharyngitis, a condition characterized by swollen and edematous mucous membranes, bleeding points, and ulceration. These symptoms are reversible when exposure is terminated.

Systemic—

Beryllium and its compounds are highly toxic substances. Entrance to the body is almost entirely by inhalation. The acute systemic effects of exposure to beryllium primarily involve the respiratory tract and are manifest by a nonproductive cough, substernal pain, moderate shortness of breath, and some weight loss. The character and speed of onset of these symptoms, as well as their severity, are dependent on the type and extent of exposure. An intense exposure, although brief, may result in severe chemical pneumonitis with pulmonary edema.

Chronic beryllium disease is an intoxication arising from inhalation of beryllium compounds, but it is not associated with inhalation of the mineral beryl. The chronic form of this disease is manifest primarily by respiratory symptoms, weakness, fatigue, and weight loss (without cough or dyspnea at the onset), followed by non-productive cough and shortness of breath. Frequently, these symptoms and detection of the disease are delayed from five to ten years following the last beryllium exposure, but they can develop during the time of exposure. The symptoms are persistent and frequently are precipitated by an illness, surgery, or pregnancy. Chronic beryllium disease usually is of long duration with exacerbations and remissions.

Chronic beryllium disease can be classified by its clinical variants according to the disability the disease process produces.

1. Asymptomatic nondisabling disease is usually diagnosed only by routine chest X-ray changes and supported by urinary or tissue assay.
2. In its mildly disabling form, the disease results in some nonpro-

ductive cough and dyspnea following unusual levels of exertion. Joint pain and weakness are common complaints. Diagnosis is by X-ray changes. Renal calculi containing beryllium may be a complication. Usually, the patient remains stable for years, but eventually shows evidence of pulmonary or myocardial failure.

3. In its moderately severe disabling form, the disease produces symptoms of distressing cough and shortness of breath, with marked x-ray changes. The liver and spleen are frequently affected, and spontaneous pneumothorax may occur. There is generally weight loss, bone and joint pain, oxygen desaturation, increase in hematocrit, disturbed liver function, hypercalciuria, and spontaneous skin lesions similar to those of Boeck's sarcoid. Lung function studies show measurable decreases in diffusing capacity. Many people in this group survive for years with proper therapy. Bouts of chills and fever carry a bad prognosis.

4. The severely disabling disease will show all of the above mentioned signs and symptoms in addition to severe physical wasting and negative nitrogen balance. Right heart failure may appear causing a severe nonproductive cough which leads to vomiting after meals. Severe lack of oxygen is the predominant problem, and spontaneous pneumothorax can be a serious complication. Death is usually due to pulmonary insufficiency or right heart failure.

MEDICAL SURVEILLANCE

Preemployment history and physical examinations for worker applicants should include chest X-rays, baseline pulmonary function tests (FVC and FEV_1), and measurement of body weight. Beryllium workers should receive a periodic health evaluation that includes: spirometry (FVC and FEV_1), medical history questionaire directed toward respiratory symptoms, and a chest X-ray. General health, liver and kidney function, and possible effects on the skin should be evaluated.

SPECIAL TESTS

Beryllium can be determined in the urine, but shows poor correlation with quantitative exposures. Tissue biopsies for beryllium content have also been utilized in diagnostic procedures, but often show no relation to the severity of the disease and indicate only that exposure has occurred.

PERSONAL PROTECTIVE METHODS

Work areas should be monitored to limit and control levels of exposure. Personnel samplers are recommended. Good housekeeping, proper maintenance, and engineering control of processing equipment and technology are essential. The importance of safe work practices and personal hygiene should be stressed. When beryllium levels exceed the accepted standards, the workers should be provided with respiratory protective devices of the appropriate class, as determined on the basis of the actual or projected atmospheric concentration of airborne beryllium at the worksite. Protective clothing should be provided all workers

who are subject to exposure in excess of the standard. This should include shoes or protective shoe covers as well as other clothing. The clothing should be reissued clean on a daily basis. Workers should shower following each shift prior to change to street clothes.

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BISMUTH AND COMPOUNDS

DESCRIPTION

Bi, bismuth, is a pinkish-silver, hard, brittle metal. It is found as the free metal in ores such as bismuttite and bismuthinite and in lead ores. Bismuth is soluble in some mineral acids and insoluble in water. Most bismuth compounds are soluble in water.

SYNONYMS

None.

POTENTIAL OCCUPATIONAL EXPOSURES

Bismuth is used as a constituent of tempering baths for steel alloys, in lowmelting point alloys which expand on cooling, in aluminum and steel alloys to increase machinability, and in printing type metal. Bismuth compounds are found primarily in pharmaceuticals as antiseptics, antacids, antiluetics, and as a medicament in the treatment of acute angina. They are also used as a contrast medium in roentgenoscopy and in cosmetics.

A partial list of occupations in which exposure may occur includes:

Chemists	Permanent magnet makers
Cosmetic workers	Pigment makers
Disinfectant makers	Solder makers
Fuse makers	Steel alloy makers
Laboratory workers	Tin lusterers

PERMISSIBLE EXPOSURE LIMITS

There is no Federal standard for bismuth or its compounds.

ROUTE OF ENTRY

Ingestion of powder or inhalation of dust.

HARMFUL EFFECTS

Local—

Bismuth and bismuth compounds have little or no effect on intact skin and mucous membrane. Absorption occurs only minimally through broken skin.

Systemic—

There is no evidence connecting bismuth and bismuth compounds

with cases of industrial poisoning. All accounts of bismuth poisoning are from the soluble compounds used previously in therapeutics. Fatalities and near fatalities have been recorded chiefly as a result of intravenous or intramuscular injection of soluble salts. Principal organs affected by poisoning are the kidneys and liver. Chronic intoxication from repeated oral or parenteral doses causes "bismuth line." This is a gum condition with black spots of buccal and colonic mucosa, superficial stomatitis, foul breath, and salivation.

MEDICAL SURVEILLANCE

No special considerations are necessary other than following good general health practices. Liver and kidney function should be followed if large amounts of soluble salts are ingested.

SPECIAL TESTS

None have been proposed.

PERSONAL PROTECTIVE METHODS

Personal hygiene should be stressed, and eating should not be permitted in work areas. Dust masks should be worn in dusty areas to prevent inadvertent ingestion of the soluble bismuth compounds.

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BORON AND COMPOUNDS
(excluding the hydrides)

DESCRIPTION

Boron, B, is a brownish-black powder and may be either crystalline or amorphous. It does not occur free in nature and is found in the minerals borax, colemanite, boronatrocalcite, and boracite. Boron is slightly soluble in water under certain conditions.

Boric acid, H_3BO_3, is a white, amorphous powder. Saturated solutions at 0 C contain 2.6% acid; at 100 C, 28% acid. Boric acid is soluble 1 gm/18 ml in cold water.

Borax, $Na_2B_4O_7.5H_2O$, is a colorless, odorless crystalline solid. Borax is slightly soluble in water.

Boron trifluoride, BF_3, is a colorless gas with a pungent, suffocating odor. It decomposes in water, forming boric acid and fluoboric acid and hydrolyzes in air giving rise to dense, white fumes.

Boron oxide, B_2O_3, is a vitreous, colorless, crystalline, hygroscopic solid and slightly soluble in water.

SYNONYMS

B, none; boric acid, boracic acid; borax, tincal; boron trifluoride, boron fluoride; boron oxide, boric oxide.

POTENTIAL OCCUPATIONAL EXPOSURES

Boron is used in metallurgy as a degasifying agent and is alloyed with aluminum, iron, and steel to increase hardness. It is also a neutron absorber in nuclear reactors.

Boric acid is a fireproofing agent for textiles, a weatherproofing agent for wood, a preservative, and an antiseptic. It is used in the manufacture of glass, pottery, enamels, glazes, cosmetics, cements, porcelain, borates, leather, carpets, hats, soaps, and artificial gems, and in tanning, printing, dyeing, painting, and photography. It is a constituent in powders, ointments, nickeling baths, electric condensors and is used for impregnating wicks and hardening steel.

Borax is used as a soldering flux, preservative against wood fungus, and as an antiseptic. It is used in the manufacture of enamels and glazes and in tanning, cleaning compounds, for fireproofing fabrics and wood, and in artificial aging of wood.

Boron trifluoride is used as a catalyst, a flux for soldering magnesium, a fumigant, for protecting molten magnesium and its alloys from oxidation and in ionization chambers to detect weak neutrons.

Boric acid is used in the manufacture of glass, enamels and glazes, in metallurgy, and in the analysis of silicates to determine SiO_2 and alkalies.

A partial list of occupations in which exposure may occur includes:

Alloy makers	Nuclear instrument makers
Antiseptic makers	Organic chemical synthesizers
Enamel makers	Tannery workers
Fumigant workers	Textile fireproofers
Glass makers	Wood workers

PERMISSIBLE EXPOSURE LIMITS

The applicable Federal standards are: Boron trifluoride 1 ppm (3 mg/m^3) as a ceiling value; and Boron oxide 15 mg/m^3.

ROUTE OF ENTRY

Inhalation of dust, fumes, and aerosols; ingestion.

HARMFUL EFFECTS

Local—

These boron compounds may produce irritation of the nasal mucous membranes, the respiratory tract, and eyes.

Systemic—

These effects vary greatly with the type of compound. Acute poisoning in man from boric acid or borax is usually the result of application of dressings, powders, or ointment to large areas of burned or abraded skin, or accidental ingestion. The signs are: nausea, abdominal pain, diarrhea and violent vomiting, sometimes bloody, which may be accompanied by headache and weakness. There is a characteristic erythematous rash followed by peeling. In severe cases, shock with fall in arterial pressure, tachycardia, and cyanosis occur. Marked CNS irrita-

tion, oliguria, and anuria may be present. The oral lethal dose in adults is over 30 grams. Little information is available on chronic oral poisoning, although it is reported to be characterized by mild GI irritation, loss of appetite, disturbed digestion, nausea, possibly vomiting, and erythymatous rash. The rash may be "hard" with a tendency to become purpuric. Dryness of skin and mucous membranes, reddening of tongue, cracking of lips, loss of hair, conjunctivitis, palpebral edema, gastro-intestinal disturbances, and kidney injury have also been observed.

Although no occupational poisonings have been reported, it was noted that workers manufacturing boric acid had some atrophic changes in respiratory mucous membranes, weakness, joint pains, and other vague symptoms. The biochemical mechanism of boron toxicity is not clear but seems to involve action on the nervous system, enzyme activity, carbohydrate metabolism, hormone function, and oxidation processes, coupled with allergic effects. Borates are excreted principally by the kidneys.

The toxic action of the halogenated borons (boron trifluoride and trichloride) is considerably influenced by their halogenated decomposition products. They are primary irritants of the nasal passages, respiratory tract, and eyes in man. Animal experiments showed a fall in inorganic phosphorous level in blood and on autopsy, pneumonia, and degenerative changes in renal tubules. Long term exposure leads to irritation of the respiratory tract, dysproteinemia, reduction in cholinesterase activity, increased nervous system lability. High concentrations showed a reduction of acetyl carbonic acid and inorganic phosphorous in blood, and dental fluorosis.

Skin and respiratory tract irritation and central nervous system effects have been reported from animal experiments with amine and alkylboranes. The alkylboranes seem to be more toxic than the amino compounds and decaborane, but less toxic than pentaborane. No toxic effects have been attributed to elemental boron.

MEDICAL SURVEILLANCE

No specific considerations are needed for boric acid or borates except for general health and liver and kidney function. In the case of boron trifluoride, the skin, eyes, and respiratory tract should receive special attention. In the case of the boranes, central nervous system and lung function will also be of special concern.

SPECIAL TESTS

None in common use.

PERSONAL PROTECTIVE METHODS

Exposed workers should be educated in the proper use of protective equipment and there should be strict adherence to ventilating provisions in work areas. Workers involved with the manufacture of boric acid should be provided with masks to prevent inhalation of dust and fumes. Where exposure is to halogenated borons, or boranes, masks and supplied air respirators are necessary in areas of dust, gas, or fume

concentration. In some areas protective clothing, gloves, and goggles may be necessary.

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BORON HYDRIDES

DESCRIPTION

Diborane: B_2H_6, boroethane, diboron hexahydride. Diborane is a colorless gas with a nauseating odor. It ignites spontaneously in moist air, and on contact with water, hydrolyzes exothermically forming hydrogen and boric acid.

Pentaborane: B_5H_9, pentaboron monohydride. Pentaborane is a colorless, volatile liquid with an unpleasant, sweetish odor. It ignites spontaneously in air, decomposes at 150 C and hydrolyzes in water.

Decaborane: $B_{10}H_{14}$, decaboron tetradecahydride. This is a white crystal with a bitter odor. It hydrolyzes very slowly in water.

SYNONYMS

Boranes, hydrogen borides.

POTENTIAL OCCUPATIONAL EXPOSURES

Diborane is used as a catalyst for olefin polymerization, a rubber vulcanizer, a reducing agent, a flame-speed accelerator, a chemical intermediate for other boron hydrides, and as a doping agent; and in rocket propellants and in the conversion of olefins to trialkyl boranes and primary alcohols.

Pentaborane is used in rocket propellants and in gasoline additives.

Decaborane is used as a catalyst in olefin polymerization, in rocket propellants, in gasoline additives, and as a vulcanizing agent for rubber.

A partial list of occupations in which exposure may occur includes:

- Dope makers
- Gasoline additive makers
- Gasoline makers
- Organic chemical synthesizers
- Plastic makers
- Rocket fuel makers
- Rubber makers

PERMISSIBLE EXPOSURE LIMITS

The applicable Federal standards are: Diborane 0.1 ppm (0.1 mg/m³); Pentaborane 0.005 ppm (0.01 mg/m³); Decaborane 0.05 (0.03 mg/m³) skin.

ROUTES OF ENTRY

Inhalation and percutaneous absorption.

HARMFUL EFFECTS

Local—

Vapors of boron hydrides are irritating to skin and mucous mem-

branes. Pentaborane and decaborane show marked irritation of skin and mucous membranes, necrotic changes, serious kerato-conjunctivitis with ulceration, and corneal opacification.

Systemic—

Pentaborane is the most toxic of boron hydrides. Intoxication is characterized predominantly by CNS signs and symptoms. Hyperexcitability, headaches, muscle twitching, convulsions, dizziness, disorientation, and unconsciousness may occur early or delayed for 24 hours or more following excessive exposure. Slight intoxication results in nausea and drowsiness. Moderate intoxication leads to headache, dizziness, nervous excitation, and hiccups. There may be muscular pains and cramps, spasms in face and extremities, behavioral changes, loss of mental concentration, incoordination, disorientation, cramps, convulsions, semi-coma, and persistent leukocytosis after 40-48 hours. Liver function tests and elevated nonprotein nitrogen and blood urea levels suggest liver and kidney damage.

Decaborane's toxic effects are similar to pentaborane. Symptoms of CNS damage predominate; however, they are not as marked as the pentaborane.

Diborane is the least toxic of the boron hydrides. In acute poisoning, the symptoms are similar to metal fume fever: tightness, heaviness and burning in chest, coughing, shortness of breath, chills, fever, pericardial pain, nausea, shivering, and drowsiness. Signs appear soon after exposure or after a latent period of up to 24 hours and persist for 1-3 days or more. Pneumonia may develop later. Reversible liver and kidney changes were seen in rats exposed to very high gas levels. This has not been noted in man. Subacute poisoning is characterized by pulmonary irritation symptoms, and if this is prolonged, CNS symptoms such as headaches, dizziness, vertigo, chills, fatigue, muscular weakness, and only infrequent transient tremors, appear. Convulsions do not occur. Chronic exposure leads to wheezing, dyspnea, tightness, dry cough, rales, and hyperventilation which persist for several years.

MEDICAL SURVEILLANCE

Preemployment and periodic physical examinations to determine the status of the workers' general health should be performed. These examinations should be concerned especially with any history of central nervous system disease, personality or behavioral changes, as well as liver, kidney, or pulmonary disease of any significant nature. Chest X-rays and blood, liver, and renal function studies may be helpful.

SPECIAL TESTS

None in common use.

PERSONAL PROTECTIVE METHODS

Constant vigilance in the storage and handling of boron hydrides is required. Continuing worker education in the use of personal protective

equipment is necessary even when maximum engineering safety measures are applied.

Adequate sanitation facilities including showers and facilities for eating away from exposure area should be provided. Workers should wash thoroughly when leaving exposure areas. Protective clothing impervious to the liquid and gas compounds are necessary. When skin is contaminated by splash or spill, immediate clothes change with thorough washing of the skin area is necessary. Showering after the shift and before changing to street clothes should be required. Masks, either dust, vapor or supplied air type depending on the compound being used in the work place, should be used by all exposed personnel and should be fullface type.

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BRASS

DESCRIPTION

Brass is a term used for alloys of copper and zinc. The ratio of the two compounds is generally 2 to 1, although different types of brass may have different proportions. Brass may contain significant quantities of lead. Bronze is also a copper alloy, usually with tin; however, the term bronze is applied to many other copper alloys, some of which contain large amounts of zinc.

SYNONYMS

None.

POTENTIAL OCCUPATIONAL EXPOSURES

Brass may be cast into bearings and other wearing surfaces, steam and water valves and fittings, electrical fittings, hardware, ornamental castings, and other equipment where special corrosion-resistance properties, pressure tightness, and good machinability are required. Wrought forms of brass such as sheets, plates, bars, shapes, wire, and tubing are also widely used.

A partial list of occupations in which exposure may occur includes:

- Bench molders
- Braziers
- Bronzers
- Core makers
- Galvanizers
- Junk metal refiners
- Welders
- Zinc founders
- Zinc smelters

PERMISSIBLE EXPOSURE LIMITS

There is no Federal standard for brass; however, there are standards for its constituents: Lead (inorganic) (0.2 mg/m^3); Zinc Oxide fume (5 mg/m^3); Copper fume (0.1 mg/m^3).

ROUTE OF ENTRY

Inhalation of fume.

HARMFUL EFFECTS

Local—

Brass dust and slivers may cause dermatitis by mechanical irritation.

Systemic—

Since zinc boils at a lower temperature than copper, the fusing of brass is attended by liberation of considerable quantities of zinc oxide. Inhalation of zinc oxide fumes may result in production of signs and symptoms of metal fume fever (see Zinc Oxide). Brass founder's ague is the name often given to metal fume fever occurring in brass-founding industry.

Brass foundings may also release sufficient amounts of lead fume to produce lead intoxication (see Lead-Inorganic).

MEDICAL SURVEILLANCE

See Zinc Oxide and/or Lead-Inorganic.

SPECIAL TESTS

Blood lead valves may be useful if lead fume or dust exposure is suspected. (See Lead.)

PERSONAL PROTECTIVE METHODS

See Zinc Oxide and/or Lead-Inorganic.

CADMIUM AND COMPOUNDS

DESCRIPTION

Cd, cadmium, is a bluish-white metal. The only cadmium mineral, greenockite, is rare; however, small amounts of cadmium are found in zinc, copper, and lead ores. It is generally produced as a by-product of these metals, particularly zinc. Cadmium is insoluble in water but is soluble in acids.

SYNONYMS

None.

POTENTIAL OCCUPATIONAL EXPOSURES

Cadmium is highly corrosion resistant and is used as a protective coating for iron, steel, and copper; it is generally applied by electro-

plating, but hot dipping and spraying are possible. Cadmium may be alloyed with copper, nickel, gold, silver, bismuth, and aluminum to form easily fusible compounds. These alloys may be used as coatings for other materials, welding electrodes, solders, etc. It is also utilized in electrodes of alkaline storage batteries, as a neutron absorber in nuclear reactors, a stabilizer for polyvinyl chloride plastics, a deoxidizer in nickel plating, an amalgam in dentistry, in the manufacture of fluorescent lamps, semiconductors, photocells, and jewelry, in process engraving, in the automobile and aircraft industries, and to charge Jones reductors.

Various cadmium compounds find use as fungicides, insecticides, nematocides, polymerization catalysts, pigments, paints, and glass; they are used in the photographic industry and in glazes. Cadmium is also a contaminant of superphosphate fertilizers.

Exposure may occur during the smelting and refining of cadmium-containing zinc, lead, and copper ores, and during spraying, welding, cutting, brazing, soldering, heat treating, melting, alloying and salvage operations which require burning of cadmium-containing materials.

A partial list of occupations in which exposure may occur includes:

- Alloy makers
- Battery makers
- Dental amalgam makers
- Engravers
- Metalizers
- Paint makers
- Pesticide workers
- Solder workers
- Textile printers
- Welders
- Zinc refiners

PERMISSIBLE EXPOSURE LIMITS

The Federal standard for cadmium fume is 0.1 mg/m^3 (as Cd) as an 8-hour TWA with an acceptable ceiling of 3 mg/m^3. For cadmium dust, the standard is 0.2 mg/m^3 (Cd) as an 8-hour TWA with an acceptable maximum ceiling of 0.6 mg/m^3. NIOSH has recommended a TWA limit of 40 μg/m^3 with a ceiling limit of 200 μg in a 5-minute sampling period.

ROUTES OF ENTRY

Inhalation or ingestion of fumes or dust.

HARMFUL EFFECTS

Local—

Cadmium is an irritant to the respiratory tract. Prolonged exposure can cause anosmia and a yellow stain or ring that gradually appears on the necks of the teeth. Cadmium compounds are poorly absorbed from the intestinal tract, but relatively well absorbed by inhalation. Skin absorption appears negligible. Once absorbed Cd has a very long half-life and is retained in the kidney and liver.

Systemic—

Acute toxicity is almost always caused by inhalation of cadmium fumes or dust which are produced when cadmium is heated. There is generally a latent period of a few hours after exposure before symptoms

develop. During the ensuing period, symptoms may appear progressively. The earliest symptom is slight irritation of the upper respiratory tract. This may be followed over the next few hours by cough, pain in the chest, sweating, and chills which resemble the symptoms of nonspecific upper respiratory infection. Eight to 24 hours following acute exposure severe pulmonary irritation may develop, with pain in the chest, dyspnea, cough, and generalized weakness. Dyspnea may become more pronounced as pulmonary edema develops. The mortality rate in acute cases is about 15%. Patients who survive may develop emphysema and cor pulmonale; recovery can be prolonged.

Chronic cadmium poisoning has been reported after prolonged exposure to cadmium oxide fumes, cadmium oxide dust, cadmium sulfides, and cadmium stearates. Heavy smoking has been reported to considerably increase tissue Cd levels. In some cases, only the respiratory tract is affected. In others the effects may be systemic due to absorption of the cadmium. Lung damage often results in a characteristic form of emphysema which in some instances is not preceded by a history of chronic bronchitis or coughing. This type of emphysema can be extremely disabling. Some studies have not shown these effects.

Systemic changes due to cadmium adsorption include damage to the kidneys with proteinuria, anemia, and elevated sedimentation rate. Of these, proteinuria (low molecular weight) is the most typical. In advanced stages of the disease, there may be increased urinary excretion of amino acids, glucose, calcium, and phosphates. These changes may lead to the formation of renal calculi. If the exposure is discontinued, there is usually no progression of the kidney damage. Mild hypochromic anemia is another systemic condition sometimes found in chronic exposure to cadmium.

In studies with experimental animals, cadmium has produced damage to the liver and central nervous system, testicular atrophy, teratogenic effects in rodents after intravenous injection of cadmium, decrease in total red cells, sarcomata, and testicular neoplasms. Hypertensive effects have also been produced. None of these conditions, however, has been found in man resulting from occupational exposure to cadmium. Heavy smoking would appear to increase the risk of cumulative toxic effects.

MEDICAL SURVEILLANCE

In preemployment physical examinations, emphasis should be given to a history of or the actual presence of significant kidney disease, smoking history, and respiratory disease. A chest X-ray and baseline pulmonary function study is recommended. Periodic examinations should emphasize the respiratory system, including pulmonary function tests, kidneys, and blood.

SPECIAL TESTS

A low molecular weight proteinuria may be the earliest indication of renal toxicity. The trichloroacetic acid test may pick this up, but

more specific quantitative studies would be preferable. If renal disease due to cadmium is present, there may also be increased excretion of calcium, amino acids, glucose, and phosphates.

PERSONAL PROTECTIVE METHODS

Most important is the requirement that each worker be adequately protected by the use of effective respiratory protection: either by dust masks, vapor canister respirators, or supplied air respirators. Clothing should be changed after each shift and clean work clothing issued each day. Food should not be eaten in contaminated work areas. Workers should shower after each shift before changing to street clothes.

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CARBONYLS

DESCRIPTION

Metal carbonyls have the general formula $Me_x(CO)_y$ in which Me is the metal and x and y are whole numbers. They are generally produced by direct reaction between carbon monoxide and the finely divided metal; however, chromium, molybdenum, and tungsten carbonyls can be produced by the Grignard method, and platinum metals, iron and rhenium carbonyls may be obtained from metal sulfides, halides, or oxides. The carbonyls react with oxidizing agents and may ignite spontaneously. Reaction with water or steam results in the liberation of carbon monoxide; and on heating, the carbonyls decompose forming carbon monoxide and the finely divided metal powder which may ignite. Some of the more important carbonyls are:

Chromium carbonyl: $Cr(CO)_6$ Colorless crystals.

Cobalt tricarbonyl: $(Co(CO)_3)_4$. Black crystal.

Cobalt tetracarbonyl: $(Co(CO)_4)_2$. Orange crystals or dark brown microscopic crystals.

Cobalt carbonyl hydride: $HCo(CO)_4$. Below −26.2 C, exists as light yellow solid. At room temperature, a gas. It begins to decompose in air above −26 C.

Cobalt nitrosocarbonyl: $Co(CO)_3(NO)$. Cherry red liquid.

Iron tetracarbonyl: $(Fe(CO)_4)_3$. Dark green lustrous crystals.

Iron pentacarbonyl: $Fe(CO)_5$. Viscous yellow liquid.

Iron nonacarbonyl: $Fe_2(CO)_5$. Yellow to orange crystals.

Iron carbonyl hydride: $H_2Fe(CO)_4$. A gas. Begins to decompose at −10 C.

Iron nitrosyl carbonyl: $Fe(NO)_2(CO)_2$. Dark red crystals.

Molybdenum hexacarbonyl: $Mo(CO)_6$. White crystals.

Nickel carbonyl: $Ni(CO)_4$. Colorless liquid.

Osmium carbonyl chloride: $Os(CO)_2Cl_3$. Dark brown. Deliquescent.

Ruthenium pentacarbonyl: $Ru(CO)_5$. Colorless liquid. Very volatile.

Tungsten carbonyl: $W(CO)_6$. Colorless crystals.

SYNONYMS

Chromium carbonyl: None.

Cobalt tricarbonyl: Tetracobalt dodecacarbonyl.

Cobalt tetracarbonyl: Dicobalt octacarbonyl.

Cobalt carbonyl hydride: Cobalt tetracarbonyl hydride.

Cobalt nitrosocarbonyl: None.

Iron tetracarbonyl: None.

Iron pentacarbonyl: None.

Iron nonacarbonyl: Enneacarbonyl.

Iron carbonyl hydride: None.

Iron nitrosyl carbonyl: None.

Molybdenum hexacarbonyl: Molybdenum carbonyl.

Nickel carbonyl: Nickel tetracarbonyl.

Osmium carbonyl chloride: None.

Ruthenium pentacarbonyl: None.

Tungsten carbonyl: None.

POTENTIAL OCCUPATIONAL EXPOSURES

Metal carbonyls are used in isolating certain metals from complex ores, in the preparation of high purity metals, for the production of carbon steel and metallizing, and as catalysts in organic synthesis. Pure metal powders from carbonyls are used in the electronics industry for radiofrequency transformers. $Fe(CO)_5$ is used as a gasoline additive in Europe and as an antidetonator.

Metal carbonyls may be formed during other processes: in the Bessemer converter in the steel industry; inadvertent introduction of carbon monoxide onto metal catalyst beds; storage of carbon monoxide in steel cylinders producing $Fe(CO)_5$; slowly flowing water or gas in an iron pipe generating $Fe(CO)_5$; and in the Fischer-Tropsch process for the liquefaction of coal.

A partial list of occupations in which exposure may occur includes:

Acetylene welders
Blast furnace workers
Metal refiners
Mond process workers
Nickel refiners
Organic chemical synthesizers
Petroleum refinery workers

PERMISSIBLE EXPOSURE LIMITS

There are no specific standards for the metal carbonyls, other than nickel carbonyl. (See under Nickel Carbonyl this section.)

ROUTES OF ENTRY

Inhalation of vapor or dust. Percutaneous absorption of liquids may occur.

HARMFUL EFFECTS

Local—

Aside from skin irritation caused by the specific metal liberated when the metal carbonyl decomposes, no local effects have been reported.

Systemic—

Metal carbonyls as a group have somewhat similar toxicological effects, although there are differences in degrees of toxicity which range from moderate to extremely mild. Nickel carbonyl is the best known and is highly toxic, capable of causing pulmonary edema. Exposures during the Mond process have been associated with an increased incidence of lung and nasal sinuses cancer. Cancer has been produced in rats in the lung, liver, and kidneys.

The toxicity of carbonyls depends in part on the toxic character of the metal component and in part on the volatility and stability of the carbonyl itself. $Ni(CO)_4$ has a very high vapor pressure, plus stability at room temperature. There are no reports of human injury following exposure to cobalt carbonyls. Cobalt tetracarbonyl has an odor so offensive at low levels of concentration that it provides an effective warning against toxic exposure. Iron pentacarbonyl may cause similar pulmonary symptoms to those of nickel carbonyl. Animal studies indicate that the inhalation of fumes and dusts of carbonyls causes respiratory irritation and disturbances to the central nervous system.

MEDICAL SURVEILLANCE

(See Nickel Carbonyl.) Preemployment physical examinations should give particular attention to the respiratory tract and skin. Periodic examinations should include the respiratory tract and nasal sinuses, smoking history as well as general health. A baseline chest X-ray should be available and pulmonary function followed.

SPECIAL TESTS

Urinary nickel level determinations for a few days after an acute

exposure may be useful. Little information is available as to the value of biochemical studies in the case of the other carbonyls.

PERSONAL PROTECTIVE METHODS

In areas where either dust or vapors of the metal carbonyls are encountered, the worker should wear appropriate supplied air respirators. Where the danger of splash or spill of liquids exists, impervious protective clothing should be used.

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CERIUM AND COMPOUNDS

DESCRIPTION

Ce, cerium, a soft, steel-gray metal, is found in the minerals monazite, cerite, and orthite. It may form either tri- or tetravalent compounds. The cerious salts are usually white and the ceric salts are yellow to orange-red. Cerium decomposes in water and is soluble in dilute mineral acids.

SYNONYMS

None.

POTENTIAL OCCUPATIONAL EXPOSURES

Cerium and its compounds are used as a catalyst in ammonia synthesis, a deoxidizer to improve the mechanical quality and refine grain size of steel, an opacifier in certain enamels, an arc-stabilizer in carbon arc lamps, an abrasive for polishing mirrors and lenses, a sedative and as a medicinal agent for vomiting during pregnancy. It is used in the manufacture of topaz yellow glass, spheroidal cast iron, incandescent gas mantles and in decolorizing glass, to prevent mildew in textiles, and to produce a vacuum in neon lamps and electronic tubes. Alloyed with aluminum, magnesium, and manganese, it increases resistance to creep and fatigue. Ferro-cerium is the pyrophoric alloy in gas cigarette lighters, and an alloy of magnesium, cerium, and zirconium is utilized for jet engine parts.

A partial list of occupations in which exposure may occur includes:

Alloy makers	Lighter flint makers
Ammonia makers	Metal refiners
Enamel makers	Phosphor makers
Glass (vitreous) makers	Rocket fuel makers
Ink makers	Textile workers

PERMISSIBLE EXPOSURE LIMITS

There is no Federal standard for cerium or its compounds.

ROUTE OF ENTRY

Inhalation of dust.

HARMFUL EFFECTS

Local—

No local effects have been reported due to cerium and its compounds.

Systemic—

There are no records of injury to human beings from either the industrial or medicinal use of cerium. The main risk to workers is from dust in mining and production areas. Recent reports in the literature describe "Cer-pneumoconiosis," a condition found in a group of graphic arts workers who use carbon arc lights in their work. Chest X-rays reveal small, miliary, homogeneously distributed infiltrates. Cer-pneumoconiosis cannot be considered a dust disease of the lung similar to silicosis. In the later stages of the reaction to the dust of carbon arc lamps, perifocal emphysema, and slight fibrosis of lungs are noted. It has been speculated that these changes may have been due to inhalation of substances containing radioactive elements of the thorium chain. To date, these views have not been confirmed by animal experimentation, autopsy, or human biopsy. Animal experimentation has demonstrated increased coagulation time from organic preparations of cerium, disturbance of lipid metabolism from cerium and its nitrates, and profound effects on metabolism and intestinal muscle causing loss of motility from cerium chloride.

MEDICAL SURVEILLANCE

Chest X-rays should be taken as a part of preemployment and periodic physical examinations.

SPECIAL TESTS

None in common use.

PERSONAL PROTECTIVE METHODS

In areas of carbon arc lights, workers should wear effective dust filters or respirators. In mining and production areas, workers should wear effective dust filters or respirators suitable for the particulate size of air borne dust.

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CHROMIUM AND ITS COMPOUNDS

DESCRIPTION

This group includes chromium trioxide (CrO_3), chromium (VI) oxide, chromic acid anhydride and its aqueous solutions. Chromium may exist in one of three valence states in compounds, +2, +3, and +6. Chromic acid, along with chromates, is in the hexavalent form.

Chromium trioxide is produced from chromite ore by roasting with alkali or lime, (calcium oxide) leaching, crystallization of the soluble chromate or dichromate followed by reaction with sulfuric acid. Chromic acid anhydride mixed with water gives chromic acid and dichromic acid.

SYNONYMS

None.

POTENTIAL OCCUPATIONAL EXPOSURES

Chromium trioxide is used in chrome plating, copper stripping, aluminum anodizing, as a catalyst, in refractories, in organic synthesis, and photography.

A partial list of occupations in which exposure may occur includes:

- Anodizers
- Copper etchers
- Electroplaters
- Glass workers
- Lithographers
- Metal workers
- Oil purifiers
- Photoengravers
- Photographers
- Process engravers
- Stainless steel workers
- Textile workers
- Welders

PERMISSIBLE EXPOSURE LIMITS

The Federal standard for chromic acid and chromates is 0.1 mg/m^3 as a ceiling concentration. The NIOSH Criteria for a Recommended Standard would set work place limits for chromic acid of 0.05 mg/m^3 as chromium trioxide as a TWA with a ceiling concentration of 0.1 mg/m^3 as chromium trioxide determined by a sampling time of 15 minutes.

ROUTES OF ENTRY

Percutaneous absorption, inhalation, and ingestion.

HARMFUL EFFECTS

Local—

In some workers, chromium compounds act as allergens which cause dermatitis to exposed skin. They may also produce pulmonary sensitization. Chromic acid has a direct corrosive effect on the skin and the mucous membranes of the upper respiratory tract; and although rare, the possibility of skin and pulmonary sensitization should be considered.

Systemic—

Chromium compounds in the +3 state are of a low order of toxicity. In the +6 state, chromium compounds are irritants and corrosive, which can enter the body by ingestion, inhalation, and through the skin. Typical industrial hazards are: inhalation of the dust and fumes released during the manufacture of dichromate from chromite ore; inhalation of chromic acid mist during the electroplating and surface treatment of metals; and skin contact in various manufacturing processes.

Acute exposures to dust or mist may cause coughing and wheezing, headache, dyspnea, pain on deep inspiration, fever, and loss of weight.

Tracheobronchial irritation and edema persist after other symptoms subside. In electroplating operations, workers may experience a variety of symptoms including lacrimation, inflammation of the conjunctiva, nasal itch and soreness, epistaxis, ulceration and perforation of the nasal septum, congested nasal mucosa and turbinates, chronic asthmatic bronchitis, dermatitis and ulceration of the skin, inflammation of laryngeal mucosa, cutaneous discoloration, and dental erosion. Hepatic injury has been reported from exposure to chromic acid used in plating baths, but appears to be rare.

Working in the chromate-producing industry increases the risk of lung cancer.

MEDICAL SURVEILLANCE

Preemployment physical examinations should include: a work history to determine past exposure to chromic acid and hexavalent chromium compounds, exposure to other carcinogens, smoking history, history of skin or pulmonary sensitization to chromium, history or presence of dermatitis, skin ulcers, or lesions of the nasal mucosa and/or perforation of the septum, and a chest X-ray. On periodic examinations an evaluation should be made of skin and respiratory complaints, especially in workers who demonstrate allergic reactions. Chest X-rays should be taken yearly for workers over age 40, and every five years for younger workers. Blood, liver, and kidney function should be evaluated periodically.

SPECIAL TESTS

Urinary chromate values have been studied in relation to exposure, but their value is questionable.

PERSONAL PROTECTIVE METHODS

Full body protective clothing should be worn in areas of chromic acid exposure, and impervious gloves, aprons, and footwear should be worn in areas where spills or splashes may contact the skin. Where chromic acid may contact the eyes by spills or splashes, impervious protective goggles or face shield should be worn. All clothing should be changed at the end of the shift and showering encouraged prior to change to street clothes. Clean clothes should be reissued at the start of the shift. Respirators should be used in areas where dust, fumes, or mist exposure exceeds Federal standards or where brief concentrations exceed the TWA, and for emergencies. Dust fumes and mist filter type respirators or supplied air respirators should be supplied all workers exposed, depending on concentration of exposure.

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COBALT AND COMPOUNDS

DESCRIPTION

Co, cobalt, is a silver-grey, hard, brittle, magnetic metal. It is relatively rare; the important mineral sources are the arsenides, sulfides, and oxidized forms. It is generally obtained as a by-product of other metals, particularly copper. Cobalt is insoluble in water, but soluble in acids.

SYNONYMS

None.

POTENTIAL OCCUPATIONAL EXPOSURES

Nickel-aluminum-cobalt alloys are used for permanent magnets. Alloys with nickel, aluminum, copper, beryllium, chromium, and molybdenum are used in the electrical, automobile, and aircraft industries. Cobalt is added to tool steels to improve their cutting qualities and is used as a binder in the manufacture of tungsten carbide tools.

Various cobalt compounds are used as pigments in enamels, glazes, and paints, as catalysts in afterburners, and in the glass, pottery, photographic, electroplating industries.

Radioactive cobalt (^{60}Co) is used in the treatment of cancer.

A partial list of occupations in which exposure may occur includes:

- Alloy makers
- Catalyst workers
- Ceramic workers
- Drug makers
- Electroplaters
- Glass colorers
- Nickel workers
- Paint dryer makers
- Porcelain colorers
- Rubber colorers
- Synthetic ink makers

PERMISSIBLE EXPOSURE LIMITS

The Federal standard for cobalt, metal fume and dust, is 0.1 mg/m^3.

ROUTE OF ENTRY

Inhalation of dust or fume.

HARMFUL EFFECTS

Local—

Cobalt dust is mildly irritating to the eyes and to a lesser extent to the skin. It is an allergen and has caused allergic sensitivity type dermatitis in some industries where only minute quantities of cobalt are used. The eruptions appear in the flexure creases of the elbow, knee, ankles, and neck. Cross sensitization occurs between cobalt and nickel, and to chromium when cobalt and chromium are combined.

Systemic—

Inhalation of cobalt dust may cause an asthma-like disease with cough and dyspnea. This situation may progress to interstitial pneumonia with marked fibrosis. Pneumoconiosis may develop which is believed to be reversible. Since cobalt dust is usually combined with other dusts, the role cobalt plays in causing the pneumoconiosis is not entirely clear.

Ingestion of cobalt or cobalt compounds is rare in industry. Vomiting, diarrhea, and a sensation of hotness may occur after ingestion or after the inhalation of excessive amounts of cobalt dust. Cardiomyopathy has also been reported, but the role of cobalt remains unclear in this situation.

MEDICAL SURVEILLANCE

In preemployment examinations, special attention should be given to a history of skin diseases, allergic dermatitis, baseline allergic respiratory diseases, and smoking history. A baseline chest X-ray should be taken. Periodic examinations should be directed toward skin and respiratory symptoms and lung function.

SPECIAL TESTS

None are in common use.

PERSONAL PROTECTIVE METHODS

Where dust levels are excessive, dust respirators should be used by all workers. Protective clothing should be issued to all workers and changed on a daily basis. Showering after each shift is encouraged prior to change to street clothes. Gloves and barrier creams may be helpful in preventing dermatitis.

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COPPER AND COMPOUNDS

DESCRIPTION

Cu, copper, is a reddish-brown metal which occurs free or in ores such as malachite, cuprite, and chalcopyrite. It may form both mono- and divalent compounds. Copper is insoluble in water, but soluble in nitric acid and hot sulfuric acid.

SYNONYMS

None.

POTENTIAL OCCUPATIONAL EXPOSURES

Metallic copper is an excellent conductor of electricity and is widely

used in the electrical industry in all gauges of wire for circuitry, coil, and armature windings, high conductivity tubes, commutator bars, etc. It is made into castings, sheets, rods, tubing, and wire, and is used in water and gas piping, roofing materials, cooking utensils, chemical and pharmaceutical equipment, and coinage. Copper forms many important alloys: Be-Ce alloy, brass, bronze, gun metal, bell metal, German silver, aluminum bronze, silicon bronze, phosphor bronze, and manganese bronze.

Copper compounds are used as insecticides, algicides, molluscicides, plant fungicides, mordants, pigments, catalysts, and as a copper supplement for pastures, and in the manufacture of powdered bronze paint and percussion caps. They are also utilized in analytical reagents, in paints for ships' bottoms, in electroplating, and in the solvent for cellulose in rayon manufacture.

A partial list of occupations in which exposure may occur includes:

Asphalt makers	Pigment makers
Battery makers	Rayon makers
Electroplaters	Solderers
Fungicide workers	Wallpaper makers
Gem colorers	Water treaters
Lithographers	Wood preservative workers

PERMISSIBLE EXPOSURE LIMITS

The Federal standard for copper fume is 0.1 mg/m^3, and for copper dusts and mists, 1 mg/m^3.

ROUTE OF ENTRY

Inhalation of dust or fume.

HARMFUL EFFECTS

Local—

Copper salts act as irritants to the intact skin causing itching, erythema, and dermatitis. In the eyes, copper salts may cause conjunctivitis and even ulceration and turbidity of the cornea. Metallic copper may cause keratinization of the hands and soles of the feet,but it is not commonly associated with industrial dermatitis.

Systemic—

Industrial exposure to copper occurs chiefly from fumes generated in welding copper-containing metals. (See Brass.) The fumes and dust cause irritation of the upper respiratory tract, metallic taste in the mouth, nausea, metal fume fever, and in some instances discoloration of the skin and hair. Inhalation of dusts, fumes, and mists of copper salts may cause congestion of the nasal mucous membranes, sometimes of the pharynx, and on occasions, ulceration with perforation of the nasal septum. If the salts reach the gastrointestinal tract, they act as irritants,producing salivation, nausea, vomiting, gastric pain, hemorrhagic gastritis, and diarrhea. It is unlikely that poisoning by ingestion in industry would

progress to a serious point as small amounts induce vomiting and empty the stomach of copper salts.

Chronic human intoxication occurs rarely and then only in individuals with Wilson's disease (hepatolenticular degeneration). This is a genetic condition caused by the pairing of abnormal autosomal recessive genes in which there is abnormally high absorption, retention, and storage of copper by the body. The disease is progressive and fatal if untreated.

MEDICAL SURVEILLANCE

Consider the skin, eyes, and respiratory system in any placement or periodic examinations.

PERSONAL PROTECTIVE METHODS

In areas where copper dust or fume is excessive, workers should be provided with proper dust or fume filters or supplied air respirator with full facepiece.

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GERMANIUM

DESCRIPTION

Ge, germanium, is a greyish-white, lustrous, brittle metalloid. It is never found free and occurs most commonly in argyrodite and germanite. It is generally produced from germanium containing minerals or as a by-product in zinc production or coal processing. Germanium is insoluble in water.

SYNONYMS

None.

POTENTIAL OCCUPATIONAL EXPOSURES

Because of its semiconductor properties, germanium is widely used in the electronic industry in rectifiers, diodes, and transistors. It is alloyed with aluminum, aluminum-magnesium, antimony, bronze, and tin to increase strength, hardness, or corrosion resistance. In the process of alloying germanium and arsenic, arsine may be released; stibine is released from the alloying of germanium and antimony. Germanium is also used in the manufacture of optical glass, lenses for infrared applications, red-fluorescing phosphors, and cathodes for electronic valves, and in electroplating, in the hydrogenation of coal, and as a catalyst, particularly at low temperatures. Certain compounds are used medically.

Industrial exposures to the dust and fumes of the metal or oxide generally occur during separation and purification of germanium, weld-

ing, multiple-zone melting operations, or cutting and grinding of crystals. Germanium tetrahydride (germanium hydride, Germane, monogermane) and other hydrides are produced by the action of a reducing acid on a germanium alloy.

A partial list of occupations in which exposure may occur includes:

- Alloy makers
- Dental alloy makers
- Electroplaters
- Glass makers
- Phosphor makers
- Rectifier makers
- Semiconductor makers
- Transistor makers
- Vacuum tube makers
- Residue workers

PERMISSIBLE EXPOSURE LIMITS

There is no Federal standard for germanium or its compounds; however, the ACGIH recently added a TLV for germanium tetrahydride of 0.2 ppm (0.6 mg/m^3).

ROUTE OF ENTRY

Inhalation of gas, vapor, fume, or dust.

HARMFUL EFFECTS

Local—

The dust of germanium dioxide is irritating to the eyes. Germanium tetrachloride causes irritation of the skin.

Systemic—

Germanium tetrachloride is an upper respiratory irritant and may cause bronchitis and pneumonitis. Prolonged exposure to high level concentrations may result in damage to the liver, kidney, and other organs. Germanium tetrahydride is a toxic hemolytic gas capable of producing kidney damage.

MEDICAL SURVEILLANCE

Consider respiratory, liver, and kidney disease in any placement or periodic examinations.

SPECIAL TESTS

None commonly used, but can be determined in urine.

PERSONAL PROTECTIVE METHODS

In dust areas, protective clothing and gloves may be necessary to protect the skin, and goggles to protect the eyes. In areas where germanium tetrachloride is in high concentrations, dust-fume masks or supplied air respirators with full facepiece should be supplied to all workers. Personal hygiene is to be encouraged, with change of clothes following each shift and showering prior to change to street clothes.

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IRON COMPOUNDS

DESCRIPTION

Fe, iron, is a malleable, silver-grey metal. Ferric oxide is a dense, dark red powder or lumps. Hematite is the most important iron ore and is generally found as red hematite (red iron ore, mainly Fe_2O_3) and brown hematite (brown iron ore, mainly limonite, a hydrated sesquioxide of iron). Magnetic iron oxide, Fe_3O_4 is black. Iron is insoluble in water. Iron oxide is soluble in hydrochloric acid.

SYNONYMS

None.

POTENTIAL OCCUPATIONAL EXPOSURES

Iron is alloyed with carbon to produce steel. The addition of other elements (e.g., manganese, silicon, chromium, vanadium, tungsten, molybdenum, titanium, niobium, phosphorus, zirconium, aluminum, copper, cobalt, and nickel) imparts special characteristic to the steel.

Occupational exposures occur during mining, transporting, and preparing of ores and during the production and refining of the metal and alloys. In addition, certain workers may be exposed while using certain iron-containing materials: welders, grinders, polishers, silver finishers, metal workers, and boiler scalers.

A partial list of occupations in which exposure may occur includes:

- Arc cutters
- Bessemer operators
- Electric arc welders
- Flame cutters
- Friction saw operators
- Metalizers
- Seam welders
- Stainless steel makers
- Steel foundry workers

PERMISSIBLE EXPOSURE LIMITS

The Federal standard for iron oxide fume is 10 mg/m³. There are no standards for other iron compounds.

ROUTE OF ENTRY

Inhalation of dust.

HARMFUL EFFECTS

Local—

Soluble iron salts, especially ferric chloride and ferric sulfate, are cutaneous irritants and their aerosols are irritating to the respiratory tract. Iron compounds as a class are not associated with any particular industrial risk.

Systemic—

The inhalation of iron oxide fumes or dust may cause a benign pneumoconiosis (siderosis). It is probable that the inhalation of pure iron oxide does not cause fibrotic pulmonary changes, whereas the inhalation of iron oxide plus certain other substances may cause injury.

On the basis of epidemiological evidence, exposure to hematite dust increases the risk of lung cancer for workers working underground, but not for surface workers. It may be, however, that hematite dust becomes carcinogenic only in combination with radioactive material, ferric oxide, or silica. There is no evidence that hematite dust or ferric oxide causes cancer in any part of the body other than the lungs.

Iron compounds derive their dangerous properties from the radical with which the iron is associated. Iron pentacarbonyl is one of the more dangerous metal carbonyls. It is highly flammable and toxic. Symptoms of overexposure closely resemble those caused by $Ni(CO)_4$ and consist of giddiness and headache, occasionally accompanied by fever, cyanosis, and cough due to pulmonary edema. Death may occur within 4 to 11 days due to pneumonia, liver damage, vascular injury, and central nervous system degeneration.

MEDICAL SURVEILLANCE

Special consideration should be given to respiratory disease and lung function in placement and periodic examinations. Smoking history should be known. Chest X-rays and pulmonary function should be evaluated periodically especially if symptoms are present.

PERSONAL PROTECTIVE METHODS

Dust masks are recommended for all workers exposed to areas of elevated dust concentrations and especially those workers in underground mines. In areas where iron oxide fumes are excessive, vapor canister masks or supplied air masks are recommended. Generally speaking, protective clothing is not necessary, but attention to personal hygiene, showering, and clothes changing should be encouraged.

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LEAD - INORGANIC

DESCRIPTION

Pb, inorganic lead, includes lead oxides, metallic lead, lead salts, and organic salts such as lead soaps, but excludes lead arsenate and organic lead compounds. Lead is a blue-grey metal which is very soft and malleable. Commercially important lead ores are galena, cerussite, anglesite, crocoisite, wulfenite, pyromorphite, matlockite, and vanadinite. Lead is slightly soluble in water in presence of nitrates, ammonium salts, and carbon dioxide.

SYNONYMS

None.

POTENTIAL OCCUPATIONAL EXPOSURES

Metallic lead is used for lining tanks, piping, and other equipment where pliability and corrosion resistance are required such as in the chemical industry in handling corrosive gases and liquids used in the manufacture of sulfuric acid; in petroleum refining; and in halogenation, sulfonation, extraction, and condensation processes; and in the building industry. It is also used as an ingredient in solder, a filler in the automobile industry, and a shielding material for X-rays and atomic radiation; in manufacture of tetraethyl lead and organic and inorganic lead compounds, pigments for paints and varnishes, storage batteries, flint glass, vitreous enameling, ceramics as a glaze, litharge rubber, plastics, and electronic devices. Lead is utilized in metallurgy and may be added to bronze, brass, steel, and other alloys to improve their characteristics. It forms alloys with antimony, tin, copper, etc. It is also used in metallizing to provide protective coatings and as a heat treatment bath in wire drawing.

Exposures to lead dust may occur during mining, smelting, and refining, and to fume, during high temperature (above 500 C) operations such as welding or spray coating of metals with molten lead.

There are numerous applications for lead compounds, some of the more common being in the plates of electric batteries and accumulators, as compounding agents in rubber manufacture, as ingredients in paints, glazes, enamels, glass, pigments, and in the chemical industry.

A partial list of occupations in which exposure may occur includes:

- Battery makers
- Brass founders
- Ceramic makers
- Enamel workers
- Glass makers
- Imitation pearl makers
- Insecticide workers
- Lubricant makers
- Match makers
- Painters
- Plumbers
- Solderers

PERMISSIBLE EXPOSURE LIMITS

The Federal standard for lead and its inorganic compounds is 0.2 mg/m^3 as a time-weighted average. The NIOSH Criteria Document recommends a time-weighted average value of 0.15 mg Pb/m^3.

ROUTES OF ENTRY

Ingestion of dust; inhalation of dust or fume.

HARMFUL EFFECTS

Local—

None.

Systemic—

The early effects of lead poisoning are nonspecific and, except by laboratory testing, are difficult to distinguish from the symptoms of minor seasonal illnesses. The symptoms are decreased physical fitness, fatigue, sleep disturbance, headache, aching bones and muscles, diges-

tive symptoms (particularly constipation), abdominal pains, and decreased appetite. These symptoms are reversible and complete recovery is possible.

Later findings include anemia, pallor, a 'lead line' on the gums, and decreased hand-grip strength. Lead colic produces an intense periodic abdominal cramping associated with severe constipation and, occasionally, nausea and vomiting. Alcohol ingestion and physical exertion may precipitate these symptoms. The peripheral nerve affected most frequently is the radial nerve. This will occur only with exposure over an extended period of time and causes "wrist drop." Recovery is slow and not always complete. When the central nervous system is affected, it is usually due to the ingestion or inhalation of large amounts of lead. This results in severe headache, convulsions, coma, delirium, and possibly death. The kidneys can also be damaged after long periods of exposure to lead, with loss of kidney function and progressive azotemia.

Because of more efficient material handling methods and biological monitoring, serious cases of lead poisoning are rare in industry today.

MEDICAL SURVEILLANCE

In preemployment physical examinations, special attention is given to neurologic and renal disease and baseline blood lead levels. Periodic physical examinations should include hemoglobin determinations, tests for blood lead levels, and evaluation of any gastrointestinal or neurologic symptoms. Renal function should be evaluated.

SPECIAL TESTS

Periodic evaluation of blood lead levels are widely used as an indicator of increased or excessive lead absorption. Other indicators are blood and urine coproporphyric III and delta amino low valence acid dehydrase (ALAD). Erythrocytic protoporphyrin determinations may also be helpful.

PERSONAL PROTECTIVE METHODS

Workers should be supplied with full body work clothing and caps (hard hats). The dust should be removed (vacuumed) before leaving after the shift. Showering after each shift prior to changing to street clothes should be encouraged. Dust and fume masks or supplied air respirators should be supplied to all employees exposed to concentrations above the TWA standard and in all emergencies. Food should not be eaten in contaminated areas.

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LEAD-ALKYL

DESCRIPTION

Both tetraethyl (TEL) and tetramethyl (TM) lead are colorless liquids; however, they are generally mixed with dyes to identify them. TEL is insoluble in water, but soluble in organic solvents. TML is only slightly soluble in organic solvents. Tetraethyl lead will decompose in bright sunlight yielding needle-like crystals of tri-, di-, and mono-ethyl lead compounds, which have a garlic odor.

SYNONYMS

Tetraethyl lead: TEL. Tetramethyl lead: TML.

POTENTIAL OCCUPATIONAL EXPOSURES

TEL and TML are used singly or together as "antiknock" ingredients in gasoline. Exposure may occur during synthesis, handling, transport, or mixing with gasoline.

A partial list of occupations in which exposure may occur includes:

Gasoline additive workers
Storage tank cleaners

PERMISSIBLE EXPOSURE LIMITS

The Federal standard for tetraethyl lead is 0.075 mg Pb/m^3 and for tetramethyl lead 0.07 mg Pb/m^3.

ROUTES OF ENTRY

Inhalation of vapor and percutaneous absorption of liquid. TML is more volatile than TEL and therefore may present more of an inhalation hazard. If the tri-, di-, and mono-ethyl lead compounds are dried, the dust may be inhaled producing the same symptomatology as TEL.

HARMFUL EFFECTS

Local—

Liquid alkyl lead may penetrate the skin without producing appreciable local injury. However, the decomposition products of TEL (i.e., mono-, di-, tri-ethyl lead compounds) in dust form may be inhaled and result in irritation of the upper respiratory tract and possibly paroxysmal sneezing. This dust, when in contact with moist skin or ocular membranes, may cause itching, burning, and transient redness. TEL itself may be irritating to the eyes.

Systemic—

The absorption of a sufficient quantity of tetraethyl lead, whether briefly at a high rate, or for prolonged periods at a lower rate, may cause acute intoxication of the central nervous system. Mild degrees of intoxication cause headache, anxiety, insomnia, nervous excitation, and minor gastrointestinal symptoms with a metallic taste in the mouth. The most noticeable clinical sign of tetraethyl lead poisoning is encephal-

opathy which may give rise to a variety of symptoms, which include mild anxiety, toxic delirium with hallucinations, delusions, convulsions, and acute toxic psychosis. Physical signs are not prominent; but bradycardia, hypotension, increased reflexes, tremor, and slight weight loss have been reported. No peripheral neuropathy has been observed. When the interval between the termination of (either brief or prolonged) exposure and the onset of symptoms is delayed (up to 8 days) the prognosis is guardedly hopeful, but when the time interval is short (few hours), an early fatal outcome may result. Recovered patients show no residual damage to the nervous system, although recovery may be prolonged.

Diagnosis depends on developing a history of exposure to organic lead compounds, followed by the onset of encephalopathy. Biochemical measurements are helpful but not diagnostic. Blood lead is usually not elevated in proportion to the degree of intoxication. Urine amino-levulinic acid, and coproporphyrin excretion will show values close to normal with no correlation with the severity of intoxication. Erythrocyte protoporphyrin also remains within normal range.

No cases of poisoning from absorption of tetramethyl lead have been found. The compound responsible for almost all cases of organic lead poisoning is tetraethyl lead. Animal experimentation, however, indicates that a similar intoxication can be caused by tetramethyl lead.

MEDICAL SURVEILLANCE

In both preemployment and periodic physical examinations, the worker's general health should be evaluated, and special attention should be given to neurologic and emotional disorders.

SPECIAL TESTS

None seem to be useful.

PERSONAL PROTECTIVE METHODS

A training program should stress the importance of personal hygiene and encourage the proper use of personal protective equipment. Showers, lavatories, and locker rooms are necessary. Workers should be required to make a complete change of clothing at the beginning and end of each shift and to shower prior to changing to street clothes. Eating should not be permitted in work areas. In areas where vapor concentrations of TEL exceed the standard, dust masks, organic vapor canister masks, or supplied air respirators should be furnished and required to be worn. In areas of spills or splash, impervious clothing should be worn and goggles furnished.

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MAGNESIUM AND COMPOUNDS

DESCRIPTION

Magnesium is a light, silvery-white metal and is a fire hazard. It is found in dolomite, magnesite, brucite, periclase, carnallite, kieserite and as a silicate in asbestos, talc, olivine, and serpentine. It is also found in sea water, brine wells, and salt deposits. It is insoluble in water and ordinary solvents.

SYNONYMS

None.

POTENTIAL OCCUPATIONAL EXPOSURES

Magnesium alloyed with manganese, aluminum, thorium, zinc, cerium, and zirconium is used in aircraft, ships, automobiles, hand tools, etc. because of its lightness. Dow metal is the general name for a large group of alloys containing over 85% magnesium. Magnesium wire and ribbon are used for degassing valves in the radio industry and in various heating appliances; as a deoxidizer and desulfurizer in copper, brass, and nickel alloys; in chemical reagents; as the powder in the manufacture of flares, incendiary bombs, tracer bullets, and flashlight powders; in the nuclear energy process; and in a cement of magnesium oxide and in magnesium chloride for floors.

A partial list of occupations in which exposure may occur includes:

- Alloy makers
- Antiseptic makers
- Battery makers
- Drug makers
- Flare makers
- Fungicide makers
- Organic chemical synthesizers
- Pigment makers
- Steel makers
- Textile workers
- Welders

PERMISSIBLE EXPOSURE LIMITS

The Federal standard for magnesium oxide fume is 15 mg/m^3.

ROUTE OF ENTRY

Inhalation of fume.

HARMFUL EFFECTS

Local—

Magnesium and magnesium compounds are mild irritants to the conjunctiva and nasal mucosa, but are not specifically toxic. Magnesium in finely divided form is readily ignited by a spark or flame, and splatters and burns at above 2,300 F. On the skin, these hot particles are capable of producing second and third degree burns, but they respond to treatment as other thermal burns do. Metallic magnesium foreign bodies in the skin cause no unusual problems in man. In animal experiments, however, they have caused "gas gangrene"—massive localized gaseous tumors with extensive necrosis.

Systemic—

Magnesium in the form of nascent magnesium oxide can cause metal fume fever if inhaled in sufficient quantity. Symptoms are analogous to those caused by zinc oxide: cough, oppression in the chest, fever, and leukocytosis. There is no evidence that inhalation of magnesium dust has led to lung injury. It has been noted that magnesium workers show a rise in serum magnesium — although no significant symptoms of ill health have been identified. Some investigators have reported higher incidence of digestive disorders and have related this to magnesium absorption, but the evidence is scant. In foundry casting operations, hazards exist from the use of fluoride fluxes and sulfur-containing inhibitors which produce fumes of fluorides and sulfur dioxide.

MEDICAL SURVEILLANCE

No specific recommendations.

SPECIAL TESTS

None.

PERSONAL PROTECTIVE METHODS

Employees should receive training in the use of personal protective equipment, proper methods of ventilation, and fire suppression. Protective clothing should be designed to prevent burns from splatters. Masks to prevent inhalation of fumes may be necessary under certain conditions, but generally this can be controlled by proper ventilation. Dust masks may be necessary in areas of dust concentration as in transfer and storage areas, but adequate ventilation generally provides sufficient protection.

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MANGANESE AND COMPOUNDS

DESCRIPTION

Mn, manganese, is a reddish-grey or silvery, soft metal. The most important ore containing manganese is pyrolusite. Manganese may also be produced from ferrous scrap used in the production of electric and open-hearth steel. Manganese decomposes in water and is soluble in dilute acid.

SYNONYMS

None

POTENTIAL OCCUPATIONAL EXPOSURES

Most of the manganese produced is used in the iron and steel industry in steel alloys, e.g., ferromanganese, silicomanganese, Manganin, spiegeleisen, and as an agent to reduce oxygen and sulfur content of molten steel. Other alloys may be formed with copper, zinc, and aluminum. Manganese and its compounds are utilized in the manufacture of dry cell batteries (MnO_2), paints, varnishes, inks, dyes, matches and fireworks, as a fertilizer, disinfectants, bleaching agent, laboratory reagent, drier for oils, an oxidizing agent in the chemical industry, particularly in the synthesis of potassium permanganate, and as a decolorizer and coloring agent in the glass and ceramics industry.

Exposure may occur during the mining, smelting and refining of manganese, in the production of various materials, and in welding operations with manganese coated rods.

A partial list of occupations in which exposure may occur includes:

Battery makers	Glass makers
Ceramic makers	Ink makers
Drug makers	Match makers
Electric arc welders	Paint makers
Feed additive makers	Varnish makers
Foundry workers	Water treaters

PERMISSIBLE EXPOSURE LIMITS

The Federal standard for manganese is 5 mg/m^3 as a ceiling value.

ROUTES OF ENTRY

Inhalation of dust or fume; limited percutaneous absorption of liquids.

HARMFUL EFFECTS

Local—

Manganese dust and fumes are only minor irritants to the eyes and mucous membranes of the respiratory tract, and apparently completely innocuous to the intact skin.

Systemic—

Chronic manganese poisoning has long been recognized as a clinical entity. The dust or fumes (manganous compounds) enter the respiratory tract and are absorbed into the blood stream. Manganese is then deposited in major body organs and has a special predilection for the liver, spleen, and certain nerve cells of the brain and spinal cord. Among workers there is a very marked variation in individual susceptibility to manganese. Some workers have worked in heavy exposure for a lifetime and have shown no signs of the disease; others have developed manganese intoxication with as little as 49 days of exposure.

The early phase of chronic manganese poisoning is most difficult to recognize, but it is also most important to recognize since early removal from the exposure may arrest the course of the disease. The onset is insidious, with apathy, anorexia, and asthenia. Headache, hypersomnia, spasms, weakness of the legs, arthralgias, and irritability are frequently noted. Manganese psychosis follows with certain definitive features: unaccountable laughter, euphoria, impulsive acts, absentmindedness, mental confusion, aggressiveness, and hallucinations. These symptoms usually disappear with the onset of true neurological disturbances, or may resolve completely with removal from manganese exposure.

Progression of the disease presents a range of neurological manifestations that can vary widely among individuals affected. Speech disturbances are common: monotonous tone, inability to speak above a whisper, difficult articulation, incoherence, even complete muteness. The face may take on masklike quality, and handwriting may be affected by micrographia. Disturbances in gait and balance occur, and frequently propulsion, retropropulsion, and lateropropulsion are affected, with no movement for protection when falling. Tremors are frequent, particularly of the tongue, arms, and legs. These will increase with intentional movements and are more frequent at night. Absolute detachment, broken by sporadic or spasmodic laughter, ensues, and as in extrapyramidal affections, there may be excessive salivation and excessive sweating. At this point the disease is indistinguishable from classical Parkinson's disease.

Chronic manganese poisoning is not a fatal disease although it is extremely disabling.

Manganese dust is no longer believed to be a causative factor in pneumonia. If there is any relationship at all, it appears to be as an aggravating factor to a preexisting condition. Freshly formed fumes have been reported to cause fever and chills similar to metal fume fever.

MEDICAL SURVEILLANCE

Preemployment physical exams should be directed toward the individual's general health with special attention to neurologic and personality abnormalities. Periodic physical examinations may be required as often as every two months. Special emphasis should be given to behavioral and neurological changes: speech defects, emotional distur-

bances, hypertonia, tremor, equilibrium, difficulty in walking or squatting, adiadochokinesis, and handwriting.

SPECIAL TESTS

There are no laboratory tests which can be used to diagnose manganese poisoning.

PERSONAL PROTECTIVE METHODS

In areas where the ceiling value standards are exceeded, dust masks or respirators are necessary. Education in the use and necessity of these devices is important.

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MERCURY - INORGANIC

DESCRIPTION

Hg, inorganic mercury, is here taken to include elemental mercury, inorganic mercury compounds, and organic mercury compounds, excluding alkyl mercury compounds. Metallic mercury is a silver-white liquid at room temperature. It occurs as the free metal or as cinnabar (HgS). Mercury is produced from the ore by roasting or reduction.

SYNONYMS

Quicksilver, hydrargyrum.

POTENTIAL OCCUPATIONAL EXPOSURES

Elemental and inorganic mercury compounds are used in the manufacture of scientific instruments (barometers, thermometers, etc.), electric equipment (meters, switches, batteries, rectifiers, etc.), mercury vapor lamps, incandescent electric lamps, X-ray tubes, artificial silk, radio valves, amalgams with copper, tin, silver, or gold, and solders with lead and tin. In the chemical industry, it is used as a fluid cathode for the electrolytic production of caustic soda (sodium hydroxide), chlorine, and acetic acid. It is utilized in gold, silver, bronze, and tin plating, tanning and dyeing, feltmaking, taxidermy, textile manufacture, photography and photoengraving, in extracting gold and silver from ores, in paints and pigments, in the preparation of drugs and disinfectants in the pharmaceutical industry, and as a chemical reagent.

The aryl mercury compounds such as phenylmercury are primarily used as disinfectants, fungicides for treating seeds, antiseptics, herbicides, preservatives, mildew-proofing agents, denaturants for ethyl alcohol, germicides, and bactericides.

Hazardous exposure may occur during mining and extraction of

mercury and in the use of mercury and its compounds. Elemental mercury readily volatilizes at room temperature.

A partial list of occupations in which exposure may occur includes:

- Amalgam makers
- Bactericide makers
- Battery makers
- Caustic soda makers
- Dental amalgam makers
- Fungicide makers
- Gold extractors
- Jewelers
- Paper makers
- Photographers
- Taxidermists

PERMISSIBLE EXPOSURE LIMITS

The Federal standard for mercury is 1 mg/10m^3 as a ceiling value. The recommended standard is 0.05 mg Hg/m^3 as a TWA.

ROUTES OF ENTRY

Inhalation of dust or vapor; percutaneous absorption of elemental mercury.

HARMFUL EFFECTS

Local—

Mercury is a primary irritant of skin and mucous membranes. It may occasionally be a skin sensitizer.

Systemic—

Acute poisoning due to mercury vapors affects the lungs primarily, in the form of acute interstitial pneumonitis, bronchitis, and bronchiolitis.

Exposure to lower levels over prolonged periods produces symptom complexes that can vary widely from individual to individual. These may include weakness, fatigability, loss of appetite, loss of weight, insomnia, indigestion, diarrhea, metallic taste in the mouth, increased salivation, soreness of mouth or throat, inflammation of gums, black line on the gums, loosening of teeth, irritability, loss of memory, and tremors of fingers, eyelids, lips, or tongue. More extensive exposures, either by daily exposures or one-time,can produce extreme irritability, excitability, anxiety, delirium with hallucinations, melancholia, or manic depressive psychosis. In general, chronic exposure produces four classical signs: gingivitis, sialorrhea, increased irritability, and muscular tremors. Rarely are all four seen together in an individual case.

Either acute or chronic exposure may produce permanent changes to affected organs and organ systems.

MEDICAL SURVEILLANCE

Preemployment and periodic examinations should be concerned especially with the skin, respiratory tract, central nervous system, and kidneys. The urine should be examined and urinary mercury levels determined periodically. Signs of weight loss, gingivitis, tremors, personality changes, and insomnia would be suggestions of possible mercury intoxication.

SPECIAL TESTS

Urine mercury determination may be helpful as an index of amount of absorption. Opinions vary as to the significance of a given level. Generally, 0.1 to 0.5 mg Hg/liter of urine is considered significant.

PERSONAL PROTECTIVE METHODS

In areas where the exposures are excessive, respiratory protection shall be provided either by full face canister type mask or supplied air respirator, depending on the concentration of mercury fumes. Above 50 mg Hg/cu m requires supplied air positive pressure fullface respirators. Full body work clothes including shoes or shoe covers and hats should be supplied, and clean work clothes should be supplied daily. Showers should be available and all employees encouraged to shower prior to change to street clothes. Work clothes should not be stored with street clothes in the same locker. Food should not be eaten in the work area.

MERCURY - ALKYL

DESCRIPTION

Methyl mercury compounds: methyl mercury dicyandiamide - $CH_3HgNHC(:NH)NHCN$. Soluble in water.

Ethyl mercury compounds: ethylmercuric chloride: C_2H_5HgCl. Insoluble in water. Ethylmercuric phosphate: $(C_2H_5Hg)PO$. Soluble in water. N-(Ethylmercuric)-p-toluenesulphonanilide: $C_6H_5N(HgC_2H_5)$-$SO_2C_6H_4CH_3$. Practically insoluble in water.

SYNONYMS

Methyl mercury compounds: Methyl mercury dicyandiamide: none. Cyano (methyl mercury) guanidine: panogen.

Ethyl mercury compounds: Ethylmercuric chloride: ceresan. Ethylmercuric phosphate: new ceresan. N-(Ethylmercuric)-p-toluenesulphonanilide: ceresan m.

POTENTIAL OCCUPATIONAL EXPOSURES

These compounds are used in treating seeds for fungi and seed-borne diseases, as timber preservatives, and disinfectants.

A partial list of occupations in which exposure may occur includes:

Disinfectant makers	Seed handlers
Fungicide makers	Wood preservers

PERMISSIBLE EXPOSURE LIMITS

The Federal standard is 0.01 mg/m^3 as an 8-hour TWA with an acceptable ceiling of 0.04 mg/m^3.

ROUTES OF ENTRY

Inhalation of dust, percutaneous absorption.

HARMFUL EFFECTS

Local—

Alkyl mercury compounds are primary skin irritants and may cause dermatitis. When deposited on the skin, they give no warning, and if contact is maintained, can cause second-degree burns. Sensitization may occur.

Systemic—

The central nervous system, including the brain, is the principal target tissue for this group of toxic compounds. Severe poisoning may produce irreversible brain damage resulting in loss of higher functions.

The effects of chronic poisoning with alkyl mercury compounds are progressive. In the early stages, there are fine tremors of the hands, and in some cases, of the face and arms. With continued exposure, tremors may become coarse and convulsive; scanning speech with moderate slurring and difficulty in pronunciation may also occur. The worker may then develop an unsteady gait of a spastic nature which can progress to severe ataxia of the arms and legs. Sensory disturbances including tunnel vision, blindness, and deafness are also common.

A later symptom, constriction of the visual fields, is rarely reversible and may be associated with loss of understanding and reason which makes the victim completely out of touch with his environment. Severe cerebral effects have been seen in infants born to mothers who had eaten large amounts of methyl mercury contaminated fish.

MEDICAL SURVEILLANCE

Preplacement and periodic physical examinations should be concerned particularly with the skin, vision, central nervous system, and kidneys. Consideration should be given to the possible effects on the fetus of alkyl mercury exposure in the mother. Constriction of visual fields may be a useful diagnostic sign. (See Mercury-Inorganic.)

SPECIAL TESTS

Blood and urine levels of mercury have been studied, especially in the case of methyl mercury. A precise correlation has not been found between exposure levels and concentrations. They may be of some value in indicating that exposure has occurred, however.

PERSONAL PROTECTIVE METHODS

(See Mercury-Inorganic.)

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MOLYBDENUM AND COMPOUNDS

DESCRIPTION

Mo, molybdenum, is a silver-white metal or a greyish-black powder. Molybdenite is the only important commercial source. This ore is often associated with copper ore. Molybdenum is insoluble in water and soluble in hot concentrated nitric and sulfuric acid.

SYNONYMS

None

POTENTIAL OCCUPATIONAL EXPOSURES

Most of the molybdenum produced is used in alloys: steel, stainless steel, tool steel, cast iron, steel mill rolls, manganese, nickel, chromium, and tungsten. The metal is used in electronic parts (contacts, spark plugs, X-ray tubes, filaments, screens, and grids for radios), induction heating elements, electrodes for glass melting, and metal spraying applications. Molybdenum compounds are utilized as lubricants; as pigments for printing inks, lacquers, paints, for coloring rubber, animal fibers, and leather, and as a mordant; as catalysts for hydrogenation cracking, alkylation, and reforming in the petroleum industry, in Fischer-Tropsch synthesis, in ammonia production, and in various oxidation-reduction and organic cracking reactions; as a coating for quartz glass; in vitreous enamels to increase adherence to steel; in fertilizers, particularly for legumes; in electroplating to form protective coatings; and in the production of tungsten.

Hazardous exposures may occur during high-temperature treatment in the fabrication and production of molybdenum products, spraying applications, or through loss of catalyst. MoO_3 sublimes above 800 C.

A partial list of occupations in which exposure may occur includes:

- Ceramic makers
- Drug makers
- Electroplaters
- Fertilizer makers
- Glass makers
- Metal platers
- Petroleum refinery workers
- Steel alloy makers
- Tannery workers
- Vacuum tube makers

PERMISSIBLE EXPOSURE LIMITS

The Federal standards are: Molybdenum: soluble compounds, 5 mg/m^3. Molybdenum: insoluble compounds, 15 mg/m^3.

ROUTE OF ENTRY

Inhalation of dust or fume.

HARMFUL EFFECTS

Local—

Molybdenum trioxide may produce irritation of the eyes and mucous membranes of the nose and throat. Dermatitis from contact with molybdenum is unknown.

Systemic—

No reports of toxic effects of molybdenum in the industrial setting have appeared. It is considered to be an essential trace element in many species, including man. Animal studies indicate that insoluble molybdenum compounds are of a low order of toxicity (e.g., disulfide, oxides, and halides). Soluble compounds (e.g., sodium molybdate) and freshly generated molybdenum fumes, however, are considerably more toxic. Inhalation of high concentrations of molybdenum trioxide dust is very irritating to animals and has caused weight loss, diarrhea, loss of muscular coordination, and a high mortality rate. Molybdenum trioxide dust is more toxic than the fumes. Large oral doses of ammonium molybdate in rabbits caused some fetal deformities. Excessive intake of molybdenum may produce signs of a copper deficiency.

MEDICAL SURVEILLANCE

Preemployment and periodic physical examinations should evaluate any irritant effects to the eyes or respiratory tract and the general health of the worker. Although molybdenum compounds are of low order of toxicity, animal experimentation indicates protective measures should be employed against the more soluble compounds and molybdenum trioxide dust and fumes. The normal intake of copper in the diet appears to be sufficient to prevent systemic toxic effects due to molybdenum poisoning.

SPECIAL TESTS

None in common use.

PERSONAL PROTECTIVE METHODS

Where dust and fumes exceed the standard, molybdenum workers should be supplied with dust masks or supplied air respirators. Full body work clothes are advisable with daily change of clothes and showering before changing to street clothes.

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NICKEL AND COMPOUNDS

DESCRIPTION

Ni, nickel, is a hard, ductile, magnetic metal with a silver-white color. It is insoluble in water and soluble in acids. It occurs free in meteorites and in ores combined with sulfur, antimony, or arsenic. Processing and refining of nickel is accomplished by either the Orford (sodium sulfide and electrolysis) or the Mond (nickel carbonyl) processes. In the latter, impure nickel powder is reacted with carbon monoxide to form gaseous nickel carbonyl which is then treated to deposit high purity metallic nickel.

SYNONYMS

None.

POTENTIAL OCCUPATIONAL EXPOSURES

Nickel forms alloys with copper, manganese, zinc, chromium, iron, molybdenum, etc. Stainless steel is the most widely used nickel alloy. An important nickel-copper alloy is Monel metal, which contains 66% nickel and 32% copper and has excellent corrosion resistance properties. Permanent magnets are alloys chiefly of nickel, cobalt, aluminum, and iron.

Elemental nickel is used in electroplating, anodizing aluminum, casting operations for machine parts, and in coinage; in the manufacture of acid-resisting and magnetic alloys, magnetic tapes, surgical and dental instruments, nickel-cadmium batteries, nickel soaps in crankcase oils, and ground-coat enamels, colored ceramics, and glass. It is used as a catalyst in the hydrogenation of fats, oils, and other chemicals, in synthetic coal oil production, and as an intermediate in the synthesis of acrylic esters for plastics.

Exposure to nickel may also occur during mining, smelting, and refining operations.

A partial list of occupations in which exposure may occur includes:

- Battery makers
- Ceramic makers
- Chemists
- Dyers
- Enamelers
- Ink makers
- Magnet makers
- Oil hydrogenators
- Paint makers
- Pen point makers
- Spark plug makers
- Textile dyers
- Varnish makers

PERMISSIBLE EXPOSURE LIMITS

The Federal standard for nickel metal and its soluble compounds is I mg/m^3 expressed as Ni.

ROUTE OF ENTRY

Inhalation of dust or fume.

HARMFUL EFFECTS

Local—

Skin sensitization is the most commonly seen toxic reaction to nickel and nickel compounds and is seen frequently in the general population. This often results in chronic eczema "Nickel itch," with lichenification resembling atopic or neurodermatitis. Nickel and its compounds are also irritants to the conjunctiva of the eye and the mucous membrane of the upper respiratory tract.

Systemic—

Elemental nickel (as deposited from inhalation of nickel carbonyl) and nickel salts are probably carcinogenic, producing an increased incidence of cancer of the lung and nasal passages. Effects on the heart

muscle, brain, liver, and kidney have been seen in animal studies. Pulmonary eosinophilia (Loeffler's syndrome) has been reported in one study to be caused by the sensitizing property of nickel. Finely divided nickel has also shown some carcinogenic effects in rats by injection, and in guinea pigs by inhalation.

MEDICAL SURVEILLANCE

Preemployment physical examinations should evaluate any history of skin allergies or asthma, other exposures to nickel or other carcinogens, smoking history, and the respiratory tract. Lung function should be studied and chest X-rays periodically evaluated. Special attention should be given to the nasal sinuses and skin.

SPECIAL TESTS

Serum and urinary nickel can be determined, although opinions vary as to their value in monitoring exposures.

PERSONAL PROTECTIVE METHODS

Full body protective clothing is advisable, as is the use of barrier creams to prevent skin sensitization and dermatitis. In areas of dust or fumes, masks or supplied air respirators are mandatory where concentrations exceed the standard limits. Clean work clothing should be provided daily; and showering should be required before changing to street clothes. No food should be eaten in work areas.

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NICKEL CARBONYL

DESCRIPTION

$Ni(CO)_4$, nickel carbonyl, is a colorless, highly volatile, flammable liquid with a musty odor. It decomposes above room temperature producing carbon monoxide and finely divided nickel. It is soluble in organic solvents.

SYNONYMS

Nickel tetracarbonyl.

POTENTIAL OCCUPATIONAL EXPOSURES

The primary use of nickel carbonyl is in the production of nickel by the Mond process. Impure nickel powder is reacted with carbon monoxide to form gaseous nickel carbonyl which is then treated to deposit high purity metallic nickel and release carbon monoxide. Other uses include gas plating, the production of nickel products; in chemical

synthesis as a catalyst, particularly for oxo reactions (addition reaction of hydrogen and carbon monoxide with unsaturated hydrocarbons to form oxygen-function compounds), e.g., synthesis of acrylic esters, and as a reactant.

A partial list of occupations in which exposure may occur includes:

Foundry workers
Gas platers
Mond process workers
Organic chemical synthesizers
Petroleum refinery workers

PERMISSIBLE EXPOSURE LIMITS

The Federal standard for nickel carbonyl is 0.001 ppm (0.007 mg/m^3).

ROUTES OF ENTRY

Inhalation of vapor. It may be possible for appreciable amounts of the liquid to be absorbed through the skin.

HARMFUL EFFECTS

Local—

Nickel dermatitis may develop. (See Nickel and Compounds.)

Systemic—

Symptoms of exposure to the toxic vapors of nickel carbonyl are of two distinct types. Immediately after exposure, symptoms consist of frontal headache, giddiness, tightness of the chest, nausea, weakness of limbs, perspiring, cough, vomiting, cold and clammy skin, and shortness of breath. Even in exposures sufficiently severe to cause death, the initial symptoms disappear quickly upon removal of the subject to fresh air. Symptoms may be so mild during this initial phase that they go unrecognized.

Severe symptoms may then develop insidiously hours or even days after exposure. The delayed syndrome usually consists of retrosternal pain, tightness in the chest, dry cough, shortness of breath, rapid respiration, cyanosis, and extreme weakness. The weakness may be so great that respiration can be sustained only by oxygen support. Fatal cases are usually preceded by convulsion and mental confusion, with death occurring from 4 - 11 days following exposure. The syndrome represents a chemical pneumonitis with adrenal cortical suppression.

Nickel carbonyl is carcinogenic to the same degree as elemental nickel. (See Nickel and Compounds.)

MEDICAL SURVEILLANCE

(See Nickel and Compounds.)

SPECIAL TESTS

Urinary nickel levels for several days after acute exposures may be helpful.

PERSONAL PROTECTIVE METHODS

(See Nickel and Compounds.)

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OSMIUM AND COMPOUNDS

DESCRIPTION

Os, osmium, is a blue-white metal. It is found in platinum ores and in the naturally occurring alloy osmiridium. Osmium when heated in air or when the finely divided form is exposed to air at room temperature oxidizes to form tetroxide (OsO_4, osmic acid). It has a nauseating odor.

SYNONYMS

None.

POTENTIAL OCCUPATIONAL EXPOSURES

Osmium may be alloyed with platinum metals, iron, cobalt, and nickel, and it forms compounds with tin and zinc. The alloy with iridium is used in the manufacture of fountain pen points, engraving tools, record player needles, electrical contacts, compass needles, fine machine bearings, and parts for watch and lock mechanisms. The metal is a catalyst in the synthesis of ammonia, and in the dehydrogenation of organic compounds. It is also used as a stain for histological examination of tissues. Osmium tetroxide is used as an oxidizing agent and as a fixative for tissues in electron microscopy. Other osmium compounds find use in photography. Osmium is no longer used in incandescent lights and in fingerprinting.

A partial list of occupations in which exposure may occur includes:

- Alloy makers
- Histology technicians
- Organic chemical synthesizers
- Synthetic ammonia makers
- Platinum hardeners

PERMISSIBLE EXPOSURE LIMITS

There is presently no Federal standard for osmium itself; the standard for osmium tetroxide is 0.002 mg/m^3.

ROUTE OF ENTRY

Inhalation of vapor or fume.

HARMFUL EFFECTS

Local—

Osmium metal is innocuous, but persons engaged in the production of the metal may be exposed to acids and chlorine vapors. Osmium

tetroxide vapors are poisonous and extremely irritating to the eyes; even in low concentrations, they may cause weeping and persistent conjunctivitis. Longer exposure can result in damage to the cornea and blindness. Contact with skin may cause discoloration (green or black) dermatitis and ulceration.

Systemic—

Inhalation of osmium tetroxide fumes is extremely irritating to the respiratory system, causing tracheitis, bronchitis, bronchial spasm, and difficulty in breathing which may last several hours. Longer exposures can cause serious inflammatory lesions of the lungs (bronchopneumonia with suppuration and gangrene). Slight kidney damage was seen in rabbits inhaling lethal concentrations of vapor for 30 minutes. Some fatty degeneration of renal tubules was seen in one fatal human case along with bronchio pneumonia following an accidental overexposure.

MEDICAL SURVEILLANCE

Consider the skin, eyes, respiratory tract, and renal function in preplacement or periodic examinations.

SPECIAL TESTS

None in common use.

PERSONAL PROTECTIVE METHODS

In areas where the concentration of osmium tetroxide fumes or vapors are excessive, fullface masks or supplied air respirators are necessary. Even low concentrations can cause severe irritation of the eyes. This can usually be prevented by proper ventilation (exhaust hoods, etc.) and the use of goggles. Gloves should be used to prevent burns of the skin and hands. Precautions should be taken to provide protection against acids and chlorine vapors in areas where the metal is produced. (See chlorine.)

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PHOSPHINE

DESCRIPTION

PH_3, phosphine, is a colorless gas with an odor of decaying fish. Phosphine presents an additional hazard in that it ignites at very low temperature. Phosphine is soluble in water 26 ml/100 ml at 17 C and in organic solvents.

SYNONYMS

Hydrogen phosphide, phosphoretted hydrogen, phosphorus trihydride.

POTENTIAL OCCUPATIONAL EXPOSURES

Phosphine is only occasionally used in industry, and exposure usually results accidentally as a by-product of various processes. Exposures may occur when acid or water comes in contact with metallic phosphides (aluminum phosphide, calcium phosphide). These two phosphides are used as insecticides or rodenticides for grain, and phosphine is generated during grain fumigation. Phosphine may also evolve during the generation of acetylene from impure calcium carbide, as well as during metal shaving, sulfuric acid tank cleaning, rust proofing, and ferrosilicon, phosphoric acid and yellow phosphorus explosive handling.

A partial list of occupations in which exposure may occur includes:

- Acetylene workers
- Cement workers
- Firemen
- Grain fumigators
- Metal refiners
- Metal slag workers
- Metallic phosphate workers
- Organic chemical synthesizers
- Rustproofers
- Welders

PERMISSIBLE EXPOSURE LIMITS

0.3 ppm (0.4 mg/m^3) is the Federal standard for occupational exposure to phosphine determined as TWA.

ROUTE OF ENTRY

Inhalation of vapor.

HARMFUL EFFECTS

Local—

Phosphine's strong odor may be nauseating. However, irritation to the eyes or skin is undocumented, and some authors indicate that lacrimation, if it occurs, results as a systemic effect rather than from local irritation.

Systemic—

Acute effects are secondary to central nervous system depression, irritation of lungs, and damage to the liver and other organs. Most common effects include weakness, fatigue, headache, vertigo, anorexia, nausea, vomiting, abdominal pain, diarrhea, tenesmus, thirst, dryness of the throat, difficulty in swallowing, and sensation of chest pressure. In severe cases staggering gait, convulsions, and coma follow. Death may occur from cardiac arrest and, more typically, pulmonary edema, which may be latent in a manner similar to nitrogen oxide intoxication.

Chronic poisoning has been suggested by some authors and symptoms have been attributed to chronic phosphorus poisoning. However, there is evidence that phosphine may be metabolized to form nontoxic phosphates, and chronic exposure of animals has failed to produce toxic effects. Compounded with the lack of human experience and of extensive commercial usage, evidence indicates that chronic poisoning per se does not occur.

MEDICAL SURVEILLANCE

No special considerations are necessary in placement or periodic examinations, other than evaluation of the respiratory system. If poisoning is suspected, workers should be observed for 48 hours due to the delayed onset of pulmonary edema.

SPECIAL TESTS

None have been used.

PERSONAL PROTECTIVE METHODS

In areas where vapors are excessive, workers should be supplied with fullface gas masks with proper cannisters or supplied air respirators.

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PHOSPHORUS AND COMPOUNDS (EXCLUDING PHOSPHINE)

DESCRIPTION

Phosphorus (white or yellow): P. Almost insoluble in water, but soluble in organic solvents.

Phosphoric acid: H_3PO_4. Soluble in water and alcohol.

Phosphorus trichloride: PCl_3. Decomposes in cold water.

Tetraphosphorus trisulfide: P_4S_3. Insoluble.

Red phosphorus is excluded in that it is a nontoxic allotrope, although it is frequently contaminated with a small amount of the yellow. White or yellow phosphorus is either a yellow or colorless, volatile, crystalline solid which darkens when exposed to light and ignites in air to form white fumes and greenish light. Phosphoric acid is also a crystal; however, it is typically encountered in a liquid form. Phosphorus pentachloride and phosphorus pentasulfide are white to pale yellow, fuming crystals, while tetraphosphorus trisulfide is a greenish-yellow crystal. Phosphorus trichloride and phosphorous oxychloride are also colorless, fuming liquids.

Elemental phosphorus does not occur free in nature, but is found in the form of phosphates. Phosphorus and phosphoric acid are prepared commercially from "phosphate rock" deposits of the Southern United

States and, at one time, from bone in Europe. Phosphorus, once formed, is immediately converted to less toxic substances, such as phosphoric acid. The other compounds are prepared directly from red phosphorus and chloride or sulfur respectively. Decomposition products of phosphorus compounds are also toxic and include hydrogen sulfide and phosphoric acid for sulfur-containing compounds.

SYNONYMS

Phosphorus: none.

Phosphoric acid: H_3PO_4, orthophosphoric acid.

Phosphorus trichloride: PCl_3, phosphorous chloride.

Phosphorus pentachloride: PCl_5, phosphoric chloride, phosphorus perchloride.

Tetraphosphorus trisulfide: P_4S_3, phosphorus sesquisulfide, trisulfurated phosphorus.

Phosphorus pentasulfide: P_2S_5, phosphoric sulfide, thiophosphoric anhydride, phosphorus persulfide.

Phosphorus oxychloride: $POCl_3$, phosphorylchloride.

POTENTIAL OCCUPATIONAL EXPOSURES

Yellow phosphorus is handled away from air so that exposure is usually limited. Phosphorus was at one time used for the production of matches or lucifers but has long since been replaced due to its chronic toxicity. Phosphorus is used in the manufacture of munitions, pyrotechnics, explosives, smoke bombs, and other incendiaries, in artificial fertilizers, rodenticides, phosphorbronze alloy, semiconductors, electroluminescent coating, and chemicals, such as, phosphoric acid and metallic phosphides. Phosphoric acid is used in the manufacture of fertilizers, phosphate salts, polyphosphates, detergents, activated carbon, animal feed, ceramics, dental cement, pharmaceuticals, soft drinks, gelatin, rust inhibitors, wax, and rubber latex. Exposure may also occur during electropolishing, engraving, photoengraving, lithograving, metal cleaning, sugar refining, and water treating. Phosphorus trichloride and phosphorus pentachloride are used in the manufacture of agricultural chemicals, chlorinated compounds, dyes, gasoline additives, acetylcellulose, phosphorus oxychloride, plasticizers, saccharin, and surfactants. Phorphorus pentasulfide and tetraphosphorus trisulfide are used in the manufacture of flotation agents, insecticides, lubricating oil, additives, ignition compounds, and matches. They are also used to introduce sulfur into agricultural, rubber, and organic chemicals.

A partial list of occupations in which exposure may occur includes:

Acetylcellulose makers
Bronze alloy makers
Chlorinated compound makers
Electroluminescent coating makers
Fertilizer makers
Fireworks makers
Hydraulic fluid makers
Incendiary makers
Metal refiners
Metallic phosphide makers
Munitions workers
Pesticide workers
Rat poison workers
Semiconductor makers
Smoke bomb makers

PERMISSIBLE EXPOSURE LIMITS

Federal standards are: Phosphorus (yellow) 0.1 mg/m³, phosphoric acid 1.0 mg/m³, phosphorus trichloride 3.0 mg/m³, phosphorus pentachloride 1.0 mg/m³, phosphorus pentasulfide 1.0 mg/m³.

ROUTE OF ENTRY

Inhalation of vapor or fumes or mist.

HARMFUL EFFECTS

Local—

Phosphorus, upon contact with skin, may result in severe burns, which are necrotic, yellowish, fluorescent under ultraviolet light, and have a garlic-like odor. Other phosphorus compounds are potent irritants of the skin, eyes, and mucous membranes of nose, throat, and respiratory tract. At 1 ppm, the Federal standard, phosphoric acid mist is irritating to unacclimated workers but is easily tolerated by acclimated workers. Localized contact dermatitis, particularly of the thighs and eczema of the face and hands, have been observed in workers manufacturing the "strike anywhere" matches containing tetraphosphorus trisulfide.

Systemic—

Acute phosphorus poisoning usually occurs as a result of accidental or suicidal ingestion. However, animal experiments indicate that acute systemic poisoning may follow skin burns. In acute cases, shock may ensue rapidly and the victim may succumb immediately. If acute attack is survived, an asymptomatic latency period of a few hours to a few days may follow. Death often occurs upon relapse from liver, kidney, cardiac, or vascular dysfunction or failure. Abnormal electrocardiograms, particularly of the QT, ST, or T wave phases, abnormal urinary and serum calcium and phosphate levels, proteinuria and aminoaciduria, and elevated serum SGPT are indicative signs. Vomitus, urine, and stools may be fluorescent in ultraviolet light, and a garlic odor of breath and eructations may be noted.

Inhalation of fumes produced by phosphorus compounds listed above may cause irritation of pulmonary tissues with resultant acute pulmonary edema. Chronic exposure may lead to cough, bronchitis, and pneumonia. The hazards of phosphorous pentasulfide are the same as for hydrogen sulfide to which it rapidly hydrolyzes in the presence of moisture.

Chronic phosphorus poisoning is a result of continued absorption of small amounts of yellow phosphorus for periods typically of ten years; however, exposures of as short as 10 months may cause phosphorus necrosis of the jaw ("phossy jaw"). Chronic intoxication is characterized by periostitis with suppuration, ulceration, necrosis, and severe deformity of the mandible and, less often, maxilla. Sequestration of bone may occur. Polymorphic leukopenia, susceptibility to bone fracture, and failure of the alveolar bone to resorb following extractions are secondary clinical signs. Carious teeth and poor dental hygiene increase susceptibility.

MEDICAL SURVEILLANCE

Special consideration should be given to the skin, eyes, jaws, teeth, respiratory tract, and liver. Preplacement medical and dental examination with X-ray of teeth is highly recommended in the case of yellow phosphorus exposure. Poor dental hygiene may increase the risk in yellow phosphorus exposures, and any required dental work should be completed before workers are assigned to areas of possible exposure. Workers experiencing any jaw injury, tooth extraction, or any abnormal dental conditions should be removed from areas of exposure and observed. Roentgenographic examinations may show necrosis; however, in order to prevent full development of sequestrae, the disease should be diagnosed in earlier stages. Liver function should be evaluated periodically. Pulmonary function tests may be useful when exposures are to the acid, chlorides, and sulfide compounds.

SPECIAL TESTS

None commonly used.

PERSONAL PROTECTIVE METHODS

Full body protective clothing including hat and face shield should be supplied to workers who may be exposed to spill, splashes, or spotters of phosphorus or phosphorus compounds. Inhalation of vapors or fumes can be prevented by proper ventilation in many cases, but in areas of higher concentration fullface mask respirators with proper canisters or supplied air respirators may be required. Continuing worker education of exposure risks for those in exposed areas is essential.

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PLATINUM AND COMPOUNDS

DESCRIPTION

Pt, platinum, is a soft, ductile, malleable, silver-white metal, insoluble in water and organic solvents. It is found in the metallic form

and as the arsenide, sperrylite. It forms complex soluble salts such as Na_2PtCl_6.

SYNONYMS

None.

POTENTIAL OCCUPATIONAL EXPOSURES

Platinum and its alloys are utilized because of their resistance to corrosion and oxidation, particularly at high temperatures, their high electrical conductivity, and their excellent catalytic properties. They are used in relays, contacts and tubes in electronic equipment, in spark plug electrodes for aircraft, and windings in high temperature electrical furnaces. Platinum alloys are used for standards for weight, length, and temperature measurement. Platinum and platinum catalysts (e.g., hexachloroplatinic acid, H_2PtCl_6) are widely used in the chemical industry in persulfuric, nitric, and sulfuric acid production, in the synthesis of organic compounds and vitamins, and for producing higher octane gasoline. They are coming into use in catalyst systems for control of exhaust pollutants from automobiles. They are used in the equipment for handling molten glass and manufacturing fibrous glass; in laboratory, medical, and dental apparatus; in electroplating; in photography; in jewelry; and in X-ray fluorescent screens.

A partial list of occupations in which exposure may occur includes:

- Alloy makers
- Catalyst workers
- Ceramic workers
- Dental alloy makers
- Drug makers
- Electronic equipment makers
- Electroplaters
- Gasoline additive makers
- Indelible ink makers
- Jewelry makers
- Laboratory ware makers
- Mirror makers
- Spark plug makers
- Zinc etchers

PERMISSIBLE EXPOSURE LIMITS

The Federal standard for soluble salts of platinum is 0.002 mg/m^3 expressed as Pt.

ROUTE OF ENTRY

Inhalation of dust or mist.

HARMFUL EFFECTS

Local—

Hazards arise from the dust, droplets, spray, or mist of complex salts of platinum, but not from the metal itself. These salts are sensitizers of the skin, nasal mucosa, and bronchi, and cause allergic phenomena. One case of contact dermatitis from wearing a ring made of platinum alloy is recorded.

Systemic—

Characteristic symptoms of poisoning occur after 2 to 6 months' exposure and include pronounced irritation of the throat and nasal pas-

sages, which result in violent sneezing and coughing; bronchial irritation, which causes respiratory distress; and irritation of the skin, which produces cracking, bleeding, and pain. Respiratory symptoms can be so severe that exposed individuals may develop status asthmaticus. After recovery, most individuals develop allergic symptoms and experience further asthma attacks when exposed to even minimal amounts of platinum dust or mists. Mild cases of dermatitis involve only erythema and urticaria of the hands and forearms. More severe cases affect the face and neck. All pathology is limited to allergic manifestations.

MEDICAL SURVEILLANCE

In preemployment and periodic physical examinations, the skin, eyes, and respiratory tract are most important. Any history of skin or pulmonary allergy should be noted, as well as exposure to other irritants or allergens, and smoking history. Periodic assessment of pulmonary function may be useful.

SPECIAL TESTS

None commonly used.

PERSONAL PROTECTIVE METHODS

In areas where dust or mist are excessive, masks or air supplied respirators should be supplied. Where droplets, mist, or spray are encountered, impervious protective clothing, gloves, and goggles should be supplied.

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SELENIUM AND COMPOUNDS

DESCRIPTION

Se, selenium, exists in three forms: a red amorphous powder, a grey form, and red crystals. Selenium, along with tellurium, is found in the sludges and sediments from electrolytic copper refining. It may also be recovered in flue dust from burning pyrites in sulfuric acid manufacture.

SYNONYMS

None.

POTENTIAL OCCUPATIONAL EXPOSURES

Most of the selenium produced is used in the manufacture of selenium rectifiers. It is utilized as a pigment for ruby glass, paints, and dyes, as a vulcanizing agent for rubber, a decolorized agent for green

glass, a chemical catalyst in the Kjeldahl test, and an insecticide; in the manufacture of electrodes, selenium photocells, selenium cells, and semiconductor fusion mixtures; in photographic toning baths; and for dehydrogenation of organic compounds. Se is used in radioactive scanning of the pancreas and for photostatic and X-ray xerography. It may be alloyed with stainless steel, copper, and cast steel.

Hydrogen selenide (selenium hydride, H_2Se) is a colorless gas with a very disagreeable odor which is soluble in water. It is not used commercially. However, it may be produced by the reaction of acids or water and metal selenides or hydrogen and soluble selenium compounds. Selenium hexafluoride (SeF_6) is a gas and is utilized as a gaseous electric insulator. Other selenium compounds are used as solvents, plasticizers, reagents for alkaloids, and flameproofing agents for textiles and wire-cable coverings.

Selenium is a contaminant in most sulfide ores of copper, gold, nickel, and silver, and exposure may occur while removing selenium from these ores.

A partial list of occupations in which exposure may occur includes:

- Arc light electrode makers
- Copper smelters
- Electric rectifier makers
- Glass makers
- Organic chemical synthesizers
- Pesticide makers
- Photographic chemical makers
- Pigment makers
- Plastic workers
- Pyrite roasters
- Rubber makers
- Semiconductor makers
- Sulfuric acid makers
- Textile workers

PERMISSIBLE EXPOSURE LIMITS

The Federal standards are: Selenium compounds (as Se): 0.2 mg/m³. Selenium hexafluoride: 0.05 ppm, 0.4 mg/m³. Hydrogen selenide: 0.05 ppm, 0.2 mg/m³.

ROUTES OF ENTRY

Inhalation of dust or vapor; percutaneous absorption of liquid; ingestion.

HARMFUL EFFECTS

Local—

Elemental selenium is considered to be relatively nonirritating and is poorly absorbed. Some selenium compounds (particularly selenium dioxide and selenium oxychloride) are strong vesicants and can cause destruction of the skin. They are strong irritants to the upper respiratory tract and eyes, and may cause irritation of the mucous membrane of the stomach. Selenium compounds also may cause dermatitis of exposed areas. Allergy to selenium dioxide has been reported in the form of an urticarial generalized rash, and may cause a pink discoloration of the eyelids and palpebral conjunctivitis ("rose-eye"). Selenium oxide also may penetrate under the free edge of the nail, causing excruciatingly painful nail beds and painful paronychia. Selenium compounds may be

absorbed through intact skin to produce systemic effects (Se sulfide in shampoo).

Selenium is considered to be an essential trace element for rats and chickens, and there is strong evidence of its essentiality in man. It is capable of antagonizing the toxic effects of certain other metals, e.g., As and Cd.

Systemic—

The effects of hydrogen selenide intoxication are similar to those caused by other irritating gases in industry: irritation of the mucous membranes of the nose, eyes, and upper respiratory tract, followed by slight tightness in the chest. These symptoms clear when the worker is removed from the exposed area. In some cases, however, pulmonary edema may develop suddenly after a latent period of six to eight hours following exposure. Selenium dioxide inhaled in large quantities may also produce pulmonary edema.

The first and most characteristic sign of selenium absorption is a garlic odor of the breath. This may be related to the excretion in the breath of small amounts of dimethyl selenide. This odor dissipates completely in seven to ten days after the worker is removed from the exposure. It cannot be relied upon as a certain guide to selenium absorption. A more subtle and earlier sign is a metallic taste in the mouth, but many workers accept this without complaint. Other systemic effects are less specific: pallor, lassitude, irritability, vague gastrointestinal symptoms (indigestion), and giddiness. Vital organs appear to escape harm from selenium absorption, but, based on the results of animal experimentation, liver and kidney damage should be regarded as possible. Liver damage and other effects have been long recognized in livestock grazing on high selenium soils. Selenium has been mentioned for its carcinogenic, anticarcinogenic, and teratogenic effects, but, to date, these effects have not been seen in man.

MEDICAL SURVEILLANCE

Preemployment and periodic examinations should consider especially the skin and eyes as well as liver, respiratory and kidney disease and function. The fingernails should be examined.

SPECIAL TESTS

Urinary selenium excretion has been used to indicate exposure in the environment and also occupational exposure. It varies with the Se content of the diet and geographic location. Dimethyl selenide can be determined in breath.

PERSONAL PROTECTIVE METHODS

Protective clothing with special emphasis on personal hygiene (showering and care of fingernails) should help prevent skin exposure and sensitization. Masks and supplied air respirators are needed in areas where concentrations of dust and vapors exceed the allowable standards. These should be equipped with fullface plates. Work clothing

should be changed daily and showering encouraged prior to change to street clothing.

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STIBINE

DESCRIPTION

SbH_3, stibine, is a colorless gas with a characteristic disagreeable odor. It is produced by dissolving zinc-antimony or magnesium-antimony in hydrochloric acid.

SYNONYMS

Antimony hydride.

POTENTIAL OCCUPATIONAL EXPOSURES

Stibine is used as a fumigating agent. Exposure to stibine usually occurs when stibine is released from antimony-containing alloys during the charging of storage batteries, when certain antimonial drosses are treated with water or acid, or when antimony-containing metals come in contact with acid. Operations generally involved are metallurgy, welding or cutting with blow torches, soldering, filling of hydrogen balloons, etching of zinc, and chemical processes.

A partial list of occupations in which exposure may occur includes:

Etchers	Storage battery workers
Solderers	Welders

PERMISSIBLE EXPOSURE LIMITS

The Federal standard is 0.1 ppm (0.5 mg/m^3).

ROUTE OF ENTRY

Inhalation of gas.

HARMFUL EFFECTS

Local—

No local effects have been noted.

Systemic—

Stibine is a powerful hemolytic and central nervous system poison. In acute poisoning, the symptoms are severe headache, nausea, weakness, abdominal and lumbar pain, slow breathing, and weak, irregular

pulse. One of the earliest signs of overexposure may be hemoglobinuria. Laboratory studies may show a profound hemolytic anemia. Death is preceded by jaundice and anuria. Chronic stibine poisoning in man has not been reported.

MEDICAL SURVEILLANCE

In preemployment and periodic examinations special attention should be given to significant blood, kidney, and liver diseases. The general health of exposed workmen should be evaluated periodically. Blood hemoglobin and urine tests for hemoglobin on persons suspected of stibine overexposure are indicated. Workers should also be advised to immediately report any red or dark urinary discoloration to the medical department. This frequently is the initial sign of stibine poisoning. (See Arsine).

SPECIAL TESTS

None in common use.

PERSONAL PROTECTIVE METHODS

In areas where stibine gas is suspected, all persons entering or working in the area should be provided with fullface gas masks or supplied air respirators.

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SILVER AND COMPOUNDS

DESCRIPTION

Ag, silver, is a white metal and is extremely ductile and malleable, insoluble in water but soluble in hot sulfuric and nitric acids.

SYNONYMS

None.

POTENTIAL OCCUPATIONAL EXPOSURES

Silver may be alloyed with copper, aluminum, cadmium, lead, or antimony; the alloys are used in the manufacture of silverware, jewelry, coins, ornaments, plates, commutators, scientific instruments, automobile bearings, and grids in storage batteries. Silver is used in chrome-nickel steels, in solders and brazing alloys, in the application of metallic films on glass and ceramics, to increase corrosion resistance to sulfuric acid, in photographic films, plates and paper, as an electroplated undercoating for nickel and chrome, as a bactericide for sterilizing water, fruit juices, vinegar, etc., in busbars and windings in electrical plants, in dental amal-

gams, and as a chemical catalyst in the synthesis of aldehydes. Because of its resistance to acetic and other food acids, it is utilized in the manufacture of pipes, valves, vats, pasteurizing coils and nozzles for the milk, vinegar, cider, brewing, and acetate rayon silk industries.

Silver compounds are used in photography, silver plating, inks, dyes, coloring glass and porcelain, etching ivory, in the manufacture of mirrors, and as analytical chemical reagents and catalysts. Some of the compounds are also of medical importance as antiseptics or astringents, and in the treatment of certain diseases, particularly in veterinary medicine.

A partial list of occupations in which exposure may occur includes:

Alloy makers
Bactericide makers
Ceramic makers
Coin makers
Chemical laboratory workers
Dental alloy makers
Drug makers
Electric equipment makers
Food product equipment makers
Glass makers
Hair dye makers
Hard solder workers
Ivory etchers
Mirror makers
Organic chemical makers
Photographic workers
Water treaters

PERMISSIBLE EXPOSURE LIMITS

The Federal standard for silver metal and soluble compounds is 0.01 mg/m^3.

ROUTES OF ENTRY

Inhalation of fumes or dust; ingestion of solutions or dust.

HARMFUL EFFECTS

Local—

The only local effect from metallic silver derives from the implant of small particles in the skin of the workmen (usually hands and fingers) which causes a permanent discoloration equivalent to the process of tattooing (local argyria). Silver nitrate dust and solutions are highly corrosive to the skin, eyes, and intestinal tract. The dust of silver nitrate may cause local irritation of the skin, burns of the conjunctiva, and blindness. Localized pigmentation of the skin and eyes may occur. The eye lesions are seen first in the caruncle, and then in the conjunctiva and cornea. The nasal septum and tonsillar pillars also are pigmentated.

Systemic—

All forms of silver are extremely cumulative once they enter body tissues, and very little is excreted. Studies on the occurrence of argyria following injection of silver arsphenamine indicate that the onset of visible argyria begins at a total dose of about 0.9 grams of silver. Generalized argyria develops when silver oxide or salts are inhaled or possibly ingested by workmen who handle compounds of silver (nitrate, fulminate, or cyanide). The condition produces no constitutional symp-

toms, but it may lead to permanent pigmentation of the skin and eyes. The workman's face, forehead, neck, hands, and forearms develop a dark, slate-grey color, uniform in distribution and varying in depth depending on the degree of exposure. Fingernails, buccal mucosa, toe nails, and covered parts of the body to a lesser degree, can also be affected by this discoloration process. The dust is also deposited in the lungs and may be regarded as a form of pneumoconiosis, although it carries no hazard of fibrosis. The existence of kidney lesions of consequence to renal function is improbable from occupational exposure.

MEDICAL SURVEILLANCE

Special attention should be given to other sources of silver exposure, e.g., medications or previous occupational exposure. Inspection of the nasal septum, eyes, and throat will generally give incidence of pigmentation before generalized argyria occurs. This will usually be seen first in the ear lobes, face, and hands.

SPECIAL TESTS

Silver is excreted principally in the feces. Urine and blood levels have not been found useful in monitoring.

PERSONAL PROTECTIVE METHODS

Workers involved with silver nitrate solution should be protected from spills and splashes by impervious protective clothing and chemical goggles. In areas of excessive dust levels, masks with fullface plates should be worn. Clean clothing should be provided daily and meals eaten in noncontaminated areas. Showers should be taken after each shift before change to street clothes.

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TELLURIUM AND COMPOUNDS

DESCRIPTION

Te, tellurium, is a semimetallic element with a bright lustre which is insoluble in water and organic solvents. It may exist in a hexagonal crystalline form or an amorphous powder. It is found in sulfide ores and is produced as a by-product of copper or bismuth refining.

SYNONYMS

Aurum paradoxum, metallum problematum.

POTENTIAL OCCUPATIONAL EXPOSURES

The primary use of tellurium is in the vulcanization of rubber. It is also used as a carbide stabilizer in cast iron, a chemical catalyst, a coloring agent in glazes and glass, a thermocoupling material in refrig-

erating equipment, and as an additive to selenium rectifiers; in alloys of lead, copper, steel, and tin for increased resistance to corrosion and stress, workability, machinability, and creep strength, and in certain culture media in bacteriology. Since tellurium is present in silver, copper, lead, and bismuth ores, exposure may occur during purification of these ores.

A partial list of occupations in which exposure may occur includes:

- Alloy makers
- Ceramic makers
- Copper refinery workers
- Electronic workers
- Enamel makers
- Foundry workers
- Glass makers
- Lead refinery workers
- Porcelain makers
- Rubber workers
- Semiconductor makers
- Silverware makers
- Stainless steel makers
- Thermoelectric device makers

PERMISSIBLE EXPOSURE LIMITS

The applicable Federal standards are: Tellurium: 0.1 mg/m^3. Tellurium hexafluoride: 0.02 ppm (mg/m^3).

ROUTES OF ENTRY

Inhalation of dust or fume; percutaneous absorption from dust.

HARMFUL EFFECTS

Local—

The literature contains no indication of any local effect from tellurium.

Systemic—

The toxicity of tellurium and its compounds is of a low order. There is no indication that either tellurium dust or fume is damaging to the skin or lungs. Inhalation of fumes may cause symptoms, however, some of which are particularly annoying socially to the worker. The most common sign of exposure are foul (garliclike) breath and perspiration, metallic taste in the mouth, and dryness. This is probably due to the presence of dimethyl telluride. These symptoms may appear after relatively short exposures at high concentrations, or longer exposures at lower concentrations, and may persist for long periods of time after the exposure has ended. Workers also complain of afternoon somnolence and loss of appetite.

Exposure to hydrogen telluride produces symptoms of headache, malaise, weakness, dizziness, and respiratory and cardiac symptoms similar to those caused by hydrogen selenide. Pulmonary irritation and the destruction of red blood cells have been reported in studies of laboratory animals exposed to hydrogen telluride.

In other animal studies, tellurium hexafluoride was found to be a respiratory irritant which caused pulmonary edema, and metallic tellurium was shown to have a teratogenic effect on the fetus of rats.

MEDICAL SURVEILLANCE

Oral hygiene and the respiratory tract should receive special attention in preplacement or periodic examinations.

SPECIAL TESTS

Urinary tellurium excretion has been studied in relation to exposure, but is of uncertain value.

PERSONAL PROTECTIVE METHODS

Clean change of work clothes is necessary for hygienic purposes, and showering after each shift before change to street clothes should be encouraged. Respiratory protection is indicated in areas where exposure to hydrogen telluride and tellurium hexafluoride fumes and dust are above the allowable limits.

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THALLIUM AND COMPOUNDS

DESCRIPTION

Tl, thallium, is a soft, heavy metal insoluble in water and organic solvents. It is usually obtained as a by-product from the flue dust generated during the roasting of pyrite ores in the smelting and refining of lead and zinc.

SYNONYMS

None.

POTENTIAL OCCUPATIONAL EXPOSURES

Thallium and its compounds are used as rodenticides, fungicides, insecticides, catalysts in certain organic reactions, and phosphor activators, in bromoiodide crystals for lenses, plates, and prisms in infrared optical instruments, in photoelectric cells, in mineralogical analysis, alloyed with mercury in low temperature thermometers, switches and closures, in high-density liquids, in dyes and pigments, and in the manufacture of optical lenses, fireworks, and imitation precious jewelry. It forms a stainless alloy with silver and a corrosion-resistant alloy with lead. Its medicinal use for epilation has been almost discontinued.

A partial list of occupations in which exposure may occur includes:

Alloy makers
Artificial diamond makers
Chlorinated compound makers
Dye makers
Fireworks makers
Gem makers
Glass makers
High refractive index makers
Infrared instrument makers
Optical glass makers
Photoelectric cell makers
Rodenticide workers

PERMISSIBLE EXPOSURE LIMITS

The Federal standard for thallium (soluble compounds) is 0.1 mg Tl/m³.

ROUTES OF ENTRY

Inhalation of dust and fume. Ingestion and percutaneous absorption of dust.

HARMFUL EFFECTS

Local—

Thallium salts may be skin irritants and sensitizers, but these effects occur rarely in industry.

Systemic—

Thallium is an extremely toxic and cumulative poison. In nonfatal occupational cases of moderate or long term exposure, early symptoms usually include fatigue, limb pain, metallic taste in the mouth and loss of hair, although loss of hair is not always present as an early symptom. Later, peripheral neuritis, proteinuria, and joint pains occur.

Occasionally, neurological signs are the presenting factor, especially in more severe poisonings. Long term exposure may produce optic atrophy, paraesthesias, and changes in pupillary and superficial tendon reflexes (slowed responses). Acute poisoning rarely occurs in industry, and is usually due to ingestion of thallium. When it occurs, gastrointestinal symptoms, abdominal colic, loss of kidney function, peripheral neuritis, strabismus, disorientation, convulsions, joint pain, and alopecia develop rapidly (within 3 days). Death is due to damage to the central nervous system.

MEDICAL SURVEILLANCE

Preplacement and periodic examinations should give special consideration to the central nervous system, gastrointestinal symptoms, and liver and kidney function. Hair loss may be a significant sign. Urine examinations may be helpful.

SPECIAL TESTS

Thallium has been determined in the urine, but the levels do not relate to degree, exposure, or to symptoms.

PERSONAL PROTECTIVE METHODS

Eating, gum chewing, and smoking should not be allowed in pro-

duction areas. Strict enforcement of high standards of personal hygiene is recommended. Appropriate respiratory protection should be used. Protective clothing, hats, goggles, and gloves may be needed to prevent dust absorption through the skin. Daily change of work clothes and showers at the end of the shift will reduce the chances of significant absorption.

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THORIUM AND COMPOUNDS

DESCRIPTION

Th, thorium, is a natural radioactive element insoluble in water and organic solvents. It occurs in the minerals monazite, thorite, and thorinite, usually mixed with its distintegration products.

SYNONYMS

None.

POTENTIAL OCCUPATIONAL EXPOSURES

Metallic thorium is used in nuclear reactors to produce nuclear fuel, in the manufacture of incandescent mantles, as an alloying material, especially with some of the lighter metals, e.g., magnesium, as a reducing agent in metallurgy, for filament coatings in incandescent lamps and vacuum tubes, as a catalyst in organic synthesis, in ceramics, and in welding electrodes.

Exposures may occur during production and use of thorium-containing materials, in the casting and machining of alloy parts, and from the fume produced during welding with thorium electrodes.

A partial list of occupations in which exposure may occur includes:

Ceramic makers	Metal refiners
Gas mantle makers	Nuclear reactor workers
Incandescent lamp makers	Organic chemical synthesizers
Magnesium alloy makers	Vacuum tube makers

PERMISSIBLE EXPOSURE LIMITS

Maximum permissible concentration for thorium under the Federal standard (see 20 CFR Part 20-Table 1) is $1X10^{-6}$ μCi/ml (air).

ROUTES OF ENTRY

Ingestion of liquid, inhalation of dust or gas, and percutaneous absorption.

HARMFUL EFFECTS

Local—

Thorium and thorium compounds are relatively inert, but some

irritant effect may occur depending on the anion present. Gas and aerosols can penetrate the body by way of the respiratory system, the digestive system, and the skin.

Systemic—

Thorium and its compounds are toxicologically inert on the basis of its chemical toxicity. Only 0.001% of an ingested dose is retained in the body. Thorium, once deposited in the body, remains for long periods of time. It has a predilection for bones, lungs, lymphatic glands, and parenchymatous tissues. Characteristic effects of the activity of thorium and its disintegration products are changes in blood forming, nervous, and reticuloendothelial systems, and functional and morphological damage to lung and bone tissue. Only much later do illness and symptoms characteristic of chronic radiation disease appear. After a considerable time, neoplasms may occur and the immunological activity of the body may be reduced. External radiation with gamma rays can occur from contact with material containing mesothorium, with thorium in large quantities, and with by-products that contain disintegration products of thorium. Thorium dioxide (thorotrast) is known to cause severe radiation damage and cancer of bone, blood vessels, liver, and other organs when administered to patients for diagnostic purposes. Its use is now forbidden for introduction into body tissues. Workers in plants where thorium dioxide is produced have not experienced either chemical or radiation injury.

MEDICAL SURVEILLANCE

Monitoring of personnel for early symptoms and changes such as abnormal leukocytes in the blood smear may be of value.

SPECIAL TESTS

In cases of chronic or acute exposure, the determination of thorium in the urine or the use of whole body radiation counts and breath radon are useful methods of monitoring the exposure dose and excretion rates.

PERSONAL PROTECTIVE METHODS

Protection of the worker is afforded by respiratory protection with either dust masks, special canister gas masks, or supplied air respirators. Protective clothing and gloves to prevent dust settling on the skin, with daily change of work clothes, and showering after each shift before change to street clothes should be routine.

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TIN AND COMPOUNDS

DESCRIPTION

Sn, tin, is a soft, silvery-white metal insoluble in water. The primary commercial source of tin is cassiterite (SnO_2, tinstone).

SYNONYMS

Stannum.

POTENTIAL OCCUPATIONAL EXPOSURES

The most important use of tin is as a protective coating for other metals such as in the food and beverage canning industry, in roofing tiles, silverwares, coated wire, household utensils, electronic components, and pistons. Common tin alloys are phosphor bronze, light brass, gun metal, high tensile brass, manganese bronze, die-casting alloys, bearing metals, type metal, and pewter. These are used as soft solders, fillers in automobile bodies, and as coatings for hydraulic brake parts, aircraft landing gear and engine parts. Metallic tin is used in the manufacture of collapsible tubes and foil for packaging.

Organic and inorganic tin compounds are important industrially in the production of drill-glass, ceramics, porcelain, enamel, glass, and inks; as a mordant it is important in the production of fungicides, anthelmintics, insecticides; as a stabilizer it is used in polyvinyl plastics and chlorinated rubber paints; and it is used in plating baths.

Exposures to tin may occur in mining, smelting, and refining, and in the production and use of tin alloys and solders.

A partial list of occupations in which exposure may occur includes:

Babbitt metal (tin, copper, antimony) makers
Brass (essentially copper and zinc) founders
Britannia metal (tin, copper, antimony) makers
Bronze (tin, copper) founders
Dye workers
Fungicide workers
Pewter makers
Pigment workers
Plastic makers
Solder makers
Textile workers
Type metal (lead, antimony, tin) makers

PERMISSIBLE EXPOSURE LIMITS

The Federal standard for organic tin compounds is 0.1 mg/m^3 and for inorganic compounds excluding the oxides it is 2.0 mg/m^3.

ROUTES OF ENTRY

Inhalation of dust. Ingestion, inhalation, or percutaneous absorption of organo-tins.

HARMFUL EFFECTS

Local—

Certain inorganic tin salts are mild irritants to the skin and mucous membranes. They may be strongly acid or basic depending on the cation or anion present. Organic tin compounds, especially tributyl and dibutyl compounds, may cause acute burns to the skin. The burns produce little pain but may itch. They heal without scarring. Clothing contaminated by vapors or liquids may cause subacute lesions and diffuse erythematoid dermatitis on the lower abdomen, thighs, and groin of workmen who handle these compounds. The lesions heal rapidly on removal from contact. The eyes are rarely involved, but accidental splashing with tributyl tin has caused lacrimation and conjunctival edema which lasted several days; there was no permanent injury.

Systemic—

Exposure to dust or fumes of inorganic tin is known to cause a benign pneumoconiosis (stannosis). This form of pneumoconosis produces distinctive progressive X-ray changes of the lungs as long as exposure persists, but there is no distinctive fibrosis, no evidence of disability, and no special complicating factors. Because tin is so radioopaque, early diagnosis is possible.

Certain organic tin compounds, especially alkyltin compounds, are highly toxic when ingested. The trialkyl and tetraalkyl compounds cause damage to the central nervous system with symptoms of headaches, dizziness, photophobia, vomiting, and urinary retention, some weakness and flacid paralysis of the limbs in the most severe cases. Percutaneous absorption of these compounds has been postulated, but to date, deaths and serious injury have resulted only from ill-advised attempts at therapeutic use by mouth. The mechanism of action of the organo-tins is not clearly understood, although triethyltin is an extremely potent inhibitor of oxidative phosphorylation. Occasionally, mild organo-tin intoxication is seen in chemical laboratories with headache, nausea, and EEG changes.

MEDICAL SURVEILLANCE

In the case of inorganic tin compounds, the skin and eyes are of particular interest. Chest X-rays may reveal that exposures have occurred. For organo-tins, preplacement and periodic examinations should include the skin, eyes, blood, central nervous system, and liver and kidney function.

SPECIAL TESTS

None in use.

PERSONAL PROTECTIVE METHODS

It is important that employees be trained in the correct use of personal protective equipment. Skin contact should be prevented by protective clothing, and, especially in the case of organic tin compounds, clean work clothes should be supplied daily and the worker required to

shower following the shift and prior to change to street clothes. In all areas of dust concentration, dust masks should be provided, and in the case of fumes, masks with proper canisters or supplied air respirators should be used.

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TITANIUM AND COMPOUNDS

DESCRIPTION

Ti, titanium, is a dark-grey, lustrous metal insoluble in water. It is brittle when cold and malleable when hot. The most important minerals containing titanium are ilmenite, rutile, perovskite, and titanite or sphene.

SYNONYMS

None.

POTENTIAL OCCUPATIONAL EXPOSURES

Titanium metal, because of its low weight, high strength, and heat resistance, is used in the aerospace and aircraft industry as tubing, fittings, fire walls, cowlings, skin sections, and jet compressors, and it is also used in surgical appliances. It is used, too, as control-wire casings in nuclear reactors, as a protective coating for mixers in the pulp-paper industry and in other situations in which protection against chlorides or acids is required, in vacuum lamp bulbs and X-ray tubes, as an addition to carbon and tungsten in electrodes and lamp filaments, and to the powder in the pyrotechnics industry. It forms alloys with iron, aluminum, tin, and vanadium of which ferrotitanium is especially important in the steel industry.

Titanium dioxide (TiO_2, rutile, anatase, titania) is a white pigment in the rubber, plastics, ceramics, paint, and varnish industries, in dermatological preparations, and is used as a starting material for other titanium compounds, as a gem, in curing concrete, and in coatings for welding rods.

Other titanium compounds are utilized in smoke screens, as mordants in dyeing, in the manufacture of cemented metal carbides, as thermal insulators, and in heat resistant surface coatings in paints and plastics.

A partial list of occupations in which exposure may occur includes:

Ceramic makers	Paper makers
Glass makers	Plastic makers
Incandescent lamp makers	Rayon makers
Ink makers	Smoke screen makers
Lacquer makers	Steel workers
Nuclear steel makers	Vacuum tube makers
Paint makers	Welding rod makers

PERMISSIBLE EXPOSURE LIMITS

The Federal standard for titanium dioxide is 15 mg/m^3. There is no standard for titanium itself or other titanium compounds.

ROUTE OF ENTRY

Inhalation of dust or fume.

HARMFUL EFFECTS

Local—

Titanium and titanium compounds are, for the most part, virtually inert and not highly toxic to man. Titanium tetrachloride, which is released into the air during maintenance of chlorinating and rectifying operations, is an exception. Titanium tetrachloride and its hydrolysis products are highly toxic and irritating. Skin exposure may cause irritation and burns, and even brief contact with the eyes may cause suppurating conjunctivitis and keratitis, followed by clouding of the cornea.

Systemic—

During the production of titanium metal, it is possible that the air may be contaminated with chlorine, hydrogen chloride, titanium tetrachloride, and similar harmful constituents. Reports of severe lung injury caused by such exposures have been recorded; in some cases the condition resembles silicotic lungs. Reports of pulmonary fibrosis due to titanium carbide are now mostly discounted, but precautions are still recommended. Titanium tetrachloride may cause injury to the upper respiratory tract and acute bronchitis.

MEDICAL SURVEILLANCE

Preemployment and periodic physical examinations should give special attention to lung disease, especially if irritant compounds are involved. Chest X-rays should be included in both examinations and pulmonary function evaluated periodically. Smoking history should be taken. Careful attention should be given to the eyes and the skin.

PERSONAL PROTECTIVE METHODS

Employees exposed to titanium tetrachloride should wear protective clothing and respirators. In areas of dust or fumes of titanium tetrachloride, all workers should be provided with goggles and dust masks, fullface gas masks, or supplied air respirators. Clothing should be

changed daily to avoid dust inhalation from clothing, and employees should be encouraged to shower before changing to street clothes.

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URANIUM AND COMPOUNDS

DESCRIPTION

U, uranium, is a hard, silvery-white amphoteric metal and is a radioactive element. In the natural state, it consists of three isotopes: U^{238} (99.28%), U^{234} (0.006%), and U^{235} (0.714%). There are over one hundred uranium minerals; those of commercial importance are the oxides and oxygenous salts. The processing of uranium ore generally involves extraction then leaching either by an acid or a carbonate method. The metal may be obtained from its halides by fused salt electrolysis.

SYNONYMS

None.

POTENTIAL OCCUPATIONAL EXPOSURES

The primary use of natural uranium is in nuclear energy as a fuel for nuclear reactors, in plutonium production, and as feeds for gaseous diffusion plants. It is also a source of radium salts. Uranium compounds are used in staining glass, glazing ceramics, and enamelling, in photographic processes, for alloying steels, and as a catalyst for chemical reactions, radiation shielding, and aircraft counterweights.

Uranium presents both chemical and radiation hazards, and exposures may occur during mining, processing of the ore, and production of uranium metal.

A partial list of occupations in which exposure may occur includes:

Atomic bomb workers	Hydrogen bomb workers
Ceramic makers	Nuclear reactor workers
Glass makers	Photographic chemical makers

PERMISSIBLE EXPOSURE LIMITS

The Federal standards are: uranium, soluble compounds, 0.05 mg/m^3; and uranium, insoluble compounds, 0.25 mg/m^3.

ROUTES OF ENTRY

Inhalation of fume, dust, or gas. The following uranium salts are reported to be capable of penetrating intact skin:

Uranyl nitrate, $UO_2(NO_3)_2.6H_2O$.

Uranyl fluoride, UO_2F_2.

Uranium pentachloride, UCl_5.

Uranium trioxide (uranyl oxide), UO_3.
Sodium diuranate (sodium uranate (VI), $Na_2U_2O_7.H_2O$).
Ammonium diuranate (ammonium uranate (VI) $(NH_4)_2U_2O_7$).
Uranium hexafluoride, UF_6.

HARMFUL EFFECTS

Local—

No toxic effects have been reported, but prolonged contact with skin should be avoided to prevent radiation injury.

Systemic—

Uranium and its compounds are highly toxic substances. The compounds which are soluble in body fluids possess the highest toxicity. Poisoning has generally occurred as a result of accidents. Acute chemical toxicity produces damage primarily to the kidneys. Kidney changes precede in time and degree the effects on the liver. Chronic poisoning with prolonged exposure gives chest findings of pneumoconiosis, pronounced blood changes, and generalized injury.

It is difficult to separate the toxic chemical effects of uranium and its compounds from their radiation effects. The chronic radiation effects are similar to those produced by ionizing radiation. Reports now confirm that carcinogenicity is related to dose and exposure time. Cancer of the lung, osteosarcoma, and lymphoma have all been reported.

MEDICAL SURVEILLANCE

Special attention should be given to the blood, lung, kidney, and liver in preemployment physical examinations. In periodic examinations, tests for blood changes, changes in chest X-rays, or for renal injury and liver damage are advisable.

SPECIAL TESTS

Uranium excretion in the urine has been used as an index of exposure. Whole body counting may also be useful.

PERSONAL PROTECTIVE METHODS

It is important that a formal monitoring system be established to measure each employee's exposure to uranium. This industry has an excellent record of safety to this hazardous material because of good industrial hygiene practices and monitoring of work practices. Protective clothing, gloves, and respirators are necessary in cases of spills and accidents, and must be worn when dealing with soluble compounds in open systems. Closed systems are essential because of the carcinogenic effects.

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VANADIUM AND COMPOUNDS

DESCRIPTION

V, vanadium, is a light grey or white, lustrous powder or fused hard lump insoluble in water. It is produced by roasting the ores, thermal decomposition of the iodide, or from petroleum residues, slags from ferrovanadium production, or soot from oil burning.

SYNONYMS

None.

POTENTIAL OCCUPATIONAL EXPOSURES

Most of the vanadium produced is used in ferrovanadium and of this, the majority is used in high speed and other alloy steels with only small amounts in tool or structural steels. It is usually combined with chromium, nickel, manganese, boron, and tungsten in steel alloys.

Vanadium pentoxide (V_2O_5) is an industrial catalyst in oxidation reactions, is used in glass and ceramic glazes, is a steel additive, and is used in welding electrode coatings. Ammonium metavanadate (NH_4VO_3) is used as an industrial catalyst, a chemical reagent, a photographic developer, and in dyeing and printing. Other vanadium compounds are utilized as mordants in dyeing, in insecticides, as catalysts, and in metallurgy.

Since vanadium itself is considered nontoxic, there is little hazard associated with mining; however, exposure to the more toxic compounds, especially the oxides, can occur during smelting and refining. Exposure may also occur in conjunction with oil-fired furnace flues.

A partial list of occupations in which exposure may occur includes:

Alloy makers	Glass makers
Ceramic makers	Organic chemical synthesizers
Dye makers	Photographic chemical makers
Ferrovanadium workers	Textile dye workers

PERMISSIBLE EXPOSURE LIMITS

The Federal standards are: V_2O_5 dust, 0.5 mg/m^3, and V_2O_5 fume, 0.1 mg/m^3, both as ceiling values.

ROUTE OF ENTRY

Inhalation of dust or fume.

HARMFUL EFFECTS

Local—

Vanadium compounds, especially vanadium pentoxide, are irritants to the eyes and skin. The initial eye symptoms are profuse lacrimation and a burning sensation of the conjuctiva. Skin lesions are of the eczematous type which itch intensely. In some cases there may be generalized urticaria. Workers may also exhibit greenish discoloration of the tongue. This same discoloration may be detectable on the butts of cigarettes smoked by vanadium workers.

Systemic—

Vanadium compounds are irritants to the respiratory tract. Entrance to the body is through inhalation of dusts or fumes. Serous or hemorrhagic rhinitis, sore throat, cough, tracheitis, bronchitis, expectoration, and chest pain, may result after even a brief exposure. More serious exposure may result in pulmonary edema and pneumonia which may be fatal. Individuals who recover may experience persistent bronchitis resembling asthma, and bouts of dyspnea; however, no chronic lung lesions have been described.

The results of experimental biochemical studies show that vanadium compounds inhibit cholesterol synthesis and the activity of the enzyme cholinesterase. A variety of other biochemical effects have been noted experimentally, but these have not been reported in relation to occupational exposures. Slightly lower cholesterol levels in blood were noted in one report, but this seems of doubtful significance.

MEDICAL SURVEILLANCE

Preemployment and periodic physical examinations should emphasize effects on the eyes, skin, and lungs.

SPECIAL TESTS

Urinary vanadium excretion may be useful as an index of exposure.

PERSONAL PROTECTIVE METHODS

Employees should receive training in personal hygiene and in the use of personal protective equipment. In certain areas, masks or respirators may be necessary to prevent inhalation of dust and fumes. Protective clothing and gloves will be helpful in preventing dermatitis. Showering after each shift before changing to street clothes is very important. Clean work clothes should be supplied daily.

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ZINC CHLORIDE

DESCRIPTION

$ZnCl_2$, zinc chloride, consists of white hexagonal, deliquescent crystals, soluble in water(1 gm/0.5 ml)and in organic solvents. It may be produced from zinc sulfide ore, zinc oxide, or zinc metal.

SYNONYMS

Butter of zinc.

POTENTIAL OCCUPATIONAL EXPOSURES

Zinc chloride is used as a wood preservative, for dry battery cells, as a soldering flux, and in textile finishing, in vulcanized fiber, reclaiming rubber, oil and gas well operations, oil refining, manufacture of parchment paper, dyes, activated carbon, chemical synthesis, dentists' cement, deodorants, disinfecting and embalming solutions, and taxidermy. It is also produced by military screening-smoke devices.

A partial list of occupations in which exposure may occur includes:

- Activated carbon makers,
- Dental cement makers
- Deodorant makers
- Disinfectant makers
- Dry cell battery makers
- Dye makers
- Embalmers
- Military personnel
- Paper makers
- Petroleum refinery workers
- Rubber workers
- Solderers
- Taxidermists
- Textile finishers

PERMISSIBLE EXPOSURE LIMITS

The Federal standard for zinc chloride fume is 1 mg/m^3.

ROUTES OF ENTRY

Inhalation of dust and fumes; ingestion.

HARMFUL EFFECTS

Local—

Solid zinc chloride is corrosive to the skin and mucous membranes. Aqueous solutions of 10% or more are also corrosive and cause primary dermatitis and chemical burns, especially at sites of minor trauma. Aqueous solutions are also extremely dangerous to the eyes, causing extreme pain, inflammation, and swelling, which may be followed by corneal ulceration. Zinc chloride may produce true sensitization of the skin in the form of eczematoid dermatitis. Ingestion of zinc chloride may cause serious corrosive effects in the esophagus and stomach, often complicated by pyloric stenosis.

Systemic—

There are no reports of inhalation of zinc chloride from industrial exposure. All reported experience with inhaled zinc chloride is based on exposures caused by military accidents. In all of those cases, there was severe irritation of the respiratory tract. In the more severe cases, acute pulmonary edema developed within two to four days following exposure. The fatalities reported were due to severe lung injury with hemorrhagic alveolitis and bronchopneumonia. In human experimentation with concentrations of 120 mg/m^3, there were complaints of irritation of the nose, throat, and chest after 2 minutes. With exposure to 80 mg/m^3 for 2 minutes, the majority of subjects experienced slight nausea, all noticed the smell, and one or two coughed.

MEDICAL SURVEILLANCE

In preemployment and periodic physical examinations, special at-

tention should be given to the skin and to the history of allergic dermatitis, as well as to exposed mucous membranes, the eyes, and the respiratory system. Chest X-rays and periodic pulmonary function studies may be helpful. Smoking history should be known.

SPECIAL TESTS

Urinary zinc excretion may be useful.

PERSONAL PROTECTIVE METHODS

Employees exposed to zinc chloride should be given instruction in personal hygiene, and in the use of personal protective equipment. Goggles should be provided in areas where splash or spill of liquid is possible. In areas with excessive dust or fume levels, respiratory protection by use of filter type dust masks or air supplied respirators with fullface pieces should be required. In areas where danger of spills or splashes exists, skin protection should be provided with rubber gloves, face shields, rubber aprons, gauntlets, suits, and rubber shoes.

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ZINC OXIDE

DESCRIPTION

ZnO, zinc oxide, is an amorphous, odorless, white or yellowish-white powder, practically insoluble in water. It is produced by oxidation of zinc or by roasting of zinc oxide ore.

SYNONYMS

Zinc white, flowers of zinc.

POTENTIAL OCCUPATIONAL EXPOSURES

Zinc oxide is primarily used as a white pigment in rubber formulations and as a vulcanizing aid. It is also used in photocopying, paints, chemicals, ceramics, lacquers, and varnishes, as a filler for plastics, in cosmetics, pharmaceuticals, and calamine lotion. Exposure may occur in the manufacture and use of zinc oxide and products, or through its formation as a fume when zinc or its alloys are heated.

A partial list of occupations in which exposure may occur includes:

- Alloy makers
- Brass foundry workers
- Ceramic makers
- Chemical synthesizers
- Cosmetic makers
- Electroplaters
- Galvanizers
- Lacquer makers
- Paint makers
- Pigment makers
- Plastic makers
- Rubber workers
- Welders

PERMISSIBLE EXPOSURE LIMITS

The Federal standard for zinc oxide fume is 5 mg/m^3.

ROUTE OF ENTRY

Inhalation of dust or fumes.

HARMFUL EFFECTS

Local—

When handled under poor hygienic conditions, zinc oxide powder may produce a dermatitis called "oxide pox." This condition is due primarily to clogging of the sebaceous glands with zinc oxide and produces a red papule with a central plug. The area rapidly becomes inflamed and the central plug develops into a pustule which itches intensely. Lesions occur in areas of the skin that are exposed or subject to heavy perspiration. These usually clear, however, in a week to ten days with good hygiene and proper care of secondary infections.

Systemic—

The syndrome of metal fume fever is the only important effect of exposure to freshly formed zinc oxide fumes and zinc oxide dusts of respirable particle size. The fumes are formed by subjecting either zinc or alloys containing zinc to high temperatures. Typically, the syndrome begins four to twelve hours after sufficient exposure to freshly formed fumes of zinc oxide. The worker first notices the presence of a sweet or metallic taste in the mouth, accompanied by dryness and irritation of the throat. Cough and shortness of breath may occur, along with general malaise, a feeling of weakness, fatigue, and pains in the muscles and joints. Fever and shaking chills then develop. Fever can range from 102 to 104 F. Profuse sweats develop and the fever subsides. The entire episode runs its course in 24 - 48 hours. During the acute period, there is an elevation of the leukocyte count (rarely above 20,000/ml), and the serum LDH may be elevated. Chest X-rays are not diagnostic.

Metal fume fever produces rapid development of tolerance or short-lived relative immunity. This may be lost, however, over a weekend or holiday, and the worker may again develop the complete syndrome when he returns to work if fume levels are sufficiently high. There are no sequelae to the attacks.

Other possible systemic effects of zinc oxide are in doubt. Cases of gastrointestinal disturbance have been reported, but most authorities agree there is no evidence of chronic industrial zinc poisoning.

MEDICAL SURVEILLANCE

Preemployment and periodic physical examinations should be made to assess the status of the general health of the worker. Examinations are also recommended following episodes of metal fume fever or intercurrent illnesses.

SPECIAL TESTS

Zinc excretion in urine can be used as an index of exposure.

PERSONAL PROTECTIVE METHODS

Employees should receive instruction in personal hygiene and in

the causes and effects of metal fume fever. Workers exposed to zinc oxide powder should be supplied with daily clean work clothes and should be required to shower before changing to street clothes. In cases of accident or where excessive fume concentrations are present, gas masks with proper canister or supplied air respirators should be provided.

ZIRCONIUM AND COMPOUNDS

DESCRIPTION

Zr, zirconium, is a greyish-white, lustrous metal in the form of platelets, flakes, or a bluish-black, amorphous powder. It is never found in the free state; the most common sources are the ores zircon and baddeleyite. It is generally produced by reduction of the chloride or iodide. The metal is very reactive, and the process is carried out under an atmosphere of inert gas. The powdered metal is a fire and explosive hazard.

SYNONYMS

None.

POTENTIAL OCCUPATIONAL EXPOSURES

Zirconium metal is used as a "getter" in vacuum tubes, a deoxidizer in metallurgy, and a substitute for platinum; it is used in priming of explosive mixtures, flashlight powders, lamp filaments, flash bulbs, and in construction rayon spinnerets. Zirconium or its alloys (nickel cobalt, niobium, tantalum) are used as lining materials for pumps and pipes, for chemical processes, and for reaction vessels. Pure zirconium is a structural material for atomic reactors, and alloyed, particularly with aluminum, it is a cladding material for fuel rods in water-moderated nuclear reactors. A zirconium-columbian alloy is an excellent superconductor.

Zircon ($ZrSiO_4$) is utilized as a foundry sand, an abrasive, a refractory in combination with zirconia, a coating for casting molds, a catalyst in alkyl and alkenyl hydrocarbon manufacture, a stabilizer in silicone rubbers, and as a gem stone; in ceramics it is used as an opacifier for glazes and enamels and in frittered glass filters. Both zircon and zirconia (zirconium oxide, ZrO_2) bricks are used as linings for glass furnaces. Zirconia itself is used in die extrusion of metals and in spout linings for pouring metals as a substitute for lime in oxyhydrogen light, as a pigment, and an abrasive; it is used, too, in incandescent lights, as well as in the manufacture of enamels, white glass, and refractory crucibles.

Other zirconium compounds are used in metal cutting tools, thermocouple jackets, waterproofing textiles, ceramics, and in treating dermatitis and poison ivy.

A partial list of occupations in which exposure may occur includes:

Abrasive makers
Ceramic workers
Crucible makers
Deodorant makers
Enamel makers
Explosive workers
Foundry workers
Glass makers
Incandescent lamp makers
Metallurgists
Pigment makers
Rayon spinneret makers
Refractory material makers
Textile waterproofers
Vacuum tube makers

PERMISSIBLE EXPOSURE LIMITS

The Federal standard for zirconium compounds is 5 mg/m³ as Zr.

ROUTE OF ENTRY

Inhalation of dust or fume.

HARMFUL EFFECTS

Local—

No ill effects from industrial exposure to zirconium have been proven. A recent study from the U.S.S.R., however, reports that some workers exposed to plumbous titanate zirconate developed a mild occupational dermatitis associated with hyperhydrosis of the hands. This condition was accompanied by subjective complaints of vertigo, sweet taste in the mouth, and general indisposition. These workers were also said to have elevated thermal and pain sensitivity, and electric permeability of the horny layer, along with increased sweating, and reduced capillary resistance.

Zircon granuloma were reported in the U.S. as early as 1956. This condition arose from the use of deodorant sticks in the axillae, but it was resolved when use was stopped. Zircon is no longer used as a deodorant. Because of a possible allergic sensitivity reaction, individuals who have experienced granuloma from zirconium should avoid dust and mist.

Systemic—

Inhalation of zirconium dust and fumes has caused no respiratory or other pathological problems. Animal experiments, however, have produced interstitial pneumonitis, peribronchial abscesses, peribronchiolar granuloma, and lobular pneumonia.

MEDICAL SURVEILLANCE

No special considerations are needed.

SPECIAL TESTS

None in common use.

PERSONAL PROTECTIVE METHODS

Employees should be trained in the correct use of personal protective equipment. In areas of dust accumulation or high fume concentrations, respiratory protection is advised either by dust mask or supplied

air respirators. Skin protection is not generally necessary, but where there is a history of zircon granuloma from deodorants, it is probably advisable.

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MISCELLANEOUS INORGANIC COMPOUNDS

AMMONIA

DESCRIPTION

NH_3, ammonia, is a colorless, strongly alkaline, and extremely soluble gas with a characteristic pungent odor.

SYNONYMS

None.

POTENTIAL OCCUPATIONAL EXPOSURES

Ammonia is used as a nitrogen source for many nitrogen-containing compounds. It is used in the production of ammonium sulfate and ammonium nitrate for fertilizers and in the manufacture of nitric acid, soda, synthetic urea, synthetic fibers, dyes, and plastics. It is also utilized as a refrigerant and in the petroleum refining, chemical, and pharmaceutical industries.

Other sources of occupational exposure include the silvering of mirrors, gluemaking, tanning of leather, and around nitriding furnaces. Ammonia is produced as a by-product in coal distillation and by the action of steam on calcium cyanamide, and from the decomposition of nitrogenous materials.

A partial list of occupations in which exposure may occur includes:

- Aluminum workers
- Annealers
- Chemical laboratory workers
- Chemical workers
- Dye makers
- Electroplate workers
- Fertilizer workers
- Galvanizers
- Glue makers
- Metal extractors
- Metal powder processors
- Mirror silverers
- Paper makers
- Paper pulp makers
- Pesticide makers
- Rayon makers
- Refrigeration workers
- Sulfuric acid workers
- Tannery workers
- Water treaters

PERMISSIBLE EXPOSURE LIMITS

The Federal standard for ammonia is an 8-hour time weighted average of 50 ppm (35 mg/m^3). NIOSH has recommended 50 ppm expressed as a ceiling and determined by a 5-minute sampling period.

ROUTE OF ENTRY

Inhalation of gas.

HARMFUL EFFECTS

Local—

Contact with anhydrous liquid ammonia or with aqueous solutions is intensely irritating to the mucous membranes, eyes, and skin. Eye symptoms range from lacrimation, blepharospasm, and palpebral edema to a rise of intraocular pressure, and other signs resembling acute-angle closure glaucoma, corneal ulceration, and blindness. There may be corrosive burns of skin or blister formation. Ammonia gas is also irritating to the eyes and to moist skin.

Systemic—

Mild to moderate exposure to the gas can produce headache, salivation, burning of throat, anosmia, perspiration, nausea, vomiting, and substernal pain. Irritation of ammonia gas in eyes and nose may be sufficiently intense to compel workers to leave the area. If escape is not possible, there may be severe irritation of the respiratory tract with the production of cough, glottal edema, bronchospasm, pulmonary edema, or respiratory arrest. Bronchitis or pneumonia may follow a severe exposure if patient survives. Urticaria is a rare allergic manifestation from inhalation of the gas.

MEDICAL SURVEILLANCE

Preemployment physical examinations for workers in ammonia exposure areas should be directed toward significant changes in the skin, eyes, and respiratory system. Persons with corneal disease, and glaucoma, or chronic respiratory diseases may suffer increased risk. Periodic examinations should include evaluation of skin, eyes, and respiratory system, and pulmonary function tests to compare with baselines established at preemployment examination.

SPECIAL TESTS

None.

PERSONAL PROTECTIVE METHODS

Where ammonia hazards exist in concentrations above the standard, respiratory, eye, and skin protection should be provided. Fullface gas masks with ammonia canister or supplied air respirators, both with full facepieces, afford good protection. In areas where exposure to liquid ammonia occurs, goggles or face shields, as well as protective clothing impervious to ammonia and including gloves, aprons, and boots should be required. Where ammonia gas or concentrated ammonia solution is

splashed in eyes, immediate flooding of the eyes with large quantities of water for 15 minutes or longer is advised, followed at once by medical examination. In heavy concentrations of ammonia gas, workers should be outfitted with complete self-contained protective suits impervious to ammonia, with supplied air source, and full headpiece and facepiece. Work clothes wetted with concentrated ammonia solutions should be changed immediately, and the exposed area of the body washed thoroughly with water.

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CALCIUM OXIDE

DESCRIPTION

CaO, calcium oxide, occurs as white or grayish-white lumps or granular powder. The presence of iron gives it a yellowish or brownish tint. It is soluble in water and acids.

SYNONYMS

Lime, burnt lime, quicklime, calx, fluxing lime.

POTENTIAL OCCUPATIONAL EXPOSURES

Calcium oxide is used as a refractory material, a binding agent in bricks, plaster, mortar, stucco and other building materials, a dehydrating agent, a flux in steel manufacturing, and a laboratory agent to absorb CO_2; in the manufacture of aluminum, magnesium, glass, pulp and paper, sodium carbonate, calcium hydroxide, chlorinated lime, calcium salts, and other chemicals; in the flotation of nonferrous ores, water and sewage treatment, soil treatment in agriculture, dehairing hides, the clarification of cane and beet sugar juice, and in fungicides, insecticides, drilling fluids, and lubricants.

A partial list of occupations in which exposure may occur includes:

- Brick masons
- Fertilizer makers
- Fungicide workers
- Glass makers
- Insecticide makers
- Metal smelters
- Mortar workers
- Paper makers
- Plaster makers
- Steel workers
- Sugar refiners
- Tannery workers
- Water treaters

PERMISSIBLE EXPOSURE LIMITS

The Federal standard for calcium oxide is 5 mg/m³.

ROUTE OF ENTRY

Inhalation of dust.

HARMFUL EFFECTS

Local—

The irritant action of calcium oxide is due primarily to its alkalinity and exothermic reaction with water. It is irritating and may be caustic to the skin, conjunctiva, cornea, and mucous membranes of upper respiratory tract, may produce burns or dermatitis with desquamation and vesicular rash, lacrimation, spasmodic blinking, ulceration, and ocular perforation, ulceration and inflammation of the respiratory passages, ulceration of nasal and buccal mucosa, and perforation of nasal septum.

Systemic—

Bronchitis and pneumonia have been reported from inhalation of dust. The lower respiratory tract is generally not affected because irritation of upper respiratory passages is so severe that workers are forced to leave the area.

MEDICAL SURVEILLANCE

Preemployment physical examinations should be directed to significant problems of the eyes, skin, and the upper respiratory tract. Periodic examinations should evaluate the skin, changes in the eyes, especially the cornea and conjunctiva, muscosal ulcerations of the nose, mouth, and nasal septum, and any pulmonary symptoms. Smoking history should be known.

SPECIAL TESTS

None in common use.

PERSONAL PROTECTIVE METHODS

In areas where workers are exposed to calcium oxide levels above the standard, protection to the skin, eyes, and respiratory tract should be provided. Skin protection can be provided by protective clothing and gloves. All dusty area workers should be provided with goggles and dust masks with proper cartridges. Personal hygiene is to be encouraged, with frequent change of work clothes and showering after each shift before change to street clothes.

CARBON DIOXIDE

DESCRIPTION

CO_2, carbon dioxide, is a colorless, odorless, non-combustible gas, soluble in water. It is commonly sold in the compressed liquid form, and the solid form (dry ice).

SYNONYMS

Carbonic acid gas, carbonic anhydride.

POTENTIAL OCCUPATIONAL EXPOSURES

Gaseous carbon dioxide is used to carbonate beverages, as a weak acid in the textile, leather, and chemical industries, in water treatment,

and in the manufacture of aspirin and white lead, for hardening molds in foundries, in food preservation, in purging tanks and pipelines, as a fire extinguisher, in foams, and in welding. Because it is relatively inert, it is utilized as a pressure medium. It is also used as a propellant in aerosols, to promote plant growth in green houses; it is used medically as a respiratory stimulant, in the manufacture of carbonates, and to produce an inert atmosphere when an explosive or flammable hazard exists. The liquid is used in fire extinguishing equipment, in cylinders for inflating life rafts, in the manufacturing of dry ice, and as a refrigerant. Dry ice is used primarily as a refrigerant.

Occupational exposure to carbon dioxide may also occur in any place where fermentation processes may deplete oxygen with the formation of carbon dioxide, e.g., in mines, silos, wells, vats, ships' holds, etc.

A partial list of occupations in which exposure may occur includes:

- Aerosol packagers
- Beverage carbonators
- Blast furnace workers
- Brewery workers
- Carbonic acid makers
- Charcoal burners
- Chemical synthesizers
- Explosive makers
- Fire extinguisher makers
- Firemen
- Foundry workers
- Grain elevator workers
- Inert atmosphere welders
- Insecticide makers
- Miners
- Refrigerating car workers
- Refrigerating plant workers
- Soda makers
- Tannery workers
- Textile workers
- Vatmen
- Well cleaners

PERMISSIBLE EXPOSURE LIMITS

The Federal standard is 5,000 ppm (9,000 mg/m^3).

ROUTE OF ENTRY

Inhalation of gas.

HARMFUL EFFECTS

Local—

Frostbite may result from contact with dry ice or gas at low temperature.

Systemic—

Carbon dioxide is a simple asphyxiant. Concentrations of 10% (100,000 ppm) can produce unconsciousness and death from oxygen deficiency. A concentration of 5% may produce shortness of breath and headache. Continuous exposure to 1.5% CO_2 may cause changes in some physiological processes. The concentration of carbon dioxide in the blood affects the rate of breathing.

MEDICAL SURVEILLANCE

No special considerations are necessary although persons with cardiovascular or pulmonary disease may be at increased risk.

SPECIAL TESTS

None in common use.

PERSONAL PROTECTIVE METHODS

Carbon dioxide is a heavy gas and accumulates at low levels in depressions and along the floor. Generally, adequate ventilation will provide sufficient protection for the worker. Where concentrations are of a high order, supplied air respirators are recommended.

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CARBON MONOXIDE

DESCRIPTION

CO, carbon monoxide, is a colorless, odorless, tasteless gas, partially soluble in water, but one which decomposes.

SYNONYMS

None.

POTENTIAL OCCUPATIONAL EXPOSURES

Carbon monoxide is used in metallurgy as a reducing agent, particularly in the Mond process for nickel; in organic synthesis, especially in the Fischer-Tropsch process for petroleum products and in the oxo reaction; and in the manufacture of metal carbonyls.

It is usually encountered in industry as a waste product of incomplete combustion of carbonaceous material (complete combustion produces CO_2). The major source of CO emission in the atmosphere is the gasoline-powered internal combustion engine. Specific industrial processes which contribute significantly to CO emission are iron foundries particularly the cupola; fluid catalytic crackers, fluid coking, and moving-bed catalytic crackers in petroleum refining; lime kilns and Kraft recovery furnaces in Kraft paper mills; furnace, channel, and thermal operations in carbon black plants; beehive coke ovens, basic oxygen furnaces, sintering of blast furnace feed in steel mills; and formaldehyde manufacture. There are numerous other operations in which a flame touches a surface that is cooler than the ignition temperature of the gaseous part of the flame where exposure to CO may occur, e.g., arc welding, automobile repair, traffic control, tunnel construction, fire fighting, mines, use of explosives, etc.

A partial list of occupations in which exposure may occur includes:

Acetylene workers	Metal oxide reducers
Blast furnace workers	Miners
Boiler room workers	Mond process workers
Brewery workers	Organic chemical synthesizers
Carbon black makers	Petroleum refinery workers
Coke oven workers	Pulp and paper workers
Diesel engine operators	Steel workers
Garage mechanics	Water gas workers

PERMISSIBLE EXPOSURE LIMITS

The present Federal standard is 50 ppm (55 mg/m^3). The standard recommended by NIOSH is 35 ppm with a ceiling value of 200 ppm. This latter value is to limit carboxyhemoglobin formation to 5% in a nonsmoker engaged in sedentary activity at normal altitude.

ROUTE OF ENTRY

Inhalation of gas.

HARMFUL EFFECTS

Local—

None.

Systemic—

Carbon monoxide combines with hemoglobin to form carboxyhemoglobin which interferes with the oxygen carrying capacity of blood, resulting in a state of tissue hypoxia. The typical signs and symptoms of acute CO poisoning are headache, dizziness, drowsiness, nausea, vomiting, collapse, coma, and death. Initially the victim is pale; later the skin and mucous membranes may be cherry-red in color. Loss of consciousness occurs at about the 50% carboxyhemoglobin level. The amount of carboxyhemoglobin formed is dependent on concentration and duration of CO exposure, ambient temperature, health, and metabolism of the individual. The formation of carboxyhemoglobin is a reversible process. Recovery from acute poisoning usually occurs without sequelae unless tissue hypoxia was severe enough to result in brain cell degeneration.

Carbon monoxide at low levels may initiate or enhance deleterious myocardial alterations in individuals with restricted coronary artery blood flow and decreased myocardial lactate production.

Severe carbon monoxide poisoning has been reported to permanently damage the extrapyramidal system, including the basal ganglia.

MEDICAL SURVEILLANCE

Preplacement and periodic medical examinations should give special attention to significant cardiovascular disease and any medical conditions which could be exacerbated by exposure to CO. Heavy smokers may be at greater risk. Methylene chloride exposure may also cause an

increase of carboxyhemoglobin. Smokers usually have higher levels of carboxyhemoglobin than nonsmokers (often 5 - 10% or more).

SPECIAL TESTS

Carboxyhemoglobin levels are reliable indicators of exposure and hazard.

PERSONAL PROTECTIVE METHODS

Under certain circumstances where carbon monoxide levels are not exceedingly high, gas masks with proper canisters can be used for short periods but are not recommended. In areas with high concentrations, self-contained air apparatus is recommended.

GRAPHITE

DESCRIPTION

Graphite is crystallized carbon and usually appears as soft, black scales. There are two types of graphite, natural and artificial.

SYNONYMS

Plumbago, black lead, mineral carbon.

POTENTIAL OCCUPATIONAL EXPOSURES

Natural graphite is used in foundry facings, steelmaking, lubricants, refractories, crucibles, pencil "lead," paints, pigments, and stove polish. Artificial graphite may be substituted for these uses with the exception of clay crucibles; other types of crucibles may be produced from artificial graphite. Additionally, it may be used as a high temperature lubricant or for electrodes. It is utilized in the electrical industry in electrodes, brushes, contacts, and electronic tube rectifier elements; as a constituent in lubricating oils and greases; to treat friction elements, such as brake linings; to prevent molds from sticking together; and in moderators in nuclear reactors.

A partial list of occupations in which exposure may occur includes:

- Brake lining makers
- Cathode ray tube makers
- Commutator brush makers
- Crucible makers
- Electrode makers
- Explosive makers
- Foundry workers
- Lubricant makers
- Match makers
- Nuclear reactor workers
- Paint makers
- Pencil lead makers
- Pigment makers
- Refractory material makers
- Steel makers
- Stove polish makers

PERMISSIBLE EXPOSURE LIMITS

The Federal standard for natural graphite is 15 mppcf.

ROUTE OF ENTRY

Inhalation of dust.

HARMFUL EFFECTS

Local—

None.

Systemic—

Exposure to natural graphite may produce a progressive and disabling pneumoconiosis similar to anthracosilicosis. Symptoms include headache, coughing, depression, decreased appetite, dyspnea, and the production of black sputum. Some individuals may be asymptomatic for many years then suddenly become disabled. It has not yet been determined whether the free crystalline silica in graphite is solely responsible for development of the disease. There is evidence that artificial graphite may be capable of producing a pneumoconiosis.

MEDICAL SURVEILLANCE

Preemployment and periodic examinations should be directed toward detecting significant respiratory disease, through chest X-rays and pulmonary function tests.

SPECIAL TESTS

None.

PERSONAL PROTECTIVE METHODS

Workers in exposed areas should be provided with dust masks with proper cartridges and should be instructed in their maintenance.

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HYDROGEN PEROXIDE

DESCRIPTION

H_2O_2, anhydrous hydrogen peroxide, is a colorless rather unstable liquid with a bitter taste. Hydrogen peroxide is completely miscible with water and is commercially sold in concentrations of 3, 35, 50, 70, and 90 percent solutions.

SYNONYMS

Peroxide, hydrogen dioxide, hydroperoxide.

POTENTIAL OCCUPATIONAL EXPOSURES

Hydrogen peroxide is used in the manufacture of acetone, antichlor, antiseptics, benzol peroxide, buttons, disinfectants, pharmaceuticals, felt hats, plastic foam, rocket fuel, and sponge rubber. It is also used in

bleaching bone, feathers, flour, fruit, fur, gelatin, glue, hair, ivory, silk, soap, straw, textiles, wax, and wood pulp, and as an oxygen source in respiratory protective equipment. Other specific occupations with potential exposure include liquor and wine agers, dyers, electroplaters, fat refiners, photographic film developers, wool printers, veterinarians, and water treaters.

A partial list of occupations in which exposure may occur includes:

- Acetone makers
- Alcoholic beverage agers
- Antichlor makers
- Antiseptic makers
- Benzol peroxide makers
- Button makers
- Disinfectant makers
- Drug makers
- Dyers
- Electroplaters
- Fat refiners
- Hide disinfectors
- Metal cleaners
- Photographic film developers
- Plastic foam makers
- Rocket fuel workers
- Sponge rubber makers
- Veterinarians
- Water treaters
- Wool printers

PERMISSIBLE EXPOSURE LIMITS

The Federal standard for hydrogen peroxide (90 percent) is 1 ppm (1.4 mg/m^3).

ROUTE OF ENTRY

Inhalation of vapor or mist.

HARMFUL EFFECTS

Local—

The skin, eyes, and mucous membranes may be irritated by concentrated vapor or mist. Bleaching and a burning sensation may occur at lower levels, while high concentrations may result in blistering and severe eye injury, which may be delayed in appearance.

Systemic—

Inhalation of vapor or mist may produce pulmonary irritation ranging from mild bronchitis to pulmonary edema. No chronic systemic effects have been observed.

MEDICAL SURVEILLANCE

Preplacement and periodic examinations should be directed to evaluation of the general health with particular reference to the skin, eyes, mucous membranes, and respiratory tract.

SPECIAL TESTS

None.

PERSONAL PROTECTIVE METHODS

In areas where concentrated hydrogen peroxide is being used, if there is danger of spill or splash, skin protection should be provided by protective clothing, gloves, goggles, and boots. Where fumes or vapor are excessive, workers should be provided with gas masks with full face pieces and proper canisters or supplied air respirators. Additional health hazards may occur from the decomposition of hydrogen peroxide. Oxygen, possibly at high pressure, may form, which may create an explosion hazard. Hydrogen peroxide is generally handled in a closed system to prevent contamination.

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HYDROGEN SULFIDE

DESCRIPTION

H_2S, hydrogen sulfide, is a flammable, colorless gas with a characteristic rotten-egg odor and is soluble in water.

SYNONYMS

Sulfuretted hydrogen, hydrosulfuric acid, stink damp.

POTENTIAL OCCUPATIONAL EXPOSURES

Hydrogen sulfide is used in the synthesis of inorganic sulfides, sulfuric acid, and organic sulfur compounds, as an analytical reagent, as a disinfectant in agriculture, and in metallurgy. It is generated in many industrial processes as a by-product and also during the decomposition of sulfur-containing organic matter, so potential for exposure exists in a variety of situations. Hydrogen sulfide is found in natural gas, volcanic gas, and in certain natural spring waters. It may also be encountered in the manufacture of barium carbonate, barium salt, cellophane, depilatories, dyes and pigments, felt, fertilizer, adhesives, viscose rayon, lithopone, synthetic petroleum products; in the processing of sugar beets; in mining, particularly where sulfide ores are present; in sewers and sewage treatment plants; during excavation of swampy or filled ground for tunnels, wells, and caissons; during drilling of oil and gas wells; in purification of hydrochloric acid and phosphates; during the low temperature carbonization of coal; in tanneries, breweries, slaughterhouses; in fat rendering; and in lithography and photoengraving.

A partial list of occupations in which exposure may occur includes:

Barium carbonate makers
Brewery workers
Caisson workers
Cellophane makers
Coke oven workers
Depilatory makers
Dye makers
Fat renderers
Felt makers
Lithographers
Miners
Natural gas makers
Paper pulp makers
Photoengravers
Rayon makers
Sewage treatment plant workers
Sewer workers
Silk makers
Slaughterhouse workers
Soap makers
Sugar beet processors
Sulfuric acid purifiers
Sulfur makers
Synthetic fiber makers
Tannery workers
Tunnel workers
Well diggers

PERMISSIBLE EXPOSURE LIMITS

The Federal standard is a ceiling value of 20 ppm (30 mg/m^3) with a maximum peak above this value for an 8-hour shift of 50 ppm (75 mg/m^3) for a maximum duration of 10 minutes once only if no other measurable exposure occurs.

ROUTE OF ENTRY

Inhalation of gas.

HARMFUL EFFECTS

Local—

Palpebral edema, bulbar conjunctivitis, keratoconjunctivitis, and ocular lesions may occur when hydrogen sulfide comes in contact with the eyes. Photophobia and lacrimation may also develop. Direct irritation of the respiratory tract may cause rhinitis, pharyngitis, bronchitis, and pneumonia. Hydrogen sulfide may penetrate deep into the lungs and cause hemorrhagic pulmonary edema. Hydrogen sulfide's irritative effects are due to the formation of alkali sulfide when the gas comes in contact with moist tissues.

Systemic—

Acute exposure may cause immediate coma which may occur with or without convulsions. Death may result with extreme rapidity from respiratory failure. Post-mortem signs include a typical greenish cyanosis of the chest and face with green casts found in viscera and blood. The toxic action of hydrogen sulfide is thought to be due to inhibition of cytochrome oxidase by binding iron which is essential for cellular respiration. Subacute exposure results in headache, dizziness, staggering gait, and excitement suggestive of neurological damage, and nausea and diarrhea suggestive of gastritis. Recovery is usually complete although rarely polyneuritis may develop as a result of vestibular and extrapyra-

midal tract damage. Tremors, weakness, and numbness of extremities may also occur. Physicians may observe a "rotten-egg" breath and abnormal electrocardiograms in victims. Systemic effects from chronic exposure to hydrogen sulfide have not been established.

MEDICAL SURVEILLANCE

Preplacement medical examinations should evaluate any preexisting neurological, eye, and respiratory conditions and any history of fainting seizures.

SPECIAL TESTS

None in common use for surveillance purposes.

PERSONAL PROTECTIVE METHODS

Hydrogen sulfide's strong odor, noticeable at low concentrations, is a poor warning sign as it may cause olfactory paralysis, and some persons are congenitally unable to smell H_2S.

Accidental exposure may occur when workers enter sewage tanks and other confined areas in which hydrogen sulfide is formed by decomposition. In a number of cases workers enter unsuspectingly and collapse almost immediately. Workers, therefore, should not enter enclosed spaces without proper precautions.

All Federal standard and other safety precautions must be observed when tanks or other confined spaces are to be entered. In areas where the exposure to hydrogen sulfide exceeds the standards, workers should be provided with fullface canister gas masks or preferably supplied air respirators.

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NITROGEN OXIDES

DESCRIPTION

Nitrogen oxides include:
Nitrous oxide: N_2O
Nitric oxide: NO
Nitrogen dioxide: NO_2

Nitrogen trioxide: N_2O_3
Nitrogen tetroxide: N_2O_4
Nitrogen pentoxide: N_2O_5
Nitric acid: HNO_3
Nitrous acid: HNO_2

Nitrous oxide, N_2O, is a colorless, noncombustible gas, sweet-tasting, and slightly soluble in water. Nitric oxide is a colorless gas slightly soluble in water. Nitric oxide combines with oxygen to form nitrogen dioxide which is a reddish-brown gas with a characteristic odor. Nitrogen dioxide exists in equilibrium with nitrogen tetroxide, and these two compounds and oxygen are in equilibrium with the crystalline nitrogen pentoxide. However, nitrogen dioxide and nitric oxide are the dissociation products of nitrogen trioxide. When nitrogen dioxide comes in contact with water, nitrous acid and nitric acid are formed. Nitric acid is a colorless liquid when pure, but on exposure to light, the liquid may turn yellowish-brown as a result of nitrogen dioxide formation. Nitric acid mist almost always contains nitrogen oxide gases and is, therefore, included in this group. Nitrogen dioxide decomposes in water, Nitrogen pentoxide is slightly soluble in water and nitric acid (70% aqueous solution) is soluble in water.

SYNONYMS

Nitrous oxide: Nitrogen monoxide
Nitric oxide: Mononitrogen monoxide
Nitrogen dioxide: None
Nitrogen trioxide: Dinitrogen trioxide, nitrous anhydride
Nitrogen tetroxide: Dinitrogen tetroxide
Nitrogen pentoxide: Nitric anhydride
Nitrous acid: None
Nitric acid: Aqua fortis, azotic acid, hydrogen nitrate

POTENTIAL OCCUPATIONAL EXPOSURES

Exposure to nitrogen oxides is typically a mixed exposure to "nitrous fumes" which may evolve from various manufacturing processes and in many other industrial situations. Exposure to nitrogen oxides may occur during the manufacture of nitric and sulfuric acid, oxidized cellulose compounds, explosives, rocket propellants, fertilizers, dyes and dyestuffs, pharmaceuticals, and various other organic and inorganic chemicals such as nitrites, nitrates, and other nitro compounds, aqua regia, arsenic acid, oxalic acid, nitrous acid, phthalic acid, and phosphoric acid. Exposure may also occur during jewelry manufacturing, etching, brazing, lithographing, metal cleaning, textile (rayon) and food bleaching, glass blowing, electroplating, gas and electric arc welding, and during the nitration of chloroform. Nitrogen oxides also occur in garages from automobile exhaust, in silos from organic material decomposition, in tunnels following blasting, and when nitric acid comes in contact with organic materials.

A partial list of occupations in which exposure may occur includes:

Braziers	Medical technicians
Dentists	Metal cleaners
Dye makers	Nurses
Fertilizer makers	Organic chemical synthesizers
Food and textile bleachers	Photoengravers
Garage workers	Physicians
Gas and electric arc welders	Silo fillers
Jewelry makers	Sulfuric acid makers

PERMISSIBLE EXPOSURE LIMITS

The Federal standards are: nitric oxide — 25 ppm (30 mg/m^3), nitrogen dioxide — 5 ppm (9 mg/m^3), and nitric acid — 2 ppm (5 mg/m^3); determined as a TWA. Currently there are no standards for the other listed compounds. NIOSH has recommended a ceiling level of 1 ppm for nitrogen dioxide.

ROUTES OF ENTRY

Inhalation of gas in the case of nitrogen oxide gases; inhalation of the mist or vapor, in the case of nitric and nitrous acids.

HARMFUL EFFECTS

Local—

Nitrogen oxide gases may produce irritation of the eyes and mucous membranes. Prolonged low level exposure may produce yellowish or brownish staining of the skin and teeth; however, this sign usually indicates nitric acid exposure. Nitric acid and nitrogen tetroxide are extremely corrosive liquids and may cause severe burns, ulcers, and necrosis of the skin, mucous membranes, and eye tissues.

Systemic—

Exposure to high concentrations of nitrogen oxides may result in severe pulmonary irritation and methemoglobinemia. The former is believed to be caused by the nitrogen dioxide portion, while the latter is mainly caused by nitric oxide. It is postulated that nitric oxide is nonirritating but the distinction is of questionable importance since nitric oxide exposure generally includes other nitrogen oxides; moreover, nitric oxide at even moderate concentrations oxidizes rapidly and spontaneously in the presence of atmospheric oxygen.

Nitrogen dioxide at high concentrations has also been shown to cause methemoglobinemia in the dog. Typically, acute exposure may produce immediate malaise, cyanosis, cough, dyspnea, chills, fever, headache, nausea, and vomiting. Collapse and death may occur if exposure is sufficiently high. When lower concentrations are encountered, there may be only mild signs of bronchial irritation followed by a five- to twelve-hour symptom-free period. Subsequently, the onset of signs and symptoms of acute pulmonary edema occur suddenly, which unfortunately may take place away from prompt medical aid.

Nitrogen oxides may be formed from green silage in amounts which,

when restrained to the confines of a silo, may constitute a serious health hazard. "Silo-filler's disease" is the name used to designate the syndrome culminating in bronchiolitis fibrosa obliterans, caused by exposure to nitrogen oxides evolved in this way.

If the acute episode is survived, bronchiolitis fibrosa obliterans may develop usually within a few days but may be latent for as long as six weeks. Victims may develop severe and increasing dyspnea which is often accompanied by fever and cyanosis. Chest roentgenogram may reveal a diffuse, reticular, and fine nodular infiltration or numerous, uniform, scattered nodular densities ranging in size from 1 to 5 mm in diameter.

Chronic exposure may result in pulmonary dysfunction with decreased vital capacity, maximum breathing capacity and lung compliance, and increased residual volume. The most common complaint is of dyspnea upon exertion. Signs include moist rales and wheezes, sporadic cough with mucopurulent expectoration, a decrease in blood pH and serum proteins, and an increase in urinary hydroxyproline and acid mucopolysaccharides. These findings are suggestive of emphysema, although they are as yet inconclusive.

The development of methemoglobinemia is typically mild and transient. In rare cases individuals may have a preexisting constantly high methemoglobin level due to a genetic defect. Such individuals are more susceptible to toxic methemoglobinemia.

MEDICAL SURVEILLANCE

Preplacement and periodic examinations should be concerned particularly with the skin, eyes, and with significant pulmonary and heart diseases. Periodic chest X-rays and pulmonary function tests may be useful. Smoking history should be known. Methemoglobin studies may be of interest if exposure to nitric oxide is present. In the case of nitric acid vapor mist exposure, dental effects may be present.

SPECIAL TESTS

None.

PERSONAL PROTECTIVE METHODS

Workers should not enter confined areas where nitrogen oxides may accumulate (for example, silos) without appropriate eye and respiratory protection.

Individuals should be equipped with supplied air respirators with full face piece or chemical goggles, and enclosed areas should be properly ventilated before entering. An observer equipped with appropriate respiratory protection should be outside the area and standing by to supply any aid needed.

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OZONE

DESCRIPTION

O_3, ozone, is a bluish gas with a characteristic pungent odor, slightly soluble in water. Ozone is found naturally in the atmosphere as a result of the action of solar radiation and electrical storms. It is also formed around electrical sources such as X-ray or ultraviolet generators, electric arcs, mercury vapor lamps, linear accelerators, and electrical discharges.

SYNONYMS

None.

POTENTIAL OCCUPATIONAL EXPOSURES

Ozone is used as an oxidizing agent in the organic chemical industry (e.g., production of azelaic acid); as a disinfectant for food in cold storage rooms and for water (e.g., public water supplies, swimming pools, sewage treatment); for bleaching textiles, waxes, flour, mineral oils and their derivatives, paper pulp, starch, and sugar; for aging liquor and wood; for processing certain perfumes, vanillin, and camphor; in treating industrial wastes; in the rapid drying of varnishes and printing inks; and in the deodorizing of feathers.

Industrial exposure often occurs around ozone generating sources, particularly during inert-gas shielded arc welding.

A partial list of occupations in which exposure may occur includes:

- Air treaters
- Arc welders
- Cold storage food preservers
- Industrial waste treaters
- Liquor agers
- Odor controllers
- Oil bleachers
- Organic chemical synthesizers
- Sewage treaters
- Textile bleachers
- Water treaters
- Wax bleachers
- Wood agers

PERMISSIBLE EXPOSURE LIMITS

The Federal standard is 0.1 ppm (0.2 mg/m^3).

ROUTES OF ENTRY

Inhalation of the gas.

HARMFUL EFFECTS

Local—

Ozone is irritating to the eyes and all mucous membranes. In human exposures, the respiratory signs and symptoms in order of increasing ozone concentrations are: dryness of upper respiratory passages; irritation of mucous membranes of nose and throat; choking, coughing, and severe fatigue; bronchial irritation, substernal soreness, and cough. Pulmonary edema may occur, sometimes several hours after exposure has ceased. In severe cases, the pulmonary edema may be fatal.

Animal experiments demonstrate that ozone causes inflammation and congestion of respiratory tract and, in acute exposure, pulmonary edema, hemorrhage, and death.

Chronic exposure of laboratory animals resulted in chronic bronchitis, bronchiolitis, emphysematous and fibrotic changes in pulmonary parenchyma.

Systemic—

Symptoms and signs of subacute exposure include headache, malaise, shortness of breath, drowsiness, reduced ability to concentrate, slowing of heart and respiration rate, visual changes, and decreased desaturation of oxyhemoglobin in capillaries. Animal experiments with chronic exposure showed aging effects and acceleration of lung tumorigenesis in lung-tumor susceptible mice.

Animal experiments further demonstrated that tolerance to acute pulmonary effects of ozone is developed and that this provided cross tolerance to other edemagenic agents. Antagonism and synergism with other chemicals also occur.

Ozone also has radiomimetic characteristics, probably related to its free-radical structure. Experimentally produced chromosomal aberrations have been observed.

MEDICAL SURVEILLANCE

Preemployment and periodic physical examinations should be concerned especially with significant respiratory diseases. Eye irritation may also be important. Chest X-rays and periodic pulmonary function tests are advisable.

SPECIAL TESTS

None.

PERSONAL PROTECTIVE METHODS

In areas of excessive concentration, gas masks with proper canister and fullface piece or goggles or the use of supplied air respirators is recommended.

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PHOSGENE

DESCRIPTION

$COCl_2$, phosgene, is a colorless, noncombustible gas with a sweet, not pleasant odor in low concentrations. In higher concentrations, it is irritating and pungent. It decomposes in water but is soluble in organic solvents.

SYNONYMS

Carbonyl chloride, carbon oxychloride, carbonic acid dichloride, chloroformyl chloride, combat gas.

POTENTIAL OCCUPATIONAL EXPOSURES

Phosgene is used in the manufacture of dyestuffs based on triphenylmethane, coal tar, and urea. It is also used in the organic synthesis of isocyanates and their derivatives, carbonic acid esters (polycarbonates), and acid chlorides. Occasional applications include its utilization in metallurgy, and in the manufacture of some insecticides and pharmaceuticals.

A partial list of occupations in which exposure may occur includes:

- Chlorinated compound makers
- Drug makers
- Dye makers
- Firemen
- Insecticide makers
- Metallurgists
- Organic chemical synthesizers

PERMISSIBLE EXPOSURE LIMITS

The Federal standard for concentrations of phosgene in air is 0.1 ppm (0.4 mg/m^3).

ROUTE OF ENTRY

Inhalation of gas.

HARMFUL EFFECTS

Local—

Conjunctivitis, lacrimation, and upper respiratory tract irritation may develop from gas. Liquid may cause severe burns.

Systemic—

Acute exposure to phosgene may produce pulmonary edema frequently preceded by a latent period of 5-6 hours but seldom longer than 12 hours. The symptoms are dizziness, chills, discomfort, thirst, increasingly tormenting cough, and viscous sputum. Sputum may then be-

come thin and foamy, and dyspnea, a feeling of suffocation, tracheal rhonci, and grey-blue cyanosis may follow. Death may result from respiratory or cardiac failure. The hazard of phosgene is increased because at low levels (205 mg/m^3) it is lacking in warning symptoms.

Chronic exposure to phosgene may result in some tolerance to acute edemagenic doses, but may cause irreversible pulmonary changes of emphysema and fibrosis. Animal experimentation has shown an increased incidence of chronic pneumonitis and acute and fibrinous pneumonia from exposure to this agent.

MEDICAL SURVEILLANCE

Preemployment medical examinations should include chest X-rays and baseline pulmonary function tests. The eyes and skin should be examined. Smoking history should be known. Periodic pulmonary function studies should be done. Workers who are known to have inhaled phosgene should remain under medical observation for at least 24 hours to insure that delayed symptoms do not occur.

SPECIAL TESTS

None.

PERSONAL PROTECTIVE METHODS

Where liquid phosgene is encountered, protective clothing should be supplied which is impervious to phosgene. Where gas is encountered above safe limits, fullface gas masks with phosgene canister or supplied air respirators should be used.

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PORTLAND CEMENT

DESCRIPTION

Portland cement is a class of hydraulic cements whose two essential constituents are tricalcium silicate and dicalcium silicate with varying amounts of alumina, tricalcium aluminate, and iron oxide. The quartz content of most is below one percent. The average composition of regular Portland cement is as follows:

CaO: 64%
SiO_2: 21%
Al_2O_3: 5.8%
FeO_3: 2.9%
MgO: 2.5%
Alkali Oxides: 1.4%
SO_3: 1.7%

SYNONYMS

None.

POTENTIAL OCCUPATIONAL EXPOSURES

Cement is used as a binding agent in mortar and concrete (a mixture of cement, gravel, and sand). Potentially hazardous exposure may occur during both the manufacture and use of cement.

A partial list of occupations in which exposure may occur includes:

- Asbestos cement workers
- Brick masons
- Bridge builders
- Building construction workers
- Burial vault builders
- Cement workers
- Concrete workers
- Drain tile makers
- Heat insulation makers
- Oil well builders
- Silo builders
- Storage tank builders
- Tunnel builders
- Water pipe makers

PERMISSIBLE EXPOSURE LIMITS

The Federal standard for Portland cement is 50 mppcf.

ROUTE OF ENTRY

Inhalation of dust.

HARMFUL EFFECTS

Local—

Exposure may produce cement dermatitis which is usually due to primary irritation from the alkaline, hygroscopic, and abrasive properties of cement. Chronic irritation of the eyes and nose may occur. In some cases, cement workers have developed an allergic sensitivity to constituents of cement such as hexavalent chromate. It is not unusual for cement dermatitis to be prolonged and to involve covered areas of the body.

Systemic—

No documented cases of pneumoconiosis or other systemic manifestations attributed to finished Portland cement exposure have been reported. Conflicting reports of pneumoconiosis from cement dust appear related to exposures that occurred in mining, quarrying, or crushing silica-containing raw materials.

MEDICAL SURVEILLANCE

Preemployment and periodic medical examinations should stress significant respiratory problems, chest X-ray, pulmonary function tests, smoking history, and allergic skin sensitivities, especially to chromates. The eyes should be examined.

SPECIAL TESTS

Patch test studies may be useful in dermatitis cases.

PERSONAL PROTECTIVE METHODS

In areas exceeding safe dust levels, masks with proper cartridges

should be provided. Gloves, barrier creams, and protective clothing (long sleeved shirts, etc.) will help protect workers subject to dermatitis. Personal hygiene is very important, and all cement workers should be encouraged to shower following each shift before changing to street clothes. Freshly laundered work clothing should be supplied on a daily basis.

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SODIUM HYDROXIDE/POTASSIUM HYDROXIDE

DESCRIPTION

NaOH, sodium hydroxide, is a white, deliquescent material sold as pellets, flakes, lumps, or sticks. It is soluble in water, alcohol, and glycerine. Aqueous solutions are known as soda lye.

KOH, potassium hydroxide, exists as white or slightly yellow deliquescent lumps, rods, or pellets. It is soluble in water. Aqueous solutions are known as lye.

SYNONYMS

Sodium hydroxide: caustic soda, caustic alkali, caustic flake, sodium hydrate.

Potassium hydroxide: potassium hydrate, caustic potash, potassa, caustic alkali.

POTENTIAL OCCUPATIONAL EXPOSURES

Sodium hydroxide is utilized to neutralize acids and make sodium salts in petroleum refining, viscose rayon, cellophane, and plastic production, and in the reclamation of rubber. It hydrolyzes fats to form soaps, and it precipitates alkaloids and most metals from aqueous solutions of their salts. It is used in the manufacture of mercerized cotton, paper, explosives, and dyestuffs, in metal cleaning, electrolytic extraction of zinc, tin plating, oxide coating, laundering, bleaching, and dishwashing, and it is used in the chemical industries.

Potassium hydroxide is used in the manufacture of liquid soap, as a mordant for wood, as a carbon dioxide absorber, in mercerizing cotton, in electroplating, photoengraving, and lithography, in printing inks, in paint and varnish removers, and in analytical chemistry, organic synthesis, and the production of other potassium compounds.

A partial list of occupations in which exposure may occur includes:

Bleachers	Paint removers
Bleach makers	Paper makers
Cellophane makers	Photoengravers
Chemical laboratory workers	Printers
Dye makers	Printing ink makers
Electroplaters	Rayon makers
Etchers	Rubber reclaimers
Explosive makers	Soap makers
Laundry workers	Textile bleachers
Lithographers	Tin platers
Mercerizers	Varnish removers
Organic chemical synthesizers	Zinc extractors

PERMISSIBLE EXPOSURE LIMITS

The Federal standard for sodium hydroxide is 2 mg/m^3. There is no standard for potassium hydroxide.

ROUTE OF ENTRY

Inhalation of dust or mist.

HARMFUL EFFECTS

Local—

Both compounds are extremely alkaline in nature and are very corrosive to body tissues. Dermatitis may result from repeated exposure to dilute solutions in the form of liquids, dusts, or mists.

Systemic—

Systemic effects are due entirely to local tissue injury. Extreme pulmonary irritation may result from inhalation of dust or mist.

MEDICAL SURVEILLANCE

The skin, eyes, and respiratory tract should receive special attention in any placement or periodic examination.

SPECIAL TESTS

None.

PERSONAL PROTECTIVE METHODS

Protection should be provided by impervious protective clothing, rubber boots, face and eye shields, and dust respirators. All skin area burns, especially of the eyes, demand immediate care by flooding with large quantities of water for 15 minutes or longer and specialized medical care.

SULFUR CHLORIDE

DESCRIPTION

S_2Cl_2, sulfur chloride, is a fuming, oily liquid with a yellowish-red

to amber color and a suffocating odor. It has an added hazard since it oxidizes and hydrolyzes to sulfur dioxide and hydrogen chloride.

SYNONYMS

Sulfur monochloride, sulfur subchloride, disulfur dichloride.

POTENTIAL OCCUPATIONAL EXPOSURES

Sulfur chloride finds use as a chlorinating agent and an intermediate in the manufacture of organic chemicals, e.g., carbon tetrachloride, and sulfur dyes, insecticides, synthetic rubber, and pharmaceuticals. Exposure may also occur during the extraction of gold, purification of sugar juice, finishing and dyeing textiles, processing vegetable oils, hardening wood, and vulcanization of rubber.

A partial list of occupations in which exposure may occur includes:

- Carbon tetrachloride makers
- Drug makers
- Gold extractors
- Insecticide makers
- Rubber workers
- Sugar juice purifiers
- Sulfur dye makers
- Synthetic rubber makers
- Textile dye makers and finishers
- Vegetable oil processors
- Wood hardeners

PERMISSIBLE EXPOSURE LIMITS

The Federal standard for sulfur chloride (sulfur monochloride) is 1 ppm (6 mg/m^3).

ROUTE OF ENTRY

Inhalation of vapor.

HARMFUL EFFECTS

Local—

Fumes, in sufficient quantity, may cause severe irritation to eyes, skin, and mucous membranes of the upper respiratory tract.

Systemic—

Although this compound is capable of producing severe pulmonary irritation, very few serious cases of industrial exposure have been reported. This is probably because the pronounced irritant effects of sulfur chloride serve as an immediate warning signal when concentration of the gas approaches a hazardous level.

MEDICAL SURVEILLANCE

Preemployment and periodic examinations should give special emphasis to the skin, eyes, and respiratory system. Pulmonary function tests may be useful. Exposures may also include sulfur dioxide and hydrochloric acid. (See these compounds).

SPECIAL TESTS

None are known to be useful.

PERSONAL PROTECTIVE METHODS

In areas where vapor levels are excessive, workers should be supplied with fullface gas masks with proper canister or supplied air respirators with full face piece. Skin protection can usually be afforded by work clothes and barrier creams, but under certain instances (spills, etc.), full impervious protective suits may be necessary.

SULFUR DIOXIDE

DESCRIPTION

SO_2, sulfur dioxide, is a colorless gas at ambient temperatures with a characteristic strong suffocating odor. It is soluble in water and organic solvents.

SYNONYMS

Sulfurous anhydride, sulfurous oxide.

POTENTIAL OCCUPATIONAL EXPOSURES

Sulfur dioxide is used in the manufacture of sodium sulfite, sulfuric acid, sulfuryl chloride, thionyl chloride, organic sulfonate, disinfectants, fumigants, glass, wine, ice, industrial and edible protein, and vapor pressure thermometers. It is also used in the bleaching of beet sugar, flour, fruit, gelatin, glue, grain, oil, straw, textiles, wicker ware, wood pulp, and wool; in the tanning of leather; in brewing and preserving; and in the refrigeration industry. Exposure may also occur in various other industrial processes as it is a by-product of ore smelting, coal and fuel oil combustion, paper manufacturing, and petroleum refining.

A partial list of occupations in which exposure may occur includes:

- Beet sugar bleachers
- Boiler water treaters
- Brewery workers
- Disinfectant makers
- Diesel engine operators and repairmen
- Firemen
- Fumigant makers
- Furnace operators
- Gelatin bleachers
- Glass makers
- Ice makers
- Ore smelter workers
- Paper makers
- Petroleum refinery workers
- Protein makers
- Refrigeration workers
- Sodium sulfite makers
- Sulfuric acid makers
- Tannery workers
- Thermometer makers (vapor)
- Wine makers
- Wood bleachers

PERMISSIBLE EXPOSURE LIMITS

The Federal standard is 5 ppm (13 mg/m^3). NIOSH has recommended lowering this standard to 2 ppm as a TWA.

ROUTE OF ENTRY

Inhalation of gas. Direct contact of gas or liquid phase on skin and mucous membranes.

HARMFUL EFFECTS

Local—

Gaseous sulfur dioxide is particularly irritating to mucous membranes of the upper respiratory tract. Chronic effects include rhinitis, dryness of the throat, and cough. Conjunctivitis, corneal burns, and corneal opacity may occur following direct contact with liquid.

Systemic—

Acute over-exposure may result in death from asphyxia. Survivors may later develop chemical bronchopneumonia with bronchiolitis obliterans. Bronchoconstriction with increased pulmonary resistance, high-pitched rales, and a tendency to prolongation of the expiratory phase may result from moderate exposure, though bronchoconstriction may be asymptomatic. The effects on pulmonary function are increased in the presence of respirable particles.

Chronic exposure may result in nasopharyngitis, fatigue, altered sense of smell, and chronic bronchitis symptoms such as dyspnea on exertion, cough, and increased mucous excretion. Transient stimulation of erythropoietic activity of the bone marrow has been reported. Slight tolerance, at least to the odor threshold, and general acclimatization are common. Sensitization in a few individuals, particularly young adults, may also develop following repeated exposures. There is some evidence that some individuals may be innately hypersusceptible to SO_2. Animal experimentation has also indicated that sulfur dioxide may be a possible co-carcinogenic agent.

MEDICAL SURVEILLANCE

Preplacement and periodic medical examinations should be concerned especially with the skin, eye, and respiratory tract. Pulmonary function should be evaluated, as well as smoking habits, and exposure to other pulmonary irritants.

SPECIAL TESTS

None commonly used.

PERSONAL PROTECTIVE METHODS

In areas where levels of sulfur dioxide gas are excessive, the worker should be supplied with fullface piece cartridge or canister respirator or with supplied air respirators. Goggles, protective clothing, and gloves should be worn if splashes with liquid are likely. Work clothing should be changed at least twice a week to freshly laundered work clothes. Showering following each work shift should be encouraged. In areas of splash or spill, impervious clothing should be supplied, but if work clothes are wetted by sulfur dioxide, they should be promptly removed and the skin area thoroughly washed.

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SULFURIC ACID

DESCRIPTION

H_2SO_4, concentrated sulfuric acid, is a colorless, odorless, oily liquid which is commercially sold at 93 to 98% H_2SO_4, the remainder being water. Fuming sulfuric acid (oleum) gives off free sulfur trioxide and is a colorless or slightly colored, viscous liquid. Sulfuric acid is soluble in water and alcohol.

SYNONYMS

Oil of vitriol, spirit of vitriol, spirit of sulfur, hydrogen sulfate.

POTENTIAL OCCUPATIONAL EXPOSURES

Sulfuric acid is used as a chemical feedstock in the manufacture of acetic acid, hydrochloric acid, citric acid, phosphoric acid, aluminum sulfate, ammonium sulfate, barium sulfate, copper sulfate, phenol, superphosphates, titanium dioxide, as well as synthetic fertilizers, nitrate explosives, artificial fibers, dyes, pharmaceuticals, detergents, glue, paint, and paper. It finds use as a dehydrating agent for esters and ethers due to its high affinity for water, as an electrolyte in storage batteries, for the hydrolysis of cellulose to obtain glucose, in the refining of mineral and vegetable oil, and in the leather industry. Other uses include fur and food processing, carbonization of wool fabrics, gas drying, uranium extraction from pitchblende, and laboratory analysis.

A partial list of occupations in which exposure may occur includes:

- Aluminum sulfate makers
- Battery makers
- Cellulose workers
- Chemical synthesizers
- Copper sulfate makers
- Detergent makers
- Dye makers
- Explosive makers
- Food processors
- Glue makers
- Jewelers
- Leather workers
- Metal cleaners
- Paint makers
- Paper makers
- Phenol makers

PERMISSIBLE EXPOSURE LIMITS

The Federal standard for sulfuric acid is 1 mg/m^3.

ROUTE OF ENTRY

Inhalation of mist.

HARMFUL EFFECTS

Local—

Burning and charring of the skin are a result of the great affinity for, and strong exothermic reaction with, water. Concentrated sulfuric acid

will effectively remove the elements of water from many organic materials with which it comes in contact. It is even more rapidly injurious to mucous membranes and exceedingly dangerous to the eyes. Ingestion causes serious burns of the mouth or perforation of the esophagus or stomach. Dilute sulfuric acid does not possess this property, but is an irritant to skin and mucous membranes due to its acidity and may cause irreparable corneal damage and blindness as well as scarring of the eyelids and face.

Systemic—

Sulfuric acid mist exposure causes irritation of the mucous membranes, including the eye, but principally the respiratory tract epithelium. The mist also causes etching of the dental enamel followed by erosion of the enamel and dentine with loss of tooth substance. Central and lateral incisors are mainly affected. Breathing high concentrations of sulfuric acid causes tickling in the nose and throat, sneezing, and coughing. At lower levels sulfuric acid causes a reflex increase in respiratory rate and diminution of depth, with reflex bronchoconstriction resulting in increased pulmonary air flow resistance. A single overexposure may lead to laryngeal, tracheobronchial, and pulmonary edema. Repeated excessive exposures over long periods have resulted in bronchitic symptoms, and rhinorrhea, lacrimation, and epistaxis. Long exposures are claimed to result in conjunctivitis, frequent respiratory infections, emphysema, and digestive disturbances.

MEDICAL SURVEILLANCE

Preplacement and periodic medical examinations should give special consideration to possible effects on the skin, eyes, teeth, and respiratory tract. Pulmonary function tests should be performed.

SPECIAL TESTS

None commonly used.

PERSONAL PROTECTIVE METHODS

In all areas where liquid sulfuric acid is handled, impervious clothing should be provided, including gloves, goggles or face mask, rubber suits, and rubber shoes. Any work clothing wetted by sulfuric acid should be immediately changed and the skin area thoroughly washed and flooded with water. In areas where mist or gas is excessive, gas masks with appropriate canister or supplied air respirators should be provided. In either instance the worker should be supplied with full face protection.

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SECTION VIII

Posterity may know we have not loosely through silence permitted things to poss away as in a dream.

—*Richard Hooker*

CHEMICAL CARCINOGENS

Kenneth Bridbord, M.D., Joseph K. Wagoner, S.D.Hyg., and Hector P. Blejer, M.D.

Recogniton of the causative role that chemicals in the workplace play in carcinogenesis dates back to 1775 when Percival Pott, a London surgeon, linked a high prevalence of scrotal cancer in young chimney sweepers to their occupational exposure to soot produced by the coal burned in the chimneys they cleaned. Today, over 200 years after Dr. Pott's discovery, workers in the United States and other industrialized countries are exposed to a multitude of chemicals, many of which are recognized as, or suspected to be, carcinogens.

Carcinogenic effects of chemicals in man are difficult to document because 1) cancers are generally not clinically evident until a lapse of up to 20 to 30 years after the first exposure has occurred and 2) chemical exposures in many workplaces are so complex that it is difficult to pinpoint the specific causal agent or agents and the concentrations of the agents primarily responsible for the ultimate carcinogenic effect manifested decades later.

For example, workers occupationally exposed to coke oven emissions are at increased risk of developing lung and kidney cancer. Yet, in spite of lengthy research and the evidence as related to cancer, all the specific carcinogens in the chemically complex coke oven exposures responsible for the increased carcinogenic risks have not been determined.

LEVEL OF EXPOSURE

There is considerable debate among scientists regarding the level of exposure to a given carcinogen required to cause cancer. It is beyond the scope of this chapter to review all of the arguments offered as to whether or not a threshold exists for chemical carcinogenesis.

We have currently no established scientific method to determine threshold levels for chemical carcinogens, if indeed such thresholds do exist. Moreover, if a threshold for a given chemical carcinogen were to exist, it would not necessarily be determinative of a safe exposure since in the industrial environment workers may be exposed to multiple carcinogenic agents which may compete for the same target site.

Multiple exposures can occur on the job, in the diet, and in the ambient and home environments. Under these circumstances, some people may have already received doses from multiple exposures in excess of any presumed threshold for any single carcinogenic chemical. Consequently, any incremental increased exposure to chemical carcinogens could then result in an increased risk of cancer, especially if this incremental exposure may already be in the area where the slope of the

dose-response curve has steepened (1). As a result, the National Institute for Occupational Safety and Health (NIOSH) has taken the position that it is not currently possible to demonstrate safe levels of exposure to all chemical carcinogens (2).

RECOGNITION OF HAZARDS

Carcinogenic hazards may be identified by epidemiologic studies of people who have been exposed to suspect chemical agents and by experiments in animals exposed to controlled amounts of chemical agents. In the workplace, the primary route of exposure is through inhalation although concomitant ingestion and skin contact can also be important. Consequently, inhalation exposure experiments in animals constitute the most relevant toxicologic approach for simulating the most prevalent exposure conditions in the workplace.

In assessing the evidence for cancer, be it from animal or epidemiologic data, one must consider the strengths and weaknesses of the individual studies as well as the consistency of evidence between studies. There are, however, no universally accepted criteria for the quality or the consistency of the data that are required before considering that a given chemical represents a carcinogenic hazard to man.

EPIDEMIOLOGIC STUDIES

Identification of carcinogenic hazards from epidemiologic data generally involves the use of cohort studies; that is, tracing the present and future mortality experience of groups of individuals exposed to a common chemical agent at or during a specific time period and comparing their mortality experience with a matched group not so exposed during the same time period.

Most frequently chosen as a control group in these studies is a group from the general population matched for age, sex, and race. By comparing the mortality experiences among the exposed and nonexposed control populations, it is possible to ascertain if exposure has increased the risk of a given cause of death. In this type of study, the calculation of a standardized mortality ratio (SMR) in order to compare the frequency of death from a given cause or causes in the exposed population with that in the control populations is extremely informative. An index of more than 1.0 indicates that an excess risk may exist in the exposed population. In such comparisons, however, because the working population may in general be healthier than the general population, an apparently less risky SMR of 0.90 to 1.0 on a specific cause of death may actually indicate an increased risk among those who have been exposed — the so-called "healthy worker effect."

As noted, the usual latent period or lapse time for development of chemically-induced cancer is about 20 years. Consequently, a mortality study which does not include an adequate proportion of workers with

long latent or lapse times following onset of exposure may yield erroneous conclusions as to the lack of health effects of those chemicals in the work environment that are being studied.

ANIMAL STUDIES

In epidemiologic studies, once a chemical or exposure condition has been shown to cause cancer, preventive measures may not be adequate to protect those who have had previous exposures, but who have not lived long enough for effects to be expressed in terms of clinical illness. A major advantage of experimental animal studies is the possibility of detecting a chemical cancer hazard earlier than if one waited for epidemiologic evidence of cancer in man to become available. Under such circumstances, preventive action can be taken much sooner.

To date, there are a number of instances in which data on cancer in experimental animals have been used to establish occupational health regulations in the United States. It is increasingly evident that experiments in animals can be important indicators of cancer risk for man. Almost all chemicals shown to be carcinogenic in man by epidemiologic studies have also been shown to be carcinogenic in appropriate animal models. Although this does not necessarily mean that a positive test for cancer in animals provides incontrovertible evidence of cancer risk for man, it does indicate that the chemical should be considered at least as a potential carcinogen for man.

Experts frequently recommend testing chemicals in more than one animal species, primarily to avoid false negative results. Nevertheless, this should not be interpreted to mean that, before a chemical can be called a carcinogen, it must be positive in two or more species tested. Naturally, however, the greater the number of studies that show that certain chemicals produce cancer in different species of laboratory animals, the greater the confidence in the conclusion that those substances pose a carcinogenic threat to man.

POTENTIAL OCCUPATIONAL EXPOSURES

The boundaries of potential occupational exposures to chemical carcinogens are ever expanding. The following occupations are some of those subject to recent investigations.

Asbestos workers
Auto repairmen
Bakery workers
Clothing pressers
Coke oven workers
Dairy industry workers
Dental laboratory technicians
Electricians
Leather workers
Photoengravers
Roofers
Rubber workers
Vinyl chloride workers

Table 4 presents a list of occupational chemicals and substances which cause, or are suspected of causing, cancer and the target organ or tissue. It should be emphasized that this list is substantially incomplete in that many, if not most, chemicals in the workplace have not been adequately tested for their carcinogenic potential. As such, however, the format of Table 4 attempts to organize a growing body of data in a manner that may be useful for physicians in making a differential diagnosis of the possible occupational etiology of cancer cases in individuals. Additionally, this list may lead physicians and other health professionals to become more aware of the magnitude of the growing problem of chemical carcinogens in the workplace. Since occupational carcinogens may effect virtually all organ systems, physicians should be alert to investigate situations where clinically evident cancer could be associated with on-the-job chemical exposures.

One helpful data source for physicians is the registry of suspected carcinogens maintained by NIOSH as a subfile of the Registry of Toxic Effects of Chemical Substances (3). This Registry contains approximately 1,500 suspect carcinogens, most of which have not been adequately tested. Their inclusion on this list does not represent a process of substantive evaluation with respect to the adequacy of scientific data related to carcinogenicity. Rather, this list is a useful starting point to ascertain the extent of data regarding carcinogenic responses for a given compound. Even with these caveats, it should be evident that a number of compounds in this list may be shown to be carcinogenic in man following more detailed evaluation.

Observations by alert physicians and alert workers have frequently helped to identify problems of carcinogenic risk in the workplace long before they might otherwise be realized. Examples of this are hepatic angiosarcoma, a rare liver cancer caused by occupational exposure to vinyl chloride, and leukemia among workers in the manufacture of styrene-butadiene rubber. A number of other occupational chemicals have been shown to produce "marker" or unusual forms of cancer, such as the pleural and peritoneal mesotheliomas due to asbestos, and hepatic angiosarcoma due to inorganic arsenic. It is likely that careful follow-up of rare cancers or unusually high incidences of common cancers may help to uncover unsuspected chemical cancer hazards among other worker populations.

Unsuspected occupational cancer problems might also be predicted on the basis of structural similarity with certain chemicals and substances already shown to cause cancer in humans or animals. For example, Table 5 lists compounds that by virtue of their structural similarity to vinyl chloride would be suspected of posing possible carcinogenic risks to man. Surveillance by alert physicians of workers exposed to these substances might help to early identify potential future problems. Similarly, it is most important for clinicians to be aware of newer data on carcinogenesis emerging from experimental studies on animals. For example, preliminary data have indicated that trichloroethylene produces liver cancer in experimental animals (4), and data from Russia have suggested a human carcinogenic lung and skin response to chloroprene (5).

Table 4. Confirmed and suspected occupational carcinogens by target organ.

Target Organ/Tissue	Occupational Carcinogen	
	Confirmed	Suspected
Bone		Beryllium
Brain	Vinyl Chloride	
Gastroenteric Tract	Asbestos	
Hematopoietic Tissue (leukemia)	Benzene Styrene Butadiene and other Rubber Manufacture Substances	
Kidney	Coke Oven Emissions	Lead
Larynx	Asbestos, Chromium	
Liver	Vinyl Chloride	Aldrin Carbon Tetrachloride Chloroform DDT Dieldrin Heptachlor PCB's Trichloroethylene
Lung	Arsenic Asbestos Bis (chloromethyl) ether Chloromethyl methyl ether Chromates Coke Oven Emissions Mustard Gas Nickel Soots and Tars Uranium Vinyl Chloride	Beryllium Cadmium Chloroprene Lead
Lymphatic Tissue		Arsenic Benzene
Nasal Cavity	Chromium, Isopropyl Oil, Nickel, Wood Dusts	
Pancreas		Benzidine PCB's
Pleural Cavity	Asbestos	
Prostate		Cadmium
Scrotum	Soots and Tars	
Skin	Arsenic Coke Oven Emissions Cutting Oils Soots and Tars	Chloroprene
Urinary Bladder	4-Aminobiphenyl Benzidine B-Naphthylamine	Auramine 4-Nitrodiphenyl Magenta

Table 5. Suspected carcinogens based upon structural similarity to vinyl chloride.

Suspected Carcinogen	Structure
Vinyl Chloride	$H_2C=CH$ $\quad\quad Cl$
Bromoprene	$H_2C=CHCH_2Br$
Chloroprene	$H_2C=CHCH_2Cl$
Epibromohydrin	$H_2C-CH-CH_2Br$ $\quad\quad\quad

IN VITRO SCREENING TESTS

In vitro screening techniques have been developed which have promise for identifying chemicals with potential for causing cancer (6). These techniques are based upon a number of end points such as mutations and inhibition of DNA repair mechanisms. Such tests are done on a number of cell systems, including human cell cultures and bacteria.

Preliminary evidence reveals that nearly 90 percent of chemicals established to be carcinogens by animal or human data give positive test results in one or more of these in vitro test systems(7) It is not known what proportion of chemicals selected at random without prior knowledge as to their carcinogenic activity will give a positive result in these tests. Strictly speaking these tests *per se* do not indicate carcinogenic activity. Nevertheless, they offer considerable promise in the identification of those chemical agents which should be tested for carcinogenicity. Thus, the practicing physician needs to know about these test procedures and results, because they may give important clues as to the existence of potentially carcinogenic chemicals.

INFORMATION SOURCES

Well over one thousand chemicals have shown at least one positive test result for carcinogenicity. These chemicals are in Suspected Carcinogens, A Subfile of the Toxic Substances List (3), published in 1975 by NIOSH and updated periodically. The International Agency on Research on Cancer has also published a series of monographs which review the carcinogenicity of a large number of chemicals (8). The National Cancer Institute (NCI) currently has approximately 300 chemicals on long-term test for cancer in animals and much new information will become available over the next several years.

Both NIOSH and NCI maintain systems which issue bulletins giving summarized, recent information on the carcinogenicity of chemicals. Copies of the bulletins may be obtained from these agencies. They are intended to make more people aware of new research information on chemicals and cancer so that appropriate medical and other precautions and action can be taken to monitor and prevent undue exposure. Additionally, such bulletins will likely lead to new research studies to clarify the existence and magnitude of suspected carcinogenic risks.

Occupational health physicians must be aware of these data sources. The information they provide on cancer risk may provide the clues which would facilitate the physicians' recognition of corroborative evidence based upon their own clinical experience. Such evidence would in turn help to stimulate adequate control actions to minimize future occupational and environmental exposure to these chemicals.

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SECTION IX

It may happen that the physician will be asked what to do in a department where all known mechanical devices for the prevention of poisonous dusts and vapors have been applied, and yet, because of the very nature of the process, there is still contamination of the air. . . .With newer poisons, it may be that the physician will have to study for himself the physiological effects in order to discover what sign or symptom can be depended on to give the needed warning of danger. . . .let me beg the industrial physician not to let the atmosphere of the factory befog his view of his special problem. His duty is to the producer, [worker], not to the product.

—Alice Hamilton

PESTICIDES *

Wesley E. Straub

This section deals with a discrete group of chemicals that are of particular importance in agriculture, pest control industries, and public health. Their use in crop production and disease control has increased with the expanding world population, and their complexity and number have increased in proportion to their expanded use.

No attempt is made to present the clinical effects of all pesticides currently in use nor to delineate definitive treatment. Some aspects of clinical treatment are presented, particularly where the information is of general application to more than one substance and is not generally available, and treatment must be rapidly instituted. Hazardous exposures may occur in both occupational and nonoccupational activities, and the physician should be on notice to consider both aspects of a worker's activities in checking for source of exposure.

In severe poisoning, the initial diagnosis and institution of appropriate treatment must be made on clinical grounds alone since there is generally insufficient time to wait for confirmatory laboratory results.

Essential to the correct diagnosis of pesticide poisoning is a high index of suspicion on the part of the physician based on 1) a history of opportunity for any adequate exposure compatible with time-dose relationships, 2) clinical manifestations, and 3) laboratory confirmation.

The toxic dose and clinical picture of poisoning vary with the compound and formulation, and possibly with the individual.

For purposes of the following discussion, the pesticides are grouped according to their chemical nature or use as organophosphates, carbamates, chlorinated hydrocarbons, bipyridyls, coumarins and indandiones, rodenticides, fungicides, herbicides, fumigants, and miscellaneous insecticides.

ORGANOPHOSPHATES

The more important organophosphates are:

Abate	Ethion
DDVP (Vapona)	Fenthion (Baytex)
Diazinon	Gardona
Dicathon	Malathion
Dimethoate (Cygon)	Naled (Dibrom)
Dursban	Parathion
EPN	

The organophosphate insecticides are characterized by the similarity of their mechanism of toxic action. They differ widely, however, in inherent toxicity and, to some extent, in rate of absorption and excretion.

The organophosphates act as irreversible inhibitors of the enzyme

*Based in some parts on the Chapter in the previous edition written by Roy L. Gibson, M.D., and Thomas H. Milby, M.D.

cholinesterase, thereby allowing the accumulation of acetylcholine at nerve endings. They are rapidly absorbed into the body by ingestion, through the intact skin, including the eye (even more efficiently through cuts, abrasions, areas of dermatitis, etc.), and by inhalation.

Dose and dose-interval affect the speed with which the toxic manifestations occur. Onset of symptoms more than 12 hours after the termination of exposure generally excludes the diagnosis of organophosphate poisoning. It must be remembered, however, that continuing exposure may occur from contaminated hair, shoes, and clothing.

The following table for parathion lists symptoms which are indicative not only for parathion, but for other organophosphate exposures.

Table 6. Signs and symptoms in patients with parathion poisoning as related to levels of cholinesterase activity.

Sign or symptoms	Total number of patients with signs or symptoms	Number of symptomatic patients in each of three levels of activity*		
		0-10% of normal	11-20% of normal	21-50% of normal
Weakness	47	14	11	22
Headache	46	14	11	21
Sweating	44	14	10	20
Nausea and vomiting	42	14	11	17
Salivation	31	13	8	10
Miosis	25	14	6	5
Dyspnea	23	14	6	3
Difficulty in walking	22	14	8	0
Diarrhea	21	9	4	8
Muscular fasciculation	20	14	6	0
Disturbance in speech	20	14	6	0
Disturbance in consciousness	19	14	5	0
Abdominal pain	15	5	4	6
Fever	15	9	6	0
Bronchopharyngeal secretion	14	10	4	0
Increased blood pressure	12	10	2	0
Loss of pupillary reflex	10	10	0	0
Cramp	9	9	0	0
Cyanosis	8	8	0	0

*As percent of value of each patient after recovery from poisoning.

Note: Modified from Nambe, T., C. T. Nolte, J. Jackrel, and D. Grob. 1971. Poisoning due to organophosphate insecticides: acute and chronic manifestations. Am. J. Med. 50:475-492. Reprinted with permission from author and publisher.

PERMISSIBLE EXPOSURE LIMITS

The Federal standards for parathion and malathion are 0.1 mg/m^3 and 15 mg/m^3, respectively. NIOSH has a recommended limit for parathion of 0.05 mg/m^3 (TWA) and a limit for methyl parathion of 0.2 mg/m^3 (TWA).

HARMFUL EFFECTS

Mild organophosphate poisoning causes symptoms of headache, fatigue, dizziness, blurred vision, excessive sweating, nausea and vomiting, stomach cramps, diarrhea, and salivation. These symptoms are similar to those of many diseases not related to pesticide exposure such as influenza, heat stroke, heat exhaustion, and gastroenteritis.

Moderately severe organophosphate poisoning causes all of the symptoms found in mild poisoning, but in addition, the patient is unable to walk, often complains of chest discomfort and tightness, exhibits marked miosis (constriction of the pupils), and exhibits muscle twitching. These symptoms might be reasonably mistaken for such conditions as pneumonia, myocardial infarction, and encephalitis.

Severe organophosphate poisoning may result in rapid onset of unconsciousness, local or generalized seizures, and other manifestations of a cholinergic crisis.

CLINICAL NOTES

An important clinical observation, in addition to those previously mentioned (high index of suspicion, history, clinical manifestations), which aids in the substantiation of the diagnosis of an anticholinesterase intoxication, is atropine refractoriness. When a larger than normal dose of atropine is given to a person not exposed to anticholinesterase pesticides, the early signs of atropine toxicity soon become apparent. These signs include dry mouth, flushed skin, increased heart rate, and dilated pupils. If the patient has anticholinesterase poisoning, large doses of atropine are required to produce these normal reactions.

The atropine test should be used with caution in patients with glaucoma. However, in acutely toxic patients suspected of organophosphate poisoning, immediate atropine therapy must be initiated even without tests of atropine refractoriness.

Acholest screening tests provide a simple but crude index of the degree of cholinesterase inhibition while offering the most immediate confirmation that is within the laboratory expertise of any hospital or clinic. Plasma cholinesterase determinations of this nature use filter paper impregnated with a pH-sensitive color reagent and can detect inhibition as low as 20 percent of serum cholinesterase. Plasma and red blood cell cholinesterase are more precise determinations, but errors have resulted from unfamiliarity of laboratory personnel with these procedures.

Treatment for parathion poisoning has been improved with the availability of 2-PAM (2-pyridine-aldoxime methiodide). Blood samples for cholinesterase determinations must be collected before 2-PAM treatment is started.

In severe cases it may be necessary to begin treatment with atropine

or 2-PAM before laboratory confirmation of significant cholinesterase depression is obtained.

TREATMENT

SPEED IS IMPERATIVE.

Principles of treatment include the immediate injection of atropine to block parasympathetic effects of the accumulated acetylocholine and of 2-PAM to reactivate the phosphorylated enzyme.

It is imperative to keep the airway open.

When the compound is ingested, it is important to act quickly to prevent any further systemic absorption.

When exposure is by skin contact, the compound should be rapidly removed by thorough rinsing or washing with water or soap and water. Compound splashed in the eye should be washed out with water, isotonic saline, or other ophthalmic irrigating solution, as available.

Wear rubber gloves while washing contact area to prevent any danger to medical personnel.

CARBAMATES

Carbamates are reversible cholinesterase inhibitors. Like organophosphates, they may be direct or delayed in action. Inhibition of the enzyme is reversed largely by hydrolysis of the carbamylated enzyme and to a lesser extent by synthesis of a new enzyme. Important carbamates are:

Baygon
Carbaryl (Sevin)
Thiram
Vapam
Zectran

PERMISSIBLE EXPOSURE LIMITS

The Federal standard for carbaryl is 5 mg/m^3.

HARMFUL EFFECTS

Signs and symptoms of intoxication may include miosis, salivation, profuse sweating, lassitude, muscle incoordination, nausea, vomiting, diarrhea, epigastric pain, tightness in the chest, etc.

CLINICAL NOTES

2-PAM and other oximes are contraindicated for routine use.

Cholinesterase reactivates rapidly after carbamate poisoning. Laboratory cholinesterase determinations may be misleading.

LABORATORY NOTES

1-Naphthol, normally found in traces, is excreted in the urine in much higher concentrations following carbaryl ingestion.

TREATMENT

SPEED IS IMPERATIVE

Principles of treatment are similar to those used in organophosphate poisoning (atropine and maintenance of adequate respiration) with the exception of the use of 2-PAM.

CHLORINATED HYDROCARBONS

Chlorinated hydrocarbon insecticides are more persistent in the environment than most other synthetic organic pesticides, and because of this, their use has recently decreased. Among the most important chlorinated hydrocarbons are the following:

Benzene Hexachloride (BHC)	Kepone
Chlordane	Heptachlor
DDT	Lindane (Isomer of BHC)
Dicofol (Kelthane)	Mirex
Dieldrin	Thiodan
Endrin	Toxaphene

HARMFUL EFFECTS

Chlorinated hydrocarbons are most efficiently absorbed by ingestion. In general, they act on the central nervous system to stimulate or depress. Signs and symptoms of toxicity, therefore, vary with the specific chemical. Symptoms have been reported as soon as 30 minutes after massive exposure, but generally develop more slowly; if this pattern of symptoms does not appear within a few hours after suspected acute exposure, another diagnosis or complicating feature must be sought.

Mild chlorinated hydrocarbon poisoning causes such symptoms as dizziness, nausea, abdominal pain, and vomiting. In chronic poisoning, loss of weight and appetite, and, in the case of endrin, temporary deafness and disorientation may occur.

Moderately severe chlorinated hydrocarbon poisoning presents mild signs followed by severe irritability, convulsive seizures, and coma. Seizures may be epileptiform in character with frothing at the mouth, facial congestion, violent convulsive movements or stiffness of the limbs, associated with stupor or coma. In severe cases, the convulsions may be continuous, with elevated body temperatures, unconsciousness, labored breathing with vigorous, rapid heart beat, and eventually death.

CLINICAL NOTES

Vomiting should NOT be induced when the ingested pesticide is in a hydrocarbon solvent. Epinephrine should not be given since chlorinated hydrocarbons may sensitize the heart to catecholamines.

LABORATORY NOTES

A high urinary level of organic chlorine or especially of p-chlorophenyl acetic acid indicates exposure to DDT or to one of the analogous compounds. The level, however, is not necessarily indicative of the severity of exposure.

TREATMENT

In cases of ingestion, gastric lavage should be performed.

Care should be taken to prevent aspiration of gastric contents. In some cases, induction of catharasis with aqueous solutions of sodium

sulfate has been of value in increasing fecal excretion and retarding absorption. Barbiturates are sometimes helpful in reducing convulsions. Respiration should be closely followed. Oil, oily cathartics (e.g., mineral oil), and epinephrine should be avoided.

BIPYRIDYLS

Bipyridyls include paraquat and diquat and are used in the form of the dichloride, dibromide, or dimethosulfate salt.

HARMFUL EFFECTS

Most reported cases involved accidental ingestion which produced proliferative changes in the lungs, cornea, lens, nasal mucosa, skin, and finger nails.

With the exception of eye lesion, illness due to occupational exposure is usually mild and is the result of skin contact.

CLINICAL NOTES

Diquat affects the lens and the gastrointestinal mucosa. It does not produce the lung changes characteristic of paraquat.

The clinical picture following accidental or suicidal ingestion of paraquat is very different. Paraquat ingestions are frequently fatal. Their management is unsatisfactory and largely symptomatic. Three clinical stages follow ingestion of as little as one ounce of paraquat.

The first is a gastrointestinal phase with burning in the mouth and throat, nausea, vomiting, abdominal pain, and diarrhea.

Several days after exposure, signs of hepatic and renal toxicity appear. These are due to central zone necrosis of the liver and acute tubular necrosis of the kidney.

Ten to 20 days after ingestion, progressive proliferative changes develop in the lungs. Hyperplastic changes in the terminal bronchioles occur with alveolar fibroblastic proliferation. Loss of lung surfactant has been demonstrated. Within a few days, death from respiratory failure occurs.

LABORATORY NOTES

Urinary studies have indicated that 90 percent of the ingested paraquat is excreted in the first 24 hours. Delayed pulmonary effects appear to be the result of an irreversible process that develops long after the initial stimulus has gone.

Paraquat is poorly absorbed from the gastrointestinal tract. Excretion data suggest that only 1 to 5 percent of the ingested material is absorbed in man. Maximal blood concentrations are reached within 4 to 6 hours after ingestion.

TREATMENT

Treatment is primarily directed toward decreasing the amount of paraquat absorbed and the concentration in the circulating blood. This may be achieved by appropriate repeated administration of large amounts of adsorbents and purgatives.

RODENTICIDES

Rodenticides of first importance include sodium fluoroacetate, strychnine, thallium sulfate, and warfarin. For information on rodenticides containing arsenic, barium, cyanide, and phosphorus, reference may be made to the appropriate chemical in the section on Chemical Hazards.

SPECIAL NOTES

Fluoroacetate is a highly toxic poison which causes central nervous system stimulation (convulsions) and cardiac arrhythmias. Specific treatment includes monacetin (monoacetin, glycerol monoacetate).

Strychnine poisoning is characterized by severe convulsion without loss of consciousness. Death is usually a result of asphyxia or involvement of vital brain centers. The compound may be identified in the urine soon after ingestion.

Thallium sulfate by ingestion or skin absorption may induce intoxication. Acute poisoning is characterized by severe gastroenteritis following a latent period of 12 to 24 hours. Other effects may include liver and kidney damage, encephalopathy, neuritis, ataxia, and alopecia. Recovery is slow. Thallium may be demonstrated in the urine.

Warfarin — See Coumarins.

Coumarins and *Indandiones* include Diphacin, Fumarin, Pival, (Pivalyn), PMP, Valone, and warfarin.

After repeated ingestion for several days, symptoms may include bleeding from the nose and gums, and into the conjunctiva, urine, and stool. Other possible symptoms are pallor, petechial rash, massive ecchymoses, hematoma of skin and joints, brain hemorrhage, etc. Shock and death may follow.

Laboratory determination of prothrombin time may be helpful in assessing the extent of exposure.

FUNGICIDES

The fungicides are a heterogeneous group of chemicals and, with the major exception of the dithiocarbamates, have been in use for many years. Many of the fungicides such as formaldehyde, furfural, phenol, tetramethylthiuram disulfide and compounds of boron, chromium, copper,

mercury, tin, and zinc (some of which are also used as herbicides and insecticides) are discussed in the section on Chemical Hazards.

The dithiocarbamates include ferbam (ferric dimethyldithiocarbamates), ziram (zinc dimethyldithiocarbamate), maneb (manganous ethylene bisdithiocarbamate), nabam (disodium ethylene bisdithiocarbamate), and zineb (zinc ethylene bis-dithiocarbamate). Their chief adverse effects are irritation of the skin, eyes, and upper respiratory tract.

HERBICIDES

Herbicides, or weed killers, may be classified as pesticide chemicals. They can kill plants on contact, or they can be translocated, that is, absorbed by one part of the plant and carried to other parts where they exert their primary toxic effect. Most of the commonly used herbicides (ammonium sulfamate, dalapon, phenoxyacetic acid derivatives (e.g., 2,4,5—T), carbamate derivatives, petroleum oils, sodium borate, Crag herbicide) have a low toxicity and have caused little difficulty among users.

Some herbicides pose more serious problems; for example, the methemoglobinemia and central nervous system depression produced by sodium chlorate. Pentachlorophenol, a metabolic stimulant, has been responsible for several deaths because of hyperthermia. Pentachlorophenol through skin absorption can also result in peripheral m otor neuropathies. Amino triazole has produced cancer in experimental animals, but there have been no untoward effects reported in man.

Herbicides with cutaneous effects include trichloroacetic acid, a corrosive irritant of the skin and mucous membranes; pentachlorophenol, a producer of a primary irritant type of contact dermatitis; and creosote, a primary irritant and photosensitizer.

Reference may be made to chemicals in the section on Chemical Hazards for the toxicity of the following herbicides: arsenic trioxide and sodium arsenate (see Arsenic), copper sulfate (see Copper and Compounds), creosote compounds, (see Cresol and Phenol), dinitrophenols (see Dinitrophenol), kerosene, and phenylmercuric acetate (see Mercury and Compounds).

FUMIGANTS

Fumigants are pesticides which may be applied in the solid, liquid, or gaseous state. A combination of high volatility with high pest toxicity is generally desired; however, compounds with low volatility may be preferred for soil fumigation. The possibility of excessive exposures exists wherever fumigants are used, as in fumigating grains, soils, clothes, furs, homes, warehouses, barns, ships, mills, freight cars, and greenhouses.

Each of the following compounds has found use as a fumigant.

Because they have other industrial applications as well, they are discussed individually in the section on Chemical Hazards.

Acrylonitrile
Carbon Disulfide
Carbon Tetrachloride
p-Dichlorobenzene (see Chlorinated Benzenes)
Dioxane
Ethylene Dibromide
Ethylene Dichloride
Ethylene Oxide
Hydrogen Cyanide
Methyl Bromide (see Bromine and Compounds)
Methylene Chloride
Methyl Formate
Naphthalene
Perchloroethylene
Propylene Dichloride
Sulfur Dioxide
Tetrachloroethane
Trichloroethylene

MISCELLANEOUS INSECTICIDES

Although the newer synthetic pesticides previously discussed in this section are becoming increasingly popular, the following compounds continue to find significant usage.

Lead Arsenate and Arsenite

These compounds enter the body by inhalation, ingestion, or percutaneous absorption. Signs and symptoms of poisoning are similar to those characteristic of lead or arsenic intoxication. Acute symptoms include nausea, vomiting, abdominal pain, diarrhea, muscle cramps, excitation, and disorientation. Chronic poisoning is manifested by anorexia, weakness, weight loss, pallor, colic, diarrhea, peripheral neuritis, hepatitis, and nephritis. A vesicular dermatitis has frequently been reported. The carcinogenic hazard from chronic arsenic exposure also cannot be ignored.

Nicotine

Nicotine is an extremely toxic alkaloid capable of producing nervous system stimulation followed by severe nervous system depression. The effects may result from ingestion, inhalation, or rapid percutaneous absorption of the material. Analysis for urinary nicotine may aid in the diagnosis.

Pyrethrum

Pyrethrum does not appear to be particularly toxic; however, pri-

mary contact dermatitis and allergic skin and pulmonary reactions have occurred following minimal exposure to the dust.

Rotenone

Rotenone is a plant extract which is more toxic than pyrethrum but, as normally used, is not excessively hazardous. Contact dermatitis and numbness of the oral mucous membranes may follow sufficient exposure.

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SECTION X

Various and manifold is the harvest of diseases reaped by certain workers from the crafts and trades that they pursue; all the profit that they get is fatal injury to their health. . . .mostly, from two causes. The first and most potent is the harmful character of the materials that they handle. . .the second cause I ascribe to certain violent and irregular motions and unnatural postures of the body, by reason of which the natural structure of the vital machine is so impaired that serious diseases gradually develop therefrom.

—Ramazzini

PHYSICAL HAZARDS

The properties, biological effects, and health hazards associated with physical agents are discussed in this chapter. A physical agent can be defined as an entity without substance (i.e., with minimal matter), yet capable of affecting the biological mechanisms of an exposed worker. The hazards considered are those associated with exposure to the following agents, which will be grouped into three classes for discussion.

RADIATION: ionizing radiation and nonionizing radiation, including ultraviolet, visible, infrared, microwave, radio frequency, and laser.

ATMOSPHERIC VARIATIONS: heat, cold, air pressure.

OSCILLATORY VIBRATIONS: noise, vibration.

Some of these agents are encountered only in specific occupational situations while others may be present in a number of working environments.

RADIATION

Eugene Moss, William Murray, Wordie Parr, Ph.D. and David Conover, Ph.D.

Radiation is energy which is emitted, transmitted, or absorbed in wave, or energetic particle, form. One can think of the wave as the disturbance that transfers energy progressively from one point to another point in a medium.

The electromagnetic (EM) waves consist of electric and magnetic forces. When these forces are disturbed, EM radiation results. The known EM radiations are grouped into a spectrum according to their frequency and/or wavelength (Table 7). The spectrum consists of a continuum of radiation ranging from below radio frequencies to above ionizing; it includes microwave, infrared, visible, and ultraviolet radiation. Although no range of radiation is sharply delineated from another and, in fact, the ranges often overlap, it is convenient to separate these ranges into the above groups because of the physical and biological effects associated with each radiation type.

The range of biological effects of exposure within the EM spectrum is extremely broad and diverse. This is further emphasized by noting the frequency bandwidth covered by the spectrum. Table 7 indicates the band width to encompass at least 10^7 but, in fact, it extends to a factor of 10^{22} Hz. The amount of energy absorbed by the worker varies considerably over this range. The safety, health, evaluation, and control problems associated with such a range of magnitudes are great. No other area in human research has a similar wide range of the hazards that need to be considered, classified, and regulated.

Table 7. Characteristics and sources of electromagnetic radiation.

Type of radiation	Frequency range (Hz)*	Wave length range**(m)	Energy per photon range (eV)†	Typical industrial sources of exposures	Estimated number of exposed workers
A. IONIZING X-ray γ-ray B-ray γ-particle Proton Neutron	$>3.0\times10^{15}$	$<1.0\times10^{-7}$	$>1.2\times10^{1}$	Electronic tubes, radiography, nuclear power plants, radiation curing, medical uses, uranium mining, sterilization processes.	7×10^{6}
B. NONIONIZING					
Ultraviolet	7.5×10^{14} to 3.0×10^{15}	1.0×10^{-7} to 4.0×10^{-7}	3.1 to 1.2×10^{1}	Lamps, welding arcs, gas discharge tubes.	86×10^{6}
Visible	4.0×10^{14} to 7.5×10^{14}	4.0 to 7.6×10^{-7}	1.6 to 3.1	Lamps, welding arcs, hot bodies.	96×10^{6}
Infrared	3.0×10^{11} to 4.0×10^{14}	7.6×10^{-7} to 1.0×10^{-3}	1.2×10^{-3} to 1.6	Lamps, welding arcs, hot bodies.	26×10^{6}
Microwaves	3.0×10^{8} to 3.0×10^{11}	1.0×10^{-3} to 1.0	1.2×10^{-6} to 1.2×10^{-3}	Klystron, magnetron.	14×10^{6}
Radio Frequencies	$<3.0\times10^{8}$	>1.0	$<1.2\times10^{-6}$	Plastic sealers, furniture glue.	7×10^{6}

*Hz = Hertz, cycle per second. **m = meter. †eV = electron volts. Note: The given ranges are only approximations.

IONIZING RADIATION

Ionizing radiation has always been a part of man's natural environment, and since the discovery of X-rays and radioactivity, it has become a part of the industrial environment of many workers. The different types of ionizing radiation vary in their penetrative powers as well as in the number of ions they produce in traversing matter. The latter is important in that biological effects vary with ion density (the number of ions produced per unit length of track).

Ionizing radiations are produced naturally by the decay of radioactive elements or artificially by such devices as X-ray machines and high energy accelerators. A radioactive nucleus is one that spontaneously changes to a lower energy state, emitting particles and often gamma rays in the process. The particles commonly emitted are alpha particles and beta particles. High energy accelerators can produce all of the above particles plus protons, neutrons, and X-rays. The following sections describe some of the more commonly encountered types of ionizing radiation.

Alpha particles, which interact readily with matter to produce ions, usually have energies of from 4 to 8 million electron volts (Mev). They travel a few centimeters in air and up to 60 microns into tissue. The high energy and short path result in a dense tract of ionization along the path of the particles, which produces serious biologic damage in the tissues with which the particles interact. Alpha particles will not penetrate the stratum corneum of the skin and thus are not an external hazard; but if alpha-emitting elements are taken into the body by inhalation or ingestion, serious internal exposure problems may result.

Beta particles interact much less readily with matter than do alpha particles and will travel up to a few centimeters into tissue or many meters in air. Exposure to external sources of beta particles is potentially hazardous, but exposure internally is more hazardous.

Protons with energies of a few Mev are produced by high-energy accelerators and are quite effective in producing tissue ionization. The path length of a proton is somewhat longer than the path of an alpha particle of equivalent energy.

Gamma rays and X-rays are electromagnetic radiations with similar properties. X-rays, in general, have longer wave lengths, lower frequencies, and, therefore, lower energies than gamma rays. Gamma rays are produced by nuclear processes, while X-rays may result from the electronic structure of the atoms or from the slowing down of high-speed electrons. X-rays and gamma rays are primarily an external hazard and their biologic effects are better known than those of any of the other ionizing radiations. Examples of gamma emitters used in industry are cobalt-60, cesium-137, and iridium-192. X-rays may also be encountered during the manufacture and use of electronic tubes and electron microscopes.

Neutrons have about the same mass as protons, but react much differently since they are electrically neutral. These uncharged nuclear

particles upon collision with matter may cause the release of all the above types of ionizing radiation. Neutrons can be produced up to several Mev by reactors, accelerators, or certain beryllium-enriched sources.

ROUTES OF ENTRY

The conditions presented by external radiation sources are entirely different from those presented by internal radiation sources which have been deposited in the body, with their attendant continuous irradiation of cells and tissue.

Entry of radiation sources into the body during occupational exposures is principally from breathing air containing particulate or gaseous radionuclides, although ingestion and skin absorption can be important.

Implantation under the skin may occur as the result of accidental skin puncture or laceration. Once inside the body, radionuclides are absorbed, metabolized, and distributed throughout the tissues and organs according to the chemical properties of the elements and compounds in which they exist. Their effects on organs or tissues depend on the type and energy of the radiation and residence time.

The effect from external radiation sources depends on the penetrating ability of the particular radiation. Thus, alpha radiation is of no concern externally, and beta is stopped in the outer tissues, the depth depending on energy. Very low energy X- or gamma radiation is attenuated quite rapidly.

HARMFUL EFFECTS

The early experience of radiation workers (including various nuclear accidents, exposure of radium dial-painters, casualties from atomic bomb explosions) and data from research projects provide clear evidence that high levels of ionizing radiation definitely create somatic damage and may induce genetic damage. The occupational somatic effects include radiodermatitis, epilation, acute radiation syndrome, cancer, leukemia, cataracts, sterility, and life span shortening. The genetic effects resulting from occupational exposures are to a great extent still unknown. Moreover, it is important to remember that a mutation produced by radiation is similar to one effected by a mutagenic chemical or to one occurring spontaneously.

In general, the sequence of events following radiation exposure may be classified into three major periods. The first period is the latent period, defined as the time lapse between the initial radiation event and the first detectable effect. Since the latent period can range from days to years, it is often divided into short-term (days or weeks) and long-term (months or years) effects. The second period, the period of demonstrable effects, occurs immediately after the latent period and is that time period when certain discrete biological effects can be observed. The final period is the recovery period.

The effects from occupational exposure to ionizing radiation are usually localized, leading to erythema or radiodermatitis. An acute radiation syndrome (ARS) episode occurs very rarely. An episode of this type involves whole body exposure exceeding 100 roentgens given in a

very short time.

Initial symptoms of ARS are nausea, vomiting, diarrhea, weakness, and shock. Following a latent period of 2 to 14 days, symptoms of fever and malaise occur. During this same period of time, hemorrhagic lesions of the skin often appear, and by the third week, epilation occurs. Painful ulceration, both internal and external, may appear over the whole body and bloody diarrhea may occur. Death may result from severe bone marrow depression if the radiation exposure level is high. The patient is also susceptible to infection of many types.

Among the long term effects are an increased incidence of carcinoma, as noted in the radium dial painters and uranium miners; the embryological effects, as noted in pregnant working women; the cataractogenic effects, as seen in certain radiologists and nuclear physicists; and shortening of the life span.

Overexposure of a worker to either an external or internal source of radiation creates a medical emergency. In serious situations involving a release of radioactive material, the degree of contamination must be determined before the individual can be admitted to a hospital. A much more detailed discussion of the medical aspects of radiation accidents is available in the volume by Saenger (1963) listed in the bibliography that follows.

POTENTIAL OCCUPATIONAL EXPOSURES

With the widespread use of radioactive isotopes in industry and the increasing use of X-ray sources, ionizing radiation exposures may occur in a wide variety of occupations. The following examples show the diversity of occupations potentially exposed to ionizing radiation.

Aircraft workers
Atomic energy plant workers
Biologists
Cathode ray tube makers
Ceramic workers
Chemists
Dental Assistants
Dentists
Dermatologists
Drug makers
Drug sterilizers
Electron microscope makers
Electron microscopists
Electrostatic eliminator operators
Embalmers
Fire alarm makers
Food preservers
Food sterilizers
Gas mantle makers
High voltage television repairmen
High voltage vacuum tube makers
High voltage vacuum tube users

Industrial fluoroscope operators
Industrial radiographers
Inspectors using, and workers in proximity to, sealed gamma ray sources (cesium-137, cobalt-60, and iridium-192)
Klystron tube operators
Liquid level gage operators
Luminous dial painters
Machinists, fabricated metal product
Military personnel
Nurses
Oil well loggers
Ore assayers
Pathologists
Petroleum refinery workers
Physicians
Physicists
Pipeline oil flow testers
Pipeline weld radiographers
Plasma torch operators
Plastic technicians
Prospectors
Radar tube makers
Radiologists
Radium laboratory workers
Radium refinery workers
Research workers
Television tube makers
Thickness gage operators
Thorium-aluminum alloy workers
Thorium-magnesium alloy workers
Thorium ore producers
Tile glazers
Uranium dye workers
Uranium mill workers
Uranium miners
Veterinarians
X-ray aides
X-ray diffraction apparatus operators
X-ray technicians
X-ray tube makers

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ULTRAVIOLET RADIATION

Ultraviolet (UV) radiation is an invisible radiant energy produced naturally by the sun and artificially by arcs operating at high temperatures. Artificial sources commonly found in industry are germicidal and black-light lamps, carbon arcs, welding and cutting torches, electric arc furnaces, and laboratory equipment.

Since the eyes and skin readily absorb UV radiation, they are particularly vulnerable to injury. The severity of radiation injury depends on factors which include exposure time, intensity of the radiation source, distance from the source, wavelength, sensitivity of the individual, and presence of sensitizing agents.

HARMFUL EFFECTS

Sunburn (erythema) is a common example of the effect of UV radiation on the skin. Repeated UV exposure of lightly pigmented individuals may result in actinic skin — a dry, brown, inelastic, wrinkled skin. There are telangiectases on the forehead, and neck movements produce lines on the nape in an angular pattern. Actinic skin is not harmful in itself, but it is a warning that conditions such as senile keratosis, squamous cell epithelioma, and basal cell epithelioma may develop.

Since UV radiation is not visible, the worker may not be aware of the danger at the time of exposure. Absorption of the radiation by the mucous membranes of the eye and eyelids can cause conjunctivitis (commonly known as "ground glass eyeball" or "welder's flash"). Lesions may also be formed on the cornea at high exposure levels (photokeratitis). Such injuries usually manifest themselves 6 to 12 hours after exposure. The injuries may be very painful and incapacitating, but impairment is usually temporary.

Photosensitizing agents have action spectra which are frequently in the ultraviolet range (Table 8). Some drugs and many plants, including figs, limes, parsnips, and pink-rot celery, carry photosensitizing chemicals believed to be furocoumarins and psoralens. Symptoms upon contact are those of an exaggerated sunburn with blisters frequently present. The most important industrial photosensitizer is coal tar which has an action spectrum in the visible range.

Ultraviolet light can also act in a nonspecific manner in the production of herpes simplex and chronic discoid lupus erythematosus.

PERSONAL PROTECTIVE METHODS

Protective measures are essential for workers exposed to high-intensity UV sources. Goggles, face shields, and masks provide protection for the eyes; protective clothing and barrier creams minimize skin exposure. Shiny metal surfaces reflect UV radiation and, when possible, should be removed from the work area. Reflections from lamp housings, walls, ceilings, and other surfaces should be reduced by coating these surfaces with a pigmented paint of low UV reflectance. Operations that produce high levels of UV should be placed behind enclosures to absorb the radiation and shield nearby workers from exposure.

POTENTIAL OCCUPATIONAL EXPOSURES

UV radiation exposure is generally present wherever occupations involve sunlamps, the outdoor sun, welding arcs, plasma torches, lasers, laboratory research, printing processes, drying and curing processes, nondestructive testing, environmental test chambers, medical devices and materials, and chemical processing and manufacturing. Occupations associated with potential UV radiation exposure include the following:

Agricultural workers
Bacteriologists
Bath attendants
Beauty salon workers
Brick masons
Cattlemen
Chemists
Construction workers
Dentists
Farmers
Fishermen
Food irradiators
Gardeners
Graphic illustrators
Greenskeepers
Horticultural workers
Laboratory workers
Lamp testers
Landscapers
Lifeguards
Lithographers
Lumberjacks
Maintenance workers
Meat curers
Metal casting inspectors
Microscopists
Military personnel
Movie projectionists
Nurses
Oilfield workers
Open pit-miners
Opticians
Optometrists
Outdoor maintenance workers
Paint and color testers
Paint curers
Physicians
Physicists
Physiological optics workers
Photo-bacteriologists
Phototherapy technicians
Pipeline workers

Table 8. Some examples of action spectra of normal and abnormal reactions in man.*

Condition	Wavelength Range (nm)	Maximum Reaction (nm)
Normal sunburn	290-320	297-307
Artificial light sources	250-320	250
Melanin pigmentation	290-320 320-480	290-310
Vitamin D production	290-310	290
Hyperbilirubinemia of prematurity	Blue visible spectrum	440-470
UV carcinogenesis	290-320	290-310
Solar urticaria	290-320 320-400 400-600	varying
Porphyria photosensitivity	380-600	400-410
Xeroderma pigmentosum	290-340	293-307
Polymorphic photodermatitis	290-320 320-400	290-320
Lupus erythematosus (LE) and discoid LE	290-320	
Solar (actinic) degeneration	290-400	?
Photoallergic reactions to halogenated salicylanilides and other related compounds	320-380	330-360
Phototoxic reactions to drugs	320-400 290-320	320-400
Psoralens (8-methoxy- and trimethylpsoralen)	320-380	330-360

*Used with permission from Sunlight and Man. T. B. Fitzpatrick et al., eds. Table 4. University of Tokyo Press, Tokyo, Japan.

Plasma torch operators
Plastic curers
Policemen (including crossing guards)
Postmen
Printers
Production workers in chemical processing
Railroad track workers
Road workers
Seamen
Ski instructors
Space simulator workers
Sportsmen
Surveyors
Textile inspectors
Tissue culture workers
Tobacco irradiators
Vitamin D synthesis workers
Welders
Welder foremen
Wood curers

VISIBLE RADIATION

Visible radiation, or light, from either the sun or artificial sources, is probably one of the more important occupational health considerations because of its major role in our daily life. Only within the last few years have investigators begun to discover various subtle physiological and biochemical responses to light.

Several human systems respond directly or indirectly to visible radiation. A direct effect has been defined as a chemical change in the composition of a tissue resulting from the absorption of light energy within the tissue. Because few direct effects of light have been documented, light is not considered a major occupational health hazard.

Indirect effects of light, however, can occur — not from absorption of light energy in tissues — but from the action of chemical signals liberated by cells in the body. Examples of this relationship of light to biological rhythms, include physical activity, sleep, food consumption, etc. Another well-known indirect effect is the inhibition of melatonin synthesis by the pineal gland which in turn affects maturation and activity of the sex gland. The various known classifications of abnormal biological reactions to light are shown in Table 9.

HARMFUL EFFECTS

One of the controversial issues associated with visible radiation is the effect of illumination on job performance. The concept exists that the levels of illumination and source luminance routinely encountered in interior working environments may constitute some type of an ocular health hazard. Studies on humans gazing at the sun and on rats and mice under common interior lighting intensities have produced evidence to indicate a hazard; these studies, however, have not been considered conclusive and the results have been disputed.

At the 1974 NIOSH symposium on illumination, several conclusions were agreed upon:

1) High levels of lighting can cause damage to the eye, i.e., retinal or macular degeneration.
2) Poor lighting conditions can cause aesthenopia.

3) With the possible exception of miner's nystagmus, such organic diseases as glaucoma, cataracts, and retinal degeneration do not result from exposure to low levels of illumination.

The consensus seemed to be that if there is sufficient illumination to perform a task reasonably well, then there is sufficient light to meet the safety criteria.

Although the etiology of asthenopia (eye strain) is debatable, it appears that repeated occurrences probably do not lead to any permanent eye damage. Workers over 40 years of age will probably encounter more symptoms of asthenopia (headache, tired eyes, irritation) since they require more light to perform a similar job than younger workers.

POTENTIAL OCCUPATIONAL EXPOSURES

Virtually all occupations offer exposure to the potential hazards of defective illumination. Some occupations, however, require unusually close, fine work and attention to detail for many hours a day. Some of these occupations are:

- Draftsmen
- Electronic equipment assemblers
- Engravers
- Jewelers
- Quality control inspectors
- Watchmakers

INFRARED RADIATION

All objects having temperatures above absolute zero emit infrared radiation (IR) as a function of temperature. In biological systems the major insult of IR occurs as a result of a rise in temperature of the absorbing tissue.

The physical factors associated with temperature rise are the wavelength, heat conduction parameters, exposure time, and total amount of energy delivered to the exposed tissue. Since IR photons are low in energy, they probably do not enter into photochemical reactions with biological systems. Molecular interaction with radiation in the IR regions are characterized by various vibrational-rotational transitions resulting in an increase in thermal energy of the molecule.

HARMFUL EFFECTS

Since the primary effect of IR on biological tissues is thermal, the skin provides its own warning mechanism by having a pain threshold below that of the burn threshold.

In the eye, however, there is no adequate warning mechanism to protect against lenticular damage. Cataracts may be produced by prolonged exposure to wavelengths at energy levels that do not normally burn the skin. The classic ocular effect observed after many years of IR exposure is a posterior cataract, sometimes called glassblower's or furnaceman's cataract. This type of cataract has a lengthy latent period (10 to 15 years) in individuals chronically exposed to IR which has made it difficult to determine threshold values.

The present etiology of IR-induced cataracts is thought to be directly correlated with the amount of energy initially absorbed by the iris and then transferred to the lens. The threat of cataract formation is primarily from wavelengths below 1400 nanometers. Longer wavelengths may produce corneal damage, the difference being due to the site of energy absorption.

The primary biological effect of IR on the retina and choroid is thermal in nature, with the amount of damage being proportionate to the length of exposure. If the radiation intensity is low enough, however, the normal retinal blood flow may be sufficient to dissipate any heat generated. Nevertheless, due to the focusing effect of the anterior ocular components, small amounts of IR radiation can produce a relatively intense point energy distribution on the retina resulting in a lesion. The effects of IR on the lid and cornea can be considered as ordinary cutaneous burns.

POTENTIAL OCCUPATIONAL EXPOSURES

A wide range of IR wavelengths representing large variations in temperature is encountered in many industries from direct (lamps) and indirect (heat) sources. Occupations potentially associated with infrared radiation exposures include the following:

- Bakers
- Blacksmiths
- Braziers
- Chemists
- Cloth inspectors
- Cooks
- Dryers, lacquer
- Electricians
- Firemen, stationary
- Foundry workers
- Furnace workers
- Gas mantle hardeners
- Glass blowers
- Glass furnace workers
- Heat treaters
- Iron workers
- Kiln operators
- Laser operators
- Motion picture machine operators
- Plasma torch operators
- Skimmers, glass
- Solderers
- Steel mill workers
- Stokers
- Welders

MICROWAVE/RADIOFREQUENCY RADIATION

Various estimates have been made of the number of workers potentially exposed to microwave/radio frequency (RF) radiation sources in industry, including one estimate of approximately 21 million workers at risk (See Reference 110). Obviously, not every worker in every job is exposed to hazardous microwave/RF radiation levels, but an exposure of even 25 percent of the workers potentially at risk would still present a significant target population. Because of the size of this potentially exposed group at work, the continuing expansion of microwave use in industry, and the lack of widely known bioeffects data, a list of references is offered in citations to the material presented. Also the following

Table 9. Classification of abnormal reactions to light in man.*

Type	Light Alone	Light + Exogenous Agent	Light + Metabolite	Light + Abnormal Skin or Disease
Genetic	Ephelides (freckles)		Porphyrias: Erythropoietic porphyria; Erythropoietic protoporphyria; Variegate porphyria (mixed porphyria); Hereditary coproporphyria	Xeroderma pigmentosum; Hartnup syndrome; Oculocutaneous albinism; Cockayne's syndrome; Rothmund-Thomson syndrome; Hailey-Hailey disease; Vitiligo; Darier-White disease; Bloom's syndrome; Phenylketonuria(?)
Chemical, phototoxic		Topical and oral drugs; Phytophotodermatitis		
Chemical, photoallergic		Topical and oral drugs		
Chemical induction of disease		Lupus erythematosus from sulfonamides, hydralazine, reserpine, griseofulvin, oral contraceptives, etc.	Porphyria from hexachlorobenzene, estrogens, griseofulvin, stilbesterol, alcohol	
Nutritional				Kwashiorkor; Pellagra
Infectious				Lymphogranuloma venereum; Herpes simplex

Table 9. Classification of abnormal reactions to light in man.* (Continued)

Type	Light Alone	Light + Exogenous Agent	Light + Metabolite	Light + Abnormal Skin or Disease
Degenerative and neoplastic	Chronic solar skin damage Solar keratoses Basal cell carcinoma Squamous cell carcinoma Malignant melanoma(?) Connective tissue degeneration (wrinkling) Telangiectasia			
Miscellaneous	Acute solar skin damage (sunburn) Hydroa aestivale and hydroa vacciniforme Disseminated superficial actinic porokeratosis	Phytophotodermatitis	Porphyria cutanea tarda	Lupus erythematosus (cutaneous and systemic) Solar urticaria Polymorphous light eruption Malignant carcinoid Pemphigus foliaceus Dermatomyositis

*Used with permission from Sunlight and Man. T. B. Fitzpatrick et al., eds. Table 1. University of Tokyo Press, Tokyo, Japan.

units of measurement are defined: μWcm^{-2} = microwatts per centimeter squared; MHz = 10^6 Hz; GHz = 10^9 Hz; $V \cdot m^{-1}$ = volts per meter; and $A \cdot m^{-1}$ = amperes per meter.

HARMFUL EFFECTS

Effects from exposure to microwave/RF radiation due to heating have been well documented, but evidence for those occurring in the absence of a tissue temperature rise is incomplete and in dispute (1-3).

Frey (4) reported an acoustic response which he postulated to be a direct auditory nerve response to microwaves. The phenomenon occurred instantaneously in human subjects exposed to power densities as low as 100 $\mu W \cdot cm^{-2}$. This specific effect was perceived as a buzz, ticking, or knocking, depending upon the pulse width and pulse repetition rate. The greatest sensitivity was observed in humans within the frequency range 300 MHz to 1200 MHz. The area directly over the temporal lobe of the brain was identified as the most sensitive area.

More recently Lebovitz (5) proposed that the cochlear hair cell structures within the vestibulo-cochlear complex of the human ear could directly respond to stimulation from pulse modulated microwave radiation. Guy (6) confirmed the reports of human auditory perception of pulse modulated microwave radiation and demonstrated a similar auditory response in cats which disappeared when the fluid was removed from the vestibulo-cochlear complex. Sommer and Von Gierke (7) confirmed the existence of this specific effect but stated that the effect was due to electromechanical transduction rather than direct stimulation of neural fibers or cortical neural tissue.

The possibility that microwave radiation could interact with the central nervous system (CNS) without detectable heating has been suggested by several investigators (8-18). Osipov (12) feels that some of the effects which have not been attributed to heating may in fact be due to microthermal heating. Microthermal heating is very localized and the temperature rise could not be detected by conventional temperature measurement techniques. Contrary to the subjective clinical complaints reported previously, recent studies [Baranski (8)] have used more objective measurements, better controls, improved statistics, a clearer description of the experimental design, and well documented experimental procedures.

In the Soviet and Eastern European literature (19-32), the following symptoms were reported as associated with 10/20-year exposure to microwave/RF radiation: headache, increased suspectibility to fatigue, diminished intellectual capabilities, dullness, partial loss of memory, decreased sexual ability, irritability, sleepiness and insomnia, and emotional instability. Objective disorders include sweating, hypotension, dyspnea, pains in the chest, sinus arrhythmias, bradycardia, and other cardiovascular problems. Electroencephalogram (EEG) recordings and the associated response times have been shown to be altered by microwave/RF exposure. Responses are characterized by initial excitation followed by inhibition. Also noted were threshold shifts generally in the direction of increasing thresholds for sensory perception, increases in the

latent period of the condition-reflex reaction, and disruption of vegetative system regulatory and compensatory functions.

Other changes that have been observed are: in the histamine level in the blood (33-36), decreased cholinesterase levels in persons and animals exposed to microwave/RF radiation (37-43); alterations in the protein fractions, ions, histamine content, hormone and enzyme levels, and immunity factors (27,33-37,44-52); leukocytosis (monocytosis, lymphocytosis, and eosinophilia); recticulocytosis; and thrombocytopenia.

Of particular significance are studies of genetic and reproductive system effects because of the possible impact in large populations over long periods of time. One study suggested a possible correlation between paternal radar exposure and mongoloidism of the progeny (53). Several occupational studies have suggested possible disturbances in human reproductive system functions (27,33,54). Animal studies with low intensity exposures report reproductive system disturbances and cases of detrimental effects on the progeny (55-59).

Changes in menstrual patterns, retarded fetal development, congenital effects in newborn babies, decreased lactation in nursing mothers, and an increased incidence of miscarriages for women working with microwaves have been reported [Marha et al. (60,61]. As a result, Czechoslovakian employment practices (60) prohibit women of reproductive capacity from working with RF radiation sources. Teratogenic effects on the fetus of a mother treated with RF radiation at the beginning of pregnancy (62) and impaired embryogenesis in humans and animals, particularly when RF irradiation occurred during the initial stages of pregnancy (63) have been reported. Rubin (64) reported on a case of microwave exposure of the human female pelvis during early pregnancy and prior to conception. The patient was irradiated during the first 59 days of pregnancy and aborted on day 67. The author warned against treating pregnant women with microwave radiation unless a careful menstrual history is taken (64).

Microwave/RF teratogenic effects have been produced in mealworm beetle pupae (65, 66), chick embryos (67,68), rats (69), and mice (70). The teratogenic effects noted in rats (69) were produced following irradiation at the same frequency (27.12 MHz) where the majority of RF power sources operate. A single acute RF exposure for ten minutes at 27.12·MHz between the first and the 16th day of pregnancy was sufficient to produce teratogenic effects in rats (69). The head, palate, limbs, tail, and abdomen were malformed following RF irradiation.

Primigravid mice irradiated at 2450 MHz with an accompanying injection of cortisone on days 11, 12, 13, and 14 of gestation gave birth to greater numbers of stillborn and deformed fetuses (71). Microwave irradiation of white CF1 mice at 2450 MHz with a mean absorbed dose rate of 107 mW/gm for four minutes produced teratogenic effects on gestation days 3, 4, 8, 10, and 12 (72).

One of the more important bioeffects related to temperature rises induced by microwave radiation in biological media is cataract formation (73-81). Kramer et al. (82) concluded that a temperature of at least

41 °C within the rabbit eye behind the lens is needed for cataractogenesis.

Weiter et al. (83) reported that the ascorbic acid content of rabbit lenses is a good index of microwave induced cataracts. The ascorbic acid content of rabbit lenses decreased with increasing microwave irradiation.

Behavioral effects observed after exposure to microwave/RF radiation indicate that the modifications of normal behavior are related to the increased body thermal burden. Thomas et al. (84) observed that a rat's ability to perceive a constant time interval was degraded by irradiation for times sufficient to cause whole body heating. Hunt et al. (85) found that irradiation of rats at 2.45 GHz caused a decrease in their ability to swim a water alley and that errors of omission in performing visual discrimination tests increased immediately following microwave irradiation. Soviet investigators, however, have concluded that behavioral effects are not primarily related to whole body heating (86).

A summary of available information on biologic effects and health hazards was presented in 1974 at an international symposium held in Warsaw, Poland (87).

Radio Frequencies Below 300 MHz

Previously, it was thought that there were no biological effects for frequencies below 300 MHz (0.3 GHz). In 1977, NIOSH, the United States Information Agency (USIA), and the United States Air Force (USAF) funded bioeffects research within the radiofrequency (RF) band which extends from 10 to 300 MHz in order to evaluate the accuracy of the 1977 standards for this frequency band. Both the ANSI C95.1 and the OSHA regulations (88) specify a frequency range from 10 MHz to 100 GHz.

Kall (89,90) studied the effects on rats exposed to RF (6 and 21 MHz) fields, similar to those found in the industrial environment. He noted that the gastrointestinal motor activity increased and cholinesterase activity decreased significantly. From the results of these studies on rats, Kall (90) recommended to the USIA that exposure standards should be modified to specify an electric field strength of 1500 $V \cdot m^{-1}$ and a magnetic field strength of 5 $A \cdot m^{-1}$ for the frequency range from 3 to 30 MHz.

The theoretical data of Lin (91) for a spherical phantom model simulating man has shown that the *magnetic field component* of the RF(1-20MHz) field may be the *most hazardous*. For RF fields close to typical RF power sources, the heating of the theoretical human phantom model due to the magnetic field component can be even larger. These conditions should be carefully considered when attempting to estimate the potential hazard from an RF radiation field.

Bawin (92,93) demonstrated EEG changes in cats and increases in calcium efflux in chicks following exposure to amplitude modulated 147 MHz fields. The amplitude modulation frequency (8-16 MHz) approached that of physiological bioelectric function rhythms. Further examples of alterations in circadian rhythms by RF exposure are effects

on the cell division rate of corneal epithelium (94,95) and changes in rate of bone marrow cell mitoses (96).

Prince et al. (97) demonstrated that RF fields (10-27 MHz) can produce a marked increase in lymphocyte mitotic activity 71 hours post-exposure in monkeys. Lovely et al. (98) reported no in vitro lymphocyte mitotic activity 48 hours following RF exposure of monkeys.

Stavinoha et al. (99) reported that near-field exposures of mice to 19 MHz caused definitive changes in lymphocytes and other white blood cells. The number of polymorphonuclear leukocytes was significantly higher and the number of lymphocytes was significantly lower in the irradiated group as compared with that of the control group. These alterations in white blood cells could not be duplicated in animals exposed in a hot air oven which elevated the colonic temperature the same amount as the RF field exposures. Czerski (96) showed alterations in the diurnal rhythm of peripheral white blood cells and granulocyte precursor mitoses in Guinea pig bone marrow. The results suggest a cumulative effect on the lymphatic system associated with decreased antibody production. This would imply that employees exposed to RF radiation might be more susceptible to infection and more susceptible to allergies encountered in the work environment.

The immunosuppressive and anti-lymphocytic actions of the adrenal glucocorticoids are well known. It is possible that immunologic effects caused by microwave/RF fields are related to glucocorticoid levels. Guillet et al. (100) reported the concentration of adrenal glucocorticoids in the blood increased following microwave irradiation.

Bollinger et al. (101) demonstrated that the uptake of H-3 Thymidine by lymphocytes isolated from mice exposed to RF fields ranged from 2.5 to 5 times that of control animals. Frazer (102) reported that the proportion of rat lymphocytes fell to 48.5% following RF irradiation. The total segmenters increased from 27.8 to 46.8%. It is not known whether these results indicate a direct effect on the cell population or an indirect thermal interaction followed by a rapid endocrine response. Froehlich (103) has shown that these effects could occur at the molecular level by either mechanism. Cody et al. (104) demonstrated alterations in the molecular spectra of biologically significant compounds, such as RNA, when exposed to RF radiation.

It is important to consider data which show that the distribution and magnitude of the absorbed power, *hence the expected biological effects,* varied greatly with exposure conditions. Bussey (105) found that the field intensity and distribution are markedly different in rats and primates. Guy et al. (106) measured field distributions in realistic models of man. The distribution of absorbed power in models of man was highly non-uniform with maximum power absorption (and associated localized temperature rises) occurring in the following locations: axilla, gonads, perineum, lateral thorax, ankles, sternum, forearms, tibia and fiibula, knees, neck, clavicle, shoulders and back. Persons industrially exposed to RF have complained of sensations of warming in many of the above mentioned locations.

Research (107-108) has indicated that the maximum amount of microwave/RF radiation is absorbed within the RF frequency range of approximately 25-26 MHz. Thus, consideration of the results of hyperthermia in the anatomical locations specified by Guy (106) should be of great interest.

For microwave/RF bioeffects dependent on power absorption, human RF exposures from approximately 25-26 MHz represent the greatest potential hazard. Industrial RF exposure measurements performed by NIOSH (109,110) show that the vast majority of industrial RF sources operate from 10-40 MHz and that more than 70% of the sources surveyed exceeded applicable personnel RF exposure standards.

POTENTIAL OCCUPATIONAL EXPOSURES

The following is a list of occupations with specific activities and/or products in which microwave/RF radiation is present and may be a potential hazard.

- Automotive workers
  - Drying of trim base panels
  - Embossing of heel pads to carpets
  - Heat-sealing body interior trim panels
  - Heat-sealing upholstery covers for seats and backs
  - Heat-sealing convertible tops
- Food products workers
  - Finish-drying of "polishing" baked goods
  - Inhibiting enzyme action
  - Melting chocolate prior to tempering
  - Thawing frozen baked goods
- Furniture and wood workers
  - Decking assembly
  - Door lamination
  - Fiberboard fabrication
  - Laminated beams
  - Lumber edge gluing
  - Plywood panel patching
  - Plywood or particleboard scarf gluing
  - Posts
  - Rafters
  - Ski lamination
  - Veneer panel gluing
- Glass fiber workers
  - Drying glass fibers on forming tubes
  - Drying roving packages
  - Drying and curing sizing on machine packages
  - Drying coatings on continuously moving strands
- Paper product workers
  - Correcting moisture profile on continuously moving webs
  - Drying twisted twine packages
  - Drying resin coatings

Paper product workers (cont.)
- Gluing paper
- Heating coating on continuous webs

Plastic heat-sealing workers
- Acetate box covers
- Advertising novelties
- Appliance covers
- Aprons
- Baby Pants
- Beach Balls
- Belts and suspenders
- Blister packages
- Book covers
- Capes
- Check book covers
- Charge cards
- Convertible tops
- Cushions
- Diaper bags
- Display boxes
- Electric blankets
- Food packages
- Fountain pens
- Garment bags
- Gas masks
- Goggles (industrial)
- Hand bags
- Hat covers
- Index cards
- Lampshades
- Liquid containers
- Luggage
- Machine covers
- Mattress covers
- Milk cartons
- Oxygen tents
- Packages
- Pharmaceuticals
- Pillow cases
- Pillow packages
- Plastic gloves
- Pool liners
- Protective clothing
- Rain apparel
- Racquet bags
- Refrigerator bags
- Shoes
- Shoe bags
- Shower curtains

Slip covers
Splatter mats
Sponge backings
Sport equipment
Tobacco pouches
Toys
Travel cases
Umbrellas
Wallets
Waterproof containers
Wire terminal covers

RF/microwave application workers
Advertising — RF excited gas display signs
Ceramics — dry ceramic bodies
Chemical — chemical activation
Electronics — tube aging and testing
Laser — RF excited gas lasers
Medical-diathermy
Scientific equipment — low temperature ashing of samples
Tobacco — dry blended tobacco and dry cigars
Welding — RF stabilized welders

Rubber products workers
Drying latex foams
Gelling latex foams
Preheating prior to molding
Preheating prior to curing latex foams

Textile workers
Drying rayon cake packages
Drying wound packages
Drying impregnated or coated yarns
Drying slasher coatings
Drying continuous webs

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LASER

The word laser is an acronym for "light amplification by stimulated emission of radiation." Lasers operate in the infrared and ultraviolet as well as visible regions. The name is descriptive of the physical principle involved in the production of the radiation. By stimulating or forcing atoms to emit photons, the resultant laser beam is coherent. This means that the photons in the beam are monochromatic and are in phase with each other. Since the laser beam is highly coherent it diverges slowly, maintains its divergency over a long distance, and has a high radiant exposure. As a result of these properties, laser light differs considerably from visible light.

At one time shortly after the development of the first working model in 1960, the laser was referred to as a solution looking for a problem. This situation has changed. By 1975 the development and marketing of new laser devices had resulted in the availability of almost 2500 models from 175 manufacturers and distributors. The most common lasers are shown in Table 10.

HARMFUL EFFECTS

The laser by virtue of its design is able to concentrate a large amount of energy in a small cross-sectional area. Consequently, individuals working with such devices encounter a potential hazard. The critical organs are the eye and skin and the resulting biological effects are similar to those produced by conventional optical radiation sources. Since energy must be absorbed to produce an effect, the degree of injury depends upon the wavelength of the laser and the capacity of the tissue to absorb energy of that particular wavelength.

The primary hazard from laser exposure is ocular damage. Overexposure of skin varies from a mild erythema to blisters and charring. Chronic or repeated exposures to laser radiation may have a long-term effect, but this is normally discounted.

Infrared radiation (700-1400 nm) has also been implicated as a cataractogenic agent. Overexposure to UV laser radiation can result in keratoconjunctivitis. Cataracts can be induced by UV lasers operating in the 300-400 nm range. Infrared lasers (1400 nm to 1 mm)present a corneal hazard only.

PERSONAL PROTECTIVE METHODS

If the radiation levels are kept below those injurious to the eye, other tissues will not be harmed. The thresholds for skin and ocular damage are the same for both UV radiation and for IR (1400 nm – 1mm)ranges. It is assumed that both organs are equally sensitive. The visible and near IR wavelengths (400-1400 nm) are readily transmitted through the ocular media to the retina. Not only is this radiation transmitted, it is focused by the lens onto the fovea centralis which concentrates the energy onto a very small spot.

Thus, in this spectral region, the eye (retina) is far more sensitive

Table 10. Common laser devices and applications.

Type	Wavelength(s)	Applications
Argon (Ar)	458-515 nm	alignment surveying instrumentation halography photocoagulation
Carbon dioxide (CO_2)	10.6 μm	material processing optical radar instrumentation surgery techniques
Dye	variable	instrumentation
Gallium arsenide (GaAs)	850-950 nm	instrumentation ranging intrusion detection communications
Helium cadmium (HeCd)	325, 442 nm	alignment surveying
Helium neon (HeNe)	632.8 nm	alignment surveying halography ranging intrusion detection communications
Neodymium glass (Nd glass) Neodymium YAG (Nd YAG)	106 μm	material processing instrumentation optical radar surgery
Ruby	694.3 nm	material processing holography photocoagulation ranging

than the skin by several orders of magnitude, and even a diffuse reflection from a high power laser can present an ocular hazard. An action spectrum has been recently developed to account for the variation in retinal sensitivity with wavelength for exposure times greater than 10 seconds. The minimum threshold dose for retinal lesions occurs at 440 nm and is thought to be due to a photochemical process rather than to a thermal mechanism as in wavelengths greater than 500 nm.

POTENTIAL OCCUPATIONAL EXPOSURES

Lasers are used in many diverse situations such as drilling holes in metals and baby bottle nipples, cutting diamonds, aligning wings on airplanes, repairing detached retinas, and taking three-dimensional pictures.

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ATMOSPHERIC VARIATIONS

Francis N. Dukes-Dobos, M.D.,
and Donald W. Badger, Ph.D.

HEAT

Four factors influence the interchange of heat between man and his environment. These are 1) air temperature, 2) air velocity, 3) moisture content of the air, and 4) radiant temperature. The industrial heat problem is one in which a combination of these factors produces a working environment which may be uncomfortable or even hazardous because of imbalance of metabolic heat production and heat loss.

The fundamental thermodynamic processes involved in heat exchange between the body and its environment may be described by the basic equation of heat balance:

$$\Delta S = M - E \pm R \pm C \qquad (1)$$

where M = rate of metabolism; ΔS = change in body heat content; E = heat loss through evaporation; R = heat loss or gain by radiation; and C = heat loss or gain through convection. Under conditions of thermal equilibrium, this becomes:

$$M = E \pm R \pm C \qquad (2)$$

Under these conditions, since equilibrium means no change in body heat content, the heat generated within the body by metabolism is completely dissipated to the environment and $\Delta S = 0$.

For purposes of temperature determination, the body can be divided into two regions, the deep region or the core, and the superficial region which is made up largely of the skin and subcutaneous tissues. The thermo-regulatory mechanisms of the body are directed at maintaining a uniform core temperature (about 37.0±0.5°C/98.6±1.0°F) while the temperature of the superficial tissues may vary within a relatively wide range according to the amount of heat received from or lost to the environment. The mean weighted skin temperature may vary within the range of 29 to 36°C/84 to 97°F.

TRANSFER MECHANISMS

When heat loss fails to keep pace with heat gain, the core temperature begins to rise. At this point certain physiologic mechanisms come into play in an attempt to increase heat loss from the body. First, there is dilation of the blood vessels of the skin and subcutaneous tissues with diversion of a large part of cardiac output to these superficial regions. There is a concomitant increase in circulating blood volume brought

about by contraction of the spleen and by dilution of the circulating blood with fluids drawn from other tissues. Cardiac output is also increased. All these circulatory adjustments enhance heat transport from the body core to the surface. Concomitantly the sweat glands become active, spreading fluid over the skin which removes the heat from the skin surface by evaporation. Under these conditions the equation (2) may be modified:

$$E = M \pm R \pm C \qquad (3)$$

to indicate that evaporative cooling must balance metabolic plus environmental heat load to maintain thermal equilibrium. If this fails, heat storage begins with the strain of increased body temperature occurring. Unchecked, this can lead to heat stroke which is often fatal, and is always more or less debilitating.

In general, industrial heat exposures may be classified as either hot-dry or as warm-moist. In the former the moisture content of the air is not excessive, so evaporative cooling is not impeded. The difficulties in hot-dry situations arise when the body absorbs more heat by radiation or convection or both than the cooling power of the sweat which man can produce and evaporate, that is: $M+R+C>E$. The human sweat producing capacity may be as high as 2 liters per hour, but over an 8-hour period a sweat rate of 1 liter per hour is considered to be the maximum which a healthy acclimatized worker can maintain day by day. Warm-moist environments may occur during the summer in areas where the outdoor air has a high moisture content or in plants where large amounts of moisture are released from the industrial processes involved, while air and radiant temperatures may be moderate. Here, the heat load from radiation and convection may not be great, but the high humidity inhibits heat loss from the body through evaporation of sweat and the same imbalance may occur.

It is apparent from the foregoing that an ordinary room thermometer (which corresponds to a dry bulb thermometer in scientific terminology) will not describe the total heat load imposed upon the worker by his job because it reacts only to the air temperature and thus informs us only about convective heat change. The globe thermometer is most widely used for assessing the radiant heat load; the wet bulb thermometer, for assessing the humidity of the air; and the anemometer, for wind velocity measurement. The metabolic heat generated within the body can be assessed by using energy requirement tables published in the literature. For more accurate determination of metabolism the oxygen consumption has to be measured.

If all these measurements are performed, it is possible to calculate the required evaporation (E_{req}) for maintaining heat equilibrium in a given work environment. The nomograms of the Belding-Hatch heat stress index (HSI) make this calculation relatively simple. Furthermore, the HSI permits the estimation of the maximum evaporative capacity of the ambient air (E_{max}). It is the ratio $\frac{E_{req}}{E_{max}} \times 100$ which gives us the HSI

value, indicative of the stressfulness of a hot job. There are other simpler heat stress indices, however, which can be used for the purpose of describing the environmental heat load. The Corrected Effective Temperature (CET) can be assessed by the use of a single nomogram and it combines the values of air temperature, humidity, and wind velocity into one number which is related to the human comfort feeling. The Wet Bulb Globe Temperature index (WBGT) is a simplified version of CET. Instead of a nomogram an equation can be used:

$$WBGT = 0.7\ NWB + 0.3\ GT \quad \text{(for indoors)} \quad (4)$$

$$WBGT = 0.7\ NWB + 0.2\ GT + 0.1\ DB \quad \text{(for outdoors)} \quad (5)$$

where NWB = natural wet bulb temperature
GT = globe temperature and
DB = dry bulb temperature.

Thus for estimating the WBGT index there is no need for wind velocity measurements which further simplifies this methodology.

ACCLIMATIZATION

Acclimatization is essential if man is to work in hot environments. This process of adaptation is characterized by the worker's ability to perform with less increase in core temperature and heart rate and less salt loss, due to a lower concentration of sodium chloride in the sweat. The greatest portion of adaptive changes in acclimatization to heat occurs within the first week. Nonadaptable individuals often abandon hot jobs within that time span. Acclimatization to heat can, however, be lost almost as rapidly as it is acquired.

The human sense of thirst is not an adequate regulator of fluid replacement during heat exposure. If workers are sweating profusely and do not replace their fluid and salt loss systematically, most of them will end up each work day in a dehydrated state. The amount of water loss considered to be still compatible with good health and high degree of fitness, provided that the water content of the body is restored by the start of the next work day, is 1.5% of the total body weight.

HARMFUL EFFECTS

Prolonged exposure to excessive heat may cause increased irritability, lassitude, decrease in morale, increased anxiety, and inability to concentrate. The results are mirrored by a general decrease in the efficiency of production and in the quality of a finished product.

The physical disabilities caused by excessive heat exposure are, in order of increasing severity, heat rash, heat cramps, heat exhaustion, and heat stroke.

Heat rash (prickly heat) may be caused by unrelieved exposure to hot and humid air as may occur in warm-moist climatic zones. The orifices of the sweat ducts become plugged due to the swelling of the moist keratin layer of the skin which leads to inflammation of the glands.

There are tiny red vesicles visible in the affected skin area and, if the affected area is extensive, sweating can be substantially impaired. As a consequence heat rash not only is a nuisance because of the discomfort it causes but also can greatly diminish the workers' capacity to tolerate heat.

Heat cramps may occur after prolonged exposure to heat with profuse perspiration and inadequate replacement of salt. The signs and symptoms of heat cramps consist of spasm and pain in the muscles of the abdomen and extremities. Albuminuria may be a transient finding.

Heat exhaustion may result from physical exertion in a hot environment when vasomotor control and cardiac output are inadequate to meet the increased demand placed upon them by peripheral vasodilatation or the plasma volume is reduced by dehydration. Signs and symptoms of heat exhaustion may include palor, lassitude, dizziness, syncope, profuse sweating, and cool moist skin. There may or may not be a mild hyperthermia, observable by rectal measurement.

Heat stroke is a serious medical condition. An important predisposing factor is excessive physical exertion. Signs and symptoms may include dizziness, nausea, severe headache, hot dry skin because of cessation of sweating, very high body temperature (usually 106°F and rising), confusion, collapse, delirium, and coma. Often circulation is also compromised to the point of shock. If cooling of the victim's body is not started immediately, irreversible damage to vital organs may develop, leading to death.

Some studies performed in Europe and in South America showed evidence that workers employed for prolonged time in hot industry have a higher morbidity rate from cardiovascular diseases.

RECOMMENDED LIMITS

Higher heat exposures than shown in Table 11 are permissible if the workers have been undergoing medical surveillance and it has been established that they are more tolerant to work in heat than the average worker. Workers should not be permitted to continue their usual work routine when their deep body temperature exceeds 38.0°C.

Table 11. Permissible heat exposure threshold limit values.

	Work load*		
Work-Rest regimen	Light	Moderate	Heavy
Continuous work	30.0	26.7	25.0
75% Work 25% Rest, Each hour	30.6	28.0	25.9
50% Work 50% Rest, Each hour	31.4	29.4	27.9
25% Work 75% Rest, Each hour	32.2	31.1	30.0

*Values are given in °C WBGT

POTENTIAL OCCUPATIONAL EXPOSURES

Animal rendering workers
Bakers
Boiler heaters
Cannery workers
Chemical plant operators working near hot containers and furnaces
Cleaners
Coke oven operators
Cooks
Foundry workers
Glass manufacturing workers
Kiln workers
Miners in deep mines
Outdoor workers during hot weather
Sailors passing hot climatic zones
Shipyard workers when cleaning cargo holds
Smelter workers
Steel and metal forgers
Textile manufacturing workers (weaving, dyeing)
Tire (rubber) manufacturing workers

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COLD

For the body to maintain thermal homeostasis in a cold environment, certain physiologic mechanisms come into play which tend to limit heat loss and increase heat production. The first mechanism is one of peripheral vasoconstriction, especially in the extremities, resulting in a marked drop in skin temperature. Body heat loss to the environment is thereby diminished. The most severe strain of this mechanism of heat conservation is chilling of the extremities so that if activity is restricted, the toes and fingers may approach freezing temperatures very rapidly.

Long before that, and in fact when their temperature drops below 15°C, the hands and fingers become insensitive, and the probability of malfunction and accidents increases.

In general, cooling stress is proportional to the total thermal gradient between the skin and the environment since this gradient determines the rate of heat loss from the body by radiation and convection. Loss of heat through the mechanism of the evaporation of perspiration is not significant at environmental temperatures lower than about 15° to 20°C. When vasoconstriction is no longer adequate to maintain body heat balance, muscular hypertonus and shivering become important mechanisms for increasing body temperature by causing metabolic heat production to increase to several times the resting rate. Not only shivering, but general physical activity acts to increase metabolic heat. With proper insulation from clothing to minimize heat loss through even a large thermal gradient, a satisfactory microclimate may be maintained with only exposed body surfaces (as the face and the digits of hands and feet) liable to excessive chilling and frostbite. However, if the garments become wet either from contact with water or due to sweating during intensive physical work, their cold insulating property will be greatly diminished.

HARMFUL EFFECTS

Frostbite occurs when there is actual freezing of the tissues with the attendant mechanical disruption of cell structure. Theoretically, the freezing point of the skin is −1°C; however, with increasing wind velocity, heat loss is greater and frostbite will occur more rapidly. Once started, freezing progresses rapidly. For example, if the wind velocity reaches 20 mph, exposed flesh will freeze within about one minute at −10°C. Furthermore, if the skin comes in direct contact with objects whose surface temperature is below freezing point, frostbite may develop in spite of warm environmental temperatures. The first warning of frostbite is often a sharp, pricking sensation. However, cold itself produces numbness and anesthesia which may permit serious freezing to develop without the warning of acute discomfort. Injury produced by frostbite may range from simple superficial injury with redness of the skin, transient anesthesia and superficial bullae to deep tissue freezing with persisting ischemia, thrombosis, deep cyanosis, and gangrene.

Trench foot or immersion foot may be caused by long continuous exposure to cold without freezing, combined with persistent dampness or actual immersion in water. This condition is due to persistent local tissue anoxia, combined with mild or severe cold with resultant injury to the capillary walls. Edema, tingling, itching, and severe pain occur and may be followed by blistering, superficial skin necrosis, and ulceration.

General hypothermia is an extreme acute problem resulting from prolonged cold exposure and heat loss. If an individual becomes fatigued during physical activity, he will be more prone to heat loss, and as exhaustion approaches, the vasoconstrictor mechanism is overpowered; then sudden vasodilatation occurs with resultant rapid loss of heat, and critical

cooling ensues. Sedative drugs and alcohol increase the danger of hypothermia.

Vascular abnormalities may be either precipitated or aggravated by cold exposures. These include chilblain (pernio), Raynaud's disease, acrocyanosis, and thromboangiitis obliterans. Workers suffering from these ailments should take special precautions to avoid chilling. Some people develop hypersensitivity reactions when exposed to cold.

RECOMMENDED LIMITS

Cold stress indices have been developed for estimating the significance of cold environments for human welfare and efficiency. Those relating insulating effect of clothing and the convective heat loss of cold air movement (wind chill) are probably most useful in predicting the impact of cold outdoor exposure.

POTENTIAL OCCUPATIONAL EXPOSURES

Occupations with potential exposure include:

Cooling room workers
Divers
Dry ice workers
Firemen
Fishermen
Ice makers
Liquified gas workers
Out-of-door workers during cold weather
Packing house workers
Refrigerated warehouse workers
Refrigeration workers

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HYPERBARIC ENVIRONMENTS

Air pressures in excess of those found at sea level (hyperbaric) are encountered in both terrestrial and aquatic environments. Sea level pressure equals 14.7 pounds per square inch, or one atmosphere absolute (ata). Occupational exposures occur in caisson or tunneling operations, where a compressed gas environment is used to exclude water or mud and to provide support for structures. Pressures encountered in such

operations range from less than 2 ata to more than 4 ata. Similarly, hyperbaric environments are encountered by divers operating underwater, whether by holding the breath while diving, breathing from a self-contained underwater breathing apparatus (SCUBA), or by breathing gas mixtures supplied by compression from the surface. While commercial divers routinely dive to depths greater than 100 meters, even in breath-holding dives to 30 meters, pressures encountered can be considerable (each 10-meter increase in sea water depth is equivalent to an increase of 1 atmosphere pressure).

PRIMARY PRESSURE PHENOMENA

Man can withstand large pressures above normal, providing air has free access to all surfaces of the body including lungs, sinuses, and the middle ear. Unequal distribution of pressure can result in barotrauma, probably the most common occupational disease of those who work in high pressure environments. Barotrauma refers to tissue damage resulting from expansion or contraction of gas spaces found within or adjacent to the body, and can occur either during compression (descent) or during decompression (ascent).

The teeth, sinuses, and ears are frequently affected by such pressure differentials. For example, gas spaces which may be present adjacent to tooth roots or fillings may be compressed during descent. Fluid or tissue forced into these spaces may cause pain either during descent or ascent. Sinus blockage, comparatively rare in divers, is probably due to occlusion of the sinus aperture by inflamed nasal mucosa which prevents equalization of pressures.

Middle ear barotrauma (aerotitis media) occurs commonly among divers. Blockage of the eustachian tube as a result of inflammation or by failure of the diver to clear the ears, creates a negative middle ear pressure during compression, with progressive inward deformation of the tympanic membrane, with possible rupture. Forceful Valsalva maneuvers under these conditions can also result in round window rupture with inner ear damage.

The lungs themselves may be subject to squeeze if the chest is compressed to a volume smaller than the residual volume of the lung, the amount of air left in the lungs following forced expiration. Lung squeeze is occasionally seen in unprotected swimmers who dive by simply holding the breath. The effect of the squeeze is to force blood and tissue fluids into the respiratory passages and alveoli. Considerable lung damage may result.

SECONDARY PRESSURE PHENOMENA

In addition to the mechanical effects there are well known problems of toxicity from the gases of air at elevated partial pressures. Also some normally toxic gases such as carbon monoxide are probably more toxic at elevated partial pressure. These phenomena have to do with molecular rather than with bulk gas characteristics.

Narcotic action of nitrogen: At 4 atmospheres of pressure or more,

the gaseous nitrogen in normal air induces a narcotic action evidenced by decreased ability to work, mood changes, and frequently, a mild to marked euphoria. The responses are similar to those associated with alcoholic intoxication. The exact cause of this cerebral disturbance is unknown. It may be noted, however, that nitrogen is highly soluble in fat, the ratio of its solubility in fat to its solubility in water being about five to one. According to the Meyer-Overton hypothesis, a gas having such a relatively high solubility ratio may act as a narcotic.

Oxygen poisoning: Inhalation of oxygen when its partial pressure exceeds two atmospheres may result in the production of the signs and symptoms of oxygen poisoning. These include tingling of fingers and toes, visual disturbances, acoustic hallucination, confusion, muscle twitching, especially about the face, nausea, and vertigo. The final result of such exposure may be the epileptiform convulsion, which ceases as soon as exposure to high oxygen partial pressures is terminated. This toxic action of oxygen is greatly enhanced by exercise or by the presence of moderate amounts of carbon dioxide. At one atmosphere, about 15 p.s.i., pure oxygen will irritate the throat although symptoms of systemic oxygen poisoning do not occur if the exposure is relatively short.

It should be noted that the greatest hazard in oxygen administration in chambers is the danger of fire. It is also true that in increased environmental pressures an increased partial pressure of oxygen enhances the fire hazard.

Effect of carbon dioxide: Carbon dioxide enhances the toxicity of oxygen and the narcotic effect of nitrogen, and in addition a higher incidence of bends has been reported in association with a rise in the CO_2 pressure. The partial pressure of CO_2 present in the breathing medium in a compressed air environment should not exceed the equivalent of 0.2 percent CO_2 at one atmosphere pressure.

DECOMPRESSION

An opposite effect to lung squeeze, expansion of air in the lungs, may occur during ascent from depths of water or during decompression in a chamber. Air in the lung at a depth of 130 feet is at 5 ata. It will increase in volume five times when decompression to normal atmospheric pressure occurs. If decompression is excessively rapid and sufficient air is not exhaled, some of the pulmonary alveoli will rupture with the formation of one or more of the following: mediastinal emphysema, pneumothorax, or air embolism. The most dangerous of these conditions is the air embolism which occurs when air, expanding in the lung, is forced into the pulmonary blood vessels and then into the left side of the heart. The arterial circulation may quickly carry the air bubbles to the brain to produce a cerebral air embolism, a condition which may be rapidly fatal if not treated promptly by decompression.

A more likely mechanical problem of a too rapidly decreased air pressure is formation of nitrogen bubbles as the gas leaves solution in blood and tissues, a situation comparable to bubble formation in carbonated beverages when the closure is broken. These bubbles of liberated

gas create circulatory impairment and local tissue damage and are responsible for the signs and symptoms of decompression sickness.

The amount of bubble formation that will occur upon decompression depends to a large extent upon 1) the amount of gas dissolved in the tissues, which in turn is dependent upon the degree and duration of exposure to pressure and upon the amount of body fat in which gas can be dissolved; 2) conditions which alter blood flow, including age, temperature, exercise, fright, and post-alcoholic state, especially if these alterations in blood flow occur during or shortly after the decompression process; and 3) the rapidity of decompression from elevated air pressure to the ambient level. The conditions can, but are less likely to, occur upon rapid ascent from ground level to high elevation in high performance aircraft.

Acute Signs and Symptoms

Bends: A relatively common manifestation of decompression sickness is a dull, throbbing type of pain which is gradual in onset, progressive and shifting in character, and frequently felt in the joints or deep in the muscles and bones. When the symptoms of bends occur, they do so in the first four to six hours in 80 percent of the cases, while the remainder will occur within 24 hours. Contributing to variations in susceptibility are such factors as age, obesity, defects to the lungs, heart impairments, temporary ill health, and individual predisposition.

Chokes: This rather specific type of asphyxia occurs less frequently than bends and is thought to be due to the accumulation in the large veins, the right side of the heart, and the pulmonary vessels of quantities of gas eliminated from the arterial circulation and from the extravascular tissues. The earliest evidence of impending chokes is a sensation of substernal distress felt during deep inspiration, especially during inhalation of tobacco smoke which elicits paroxysmal coughing. These attacks of coughing may proceed to loss of consciousness with all of the signs and symptoms of a true shocklike syndrome.

Paralysis: The most serious complication of decompression sickness is paralysis. Spastic paraplegia or monoplegia involving the lower extremities may follow the formation of bubbles in the blood vessels and tissues of the spinal cord. Immediate and prolonged decompression usually brings about rapid recovery even following paraplegia. Cerebral involvement is very rare.

Chronic Symptoms

Aseptic bone necrosis: The most likely chronic sequela of repeated compressed air exposure is termed aseptic bone necrosis. This condition is thought to be caused by the occlusion of small arteries in the bone by bubbles of nitrogen followed by infarction in the involved area. The sites of predilection for the occurrence of occlusion and necrosis, as seen in this process, are the lower femoral diaphysis, the upper tibial diaphysis, and the head and neck of the humerus and the femur. These lesions are usually multiple and tend to be bilaterally symmetrical.

Aseptic bone necrosis is usually asymptomatic unless joint surfaces are involved, in which case pain may be a symptom. Complete collapse of the affected joint has been known to occur. Healing takes place through an osteocondensing process. This increase in density may appear on roentgenographic examination as a snowcap on the top of the articular surface.

Dysbaric osteonecrosis: A significant incidence of dysbaric osteonecrosis has been recognized in caisson workers in the USA and England. It has also been recognized to occur in Royal Navy divers, among commercial divers working in USA coastal waters, in Japanese breathholding divers, and even in U.S. Air Force pilots. There appears to be a correlation between the disease and the number of decompressions undergone by an individual, frequency of exposure, magnitude of pressure, and frequency of dysbarism-related incidents. It is uncertain whether the disease can be prevented by adherence to recommended decompression schedules.

HYPOBARIC ENVIRONMENTS

Two rather distinct types of occupational exposure to hypobaric environments exist: high altitude and low altitude.

HIGH ALTITUDE SYMPTOMS

Among pilots and air crews engaged in operation of high performance aircraft at extremely high altitudes (in excess of 30,000 feet), the greatest single potential hazard is hypoxia. Deprivation of oxygen at these altitudes results in rapid loss of consciousness. Exposure to these reduced pressures (dysbarism) may also produce symptoms similar to those encountered by rapid decompression in divers. Bends, chokes, neurological disorders, aeroembolism, aerodontalgia, aerotitis, and aerosinusitis have all been described in air crewmen.

Dysbarism may be complicated by a type of neurogenic peripheral circulatory failure or primary decompression shock consisting of any or all of the following manifestations: intense pallor, profuse sweating, faintness and dizziness, nausea, vomiting, and loss of consciousness. These symptoms are usually relieved rapidly by descent from altitude.

OTHER ALTITUDE SYMPTOMS

Potential occupational hazards also exist at much lower altitudes, where the effects of hypoxia are evidenced by impaired judgment and performance, and a general feeling of malaise. Acute mountain sickness (AMS) is considered a definite clinical syndrome characterized by overwhelming depression, severe headache, nausea, vomiting, and loss of appetite. Particularly characteristic is irritability of the subject. Virtually all sojourners develop one or more symptoms, although the severity of such symptoms varies widely among subjects. Peak severity is reached within 48 hours and symptoms disappear over the following 2 to 4 days.

PULMONARY EDEMA

Of considerable concern to the physician is the not infrequent occurrence of high altitude pulmonary edema. This circulatory disturbance appears more frequently in children than adults, and frequently occurs when altitude–acclimatized subjects return from sojourning at sea level. There is a strong tendency for it to recur repeatedly in susceptible subjects. The condition usually begins with progressive cough and dyspnea, and is associated with elevated pulmonary arterial pressures. Treatment with O_2 or return to sea level usually abolishes symptoms rapidly.

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OSCILLATORY VIBRATIONS

Terry L. Henderson, Ph.D., Derek E. Dunn, Ph.D., R. J. Nozza, M.A., and Don Wasserman, M.S.E.E.

Research and study continue in efforts to verify noise tolerance limits for protecting human hearing. The effects of different vibration conditions for causing pain and injury or illness to individuals exposed are also under study. The following sections offer a broad overview of present findings in relation to recognizing the occupational origin of certain symptoms due to exposure to oscillatory vibrations.

NOISE

Advanced mechanization has created excessive noise in many occupations. Various aspects of noise exposure have been correlated with hearing loss, and proposed exposure limits for protecting against hearing loss have been developed.

DESCRIPTION

Noise is generally identified as unwanted sound. The word "sound" itself can be used to mean either a physical pressure oscillation (alternate increases and decreases in normal atmospheric pressure caused by a rapidly vibrating object) or the resulting subjective auditory sensation that occurs when the hearing mechanism is stimulated. The rate of vibration of the object corresponds to the frequency of sound expressed in Hertz (Hz), the unit of frequency corresponding to one vibration cycle per second.

The frequency range of audible sounds for healthy young ears is usually considered to extend from 20 to 20,000 Hz although there is evidence to indicate that the range of man's hearing extends beyond these limits. The simplest type of sound, called a pure tone, consists of a very regular oscillation at a single frequency. This sound may be produced by a tuning fork or electric means. In contrast, music, speech, and noise, each containing a collection of different frequency sounds, are called complex sounds.

The pattern of distribution of acoustical energy at the various frequencies is referred to as the *spectrum* of the sound. The frequencies comprising speech are found principally between 250 and 3,000 Hz. This is, therefore, considered to be the most important range of frequencies, since hearing loss for speech sounds would handicap the individual in most daily activities.

MEASUREMENT

Sound pressure level (SPL) measurements are based upon the average (root mean square) amplitude of the pressure changes constituting the sound stimulus and are directly related to the intensity or energy characteristics of the sound. The unit of measurement is the "decibel," abbreviated "dB." Instruments are readily available for measurement of SPL. When measuring intense noises, one usually uses the "A-weighting" feature that is incorporated into most sound level meters in order to partially simulate the response to the human ear. If so, then the abbreviation for decibel is modified to "dB(A)."

The process of measuring the environmental sound level presents problems because the sound levels are apt to be variable and to change rapidly with time as well as with position. Although negative dB levels (below zero) are theoretically possible, and extremely high levels may be encountered at the exhaust of a turbojet or rocket, with rare exception the environmental sound level will lie within the range of 20 to 125 dB(A).

Doubling the number of noisy machines in a room does not double

the sound level (it will probably increase by only about 3 dB). The sound level in a room will depend upon 1) the total amount of sound energy being produced within the room or leaking into the room from the outside, 2) how thoroughly the room is enclosed, i.e., how well sound is prevented from leaking out, 3) how acoustically absorbent the walls and contents of the room are, 4) the size and shape of the room, and 5) the distance to the sound source and to reflecting or shielding surfaces.

TYPES

Noise, commonly defined as unwanted sound, covers the range of sound which is implicated in harmful effects. Noise can be classified into many different types, including wide-band noise, narrow-band noise, and impulse noise. To describe the spectrum of a noise the audible frequency range is usually divided into eight frequency bands, each one-octave wide, and SPL measurements are made in each band using a special sound level meter. A wide-band noise is one where the acoustical energy is distributed over a large range of frequencies. Examples of wide-band noise can be found in the weaving room of a textile mill and in jet aircraft operations.

Narrow-band noises, with most of their energy confined to a narrow range of frequencies, normally produce a definite pitch sensation. For a true narrow-band noise, only a single octave band will contain a significant SPL. The noise caused by a circular saw, planer, or other power cutting tools is occasionally of the narrow-band type, but usually there is some spreading of the acoustic energy to several of the octave bands.

The impulse type of noise consists of transient pulses, occurring in repetitive or nonrepetitive fashion. The operation of a rivet gun or a pneumatic hammer usually produces repetitive impulse noise. The firing of a gun is an example of non-repetitive impulse noise.

HARMFUL EFFECTS

Exposure to intense noise causes hearing losses which may be temporary, permanent, or a combination of the two. These impairments are reflected by elevated thresholds of audibility for discrete frequency sounds, with the increase in dB required to hear such sounds being used as a measure of the loss. Temporary hearing losses, also called auditory fatigue, represent threshold losses which are recoverable after a period of time away from the noise. Such losses may occur after only a few minutes of exposure to intense noise. With prolonged and repeated exposures (months or years) to the same noise level, there may be only partial recovery of the threshold losses, the residual loss being indicative of a developing permanent hearing impairment.

Temporary

Temporary hearing impairment has been extensively studied in relation to various conditions of noise exposure. Findings include the following:

1) Typical industrial noise exposures produce the largest temporary hearing losses at test frequencies of 4,000 and 6,000 Hz. The actual

pattern of loss depends upon the spectrum of the noise itself. The greatest portion of the loss occurs within the first 2 hours of exposure. Recovery from such losses is greatest within 1 or 2 hours after exposure.

2) The amount of temporary hearing loss from a given amount of noise varies considerably from individual to individual. For example, losses at a given frequency due to noise intensities of 100 dB(A) may range from 0 to more than 30 dB.

3) Low frequency noise, below 300 Hz, must be considerably more intense than middle or high frequency noise to produce significant threshold losses.

4) Considerably fewer temporary hearing losses result from intermittent than from continuous noise exposure, even though the total amount of noise exposure is the same in both instances.

Permanent

The permanent hearing loss that is seen in workers who have been exposed to noise daily for a period of many years is very similar to the pattern of temporary hearing loss except that the permanent loss is not recoverable and does not respond to any known treatment or cure.

Exposure to intense noise, however, is only one cause of permanent hearing damage. Other causes may be disease, mechanical injury, and use of drugs. The time and nature of onset of the loss, the pattern of hearing loss for different frequencies, the findings of an otologic examination and medical history are factors in determining whether a case of permanent hearing damage might be due to noise exposure or other causes. Once these causes have been excluded from the etiology of hearing damage, the losses attributable to the aging process (presbycusis) must be considered. Curves showing the usual deterioration in hearing with increasing age are used to differentiate the amount of hearing loss due to noise exposure from that due to the aging process.

Figure 6 illustrates median shifts in hearing acuity of a group of noise-exposed jute weavers. (Corrections for the effects of aging were used to determine the effects due solely to noise.)

Although no direct physiological link has been established between temporary and permanent hearing loss, the similarities have led to some tentative conclusions, including the notion that permanent loss represents the long term accumulation of residual losses from incomplete recovery to repeated, daily temporary hearing loss. Evidence from noise-exposed groups of workers shows that permanent threshold losses caused by noise initially appear in the region 3,000 to 6,000 Hz and are most prominent at 4,000 Hz. With continued exposure, the losses in hearing become greater and occur at frequencies above and below the 3,000 to 6,000 Hz range until eventually losses are shown at most frequencies.

The losses in hearing due to exposure to intense occupational noise (105 dB(A) or above) tend to reach a plateau at certain frequencies (most notably 4,000 Hz) after about 10 years of exposure; further losses in hearing at the frequency then develop more slowly, and may be accounted for substantially by the aging process. Since the hearing loss for such

frequencies which result from a 10-year exposure to noise appears to approximate the temporary hearing loss resulting from a single day's exposure, it is possible that, when validated, the use of temporary threshold losses as a susceptibility index for predicting permanent noise-induced hearing losses may be a useful screening tool.

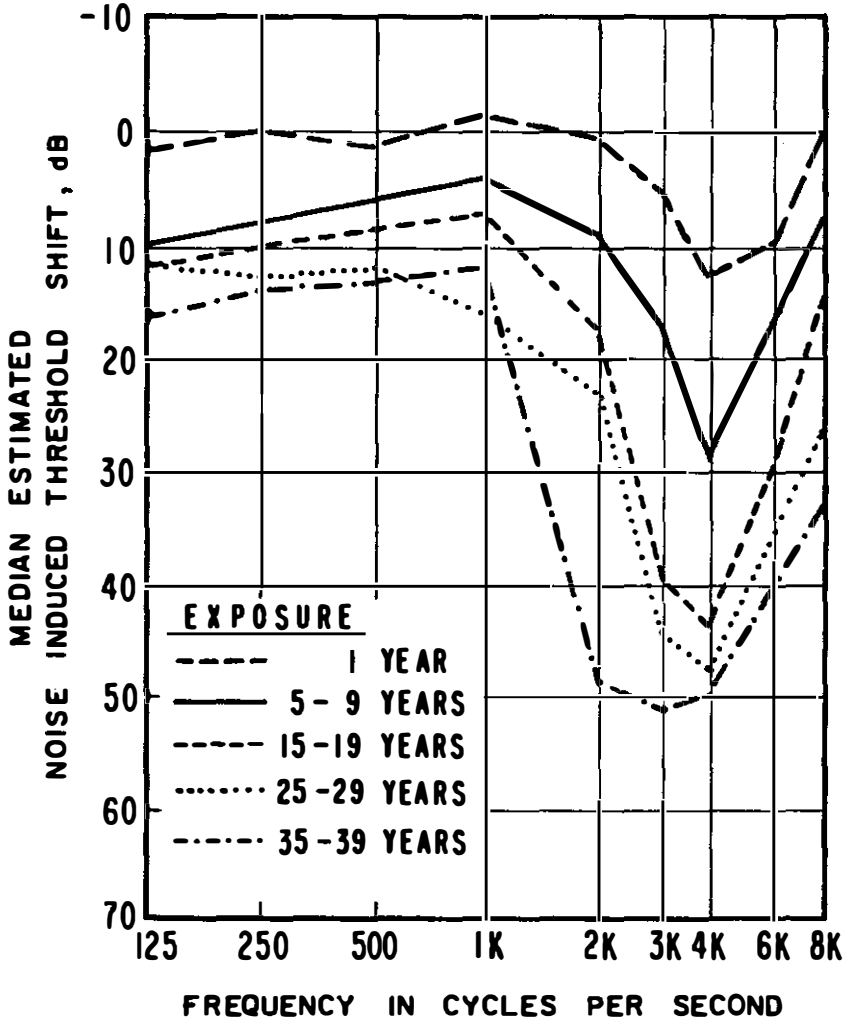

Figure 6. Median permanent threshold shifts in hearing levels as a function of exposure years to jute weaving noise. (Data taken from Taylor, et al. [Ref. Taylor W. A., A. Mair, and W. Burns. 1965. Study of noise and hearing in jute weaving. J. Acoust. Soc. Am. 48:524-530]). (Figure from Criteria for a Recommended Standard . . . Occupational Exposure to Noise.)

Communications Interference

Noise which is not intense enough to cause hearing damage may still disrupt speech communication and the hearing of other desired sounds. Such disruptions will affect performance on those jobs which depend upon reliable speech communication and may contribute to job stress. More important, however, is the fact that the inability to hear commands or danger signals due to excessive noise increases the probability of severe accidents. Ear protectors are no solution because when they are worn, shouting will be necessary for communication, which may lead to hoarseness, and communication is still not assured.

Physiologic Effects

Physiologic reactions to a noise of sudden onset represent a typical startle pattern. There is a rise in blood pressure, an increase in sweating, an increase in heart rate, changes in breathing, and sharp contractions of the muscles over the whole body. These changes are often regarded as an emergency reaction of the body, increasing the effectiveness of any muscular exertion which may be required. However desirable in emergencies, these changes are not desirable for long periods since they could interfere with other necessary activities. Fortunately, these physiologic reactions subside with repeated presentations of the noise.

For performance on a task to remain unimpaired by noise, man must exert greater effort than would be necessary under quiet conditions. When measures of energy expenditure — for example, oxygen consumption and heart rate — are made during the early stages of work under noisy conditions they show variations which are indicative of increased effort. Measurements in later stages under continued exposure, however, show responses return to their normal level.

RECOMMENDED PROTECTIVE METHODS

Those controlling the individual company hearing conservation activities should insure that no permanent hearing loss occurs among the employees.

PERMISSIBLE EXPOSURE LIMITS

Noise dose limits are now required for workplaces to minimize hearing loss from occupational exposure. Although louder noise is allowed for brief periods during the workday, the mandatory noise level limitation is 90 dB(A) for 8 hours exposure. As noted earlier, the "(A)" denotes the use of the A-weighting scale of the sound level meter, which takes into account the relative effectiveness of different frequencies of the noise spectrum in producing hearing damage.

AUDIOMETRIC TESTING

Since early noise-induced losses almost always occur at frequencies slightly above the so-called speech range, substantial impairments in hearing can occur without the individual's being aware of it. Impairments in the perception of speech may not become noticeable until losses for the speech frequencies are 20 dB or more.

An effective audiometric testing program can be provided by utilizing either company personnel and equipment or the services of an independent organization on a contract basis.

The audiometric test is a simple means of evaluating a person's hearing acuity. An audiogram, which is a graph of hearing vs. frequency, is the product of an audometric evaluation. An audiogram should be obtained periodically for any employee working in a noise-related job in order to monitor changes, if any, in hearing status. The initial or baseline audiogram should be obtained prior to an employee's first day on a new assignment. The evaluation supplies the employer with not only valuable information concerning the worker's ability to perform the job safely and competently, but also documentation of the employee's hearing at that date in the event of a future claim for hearing loss compensation.

In addition, the aging process, possible use of ototoxic drugs, off-the-job activities, over-susceptibility to noise, past medical problems, and former work experiences, must all be taken into consideration in a comprehensive hearing evaluation. Most pertinent information can be acquired when a full history is obtained at the preemployment examination and supplemented at periodic tests.

Audiometric monitoring can be effective in protecting the worker from incurring a significant hearing loss. Changes in a worker's hearing may indicate that the hearing conservation program is failing. Noise measurements should be made to determine if there has been a change in the noise levels or work patterns since the last noise survey was done. A change in the noise level may be due to a modification of manufacturing technique, a malfunction of existing equipment, or the addition of new equipment. One should then determine if the noise control measures are actually providing the attenuation anticipated and whether administrative measures to reduce individual exposure doses are adequate. It is very important to confirm that proper conditions of fit are being maintained for any ear protectors being used. The worker may need a refresher course on the use of ear protectors and the benefits of cooperating with the hearing conservation effort. Unless the changes revealed by the audiometric monitoring lead to effective correction action, further hearing losses will not be prevented and the monitoring program will have been rendered valueless.

When hearing testing is done, certain conventions must be followed. A suitable quiet environment for the tests is particularly important. The equipment used must meet fairly rigid specifications. The testing apparatus (audiometer, head phones, test room) must be calibrated and procedure must be standardized in order to insure the comparability of a test taken at one place and time with those done at some later time in perhaps other workplaces.

The equipment is not the only component that needs careful attention in audiometric testing. The person responsible for the monitoring program must be properly trained in the use of the audiometer, the meaning of the audiogram, dealing with the industrial worker, and in the providing of ear protection.

EAR PROTECTION DEVICES

The subject of fitting of ear protection deserves special attention. There are a variety of different types of devices available, each requiring special procedures and considerations for obtaining proper fit. An employee may require several trials with different types and sizes of plugs before a suitable combination of comfort and protection is obtained. The decibel attenuation data provided by the manufacturer do not reflect the inherent physical property of the device, but rather an optimistic level of performance to be expected under good conditions of fit, which cannot usually be maintained in the workplace.

POTENTIAL OCCUPATIONAL EXPOSURES

Noise is the most widespread of all the occupational exposures and may be encountered in almost any occupation.

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VIBRATION

Approximately 8 million workers in the United States are exposed to occupational vibration (1). Most of these are workers in transportation (e.g., truck and bus driving), farming, and construction; other exposed workers are users of chain saws, pneumatic tools, and vibratory electrical hand tools.

TERMINOLOGY

Vibration refers to any horizontal or vertical back-and-forth motion of matter. With reference to man, vibration usually is subdivided into wholebody and segmental vibration.

Wholebody vibration is vibration transmitted to the entire human

body through some supporting structure such as a vehicle seat or building floor.

Segmental vibration is vibration applied locally to specific body parts, such as the hands and feet, by a vibrating hand tool, for example.

Vibratory frequency expressed in Hertz (Hz) describes the cyclic nature of vibration. For wholebody vibration the 2 to 100 Hz range is of interest; for segmental vibration the range of about 8 to 1,500 Hz is of interest.

Displacement refers to the distance between the normal resting position of an object and its position at a given time in its vibratory cycle.

Velocity refers to the time rate of change and displacement and is expressed in ft/sec. or meters/sec.

Acceleration refers to the time rate of change of velocity (i.e., the rate at which the velocity of the vibratory motion changes in direction). Acceleration has been the most frequently used measure of vibratory magnitude because of the ease of measurement and the fact that from this single measure, both vibration velocity and displacement can be easily derived with electronic integration. Acceleration is measured in gravitational (g) units, expressed also in meters/sec. where 1 g = 9.8 m/sec.

Resonance refers to the human body tendency to act in concert with externally generated vibration and to actually amplify the vibration; for example, at 5 Hz man's wholebody is in "resonance" with a vibratory source; if, for example, at 5 Hz a vibratory magnitude of 1 g were applied to a human subject's buttocks, one could expect to measure as much as 2.5 g vibratory magnitude at the cranial level; thus, the body has intensified the actual applied vibration by a factor of 2.5 (g=gravitational force at sea level).

WHOLEBODY VIBRATION

Chronic effects of vibration are not adequately known. Short-term human and animal studies (2) however, have shown that wholebody vibration may be regarded as a "generalized stressor" and may affect multiple body parts and organs depending on the vibration characteristics. For example, for man the principal wholebody resonance occurs at 5 Hz (mostly accounting for the resonance characteristics of the trunk and upper torso); however, the head-shoulder system can resonate in the frequency range of 20 to 30 Hz; and the eyeballs can resonate in the 60 to 90 Hz range. Other body parts can resonate at other frequencies. In general, the larger the system mass, the lower the resonant frequency.

HARMFUL EFFECTS

Animal Studies: A study of rats exposed to vibration revealed a drop in lymphocyte count, an increase in granulocyte count, an increased leukocytic alkaline phosphatase activity, faster red-cell sedimentation, higher plasma and erythrocyte chloride levels, and a lowering of ascorbic acid and ATP levels of the erythrocytes (3). In a study of liver and kidney function of rats exposed to vibration, ischemia of the liver and kidneys resulted after a single hour of exposure; hyperemia resulted in these organs after 10 days' exposure, and after 21 days of exposure, portions

of the vascular system ceased functioning (4). In a laboratory study on the effects on monkeys of exposure to vibration, gastro-intestinal bleeding and lowered hematocrits were noted during the exposure and multiple lesions of the gastric mucosa were seen at necropsy (5).

Human Studies. Studies of human subjects (6,7,8,9) have shown that during wholebody vibration there are increases in oxygen consumption and pulmonary ventilation. If human subjects are exposed to intense vibration, they may have difficulty in maintaining steady posture.

One study of 78 Russian concrete workers exposed to wholebody vibration showed marked changes in bone structure involving spondylitis deformations, intervertebral osteochondrosis, and calcification of the intervertebral discs and Schmorl's nodes (10). Hypoglycemia, hypocholesteremia, and low ascorbic-acid levels in concrete workers exposed to occupational vibration have also been reported (11). Gastrointestinal tract changes in gastric secretions and peristaltic motility have been noted in human (12) and animal (13) studies. Changes in nerve-conduction velocities due to vibration have also been reported (14).

In one Polish study of agricultural and forestry workers a clinical description of so-called vibration sickness is found:

> The first stage is marked by epigastralgia, distension, nausea, loss of weight, drop in visual acuity, insomnia, disorders of the labyrinth, colonic cramps, etc. The second stage is marked by more intense pain concentrated in the muscular and osteoarticular systems. Objective examinations of the workers disclosed muscular atrophy and trophic skin lesions. It is apparent that it is difficult to determine the critical moment at which pathological changes set in, especially due to differences in individual sensitivity to vibration (15).

Safety Implications

In the human-performance area, with its possible safety implications, studies of vibration have shown that the lowest subjective-discomfort-tolerance level occurs around the 5-Hz resonant point. Manual tracking capability is also most seriously affected at this 5-Hz point. Visual acuity is severely impaired in the 1- to 25-Hz range.

On the other hand, performance of tasks such as those involving pattern recognition, reaction time, and monitoring appears not to be affected by exposure to vibration (16,17). Simulated heavy equipment driving tests which compared the effects of a mixture of simultaneous vibratory frequencies (similar to actual occupational vibration) revealed that human subjects performed worse under the mixed conditions, gradually improving as the mixture was replaced by nonresonant single sinusoidal vibratory conditions (18).

SEGMENTAL VIBRATION

Segmental vibration, unlike wholebody vibration, appears to be more a localized stressor creating injury to the fingers and hands of exposed workers using such vibratory hand tools as chain saws, pneumatic chipping hammers and picks, and electrically operated rotary grinders. Extensive use of such tools (especially in cold environments) has elicited the so-called Raynaud's phenomenon (i.e., "dead hand" or "vibration white fingers" (VWF)). This condition is characterized by numbness and blanching of the fingers with probable loss of muscular control and

Table 12. Stages of Raynaud's phenomenon.

Stage	Condition of digits	Work and social interference
0	No blanching of digits	No complaints
0_T	Intermittent tingling	No interference with activities
O_N	Intermittent numbness	
1	Blanching of one or more fingertips with or without tingling and numbness	No interference with activities
2	Blanching of one or more complete fingers with numbness, usually only in winter	Slight interference with home and social activities and interference at work
3	Extensive blanching, usually all fingers bilateral, with frequent episodes, summer and winter	Definite interference at work, at home, and with social activities and hobbies
4	Extensive blanching with all fingers involved and frequent episodes summer and winter	Occupation change required because of severity of signs and symptoms

reduction of sensitivity to heat, cold, and pain. Taylor (19) has developed and utilized a clinical classification scheme (Table 12) for assessing the extent of Raynaud's phenomenon:

Localized vibratory effects are not limited to Raynaud's phenomenon. Studies have shown changes in bone (20) and development of muscular weakness and degenerative alterations (21,22), primarily in the ulnar and median nerves. Also reported are cases of vascular changes and muscle atrophy (23,24), tenosynovitis (24), and Dupuytren's disease (24, 25) as well as cysts on some of the bones of the hand (24).

POTENTIAL OCCUPATIONAL EXPOSURES

Wholebody Vibration

Truck drivers
Bus drivers
Heavy equipment operators
Farm vehicle and tractor operators
Foundry workers (mold shakeout, fork lift trucks, overhead cranes)
Railroads (engineers, conductors, track repair workers)
Fork lift operators
Overhead crane operators
Textile machine operators
Metal (refining, mills, manufacturing) operations (rolling operations, fork lift trucks, overhead cranes, stamping operations, electric arc furnace operations)

Machine tool operators
Quarry workers
Mining (strip, and underground mining using automatic mining machines)
Vehicular body stamping operators
Lumber mills, saw plants, plywood plants, wood products manufacturing operations
Printing and publishing, press operators
Shoe manufacturing (routing operators)

Hand-Arm (Segmental) Vibration

Chain sawyers
Pneumatic tool operators (chippers, staple gun operators, construction, and road operation)
Mining (jack leg and hand tool)
Electrical grinder operators (rotary, stand, swing grinders)
Metal extrusion operators
Wood products manufacturing

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9. Hood, Jr., W. B., R. H. Murray, C. W. Urschel, J. A. Bowers, and J. G. Clark. 1966. Cardiopulmonary effects of wholebody vibration in man. J. Appl. Physiol. 21:1725.
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16. Grether, W. F. 1971. Vibration and human performance. Hum. Factors 13:203.
17. Shoenberger, R. W. 1972. Human response to wholebody vibration. Percept. Mot. Skills (Mono. Suppl. 1) 34:127.
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22. Steward, A. M. 1970. Vibration syndrome. Br. J. Ind. Med. 21:19.
23. Pramatarov, I. 1965. Clinical picture of vibration sickness in miners in the Rhodype mining region. Sar. Med. 16:94.
24. Trantola, A. 1967. Complex vibration syndrome (Raynaud's phenomenon. Dupuytren's disease, microcysts of the navicular bone, and paroxsical atrial flutter). Folia Med. 1:184.
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SECTION XI

To assure safe and healthful working conditions for working men and women; . . . by providing medical criteria which will assure insofar as practicable that no employee will suffer diminished health, functional capacity, or life expectancy as a result of his work experience; . . .by providing for training programs to increase the number and competence of personnel engaged in the field of occupational safety and health; . . .

—Public Law 91-596, Occupational Safety and Health Act of 1970.

SOURCES OF CONSULTATION

William R. Lee, M.L.S.

Following a review of the pertinent literature, the investigator may still feel the need for expert consultation owing to the often difficult task of identifying an occupational etiology with a patient's illness. This section identifies various official and nonofficial agencies to which the health professional may turn for advice on occupational health. It includes international organizations, Federal and other national agencies, state and local agencies, and schools of public health.

There are certain nonofficial agencies and organizations not given here, in particular insurance companies (especially those issuing workers' compensation insurance policies) and professional groups engaged in private consultation. Information about these latter organizations may be found under Directories in the Reference Aids portion of this section.

For a complete listing of state and local sources, refer to NIOSH's annual *Directory of Governmental Occupational Safety and Health Personnel* which gives a complete listing of state and local agencies.

INTERNATIONAL ORGANIZATIONS

Commission of the European Communities. 23-27 Avenue de la Joyeuse Entree, Brussels, Belgium.

Czechoslovak Committee of MAC, Jaroslav Teisinger, M.D., Director Ustav Hygieny, Prace a Chorob z Povolani Vinohrady, Srovarova 48 Prague 10, Czechoslovakia.

International Commission of Radiological Protection (ICRP). Radiofysiska Institutionen, Stockholm 60, Sweden.

International Ergonomics Association (IEA). Clanbusstrasse 25, Zurich, Switzerland.

International Occupational Safety and Health Information Centre (CIS). International Labour Office. 154 Rue de Lausanne, 1211 Geneva 22, Switzerland.

International Organization for Standardization (ISO). 1, Rue de Varembe, Geneva, Switzerland.

International Radiation Protection Association (IRPA). Oak Ridge National Laboratory, Oak Ridge, Tennessee 37830.

Permanent Commission and International Association on Occupational Health. Clinica del Lavoro, Via San Barnaba, Milan, Italy.

World Health Organization (WHO). Avenue Appia, Geneva, Switzerland.

NATIONAL ORGANIZATIONS IN THE UNITED STATES

Air Pollution Control Association. 4450 Fifth Avenue, Pittsburgh, Pennsylvania 15213.

The American Academy of Occupational Medicine. 150 North Wacker Drive, Chicago, Illinois 60606.

American Association of Occupational Health Nurses, Inc. (AAOHN). 79 Madison Avenue, New York, New York 10016.

American Board for Occupational Health Nurses, Inc. (ABOHN). 521 W. Westfield Avenue, Roselle Park, New Jersey 07204.

American Chemical Society. 1155 Sixteenth Street, N.W., Washington, D.C. 20036.

The American College of Preventive Medicine. Bryn Mawr, Pennsylvania 19010.

American Conference of Governmental Industrial Hygienists. P.O. Box 1937, Cincinnati, Ohio 45201.

American Hospital Association (AHA). 840 North Lake Shore Drive, Chicago, Illinois 60611.

American Industrial Hygiene Association. c/o William E. McCormick, Managing Director. 66 So. Miller Road, Akron, Ohio 44313.

American Medical Association. 535 North Dearborn Street, Chicago, Illinois 60610.

American National Standards Institute. 1430 Broadway, New York, New York 10018.

American Nurses' Association (ANA). 2420 Pershing Road, Kansas City, Missouri 64108.

American Occupational Medical Association. 150 North Wacker Drive, Chicago, Illinois 60606.

American Public Health Association. 1015 Eighteenth Street, N. W., Washington, D.C. 20036.

American Society for Testing and Materials. 1916 Race Street, Philadelphia, Pennsylvania 19103.

American Society of Tropical Medicine and Hygiene. P.O. Box 15208 Emory University Branch, Atlanta, Georgia 30333.

Chemical Industry Institute of Toxicology. Research Triangle Park, North Carolina 27709.

Industrial Health Foundation, Inc. 5231 Centre Avenue, Pittsburgh, Pennsylvania 15232.

Manufacturing Chemists' Association, Inc. 1825 Connecticut Avenue, N.W., Washington, D.C. 20009.

National Safety Council. 425 N. Michigan Avenue, Chicago, Illinois 60611.

Society of Toxicology. Robert A. Scala, Secretary. Medical Research Division, Esso Research and Engineering Company, P.O. Box 45, Linden, New Jersey 07036.

SCHOOLS OF PUBLIC HEALTH IN THE UNITED STATES AND CANADA

Columbia University School of Public Health and Administrative Medicine. 600 West 168th Street, New York, New York 10032.

Harvard University School of Public Health. 55 Shattuck Street, Boston, Massachusetts 02115.

Johns Hopkins University School of Hygiene and Public Health. 615 North Wolfe Street, Baltimore, Maryland 21205.

Loma Linda University School of Public Health. Loma Linda, California 92354.

Tulane University School of Public Health and Tropical Medicine. 1430 Tulane Avenue, New Orleans, Louisiana 70112.

University of California at Los Angeles School of Public Health. Los Angeles, California 90024.

University of California School of Public Health. Earl Warren Hall, Berkeley, California 94720.

University of Hawaii School of Public Health. 1860 East West Road, Honolulu, Hawaii 96822.

University of Illinois at the Medical Center, School of Public Health. P. O. Box 6998, Chicago, Illinois 60680.

University of Massachusetts, School of Health Sciences. Amherst, Massachusetts 01002.

University of Michigan, School of Public Health. Ann Arbor, Michigan 48104.

University of Minnesota School of Public Health. 1325 Mayo Memorial Building, Minneapolis, Minnesota 55455.

University of North Carolina School of Public Health. Chapel Hill, North Carolina 27514.

University of Oklahoma School of Health. 800 North East 13th Street, Oklahoma City, Oklahoma 73106.

University of Pittsburgh Graduate School of Public Health. Pittsburgh, Pennsylvania 15213.

University of Puerto Rico School of Public Health. Medical Sciences Campus, GPO Box 5067, San Juan, Puerto Rico 00905. (Teaching in Spanish.)

University of Texas at Houston, School of Public Health. P. O. Box 20186, Astrodome Station, Houston, Texas 77025.

University of Toronto School of Hygiene. Toronto 5, Ontario, Canada.

University of Washington School of Public Health and Community Medicine. F 356d Health Sciences Building, Mail Drop SC-30, Seattle, Washington 98195.

Yale University Department of Epidemiology and Public Health. School of Medicine, 60 College Street, New Haven, Connecticut 06510.

STATE AGENCIES

Agencies designated as of 1977 to administer state activities under the Occupational Safety and Health Act

Alabama
Alabama State Department of Labor
600 Administrative Building
64 North Union Street
Montgomery, Alabama 36104

Alaska
Department of Labor
Post Office Box 1149
Juneau, Alaska 99801

American Samoa
Director
Department of Manpower Resources
Pago Pago, American Samoa 96920

Arizona
Occupational Safety and Health Division
Industrial Commission of Arizona
P.O. Box 19070
Phoenix, Arizona 85005

Arkansas
Department of Labor
Capitol Hill Building
Little Rock, Arkansas 72201

California
Agriculture and Services Agency
1220 N Street, Room 144
Sacramento, California 95814

Colorado
Department of Labor and Employment
200 East Ninth Avenue
Denver, Colorado 80203

Connecticut
Connecticut Department of Health
79 Elm Street
Hartford, Connecticut 06115

Connecticut Department of Labor
200 Folly Brook Boulevard
Wethersfield, Connecticut 06109

Delaware
Department of Labor
801 West Street
Wilmington, Delaware 19899

District of Columbia

Gov't. of the District of Columbia
Minimum Wage and Industrial Safety Board
2900 Newton St., N.E.
Washington, D.C. 20018

Florida

Division of Labor
Ashley Building
1321 Executive Center Drive, East
Tallahassee, Florida 32301

Guam

Occupational Safety and Health
Government of Guam
P.O. Box 2950
Agana, Guam 96910

Hawaii

Director of Labor and Industrial Relations
825 Mililani Street
Honolulu, Hawaii 96813

Idaho

Department of Labor and Industrial Services
Industrial Administration Building
317 Main Street
Boise, Idaho 83702

Illinois

Department of Labor
910 South Michigan Avenue, 18th Floor
Chicago, Illinois 60605

Illinois Industrial Commission
160 North LaSalle Street
Chicago, Illinois 60601

Indiana

Indiana Division of Labor
Indiana State Office Bldg., Rm. 1013
100 North Senate Avenue
Indianapolis, Indiana 46204

Iowa

Bureau of Labor, State House
East 7th and Court Avenue
Des Moines, Iowa 50319

Kansas

Department of Labor
401 Topeka Avenue
Topeka, Kansas 66603

Kentucky

Kentucky Department of Labor
State Office Building Annex
Frankfort, Kentucky 40601

Louisiana

Department of Labor
Post Office Box 44063
Baton Rouge, Louisiana 70804

Louisiana Health and Human Resources Administration
Division of Health
c/o Charity Hospital — Advisory Board Office
1532 Tulane Avenue
New Orleans, La. 70112

Maine

Department of Manpower Affairs
20 Union Street
Augusta, Maine 04330

Maryland

Department of Licensing and Regulation
Division of Labor and Industry
203 E. Baltimore Street
Baltimore, Maryland 21202

Massachusetts

Department of Labor and Industries
Leverett Saltonstall Office Building
100 Cambridge Street
Boston, Massachusetts 02202

Michigan

Michigan Department of Labor
300 East Michigan Avenue
Lansing, Michigan 48926

Department of Public Health
3500 North Logan Street
Lansing, Michigan 48914

Minnesota

Department of Labor and Industry
5th Floor, Space Center Building
444 Lafayette Road
St. Paul, Minnesota 55101

Mississippi

State Board of Health
Post Office Box 1700
Jackson, Mississippi 39205

Missouri

Division of Labor Standards
P.O. Box 449
Jefferson City, Missouri 65101

Montana

Workmen's Compensation Division
815 Front Street
Helena, Montana 59601

Nebraska

Department of Labor
State Capitol
Lincoln, Nebraska 68509

Nevada

Department of Occupational Safety
and Health
515 East Musser Street
Carson City, Nevada 89701

New Hampshire

Department of Labor
1 Pillsbury Street
Concord, New Hampshire 03301

Occupational Health Service
Bureau of Occupational Health
Hazen Drive
Concord, New Hampshire 03301

New Jersey

Department of Labor and Industry
P.O. Box 5
Trenton, New Jersey 08625

New Mexico

Environmental Improvement Agency
P.O. Box 2348
Sante Fe, New Mexico 87501

New York

Department of Labor
State Campus
Albany, New York 12226

North Carolina

Department of Labor
Post Office Box 27407
11 West Edenton Street
Raleigh, North Carolina 27611

North Dakota

Workmen's Compensation Bureau
State Capitol
Bismarck, North Dakota 58501

Ohio

Division of Occupational Safety and Health
Department of Industrial Relations
2323 W. Fifth Avenue
Columbus, Ohio 43204

Oklahoma

Commissioner of Labor
State of Oklahoma
State Capitol, Room 5
Oklahoma City, Oklahoma 73105

Oregon

Workmen's Compensation Board
Labor and Industries Building
Salem, Oregon 97310

Pennsylvania

Department of Labor and Industry
1700 Labor and Industry Building
Harrisburg, Pennsylvania 17120

Environmental Resources
519 South Office Building
Harrisburg, Pennsylvania 17120

Puerto Rico

Secretary of Labor
Commonwealth of Puerto Rico
414 Barbosa Avenue
San Juan, Puerto Rico 00917

Rhode Island

Division of Occupational Safety
Department of Labor
235 Promenade Street
Providence, Rhode Island 02903

South Carolina

Department of Labor
3600 Forest Drive
Post Office Box 11329
Columbia, South Carolina 29211

South Dakota

State Department of Health
Office Building Number 2
Pierre, South Dakota 57501

Tennessee

Tennessee Department of Labor
C1-100 Cordell Hull Building
Nashville, Tennessee 37219

Tennessee Department of Public Health
344 Cordell Hull Building
Nashville, Tennessee 37219

Texas

Texas State Department of Health and State Safety Engineer
Texas Occupational Safety Board
1100 West 49th Street
Austin, Texas 78756

Utah

Industrial Commission
350 East Fifth South
Salt Lake City, Utah 84111

Vermont

Department of Labor and Industry
Vermont State Office Building
Montpelier, Vermont 05602

Virgin Islands

Division of Occupational Safety and Health
Department of Labor
Government Complex Building 2
Lagoon Street, Room 207
Fredriksted, St. Croix, V.I. 00840

Virginia

Department of Labor and Industry
Post Office Box 1814
Ninth Street Office Building
Richmond, Virginia 23214

State Department of Health
James Madison Building
109 Governor Street
Richmond, Virginia 23219

Washington

Department of Labor and Industries
General Administration Building
Olympia, Washington 98504

West Virginia

West Virginia Department of Health
State Capitol
Charleston, West Virginia 25305

West Virginia Department of Labor
State Capitol
Charleston, West Virginia 25305

West Virginia Insurance Commission
State Capitol
Charleston, West Virginia 25305

Wisconsin

Department of Industry, Labor and Human Relations
201 E. Washington Avenue
Post Office Box 2209
Madison, Wisconsin 53701

Wyoming

Occupational Health and Safety Department
200 E. 8th Avenue, P.O. Box 2186
Cheyenne, Wyoming 82002

UNITED STATES FEDERAL AGENCIES

Atomic Energy Commission, Division of Biomedical and Environmental Research. See Nuclear Regulatory Commission.

Department of Commerce.
- National Bureau of Standards. Washington, D.C. 20234.
- National Technical Information Service (NTIS). 5285 Port Royal Road, Springfield, Virginia 22151.

Department of Health, Education, and Welfare

Public Health Service.
- Center for Disease Control. Atlanta, Georgia 30333.
  - National Institute for Occupational Safety and Health (NIOSH). Parklawn Building, 5600 Fishers Lane, Rockville, Maryland 20852; also located at 4676 Columbia Parkway, Cincinnati, Ohio 45226 and 944 Chestnut Ridge Road, Morgantown, West Virginia 26505.
- Food and Drug Administration.
  - Bureau of Radiological Health. 12720 Twinbrook Pkwy. Rockville, Md. 20852.
  - National Clearinghouse for Poison Control Centers. 5401 Westbard Ave., Bethesda, Md. 20016.

Health Resources Administration
- National Center for Health Statistics. 330 Independence Avenue, S.W., Washington, D.C. 20201.

National Institutes of Health.
- National Cancer Institute. Bethesda, Maryland 20014.
- National Clearinghouse for Mental Health Information (NCMHI),
- National Institute of Mental Health. 5600 Fishers Lane, Rockville, Maryland 20852.
- National Heart and Lung Institute. Bethesda, Maryland 20014.
- National Institute of Environmental Health Sciences (NIEHS). P.O. Box 12233, Research Triangle Park, North Carolina 27709.
- National Library of Medicine. 8600 Rockville Pike, Bethesda, Maryland 20014.

Department of the Interior.

Bureau of Mines. C Street between Eighteenth and Nineteenth Streets, N.W., Washington, D.C. 20240.

Mining Enforcement and Safety Administration. C Street between Eighteenth and Nineteenth Streets, N.W., Washington, D.C. 20240.

Department of Labor.

Bureau of Labor Statistics. 200 Constitution Ave., N.W., Washington, D.C. 20210.

Occupational Safety and Health Administration. 200 Constitution Ave., N.W., Washington, D.C. 20210.

Environmental Protection Agency.

Air and Water Programs Office. 401 M Street, S.W., Washington, D.C. 20460.

Pesticides Programs Office. 401 M. Street, S.W., Washington, D.C. 20460.

Radiation Programs Office. Rockville, Maryland 20852.

Water Program Operations Office. 401 M Street, S.W., Washington, D.C. 20460.

Air Pollution Technical Information Center (APTIC), Office of Technical Information and Publications (OTIP), Air Pollution Control Office (APCO). P. O. Box 12055, Research Triangle Park, North Carolina 27709.

National Environmental Research Center. Research Triangle Park, North Carolina 27711 (also located in Cincinnati, Ohio 45268; Corvallis, Oregon 97330; and Las Vegas, Nevada 89114).

Western Environmental Research Laboratory. P. O. Box 15027, Las Vegas, Nevada 89114.

National Aeronautics and Space Administration. 400 Maryland Ave. SW, Washington, D.C. 20546.

National Council for Radiation Protection and Measurements (NCRP). 7610 Woodmont Ave., S.E. 1016, Washington, D.C. 20014.

National Referral Center, Library of Congress, Science and Technology Division. 10 First Street, N.W., Washington, D.C. 20540.

Nuclear Regulatory Commission, 1717 H Street NW, Washington, D.C. 20555.

Smithsonian Science Information Exchange (SSIE), Smithsonian Institution. 1730 M Street, N.W., Washington, D.C. 20036.

U.S. Government Printing Office. Washington, D.C. 20402.

REFERENCE AIDS

William R. Lee, M.L.S.

The occupational health reference aids listed in this section supplement the references appended to other sections. The material includes indexes, abstract journals, occupational health journals, bibliographies, and texts.

ABSTRACTS

Abstracts on Health Effects of Environmental Pollutants (HEEP). BioSciences Information Service of Biological Abstracts, 2100 Arch Street, Philadelphia, Pennsylvania 19103. A monthly abstract journal for current information on environmental pollution, including occupational health. 1972—

Abstracts on Hygiene, formerly Bulletin of Hygiene. Bureau of Hygiene and Tropical Diseases, Keppel Street, London WC1 E 7HT, England. A monthly abstract journal of the world literature; includes a section on occupational hygiene. 1926—

Air Pollution Abstracts, formerly NAPCA Abstract Bulletin. Environmental Protection Agency, Office of Air Programs, Air Pollution Technical Information Center (APTIC), Research Triangle Park, North Carolina 27711. A monthly abstract bulletin reporting technical literature recently accessioned by the APTIC. 1970—

Analytical Abstracts, dealing with all branches of analytical chemistry. Society of Analytical Chemistry, Cambridge, England. Monthly with annual author and subject indexes; includes: general analytical chemistry, inorganic analyses, organic analyses, biochemistry, general techniques, and apparatus. 1954—

Biological Abstracts. BioSciences Information Service of Biological Abstracts, 2100 Arch Street, Philadelphia, Pennsylvania 19103. A semimonthly with annual index, it provides abstracts of more than 140,000 research papers derived from approximately 7600 journals from some 100 countries. 1927—

Bioresearch Today, Industrial Health and Toxicology. BioSciences Information Service of Biological Abstracts, 2100 Arch Street, Philadelphia, Pennsylvania 19103. A monthly current awareness publication containing selected abstracts from two consecutive issues of Biological Abstracts. 1972—

CIS Abstracts, replacing CIS Cards (1960-73) and Occupational Safety and Health Abstracts (1963-1973). International Occupational Safety and Health Information Centre (CIS), International Labour Office, Geneva, Switzerland. Abstracts between 30,000 and 40,000 articles, books, regulations, proceedings, etc., on all aspects of occu-

pational safety and health with items grouped under 40 headings in the twelve annual issues. 1974—

Chemical Abstracts, Key to the World's Chemical Literature. American Chemical Society, Ohio State University, Columbus, Ohio 43210 and 1155 16th Street, NW, Washington, D.C. 20036. Published biweekly with indexes issued annually with five and ten year cumulations. 1907—

Digest of Neurology and Psychiatry. Institute of Living, Retreat Avenue, Hartford, Connecticut 06106. Ten issues annually covering literature in psychiatry, neurology, and their allied fields, including occupational psychiatry. 1932—

Environment Information Access. Environment Information Center, Inc., 124 East 39th Street, New York 10016. A semimonthly reference and research service for environmental affairs. 1971—

Ergonomics Abstracts. Ergonomics Information-Analysis Centre, University of Birmingham, England. A quarterly informative abstracting service; includes book reviews, classified index, and an index of applications. 1968—

Excerpta Medica. The International Medical Abstracting Service, Nassau Building, 228 Alexander Street, Princeton, New Jersey 08540. Forty separate monthly abstract journals covering the international literature in most areas of medicine and health.
Section 17, Public Health, Social Medicine and Hygiene. 1955-
Section 35, Occupational Health and Industrial Medicine. 1971-
Section 46, Environmental Health and Pollution Control. 1971—

Government Reports Announcements, continues US Government Research and Development Reports. National Technical Information Service (NTIS), US Department of Commerce, Springfield, Virginia 22151. A semimonthly listing of technical reports from government-sponsored research made available to industry and the general public. An index is published separately. 1946—

Industrial Hygiene Digest. Industrial Health Foundation, Inc., 5231 Centre Avenue, Pittsburgh, Pennsylvania 15232. A monthly abstract journal selecting articles from over 500 journals on environmental and occupational health. 1937—

Nuclear Science Abstracts. US Atomic Energy Commission, Division of Technical Information Extension, Box 62, Oak Ridge, Tennessee 37830. Issued semimonthly with a five year cumulative index. Includes a patent index. 1947—

Pesticides Abstracts (Pestab), formerly Health Aspects of Pesticides Abstract Bulletin (HAPAB). US Environmental Protection Agency, 401 M Street SW, Washington, D.C. 20460. Treats health aspects of pesticides in humans and animals; poisoning treatment; pesticide residue analysis and monitoring.

Safety in Mines Abstracts. Safety in Mines Research Establishment, Department of Trade and Industry, Sheffield, England. Bimonthly references and abstracts; includes author and subject indexes. 1952—

Thermal Abstracts. Heating and Ventilating Research Association,

Bracknell, Berkshire, England. Includes several sections covering heating services, noise and vibration, instrumentation, building materials, structures, and processes. Includes annual subject and author indexes. 1966—

BIBLIOGRAPHIES

Bibliography of Occupational Health; Occupational Health and Related Publications from the Public Health Service. U.S. Public Health Service Division of Occupational Health, Washington, D.C. Published between 1953 and 1963 this source list continues Hyslop and Gafafer (below).

Bibliography of Occupational Medicine. International Labour Office, Geneva, Switzerland. Supersedes its Bibliography of Industrial Hygiene. 1948—

CIS Bibliography. International Occupational Safety and Health Information Centre, International Labour Office, Geneva, Switzerland. 1962?—

Department of Environmental, Public, and Occupational Health of the American Medical Association: Publications List. The Association, 535 North Dearborn Street, Chicago, Illinois 60610. Continues Publications of the Council on Occupational Health. Revised periodically. 1972.

Gauvain, S.: Occupational Health, a guide to sources of information. Society of Occupational Medicine at the Royal College of Surgeons, Lincoln's Inn Fields, London, W.C.2. Gives both bibliographical and directory information in the field; organized by subject. 1968.

Hyslop, F.L., Gafafer, W.M., Bibliography of Occupational Health; Occupational Health and Related Publications from the Public Health Service, 1909-1953, Public Health Bibliography Series No. 9, Public Health Service Publication No. 300. 1954.

NLM Current Catalog. National Library of Medicine, 8600 Rockville Pike, Bethesda, Maryland 20014. A quarterly, annually cumulated bibliographic listing of publications catalogued by the Library; includes subject and name sections. Continues: The National Library of Medicine Catalog (1955-1965), The Armed Forces Medical Library Catalog (1950-1954), and ultimately that portion of the Index-Catalogue of the Library of the Surgeon General's Office (1880-1961) devoted to the catalog citations of monographic literature.

Occupational Health Bookshelf, selected references as recommended by the Industrial Medical Association. Journal of Occupational Medicine, v. 10, No. 8, August 1968, pp. 414-422.

A Review of Mental Health in Industry, Based on a Survey of the Literature. U.S. National Institute of Mental Health, Community Services Branch. 1958.

Registry of Toxic Effects of Chemical Substances, formerly Toxic Substances List. National Institute for Occupational Safety and Health,

Rockville, Maryland 20852. An annual ready reference list of potentially toxic chemicals found in the workplace. Includes references to the most important literature.

DATA SHEETS, GUIDES, MANUALS

AIHA Analytical Guides, previously sold as Analytical Abstracts. American Industrial Hygiene Association, 66 South Miller Road, Akron, Ohio 44313. A convenient source of information on analytical methods related to the field of industrial hygiene chemistry.

AIHA Hygienic Guide Series. American Industrial Hygiene Association, 66 South Miller Road, Akron, Ohio 44313. Separate data sheets on specific substances giving hygienic standards, properties, industrial hygiene practice, specific procedures, and references.

"ANSI Standards," Z37 Series, Acceptable Concentrations of Toxic Dusts and Gases. American National Standards Institute, 1430 Broadway, New York 10018. These Guides represent a consensus of interested parties concerning minimum safety requirements for the storage, transportation, and handling of toxic substances and are intended to aid the manufacturer, the consumer, and the general public.

ASTM Standards with Related Material. American Society for Testing and Materials, 1916 Race Street, Philadelphia, Pennsylvania 19103. Annual.

CIS Information Sheets. International Occupational Safety and Health Centre (CIS), International Labour Office, Geneva, Switzerland. Pamphlets on general or specific occupational safety and health topics; published irregularly.

Data Sheets. Manufacturing Chemists' Association, 1825 Connecticut Avenue, NW, Washington, D.C. 20009. Includes information on properties, hazards, handling, storage, hazard control, employee safety, medical management, etc. of specific chemicals. Thus far about 100 data sheets have been compiled.

Industrial Safety Data Sheets. National Safety Council, 425 North Michigan Avenue, Chicago, Illinois 60611. Information and recommendations regarding the safe handling of chemicals and safe practices in the work environment.

NIOSH Manual of Analytical Methods. National Institute for Occupational Safety and Health, 4676 Columbia Parkway, Cincinnati, Ohio 45226. These are the methods that chemists in the NIOSH Physical and Chemical Analysis Branch have used for industrial hygiene analysis (either found in the literature or developed by the Branch). 1974.

Safety Guides. Manufacturing Chemists' Association, 1825 Connecticut Avenue, NW, Washington, D.C. 20009. Information and recommendations pertaining to safe practices in the work envrionment.

TLVs, Threshold Limit Values for Chemical Substances and Physical

Agents in the Workroom Environment... American Conference of Governmental Industrial Hygienists, P.O. Box 1937, Cincinnati, Ohio 45201. Annual; threshold limits based on information from industrial experience, experimental human and animal studies, intended for use in the practice of industrial hygiene.

DIRECTORIES

American Industrial Hygiene Association Membership Book. AIHA, 66 South Miller Road, Akron, Ohio 44313. Annual.

American Occupational Medical Association Membership Directory. AOMA, 150 N. Wacker Drive, Chicago, Illinois 60606. Annual.

Chemical Guide to the United States, Seventh Edition. Noyes Data Corporation, Park Ridge, New Jersey. Describes over 400 of the largest US chemical firms with index to companies; no subject index. 1973.

Directory of Chemical Producers. Stanford Research Institute, 855 Oak Grove, Menlo Park, California 94025. Four volumes published continuously on a quarterly installment basis listing a total of 1,600 chemical producers and 10,000 individual commercial chemicals, arranged alphabetically by company, product, and region.

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