REPORT 4

PULP AND PAPER MILLS

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16. Abstract (Limit: 200 words) The various hazards present in the many steps used in the production of products from pulp and paper mills were reviewed and discussed. The biological effects of 43 chemical, physical and dust hazards were detailed in this report. Dust hazards included exposures to wood dust, mold and bagasse dusts and fibrogenic dusts. Physical hazards included high heat and humidity, and noise. Raw materials and chemical intermediates discussed included calcium-oxide (1305788), magnesium-oxide (1309484), pulping liquors, sodium-hydroxide (1310732), sulfate, sulfites, sulfides, sulfur (7704349) and sulfuric-acid (7664939). Pulp bleaching agents were discussed along with papermaking additives, contaminants and/or byproducts, and pulping or combustion effluents. Sampling and analytical techniques for physical and chemical hazards were discussed. Engineering controls for hazards in pulp and paper mills were reviewed. OSHA regulations governing pulp and paper mills were evaluated.

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PREFACE

Report 4 on Pulp and Paper Mills revises and updates the material in Report 3, and presents additional material.

Report 3, which was the rough draft of Chapter II, Biologic Effects of Exposure, included a discussion of the biologic effects of 35 distinct chemical, physical, and dust hazards found in pulp and paper mills. For those hazards for which NIOSH criteria documents were available (about half), relevant material from the documents was summarized. Computer and manual searches were performed for primary literature available since the publication of the criteria documents. The relevant literature was acquired and discussed in Report 3 and in the present report. For hazards for which no NIOSH criteria document was available, standard reference material on biologic effects was discussed as well as primary literature review articles, where available.

In addition to the changes in Report 3 suggested by the NIOSH criteria manager and the updated material on biologic effects (including carcinogenicity and epidemiologic studies), Report 4 contains the following additional material:

- information on biologic effects of these additional hazards: aluminum sulfate (alum), hydrazine, kaolin, kerosene, magnesium oxide, and rosin
- information on biologic effects of noise, heat, and humidity

- sampling data from OSHA inspections from 1972-1978 listing hazards sampled in SIC 2611, 2621, 2631, and 2661 that exceeded OSHA standards
- a discussion of engineering controls for physical hazards in pulp and paper mills, particularly material dealing with noise-reduction techniques
- a preliminary evaluation of OSHA regulations governing pulp and paper mills (29 CFR 1910.261)
- a preliminary discussion of selected sampling and analytical techniques for physical and dust hazards and a tabular summary of relevant NIOSH sampling and analytical techniques for some chemical hazards found in pulp and paper mills.

Revision of previous material includes: (a) new employment figures for the relevant SIC codes for 1978, (b) comparison of the incidence of illnesses and injuries producing lost workdays in the pulp and paper industry with those in other SIC 2-digit manufacturing industries, (c) new injury, illness, and accident information, and (d) a new biologic effects format where uses and properties of hazards are integrated with biologic information.

I. INTRODUCTION AND OVERVIEW

The manufacture of pulp and paper is one of the world's oldest and largest industries. According to the 1980 Kline Guide to the Paper Industry (4th edition), paper and allied products is the ninth largest manufacturing industry in the United States, accounting for nearly 3.5% of the value of all manufacturing (NO3293).

Production of paper and paperboard in the US in 1978 was 62.1 million tons, with an estimated increase to 64.3 tons in 1979 (NO3293). Production capacity of 75 million tons by 1982 is forecast by the American Paper Institute (NO2259). In 1978, the US produced 34.5% of the world's paper and paperboard supply and consumed 38% (about 642 pounds per person). By comparison, the next largest per person consumption of paper in the world was 425 pounds, in Canada (NO3293).

There are many different manufacturing processes and industrial operations associated with the manufacture of paper from pulp. These range from debarking logs in the wood yard of a pulp mill to winding, cutting, and packaging paper after it is formed from pulp on a papermaking machine. Physical hazards, such as noise, high heat, and high humidity, and dust hazards are present in many operations in pulp and paper mills.

Each year, millions of tons of chemicals are used in pulping, bleaching, papermaking, and conversion processes. The pulp and paper

industry is the sixth largest consumer of chemicals, preceded only by the chemical industry and the textile, rubber and plastics fabricating, agricultural, and petroleum refining industries (NO3293). About one-quarter ton of chemicals is used in the manufacture of every ton of paper and paperboard. The major chemicals used are inorganic chemicals such as sodium sulfate for pulping, chlorine and sodium hydroxide for bleaching, and mineral pigments, binders, fillers, resins, and specialty chemicals for papermaking (NO3293).

Literally hundreds of chemicals are used as additives in papermaking. A survey of the individual biologic effects of these agents would be an unprecedented undertaking. The extent to which various additives are used throughout the pulp and paper industry is not fully known; use of additives depends upon the specific type of paper product produced. Information on uses of chemical agents is presented in this report along with data on their biologic effects.

The uses and biologic effects of 43 chemical, physical, and dust hazards are discussed in this report. These hazards are listed in Table VI-1 and were selected on the basis of (a) their use or presence in pulping and papermaking production operations, and (b) their hazard or toxic potential, as indicated by existence of criteria documents (22 agents), OSHA standards and hazard codes, and presence in the National Occupational Hazard Survey (NOHS) data base.

This report includes data on biologic effects extracted from these documents, as well as material obtained from primary literature and

review articles that were published after the criteria documents were written. For hazards for which no criteria documents exist, material has been obtained from standard references and available primary literature.

The NOHS data base contains nearly 1,000 chemical and physical hazards in the Standard Industrial Classification (SIC) codes comprising pulp and paper mills. The relevant SIC codes are 2611, 2621, 2631, and 2661. Although SIC 2611 (pulp mills) was not surveyed in the NOHS, potential pulping process hazards are included in SIC 2621, in which integrated pulp and paper mills are classified. Not all agents listed in Table VI-1 were found in the NOHS data base for SIC codes 2621, 2631, and 2661. For example, toxic effluents resulting from alkaline pulping processes, such as hydrogen sulfide and methyl mercaptan, did not appear in the NOHS data. The NOHS data are discussed further in the Extent of Exposure section.

Hazards will be grouped, for discussion of biologic effects, into the following categories:

(a) Dust hazards

- wood dust
- mold and bagasse dusts
- fibrogenic dusts

(b) Physical hazards

- heat and humidity (heat stress)
- noise

(c) Raw materials or chemical intermediates

- Calcium oxide
- magnesium oxide

- pulping liquors
- sodium hydroxide
- sulfates, sulfites, sulfides, and sulfur
- sulfuric acid

(d) Pulp bleaching agents

- chlorine
- chlorine dioxide
- sodium chlorate
- sodium hypochlorite

(e) Papermaking additives

- acrylamide
- alum
- carbon black
- formaldehyde and melamine- and urea-formaldehyde resins
- kaolin
- rosin
- silicates
- titanium dioxide
- zinc oxide

(f) Contaminants and/or byproducts

- dimethyl sulfoxide
- hydrazine
- kerosene
- polychlorinated biphenyls
- polynuclear aromatic hydrocarbons
- tall oil
- turpentine
- vanillin

(g) Pulping or combustion effluents

- carbon monoxide
- dimethyl disulfide
- dimethyl sulfide
- hydrogen sulfide
- methyl mercaptan
- sulfur dioxide.

For the above hazards, Table VI-2 presents (1) the OSHA exposure standard, (2) the recommended NIOSH exposure level described in the relevant criteria document, and (3) the American Conference of

Governmental Industrial Hygienists' (ACGIH) recommended exposure level (usually a threshold limit value--TLV).

Operation in confined spaces is a general industrial condition that may be hazardous to workers. The hazard, confined spaces, is included in Tables VI-1 and VI-2 for completeness, documenting the existence of recommended exposure conditions. Physical trauma, another physical hazard, is discussed in the subsection Sources of Exposure.

The remainder of this report discusses extent of exposure, uses and properties of specific hazards and their biologic effects in humans and animals, information on carcinogenicity, mutagenicity, teratogenicity, and reproductive effects, epidemiologic studies, and control of exposure.

II. EXTENT OF EXPOSURE

Pulp and paper mills are included under SIC codes 2611, 2621, 2631 and 2661. In 1979, there were 425 pulp mills, 730 paper and board mills, and 4,500 converting plants in the United States. A total of 659,000 workers were employed in the pulp and paper industry and 511,000 of these were production workers (NO3293).

Numbers of workers in pulp and papermills in 1978 and previous years are presented in Table VI-3. Paper mills, including integrated pulp and paper mills, employ the greatest number of these workers, followed by paperboard mills, pulp mills, and building paper and building board mills. The number of workers in pulp, paper, and paperboard mills has been increasing, after a low point in 1975-76. More variable employment patterns are characteristic of building paper and board mills.

National Occupational Hazard Survey (NOHS) Data

Examination and documentation of the chemical and physical agents in the pulp and paper mill industry has yielded two lists of agents. One list has resulted from an examination of the NOHS data base, which ranked nearly 1,000 chemical and physical hazards. A second list, Table VI-1, containing 43 separate hazards, has resulted from our review of the literature on the basis of the criteria discussed in the Introduction.

The 1972-1974 NOHS included visits to facilities in SIC Group 26, but only to facilities in three of the four SIC classifications under discussion in this report. SIC 2611 (pulp mills) was not included in the survey. Approximately 5,700 employees were observed during the NOHS walk-through survey. Eighty-nine occupations (coded according to Bureau of the Census criteria) were recorded. Fifty-one of the occupations were directly related to production or manufacturing process activities in pulp and paper mills; 29 occupations were nonprocess related; and 9 occupations were classified as miscellaneous. The occupations observed during the NOHS survey, stratified into the above-mentioned classes, are presented in Tables VI-4, VI-5, and VI-6.

The majority of observations recorded during the NOHS visits were for production workers (approximately 4,950 workers). About 520 workers were tallied in the nonproduction occupations. Observations recorded during the walk-through surveys included documentation of actual worker-identified exposures, and labeled or worker-identified chemical or physical agents. "Generic" agents, ie, agents presumed to be present in a trade-name product or manufacturing process, were not included in further examination of the NOHS data. Since 1974, when the original NOHS data base was established, at least 80% of trade-named products have been resolved into specific components or ingredients by NIOSH (written communication, Ken Kreitel, NIOSH-Cin, December 7, 1979).

The list of chemical and physical agents relevant to pulp and paper mills in the NOHS data base includes not only specific chemical and physical hazard names but general hazards, eg, waterless hand cleaner or

ink. As specified in the documentation for the NOHS protocol (N00957), the presence of water in the workplace was not recorded as a specific hazard; however, water does appear on the NOHS list because it was listed as a component of a trade-name product.

Using only the NOHS sample of production workers observed in industries under SIC codes 2621, 2631, and 2661, the number of persons exposed (the sum of actual and trade-name exposures) was divided by tne total number of production workers for a calculation of the percent of workers exposed. No extrapolation to the total exposed US worker population was attempted. The percentage of exposure in this sample was used to rank the agents for the production workers. Eight hundred and eignty-nine chemical and physical agents were recorded for the production workers in industries under SIC codes 2621, 2631, and 2661 (Table VI-7).

The list of 889 agents includes eight International Agency for Research on Cancer (IARC)-suspected animal or human carcinogens to which 5% or less of the workers were exposed, either as an actual exposure or as an ingredient in a trade-name product. The IARC-suspected carcinogens and the proportion of the surveyed production work force exposed to these agents are presented in Table VI-8.

In the entire employee population of industries surveyed under SIC Group No. 26, 15 carcinogens were identified. The identification was either by inclusion in IARC's classification of agents or from a classification according to OSHA's criteria for Class I or II carcinogens. Table VI-9 lists the carcinogens, the occupations, and the

industry (SIC four-digit classification) in which the exposure was noted. Additionally, the occupation is noted as either a production/process, nonprocess-related, or a miscellaneous occupation, and the number of carcinogens reported for each occupation is included.

When our list of major hazards or agents derived from the review of the literature (Table VI-1) was compared with the list of hazards or agents reported in the NOHS data base for the production workers in industries under SIC codes 2621, 2631 and 2661 (Table VI-7), only 29 of the 43 hazards were registered. Table VI-10 presents this hazard list for production workers. The hazard lists for nonproduction and miscellaneous workers are presented in Tables VI-11 and VI-12, respectively.

Continuous noise and potential (intermittent) noise were the most frequently reported hazards in the truncated list (48.8 and 20.5% of the workers exposed). Exposure to asbestos, the only carcinogen on the truncated list, was observed in only 5% of surveyed workers. The majority of the asbestos exposures were seen in different occupations in SIC 2661 (building paper and building board mills). Only Asbestos-and-Insulation Workers (Census Occupation Code 601) were exposed to asbestos in SIC 2621 (Table VI-9). No reports of asbestos exposure were noted for SIC Code 2631.

Sources of Exposure

Workers in pulp and paper mills are exposed to numerous hazards whose biologic effects will be considered in a subsequent section. General health hazards to which workers are exposed include:

- dusts
- explosions and fires
- noise and vibration
- hot, humid environments
- noxious odors
- physical trauma.

The various dusts to which workers may be exposed include wood dust (chipping, grinding, and debarking of logs; pulp digester loading) starch dust, cork dust, crepe dust, lime dust, alum dust, cotton dust, bagasse (sugar cane) dust, sulfur dust (sulfite mills), and dusts from pigments, molds, and particulates (NO2762, NO2396). Many of these dusts are present during the mixing of coating and filler materials which are added to paper.

Dust, in addition to its irritant and respiratory effects, may also be an explosion hazard. Explosions or fires have been reported in paper mills at black liquor recovery units in kraft mills, steam explosions at papermaking machines, air compressor explosions, and industrial gas explosions. Fires have resulted from mishandling of flammable liquids, electrical short circuits, and materials used in building board manufacture (NO2687, NO2688, NO2396).

Noise in pulp and paper manufacturing occurs within the 300-2400 Hz frequency band and has been associated with bark removal, gang saws, chippers, and grinders during wood preparation in the wood yard. In the pulp and paper mill, high noise levels have been associated with blowing of pulp digesters, black liquor evaporators, Jordan refiners, and the operation of papermaking machines (especially the couch roll on the Fourdrinier machine) (NO2692). These generalizations were confirmed in a plant site visit to a large-capacity bleached kraft integrated pulp and paper mill (NO3297). High noise levels were encountered in the wood yard (pulp chipping), pulp mill (digester blowing, refiner operation), and the paper mill (conversation near the Fourdrinier paper machine was possible only by shouting directly into someone's ear).

Heat stress in pulp and papermaking is a potential hazard due to the hot, wet processes used. Temperatures exceed 140 C throughout the papermaking process. The evaporation of large amounts of water during the drying of the paper sheet by steam-heated rolls adds water to the air. To a significant extent, quality control and production considerations limit the amount of heat and water in the drying process, as well as in earlier stages, and limit the release to the workplace environment. Elevated heat and humidity were also apparent during the plant site visit in the black liquor recovery area, bleaching area, and near the papermaking machines (NO3297). Drying rooms and pulp washing areas are other sources of excess heat and humidity (NO2396).

Major sources of malodorous emissions from sulfate pulping processes are black liquor recovery furnaces, direct contact evaporators, brown stock washers, knotter hoods, black liquor oxidation vents, condensate strippers, smelt dissolving tanks, and the lime kiln (NO2693, NO2696, NO2571). Malodorous emissions are usually reduced sulfur effluents of alkaline pulping operations, such as hydrogen sulfide, methyl and other mercaptans, dimethyl sulfide, and dimethyl disulfide (NO2571). Also, sulfur dioxide, as an effluent of both alkaline and acid pulping operations, may pose a health hazard (NO2683). Other effluents that may pose a health hazard include chlorine gas, chlorine dioxide, hypochlorite bleaching effluent, ammonia, and turpentine (NO2396).

Physical trauma is the result of exposure to hazards in pulp and paper mills. It includes injuries to limbs and other parts of the body by equipment, machinery or as the result of accidents, such as falls. Skin and mucous membrane irritation and damage produced by chemicals and physical hazards may also be termed physical trauma.

Accidents, Illnesses, and Injuries

(a) Comparison of Pulp and Paper Industry with Other Industries

Compared to other SIC 2-digit manufacturing industries, workers in the pulp and paper industry have a relatively high incidence rate of illnesses and injuries. Figure II-1 presents illness and injury incidence rates in 1978 for workers in all SIC 2-digit manufacturing industries, obtained from the Bureau of Labor Statistics (NO2500). Of

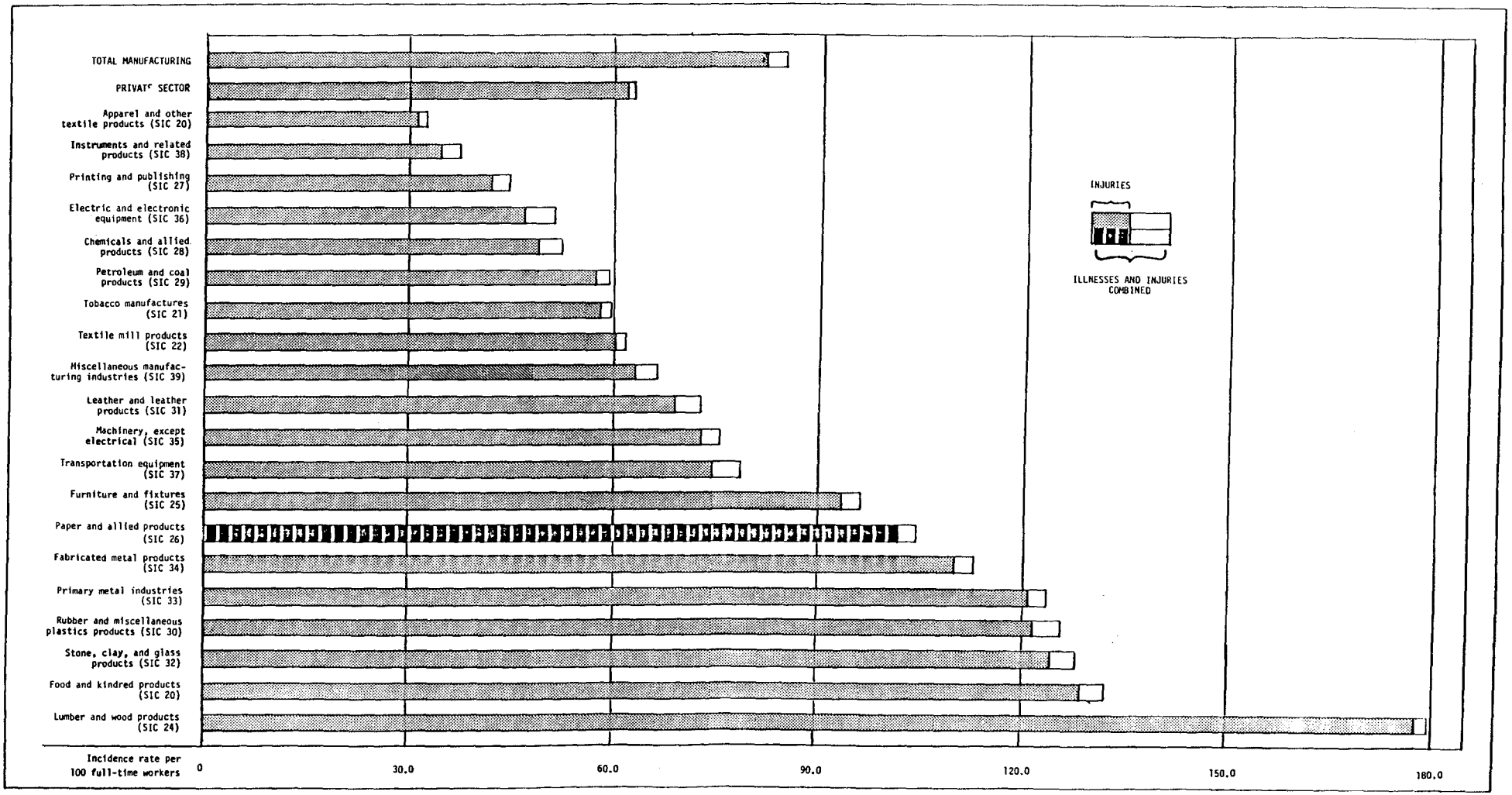

FIGURE II-1. OCCUPATIONAL INJURIES AND ILLNESSES IN US BY MANUFACTURING INDUSTRY, 1978

the 20 listed manufacturing industries, 7 industries' workers had a lost-workday incidence rate due to illnesses and injuries of greater than 100 lost workdays per 100 full-time workers. Workers in the paper and allied products industry are included in this group; in 1978, 103.3 lost workdays involving illnesses and injuries occurred for every 100 full-time workers in SIC 26. This represented an increase of 1.6% above the 1977 illness and injury incidence rate of 101.6 lost workdays (N02500).

Table VI-13 presents lost workday illness and injury incidence rates for workers in pulp and paper mills within SIC 26 for several previous years (N02499, N02500, N02764, N02766). These data indicate that workers in building paper and building board mills (SIC 2661) had the highest incidence rate for lost workdays from injuries and illnesses, although there were fewer workers in these mills than in pulp, paper, or paperboard mills. Over the period 1975-1978, workers in paper mills had the lowest injury and illness incidence rates of employees in the four SIC codes and these rates were appreciably below industry-wide incidence rates. There is no apparent reason for these differences in injury and illness rates in the various SIC codes related to pulp and paper mills.

Based on unpublished data from the Bureau of Labor Statistics, 50 fatalities occurred in the pulp and paper industry in 1976. Table VI-14 ranks 2-digit SIC manufacturing industries on the basis of fatal accidents or injuries and also on illness and injury rates causing lost workdays, for 1976. Eight of the 20 industries had a greater number of fatalities than the paper and allied products industry, while 6 of the

industries had a greater incidence of illnesses and injuries causing lost workdays. The position or ranking of the paper and allied products industry is similar to its position in 1978 relative to incidence of illnesses and injuries in manufacturing industries.

(b) Causes of Accidents in Pulp and Paper Mills

An examination of causes of fatal accidents in various industries in Oregon from 1966-1973 (NO2725) indicates that fatal accidents in pulp and paper mills represented approximately 2% of the total number of fatal accidents reported by the Workmen's Compensation Board of Oregon. Only 2 of the 10 fatal accidents in pulp and paper mills were due to chemical exposure (both deaths resulted from sulfur dioxide inhalation). The other fatalities resulted from physical trauma. Specific causes of death included: caught in winder, run over by log truck, broken neck during chip handling, struck by falling equipment, and a 32-foot fall.

In a paper published in 1974, Taylor and Gardner (NO2532) analyzed the causes of 86 explosions of chemical recovery furnaces in kraft pulping mills from 1948-1973. Hazards of this type were relatively frequent during this period (8 explosions in 1971), and most (69%) were due to smelt-water explosions. Although the use of instrument monitors and automatic controls could reduce the known causes of these explosions, unrecognized hazards related to the operation of these systems accounted for 27% of the smelt-water explosions.

Riley (NO2738) reported a study of the causes of 39 serious injuries to paper machine workers reported to the American Paper Institute during 1966-1978. The wet end of the machine accounted for 18% of the injuries, which included falling from an elevation (3 injuries) and encounters with (caught in or between) in-running roll nips (4 injuries). In the dryer area (20% of injuries), injuries occurred from breaking of dryer drums, being cut by "doctor blades" while removing material from rolls, and cuts and eye injuries from removing paper from the machine. Eighteen percent of the injuries occurred at the takeoff end of the paper machine, and 26% occurred while handling rolls of paper. Most of the latter injuries were due to the failure of employees to remove hands or fingers from crane hooks when hooking to a roll shaft. Four injuries were due to falling or slipping rolls of paper. Riley concluded that most of the accidents could have been prevented.

A preliminary examination of summaries of OSHA MIS accident data for 1972-1980 (NO3126) indicates that unspecified hazards, including unidentified physical and chemical hazards, accounted for 46% of the reported accident entries. In-depth descriptions of accidents and injuries in SIC codes 2611, 2621, 2631, and 2661 have been requested, as well as fatality reports for SIC 2-digit manufacturing industries.

OSHA Sampling Data

During the period from July 1972 through April 1980, OSHA conducted tests at about 1,265 sites in the pulp and paper industry for the

presence of various hazards. Approximately 11 different physical hazards, and 60 chemical hazards, were identified in SIC codes 2611, 2621, 2631, and 2661 and are listed in Table VI-15.

As shown in Table VI-15, physical hazards accounted for a disproportionate number of sites (and affected employees) where OSHA standards were exceeded. Noise was clearly the most prevalent hazard of all those exceeding OSHA standards, comprising 84% of the 403 sites sampled where OSHA standards for chemical or physical hazards were exceeded. At these sites, a total of 13,479 employees were affected by excessive noise, representing 91% of the 14,814 employees affected by hazards that exceeded OSHA standards.

Ranked on the basis of the number of employees affected at sites where OSHA standards were exceeded, the 10 most serious hazards were:

Hazard	No. of employees affected
Noise	13,479 (336 sites)
Electrical shock	854 (7 sites)
Improper illumination	100 (1 site)
Dusts	88 (23 sites)
Carbon monoxide	83 (2 sites)
High or low pressure	73 (70 sites)
Sulfur dioxide	39 (7 sites)
Chlorine	15 (2 sites)
Hydrogen sulfide	11 (2 sites)
Methyl ethyl ketone	7 (2 sites)

In many instances, sampling tests were carried out for these and other hazards at sites where the OSHA standard was not exceeded. This was especially true for some of the chemical hazards such as chlorine, carbon monoxide, and ammonia. Heat stress, a hazard which might be

expected to contribute to worker discomfort in various pulp and paper mill operations, was sampled at only five sites. Data from none of these tests exceeded OSHA standards.

Sampling and Analytical Techniques for Physical and Chemical Hazards

Measurement of worker exposures is an important industrial hygiene function. The data generated are used to evaluate the hazards to workers in their jobs, to measure the effectiveness of engineering controls, and to establish a data base for future evaluation of exposure standards.

An important consideration in the establishment of a sampling program must be the recognition that only a small number of the total exposures experienced by workers can be measured. Thus, the individual responsible for the sampling program must judiciously choose for sampling those jobs which represent the highest risk. The statistical evaluation of the results can be done according to the Handbook of Statistical Tests for Evaluating Employee Exposure to Air Contaminants (NIOSH, Publication No. 75-147) and the Occupational Exposure Sampling Strategy Manual (NIOSH, Publication No. 77-173).

Sampling methods can be categorized according to the type of device that is used to collect the sample. Sampling for solid materials present as dusts or fumes can be done using filters or impingers. Filters collect particles by electrostatic forces as well as mechanical filtration. Impingers collect particles by impacting the particles in a high velocity air stream on a plate submerged in a liquid. The liquid

may contain materials to react with the materials of interest, in order to eliminate losses due to degradation or to allow field analysis of the sample.

Gases and vapors are collected on solid sorbents or by bubblers. The materials are adsorbed onto the finely divided solid sorbents and can be stored for later analysis. In the case of bubblers, the gases are dissolved in the liquid medium and/or react with other materials. As with impinger samples, field analysis is possible.

Analytical methods cover the range of available techniques common to chemical analysis. Gas chromatography, atomic absorption, spectrophotometry, x-ray diffraction, and ion-specific electrodes are some of the techniques. Two principal concerns should be addressed in the analysis of the samples. First, adequate quality control procedures, including analysis of spiked samples, should be a regular part of the program. Second, the analyst should be alert to the possibility of interferences in field samples that are not likely to occur in laboratory-prepared samples.

Table VI-16 is a list of principal chemical hazards, including the sampling method and analytical procedure recommended by NIOSH, and work areas, processes, or equipment where workers are likely to be exposed to the materials.

It is important to note that there are many chemicals used in pulp and paper mills that are not included in Table VI-16. For the most part,

these chemicals are not used in significant quantities. For example, resin monomers may be present because of incomplete stripping of the resin, but are generally present at very low levels. In other cases, such as rosin sizing, little information on the chemicals exists.

For the physical hazards of noise and heat, devices have been developed commercially to measure these exposures. Noise exposure devices must meet the requirements of the American National Standards Institute (ANSI) for a Type II sound level meter. These requirements specify limits of instrument response to ensure comparability of data.

Heat stress can be measured with commercial instruments or homemade devices. A standard thermometer, a wet bulb thermometer, and a globe thermometer are all that are needed. In any measurement system, calibration of instruments is an important aspect of a complete study. All field devices should be checked before and after use for accurate calibration. All calibrations should be traceable to a primary standard. Records of calibration and checks become part of the data for a particular evaluation.

III. BIOLOGIC EFFECTS OF EXPOSURE

Uses and Biologic Effects of Physical and Chemical Hazards in Humans and Animals

This section discusses uses and biologic effects of the hazards listed in Table VI-1. The hazards are grouped into categories of use in manufacturing operations in the pulp and paper industry; material on uses and properties of each hazard or (where appropriate) group of hazards precedes the discussion of biologic effects. This material provides a context within which exposure data can be evaluated. Table IV-17 summarizes human and animal toxicity data for agents discussed in this section.

Most of the hazards are discussed in only one use category. An exception is asbestos. Asbestos is a dust hazard, and biologic effects of dust exposure are discussed in the section on physical hazards. Chrysotile asbestos is a form of silica used as an additive (filler) in papermaking, and therefore its biologic effects are discussed under the category of papermaking additives.

(a) Dust Hazards

Exposure to dusts is prevalent at various locations in the wood yard. Debarking logs, and cutting, grinding, and chipping operations

produce wood dust. The mixing of coating and filler materials which are added to paper also produces dusts which may pose a health hazard to workers.

(1) Wood Dust

Paper manufacturers mainly use wood pulp as a raw material. Wood pulp is obtained from coniferous and deciduous trees by grinding and/or chemical treatment. Wood dust is produced in the process of sawing the stems of trees into manageable lengths, or during the chipping of logs in preparation for chemical digestion.

A review of the literature on health hazards from wood dust identifies contact dermatitis, allergic respiratory reactions, systemic poisoning, and carcinoma of the nasal sinus as the major disease entities. However, they are not generally associated with all types of wood or with all operations involving exposure to wood dust.

Contact dermatitis, caused by both primary irritants and allergic sensitization, has been reported (N02774). Generally, the causes have been attributed to specific chemical components of the wood, although mechanical irritation of the skin by wood dust is possible. In the case of allergic sensitization, proposed chemical mediators are saponins and quinones (N02774). Types of wood identified inlcude western Red Cedar and cocabolla, which have produced contact dermatitis confirmed by patch test or intracutaneous injection (N02772). McCord identified 50 varieties of wood which can produce dermatitis (N02770).

Allergic respiratory reactions to a number of woods during inhalation studies have also been confirmed. Data indicating the production of wheal and flare responses to skin tests of wood extracts, and the production of reversible airway obstruction, suggest an immunoglobin E-mediated response (NO2775). In the case of cocabolla sensitivity, desensitization was was accomplished using a series of 18 injections of a 2% saline solution of wood dust (NO2774).

Cancer of the nasal sinus has been associated with exposure to wood dust. The bulk of these sources of exposure occur in the furniture industry. In England, adenocarcinoma of the nasal sinuses in woodworkers in the furniture industry was made a prescribed (compensable) disease in 1969. However, there is no firm evidence implicating specific varieties of wood with development of nasal adenocarcinoma.

Several reports discussed in the wood dust draft document (NIOSH, written communication, May 1980) have associated lung cancer and Hodgkin's disease with pulp and/or paper industries. Greene calculated a relative risk of 1.4 for the development of Hodgkin's disease for people with occupational exposure to wood and wood pulp. Decoufle identified cancer of the larynx, buccal cavity, pharynx, colon, and rectum as more common in paperworkers than lung cancer. This finding is notable, since lung cancer accounts for 20% of deaths due to cancer in white males, far more than any other cause, except for gastrointestinal cancer in older age groups. Milham found excess multiple myeloma and cancer of the small intestine using proportional mortality ratio (PMR) in pulp and paper workers. The studies cited above associating pulp and papermaking with

nasal cancers have been based on death certificate surveys or on a comparison of death rates between counties with or without papermaking industry. No studies have been identified which examine standard mortality ratios for workers in the pulp and paper industry when compared to other groups. This is in contrast to a number of studies from several parts of the world which have identified nasal sinus cancer patients as more likely to be furniture woodworkers than any other occupation.

Systemic poisoning effects from wood dust components have also been described, but specific varieties of wood produce different symptoms (NO2770). No studies have been identified which describe general systemic injury due to exposure to wood dust.

Hazards to workers in the pulp and paper industry from wood dust occur in those operations carried out before the wood is wetted. An important distinction between pulp and paper industries and the wood finishing industry is that wood finishing involves several operations, such as shaping, cutting, and sanding, which generate large amounts of fine wood dust. Thus, human effects associated with fine wood dust may not be as prevalent in the pulp and paper industry as in the wood finishing industry.

(2) Mold and Bagasse Dusts

Exposure to airborne biologic agents or their metabolic products can occur in pulp processing. The use of wood, bagasse, and cotton products is the source of these agents.

Molds are found in fresh and stored wood. A disease called wood pulp worker's disease has been described by Schleuter et al (NO2535). The illness is a hypersensitivity pneumonitis characterized by recurrent episodes of dyspnea, chills, and fever, usually occurring at night. Weight loss was reported. In both cases reported by Schleuter et al, the patients sought medical treatment, but the cause of the disease was not discovered for a number of years. The patients improved without specific therapy after prolonged periods (3-6 weeks) away from work. They responded specifically to inhalation challenge to an extract of the fungus, Alternaria. The challenge resulted in slight reactions immediately, with much more severe reactions occurring after 6-8 hours. Steroids were administered and signs and symptoms cleared in 24 hours. In both patients, permanent lung disability occurred, ascribed by the author to the delay in accurate diagnosis and continued exposure. Lung biopsies of both patients showed chronic interstitial pneumonitis in both cases.

Earlier, Emanual and coworkers (cited in NO2535) identified Cryptostroma corticale as the offending organism in a respiratory disease first described by Towey et al. In this case the spores were associated with maple logs. According to Schlueter et al (NO2535), the disease described by Towey et al was a granulomatous interstitial pneumonitis. Precipitating antibodies against the extract of the organism were present in sera of affected workers.

Another disease thought to be caused by a biologic agent is bagassosis. Bagasse, or dried sugar cane fiber, has been used in some

parts of the US where sugar cane is processed to manufacture paper. According to Lehrer et al (NO2555), Lacey demonstrated in 1971 that Thermoactinomyces sacchari produced antigens which were largely the etiologic factor in bagassosis. In a case described by Buechner (NO2704), the disease was initially characterized by severe cough, producing dark, mucoid sputum. Shortly, dyspnea and chest pain, aggravated by deep inspiration and coughing, appeared. Marked weakness, nausea, loss of appetite and weight, and chills were other sensations. On hospitalization the patient recovered with no specific therapy. The patient experienced similar and more pronounced symptoms about six months later after re-exposure to bagasse, which had been stored for a period of time. X-ray diagnosis revealed infiltration of the right upper lobe of the lung, and tuberculosis (Tb) was diagnosed. During Tb therapy, no further exposure to bagasse occurred. Six months after Tb therapy, the patient again suffered the same pronounced symptoms after exposure to bagasse at work. In addition, fever, cramps in the lower legs, low back pain, and anorexia were noted. He recovered upon cessation of exposure, and follow-up x-rays showed clearing of lung densities one month after cessation of exposure.

Boonpucknavig et al (NO2958) reported dyspnea, productive cough and fever, along with radiologic evidence of interstitial infiltration, in six workers exposed to bagasse. Not all signs and symptoms were present in all patients. The duration of exposure ranged from 15 to 90 days. Table III-1 summarizes the microscopic changes observed in lung tissue at biopsy.

TABLE III-1

SUMMARY OF FINDINGS ON MICROSCOPIC EXAMINATION

OF LUNG BIOPSY OF SIX WORKERS WITH BAGASSOSIS

Case	Proliferation of Granular Pneumocytes	Proliferation of Alveolar Macrophages	Proliferation of Foreign-body Granulomas	Bronchiolitis	Fibrosis
1	Mild	Severe	Moderate	Mild	Moderate
2	Mild	Severe	Moderate	Moderate	Severe
3	Mild	Moderate	Mild	Negative	Moderate
4	Negative	Mild	Negative	Mild	Negative
5	Mild	Moderate	Moderate	Moderate	Moderate
6	Negative	Mild	Mild	Mild	Mild

Adapted from reference N02958

(3) Fibrogenic Dusts

Hamilton and Hardy (N00916) identified asbestos, silica, talc and diatomaceous earths as fibrogenic dusts. Upon inhalation of these materials over a period of time, a tissue reaction occurs in which normal lung tissue is replaced with fibrous tissue. This leads to reduced pulmonary function by a reduction in the total amount of lung surface available for gas exchange and/or a reduction in the elasticity of the lung. A consequence is a limitation of the amount of air that can be brought to the gas exchange areas. Thus, the ability of the lungs to oxygenate the blood is reduced, which results in a reduction of the total supply of oxygen to the blood. In severe cases, this reduced oxygenation impairs work capacity, leading to

dyspnea or labored breathing at the slightest effort. Casarett (N00911) has listed chemical factors in the various forms of dust that are associated with the production of silicosis and pneumoconiosis, and these data are summarized in Table III-2.

Asbestos is a generic term for crystalline fibers of the serpentine and amphibole family of minerals (N00912). Fibers of asbestos which are respirable and can penetrate into the alveoli are generated in many industrial processes. Asbestos fibers greater than 3 m in diameter are not respirable (N00912). In the lung, these fibers produce a diffuse, nonmodular type of fibrosis that spreads along the supporting structures of the lung, leading to destruction of the alveoli and vascular bed (N00912). This fibrosis causes restrictive impairment, reducing lung volume, vital capacity, and residual volume without increasing the resistance to flow in the lung. Radiographic changes are small, poorly defined, irregular densities in contrast to those seen in silicosis (N00912). Sander (N00891) compared the radiographic changes of silicosis and asbestosis, and these effects are summarized in Table III-3.

Diatomaceous earths have been shown to produce a diffuse fibrosis, lung function abnormalities, and radiographic changes (N00916). Reports from Europe summarized by Ahlimark et al (cf N00916) suggest that "natural powders" of amorphous silica produce radiographic changes with little or no disability. Calcined or flux calcined powders, in contrast, were reported to cause pulmonary fibrosis. Although similar in many respects to silicosis, not all of the changes or symptoms seen were the same. For example, tuberculosis susceptibility is frequently enchanced by silicosis. Yet, a study of the

TABLE III-2

DUST FORMS SIGNIFICANT IN THE PRODUCTION OF SILICOSIS AND RELATED FORMS OF PNEUMOCONIOSIS

Dust Type	Etiologic and Chemical Factors	Site of Reaction	Type of Response	Designation
Quartz	Silica, SiO_2	Lung parenchyma Lymph nodes Hilus	Collagen with some reticular nodes	Silicosis (tuberculosis)
Coal	Coal plus SiO_2 and others	Lung parenchyma Lumph nodes Hilus	Primarily collagen nodules	Anthraco -silicosis
Graphite	Finely divided coal plus SiO_2 and others	Lung parenchyma Lymph nodes Hilus	Primarily collagen nodules	Graphite lung
Kaolin	$Al_2(Si_2O_5)OH_4$ plus crystal-line SiO_2	Lung parenchyma Lymph nodes Hilus	Primarily collagen nodules	Kaolinosis
Diatoms	Amorphous SiO_2	Lung parenchyma	Diffuse fibrosis	Diatom lung (kieselguhr lung)
Talc	$Mg_6(SiO_2)OH_4$	Lung parenchyma Lymph nodes	Collagen nodes Granulomata	Pleural sclerosis
Asbestos (silicates)	Fibrous silicates (Mg, Ca, and others)	Lung parenchyma	Diffuse fibrosis	Asbestosis

Adapted from reference N00911

TABLE III-3

RADIOGRAPHIC CHARACTERIZATION OF ASBESTOSIS AND SILICOSIS

Asbestosis	Silicosis
Diffuse fibrosis (film may have ground glass appearance from pleural involvement)	Nodular fibrosis
Findings may be either bilateral or unilateral	Findings characteristically bilateral
Lesions largely in the lower one half or two thirds of the lung fields	Lesions predominantly in the upper two thirds of the lung fields
Emphysema in upper portion of lung fields	Emphysema in lower portion of lung fields
Borderline cases difficult to recognize	Borderline cases more easily recognized.

Adapted from reference N00891

diatomite industry by the US Public Health Service in 1953-54 revealed no evidence of an association between x-ray changes observed in silicosis and tuberculosis. The extent to which this lack of association with tubercular changes is characteristic of other pneumoconioses is uncertain.

The radiographic changes associated with diatomaceous earth fibroses have been described as linear, modular or coalescent (N00916). Lung function abnormalities reportedly show no characteristic pattern.

Talcosis is a pulmonary fibrosis with nonspecific tissue changes (N00916). Radiographic changes show varying degrees of widespread modulation, usually with thickened pleura (Kleinfeld, as cited in N00916). Impaired ventilatory function and diffusing capacity of the lung are present. Cyanosis and clubbing occur with progression. Not all talcs produce fibrosis. As talc is found naturally in both fibrous and nonfibrous forms, there is disagreement on the degree of pathogenicity of the two forms.

(b) Physical hazards

(1) Heat and Humidity (heat stress)

(A) Biologic Mechanisms

Exposure to high ambient temperatures, hot processes, high humidity and heavy work affects the ability of humans to maintain stable body temperatures. These factors interact with normal body processes in a complex fashion which can lead to major clinical disorders, psychological disturbances, and early fatigue, as well as physical burn injuries. Excessive heat exposure is also known to potentiate the effects of chemical exposure (N02778), but this interaction has not been thoroughly investigated.

Environmental factors interact with the body by (a) adding to the heat load of the body and (b) reducing the body's rate of heat loss (N01143). These environmental factors are temperature, air speed,

humidity, and radiant heat load. In pulp and papermaking, all of these factors can be present at physiologically important levels, although radiant heat is not likely to be significant. No data are currently available on measurements of these factors.

The three major clinical heat-induced disorders are heat stroke, heat exhaustion, and heat cramps. Heat stroke, the most serious of the three, is characterized by hot, dry skin that is red or patchy, hyperthermia (excessive body temperature), mental confusion, delirium, loss of consciousness, convulsions, and coma (NO2778). Body temperature can exceed 106 F (41 C) and frequently rises rapidly. The basic mechanism of heat stroke is as follows: sweating ceases, eliminating the primary means of heat removal and, thus, temperature control. Since evaporation of sweat is necessary to eliminate metabolic as well as environmental heat, the lack of sweating results in storage of the metabolic heat. As the body's internal temperature rises, metabolism increases, thus creating a self-sustaining cycle leading to a rapid increase in body temperature. The treatment of heat stroke is a major medical emergency, requiring rapid cooling and observation for shock.

Heat exhaustion is caused by excessive body water loss as a result of insufficient water and/or salt intake (NO2778). The symptoms are general weakness or fatigue, giddiness, nausea, and headache. The skin is clammy and moist, and color may be pale, muddy, or flushed. Internal body temperature may be slightly elevated, and occasionally the patient may faint due to low blood pressure. Upon correcting the salt/water balance, recovery is usually prompt and may occur spontaneously with rest

in a cool place. In severe cases, medical treatment is necessary. Heat exhaustion is more likely to occur in unacclimatized workers.

When salt lost through sweating is not replaced, heat cramps, which are painful muscle spasms, result. Treatment is ingestion of salted liquids, and the illness can be prevented by increasing salt intake at meals.

Heat stress imposes significant demands on the cardiovascular system, reducing the capacity for work. It also reduces mental alertness and has been shown to contribute to increased accident frequencies.

(B) Experimental Studies of Effects of Heat and Humidity

Strauss et al (N03200) investigated the effect of heat and humidity on exercise-induced asthmatic airway obstruction. Seven subjects (1 male, 6 female) exercised at high rates under several different sets of environmental conditions. Four combinations of air temperature and humidity were used; air temperature at ambient levels (23-27 C) and humidity at ambient levels (50% humidity): air at body temperature (37 C) and humidity at ambient levels (50%); air at ambient temperatures and saturated with water (100%); air at body temperature (37 C) and saturated with water (100%). In these subjects, stress-induced airway obstruction occurred at ambient humidity levels, but was mitigated by increasing the water content of the air. At ambient temperature with saturated air, reductions in pulmonary functions were

lessened, and at body temperature, with saturated air, no response was induced. These results were not considered conclusive, but emphasize the problems of asthmatics in hot industries.

Shapiro et al (N03188) showed that intraocular pressure increases in individuals who are not acclimated to hot environments when exposed to heat. Interocular pressure does not appreciably change in those individuals who were acclimated to heat. Cohen (N03193) reported that healing of wounds in mice that were exposed to heat took longer than healing of wounds in unstressed animals. Comparisons were also made between animals exposed to heat before injury and those exposed during healing. There was no significant difference in the total number of days to healing between these groups.

Knecht et al (N03196) exposed rats to heat sufficient to raise core temperatures to approximately 40 C in males and 41 C in females. The authors suggested an effect on male reproductive ability due to decreases in the percent of males copulating after heat exposure. Increases in the number of resorptions and percent of dams with 2 or more resorptions in females mated with treated males were also noted. None of the above findings was statistically significant, and other parameters of reproductive ability were unimpaired. During lactation, dams that had been exposed to heat began to lose whole litters and, intolerant to daily exposures themselves, began to die.

A study of the relationship between anencephalic pregnancies and maternal hyperthermia in 63 women was reported by Miller et al (N03199)

in 1978. Seven of 63 mothers (11%) reported a history of hyperthermia during the period in which anterior neural-groove closure in the fetus is presumed to occur. Five women reported fever secondary to infection and two reported incidents associated with sauna bathing. Compared with two control groups, the total incidence of hyperthermia in the anencephalic pregnancies was highly significant. In a related study, 3 of 6 mothers who delivered anencephalic children in a 9-month period reported histories of hyperthermia during the critical period, with two due to fever and one to sauna. The authors suggested tnat hyperthermia, whatever the source, is possibly a cause of anencephaly, and quoted animal studies supporting their conclusion.

In contrast, Rapola et al (NO3198) took issue with the suggestion that sauna bathing was related to anencephaly. They based their conclusion on the high incidence of sauna bathing in Finland compared to the low incidence of anencephaly reported for that country.

(2) Noise

NIOSH (NO2905) has categorized the effects of occupational noise exposure into four groups. They are:

- temporary and permanent losses in hearing sensitivity
- physical and psychological disorders
- interference with speech communication or the reception of other wanted sounds
- disruption of job performance.

Hearing loss can result from physical damage to the ear, such as rupture of the eardrum and dislocation of the bones of the middle ear, and from injury to the hair cells of the inner ear. Permanent injury to the inner ear is quite serious because degeneration of the nerve cells occurs with injury to the hair cells (NO1143).

Levels of noise known to cause permanent hearing damage exceed 60-80 dBA (decibels A-weighted)(NO2905). However, there is also a time component to the exposure pattern which affects hearing loss. The current OSHA standard limits noise exposure to 90 dBA over an 8-hour workday, with an increase of 5 dBA permitted for each halving of the length of exposure, up to 115 dBA. NIOSH (NO2905) has recommended that the standard be lowered to 85 dBA over an 8-hour period, with an increase of 5 dBA permitted for each halving of the exposure period. This recommendation would minimize (1) unacceptable noise-induced hearing loss which interferes with speech communication, and (2) an increase (no more than 15%) in the number of people, in a population aged 50-65 years with a minimum 20 years of exposure, that will suffer impaired speech communication compared with a nonexposed group (NO2905).

There are a number of reports in the literature regarding physical and psychological effects which were considered by NIOSH in recommending the above standard. No conclusive evidence existed at that time indicating that excessive noise caused nonauditory detrimental health effects (NO2905). The question at this time is still unanswered.

A number of reports on nonauditory biologic effects of noise have been published. It is clear that noise of short duration and sufficient loudness stimulates physiological changes associated with the startle response (N01143). The literature is less clear on whether these changes persist in the presence of continuous high intensity noise or intermittent high intensity noise.

Cartwright and Thompson (N03247) addressed the question of how broadband noise affects the cardiovascular system in normal, resting adults. They exposed 20 subjects (11 female, 9 male) to 91 dBA for 1 hour, measuring blood pressure and pulse rate. One-half-hour periods of quiet preceded and followed the 1 h noise period. Post-exposure audiograms generally revealed a temporary threshold shift (TTS). The authors evaluated their data by observation of graphical representations. during the test period (1/2 hour quiet, 1 hour noise, 1/2 hour quiet). When values for certain periods of the test runs were compared to corresponding time periods in control runs, only 2 of 20 comparisons were statistically significant at the $\alpha = 0.05$ level. The authors concluded, however, that the changes observed in these two measurements were not related to the experimental conditions, especially since the confidence level for detecting changes as small as $\pm$ 6 beats per minute in heart rate and/or $\pm$ 6 mm Hg in blood pressure was 97.5%.

In 1979, Ickes et al (N03207) reported the results of exposure of young adults to 100 dBA "white" noise for 15 minutes. They measured vasoconstriction by changes in the reflectance of the light from skin, which is affected by the presence of blood near the surface. They

hypothesized that, if changes in vasoconstriction due to noise were controlled by the autonomic nervous system, persons with different personality types would respond differently. These personality types had been shown by Rosenman in 1964 to have characteristic autonomic nervous system responses to stress-prone behavior (N03207). Ickes et al were able to demonstrate a distinction in response between males of different types, but not between females of different types. Further, the changes in Type A males (aggressive types) persisted after the noise was terminated. The authors discussed causes of these two phenomena and the relation of vasoconstriction to hearing loss, but provided no data to support their speculations.

Loeb et al (N03038) in 1976 evaluated the effect of noise on the performance of tasks assessing various nonauditory functions. There was improvement in visual acuity, an increase in pupil size, a decrement in detection of lights during impact noise exposure, non color-specific increase in critical flicker frequency, and a slight retardation of dark adaptation during continuous noise exposure. Analyses of variance, however, revealed that time period was the only significant source of variation in these experiments; noise exposure condition was not significantly affected. These data indicated that noise, per se, had no effect on the variables that were measured.

In another study of the effects of noise on vision, Hermann et al (N03209) tested the suggestion that excessive noise has a detrimental effect on depth perception. Using the Howard-Dolman apparatus, subjects were tested for their ability to line up two rods in the presence of

noise of 85, 90, 95, 100, 105 and 110 dBA intensity for 5 minutes compared with their ability in the absence of noise. Groups of 59 and 28 subjects were tested according to 5 procedures. Compared with controls, no significant differences could be measured in any of the test procedures. The authors therefore concluded that depth perception effects were too small to be significant in establishing exposure standards.

The relationship between noise and hypertension was retrospectively studied by Malchaire and Mullier in males in a 1979 report (NO3201). Data on hearing acuity and blood pressure were collected in three groups of people by trained audiometric technicians and occupational physicians over a 3 to 4 year period. Individuals from 2 manufacturing facilities were grouped on the basis of World Health Organization definitions of normotensive, hypertensive, or borderline hypertensive states. Individuals with histories of cardiovascular problems, year-to-year variation in systolic blood pressure greater than $\pm$ 30 mm Hg, and hearing impairments due to causes other than noise were eliminated from the study. Noise levels in both manufacturing facilities averaged 95 $\pm$ 1 dBA.

Malchaire and Mullier (NO3201) found a statistically significant correlation between hearing impairment (a 25 dB or greater hearing threshold) and presence of borderline hypertensive state in 30-39 year-old males. The authors concluded that exposure to 95 dBA did not increase the prevalence of hypertension in males, but admitted the possibility of a relationship at higher levels. Further, they indicated that the conclusion could not be exptrapolated to include groups with persons predisposed to cardiovascular problems.

The study generally was well designed. Certainly the data base appears to be well-documented and to meet current standards on the determination of exposure, hearing acuity, blood pressure, and size. However, the slight elevation in blood pressure in hearing impaired individuals in the 30-39 year age groups would seem to preclude a total rejection of a possible relationship between noise and hypertension. If persons susceptible to hearing impairment and hypertension remove themselves from the exposure in their 30's and 40's, then the older age group might have fewer hypertensives than would occur if individuals could not remove themselves from exposure. The authors do not adequately explain this finding.

Rontal et al (N03205) reported that vocal chord dysfunction was present in 7% of patients with noise-induced hearing loss. Further, 23 of 26 patients with vocal chord nodules claimed they had to shout at work to be heard. Most of the 26 patients were smokers, and all worked in dusty environments. Injury to the vocal chords can arise from employment in a noisy job where there is need for verbal interaction between workers.

It is frequently hypothesized that continuous noise negatively affects performance of manual tasks. This hypothesis, first presented in the 1950's, was challenged by Poulton (N03203) in 1980. He asserted that in all of the experimental studies which he reviewed relating continuous noise exposure to decreases in performance of motor skills, an artifact was involved. The high noise level masked an auditory cue from the task equipment which assisted the subject in the performance of the task in the low noise control situation. That is, in the absence of noise, the

subject heard the click of microswitches or timers, which told him to do something. The clicks were not heard in the presence of intense noise, due to masking. This possibility is certainly plausible, and Poulton presented a convincing argument.

In conclusion, noise is believed to affect people in ways unrelated to hearing loss. The observation of Rontal et al (NO3205) on vocal chord dysfunction in noisy environments is important. Effects on the cardiovascular and visual systems, and behavioral effects, have been observed only at those levels which are known to produce hearing losses and/or temporary threshold shift. It is still not clear whether these effects occur because the individual, sensing the noise through hearing, is stimulated to the same degree. That is, does noise affect our bodies because we hear it, or because it has a direct effect on other bodily systems?

It should also be noted that noise levels interfering with sound reception may not be damaging to hearing, and criteria for ensuring adequate speech and reception are available from a number of sources (NO2905). Changes in job performance due to noise have also been investigated, but data supporting unequivocal conclusions are lacking (NO2905).

(c) Raw Materials or Chemical Intermediates

These are chemical agents or mixtures of chemicals which are used in or formed during pulping operations. As discused below, the complex

mixtures of chemicals (liquors) used to dissolve lignin and other components of wood pulp rely largely on chemical recovery operations for efficient use. Sodium sulfate, sulfur, magnesium oxide, and a few additional agents are specifically used as raw materials. Most of the other chemicals are byproducts of intermediate chemical reactions and are recovered for further use. Although various additives may be considered raw materials, they will be discussed in a subsequent section.

(1) Calcium Oxide

Calcium oxide is used in the recovery of sodium hydroxide, which is a major component of alkaline pulping liquor (see below). Calcium oxide (lime) is caustic to tissue, as it reacts with water to form calcium hydroxide with the evolution of heat. Calcium hydroxide is considered an important industrial hazard since it may cause dermatitis, bronchitis, and pneumonia (N00919, N03148). The heat produced by the reaction of the oxide on skin or mucous membrane to form the hydroxide produces a thermal and caustic burn (N00596). Calcium hydroxide, because of low aqueous solubility, is not as corrosive as calcium oxide. Calcium oxide may also react with carbon dioxide in air to form calcium carbonate, which may alter toxic effects of calcium oxide (N03148).

Calcium oxide burns are especially hazardous to the eye, since the reaction of the oxide with the eye's moisture and protein produces products that can be difficult to remove by the usual irrigation techniques. These products tend to lodge in reservoirs that liberate calcium hydroxide over long periods of time if not removed promptly

(N00911). Lacrimation, spasmodic blinking, ulceration, and ocular perforation may result from contact with calcium oxide (N03148).

(2) Magnesium Oxide

Magnesium oxide is used in the production of magnesium bisulfite, a major constituent of acid pulping liquor. Magnesium oxide is a noncombustible white powder that is slightly soluble in water (N00917). There is little danger from ingestion because of very slow absorption from the gastrointestinal tract. If appreciable absorption does occur, signs of magnesium intoxication may occur. These include central nervous system depression and eventual respiratory paralysis (N00596). Toxicity, in the form of a fever and leucocytosis, may occur as a result of inhalation of magnesium oxide fumes (N00919). In powder form, magnesium oxide may irritate mucous membranes, resulting in a chronic nasopharyngitis (N00825).

(3) Pulping Liquors

Pulping liquors are complex mixtures of chemicals used to dissolve lignin in pulp fibers prior to the bleaching and mechanical processes that prepare the pulp for the papermaking machine. The lignin in pulp is dissolved by alkaline chemicals in the kraft process or acid liquors in the sulfite process. In the kraft process, pulp is cooked (digested) in a "white liquor," consisting primarily of sodium hydroxide and some sodium sulfide. In another alkaline process (the soda process), elemental sulfur is added to the liquor to improve subsequent pulp

bleaching ability and yield (NO2381). The alkalis hydrolyze lignin in the pulp and also react with other constituents to form alkali lignins and reduced sulfur effluents such as hydrogen sulfide, methyl mercaptan, dimethyl sulfide, and dimethyl disulfide. The biologic effects of these effluents will be discussed in a subsequent section. The spent cooking liquor, known as "black liquor," contains unwanted residues, which are concentrated and then burned, and other chemicals that will be recovered for further pulp digestion. Sodium hydroxide is recovered by (a) burning black liquor in a recovery furnace to yield sodium carbonate in a "green liquor," (b) adding calcium oxide (lime) from a lime kiln to water to form calcium hydroxide (slaked lime), and (c) mixing slaked lime with sodium carbonate to yield sodium hydroxide. The other product from this last reaction is calcium carbonate, which is heated in a kiln to recover the lime. Both sodium hydroxide and sodium sulfide are lost in digesting and recovery processes, and sodium sulfate is added as make-up to the pulp digester.

In the acid (sulfite) process, sulfonation of the lignin in wood with sodium bisulfite occurs. Prior to this process, sulfur is melted and oxidized to sulfur dioxide, which combines with water in an absorption tower to form sulfuric and sulfurous acids. These acids and sulfur dioxide combine with magnesium carbonate and magnesium hydroxide, respectively, to form magnesium bisulfite--which hydrolyzes the lignin. The spent cooking liquor is called "red liquor," disposal of which was a serious pollution problem when calcium, rather than magnesium bisulfite, was used in the cooking liquor. Magnesium, but not calcium bisulfite, is recoverable by burning red liquor. The magnesium oxide that is formed is

slaked to magnesium hydroxide, which, when combined with the sulfur dioxide that is also formed during burning of the red liquor, forms magnesium bisulfite.

Studies of the biologic effects of the complex chemicals in pulping liquors have been performed entirely on aquatic organisms. The acute toxicity to rainbow trout of sulfite pulp waste liquor was studied by Griffin and West in 1976 (N03044). Using standard American Public Health Association (APHA) procedures, static bioassays were conducted on trout acclimated to aquaria, each containing 30 liters of neutral sulfite waste liquor from a pulp mill or water during the test period. Percent survival to 96 hours was plotted against sulfite liquor concentration to determine the LC_{50}. For each bioasay, fresh liquor was analyzed for sulfur dioxide, ammonia, pH, biochemical oxygen demand (BOD), filterable solids, and transmittance. Oxygen content of aquaria did not fall below 5 mg/l. The mean LC_{50} was 0.82%, with confidence limits of 0.08-1.56%. Regression analysis indicated significant correlation coefficients between the LC_{50} and lignin content (0.96), specific gravity (0.90), sulfur dioxide (0.81), ammonia (0.78), and BOD (0.77). In this study, mean sulfur dioxide content of the liquor was 290 mg/l, ammonia concentration was 135 mg/l, and lignin concentration (as calcium lignin sulfonate) was 0.92 g/l.

Toxicity to rainbow trout of commercial sodium lignin sulfonate powder was assessed by Roald in a study reported in 1977 (N03042). Standard APHA procedures were used to determine LC_{50} values in groups of 10 fish for lignosulfonate concentrations ranging from

0.625-20.0 g/l. Two or three replications were run at each concentration, dissolved oxygen was maintained at 80-95% air saturation, and average ammonia concentration was 0.004 mg/m^3. The 48-hour LC_{50} was calculated as 82,896 mg/m^3 (with confidence limits of 64,727-106,062 mg/m^3). The author indicated that 100% survival was observed at lignosulfonate concentrations as high as 21,292 mg/m^3 and concluded that lignosulfonate may be an important factor in the toxicity to fish of spent sulfite waste liquor. Other factors include the lowered pH of the water and lower oxygen availability in the effluent-contaminated water.

The studies by Griffin and West (N03044) and Roald (N03042) are not comparable. Commercial lignosulfonate preparations may not represent the exact chemical milieu of sulfite liquors, although control of lignosulfonate concentration is possible. Moreover, although a molecular weight of 250 is assumed for the lignin sulfonate monomer, the molecular weight in a spent cooking liquor may be as high as 100,000 (N00808). Cooking conditions may also modify the average molecular weight of lignosulfonate.

In 1979 EPA published a review by Hutchins on the toxicity of pulp and paper mill effluents to various aquatic species (N02896). The author concluded that untreated mill effluents in concentrations as low as 2% could be acutely toxic to fish, and that even treated effluents could produce sporadic increases in toxicity. Several resin acids (abietic, dehydroabietic, isopimaric, palustric, pimaric, sandaracopimaric) appear responsible for most of the toxicity in nonbleached pulp. Chlorinated compounds (including chlorinated phenols, resin acids, and stearic acids)

contribute most of the toxicity to bleached pulp effluent. In nonchlorinated kraft effluents lignin contributes little toxicity, but if kraft pulp is acid-chlorine bleached, the chlorinated lignins can exhibit a high degree of toxicity. Toxicity of sulfite waste liquors has received less attention partly because effluents are not very toxic to aquatic life after BOD-reduction treatment, and because there are relatively few sulfite plants compared to mills using the kraft process.

In addition to general toxicity (lethality), sublethal exposures of aquatic organisms to pulping liquors may affect functions such as growth rate, coughing reflex, and temperature tolerance at less than one-tenth of the 96-hour LC_{50}. Respiration and circulatory function may be affected by higher concentrations (N02896).

(4) Sodium Hydroxide

Sodium hydroxide is a major constitutent of alkaline pulping liquor, as discussed above. Sodium hydroxide is also used for caustic extraction after the first chlorine stage during bleaching. It is a strong alkali, producing burns and ulceration on contact with tissue. As reported in the 1976 NIOSH criteria document on sodium hydroxide, principal hazards include eye contact, skin contact, ingestion, and dust or vapor inhalation (N02502). Eye injury (conjunctivitis and corneal burns) has probably been the most severe effect from contact with sodium hydroxide (N02502). Inhalation of the dust or mist may cause damage to the upper respiratory tract varying from a mild irritation of the mucous membrane to a severe pneumonitis. The dust may also cause an irritative

dermatitis (N00919). Ingestion may result in nausea and abdominal pain with diarrhea, gastrointestinal tract perforation, and cardiovascular collapse (N00919).

In a 1979 paper, Cooper et al (N03057) reviewed the available data for sodium hydroxide aerosols and discussed why they consider the OSHA standard and NIOSH recommendation of 2 mg/m^3 insufficient to protect workers' health. The authors emphasized that the few published studies on the toxicity of sodium hydroxide do not consider the fact that the toxicity of a sodium hydroxide dust or aerosol will be determined by factors other than sodium hydroxide concentration. Depending on the amount of water vapor and carbon dioxide present, the hydroxide will react with these agents to form the less alkaline sodium carbonate; this reaction will occur whether the hydroxide is in the form of droplets or solid particles. At relative humidities greater than 95%, sodium bicarbonate may also be present.

Moreover, the authors (N03057) suggested that the work done in determining human response to sodium hydroxide aerosols has been ill-defined, and conclude: (a) the eye may be the organ most vulnerable to sodium hydroxide aerosol damage; (b) improved sampling and exposure evaluation techniques should be used to prevent formation of the carbonate before hydroxide is delivered to its capture site; (c) physiochemical behavior of sodium hydroxide aerosols should be studied more fully so as to better analyze available dose-response data; and (d) the standard for sodium hydroxide should be reconsidered and a standard developed for sodium carbonate.

(5) Sulfates, Sulfites, Sulfides, and Sulfur

These agents are mainly constituents of pulping liquors. Sodium sulfate (salt cake) is added to the recovery furnace in alkaline (kraft) pulping processes to replace spent sodium and sulfide ions. Magnesium and calcium bisulfite are major constituents of acid pulping liquors. Sodium sulfide is a constituent of alkaline pulping liquor. Elemental sulfur is a raw material that is burned to form sulfur dioxide which is used to produce the major constituents of acid pulping liquors.

Sodium sulfate, used in its anhydrous form, is slowly absorbed from the gastrointestinal tract. The sulfate ion is said to be nontoxic (N00919). A study by Sackner and colleagues (cited in N02521) tested various particulate sulfate aerosols in 10-minute exposures of 10, 100, and 1,000 mg/m^3 concentrations in asthmatic children and normal adults. Tests of pulmonary function, made before and after exposure, revealed no consistent toxic effects immediately and three months after testing. Also cited in N02521 was an attempt to correlate absenteeism in workers with atmospheric particulate sulfates. Sackner et al concluded that no significant effect on absenteeism could be attributed to particulate sulfate concentrations up to 35 mg/m^3.

The bisulfites, including magnesium bisulfite (sulfite pulping liquor), are strong irritants to skin, mucous membranes, and eyes, and when inhaled or ingested (N00919). Respiratory effects resemble those of sulfur dioxide, which is emitted when bisulfites are heated or when they contact acids (N00919).

The effects of sodium sulfide are similar to those of sodium hydroxide and other alkalies. They are skin irritants and are corrosive if ingested (N00919). Hydrogen sulfide may be produced, with subsequent systemic poisoning, if sulfides are ingested and gastric acidity is high (N00596).

Sulfur has a very low toxicity; survival after ingestion of 60 grams of sulfur over a period of 24 hours has been reported (N00596). Ingestion of large amounts may lead to the production of hydrogen sulfide (due to bacterial action in colon) and subsequent systemic toxicity (N00596). Chronic inhalation of sulfur dust can cause irritation of mucous membranes and other effects of nuisance dusts (N00919).

(6) Sulfuric Acid

Sulfuric acid is used in several processes in pulp and paper mills. Both sulfuric and sulfurous acids are formed during the synthesis of calcium or magnesium bisulfite cooking liquor in the sulfite pulping process. The acids are formed in a tower, where sulfur dioxide is combined with water; the acids then react with carbonates and hydroxides to form bisulfite, and are present in the sulfite effluent. Sulfuric acid is also used to manufacture chlorine dioxide at some bleached pulp mills by reaction with sodium chlorate, although hydrochloric acid may be used for this purpose (N00807). Sulfuric acid in dilute solutions (eg, 3%) may also be used with alum (aluminum sulfate) to precipitate rosin during sizing of paper (N02381).

Sulfuric acid is extremely irritating to skin and mucous membranes. In concentrated form, it is corrosive and destructive to animal tissue by virtue of its oxidizing and dehydrating effect and consequent liberation of heat (N02496). Corrosive burns may result from skin contact, ingestion, or inhalation of fumes, and a chemical pneumonitis may occur after exposure to the vapors (N00596). According to the 1974 NIOSH criteria document on sulfuric acid (N02496), other effects of exposure to sulfuric acid vapors include pulmonary edema, chronic pulmonary fibrosis, bronchiectasis, and emphysema. Subjective effects, including throat tickling and scratching, have been reported at concentrations less than the recommended standard of 1 mg/m^3.

Studies of biologic effects of sulfuric acid emphasize effects of aerosols or mists, either alone or in combination with airborne particulates or atmospheric pollutants. In one study, groups of cynomolgus monkeys were exposed to mixtures of sulfuric acid mist, sulfur dioxide, and fly ash for periods of up to 78 weeks (N03052). The data indicated that exposure to sulfuric acid mist at concentrations between 0.1 and 1.0 mg/m^3 induced slight changes in bronchial mucosa characterized by goblet cell hypertrophy and hyperplasia. Above 2.5 mg/m^3, impairment in pulmonary ventilation (increases in pulmonary flow resistance) occurred, in addition to histopathologic changes. These effects occurred at particle sizes of 4 microns or less and were attributed by the authors to the presence of the acid alone, despite marked variability in the presence of histopathologic changes.

Guinea pigs were exposed to 10 mg/m^3 of sulfuric acid mist for 6 hours/day, 5 d/wk for 6 months, in a study by Cavender and colleagues in 1978 (N03059). The animals showed only mild tracheal changes (loss of epithelial cilia) and exhibited minimal proliferation of alveolar macrophages. No pulmonary lesions were seen and sulfuric acid mist did not enhance microscopic pulmonary lesions in animals exposed to a combination of ozone (1 mg/m^3) and sulfuric acid mist (N03059). A rat lung in vitro model has been used to demonstrate synergistic effects of mixtures of ozone and sulfuric acid mists on the secretion of mucus glycoproteins by lung homogenates and tracheal explants (N03062). Although sulfuric acid aerosol concentrations of 1 mg/m^3 exhibited synergism with 1 mg/m^3 ozone in these experiments, no significant effect of sulfuric acid mist alone was observed at the 1 mg/m^3 concentration. Earlier work (cited in N03062) indicated an increased rate of mucus glycoprotein secretion with a sulfuric acid mist concentration of 137 mg/m^3.

As sulfuric acid may form in the atmosphere by oxidation of sulfur dioxide on such surfaces as particulate carbon, effects of sulfuric acid mist alone and in combination with carbon particles have been studied in rodents (cf N03058, N03065). Mice exposed to the combination exhibit alterations of immunoglobulin titer, depression of antibody response to spleen cell antigenic stimulation, and decreased resistance to respiratory infection (N03058). Cytotoxic effects to hamster tracheal tissue of sulfuric acid-carbon particle aerosols containing 1.1 mg/m^3 acid concentrations and 1.5 mg/m^3 carbon particle concentrations were significantly greater than effects produced by either acid mist or carbon particle exposure alone (N03065).

(d) Pulp Bleaching Agents

Bleaches remove brown lignin coloring material from the pulp. Acid pulps can be bleached by sequential treatment with chlorine, caustic soda extraction, and chlorine dioxide (CED sequence). Sulfate pulps require a CEDED sequence, involving an additional caustic extraction and chlorine dioxide stage. Occasionally, a sodium hypochlorite bleaching stage precedes the first chlorine dioxide stage, to give the sequence CEHDED (N00808). Chlorine dioxide is used because it is capable of producing very bright pulps and is not degrading to pulps. Chlorine dioxide is formed by the reaction of sodium chlorate and sulfuric acid. A relatively recent development is the use of an oxygen stage that precedes chlorine and chlorine dioxide bleaching. Oxygen treatment reduces the amount of lignin in the washed pulp effluent.

(1) Chlorine

The 1976 NIOSH criteria document on chlorine (N02501) reported that chlorine's irritating properties make it a serious respiratory hazard, as well as a skin and eye irritant. Chronic lung diseases have been reported in persons accidentally exposed to chlorine at high concentrations and during the bleaching of waste paper stock using nonautomated procedures (N02730). Also, accidental exposures to massive concentrations have on occasion been associated with electrocardiographic changes, instances of significant sinus tachycardia, isolated ventricular extra-systoles, and signs of repolarization disturbances of the left ventricle. Historically, various exposure standards have been set in

order to minimize chronic changes in the lungs, accelerated aging, and erosion of the teeth.

In Occupational Diseases (NO3146), NIOSH reported that prolonged exposure to low concentrations of chlorine may cause chloracne, and that 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. Extensive exposure may result in acute respiratory distress manifested by cough, hemoptysis, chest pain, dyspnea, and cyanosis. In addition, tracheobronchitis, pulmonary edema, and pneumonia may supervene.

The National Research Council (NO2547), in a report to EPA, indicated that worker exposure to low (0.02-4.1 mg/m^3) time-weighted-average chlorine concentrations did not produce significant pulmonary changes when compared with a matched control cohort. However, there are individuals, such as heavy smokers and those with cardiovascular problems, who may be particularly susceptible to adverse effects of exposure to chlorine at environmental concentrations.

A number of animal toxicity studies were reviewed and reported in the NIOSH chlorine criteria document (NO2501). The majority of these consisted of exposing the test animals, generally dogs, to varying atmospheric chlorine concentrations, observing the characteristic behavioral pathology, and then conducting autopsies to evaluate gross pathology and histopathology. These tests clearly documented the irritative and acute inflammatory reactions caused by exposure to

chlorine gas. They demonstrated the ability of chlorine to induce such responses as dilation and contraction of bronchi resulting in alternating patches of acute emphysema and atelectasis, edema, bronchiolitis obliterans, and, frequently, pulmonary infection resulting in pneumonia.

Recently, a subacute study conducted by Barrow et al (NO3075) investigated the pulmonary, hepatic and renal effects of exposures to relatively low concentrations (2.9, 8.8, and 26.5 mg/m^3) of chlorine in Fischer 344 rats. Groups of 10 rats (5 male, 5 female) were exposed to the chlorine for 6 h/d, 5 d/wk, for 6 weeks. Urinalysis revealed the urine specific gravity to be elevated at all exposure concentrations in the females and at 8.8 and 26.5 mg/m^3 in the males. Females exposed to the highest chlorine concentrations (26.5 mg/m^3) had increased hematocrit and white blood cell counts. Elevations in alkaline phosphatase activity, blood urea nitrogen, gamma-glutamyl transpeptidase, and serum glutamic-pyruvic transaminases occurred at 26.5 mg/m^3; alkaline phosphatase was elevated in rats of both sexes exposed to chlorine at 8.8 mg/m^3. Numerous histopathologic changes were observed in the respiratory tracts of rats of both sexes exposed to the various concentrations of chlorine. The kidneys of male rats exposed to the highest concentration were found to have increased eosinophilic cytoplasmic homogeneity and decreased granularity of the epithelial cells of the proximal convoluted tubules. The livers of rats of both sexes exposed to 26.5 or 8.8 mg/m^3 of chlorine had an increased hepatocellular cytoplasmic vacuolation. The authors concluded that their data showed that repeated exposure of Fischer 344 rats to chlorine resulted in pulmonary effects at all levels of chlorine used, and hepatic and renal effects at 26.5 and 8.8 mg/m^3.

Dodd et al (N03072), in another recent study, examined lung sulfhydryl and enzymatic changes in Fischer 344 rats following inhalation of chlorine at concentrations of 35.4 mg/m^3 for 6 h/d, for 1, 5, or 10 days. The primary reason for conducting these experiments was to investigate the postulate that respiratory tract irritation caused by chlorine may be associated with tissue sulfhydryl oxidation. The authors concluded that since oxidation of lung sulfhydryl content was not observed immediately following chlorine exposure, the observed increases in lung sulfhydryl and enzymatic activities during recovery periods may reflect reparative processes subsequent to the chlorine-induced lung damages.

(2) Chlorine Dioxide

Chlorine dioxide is a reddish-yellow gas with a characteristic odor that can be distinguished from that of chlorine (N00825, N02703). The NISOH/OSHA Pocket Guide to Chemical Hazards (N00872) reports that exposure to chlorine dioxide may cause irritation of the eyes, nose, and throat. Also, exposure may induce coughing, headache, and rhinorrhea, and may cause conjunctivitis, bronchitis, pulmonary edema, chronic bronchitis, emphysema, and gastrointestinal disturbances. Papers detailing experiments investigating the inhalation toxicity of chlorine dioxide are few in number and lacking in quality.

Dalhamn (N02707) exposed rats to several concentration of chlorine dioxide for various periods of time. Animals exposed to 730 mg/m^3 for 2 hours exhibited ocular discharges, epitaxis, and pulmonary edema. One

animal died after 1 hour. Daily exposure of animals to 28 mg/m^3 resulted in marked weight loss and death after 13 days. In exposed animals kidney and liver congestion was observed. Exposure of animals to 0.3 mg/m^3 for 5 h/d for 10 weeks resulted in no overt changes but respiratory infection was present in 3 of the control animals. The exposure procedure itself may have induced these infections, since 2 of 6 animals exposed to compressed air for 5 h/d for 10 days developed purulent bronchitis.

A review of clinical and animal studies of effects of chlorine dioxide exposure was published by Gloemme and Lundgren in 1957 (NO2703). The clinical studies were mainly case study descriptions where concentrations of chlorine dioxide were not specified. Qualitative effects of acute and chronic exposure were mainly respiratory (cough, bronchitis) and gastrointestinal (nausea, vomiting). Results of the animal experiments were difficult to assess due to the manner of data presentation and because of the use of widely different concentrations and exposure times.

Recent animal toxicity studies conducted on chlorine dioxide focused on the effects caused by ingestion of the chemical (NO2934). The rationale for utilizing this route of exposure is that chlorine dioxide is being used as a disinfectant in potable water supplies. Calabrese et al (NO2934), in a recent (1978) survey of literature dealing with the health effects of chlorine dioxide, reported that chlorine dioxide along with chlorate and chlorite ions can oxidize hemoglobin to methemoglobin in vivo. Since methemoglobin is incapable of combining reversibly with

oxygen, exposure to these chemicals results in a decreased oxygen-carrying capacity of the blood; therefore, an individual exposed to these chemicals could become cyanotic. Unfortunately, no animal studies have been conducted that investigate whether methemoglobinemia may develop in animals that are acutely or chronically exposed to chlorine dioxide.

(3) Sodium Chlorate

The Handbook of Industrial Toxicology (N00825) reported that the primary symptoms of overexposure to sodium chlorate are conjunctivitis, irritation of the skin, nausea, vomiting, abdominal pain, diarrhea, and anuria. As in the cases of chlorine dioxide and chlorite, the chlorate ion can oxidize hemoglobin to methemoglobin and the reaction is autocatalytic (N02934). The literature reports that cyanosis has been observed in individuals suffering acute chlorate poisoning and that the actual pathologic condition of methemoglobinemia has been and can be documented through application of the appropriate diagnostic tests.

No applicable animal toxicity studies investigating the effects of exposure to sodium chlorite were found.

(4) Sodium Hypochlorite

Plunkett's Handbook of Industrial Toxicology (N00825) stated that occupational exposure to airborne sodium hypochlorite may result in irritation of the eyes and skin with vesiculation and eczema. Inhalation

of sodium hypochlorite may cause cough, edema of the pharynx and larynx, and possibly pulmonary edema.

As mentioned above, Calabrese et al (NO2934) reported that the chlorite ion can oxidize hemoglobin to methemoglobin in vivo. They also reported that this particular biochemical reaction has been described as autocatalytic. Unfortunately, the authors did not comment upon the seriousness of this hazard in an occupational exposure frame of reference.

Animal toxicity studies investigating the effects of inhaled sodium hypochlorite have not been found.

In a 1980 paper, Cunningham (NO2953) reported the results of four experiments conducted to determine the effects of sodium hypochlorite on growth of weanling rats and guinea pigs. Relative to control groups, statistically significant increases in body weight gains were observed in: (a) male rats receiving the equivalent of 4 mg/kg available chlorine in their drinking water for 6 weeks, and (b) female rats receiving the equivalent of 8 mg/kg available chlorine by gavage twice daily for 2 weeks. In other experiments reported in this paper the increases in body weight gains relative to control animals were not statistically significant.

The basis for the improvement in growth rate resulting from sodium hypochlorite ingestion is not known. Cunningham (NO2953) speculated that hypochlorite in low concentrations could be acting as an antibiotic to counteract low-level infective agents in the gastrointestinal tract.

Alternatively, hypochlorite might inhibit nonpathogenic microorganisms that would compete with the animal for the energy provided by the ingested food.

(e) Papermaking Additives

There are literally hundreds of chemicals used as additives in pulping and papermaking operations. These range from agents that directly influence paper properties, such as fillers, coatings and dyes, to agents that control microorganisms and organic deposits on equipment. Only a few of the more important or widely-used additives will be discussed in this report. A complete discussion will be deferred until more information is obtained on the extent to which known additives are used in contemporary pulp and paper manufacturing operations.

(1) Acrylamide

Acrylamide is used in polymeric forms as synthetic wet-end adhesives. These agents promote bonding of pulp fibers and may be added to pulp stock after beating or refining or during various operations on the papermaking machine (N02668). A typical synthetic acrylamide adhesive is polyacrylamide, to which is added acrylic acid (about 10%) and aluminum hydroxide to produce the final complex polymer resin (N02668).

Monomeric acrylamide is a well-known neurotoxin and may also cause eye irritation and dermatitis in humans (N02929). Neurotoxic effects

include numbness, touch tenderness, coldness of extremities, and weakness. This material can be absorbed easily through the unbroken skin (N00919).

The toxicology of monomeric and polymeric acrylamide has been studied in several species. In a 1964 study, McCollister and colleagues (N02690) administered acrylamide to rats, guinea pigs, and rabbits in single oral doses (2.5-12.6%), in 90-day feeding studies (equivalent to 0.3-40.0 mg/kg/day), and in 1-year oral feeding studies in cats and monkeys (0.03-10.0 mg/kg/day). In addition, rabbits received skin applications of a 12.5% aqueous solution at dosages from 0.63-1.0 g/kg and administration of 10% and 40% aqueous solutions to the eye.

Severe but completely reversible neurotoxic effects (loss of proprioceptive control of hindquarter movement) were observed in rats fed acrylamide monomer in doses above 11 mg/kg/day (N02690). At a dose level of 30 mg/kg/d (provided by addition of acrylamide monomer to the polymer) severe mortality resulted, but the survivors recovered normal function when placed on the control diet. In the cat, 0.3 mg/kg/day of the monomer produced no signs of toxicity after 1 year. Doses of 3 and 10 mg/kg/day produced toxic signs in cats and monkeys, respectively. In rabbits, slight weight loss and reddening of skin occurred at 0.5 g/kg (monomer), and a 10% aqueous solution caused slight conjunctival irritation.

The authors (N02690) concluded that acrylamide is moderately toxic by acute oral administration to rodents and by parenteral and oral

administration to cats and monkeys. It was readily absorbed through the skin from aqueous solution, but toxicity to eye and skin was relatively low. The oral toxicity of the polymer appeared to be much lower than that of the monomer. The extent to which the monomer was absorbed through the skin was not assessed.

To study the relationship of acrylamide neurotoxicity to brain neurochemistry, Howland et al (N03262) performed experiments where the activity of an isoenzyme of enolase was assessed in brain and peripheral nerve tissue of rats. Male Sprague-Dawley rats received 11 daily 50 mg/kg parenteral injections of monomeric acrylamide or saline. At the end of this period, acrylamide-injected animals exhibited neurotoxic signs, including ataxia and hind limb paralysis. Neurochemical analysis indicated that the neuron-specific enolase (NSE) activity in acrylamide-treated rats was 60% of the level observed in controls, and was not detectable in sciatic nerves. In additional experiments, 10 mM of acrylamide completely inhibited total enolase activity of rat brain soluble fractions.

Howland et al (N03262) interpreted these results as supporting the hypothesis that acrylamide neuropathy may be related to the disruption of neural energy metabolism in the brain. However, previous studies on effects of acrylamide on oxygen consumption and high energy phosphate levels (cited in N03262), showed no effects of acrylamide treatment. The reported observation that only NSE and not total enolase activity was depressed by acrylamide suggests that effects of acrylamide on oxygen

on isolated glial and neuronal fractions.

(2) Alum (aluminum sulfate)

Alum (aluminum sulfate) is used mainly to precipitate dissolved rosin that is used for paper sizing. An additional use is that of controlling the pH of pulp stock solutions. About two-thirds of the alum produced in this country is used in the manufacture of pulp and paper (N00874).

Alum is a white crystalline powder that is soluble in water (N00917). Although aluminum sulfate is generally regarded as exhibiting low toxicity, ingestion of 30 grams on 2 occasions by an adult human resulted in death (N00596). This lethal effect was attributed to the corrosive action of sulfuric acid that may have been formed from hydrolysis of the salt (N00596). Other human effects resulting from ingestion of concentrated solutions include gasteroenteritis, necrosis of gum tissue, incoordination, clonic muscular contractions, and evidence of nephritis (N00596).

There are few reported studies of the toxicity of aluminum sulfate in animals. An acute oral LD_{50} of 6.1 g/kg has been reported in mice (N00596). Toxicity by the intraperitoneal route in this species is greater, and a LD_{50} of 270 mg/kg was reported in RTECS (N00893). There is neither an OSHA standard nor an ACGIH recommended exposure level for aluminum sulfate.

(3) Carbon black

Although a variety of dyes and pigments are used as coloring agents in papermaking, specific information indicating which dyes are in contemporary use is not available. However, one such agent--carbon black--is used as a basic constituent of carbon paper and a pigment in colored paper. An OSHA standard of 3.5 mg/m^3 was developed to protect against carcinogenesis and respiratory effects associated with exposure to carbon black (N02904). Although skin cancer and cancer of the nasal sinuses have been associated with carbon dust, it is not clear whether such effects are due to carbon particles, coal tar products which adhere to them, or a combination of these factors (N00919).

The 1978 NIOSH criteria document on carbon black (N02904) indicates that adverse pulmonary and cardiac effects may result from exposure to the dust, but that the reliability of these effects has not been determined.

(4) Formaldehyde and Melamine- and Urea-Formaldehyde Resins

Biologic effects of formaldehyde in pulp and paper mills are due to exposure to this agent as a component of melamine-formaldehyde and urea-formaldehyde resins used in coating mixtures. These amino resins are used to impart wet strength to paper and are usually added continuously to pulp stock after refining (N02437). These resins may be water-soluble syrups or insoluble powders that are added to the pulp in suspension form (N00917). These resins are formed by reacting urea or

melamine with formaldehyde to form dimethylol urea or trimethylol melamine, respectively. Addition of acid results in a polymerized colloidal dispersion that is heat cured to form the resin. These resins must be used under mildly acidic conditions in the paper machine system (N02437). The desire for wet-strength resins to be used under alkaline conditions has also resulted in the development of polyamide-epichlorohydrin resins.

Formaldehyde is very irritating to mucous membranes of the skin, eye, and respiratory tract, and formaldehyde vapors may produce a contact dermatitis (N00896). Prior exposure of skin to formaldehyde may sensitize the individuals to subsequent exposures. Acute irritation of the respiratory tract has resulted in pulmonary edema, pneumonitis, and death (N02497).

The 1976 NIOSH criteria document on formaldehyde (N02497) reviewed studies in the Russian literature indicating altered visual sensitivity and changes in brain electrical activity at a concentration of 1 mg/m^3. However, the authors of a 1976 NIOSH report entitled "Irritant Effects of Industrial Chemicals" (N02930) concluded that there was no relationship between occurrence of eye disorders and chronic formaldehyde exposure. In addition, no acute effects of formaldehyde on visual function tests were observed in workers exposed to formaldehyde levels averaging 0.5 mg/m^3.

In a 1979 paper, Loomis (N03100) reviewed clinical and animal data on formaldehyde toxicity. He concluded that the only generalization that

could be made from the few chronic studies reported in the literature was that chronic exposure to concentrations greater than approximately 1.2 mg/m^3 would entail significant risk of irritant effects on respiratory passages, eyes, and exposed areas of skin. Formaldehyde possesses both primary irritant as well as sensitizing actions, so that observable effects at or below 0.6 mg/m^3 may be mediated by the immune system. In such cases, no completely safe, innocuous exposure levels can be established and continued exposure may lead to sensitization.

A subject of controversy, Loomis observed, is the degree to which chronic exposure to formaldehyde concentrations that do not produce observable effects may predispose individuals to respiratory infections (N03100). Epidemologic studies provide some positve evidence, but chronic levels of exposure are generally higher than concentrations producing minimal irritative effects. The resolution of this issue by appropriately controlled studies could be of value, especially in view of the widespread exposure of the general population to gaseous formaldehyde from various formaldehyde resins used in paper board and textile products.

A 1961 report (N02706) describes 16 cases of dermatitis in workers in a plant in New Zealand that manufactured melamine-, urea-, and phenolic-formaldehyde resins for paper treatment. Despite wearing protective gloves and barrier creams, workers carrying rolls of treated kraft paper developed erythema of the face (including eyelids, neck, and forearms). Workers handling resins and brushing resin-impregnated sheets developed severe dermatitis of the hands and neck. The highest formaldehyde levels were measured between 20.0 and 37.4 mg/m^3 (5-10 times the OSHA standard).

By contrast, no statistically significant effect on visual function tests of workers were found during exposure to formaldehyde levels that averaged 0.4 ppm, although changes in visual sensitivity and brain electrical activity have been reported in workers exposed to 0.8 ppm (N02497).

(5) Kaolin

Kaolin is the predominant hydrated aluminum silicate clay filler or coating agent used in paper manufacture. It is insoluble in water. Kaolin may act as an allergen, and its toxicity depends upon its crystalline silica content when in dust form (N00919). Dust retention and fibrosis may result from kaolin exposure (N00825). Gosselin reports that mice have been successfully fed diets containing 80% kaolin for experimental purposes (N00596).

(6) Rosin

Various monocarboxylic resin acids of alkylated hydrophenanthrenes comprise commercial rosin (N02437). Rosin is the major sizing (resistance to wetting) material for paper and paper products in the United States. There are three main types of rosin: gum rosin, wood rosin, and tall oil rosin. Rosin sizing is supplied commercially mainly as an aqueous paste, water suspension, or saponified powder which is vigorously mixed with warm water to form an emulsion for use in the paper mill (N02437). Sizing involves the separate addition of

rosin and alum (aluminum sulfate) to the pulp slurry, or to the size press on the paper machine. The alum precipitates the rosin sizing which bonds the pulp fibers.

Rosin is a complex mixture of abietic and primaric resin acids derived mainly from the wood of pine trees (N00917). Rosin has neither an OSHA standard nor an ACGIH recommended exposure level. Toxicity data and information on biologic effects of rosin are scarce. Rosin is said to be an allergen, and its pyrolysis products (as in fumes from soldering fluxes) are tissue irritants (N00919).

In a 1980 study using rats, Zitting (N03244) assessed effects of exposure to rosin fumes on levels of certain enzymes involved in the metabolic detoxification of xenobiotic agents. Male Wistar rats were exposed to fumes from heated rosin of unspecified composition 5 d/wk for 2 weeks. Mean rosin concentration in the exposure chamber was 220 $\pm$ 80 mg/m^3 during the 4 daily 15-minute exposure sessions. One hour after the last exposure session, enzyme levels were measured in lung and liver. Statistically significant decreases in tissue reduced glutathione and microsmal p-nitroanisole demethylase and increases in cytochrome c reductase and ethoxycoumarin-o-deethylase occurred in liver. The only change in lung enzymes was a reduction in the level of ethoxycoumarin-o-deethylase. Since depletion of reduced glutathione has been related to the toxicity of electrophilic compounds, the author suggested that electrophilic agents may exist in rosin fumes.

(7) Silicates and Clays

At least three forms of silica are used as additives in papermaking. Generally, they are biologically inert except when inhaled. In their crystalline form they may be considered dust hazards; their biologic effects are similar to those of other nuisance dusts and will be discussed in the section on dust hazards.

Talc is a mineral product which is essentially a hydrated magnesium silicate, and which may contain calcium carbonate (NO2437). The particle size of industrial talcs ranges from less than 0.5 um to greater than 10 um. Talc is used as a filler in various types of printing paper and in lined boards.

Diatomite is an amorphous silica which may contain some alumina, iron, and magnesium. As a filler, diatomite is either added dry to the pulper or may be pre-slurried (NO2437). Diatomite may also be used as milled or after flux calcining, which changes the physical structure to forms of crystalline silica (cristobalite and tridymite) that are determined by kiln temperature (NO2908).

Chrysotile asbestos and blends of chrysotile asbestos and titanium dioxide are used as paper fillers. These agents are usually supplied in pellet form and are disintegrated by dry crushing before addition to the furnish (NO2437).

In addition to use as a paper filler, asbestos is a component of the felt fabric loops that are used on papermaking machines to carry the sheet of paper or paperboard through various presses and other operations performed on the machine. Two areas where asbestos felts are used are at the wet-end press section and (as canvas-asbestos duck fabric) at the dryer and after dryer sections of papermaking machines (N02437).

It should be emphasized that the potential human hazards of the silicates will depend on the degree to which the materials are contaminated with crystalline quartz silica. Based on x-ray diffraction studies, diatomaceous earths and other amorphous silica materials are frequently contaminated with crystalline silica (N03085). Since even small amounts of crystalline silica can produce a lung fibrosis (N02908), it is necessary that the crystalline silica content of amorphous silica additives be known. As shown in Table VI-2, the recommended concentrations of crystalline silica are lower than those of amorphous silica. It has been recommended that standards for silica exposure be based on human and animal studies using products that are well characterized in terms of presence or absence of crystalline forms of silica, by the use of x-ray diffraction techniques (N03085).

Diatomaceous earth dust may produce fibrosis of the lungs, although this effect is less likely than after exposure to crystalline silica. An oral LD_{50} of 3160 mg/kg for diatomite has been reported for the rat (N00919).

When exposure to crystalline silica occurs, it may include exposure to fibers containing crystals of quartz, tridymite or cristobalite. Silicosis is a form of pneumoconiosis (whose biologic effects were more fully discussed in the section on dust hazards) caused by inhalation and pulmonary deposition of silica. Symptoms include cough, dyspnea, wheezing, and repeated nonspecific chest illnesses (NO2908). The accepted diagnostic lesion of silicosis is a 1-10 mm diameter nodule composed of concentrically arranged bundles of collagen found in the lymphatic system and beneath the pleura in the lungs (NO2908). In animal studies, the most fibrogenic form appears to be tridymite, followed by cristobalite and quartz (NO2908).

(8) Titanium Dioxide

Titanium dioxide is a white pigment that is available commercially in two crystal lattice forms: anatase and rutile. The anatase form was primarily used as a filler in the pulp and paper industry until after World War II (NO2437). Now, this compound is used primarily as a coating pigment. Titanium dioxide and other titanium pigments have usually been purchased by mills in 50 lb kraft bags, printed with water-soluble inks, so that the bag and its contents can form part of the furnish (raw material) added to the beater (NO2437). Although titanium dioxide may be physiologically inert (though possibly posing a dust hazard), the formation of titanium tetrachloride from titanium dioxide during industrial chlorination reactions could pose a hazard. Since bleaching usually precedes the addition of pigments, formation of titanium tetrachloride under these conditions would be unlikely.

Titanium dioxide is considered physiologically inert, as no cases of intoxication have been reported in the literature. It is given a toxicity rating of 1 according to the system of Gleason (N00596), which indicates a probable oral lethal dose in humans above 15 g/kg. A case has been described where no apparent harm or distress resulted from the ingestion of one pound (0.45 kg) of titanium dioxide by a human (N00596). The dusts of titanium may be considered nuisance dusts (N00919), but it is not clear from available evidence whether pneumoconiosis may result from exposure to titanium dioxide (N00916). Some clinical studies, cited in N02945, have reported that long-term exposure to titanium dioxide pigments may produce fibrotic changes in the lungs. Such changes, however, could not be linked to these pigments alone, since the titanium dioxide pigments are usually covered with coatings. In a study using in vitro hemolysis of human erythrocytes as a model for assessing biologic activity of dusts, Zitting and Skytta (N02945) reported that both coated and uncoated rutile pigments hemolyzed erythrocytes only to a degree slightly above that exhibited by a control sample of microgranular cellulose. The anatase form, used in papermaking, produced levels of hemolysis ranging from 20-40%. On the basis of these data and data from a preliminary study assessing the potential of titanium dioxide for fibrosis formation in rat lung, the authors concluded that there is a possibility of potential occupational lung disease due to inhalation of the anatase form of titanium dioxide pigment (N02945).

Various animal studies using titanium dioxide were reviewed by Browning in a 1961 article (cf N00916). Browning indicated that a 1936

study provided evidence for lung fibrosis ("titanicosis") in some animal species that were administered unspecified concentrations of titanium dioxide by inhalation. Although considered inert, certain forms of titanium dioxide pigment (the anatase form) might be biologically active. However, negative results were obtained in a preliminary study where potential fibrogenicity in rat lung tissue after titanium dioxide administration was assessed by measuring proline hydroxylase activity, which has been associated with changes in collagen synthesis (NO2945). This study provided a rather short innoculation period and used small doses of pigments, thus firm conclusions about lung damage due to titanium dioxide could not be made.

Wilska (NO2946) discussed the health hazards of exposure to titanium dioxide pigments. He emphasized the problems of interpreting studies of respiratory effects of titanium dioxide exposure. Pure titanium dioxide does not exist outside of the chemical laboratory. Components used for coating titanium dioxide particles may be of sufficient size to cause lung irritation and damage. Moreover, Wilska (NO2946) indicated that the chemical composition of the coatings may also be implicated in biologic effects of exposure to titanium pigments. Such pigments may contain oxides of aluminium, silicon, and zinc as well as iron, chromium, vanadium, and lead.

(9) Zinc Oxide

Zinc oxide is one member of a class of pigment fillers (NO2437). Its most important use is as a coating in photoconductive

reproduction papers. Particle sizes of zinc oxide used in the paper industry average 0.25 um, with an upper limit of 1.0 um. Most zinc oxide coatings are solvent-based, and incorporate resins, plasticizers, dispersants, and dyes (NO2437).

Reports of skin lesions from prolonged skin contact with zinc oxide have been discussed in the 1976 NIOSH criteria document on zinc oxide (NO2917). In addition, a study by Drinker was described which suggested that metal fume fever may be caused by the inhalation of clouds of zinc oxide powder. Zinc, when heated (as in welding operations), gives off fumes of zinc oxide. These fumes may induce an influenza-like illness whose symptoms include chills, fever, aches, dry cough, and vomiting (NO0596). The criteria document also discussed studies describing similar changes in the lungs of workers exposed to zinc powder or zinc oxide powder. These changes included naso-pharyngitis and laryngitis.

Zinc oxide has been used for many years in the topical treatment of skin disorders and has been shown to be absorbed systemically after topical administration to intact skin of rats and in humans with burns treated with zinc tape (cited in NO3092). Thus, long-term exposure to zinc oxide dusts or materials might produce eventual systemic effects, but the likelihood of this effect from exposure to zinc oxide pigments in the paper industry would appear small.

(f) Contaminants or Byproducts

The major byproducts of sulfate pulping operations are turpentine and tall oil. Both agents are usually recovered and sold for industrial or consumer purposes. Tall oil may be further refined and used in the cosmetics industry and as rosin sizing in the paper industry. Vanillin, one byproduct of acid pulping operations, is used as a flavoring agent and in the pharmaceutical industry.

For purposes of this report, contaminants will be defined as those potentially hazardous chemicals used in the pulp and paper manufacturing operations whose presence is not essential to or desired in the finished product.

(1) Dimethyl Sulfoxide (DMSO)

DMSO is reported to be a byproduct of wood pulping (N00896), formed from the oxidation of dimethyl sulfide by oxides of nitrogen, and found in sulfide waste liquors (N00917). Although DMSO has been used extensively as an industrial solvent, it has received wide publicity in the lay press as a potential topical analgesic and anti-inflammatory agent (N02897). This agent has been under intensive pharmcologic and clinical study because of its potential therapeutic use and as a possible carrier to enhance absorption of other agents (N00896). Clinical trials were suspended by the FDA in 1965 because of reports of cataract formation and teratogenic effects in animals. In late 1966 clinical trials were resumed under strict FDA guidelines mandating that use of

DMSO be restricted to cutaneous applicaton in serious conditions, such as scleroderma and severe rheumatoid arthritis, for which no satisfactory therapy is available (NO2697).

DMSO is a colorless hygroscopic liquid that readily penetrates intact skin and other tissues. Applied cutaneously, DMSO is relatively nontoxic and has a probable human oral LD_{50} greater than 15 g/kg (NO0596). DMSO is relatively nontoxic to animals; even by parenteral routes of administration, the LD_{50} in rodents and dogs exceeds 15 g/kg. After large doses, restlessness occurs followed by coma, hypothermia, and death. Intravascular hemolysis, renal tubular injury, and liver necrosis have been reported in experimental animals (NO0596).

DMSO produces a variety of pharmacologic effects in animals, including reduction of motor activity, potentiation of effects of barbiturates, protection of rats against lethal effects of x-irradiation, and enhancement of stress-related enzyme changes (cf NO2936). In a study using rats, Kocsis and colleagues (NO2936) reported that DMSO enhanced the lethality of benzene and chlorobenzene in mice; however, DMSO decreased the toxicity of the cholinesterase inhibitors paraxon and octamethyl pyrophosphoramide. Dimethyl sulfone, the major metabolite of DMSO (which persists for as long as 3 weeks after percutaneous administration in man), was not effective in increasing the lethality of the benzene solvents in rats.

(2) Hydrazine

One of the uses of this highly toxic carcinogen is as a corrosion inhibitor (N00917). Hydrazine acts as a scavenger by removing dissolved oxygen from aqueous solutions containing corrosive agents (N03291). The extent to which hydrazine is used for this purpose in pulp and paper mills is not known. The data in Table VI-8 indicate that hydrazine was seen in the NOHS in four trade-name ingredient exposures. A full discussion of the toxicity of this agent will be deferred until further evidence of hydrazine's use in pulp and paper manufacturing is obtained.

Hydrazine, a colorless hygroscopic liquid at room temperature, may easily explode when exposed to heat or oxidizing agents (N00917). It is a strong reducing agent that produces toxic effects by all routes of administration in animals and is easily absorbed through the skin. In humans, hydrazine may produce dermatitis and damage to the eye, liver, and red blood cells. Hydrazine is a documented carcinogen in animals, especially in lung tissue (N00919). Other effects in animals include weight loss, fatty liver, kidney and heart anemia, hypoglycemia, inhibition of insulin release, vomiting, impaired renal function, and convulsions.

The 1978 NIOSH criteria document on hydrazines (N03265) recommended a ceiling exposure limit of 0.04 mg/m^3 for any 2-hour period. This recommendation was based on the severity of toxic and carcinogenic effects and on the conclusion that no demonstrably safe level of exposure was apparent from the available evidence.

(3) Kerosene

Pitch is the collective name for the unsaponified resins and fats present in pulping liquors, especially those in acid pulping (N02397). For many years, kerosene was used to control the buildup of pitch in pulping and papermaking equipment, and it is still used to some extent for this purpose. Kerosene is an oily, liquid mixture of petroleum hydrocarbons (mainly of the methane series) with explosive limits in air of 0.7-5.0% (N00917, N00919). Its toxicity is low if ingested, but this can result in gastrointestinal irritation with nausea and vomiting (N00919). The inhalation toxicity of kerosene is high, and aspiration into the lungs during ingestion may be fatal (N00596). An acute, hemorrhagic bronchopneumonia is a characteristic effect of kerosene aspiration.

Due to its low volatility, kerosene is usually not hazardous as a vapor. If high concentrations are inhaled, headache followed by central nervous system depression may occur (N00596). Gosselin et al (N00596) indicated that inhalation of jet airplane fuel, which is similar to kerosene, produces intoxication and that there is a narrow margin between concentrations inducing narcosis and those producing lethal effects.

(4) Polychlorinated Biphenyls (PCB's)

PCB's were first discovered in the pulp and paper industry when folding cartons for packaging dried fruit were analyzed. The source of

PCB contamination was found to be carbonless carbon paperstock, in which PCB was used as a component of the dye carrier system (NO2716). In a study reported in 1973, a variety of grades of pulp, paper, and paperboard from samples furnished by the American Paper Institute (API) were analyzed for the PCB's Aroclor 1242 and 1254 (NO2700). In this study, 11% of the grades of paper contained PCB residues in excess of 126 mg/m^3 (some as high as 1,257 mg/m^3), including virgin bond paper and newsprint.

In a 1975 meeting on PCB's in the environment, one representative of the API indicated that the paper industry merely re-circulates PCB's that are already present in the environment and which are unavoidably included in its manufacturing output (Aroclor 1242) through the use of recycled paperstock that may contain PCB's (NO3133 Abst). At this same meeting, a representative of the Wisconsin Paper Council stated that of the commercially available PCB's, those involved in carbonless paper were among the least stable, least environmentally persistent, and least susceptible to bioaccumulation (NO3141 Abst).

As indicated in the 1977 NIOSH criteria document (NO2926), PCB's are mixtures of chlorinated biphenyls that have a variety of biologic effects in humans, including digestive disturbances and anorexia, eye irriation, chloracne, skin and nail pigmentation, liver damage, impotence, and the potential for inducing carcinogenic and teratogenic effects. Their primary effects are on the skin (chloracne) and liver (NO0917).

As with certain pesticides such as DDT, some PCB's (such as Aroclor 1254) inhibit human leucocyte functions (mitogenic response to

phytohemagglutinin). On the basis of in vitro experiments, it has been proposed that inhibition of mitochondrial respiration, which leads to a decrease in intracellular adenosine triphosphate concentration in lymphocytes, may be responsible for Aroclor's effect on leucocyte function (NO3106).

Due to their complexity, the toxicity of PCB's is difficult to determine. Splenic atrophy and chemical porphyria appear to be general responses to PCB ingestion (NOO596). PCB's are stored in body fat, and their toxicity may be potentiated by halogenated hydrocarbon solvents such as carbon tetrachloride (NOO596). Dermal toxicity in animals is higher than that produced by other routes of administration. Adverse effects of PCB's from general environmental contamination have not been reported. However, a disease known as "Yusho" was reported in Japan in 1968 which was due to ingestion of rice bran oil contaminated with PCB's. Signs of the disease included acneiform eruptions, secretion from meibomian glands of the eye, hyperpigmentation of skin, nails, and mucous membranes, swelling of the upper eyelids, and hyperemia of the conjunctivae (NO2926).

(5) Polynuclear Aromatic Hydrocarbons

Polynuclear aromatic hydrocarbons, including benz(a)anthracene, benz(a)pyrene, fluoranthene, phenanthrene, and others have been identified in an analysis of sodium sulfate dust from the electrostatic precipitator of a recovery furnance in a pulp mill (NO2678). The extent to which actual and potential carcinogens are represented in sulfate pulping effluents, in general, is not known.

(6) Tall Oil

Tall oil (tall oil rosin), or tallol, is one of the major recovered byproducts of the treatment of pine wood with the alkaline digesting liquor of the kraft (sulfate) pulping process. Tall oil is the material that is separated from the soap that is formed when the rosin acids and fatty acids of wood from pine trees react with the alkaline salts of the digesting liquor (NO2955). In addition to rosin acids (primarily abietic acids) and fatty acids, tall oil contains an unsaponifiable fraction consisting of long-chain alcohols and sterols (NO0896).

Tall oil is a dark brown liquid with an acrid odor and is a mild allergen (NO0917). Tall oil has neither an OSHA standard nor a TLV adopted by ACGIH. A literature review, summarizing available scientific data on the safety of tall oil as a food ingredient, was published in 1973 (NO3217). The lowest reported acute LD_{50} for oral administration of tall oil rosin was 4,600 mg/kg in guinea pigs and mice. Toxicity of various esters of tall oil fatty acids in rats was less than that of tall oil rosin; reported LD_{50} values for oral administration ranged from 10,000-53,800 mg/kg in rats.

Lifetime carcinogenicity studies in mice using epoxidized tall oil products indicated a lack of tumorigenic potential of tall oil (reviewed in NO3217). These negative results were confirmed in dogs and rats fed 1% refined tall oil rosin in their diet over a 2-year period. Mutagenic effects of oxidized oleic acid were observed in chromosomes of mouse bone marrow cells.

(7) Turpentine

Crude or sulfate turpentine, recovered from pulp digestion in the kraft process, is another major byproduct of sulfate pulping where wood from pine trees of high resin content is used. Composition of this turpentine is approximately 50-60% alpha-pinene, 15-20% beta-pinene, and 10-15% monocyclic terpenes (NO2381). Other components may include limonene, phellandrene, and Δ^3-carene. The terpene p-cymene is also a component of sulfite turpentine formed during acid pulping of firs and spruces (NO2668).

Turpentine is irritating to eyes, nose, and bronchi, and contact dermatitis may result from exposure to the liquid (NO3143).

In a study using a selected group of patients suspected of having allergic eczematous contact dermatitis, tests of allergic reactions to test substances were carried out with standard trays of contact allergens in an unspecified manner (NO2951). Allergic responses were graded on a scale of 0 to 4. Of the 66 patients receiving a 10% suspension of turpentine in olive oil, 50 exhibited reactions in the lowest two categories of severity, and 16 exhibited reactions of moderate severity. The authors concluded that weak reactions, such as these, are evidence of allergic sensitization but are below the level for the occurrence of clinical (skin) disease.

Vapor inhalation and percutaneous absorption are the most frequent routes of occupational exposure for turpentine. Both routes of exposure

depression and kidney and bladder damage (chronic nephritis with albuminuria and hematuria) (N03143). Specific symptomatology includes: burning pain in throat; coughing and choking; dyspnea; transient excitement followed by ataxia, delirium and stupor; fever; and tachycardia. Odor of turpentine may be on breath and urine may have an odor of violets. Death is usually due to respiratory failure (N00596). Aspiration may lead to pulmonary edema and pneumonitis (N00596). Since turpentine is readily absorbed from skin, respiratory tract, and gut, many accidental poisonings have been reported. Ingestion of as little as 15 ml has been fatal to a child (N00596).

Turpentine is a gastrointestinal tract irritant in animals and may produce pneumonia after aspiration (N03186). Chronic low-level exposure induces hepatic microsomal enzymes in rats (N00596), and this mechanism appears responsible for the decreased barbiturate sleeping time reported in rats pretreated with turpentine, as reported by Sperling and Ewenike (cf N02949).

In a 1969 review of the toxicology of turpentine, Sperling (N03135) indicated that central nervous system effects induced by nonlethal vapor exposures appeared to be similar in various species. However, concentrations required to produce similar effects varied. For example, ataxia and convulsions were observed in guinea pigs, rabbits, and cats exposed to 4-6 mg/l for at least 1 hour; rats and mice required longer exposures for occurrence of similar effects.

Savolainen and Pfaffli (N02949) assessed neurochemical effects of exposure of male rats to 1,697 mg/m^3 (12 umol/liter) of commercial turpentine for 8 weeks, 6 h/d. Brain, blood, and perinephric fat samples were taken from experimental and sham-exposed animals 1,4,5,6,7 and 8 weeks after beginning the experiment. Although no behavioral changes were observed, and food and water intake was similar to that of controls, exposed animals exhibited metabolic and enzymatic changes. These included accumulation of alpha-pinene in fat and brain, and reduction in brain RNA content after 1 week of exposure. Serum nonspecific cholinesterase activity showed a transient decrease. The authors concluded that despite an exposure level one-seventh the LC_{50} for a 6 hour exposure, turpentine accumulated in the body, especially in fat, and produced neurochemical changes. However, the body solvent content may remain stable despite continued exposure, and no appreciable chronic effects were measured within 8 weeks of exposure. The authors speculated that oxidative removal of the solvent may be small with respect to turpentine load, and that the relatively minor effects observed were due to the lack of formation of potentially neurotoxic oxidized intermediates. It is claimed that turpentine oxidation products (at least those produced by prolonged exposure to air) are more toxic and irritating than turpentine itself (N00596).

(8) Vanillin

Vanillin (3-methoxy-4-hydroxybenzaldehyde) is a byproduct extracted from lignin and found in sulfite pulping waste liquor (N00917). It is used as a flavoring and in the manufacture of perfumes and pharmaceuticals.

Vanillin in crystalline form is slightly soluble in water and freely soluble in organic solvents and caustic solutions (N00808). This agent has been reported to exhibit low toxicity, but ingestion of an unspecified high dose has resulted in hyperpnea, dyspnea, muscular weakness, and death due to circulatory failure (N00596). The lowest probable oral lethal dose in humans is reported as 500 mg/kg (N00893). The ACGIH has not published a recommended exposure level for vanillin.

(g) Pulping or Combustion Effluents

Major pulping effluents include sulfur dioxide and total reduced sulfur (TRS) compounds, including hydrogen sulfide, methyl mercaptan, dimethyl sulfide, and dimethyl disulfide. Emission levels of these agents have been reduced to conform with regulations established under Section III(d) of the Clean Air Act, as prescribed by the Environmental Protection Agency (EPA). Criteria by which states may develop emission standards for existing kraft pulping mills are described in a recent EPA publication (N02571).

Even with reduced levels, emissions from digesters, recovery furnaces, and other equipment where pulping effluents are generated pose a health hazard. Sensory adaptation may occur to agents such as hydrogen sulfide, even at high concentrations, and exposure may be prolonged or may occur at high concentrations without any warning of an impending toxic reaction.

TRS emissions from kraft pulping may be merely the more obvious hazard from this operation. Polynuclear aromatic hydrocarbons, including benzopyrenes and anthracenes, have been qualitatively identified in an analysis of sodium sulfate dust from the electrostatic precipitator of a recovery furnance (N02678). The extent to which such agents would be routinely found in kraft process maintenance operations is not known.

(1) Carbon Monoxide

The human health hazards associated with exposure to carbon monoxide (CO) are well known and documented. According to the NIOSH publication Occupational Diseases (N03149), 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 carbon monoxide 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 (N03149). 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, and the 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. Exposure of individuals with coronary artery disease to CO concentrations resulting in carboxyhemoglobin levels greater than 5% may produce deleterious effects on the heart, including restricted coronary artery blood flow and decreased myocardial lactate production (N02723, N03149).

The factors cited above, plus evidence cited in the 1972 NIOSH criteria document on carbon monoxide, led to a NIOSH recommended 8-hour time-weighted average exposure level of 41 mg/m^3 (N03142). Although this standard was based, in part, on CO cocentrations that were not expected to increase employees' carboxyhemoglobin levels above 5%, carboxyhemoglobin levels in chronic cigarette smokers are purportedly at or near this level without additional environmental exposure (N02723, N03142).

The NIOSH criteria document on CO also indicated that there is disagreement about conditions of CO exposure that produce specific biologic effects, especially at concentrations below 117 mg/m^3 (N03142). In particular, assessment of neurophysiologic effects of CO exposure by analysis of electroencephalograms, visual evoked responses, and conditioned behavioral responses may produce different conclusions.

(2) Dimethyl Disulfide

No reports of human health effects caused by exposure to dimethyl disulfide were found. Data on the human effects are exceedingly limited. The National Fire Protection Association reported in its Fire Protection Guide on Hazardous Materials (N00733) that dimethyl sulfide has a disagreeable odor and is a moderate eye irritant. The fourth edition of Dangerous Properties of Industrial Materials (N03153) stated that toxicity details are unknown, but that dimethyl sulfide is probably highly toxic. It also reported that alkaline sulfides are similar in

action to alkalis; they cause softening and irritation of the skin and are corrosive and irritating to moist tissues because they liberate hydrogen sulfide and free alkali.

Data from animal studies indicate that exposure of animals to high concentrations of dimethyl disulfide may result in pulmonary edema, convulsions, and respiratory arrest (N03096).

(3) Dimethyl Sulfide

Dimethyl sulfide (DMS) is a minor metabolite present after the administration of dimethyl sulfoxide (DMSO) in several species (N02935). Since DMS may also be converted to DMSO after DMS administration (N02935), studies were performed to determine whether similar biologic effects resulted after administration of DMS, DMSO, and dimethyl sulfone (N02936). DMS, in a dose of 1.25 g/kg, was as effective as 5.0 g/kg of DMSO in reducing body temperatures of rats 1 hour after parenteral administration of these agents. DMS was also more effective than DMSO in reducing motor activity; DMS, in a dose of 0.5 g/kg, reduced running-wheel activity of mice to 5% of control values, whereas 5.5 g/kg of DMSO reduced activity to 14% of control values. Unlike DMSO, however, DMS was ineffective in antagonizing the lethal effects of cholinesterase inhibitors in mice (N02936). The authors concluded that additional research is needed to determine to what extent the metabolites of DMSO (such as DMS) share or are responsible for effects reported for DMSO.

Studies of chronic toxicity of DMS are scarce. Butterworth et al (NO2935) studied the short-term toxicity of DMS in male and female rats receiving various daily oral doses for 14 weeks. No effects of DMS, in doses up to 250 mg/kg day, on body weight gain or food and water intake were observed. Serum enzyme levels and results of hematological examinations and renal concentration tests were within control levels. Although some organ weights at autopsy were found to differ between experimental and control groups, the effects were not dose-related and were not reliable for both sexes.

(4) Hydrogen Sulfide

The 1977 NIOSH criteria document on hydrogen sulfide (NO2493) stated that hydrogen sulfide exposure is a leading cause of sudden death in the workplace. Brief exposure at high concentrations have caused conjunctivitis and keratitis, and exposures at very high concentrations have caused unconsciousness, respiratory paralysis, and death. Though the criteria document reported that conclusive evidence of adverse health effects from chronic, low-level exposures to hydrogen sulfide were not found, there is some evidence that hydrogen sulfide in low concentrations, either by itself or in combination with other chemicals, has caused nervous system, cardiovascular, and gastrointestinal disorders, and effects on the eyes.

NIOSH's Occupational Diseases (NO3150) reported that palpebral edema, bulbar conjunctivitis, keratoconjunctivitis, and ocular lesions may occur when hydrogen sulfide contacts the eyes. In addition,

photophobia and lacrimation may also develop. Exposures may cause direct irritation of the respiratory tract, which may result in rhinitis, pharyngitis, bronchitis, and pneumonia. If hydrogen sulfide penetrates deeply into the lungs, such as might occur in a hyperventilating worker, hemorrhagic pulmonary edema may result. Reportedly, the irritative effects of hydrogen sulfide are due to the formation of alkali sulfide when the gas contacts moist tissues.

Occupational Diseases (N03150) compiled and reported the following systemic harmful effects of hydrogen sulfide exposure. Acute exposure may cause immediate coma, with or without accompanying convulsions, and death may result from respiratory failure. Subacute exposures generally result in headache, dizziness, staggering gait, and excitement suggestive of neurologic damage, and nausea and diarrhea suggestive of gastritis. In addition, tremors, weakness, and numbness of the extremities may result, and frequently poisoning victims exhibit abnormal electrocardiograms. It is currently thought that the toxic actions of hydrogen sulfide are due to its ability to bind iron and thus inhibit the cytochrome oxidase system, which is essential for cellular respiration.

The NIOSH criteria document on hydrogen sulfide (N02493) stated that the effects of hydrogen sulfide on humans and animals are similar. The criteria document concentrated, therefore, on studies which have firmer data on environmental concentrations than most human case studies and on studies that described results which could not ethically be obtained with human subjects. The most notable of the studies reported was one in which three rhesus monkeys were exposed to hydrogen sulfide

concentrations of 709 mg/m^3 for varying (less than 25 continuous minutes) amounts of time. Autopsies revealed extensive neuropathology including necrosis of the parietal and occipital cortex, necrosis, hyperemia, and gliosis of the basal ganglia, a decrease in the number of Purkinje cells in the cerebellar cortex, and isolated accumulation of glial cells in the cerebellar folia. One monkey exhibited moderate hyperemia of the liver. The authors concluded that the brain, particularly the motor cells of the cerebellum, was the principal target of inhaled hydrogen sulfide.

A recent (1980) study by Savolainen et al (N03127) examined the biochemical effects of repeated subclinical hydrogen sulfide intoxications in the brains of mice. The mice were exposed 4 times to hydrogen sulfide, at a concentration of 142 mg/m^3 for 2 hours at 4-day intervals, and showed increasing inhibition of the cerebral cytochrome oxidase activity. The authors concluded that the effects of repeated exposures to hydrogen sulfide were, in some respects, cumulative. They hypothesized that similar inhibition of cytochrome oxidase might also take place in the human brain, which could lead to the increased headache frequency, increased fatigue, and slow deterioration of mental functions sometimes seen in chronic human hydrogen sulfide toxicity.

(5) Methyl Mercaptan (methanethiol)

The 1978 NIOSH criteria document on monothiols (N02907) stated that the primary reason for concern about employee exposure to thiols (mercaptans) was that they caused mucousal irritation, respiratory

changes leading to respiratory failure, and muscular weakness culminating in paralysis, mild to severe cyanosis, coma, and death.

Occupational Diseases (NO3145) reported that, in addition to the irritating effect of mercaptan vapors on skin, eyes, and mucous membranes of the upper respiratory tract, liquid mercaptans may cause contact dermatitis. This same source reported that, in terms of systemic effects, methyl mercaptan acts toxicologically like hydrogen sulfide and may depress the central nervous system, resulting in respiratory paralysis and death. Also, individuals that survive severe exposures may suffer from headache, dizziness, staggering gait, nausea, and vomiting. In cases where there is moderate to severe respiratory tract irritation, pulmonary edema may develop and there is some possibility of renal and hepatic damage. In at least one well documented case of methyl mercaptan poisoning (cited in NO3145), the victim developed acute anemia and methemoglobinemia.

The NIOSH criteria document on monothiols (mercaptans) (NO2907) reviewed a number of animal toxicity studies dealing with the effects of inhaled methanethiol (methyl mercaptan). Of the studies reviewed, several utilized very high exposure concentrations (ie, greater than 2,000 mg/m^3). Because of the exceedingly high concentrations used in these experiments, the results obtained do not contribute to the understanding of hazards presented by the concentrations of methyl mercaptan usually encountered in the workplace. The criteria document (NO2907) does, however, review subchronic toxicity studies in which animals are exposed to levels of methyl mercaptan similar to those which

a worker may encounter in the workplace. Among the effects observed in rats in these studies were desynchronization of the EEG, persistent disturbances of breathing, irregular heartbeats, and alterations in biochemistry. Such alterations included increased concentrations of carbon dioxide, SH, cholesterol, and lactic and pyruvic acids in the blood. Also observed were a decrease in the rats' rate of growth, increased weight of the heart, and altered distribution of corticosteroids in the adrenal tissue.

In another study reviewed in the criteria document (NO2907), rats were exposed to methyl mercaptan at 98 $\pm$ 9.8 mg/m^3 for 90 days. The exposed animals exhibited a number of hematologic changes, when compared with values determined before the experiment, including statistically significant elevations of their reticulocyte counts, hematocrits, hemoglobin concentrations, mean corpuscular volumes, and mean corpuscular hemoglobin values. In addition, when the experimental values were compared with the values from the control animals kept under duplicate conditions for the full 90 days, statistically significant decreases in erythrocyte and platelet counts and hemoglobin concentration and increases in leukocyte and reticulocyte counts were observed. The criteria document (NO2907) reported the experimenters concluded that the hemotologic changes observed could have resulted from the stresses to which the animals were subjected but considered the effects slight.

Studies concerned with the inhibition of mitochondrial respiration by methyl mercaptan in biochemical preparations have appeared in the literature in recent years (NO3097, NO3098). The primary reason for this

activity is that methyl mercaptan has been implicated as being at least partially responsible for the pathologic symptom known as fetor hepaticus, the unpleasant breath-odor of comatose patients with severe liver disease (N03098).

(6) Sulfur Dioxide

The OSHA Economic Impact Statement for sulfur dioxide (N02546) reported that symptoms of acute exposure to sulfur include irritation of the eyes, mucosal membranes and upper respiratory tract, and that potential effects of chronic exposure include nasopharangitis, reduction of pulmonary function, increased resistance to air flow, and promotion of cancer.

According to the 1974 NIOSH criteria document on sulfur dioxide (N02927), the rapidity with which sulfur dioxide forms sulfurous acid on contact with moist, mucous membranes explains its prominent biological effect in man: severe irritation. The criteria document also stated that in response to pulmonary irritation caused by inhalation of sulfur dioxide, reflex bronchoconstriction with possible resulting increase in pulmonary flow resistance and increase in mucus secretion results. Also reported were cases of suppurative bronchitis, influenza, and asthma-like attacks, and, where the concentration of sulfur dioxide was extremely high, asphyxia or severe chemical bronchopneumonia with bronchiolitis obliterans.

The criteria document on sulfur dioxide (NO2927) observed that although a considerable amount of experimental work has been reported on exposure of animals to sulfur dioxide, much of the information has been duplicated by human experiments, especially at exposure levels which are pertinent to the development of an occupational exposure standard; that, in general, man is considered to be more sensitive than other mammals to the effects of sulfur dioxide in ranges commonly employed experimentally, with the possible exception of the domestic cat; and that the effect of sulfur dioxide on all mammals is qualitatively the same--that of respiratory and mucous membrane irritation and reflex bronchoconstriction with increased airway resistance.

As a further indication that animal studies tend not to provide pertinent information about the human health hazard associated with exposure to sulfur dioxide, OSHA's economic impact statement on sulfur dioxide (NO2546) reported a study in which guinea pigs were exposed to sulfur dioxide in concentrations of 0.3, 2.7, and 15.3 mg/m^3 continuously for 1 year. At the end of the exposure period no changes were found in a variety of pulmonary and cardiac function measurements, blood and clinical chemical studies, gas uptake, or histology of the respiratory tract that were attributable to the sulfur dioxide exposure.

Carcinogenicity, Mutagenicity, and Teratogenicity

(a) Asbestos

The carcinogenicity of various commercial forms of asbestos has been studied in different animals species via various routes of administration.

(1) Inhalation and Intratracheal Installation

Experiments performed on mice did not provide conclusive evidence of the carcinogenicity of asbestos. For instance, in 1957 Lynch et al (NO3176) exposed AC/Fl hybrid mice to commercial chrysotile. Multiple pulmonary adenomas were observed in 45.7% of the experimental group as compared to 36.0% of the control. The difference in the incidence of adenoma occurrence between the control and experimental groups was not statistically significant. Experiments performed by Reeves et al (NO3175) in 1974, in which Swiss mice were exposed to 50 mg/m^3 of crocidolite, amosite, or chrysotile for 4 h/d, 4 d/wk for 2 years, resulted in papillary carcinomas in 2 of the animals exposed to crocidolite and in 1 of the animals in the control group.

Rats, on the other hand, showed more susceptibility than mice to the carcinogenic effect of asbestos. Carcinomas of the lung, adenocarcinomas, and squamous cell carcinomas were reported in rats exposed to 86 mg/m^3 of chrysotile, 30 h/wk, for 16 months (Gross et al, 1967 (NO3174)); in 1971, Reeves et al (NO3173) reported on the squamous carcinoma of the bronchus in 2 of 31 rats exposed to 49 mg/m^3 of crocidolite for 16 h/wk for 2 years; and on pulmonary adenomatosis in 5 of 40 animals exposed to chrysotile. Interestingly, in 1974 Reeves et al (NO3175) found no tumors in Charles River CD rats exposed 16 h/wk for 2 years to 50 mg/m^3 of crocidolite, amosite, or chrysotile. Reeves' results are in sharp contrast with those of Wagner et al (NO3172), who in 1974 reported results of experiments performed on CD Wistar rats exposed to Canadian chrysotile at a level of 12 mg/m^3, 7 h/d for 5 d/wk, for

periods of 1 day and 3, 12 and 24 months. Eleven animals showed mesotheliomas of the lung, 2 of which were exposed for 1 day only. Similar results were reported later by the same authors (Wagner et al, (N03156)).

Experiments in which chrysotile asbestos was installed intratracheally were designed to study the co-carcinogenesis of asbestos with benzo(a)pyrene. Shabad et al (N03171) found lung papillomas, epidermoid carcinomas, reticulosarcomas, or pleural mesotheliomas in rats injected intratracheally with Russian chrysotile on which benzo(a)pyrene was adsorbed. No lung tumors or mesotheliomas were observed in animals injected with chrysotile or benzo(a)pyrene alone, during or up to 28 months after the administration.

(2) Intrapleural Injection

Intrapleural injection of various types of commercial asbestos led to the formation of mesotheliomas in CD Wistar rats. Doses ranged from 0.5 mg of chrysotile or crocidolite (Wagner et al (N03172)) to 40 mg of asbestos dust (Stanton and Wrench, 1972 (N03170)), and even included 60 mg of Russian chrysotile (Pylev and Shabad (N03155)). The incidence of mesotheliomas varied from one type of commercial asbestos to the other; Wagner et al (N03172) in 1974 found that the highest incidence (61%) occurred with crocidolite and the lowest (19%) with Rhodesian chrysotile; in between were amosite (36% incidence), anthophyllite (34%), and Canadian chrysotile (30%).

Asbestos injected intrapleurally produced mesotheliomas in various species, eg, rats (Donna (N03169); Reeves et al (N03174), hamsters (Smith et al (N03168)), and rabbits (Reeves et al (N03174)).

Intrapleural injection of other materials such as glass fiber, in a fiber form comparable in diameter to that of asbestos, induced mesotheliomas, as reported by Stanton (N03154) and Wagner et al (N03156).

(3) Intraperitoneal Injection

A suspension of chrysotile or crocidolite injected intraperitoneally in Charles River CD rats in doses of 0.3, 0.5 or 1.0 ml induced peritoneal mesotheliomas (Reeves et al (N03173)). Similar results were obtained by Maltoni and Annoscia (N03187) using Sprague-Dawley rats.

(4) Oral Administration

Experiments in which chrysotile was given orally (mixed with malted milk powder) in a dose of 100 mg/day for 100 days resulted in 1 of 32 Wistar SPF rats developing gastric leiomyosarcomas; no tumors were observed in any of the 16 control rats that were fed malted milk only (Wagner et al (N03156)).

There is growing evidence that 90% of asbestos related cancers are the result of the co-carcinogenic effects of asbestos inhalation and cigarette smoking. To prove this theory, Lakowitz and Bevan (N03083,

N03116) studied the microsomal availability of benzo(a)pyrene when adsorbed on fibrous (anthophyllite and Canadian chrysotile) and nonfibrous (hematite and silica) particulates. They found that adsorption of benzo(a)pyrene to all four mineral particulates resulted in enhanced microsomal availability of benzo(a)pyrene, and that fibrous minerals are superior to nonfibrous ones in increasing the microsomal uptake of benzo(a)pyrene.

Studies on occupational exposure of miners and millers to talc containing asbestos demonstrated an increased number of deaths ascribed to bronchogenic cancer (N02906). Exposure to asbestos was found to be an occupational hazard (N03068).

Chromosomal aberrations in Chinese hamster cells were observed after the addition of 0.01 mg/ml of chrysotile and crocidolite to the culture medium (Sinock and Seabright, (N03167)).

(b) Formaldehyde

In 1963 Horton et al (N03179), studying lung changes from exposure to formaldehyde, exposed a group of mice to 50 mg/m^3 of formaldehyde, 1 h/d for 35 weeks; after 35 weeks, 23 of the mice were killed. The 37 remaining mice were then exposed to 150 mg/m^3 for an additional 35 weeks. Microscopic examination revealed early structural changes in the respiratory tissues but no tumors were found. The experiment was not of sufficient duration to justify the negative findings.

Formaldehyde, when present at high concentrations (624 to 3,747 mg/m^3), reacts with inorganic chlorides or hydrogen chloride to form bis-chloromethyl ether (BCME), which is a potent carcinogen. However, at the low concentration encountered in the industrial environment, no evidence of BCME formation was found (Tou and Kallous, (N03178)).

To determine the effect of formaldehyde on reproduction, Gofmekler (N03177) exposed female rats to 0.012 and 1 mg/m^3 for 10-15 days before impregnation. In both groups, mean duration of pregnancy was increased by 14-15% over that of the control. Offspring from exposed dams showed higher total body weight and weight of adrenal glands than those of the controls or control group. However, the lung and liver weights of the offspring of the exposed group were less than those of the control groups.

Formaldehyde was tested for mutagenicity in the Salmonella typhimurium test system using two strains of TA 100 (induction of substitution) and TA 98 (induction of frame shift) (N03102 Abst). The mutagenicity of formaldehyde was very weak and appeared only within a limited range of concentration.

(c) Aroclor 1242 and Aroclor 1254.

Aroclor, a trade name for polychlorinated biphenyls, is usually followed by a number, the last two digits of which denote the percent by weight of chlorine in the mixtures, eg, Aroclor 1242 and 1254 contain 42% and 54% chlorine, respectively. However, regardless of the degree of chlorination, all adequately tested PCB's have shown carcinogenic

activity. Since Aroclor 1242 and 1254 are the PCB's found as contaminants in the pulp and paper industry, carcinogenicity studies using these two compounds are of interest. In these studies both mice and rats developed hepatomas after ingesting Aroclor 1242 and 1254 for a specified period of time. In 1974, Kimbrough and Linder (N03184) fed BALB/cj male mice with 300 ppm of Aroclor for 11 months. Nine of 22 animals developed hepatocarcinomas and neoplastic nodules; only 1 mouse of 173 control groups developed hepatocarcinoma.

Studies reported in the Federal Register (42:6532-55, 1977) showed that rats receiving 108 and 1,079 mg/m^3 of Aroclor 1242, and 126 and 1,257 mg/m^3 of Aroclor 1254, for 24 months, developed hepatomas and cholangiohepatomas. At 108 mg/m^3 of Aroclor 1242, 2 of 10 animals developed liver tumors; at 126 mg/m^3 of Aroclor 1254, 3 of 26 developed liver tumors. Animals which received the higher concentration showed a higher rate of liver tumors: 11/20 and 19/27 for Aroclor 1242 and 1254, respectively. However, in another study (N02926), sponsored by the National Cancer Institute (NCI), Aroclor 1254 fed to rats at levels of 314, 629, and 1,257 mg/m^3 failed to induce any tumors in female rats; in male rats, only 1 animal from the group that received 629 mg/m^3, and 2 from the group that received 1,257 mg/m^3, developed hepatocellular carcinomas. Leukemias were also reported (N02926).

Berry et al (N03110) studied the tumor promoting ability of Aroclor 1254 in a two-stage mouse skin tumorigenesis assay. When given in a dose of 100 ug twice weekly, Aroclor was not a promoter of skin tumors.

In 1977 Kerkvliet and Kimeldorf (N03111), utilizing the transplantable Walker 256 carcinosarcoma in rats, demonstrated that Aroclor 1254, given orally at levels of 1,257-10,059 mg/m^3, not only is devoid of tumorigenic activity but inhibits the growth of this type of transplantable tumor in rats.

PCB's have little mutagenic potential. However, Popper et al (N03183) claimed that PCB's may alter the mutagenicity and carcinogenicity of other compounds. Administration of Aroclor 1254 for 7 days at 50 mg/kg/day to male rats failed to induce any significant chromosomal changes in the testes of these animals (Dikshith et al (N03182)). In another experiment (Green et al (N03181)), neither Aroclor 1242 nor Aroclor 1254 produced any chromosomal aberrations in spermatagonial or bone marrow cells of rats given 500 mg/kg/day of Aroclor 1242 for 4 days, or 300 mg/kg/day of Aroclor 1254 for 5 days.

Shahin et al (N03105) also found that Aroclor 1254 was not mutagenic in TA 1538 or TA 98. However, the presence of PCB's in Aroclor-induced rat-liver homogenate (induced S9) was identified. These results were not shared by Zeiger et al (N03104), who found no effect due to Aroclor 1254 on liver-microsomal or S9 protein concentrations. However, they found that benzo(a)pyrene mutagenicity was enchanced with Aroclor 1254 treatment (N03104).

Two-generation feeding studies, in which Aroclor 1254 was given in a range of 13-1,257 mg/m^3, were performed by Linder et al (N03180). No terata were reported, although animals that received 251-1,257 mg/m^3 of Aroclor 1254 showed reduced litter size.

(d) Wood Dust

Both epidemiologic data and experimental studies in animals have shown that exposure to wood dust is apt to produce nasopharyngeal tumors. In 1968, Gignoux et al (N03185) reported on 52 patients with malignant tumors of the ethmoid and maxillary sinuses, 16 of whom were woodworkers. Fifteen of the 16 tumors were adenocarcinomas.

(e) Titanium Dioxide

Exposure of Fischer 344 rats and B6C3F1 mice of both sexes to 83,091 or 166,181 mg/m^3 of titanium dioxide in food for 104 weeks did not result in a signficant increase in the number of tumors of treated groups over the control group (N03039).

(f) Heat

By virtue of their ability to regulate body temperature, homeotherms can accommodate reasonable changes in environmental temperature. Although the effects of heat on various physiologic systems of both animals and man have been well studied, little attention has been given to the effects of heat on fetuses. Both animal and human studies are scarce, and any teratogenic effect of heat is still to be determined.

Knecht et al (N03196) studied the effect of periodic short-term exposure to heat on Charles River rats. Animals were exposed in a chamber maintained at 38.2 $\pm$ 0.7 C and 50 $\pm$ 8% relative humidity, for a

period lasting from 2 weeks before gestation to 21 days postpartum. There was no observable effect on the rate of copulation or conception nor on the duration of gestation, when compared with control groups. Heat had no significant effect on the litter size at birth, although mean pup weights from heated dams were significantly lower at birth than those of the controls. Also, there was a fourfold increase in the number of dams with multiple resorption in the heated group, when compared with the control group. Similar results were obtained when males were exposed to heat for 4 weeks prior to copulation. No deformities were reported. There was no mention by the authors of any food analysis for compounds that could produce the same effect, nor was there mention of a hormonal level estimation being performed. It could be speculated that heated animals will have less of a tendency to consume food than the control animals. Starvation could very well lead to the effects reported in the experiment.

Pont (N03197) reported unsubstantiated deformities in kittens whose mother during pregnancy had the habit of stretching out on top of a storage radiator. However, there is no scientific evidence for this conclusion.

Observations of the effect of heat on humans have led to substantial controversy. Miller et al (N03199) attributed the anencephaly of seven infants to hyperthermia: five mothers had fevers ranging from 38.9 to 40.0 C at the time of the closure of the anterior neural-grove, and two had possible hyperthermia episodes as a consequence of sauna bathing. The correlation of the cause of fetal anencephaly to

heat in the mothers is not clear. Careful examination of these five cases revealed that the fevers were associated with five different illnesses: "flu-like" respiratory illness, pyelonephritis, streptococcal pharyngitis, and viral gastroenteritis, and one fever of unknown etiology. Such conditions could very well have been due to the toxins present in the body and not to heat per se. As for the two cases of teratogenicity that were blamed on sauna bathing, it is worth referring to the letter by Rapola et al (NO3198). The authors claim that Finnish mothers enjoy the sauna during the susceptible period, yet there is a very low incidence (0.032%) of anencephaly in Finland. In fact, they advocated that the "joy of a sauna bath should not be banned to pregnant women or women of childbearing age."

Epidemiologic Studies

The visibility of the pulp and paper mill industry is high, both among the public and the scientific/medical community. The subjective olfactory offensiveness of the environment in and around a pulp and paper mill (NO2554) may result in the public's association of a personal sensory experience to an apparent health risk. Likewise, the geographic distribution of wood-using industries (construction, furniture, paper) has prompted investigations to associate wood, wood dust, and the manufacture of wood products with the occurrence of disease.

Blot and Fraumeni (NO2721) and Mason (NO2561), using geo-coded death certificates for a correlation of malignant neoplasm mortality and industrial activity, have suggested broad associations between some

industries and particular cancers. Among their suggestions is an association between lung cancer and wood-using industries. With further refinement of this geo-coding methodology, Harrington et al (NO2508) have shown a urban-rural difference in risk for lung cancer in wood-using occupations and industries. They reported no elevated risk for lung cancer in urban workers in the wood and paper industries but a threefold increase in risk in rural workers in the wood and paper industries. The usual epidemiologic expectation is for urban risk to be higher than rural risk, especially in the same industry/occupation classification.

In an earlier study (1968), Acheson (NO2776) implicated increased risk for nasal adenocarcinoma from wood (dust) exposure and stated that the polishes, lacquers, and varnishes were unlikely to be implicated etiologically. Also, Milham (NO3134, NO2724) reported an increased risk of not only Hodgkins disease but also other malignant neoplasms in wood-using industries, specifically the pulp and paper mill industries. Milham based his studies only on death certificate data for the underlying cause-of-death and reported lifetime occupation.

Ferris et al's (NO2733) (1979) completion of a cohort follow-up study of a previously studied pulp and paper mill workplace (NO2536) showed no increase in morbidity or mortality in the group. Though the methodology was prudent, the small population and an observation period of only 10 years diminish the statistical value of their conclusion. Ferris et al's (NO2733) approach, however, is more acceptable in epidemiologic terms than the global association of Blot and Fraumeni (NO2721) or Milham (NO3134, NO2724).

Nonmalignant respiratory dysfunction has also been reported in pulp and paper mills (N02698, N02536, N02260). Documentation of specific associations of respiratory impairment has been made in OSHA regulations minimizing exposure in the workplace and in the NIOSH agent-specific criteria documents and technical report cited in Table VI-2.

As discussed above in the Extent of Exposure section, which includes data from NOHS, noise is the most prevalent hazard for all production or process areas of the industry. This physical hazard has been a subject of a NIOSH criteria document (N02905); OSHA regulations to minimize the risk to the US worker are in effect.

IV. CONTROL OF EXPOSURE

This section contains information on engineering control of specific hazards (especially noise) in pulp and paper mills and an evaluation of OSHA standards governing pulp and paper mills (29 CFR 1910.261). A detailed consideration of engineering control procedures for pulp and paper mills will be deferred until information from the NIOSH-sponsored control technology assessment of the pulp and paper industry has been received.

Engineering Controls

Protection of employees in pulp and paper operations requires attention to the control of noise, chemical agents, and nip hazards. Other sources of illness and injury, such as heat and materials handling, have no aspects unique or peculiar to pulp and paper operations, and therefore are not covered in this discussion. It should be pointed out, however, that the use of eye protection and respirators in dusty areas and in areas near paper machine equipment may pose special problems in the hot, humid environment encountered by workers. Periodic inspection of equipment--especially cast iron dryer drums of older paper machines, which may crack after long years of service--may prevent serious injury to paper machine workers (NO2738).

(a) Nip Hazards

The large number of nip hazards in papermaking arises from the numerous rolls and cylinders used to carry and/or treat the paper as it is being made. Williams (NO2375) discussed these in detail, from the wire screen section to the calendar presses. He suggested, in addition to standard practices on guarding of power transmission apparatus, that many of the nip points can be adequately guarded or modified to eliminate the nip point on the operator's side. Designs for some types of nip guards were also suggested.

(b) Noise

Noise hazards in pulp and paper operations are significant. Noise levels were measured as early as 1954. More recently, Cook and Giever (NO2692) reported levels ranging from a high of 111 dBC in Jordan refiners to a low of 93 dBC at evaporator operations (see Table VI-18). Jensen (NO2750) reported levels ranging from 88 to 110 dBA. These reports are not directly comparable, since different weighting scales were used in determining the levels.

Attempts at control of noise levels in papermaking were introduced in the 1950's. Davis and Caston (NO3162) in 1955 described pulp and paper mill noise sources in terms of octave band analyses. Noise sources which they identified were chippers, refiners, barkers, slasher saws, vacuum pumps, air jets, and tissue machines.

Over the years, noise controls have been applied to suction rolls on paper machines, Jordan refiners, and gear noise. More recently, Lund (NO3140) reported on control of corrugated paper machine flutes. Enclosure of the flutes and machine, as well as operator isolation, were attempted. Only operator enclosure reduced exposure below the OSHA limit, and only if the operator remained in the booth for a sufficient period of time.

No reports of complete attempts to control noise were located. By complete attempts, we mean providing known effective means of noise reduction in an entire operation, so that the total benefit of current technological capability can be tested. Thus, the extent to which noise in paper mills can be reduced as well as the corresponding costs in lost production are unknown at this time.

During the single plant visit to an integrated pulp and paper mill, noise levels in the wood yard were intermittent and estimated to be as high as 120 dBA (NO3297). Noise levels were so intense in the papermaking area that communication could occur only by shouting in someone's ear. Clearly, these noise levels present a hazard if workers are exposed for significant periods of time.

(c) Chemical Agents

Chemical hazards in pulp and paper operations depend almost entirely on the type of paper being made and the process used to make it. In chemical process pulp operations, sulfur derivatives present an

occupational hazard as well as a community nuisance. The most common way to control these products is to burn exhausted air in the lime recovery kiln. The reduced sulfur compounds, such as H_2S, dimethyl sulfide, and mercaptans, are produced in the chemical recovery furnace as well as in other parts of the process. Lime recovery is the only process in which stoichiometric combustion is desired.

Control of chemical agents in solid form consists principally of exhaust ventilation at tanks where the material is dissolved or slurried prior to use in the process. In larger mills, products which are used in large quantities are frequently handled in bulk. No reports of employee exposure levels have been found in the literature.

Evaluation of OSHA Regulations Governing Pulp and Paper Mills

OSHA has specific regulations governing pulp and paper making operations in 29 CFR 1910.261. In all cases, the specific standard under paragraph 1910.261 applies, as do the general industry standards to the extent that a situation is not covered in 1910.261. The first section, paragraph (a), incorporates the consensus standards of ANSI, the Institute of Makers of Explosives, and the American Society of Mechanical Engineers. These cover such areas as walking and working surfaces, wood ladders, and powered industrial trucks. There are 27 consensus standards in all, of which 17 represent duplication of other provisions of part 1910.

The other paragraphs in section 261 apply to specific operations in pulp and paper manufacturing. For example, paragraph (c) applies to handling and storage of pulpwood and pulp chips. Within this section, specifications for clearances along railroad tracks, personal protective equipment, gang planks, and other items are included. Some of these duplicate other general industry standards. For example, to the extent that eye protection is required where there is a possibility of eye injury, then requiring eye protection in a pulp chipping operation may be considered repetitive.

In other cases, the standard is incomplete. That is, the standard only covers a portion of the logical sequence of events which must be followed in a given situation for safe operations. For example, paragraph (b)(5) requires that air in vessels be tested for oxygen deficiency and for presence of toxic and flammable vapors prior to entry, but is less specific than the general industry standard [1910.94 (d)(11)] about what is permissible activity and what is not.

Several recommendations can be made regarding these standards. First, where ANSI standards cover problems addressed by OSHA standards, the OSHA reference should be included. The ANSI standards can be included as guides or appendices, but should not take precedence over OSHA's own requirements. Second, where there is no OSHA standard, the ANSI requirements, or some logical extraction of them, should be made part of the general industry standards. For example, if the lighting standard is applicable to pulp and paper mills, it is difficult to see why it is not applicable to other manufacturing facilities.

The standards could be simplified somewhat by using tables for items such as personal protective equipment, guarding of nip points, and vessel entry. Each section covering specific operations has paragraphs addressing these points, and these could be eliminated. In addition, cross-references to other OSHA standards should be included.

Generallly, the OSHA standards appear to address the major hazards of pulp and paper manufacturing. Whether they address the major causes of injury and illness is another question. An evaluation of the data contained in OSHA accident statistics and reports will be included in a later report. The evaluation will address the above question by attempting to analyze causes and correlating causes with various sections of the standards. This will be done as soon as the OSHA data are received.

V. REFERENCES

N00596 Gosselin RE, Hodge HC, Smith RP, Gleason MN: Clinical Toxicology of Commercial Products, ed 4. Baltimore, The Williams and Wilkins Co, 1976

N00600 Verschueren K: Handbook of Environmental Data on Organic Chemicals. New York, Van Nostrand Reinhold Co, 1977

N00601 Shreve RN, Brink JA Jr: Chemical Process Industries, ed 4. New York, McGraw-Hill Book Company, 1977, 814 pp

N00733 Fire Protection Guide on Hazardous Materials, ed 6. Boston, National Fire Protection Association, 1977

N00807 Grayson M (ed.): Kirk-Othmer Encylopedia of Chemical Technology, ed 3. New York, John Wiley and Sons Inc, 1979

N00808 Standen A (ed.): Kirk-Othmer Encyclopedia of Chemical Technology, ed 2. New York, John Wiley and Sons Inc, 1967

N00825 Plunkett ER: Handbook of Industrial Toxicology. New York, Chemical Publishing Co, 1976

N00872 Mackison FW, Stricoff RS, Partridge LJ Jr: NIOSH/OSHA Pocket Guide to Chemical Hazards, DHEW Publication No. (NIOSH) 78-210. US Dept of Health, Education, and Welfare, Public Health Service, Center for Disease Control, National Institute for Occupational Safety and Health, 1977

N00874 Lowenheim FA, Moran MK (eds.): Faith, Keyes, and Clark's Industrial Chemicals, ed 4. New York, John Wiley and Sons Inc, 1975, 904 pp

N00891 Patty FA, Fassett DW, Irish DD (eds.): Industrial Hygiene and Toxicology, ed 2 rev. New York, John Wiley and Sons Inc, 1963

N00893 Lewis RJ Sr, Tatken RL (eds.): Registry of Toxic Effects of Chemical Substances. Cincinnati, US Dept of Health, Education, and Welfare, Public Health Service, Center for Disease Control, National Institute for Occupational Safety and Health, 1979

N00896 Windholz M, Budavari S, Stroumtsos LY, Fertig MN (eds.): The Merck Index, ed 9. Rahway, NJ, Merck & Co, 1976

N00911 Casarett LJ, Doull J (eds.): Toxicology--The Basic Science of Poisons. New York, Macmillan Publishing Co Inc, 1975

N00912 Clayton GD, Clayton FE (eds.): Patty's Industrial Hygiene and Toxicology, ed 3. New York, John Wiley and Sons Inc, 1979

N00916 Hamilton A, Hardy HL: Industrial Toxicology, ed 3. Acton, MA, Publishing Sciences Group, 1974

N00917 Hawley GG (ed.): The Condensed Chemical Dictionary, ed 9. New York, Van Nostrand Reinhold Co, 1977

N00919 Sax NI: Dangerous Properties of Industrial Materials, ed 5. New York, Van Nostrand Reinhold Co, 1979

N00957 National Occupational Hazard Survey. Cincinnati, US Dept of Health, Education, and Welfare, Public Health Service, Center for Disease Control, National Institute for Occupational Safety and Health, Division of Surveillance, Hazard Evaluations, and Field Studies, 1974-1980, 3 vols

N01143 The Industrial Environment--Its Evaluation and Control. US Dept of Health Education, and Welfare, Public Health Service, Center for Disease Control, National Institute for Occupational Safety and Health, 1973

N02259 Paper/Paperboard Woodpulp Capacity, Fiber Consumption. New York, American Paper Institute, 1979, 24 pp

N02260 Powell River Health Study. Unpublished report submitted to the National Council of the Paper Industry for Air and Stream Improvement by the Occupational Lung Disease Unit, University of British Columbia Dept of Medicine, Vancouver General Hospital, July 31, 1979, 179 pp

N02375 Wisner A: Evaluation of Vibrations at Various Workplaces in the Iron and Steel Industry, Report No. 11. Commission des Communautes Europeenes Laboratorie de Physiologie du Travail et d'Ergonomie du Conservatoire National des Arts et Metiers, 1969

N02381 Casey JP: Coating with resinous materials, in Pulp and Paper Chemistry and Chemical Technology. New York, Interscience, 1952, vol 2, pp 1271-1318

N02396 Ball WL: Paper, paper pulp industry, in Encyclopedia of Occupational Health and Safety. Geneva, International Labour Office, 1972, pp 997-1000

N02397 Libby CE (ed.): Pulp and Paper Science and Technology. New York, McGraw-Hill, 1962, 2 vols

N02437 Alkaline pulping recovery units, in Britt KW (ed.) : Handbook of Pulp and Paper Technology, ed 2. New York, Van Nostrand Reinhold, 1970, pp 239-46

N02486 Chemicals with Sufficient Evidence of Carcinogenicity in Experimental Animals--IARC Monographs Volumes 1-17, IARC Working Group Report. Lyon, International Agency for Research in Cancer, 1978, 20 pp

N02493 Criteria for a Recommended Standard--Occupational Exposure to Hydrogen Sulfide, DHEW (NIOSH) Publication No. 77-158. Cincinnati, US Dept of Health, Education, and Welfare, Public Health Service, Center for Disease Control, National Institute for Occupational Safety and Health, 1977, 149 pp

N02496 Criteria for a Recommended Standard--Occupational Exposure to Sulfuric Acid, HEW Publication No. (NIOSH) 74-128. Cincinnati, US Dept of Health, Education, and Welfare, Public Health Service, Center for Disease Control National Institute for Occupational Safety and Health, 1974, 90 pp

N02497 Criteria for a Recommended Standard--Occupational Exposure to Formaldehyde, DHEW (NIOSH) Publication No. 77-126. Cincinnati, US Dept of Health, Education, and Welfare, Public Health Service, Center for Disease Control, National Institute for Occupational Safety and Health, 1976, 165 pp

N02499 Occupational Injuries and Illnesses in the United States by Industry, 1976, Bulletin 2019. US Dept of Labor, Bureau of Labor Statistics, 1979, 114 pp

N02500 Occupational Injuries and Illnesses in the United States by Industry, 1978, Summary, Report 586. US Dept of Labor, Bureau of Labor Statistics, 1980, 25 pp

N02501 Criteria for a Recommended Standard--Occupational Exposure to Chlorine, DHEW (NIOSH) Publication No. 76-170. Cincinnati, US Dept of Health, Education, and Welfare, Public Health Service, Center for Disease Control, National Institute for Occupational Safety and Health, 1976, 155 pp

N02502 Criteria for a Recommended Standard--Occupational Exposure to Sodium Hydroxide, HEW Publication No. (NIOSH) 76-105. Cincinnati, US Dept of Health, Education, and Welfare, Public Health Service, Center for Disease Control, National Institute for Occupational Safety and Health, 1975, 93 pp

N02508 Harrington JM, Blot WJ, Hoover RN, Housworth WJ, Heath CW Jr, Fraumeni JF Jr: Lung cancer in coastal Georgia--A death certificate analysis of occupation--Brief communication. J Nat Cancer Inst 60:295-98, 1978

N02521 Joyce TW, Dugal HS: How atmospheric sulfate standards relate to the pulp and paper industry. Pap Trade J 162:50-51, 1978

N02532 Taylor ML, Gardner HS: Causes of recovery boiler explosions, in TAPPI Eng Conference, Seattle, Oct 21-24, 1974, pp 161-64

N02535 Schlueter DP, Fink JN, Hensley GT: Wood pulp workers' disease--A hypersensitivity pneumonitis caused by alternaria. Ann Intern Med 77:907-14, 1972

NO2536 Ferris BG Jr, Burgess WA, Worcester J: Prevalence of chronic respiratory disease in a pulp mill and a paper mill in the United States. Brit J Ind Med 24:26-37, 1967

NO2546 Technological Feasibility Assessment and Inflationary Impact Statement of the Proposed Standard for Sulfur Dioxide, Report No. OSHA-IIS-77-0218. US Dept of Labor, Occupational Safety and Health Administration, 1977, 319 pp

NO2547 Chlorine and Hydrogen Chloride, Publication No. EPA-600/1-76-020. Research Triangle Park, NC, US Environmental Protection Agency, Office of Research and Development, Health Effects Research Laboratory, 1976, 396 pp

NO2554 Deane M, Sanders G: Health effects of exposure to community odors from pulp mills, Eureka, CA, 1971. Environ Res 14:164-81, 1977

NO2555 Lehrer SB, Turer E, Weill H, Salvaggio JE: Elimination of bagassosis in Louisiana paper manufacturing plant workers. Clin Allergy 8:15-20, 1978

NO2561 Mason T: Statistics point to high-cancer localities. Int J Occup Health Saf 46:44-48, 1977

NO2571 Kraft Pulping, Control of TRS Emissions from Existing Mills, Publication No. EPA-450/2-78-003b. US Environmental Protection Agency, Office of Air Quality Planning and Standards, 1979, 200 pp

NO2607 Mozell MM, White DC: Behavioral effects of whole body vibration. Aviation Medicine 29:716-24, 1958

NO2608 Sinitsyn SN, Rumyantsev GI, Voranova KV: Some changes in carbohydrate metabolism under the influence of a whole-body vibration. Hyg Sanit 29(5):58-62, 1964

NO2668 MacDonald RG, Franklin JN (eds.): Pulp and Paper Manufacture, ed 2. New York, McGraw-Hill, 1969, 3 vols

NO2678 Hendrickson ER, Walker CG, Chapnerkar VD: Identification of non-sulfur organic compounds in stack gases from pulp mills. Amer Ind Hyg Assoc J 24:121-26, 1963

NO2683 Feiner B, Marlow S: Industrial hygiene in pulp mills. N Y State Dep Labor Div Ind Hyg Mon Rev 35(7):25-28, 1956

NO2687 Fires and explosions. Accidents (74):12-27, 1968

NO2688 Explosions--the boom is getting louder. Factory Mutual Record 47(6):15-16, 1970

NO2690 McCollister DD, Oyen F, Rowe VK: Toxicology of acrylamide. Toxicol Appl Pharmacol 6:172-81, 1964

N02692 Cook WA, Giever PM: Noise exposures in pulp and paper production. Amer Ind Hyg Assoc J 30:484-86, 1969

N02693 Jones KH, Thomas JF, Brink DL: Control of malodors from Kraft recovery operations by pyrolysis. J Air Pollut Control Assoc 19:501-4, 1969

N02696 Walther JE, Amberg HR: Odor control in the Kraft pulp industry. Chem Eng Prog 66:73-80, 1970

N02697 DMSO, dimethylsulfoxide. J Occup Med (Lett) 13(1):44, 1971

N02698 Skalpe IO: Long-term effects of sulfur dioxide exposure in pulp mills. Br J Ind Med 21:69-73, 1964

N02700 Shahied SI, Stanovick RP, McInturff DE, Missaghi E: Determination of polychlorinated biphenyl (PCB's) residues in grades of pulp, paper and paperboard. Bull Environ Contam Toxicol 10:80-87, 1973

N02703 Gloemme J, Lundgren KD: Health hazards from chlorine dioxide. AMA Arch Ind Health 16:169-76, 1957

N02704 Buechner HA: Bagassosis. J Am Med Assoc 174:1237-40, 1960

N02706 Glass WI: An outbreak of formaldehyde dermatitis. N Z Med J 60(349):423-27, 1961

N02707 Dalhamn T: Chlorine dioxide. AMA Arch Indust Hyg 15:101-7, 1957

N02715 Dahl CB: Reduction of noise from paper machines suction rolls and rewinders. AMA Arch and Health 17:319-26, 1958

N02716 Trout PE: PCB and the paper industry--A progress report. Environ Health Perspect 1:63-65, 1972

N02721 Blot WJ, Fraumeni JF Jr: Geographic patterns of lung cancer--Industrial correlations. Am J Epidemiol 103:539-50, 1976

N02723 Ruhe RL: Health Hazard Evaluation Determination, Report No. 77-9-391, Corrugated Paper Company, Westboro, Massachusetts. Cincinnati, Dept of Health, Education, and Welfare, Public Health Service, Center for Disease Control, National Institute for Occupational Safety and Health, Hazard Evaluation Services Branch, 1977, 8 pp

N02724 Milham S Jr: Methods in occupational mortality studies. J Occup Med 17:581-85, 1975

N02725 Oregon Industry Fatalities, 1966-1973. Salem, OR, Accident Prevention Division, State of Oregon Workmen's Compensation Board, 1974, 32 pp

N02730 McCord CP: Industrial poisoning from low concentrations of chlorine gas. J Am Med Assoc 86:1687-88, 1926

N02733 Ferris BG Jr, Puleo S, Chen HY: Mortality and morbidity in a pulp and a paper mill in the United States--A ten-year follow-up. Br J Ind Med 36:127-34, 1979

N02738 Riley RG: Paper machine injuries: how they happen and how to prevent them. Pulp Paper 53:117-19, 1979

N02750 Jensen P: Noise control: its inherent problems and the approach to take in resolving them. Pap Trade J 162:30-33, 1978

N02762 Boyle GM: Dust explosion hazards in the paper industry. Paper Tech 2:35-40, 1970

N02764 Occupational Injuries and Illnesses in the United States by Industry, 1974, Bulletin 1932. US Dept of Labor, Bureau of Labor Statistics, 1976, 143 pp

N02766 Occupational Injuries and Illnesses in the United States by Industry, 1973, Bulletin 1874. US Dept of Labor, Bureau of Labor Statistics, 1975, 143 pp

N02770 McCord CP: The toxic properties of some timber woods. Ind Med Surg 27:202-04, 1958

N02772 Ishizaki T, Shida T, Miyamoto t, Matsumara Y, Mizuno K, Tomaru M: Occupational asthma from western red cedar dust (Thuja plicata) in furniture factory workers. J Occup Med 15: 580-85, 1973

N02774 Eaton KK: Respiratory allergy to exotic wood dust. Clin Allergy 3:307-10, 1973

N02775 Booth BH, LeFoldt RH, Moffitt EM: Wood dust hypersensitivity. J Allergy Clin Immunol 57:352, 1976

N02776 Acheson ED, Cowdell RH, Hadfield E, Macbeth RG: Nasal cancer in wood workers in the furniture industry. Br Med J 2:587-96, 1968

N02778 Criteria for a Recommended Standard--Occupational Exposure to Hot Environments, HSM 72-10269. US Dept of Health, Education, and Welfare, Health Services and Mental Health Administration, National Institute for Occupational Safety and Health, 1972, 110 pp

N02875 Criteria for a Recommended Standard--Working in Confined Spaces, DHEW (NIOSH) Publication, No. 80-106. US Dept of Health, Education, and Welfare, Public Health Service, Center for Disease Control, National Institute for Occupational Safety and Health, 1979, 68 pp

N02896 Hutchins FE: Toxicity of Pulp and Paper Mill Effluent--A Literature Review, Publication No. EPA-600/3-79-013. Corvallis, OR, US Environmental Protection Agency, Environmental Research Laboratory, 1979, 43pp

NO2897 Arena JM: Poisoning--Toxicology, Symptoms, Treatments, ed 4. Springfield, IL, Charles C. Thomas, 1978

NO2902 Criteria for a Recommended Standard--Occupational Exposure to a Cotton Dust, HEW Publication No. (NIOSH) 75-118. US Dept of Health, Education, and Welfare, Public Health Service, Center for Disease Control, National Institute for Occupational Safety and Health, 1974, 159 pp

NO2903 Revised Recommended Asbestos Standard, DHEW (NIOSH) Publication No. 77-169. US Dept of Health, Education, and Welfare, Public Health Service, Center for Disease Control, National Institute for Occupational Safety and Health, 1976, 96 pp

NO2904 Criteria for a Recommended Standard--Occupational Exposure to Carbon Black, DHEW (NIOSH) Publication No. 78-204. US Dept of Health, Education, and Welfare, Public Health Service, Center for Disease Control, National Institute for Occupational Safety and Health, 1978, 99 pp

NO2905 Criteria for a Recommended Standard--Occupational Exposure to Noise, HSM 73-11001. US Dept of Health, Education, and Welfare, Health Services and Mental Health Administration, National Institute for Occupational Safety and Health, 1972, 150 pp

NO2906 Dement JM, Zumwalde RD, Gamble JF, Fellner W, DeMeo MJ, Brown DP, Wagoner JK: Occupational Exposure to Talc Containing Asbestos, Morbidity, Mortality, and Environmental Studies of Miners and Millers, DHEW (NIOSH) Publication No. 80-115. US Dept of Health, Education, and Welfare, Public Health Service, Center for Disease Control, National Institute for Occupational Safety and Health, 1980, 106 pp

NO2907 Criteria for a Recommended Standard--Occupational Exposure to n-Alkane mono thiols, Cyclohexanethiol, Benzenethiol, DHEW (NIOSH) Publication No. 78-213. Dept of Health, Education, and Welfare, Public Health Service, Center for Disease Control, National Institute for Occupational Safety and Health, 1978, 129 pp

NO2908 Criteria for a Recommended Standard--Occupational Exposure to Crystalline Silica, HEW Publication No. (NIOSH) 75-120. US Dept of Health, Education, and Welfare, Public Health Service, Center for Disease Control, National Institute for Occupational Safety and Health, 1975, 121 pp

NO2917 Criteria for a Recommended Standard--Occupational Exposure to Zinc Oxide, DHEW Publication No. (NIOSH) 76-104. Cincinnati, US Dept of Health, Education, and Welfare, Public Health Service, Center for Disease Control, National Institute for Occupational Safety and Health, 1976, 87 pp

NO2926 Criteria for a Recommended Standard--Occupational Exposure to Polychlorinated Biphenyls (PCBs), DHEW (NIOSH) Publication No. 77-225. Cincinnati, US Dept of Health, Education, and Welfare, Public Health Service, Center for Disease Control, National Institute for Occupational Safety and Health, 1977, 224 pp

NO2927 Criteria for a Recommended Standard--Occupational Exposure to Sulfur Dioxide, Publication No. TR-107-74. US Dept of Health, Education, and Welfare, Public Health Service, Center for Disease Control, National Institute for Occupational Safety and Health, 1974, 112 pp

NO2929 Criteria for a Recommended Standard--Occupational Exposure to Acrylamide, DHEW (NIOSH) Publication No. 77-112. Cincinnati, US Dept of Health, Education, and Welfare, Public Health Service, Center for Disease Control, National Institute for Occupational Safety and Health, 1976, 127 pp

NO2930 Wayne LG, Bryan RJ, Ziedman K: Irritant Effects of Industrial Chemicals--Formaldehyde, DHEW (NIOSH) No. 77-117. Cincinnati, US Dept of Health, Education, and Welfare, Public Health Service, Center for Disease Control, National Institute for Occupational Safety and Health, 1976, 144 pp

NO2934 Calabrese EJ, Moore GS, Tuthill RW: The health effects of chlorine dioxide as a disinfectant in potable water--A literature survey. J Environ Health 41:26-31, 1978

NO2935 Butterworth KR, Carpanini F MB, Gaunt IF, Hardy J, Kiss IS, Gangolli SD: Short-term toxicity of dimethyl sulphide in the rat. Food Cosmet Toxicol 13:15-22, 1975

NO2936 Kocsis JJ, Harkaway S, Snyder R: Biological effects of the metabolites of dimethyl sulfoxide. Ann N Y Acad Sci 243:104-09, 1975

NO2945 Zitting A, Skytta E: Biological activity of titanium dioxides. Int Arch Occup Environ Health 43:93-97, 1979

NO2946 Wilska S: Titanium dioxide pigment--A potential health hazard? FATIPEC Congr 13:79-84, 1976

NO2949 Savolainen H, Pfaffli P: Effects of long-term turpentine inhalation on rat brain protein metabolism. Chem Biol Interact 21:271-76, 1978

NO2951 Baer RL, Ramsey DL, Biondi E: The most common contact allergens. Arch Dermatol 108:74-78, 1973

NO2953 Cunningham HM: Effect of sodium hypochlorite on the growth of rats and guinea pigs. Am J Vet Res 41:295-97, 1980

NO2955 Adams AB Jr: Air and water pollution control in crude tall oil manufacture in the pulp and paper industry. J Am Oil Chem Soc 50:498-500, 1973

N02958 Boonpucknavig V, Bhamarapravati N, Kamtorn P, Sukumalchandra Y: Bagassosis--A histopathologic study of pulmonary biopsies from six cases. Am J Clin Pathol 59:461-72, 1973

N03038 Loeb M, Jones PD, Cohen A: Effect of Noise on Non-Auditory Sensory Functions and Performance. Cincinnati, US Dept of Health, Education, and Welfare, Public Health Service, National Institute for Occupational Safety and Health, Division of Biomedical and Behavioral Sciences, 1976, 122 pp

N03039 Bioassay of Titanium Dioxide for Possible Carcinogenicity CAS No. 13463-67-7, Report No. NCI-CG-TR-97. Bethesda, MD, US Dept of Health, Education, and Welfare, Public Health Service, National Cancer Institute, 1978, 114 pp

N03042 Roald SO: Acute toxicity of lignosulfonates on rainbow trout (Salmo Gairdneri). Bull Environ Contam Toxicol 17:702-06, 1977

N03044 Griffin JM, West JL: Acute toxicity of ammonia-base neutral sulfite pulp mill waste liquor to rainbow trout. Bull Environ Contam Toxicol 15:608-12, 1976

N03052 Alarie YC, Krumm AA, Busey WM, Ulrich CE, Kantz RJ II: Long-term exposure to sulfur dioxide, sulfuric acid mist, fly ash, and their mixtures--Results of studies in monkeys and guinea pigs. Arch Environ Health 30:254-62, 1975

N03057 Cooper DW, Underhill DW, Ellenbecker MJ: A critique of the US standard for industrial exposure to sodium hydroxide aerosols. Am Ind Hyg Assoc J 40:365-71, 1979

N03058 Fenters JD, Bradof JN, Aranyi C, Ketels K, Ehrlich R, Gardner DE: Health effects of long-term inhalation of sulfuric acid mist-carbon particle mixtures. Environ Res 19:244-57, 1979

N03059 Cavender FL, Singh B, Cockrell BY: Effects in rats and guinea pigs of six month exposures to sulfuric acid mist, ozone and their combination. J Toxicol Environ Health 4:845-52, 1978

N03062 Last JA, Cross CE: A new model for health effects of air pollutants--Evidence for synergistic effects of mixtures of ozone and sulfuric acid aerosols on rat lungs. J Lab Clin Med 91:328-39, 1978

N03065 Schiff LJ, Bryne MM, Fenters JD, Graham JA, Gardner DE: Cytotoxic effects of sulfuric acid mist, carbon particulates, and their mixtures on hamster tracheal epithelium. Environ Res 19:339-54, 1979

N03068 Tomatis L, Agthe C, Bartsch H, Huff J, Montesano R, Saracci R, Walker E, Wilbourn J: Evaluation of the carcinogenicity of chemicals: A review of the monograph program of the International Agency for Research on Cancer (1971 to 1977). Cancer Res 38:877-85, 1978

N03072 Dodd DE, Bus JS, Barrow CS: Lung sulfhydryl changes in rats following chlorine inhalation. Toxicol Appl Pharmacol 52:199-208, 1980

N03075 Barrow CS, Kociba RJ, Rampy LW, Keyes DG, Albee RR: An inhalation toxicity study of chlorine in Fischer 344 rats following 30 days of exposure. Toxicol Appl Pharmacol 49:77-88, 1979

N03083 Lakowicz JR, Bevan DR: Effects of asbestos, iron oxide, silica, and carbon black on the microsomal availability of benzo(a)alpyrene. Biochemistry 18:5170-76, 1979

N03085 Bell ZG Jr, Dunnom DD, Lott H: Basis for exposure standards for amorphous silica dusts. Am Ind Hyg Assoc J 39:418-21, 1978

N03092 Hallmans G, Liden S: Penetration of zinc-65 through the skin of rats. Acta Derm Venereol 59:105-12, 1979

N03096 Susman JL, Hornig JF, Thomae SC, Smith RP: Pulmonary excretion of hydrogen sulfide, methanethiol, dimethyl sulfide and dimethyl disulfide in mice. Drug Chem Toxicol 1:327-38, 1979

N03097 Vahlkamp T, Meijer AJ, Wilms J, Chamuleau RAFM: Inhibition of mitochondrial electron transfer in rats by ethanethiol and methanethiol. Clin Sci 56:147-56, 1979

N03098 Waller RL: Mathanethiol inhibition of mitochondrial respiration. Toxicol Appl Pharmacol 42:111-17, 1977

N03100 Loomis TA: Formaldehyde toxicity. Arch Pathol Lab Med 103:321-24,1979

N03102 Sasaki Y, Endo R: Mutagenicity of aldehydes in Salmonella typhimurium. Mutat Res 54:251-52, 1978 (Abst)

N03104 Zeiger E, Chhabra RS, Margolin BH: Effects of the hepatic S9 fraction from Aroclor 1254-treated rats on the mutagenicity of benzo(a)pyrene and 2-aminoanthracene in the Salmonella microsome assay. Mutat Res 64:379-89, 1979

N03105 Shahin MM, Andrillon P, Goetz N, Bore P, Bugaut A, Kalopissis G: Studies on the mutagenicity of p-phenylenediamine in Salmonella typhimurium. Presence of PCB's in rat-liver microsomal fraction induced by Aroclor. Mutat Res 68:327-36, 1979

N03106 Lee TP, Park BH: Biochemical basis of Aroclor 1254 and pesticide toxicity in vitro. 1. Effects on intracellular ATP concentration. Res Commun Chem Pathol Pharmacol 25:597-605, 1979

N03110 Berry DL, DiGiovanni J, Juchau MR, Bracken WM, Gleason GL, Slaga TJ: Lack of tumor-promoting ability of certain environmental chemicals in a two-stage mouse skin tumorigenesis assay. Res Commun Chem Pathol Pharmacol 20:101-08, 1978

N03111 Kerkvliet NI, Kimeldorf DJ: Inhibition of tumor growth in rats by feeding a polychlorinated biphenyl, Aroclor 1254. Bull Environ Contam Toxicol 18:243-46, 1977

N03116 Lakowicz JR, Bevan DR: Benzo(a)pyrene uptake into rat liver microsomes--Effects of adsorption of benzo(a)pyrene to asbestos and non-fibrous mineral particulates. Chem Biol Interact 29:129-38, 1980

N03126 Test Sample and Accident Data for SICs 2611, 2621, 2631, 2661 with All Hazardous Substances 7/72 - 4/80 from Management Information System. US Dept of Labor, Occupational Safety and Health Administration, 1980, 59 pp

N03127 Savolainen H, Tenhunen R, Elovaara E, Tossavainen A: Cumulative biochemical effects of repeated subclinical hydrogen sulfide intoxication in mouse brain. Int Arch Occup Environ Health 46:87-97, 1980

N03133 Trout PE: View of the paper industry on the occurrence of PCB's in the environment and the need for regulation, in Proceedings of the National Conference on PCB's, EPA-560/6-75-004. Chicago, Nov 1975, pp 359-61 (Abst)

N03134 Milham S, Hesser JE: Hodgkin's disease in woodworkers. Lancet 2:136-37, 1967

N03135 Sperling F: In vivo and in vitro toxicology of turpentine. Clin Toxicol 2:21-35, 1969

N03140 Lund AO: Noise controls for paper corrugators. Am Ind Hyg Assoc J 39:824-27, 1978

N03141 Haney S: Statement relating to polychlorinated biphenyls on behalf of the Wisconsin Paper Council, in Proceedings of the National Conference on PCB's, EPA-560/6-75-004. Chicago, Nov 1975, pp 362-64 (Abst)

N03142 Criteria for a Recommended Standard--Occupational Exposure to Carbon Monoxide, HSM 73-11000. US Dept of Health, Education, and Welfare, Health Services and Mental Health Administration, National Institute for Occupational Safety and Health, 1972.

N03143 Turpentine, in Key MM, Henschel AF, Butler J, Ligo RN, Tabershaw IR, Ede L (eds.): Occupational Diseases--A Guide to Their Recognition, DHEW (NIOSH) Publication No. 77-181, rev ed. US Dept of Health, Education, and Welfare, Public Health Service, Center for Disease Control, National Institute for Occupational Safety and Health, 1977, pp 148-50

N03145 Mercaptans, in Key MM, Henschel AF, Butler J, Ligo RN, Tabershaw IR, Ede L (eds.): Occupational Diseases--A Guide to Their Recognition, DHEW (NIOSH) Publication No. 77-181, rev ed. US Dept of Health, Education, and Welfare, Public Health Service, Center for Disease Control, National Institute for Occupational Safety and Health, 1977, pp 310-12

N03146 Chlorine, in Key MM, Henschel AF, Butler J, Ligo RN, Tabershaw IR, Ede L (eds.): Occupational Diseases--A Guide to Their Recognition, DHEW (NIOSH) Publication No. 77-181, rev ed. US Dept of Health, Education, and Welfare, Public Health Service, Center for Disease Control, National Institute for Occupational Safety and Health, 1977, pp 317-18

N03148 Calcium oxide, in Key MM, Henschel AF, Butler J, Ligo RN, Tabershaw IR, Ede L (eds.): Occupational Diseases--A Guide to Their Recognition, DHEW (NIOSH) Publication No. 77-181, rev ed. US Dept of Health, Education, and Welfare, Public Health Service, Center for Disease Control, National Institute for Occupational Safety and Health, 1977, pp 414-15

N03149 Carbon monoxide, in Key MM, Henschel AF, Butler J, Ligo RN, Tabershaw IR, Ede L (eds.): Occupational Diseases--A Guide to Their Recognition, DHEW (NIOSH) Publication No. 77-181, rev ed. US Dept of Health, Education, and Welfare, Public Health Service, Center for Disease Control, National Institute for Occupational Safety and Health, 1977, pp 417-19

N03150 Hydrogen sulfide, in Key MM, Henschel AF, Butler J, Ligo RN, Tabershaw IR, Ede L (eds.): Occupational Diseases--A Guide to Their Recognition, DHEW (NIOSH) Publication No. 77-181, rev ed. US Dept of Health, Education, and Welfare, Public Health Service, Center for Disease Control, National Institute for Occupational Safety and Health, 1977, pp 422-24

N03153 Sax NI: Dangerous Properties of Industrial Materials, ed 4. New York, Van Nostrand Reinhold, 1975

N03154 Stanton MF: Some aetiological considerations of fibre carcinogenesis, in Bogovski P, Gilson JC, Timbrell V, Wagner JC (eds.): Proceedings of the Conference on Biological Effects of Asbestos. Lyon, World Health Organization, 1973

N03155 Pylev LN, Shabad LM: Some results of experimental studies in asbestos carcinogenesis, in Bogovski P, Gilson JC, Timbrell V, Wagner JC (eds.): Proceedings of the Conference on Biological Effects of Asbestos. Lyon, World Health Organization, 1973

N03156 Wagner JC, Berry G, Cooke TJ, Hill RJ, Pooley FD, Skidmore JW: Animal experiments with talc, in Fourth International Symposium on Inhaled Particles and Vapors, Edinborough, Sept 1975

N03162 Davis MN, Caston RH: Noise problems in pulp and paper mills. TAPPI 38:359-64, 1955

N03167 Sinock A, Seabright M: Induction of chromosome changes in Chinese hamster cells by exposure to asbestos fibres. Nature 257:56-58, 1975

N03168 Smith WE, Miller L, Elsasser RE, Hubert DD: Tests for carcinogenicity of asbestos. Ann NY Acad Sci 132:456-88, 1965

N03169 Donna A: [Experimental tumors from asbestos (chrysotile, crocidolite, and amosite) in the Sprague-Dawley rat]. Med Lav 61:1-32, 1970 (Ita) (Abst)

N03170 Stanton MF, Wrench C: Mechanisms of mesothelioma induction with asbestos and fibrous glass. J Natl Cancer Inst 48:797-821, 1972

N03171 Shabad LM, Pylev LN, Krivosheeva LV, Kulagina TF, Nemenko BA: Experimental studies on asbestos carcinogenicity. J Natl Cancer Inst 52:1175-87, 1974

N03172 Wagner JC, Berry G, Skidmore JW, Timbrell V: The effect of the inhalation of asbestos in rats. Br J Cancer 29:252-69, 1974

N03173 Reeves AL, Puro HE, Smith RG, Vorwald AJ: Experimental asbestos carcinogenesis. Environ Res 4:496-511, 1971

N03174 Gross P, deTreville RTP, Tolker EB, Kaschak M, Babyak MA: Experimental asbestosis--The development of lung cancer in rats with pulmonary deposits of chrysotile asbestos dust. Arch Envir Health 15:343-55, 1967

N03175 Reeves AL, Puro HE, Smith RG: Inhalation carcinogenesis from various forms of asbestos. Environ Res 8:178-202, 1974

N03176 Lynch KM, McIver FA, Cain JR: Pulmonary tumors in mice exposed to asbestos dust. Arch Ind Health 15:207-14, 1957

N03177 Gofmekler VA: Effect of embryonic development of benzene and formaldehyde in inhalation experiments. Hyg Sanit 33:327-31, 1968

N03178 Tou JC, Kallos GJ: Possible formation of bis(chloromethyl) ether from the reactions of formaldehyde and chloride ion. Anal Chem 48:958-63, 1976

N03179 Horton AW, Tye R, Stemmer KL: Experimental carcinogenesis of the lung--Inhalation of gaseous formaldehyde or an aerosol of coal tar by C3H mice . J Natl Cancer Inst 30:31-40, 1963

N03180 Linder RE, Gaines TB, Kimbrough RD: The effect of polychlorinated biphenyls on rat reproduction. Food Cosmet Toxicol 12:63-77, 1974

N03181 Green S, Sauro FM, Friedman L: Lack of dominant lethality in rats treated with polychlorinated biphenyls (Aroclors 1242 and 1254). Food Cosmet Toxicol 13:14-22, 1975

N03182 Dikshith TSS, Rockwood W, Abraham R, Coulston F: Effects of polychlorinated biphenyl (Aroclor 1254) on rat testis. Exp Mol Pathol 22:376-85, 1975

N03183 Popper H, Czygan P, Greim H, Schaffner F, Garro AJ: Mutagenicity of primary and secondary carcinogens altered by normal and induced hepatic microsomes (37103). Proc Soc Exp Biol Med 142:727-29, 1973

N03184 Kimbrough RD, Squire RA, Linder RE, Strandberg JD, Montaili RJ, Burse VW: Induction of liver tumors in Sherman strain female rats by polychlorinated biphenyl Aroclor 1260. J Natl Cancer Inst 55:1453-59, 1975

N03185 Gignoux M, Martin H, Cajgfinger H: Ethmoidomaxillary cancer in cabinet makers--Occupational etiology. Ann Oto Laryngol 85:831-33, 1968

N03186 Rossoff I: Handbook of Veterinary Drugs--A Compendium for Research & Clinical Use. New York Springer Publishing Co., 1974, p 627

N03187 Maltoni C, Annoscia C: Mesotheliomas in rats following the intraperitoneal injection of crocidolite, in Maltoni C, Davis W (eds): Characterization of Human Tumors--Proceedings of the Fifth International Symposium on the Biological Characterization of Human Tumors, Bologna, 4-6 April 1973

N03188 Shapiro A, Shoenfeld Y, Shapiro Y: The effect of exposure to heat on intraocular pressure. Albrecht Von Graefes Arch Klin Exp Ophthalmol 210:183-85, 1979

N03192 Cooper RA: Heat and neuroleptics a deadly combination. Am J Psychiatry (Lett) 136(4A):466-67, 1979

N03193 Cohen I: Stress and wound healing. Acta Anat (Basel) 103:134-41, 1979

N03196 Knecht EA, Wright GL, Toraason MA: Periodic short-term heat exposure and reproductive function in male and female rats. Can J Physiol Pharmacol 56:747-53, 1978

N03197 Pout DD: Heat induced deformity. Vet Rec (Lett) 102:427, 1978

N03198 Rapola J, Saxen L, Granroth G: Anencephaly and the sauna. Lancet (Lett) 1(8074):1162, 1978

N03199 Miller P, Smith DW, Shepard TH: Maternal hyperthermia as a possible cause of anencephaly. Lancet 1(8063):519-20, 1978

N03200 Strauss RH, McFadden ER Jr, Ingram RH Jr, Deal EC Jr, Jaeger JJ: Influence of heat and humidity on the airway obstruction induced by exercise in asthma. J Clin Invest 61:433-40, 1978

N03201 Malchaire JB, Mullier M: Occupational exposure to noise and hypertension a retrospective study. Ann Occup Hyg 22:63-66, 1979

NO3203 Poulton EC: Psychology of the scientist XLI continuous noise can degrade performance when using badly designed equipment a case history. Percept Mot Skills 50:319-30, 1980

NO3205 Rontal E, Rontal M, Jacob HJ, Rolnick MI: Vocal cord dysfunction an industrial health hazard. Ann Otol Rhinol Laryngol 88:818-21, 1979

NO3207 Ickes WK, Espili J: Patten a personality and noise induced vasoconstriction. J Speech Hear Res 22(2):334-342, 1979

NO3209 Hermann ER, Hesse CS, Hoyle ER, Leopold AC, Standard JJ: Influence of sonic noise on human stereoscopic depth perception. Am Ind Hyg Assoc J 40:427-35, 1979

NO3217 Scientific Literature Reviews on Generally Recognized as Safe (GRAS) Food Ingredients-Tall Oil, Food and Drug Administration, Springfield, VA, National Technical Information Services, 1973, 82 pp (NTIS PB 228 556)

NO3244 Zitting A: The effects of the fumes from heated rosin (colophony) on tissue glutathione and xenobiotic metabolism in rat lung and liver. Res Commun Chem Pathol Pharmacol 27:563-69, 1980

NO3247 Cartwright LB, Thompson RN: The effects of broadband noise on the cardiovascular system in normal resting adults. Am Ind Hyg Assoc J 36:653-58, 1975

NO3262 Howland RD, Vyas IL, Lowndes HE: The etiology of acrylamide neuropathy--possible involvement of neuron specific enolase. Brain Res 190:529-535, 1980

NO3265 Criteria for a Recommended Standard--Occupational Exposure to Hydrazines, DHEW (NIOSH) Publication No. 78-172. Cincinnati, US Dept of Health, Education, and Welfare, Public Health Service, Center for Disease Control, National Institute for Occupational Safety and Health, 1978, 269 pp

NO3270 Free Chlorine in Air--Physical and Chemical Analysis Branch Method No. 209, in NIOSH Manual of Analytical Methods, ed 2, DHEW (NIOSH) Publication No. 77-157-A. Cincinnati, US Dept of Health, Education, and Welfare, Public Health Service, Center for Disease Control, National Institute for Occupational Safety and Health, 1977, vol 1, pp 209-1 to 209-3

NO3271 Hydrazine in Air--Physical and Chemical Analysis Branch Method No. 248, in NIOSH Manual of Analytical Methods, ed 2, DHEW (NIOSH) Publication No. 77-157-A. Cincinnati, US Dept of Health, Education, and Welfare, Public Health Service, Center for Disease Control, National Institute for Occupational Safety and Health, 1977, vol 1, pp 248-1 to 248-6

N03272 Polynuclear Aromatic Compounds in Air--Physical and Chemical Analysis Branch Method No. 183, in NIOSH Manual of Analytical Methods, ed 2, DHEW (NIOSH) Publication No. 77-157-A. Cincinnati, US Dept of Health, Education, and Welfare, Public Health Service, Center for Disease Control, National Institute for Occupational Safety and Health, 1977, vol 1, pp 183-1 to 183-16

N03273 Benzo(A)Pyrene in Air--Physical and Chemical Analysis Branch Method No. 186, in NIOSH Manual of Analytical Methods, ed 2, DHEW (NIOSH) Publication No. 77-157-A. Cincinnati, US Dept of Health, Education, and Welfare, Public Health Service, Center for Disease Control, National Institute for Occupational Safety and Health, 1977, vol 1, pp 186-1 to 186-15

N03274 Butadiene--Standards Completion Program Validated Method No. S-91, in NIOSH Manual of Analytical Methods, ed 2, DHEW (NIOSH) Publication No. 77-157-B. Cincinnati, US Dept of Health, Education, and Welfare, Public Health Service, Center for Disease Control, National Institute for Occupational Safety and Health, 1977, vol 2, pp S91-1 to S91-9

N03275 Hydrogen Sulfide--Standards Completion Program Validated Method No. S-4, in NIOSH Manual of Analytical Methods, ed 2, DHEW (NIOSH) Publication No. 77-157-B. Cincinnati, US Dept of Health, Education, and Welfare, Public Health Service, Center for Disease Control, National Institute for Occupational Safety and Health, 1977, vol 2, pp S4-1 to S5-1

N03276 Turpentine--Standards Completion Program Validated Method No. S-88, in NIOSH Manual of Analytical Methods, ed 2, DHEW (NIOSH) Publication No. 77-157-B. Cincinnati, US Dept of Health, Education, and Welfare, Public Health Service, Center for Disease Control, National Institute for Occupational Safety and Health, 1977, vol 2, pp S88-1 to S88-9

N03277 Polychlorinated Biphenyls (PCB) in Air--Physical and Chemical Analysis Branch Method No. 253, in NIOSH Manual of Analytical Methods, ed 2, DHEW (NIOSH) Publication No. 77-157-A. Cincinnati, US Dept of Health, Education, and Welfare, Public Health Service, Center for Disease Control, National Institute for Occupational Safety and Health, 1977, vol 1, pp 253-1 to 253-7

N03278 Zinc Oxide in Air--Physical and Chemical Analysis Branch Method No. 222, in NIOSH Manual of Analytical Methods, ed 2, DHEW (NIOSH) Publication No. 77-157-A. Cincinnati, US Dept of Health, Education, and Welfare, Public Health Service, Center for Disease Control, National Institute for Occupational Safety and Health, 1977, vol 1, pp 222-1 to 253-7

N03279 Calcium Oxide--Standards Completion Program Validated Method No. S-205, in NIOSH Manual of Analytical Methods, ed 2, DHEW (NIOSH) Publication No. 77-157-C. Cincinnati, US Dept of Health, Education, and Welfare, Public Health Service, Centers for Disease Control, National Institute for Occupational Safety and Health, 1977, vol 3, pp S205-1 to S205-6

N03280 Carbon Black--Standards Completion Program Validated Method No. S-262, in NIOSH Manual of Analytical Methods, ed 2, DHEW (NIOSH) Publication No. 77-157-C. Cincinnati, US Dept of Health, Education, and Welfare, Public Health Service, Center for Disease Control, National Institute for Occupational Safety and Health, 1977, vol 3, pp S262-1 to S262-5

N03281 Hydrazine--Standards Completion Program Validated Method No. S-237, in NIOSH Manual of Analytical Methods, ed 2, DHEW (NIOSH) Publication No. 77-157-C. Cincinnati, US Dept of Health, Education, and Welfare, Public Health Service, Center for Disease Control, National Institute for Occupational Safety and Health, 1977, vol 3, pp S237-1 to S237-6

N03282 Silica, Crystalline--Standards Completion Program Validated Method No. S-315, in NIOSH Manual of Analytical Methods, ed 2, DHEW (NIOSH) Publication No. 77-157-C. Cincinnati, US Dept of Health, Education, and Welfare, Public Health Service, Center for Disease Control, National Institute for Occupational Safety and Health, 1977, vol 3, pp S315-1 to S315-13

N03283 Sulfuric Acid--Standards Completion Program Validated Method No. S-174, in NIOSH Manual of Analytical Methods, ed 2, DHEW (NIOSH) Publication No. 77-157-C. Cincinnati, US Dept of Health, Education, and Welfare, Public Health Service, Center for Disease Control, National Institute for Occupational Safety and Health, 1977, vol 3, pp S174-1 to S174-8

N03284 Magnesium Oxide Fume--Standards Completion Program Validated Method No. S-369, in NIOSH Manual of Analytical Methods, ed 2, DHEW (NIOSH) Publication No. 77-157-C. Cincinnati, US Dept of Health, Education, and Welfare, Public Health Service, Center for Disease Control, National Institute for Occupational Safety and Health, 1977, vol 3, pp S369-1 to S369-7

N03286 Sodium Hydroxide--Standards Completion Program Validated Method No. S-381, in NIOSH Manual of Analytical Methods, ed 2, DHEW (NIOSH) Publication No. 78-175. Cincinnati, US Dept of Health, Education, and Welfare, Public Health Service, Center for Disease Control, National Institute for Occupational Safety and Health, 1978, vol 4, pp S381-1 to S381-8

N03287 Zinc Oxide--Standards Completion Program Validated Method No. S-383, in NIOSH Manual of Analytical Methods, ed 2, DHEW (NIOSH) Publication No. 78-175. Cincinnati, US Dept of Health, Education, and Welfare, Public Health Service, Center for Disease Control, National Institute for Occupational Safety and Health, 1978, vol 4, pp S316-1 to S316-9

N03288 Free Silica (Quartz, Cristobalite, Tridymite) in Airborne Dust--Physical and Chemical Analysis Branch Method No. 259, in NIOSH Manual of Analytical Methods, ed 2, DHEW (NIOSH) Publication No. 79-141. Cincinnati, US Dept of Health, Education, and Welfare, Public Health Service, Center for Disease Control, National Institute for Occupational Safety and Health, 1979, vol 5, pp 259-1 to 259-3

N03289 Sulfate, Sulfite and Sulfur Dioxide--Physical and Chemical Analysis Branch Method No. 268, in NIOSH Manual of Analytical Methods, ed 2, DHEW (NIOSH) Publication No. 79-141. Cincinnati, US Dept of Health, Education, and Welfare, Public Health Service, Center for Disease Control, National Institute for Occupational Safety and Health, 1979, vol 5, pp 268-1 to 268-7

N03290 Carbon Monoxide--Standards Completion Program Validated Method No. S-340, in NIOSH Manual of Analytical Methods, ed 2, DHEW (NIOSH) Publication No. 78-175. Cincinnati, US Dept of Health, Education, and Welfare, Public Health Service, Center for Disease Control, National Institute for Occupational Safety and Health, 1978, pp S340-1 to S340-5

N03291 Fontana MG, Greene ND: Corrosion Engineering, ed 2. New York, McGraw-Hill Book Company, 1978, p 198

N03293 Huber JE (ed): Kline Guide to the Paper Industry, ed 4. Fairfield, NJ, Charles H Kline, 1980

N03294 Asbestos fibers in air--Physical and Chemical Analysis Branch Method No. 239, in NIOSH Manual of Analytical Methods, ed 2, DHEW (NIOSH) Publication No. 77-157-A. Cincinnati, US Dept of Health, Education, and Welfare, Public Health Service, Center for Disease Control, National Institute for Occupational Safety and Health, 1977, vol 1, pp 239-1 to 239-21

N03295 Styrene--Standards Completion Program Validation Method No. S-30, in NIOSH Manual of Analytical Methods, ed 2, DHEW (NIOSH) Publication No. 77-157-B. Cincinnati, US Dept of Health, Education, and Welfare, Public Health Service, Center for Disease Control, National Institute for Occupational Safety and Health, 1977, vol 2, pp S30-1 to S30-8

N03296 Epichlorohydrin--Standards Completion Program Validation Method No. S-118, in NIOSH Manual of Analytical Methods, ed 2, DHEW (NIOSH) Publication No. 77-157-B. Cincinnnati, US Dept of Health, Education, and Welfare, Public Health Service, Center for Disease Control, National Institute for Occupational Safety and Health, 1977, vol 2, pp S118-1 to S118-8

N03297 Pulp and Paper Mills Plant Observation Report and Evaluation. Philadelphia, Franklin Research Center, May 1980, 40 pp (submitted to NIOSH under Contract No. 210-79-0091)

N03298 Formaldehyde--Standards Completion Program Validation Method No. S-327, in NIOSH Manual of Analytical Methods, ed 2, DHEW (NIOSH) Publication No. 78-175. Cincinnati, US Dept of Health, Education, and Welfare, Public Health Service, Center for Disease Control, National Institute for Occupational Safety and Health, 1978, vol 4, pp S327-1 to S327-8

N03299 Carbon Monoxide--Sampling Data Sheet No. S-340, in NIOSH Manual of Sampling Data Sheets Supplement to 1977 Edition, DHEW (NIOSH) Publication No. 78-189. Cincinnati, US Dept of Health, Education, and Welfare, Public Health Service, Center for Disease Control, National Institute for Occupational Safety and Health, 1978, pp 340-1 to S340-2

N03300 Evaluation of dust samples, in Patty FA (ed.): Industrial Hygiene and Toxicology, ed 2. New York, Interscience, 1958, vol 1, pp 195-209

TABLE VI-1

CHEMICAL AND PHYSICAL HAZARDS ASSOCIATED WITH THE MANUFACTURE OF PULP AND PAPER

Hazard	CAS #	RTECS #	OSHA #**	NOHS #***
*Acrylamide	79-06-1	AS3325000	0115	03540
Aluminum sulfate (alum)	10043-01-3	BD1700000	--	04620
*Aroclor 1242 (PCB)	53469-21-9	TQ1356000	--	--
*Aroclor 1254 (PCB)	27323-18-8	TQ1360000	--	--
*Asbestos	1332-21-4	CI6475000	9020	90310
Bagasse (sugar cane) dust	--	--	--	--
Calcium oxide (lime)	1305-78-8	EW3100000	0520	15755
*Carbon black	7440-44-0	FF5250000	0527	80245
*Carbon monoxide	630-08-0	FG3500000	17460	17460
*Chlorine	7782-50-5	FO2100000	0640	18040
Chlorine dioxide	10049-04-4	FO3000000	0614	18045
*Confined spaces	--	--	--	--
*Cotton dust	--	GN2275000	0735	M2326
Dimethyl disulfide	624-92-0	JO1927500	--	--
Dimethyl sulfide	75-18-3	PV5075000	--	84745
Dimethyl sulfoxide	67-68-5	PV6210000	--	80564
*Formaldehyde	50-00-0	LP8925000	1290	33640
*Heat	--	--	8330	--
*Humidity (high)	--	--	--	--
*Hydrazine	302-01-2	MU7175000	1390	38110
*Hydrogen sulfide	7783-06-4	MX1225000	1480	38620
Kaolin	1332-58-7	--	--	41775
*Kerosene	8008-20-6	OA5500000	1570	--
Magnesium oxide	1309-48-4	OM3850000	--	80298
Melamine formaldehyde resins	9003-08-1	--	--	82126
*Methyl mercaptan	74-93-1	PB4375000	1643	45850
*Noise, continuous	--	--	8110	--
*Noise, intermittent	--	--	8120	--
Rosin	8050-09-7	--	--	80164
*Silica, crystalline (diatomite, cristobalite, and tridymite)	60676-86-0	VV7330000	9050	M4331
*Sodium hydroxide	1310-73-2	WB4900000	2260	69070
Sodium chlorate	7775-09-9	FO0525000	--	68870
Sodium hypochlorite	7681-52-9	--	--	69090
Sodium sulfate	7757-82-6	WE1650000	--	81663
Sodium sulfide	1313-82-2	WE1905000	--	69460
Sulfur	7704-34-9	WS4250000	--	70845
*Sulfur dioxide	7446-09-5	WS4550000	2290	70860
*Sulfuric acid	7664-93-9	WS5600000	2310	70870
Sulfurous acid	7782-99-2	WT2775000	--	82237
Talc (fibrous)	14807-96-6	WW2710000	9030	71055
Tall oil	8002-26-4	--	--	80216
Titanium dioxide	13463-67-7	XR2275000	2440	A1358
Turpentine	8006-64-2	Y08400000	2540	74990

TABLE VI-1 (CONTINUED)

CHEMICAL AND PHYSICAL HAZARDS ASSOCIATED WITH THE MANUFACTURE OF PULP AND PAPER

Hazard	CAS #	RTECS #	OSHA #**	NOHS #***
Urea formaldehyde (resins)	9011-05-6	--	--	M3046
Vanillin	121-33-5	--	--	M0609
Wood dust	--	--	9210	M1327
*Zinc oxide	1314-13-2	ZH4810000	2610	77190

*Subject of NIOSH criteria document
**OSHA Hazardous Substance Code (MIS)
***NOHS Hazard Code

TABLE VI-2

OSHA STANDARDS AND RECOMMENDED EXPOSURE LEVELS FOR PHYSICAL AND CHEMICAL HAZARDS IN PULP AND PAPER MILLS

Hazards	OSHA Standards[a] (mg/m^3)*	ACGIH Recommendation[b] (mg/m^3)*	NIOSH Recommendation[c] (mg/m^3)*	NIOSH Reference
Physical and Dust hazards				
Wood dust	--	1.0**	0.5***	--
Cotton dusts	1.0	0.2	0.2	N02902
Asbestos	2.0 fb/cm^3 (10 fb/cm^3, ceiling level)	5.0 fb/cm^3	0.5 fb/cm^3 > 5 um in length	N02903
Talc				
nonasbestos	20 mppcf****	20 mppcf	20 mppcf	N02906
fibrous	2.0 fb/cm^3 (air) (10.0 fb/cm^3, ceiling level)	5.0 fb/cm^3 > 1 um in length	0.1 fb/cm^3 (air) 0.5 fb/cm^3 (ceiling level, 15 min)	N02906
Hot, humid environments	--	--	d	N02778
Noise	--	--	e	N02905
Confined spaces	--	--	f	N02875
Raw materials or chemical intermediates				
Sulfuric acid	1.0	1.0	1.0	N02496
Sodium sulfate	--	--	--	--
Sodium sulfide	--	--	--	--
Sulfur	--	--	--	--
Sodium hydroxide	2.0	2.0	2.0 (15 min)	N02502
Magnesium oxide (fumes)	15.0	10.0	--	--
Pulp bleaching agents				
Chlorine	3.0	3.0	2.0 (ceiling level, 15 min)	N02501
Chlorine dioxide	0.3	0.3	--	--
Sodium hypochlorite	--	--	--	--
Sodium chlorate	--	--	--	--
Papermaking additives				
Titanium dioxide	15.0	10.0	--	--
Acrylamide	0.3 (skin)	0.3	0.3 (air)	N02929
Formaldehyde	3.7 (6.2, ceiling level) 12.5, maximum peak (10 min)	3.0	1.2 (30 min)	N02497
Melamine formaldehyde resin (polymer)	--	--	--	--
Urea-formaldehyde resin (polymer)	--	--	--	--

TABLE VI-2 (CONTINUED)

OSHA STANDARDS AND RECOMMENDED EXPOSURE LEVELS FOR PHYSICAL AND CHEMICAL HAZARDS IN PULP AND PAPER MILLS

Hazards	OSHA Standards[a] (mg/m^3)*	ACGIH Recommendation[b] (mg/m^3)*	NIOSH Recommendation[c] (mg/m^3)*	NIOSH Reference
Zinc oxide (fume)	5.0	5.0	5.0 (15.0,ceiling level, 15 min)	N02917
Crystalline silica	5/(% SiO_2 + 2) respirable dust	0.5 fb/cm^3	50 ug/m^3	N02908
Alum (aluminum sulfate)	--	--	--	--
Rosin	--	0.1 (solder fumes)	--	--
Amorphous (diatomaceous) silica	80.0/(% SiO_2)	1.5	50 ug/m^3	--
Carbon black	3.5	3.5	3.5 (air)	N02904
Contaminants or byproducts				
Turpentine	560.0	560.0	--	--
Tall oil	--	--	--	--
Polychlorinated biphenyls	0.5-1.0	0.5-1.0	1 ug/m^3 (air)	N02926
Aroclor 1242	1.0 (skin)	1.0 (skin)	--	--
Aroclor 1254	0.5 (skin)	1.0 (skin)	--	--
Dimethyl sulfoxide	--	--	--	--
Hydrazine	1.3	0.1	0.04 (2 hr)	N03265
Kerosene	--	--	--	--
Vanillin	--	--	--	--
Pulping effluents				
Sulfur dioxide	13.0	13.0	5.0	N02927
Hydrogen sulfide	28.3 ceiling level, and 70.9 max peak (one exposure, 10 min duration)	15.0	15.0 (10 min)	N02493
Methyl mercaptan	20.0	1.0	1.0	N02907
Dimethyl sulfide	--	--	--	--
Dimethyl disulfide	--	19.6	--	--
Carbon monoxide	55.0	55.0	40.8 (233.0, ceiling level)	N03142

*Unless otherwise noted
**This value is for hardwood, as in furniture making
***Written communication, draft NIOSH criteria document on wood dust, May 1980
****mppcf = million particles per cubic foot

TABLE VI-2 (CONTINUED)

OSHA STANDARDS AND RECOMMENDED EXPOSURE LEVELS FOR PHYSICAL AND CHEMICAL HAZARDS
IN PULP AND PAPER MILLS

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[a] OSHA Safety and Health Standards (29 CFR 1910), revised 1978

[b] Threshold Limit Values, American Conference of Governmental Industrial Hygienists, 1979

[c] NIOSH Criteria Document

[d] For sedentary jobs where continuous unimpaired mental performance is required, no employee shall be exposed to conditions which exceed the limits set forth in Figure A, below. No employee should be permitted to work without protective observation at high heat stress levels. When exposure of an employee is continuous for 1 hour or intermittent for a period of 2 hours and the time-weighted average Wet Bulb Globe Temperature (WBGT) exceeds 79 F for men or 76 F for women, then practices shall be initiated to ensure that the employee's body care temperature does not exceed 100.4 F. The WBGT for indoor exposure shall be calculated as: 0.7 WB + 0.3 GT, where WB = the natural wet bulb temperature obtained with a wetted sensor exposed to the natural air movement (unaspirated), and GT = globe thermometer temperature. The WGBT for outdoor sunlit exposure shall be calculated as: 0.7 WB + 0.2 GT + 0.1 DB, where DB = dry bulb temperature.

[e] Occupational noise exposure shall be controlled so that no worker shall be exposed in excess of the limit described as line B in Figure B, below. New installations shall be designed with noise control so that the noise exposure does not exceed the limits described as line A in Figure B. For noise exposures consisting of two or more periods of exposure at different levels, the Daily Noise Dose, D, shall not exceed unity. Line A or line B, as applicable, shall be used in computing the Daily Noise Dose. At no time shall any worker be exposed to effective noise levels exceeding 115 dBA-Slow. "Daily Noise Dose" means that value for D derived from the equation: $D = C_1/T_1 + C_2/T_2 + \ldots + C_n/T_n$, where C_1, C_2 ... C_n are the actual durations of exposure for an employee at the various noise levels, T_1, T_2 ... T_n are the respective duration limits obtained from Figure B, and D is the Daily Noise Dose.

[f] Lifelines and safety harness shall be worn by anyone entering closed vessels, tanks, chip bins, and similar equipment, and a person shall be stationed outside in a position to handle the line and to summon assistance in case of emergency. The air in the vessels shall be tested for oxygen deficiency and the presence of both toxic and explosive gases and vapors, before entry is permitted into closed vessels, tanks, etc. Self-contained air- or oxygen-supply masks shall be readily available in case of emergency. Work shall not be done on equipment under conditions where an injury would result if a valve were unexpectedly opened or closed unless the valve has been locked in a safe position.

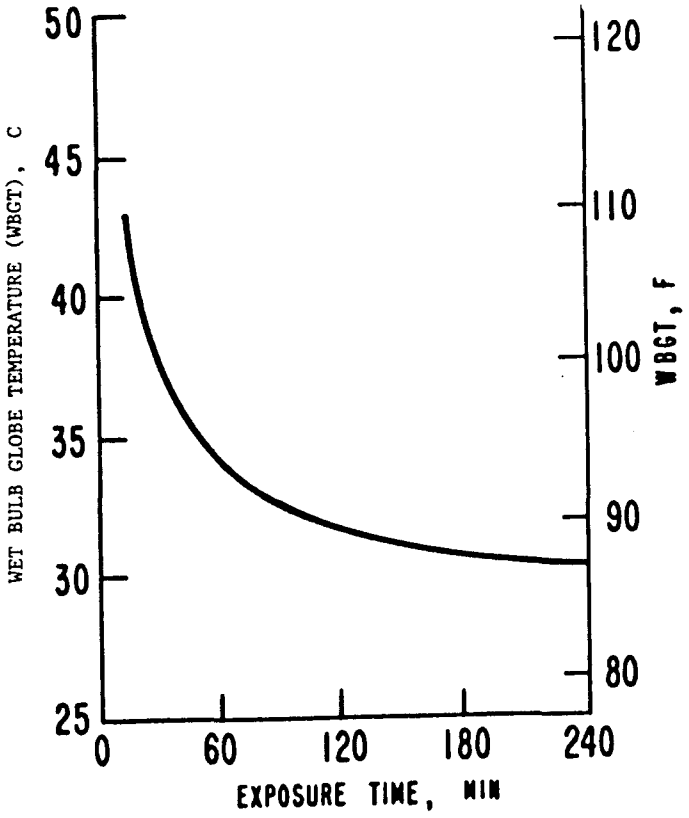

FIGURE A. UPPER LIMITS OF EXPOSURE FOR UNIMPAIRED MENTAL PERFORMANCE

Source: NO2778

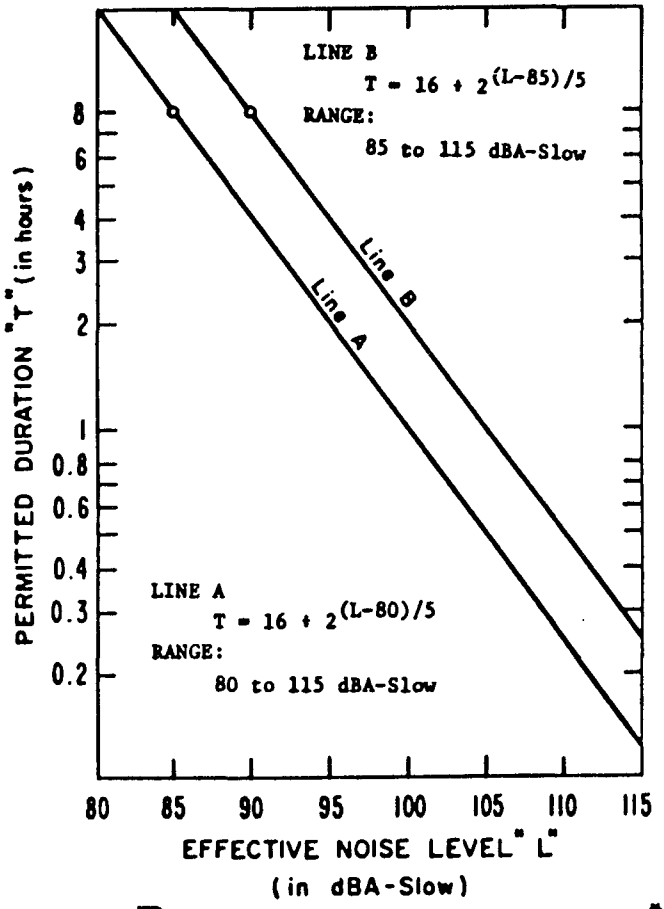

Figure B. Permitted duration vs. noise level.*

*The indicated duration limits which exceed 8 hours are to be used only for purposes of computing Daily Noise Dose and are not to be regarded as defining noise exposure limits for work days which exceed 8 hours.

Source: NO2778

TABLE VI-3

NUMBER OF WORKERS IN PULP AND PAPER MILLS

(in thousands)

SIC Classification	1972	1973	1974	1975	1976	1977	1978
2611--Pulp mills	13.5	14.4	11.6	14.0	14.5	17.6	17.3
2621--Paper mills	137.0	139.4	128.7	118.3	125.3	126.5	126.4
2631--Paperboard mills	67.9	68.0	71.0	63.2	67.0	69.3	68.9
2661--Building paper and building board mills	11.0	11.4	13.2	11.8	10.5	8.8	10.8

Source: County Business Patterns. US Dept of Commerce, Bureau of the Census, 1972 through 1978 (Mr. Steven Newell, Bureau of Labor Statistics, written communication, October 1980)

TABLE VI-4

OCCUPATIONS OF PRODUCTION WORKERS
IN PULP AND PAPER MILLS

Census Occupation Code	Description of Occupation
162	ENGINEERING AND SCIENCE TECHNICIANS, N.E.C.
190	PAINTERS AND SCULPTORS
381	STOCK CLERKS AND STOREKEEPERS
392	WEIGHERS
410	BRICKMASONS AND STONEMASONS
412	BULLDOZER OPERATORS
415	CARPENTERS
424	CRANEMEN, DERRICKMEN, AND HOISTMEN
430	ELECTRICIANS
436	EXCAVATING, GRADING, AND ROAD MACHINE OPERATORS; EXC. BULLDOZER
441	FOREMEN, N.E.C.
452	INSPECTORS, N.E.C.
461	MACHINISTS
492	MISCELLANEOUS MECHANICS AND REPAIRMEN
495	NOT SPECIFIED MECHANICS AND REPAIRMEN
502	MILLWRIGHTS
510	PAINTERS, CONSTRUCTION AND MAINTENANCE
522	PLUMBERS AND PIPE FITTERS
525	POWER STATION OPERATORS
530	PRESSMEN AND PLATE PRINTERS, PRINTING
535	SHEETMETAL WORKERS AND TINSMITHS
545	STATIONARY ENGINEERS
601	ASBESTOS AND INSULATION WORKERS
602	ASSEMBLERS
610	CHECKERS, EXAMINERS, AND INSPECTORS; MANUFACTURING
612	CUTTING OPERATIVES, N.E.C.
620	DYERS
624	GRADERS AND SORTERS, MANUFACTURING
641	MIXING OPERATIVES
642	OILERS AND GREASERS, EXC. AUTO
643	PACKERS AND WRAPPERS, EXCEPT MEAT AND PRODUCE
651	GRINDING MACHINE OPERATIVES
660	RIVETERS AND FASTENERS
662	SAWYERS
666	STATIONARY FIREMEN
680	WELDERS AND FLAME-CUTTERS
681	WINDING OPERATIVES, N.E.C.
690	MACHINE OPERATIVES, MISCELLANEOUS SPECIFIED
692	MACHINE OPERATIVES, NOT SPECIFIED
694	MISCELLANEOUS OPERATIVES

TABLE V1-4 (CONTINUED)

OCCUPATIONS OF PRODUCTION WORKERS
IN PULP AND PAPER MILLS

Census Occupation Code	Description of Occupation
695	NOT SPECIFIED OPERATIVES
706	FORK LIFT AND TOW MOTOR OPERATIVES
753	FREIGHT AND MATERIAL HANDLERS
755	GARDENERS AND GROUNDSKEEPERS, EXC. FARM
762	STOCK HANDLERS
764	VEHICLE WASHERS AND EQUIPMENT CLEANERS
770	WAREHOUSEMEN, N.E.C.
780	MISCELLANEOUS LABORERS
785	NOT SPECIFIED LABORERS
902	CLEANERS AND CHARWOMEN
903	JANITORS AND SEXTONS

TABLE VI-5

OCCUPATIONS OF NONPRODUCTION WORKERS
IN PULP AND PAPER MILLS

Census Occupation Code	Description of Occupation
1	ACCOUNTANTS
45	CHEMISTS
55	OPERATIONS AND SYSTEMS RESEARCHERS AND ANALYSTS
56	PERSONNEL AND LABOR RELATIONS WORKERS
75	REGISTERED NURSES
85	HEALTH TECHNOLOGISTS AND TECHNICIANS, N.E.C.
191	PHOTOGRAPHERS
195	RESEARCH WORKERS, NOT SPECIFIED
245	MANAGERS AND ADMINISTRATORS, N.E.C.
280	SALESMEN AND SALES CLERKS, N.E.C.
303	BILLING CLERK
305	BOOKKEEPERS
312	CLERICAL SUPERVISORS, N.E.C.
332	MAIL HANDLERS, EXCEPT POST OFFICE
343	COMPUTER AND PERIPHERAL EQUIPMENT OPERATORS
344	DUPLICATING MACHINE OPERATORS
345	KEY PUNCH OPERATORS
360	PAYROLL AND TIMEKEEPING CLERKS
372	SECRETARIES, N.E.C.
376	STENOGRAPHERS
391	TYPISTS
394	MISCELLANEOUS CLERICAL WORKERS
395	NOT SPECIFIED CLERICAL WORKERS
405	BOOKBINDERS
422	COMPOSITORS AND TYPESETTERS
484	OFFICE MACHINE
645	PHOTOGRAPHIC PROCESS WORKERS
914	FOOD COUNTER AND FOUNTAIN WORKERS
943	ELEVATOR OPERATORS

Source: Bureau of the Census, Dept of Commerce

TABLE VI-6

OCCUPATIONS OF MISCELLANEOUS WORKERS
IN PULP AND PAPER MILLS

Census Occupation Code	Description of Occupation
13	INDUSTRIAL ENGINEERS
23	ENGINEERS, N.E.C.
154	INDUSTRIAL ENGINEERING TECHNICIANS
323	EXPEDITERS AND PRODUCTION CONTROLLERS
374	SHIPPING AND RECEIVING CLERKS
473	AUTOMOBILE MECHANICS
481	HEAVY EQUIPMENT MECHANICS, INCL. DIESEL
514	PATTERN AND MODEL MAKERS, EXC. PAPER
644	PAINTERS, MANUFACTURED ARTICLES

Source: Bureau of the Census, Dept of Commerce

TABLE VI-7

NOHS DATA ON PRODUCTION WORKER EXPOSURES TO HAZARDS

Agent Name	CAS #	NIOSH Hazard Number	Percent of Group Exposed	Actual # Exposures to Agent	Trade-Name Ingredient Exposures
CONTINUOUS NOISE	--	P0610	48.8	2404	0
WATERLESS HAND CLEANER	--	76612	30.9	1521	0
WATER	7732-18-5	M1000	25.9	0	1274
POTENTIAL CONTINUOUS NOISE	8077-20-1	P0615	20.5	1007	0
UNDEFINED CHEMICAL	--	MMMMM	18.6	0	916
GLUE-OTHER	--	92460	16.1	792	0
PRODUCTS OF COMBUSTION-PROPANE	--	60719	14.8	727	0
PAPER	--	94140	12.7	627	0
MINERAL OIL	8020-83-5	M0603	12.2	20	582
STARCH	9005-25-8	69738	11.7	388	186
SOLVENT	--	92910	11.2	549	0
OIL, LUBE	--	52138	11.1	547	0
SODIUM CARBONATE	497-19-8	68850	10.4	365	147
DEFOAMER AGENT	--	92200	9.6	471	0
PROPYLENE GLYCOL	57-55-6	63525	9.5	70	397
STEEL	12597-69-2	17475	9.3	456	0
OIL, FUEL NO. 1	--	91115	9.2	378	76
GRINDING WHEEL DUST	--	35507	9.2	451	0
ASPHALT	8052-42-4	90320	8.5	397	22
FATTY ACIDS	--	M0218	8.3	0	409
SODIUM CHLORIDE	7647-14-5	68880	8.2	183	222
DYES	--	92290	8.1	397	0
PETROLEUM SPIRITS	--	M2829	7.8	81	305
ALUMINUM SULFATE	10043-01-3	04620	7.7	377	0
ULTRAVIOLET RADIATION	--	P0430	7.4	366	0
DICHLORODIFLUOROMETHANE	75-71-8	24095	7.3	18	341
OIL, PENETRATING	--	52143	7.2	353	0
TITANIUM OXIDE	13463-67-7	A1358	7.2	126	226
ETHYL ALCOHOL	64-17-5	31500	7.0	64	281
INK	--	92520	6.9	342	0
SODIUM HYDROXIDE	1310-73-2	69070	6.8	218	119
PRODUCTS OF COMBUSTION-GASOLINE (LEADED)	--	60713	6.8	337	0
ADDITIVE	--	80036	6.7	332	0
WOOD PULP SOLUTION	--	82910	6.7	332	0
LUBRICANT	--	92570	6.7	332	0
WAX	8063-08-9	76614	6.7	264	66
SILICA, AMORPHOUS FUSED	--	84055	6.5	57	261
STEEL, OXIDES OF	--	80124	6.3	309	0
POLYETHYLENE	9002-88-4	M0907	6.1	0	301
INFRARED RADIATION	--	P0410	6.1	298	0
TOLUENE	108-88-3	73300	6.0	155	138
WOODS	--	94220	5.7	282	0
NAPHTHENIC BASE OIL	--	M3358	5.7	0	279
UNIDENTIFIED CHEMICAL	--	75158	5.7	279	0

Agent Name	CAS #	NIOSH Hazard Number	Percent of Group Exposed	Actual # Exposures to Agent	Trade-Name Ingredient Exposures
ISOPROPYL ALCOHOL	67-63-0	40987	5.6	118	157
METHANOL	67-56-1	45930	5.6	56	218
WELDING RODS	--	76618	5.5	273	0
HYDROGEN CHLORIDE	7647-01-0	38580	5.5	206	66
DICHLOROMETHANE	75-09-2	47270	5.4	31	234
ACETYLENE	74-86-2	03298	5.3	263	0
CARBON ARC LAMP GASES	--	76616	5.3	263	0
SODIUM SULFATE	7757-82-6	81663	5.2	1	257
CALCIUM CARBONATE	471-34-1	15705	5.2	177	78
OLEIC ACID	112-80-1	52190	5.2	0	254
SILICONES	--	84183	5.1	149	102
CLEANING COMPOUND	--	92160	5.1	250	0
GREASE	--	92470	5.1	250	0
ASBESTOS	1332-21-4	90310	5.0	248	0
ETHYL ACETATE	141-78-6	31470	4.8	109	125
AMMONIA	7664-41-7	05250	4.8	141	93
OIL, CUTTING	--	52131	4.8	234	0
CELLULOSE, NITRATE	9004-70-0	81779	4.7	0	231
MOLD RELEASE	--	92610	4.7	229	0
BRIGHT STOCK	--	M0717	4.6	0	227
WELDING GASES, GAS	--	76617	4.6	225	0
BUTANONE, 2-	78-93-3	13980	4.4	113	106
NEUTRAL OIL	--	M0716	4.4	0	216
ALUMINUM OXIDE	1344-28-1	20265	4.3	18	192
DEXTRIN	9004-53-9	83807	4.2	0	209
CARBON	7440-44-0	80243	4.1	18	186
DESCUMMING AGENT	--	92250	4.1	202	0
ROSIN	8050-09-7	80164	4.1	12	189
COATING	--	19769	4.1	201	0
POLYETHYLENE GLYCOL MONO(OCTYLPHENYL) ETHER	9036-19-5	81435	4.1	0	200
C.I. 74160-PIGMENT BLUE 15	147-14-8	M0910	4.0	0	196
ACETIC ACID	64-19-7	01568	3.9	66	128
PARAFFIN	8002-74-2	M0600	3.9	57	136
ADHESIVES	--	92030	3.9	193	0
TRICHLOROETHANE, 1,1,1-	71-55-6	46970	3.9	23	167
SULFURIC ACID	7664-93-9	70870	3.9	92	98
ALCOHOL	64-17-5	M0238	3.9	112	78
ABSORBENT	--	92021	3.8	188	0
STEARIC ACID	57-11-4	69740	3.8	0	187
PETROLEUM OIL	--	M4242	3.8	0	187
GLYCEROL	56-81-5	35085	3.8	85	100
CLAYS	--	90590	3.8	156	29
CELLULOSE	9004-34-6	17683	3.7	184	0
CHROMIC ACID, LEAD(2+) SALT (1:1)	7758-97-6	81876	3.6	0	179
OIL, MOTOR	--	52141	3.6	98	79

Agent Name	CAS #	NIOSH Hazard Number	Percent of Group Exposed	Actual # Exposures to Agent	Trade-Name Ingredient Exposures
SIZING COMPOUND	--	80002	3.6	177	0
UREA	57-13-6	80231	3.6	6	170
ETHOXYETHANOL, 2-	110-80-5	31350	3.5	75	97
C.I. 52015-BASIC BLUE 9	61-73-4	M1469	3.5	0	170
POLYETHYLENE GLYCOL ETHER	25322-68-3	80189	3.5	57	113
C.I. 77266-PIGMENT BLACK 7	1333-86-4	M0647	3.4	0	169
FORMALDEHYDE	50-00-0	33640	3.3	43	121
SURFACTANTS	--	M1596	3.3	0	163
ALKANES	--	M0388	3.3	0	161
C.I. 23850-DIRECT BLUE 14	72-57-1	M2160	3.3	0	161
ETHYLHEXYL) PHTHALATE, BIS(2-	117-81-7	M0347	3.3	16	144
PROPANE	74-98-6	26615	3.3	33	127
CALCIUM HYDROXIDE	1305-62-0	15743	3.2	39	120
SODIUM POLYACRYLATE	9003-04-7	83142	3.2	0	158
SODIUM HYPOCHLORITE	7681-52-9	69090	3.2	8	149
COAGULANT	--	19765	3.2	156	0
VIBRATION	--	P0650	3.1	155	0
BLEACHING AGENTS	--	92100	3.1	154	0
TRIAZINE-SULFONAMIDE RESIN	--	M4221	3.1	0	153
LANOLIN	8006-54-0	80051	3.1	0	152
TALC	14807-96-6	71055	3.1	125	26
DIMETHICONE	9006-65-9	80900	3.0	0	150
AMINES	--	80177	3.0	0	149
C.I. 77891-PIGMENT WHITE 6	62338-64-1	M0913	3.0	0	149
ALUMINUM HYDROXIDE (AL(OH)3)	21645-51-2	80390	3.0	9	139
LATEX	--	91150	3.0	147	0
DICYANDIAMIDE	461-58-5	83038	2.9	0	144
ACRYLIC RESINS	--	80750	2.9	16	127
DIETHYLENE GLYCOL MONOETHYL ETHER	111-90-0	83102	2.9	0	142
C.I. 40000-DIRECT YELLOW 11	1325-37-7	M1422	2.9	0	141
C.I. 45160:1-PIGMENT RED 81	12224-98-5	M0937	2.8	0	139
PHOSPHORIC ACID	7664-38-2	58520	2.8	3	135
RUBBER, CHLOROPRENE	--	82884	2.8	0	137
POLYMERS, ORGANIC	--	64248	2.8	136	0
TRICHLOROFLUOROMETHANE	75-69-4	33565	2.7	0	135
C.I. 74180-DIRECT BLUE 86	1330-38-7	M2255	2.7	0	134
DIETHANOLAMINE	111-42-2	24615	2.7	0	133
PHENOL, POLYMER WITH FORMALDEHYDE	9003-35-4	80184	2.7	3	129
C.I. 29200- DIRECT RED 72	8005-64-9	M1419	2.7	0	131
C.I. 24401-DIRECT BLUE 218	10401-50-0	M2161	2.7	0	131
SHELLAC	9000-59-3	68512	2.7	16	115
POLYVINYL ACETATE	9003-20-7	81943	2.7	25	106
REAGENT	--	80088	2.7	131	0
PRODUCTS OF ROSIN CORE SOLDER	--	80172	2.7	131	0
SEALANT	--	92865	2.6	128	0

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LEAD SULFATE	7446-14-2	84546	2.6	0	127
DYE SOLVENT	--	92300	2.6	127	0
MICROCRYSTALLINE WAX	--	M0602	2.6	0	126
SODIUM ALUMINATE	1302-42-7	81975	2.6	126	0
DYE STUFF	--	92310	2.5	125	0
DIMETHYL SULFATE	77-78-1	26880	2.5	0	122
C.I. 42595:2-PIGMENT BLUE 1	1325-87-7	M2200	2.5	0	122
SURFACTANT	--	70995	2.5	122	0
ISOPROPYL ACETATE	108-21-4	40984	2.5	0	121
ACETONE	67-64-1	02820	2.5	69	52
DECOMPOSITION PRODUCTS-FLUX	--	80125	2.4	120	0
AMINOETHYLETHANOL AMINE	111-41-1	84805	2.4	0	119
C.I. 29000-DIRECT YELLOW 44	8005-52-5	M2174	2.4	0	119
C.I. 27680-DIRECT RED 16	6227-02-7	M3048	2.4	0	119
POLY(OXY-1,2-ETHANEDIYL), ALPHA-(4-NONYLPHENYL)-OMEGA-HYDROXY-	26027-38-3	M4862	2.4	0	119
C.I. 12355-PIGMENT RED 23	6471-49-4	M2113	2.4	0	118
C.I. 77600-PIGMENT YELLOW 34	1344-37-2	M0926	2.4	0	117
PROPANOL, 1-	71-23-8	M0256	2.4	4	112
MALEIC MODIFIED PENTA ESTER OF RESIN	--	M0640	2.3	0	115
C.I. 45160-BASIC RED 1	989-38-8	M2209	2.3	0	115
DIETHYLAMINOETHANOL	100-37-8	24930	2.3	0	114
C.I. 15630-PIGMENT RED 49	1248-18-6	M0915	2.3	0	113
FLUX	--	92410	2.3	113	0
LACTOL SPIRITS	--	M0674	2.3	0	112
POLYAMIDE RESINS	--	80186	2.2	0	109
XYLENE	1330-20-7	76720	2.2	29	80
HYDROXYETHYL STARCH	9005-27-0	M2691	2.2	0	108
HEPTANE	142-82-5	36060	2.2	0	106
C.I. 77605-PIGMENT RED 104	12656-85-8	M0916	2.2	0	106
POLYAMIDE RESIN	--	M3873	2.2	0	106
WETTING AGENT	--	92980	2.1	53	52
SODIUM BORATE	1333-73-9	80517	2.1	58	47
SODIUM GLUCONATE	527-07-1	81987	2.1	0	104
COPPER(II) SULFATE (1:1)	7758-98-7	20900	2.1	103	0
CHLOROETHANE	75-00-3	31970	2.1	0	102
MALEIC RESIN	--	83017	2.1	0	101
C.I. 77510-PIGMENT BLUE 27; C.I. 77520-PIGMENT BLUE 27	25869-98-1	M0650	2.1	0	101
STYRENATED ACRYLIC RESIN	--	M1954	2.0	0	100
SODIUM PYROPHOSPHATE, TETRA	7722-88-5	80224	2.0	11	88
POLYVINYL ALCOHOL	9002-89-5	81944	2.0	36	62
OIL, LINSEED	--	80053	1.9	0	95
C.I. 21100-PIGMENT YELLOW 13	5102-83-0	M0914	1.9	0	95
GRAPHITE	7782-42-5	17366	1.9	28	67
ETHANE, 1,1,2-TRICHLORO-1,2,2-TRIFLUORO-	76-13-1	74010	1.9	0	94
TALL OIL	8002-26-4	80216	1.9	0	94

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MALEIC ROSIN	--	M1567	1.9	0	94
ACETIC ACID ÉTHENYL ESTER, POLYMER WITH CHLOROETHENE	9003-22-9	M1750	1.9	0	94
ETHYLENE GLYCOL	107-21-1	32385	1.9	28	65
AMMONIUM HYDROXIDE	1336-21-6	06145	1.9	62	31
METHYL-2-PENTANONE, 4-	108-10-1	37510	1.9	0	92
C.I. 45175-BASIC VIOLET 11	2390-63-8	M2212	1.9	0	92
C.I. 74260-PIGMENT GREEN 7	1328-53-6	M0912	1.8	0	91
CASTOR OIL	8001-79-4	M1030	1.8	0	91
CALCIUM STEARATE	1592-23-0	15765	1.8	12	79
PROPYLENE GLYCOL METHYL ETHER	107-98-2	81815	1.8	0	90
C.I. 15585-PIGMENT RED 53	2092-56-0	M0645	1.8	0	90
C.I. 15850-PIGMENT RED 57	5858-81-1	M0646	1.8	0	90
DISINFECTANT	--	92260	1.8	89	0
PROPYL ACETATE	109-60-4	63265	1.8	0	88
ALUMINUM PHOSPHATE	7784-30-7	81696	1.8	0	88
ETHYLCELLULOSE	9004-57-3	82229	1.8	0	88
LEAD FLUORIDE	7783-46-2	83015	1.8	0	88
METHYL METHACRYLATE POLYMER	9011-14-7	M0527	1.8	0	88
FORMALDEHYDE-CYCLIC UREIDE-SULFONAMIDE RESIN	--	T0470	1.8	0	88
STYRENATED ALKYD RESIN	--	M0681	1.8	0	87
TRIAZINE-FORMALDEHYDE	--	M1911	1.8	0	87
CAUSTIC CLEANER	--	92140	1.8	87	0
C.I. 45170-BASIC VIOLET 10	81-88-9	84376	1.7	0	85
EPOXIDIZED SOYBEAN OIL	8013-07-8	M0773	1.7	0	85
CEMENT-PORTLAND	--	17695	1.7	85	0
DECOMPOSITION PRODUCTS-POLYMER, POLYETHYLENE	9002-88-4	22749	1.7	85	0
DIETHYL PHTHALATE	84-66-2	81806	1.7	0	84
BARIUM SULFATE	7727-43-7	80611	1.7	0	83
SUCROSE	57-50-1	81515	1.7	0	83
STYRENE-ALLYL ALCOHOL COPOLYMER	25119-62-4	M2415	1.7	0	82
CEMENT-GLUE	--	17494	1.6	81	0
DIETHYLENE GLYCOL MONOBUTYL ETHER	112-34-5	80343	1.6	0	80
OIL, COCONUT	--	82934	1.6	0	80
TALLOW FATTY ACID	61790-37-2	M1174	1.6	0	80
NAPHTHENIC NEUTRAL OIL	--	M4648	1.6	0	80
BUTYL ACETATE	123-86-4	14380	1.6	46	34
MALEIC ANHYDRIDE	108-31-6	43660	1.6	0	79
ESTER GUMS	--	M1022	1.6	0	79
C.I. 77007-PIGMENT BLUE 29	57455-37-5	M1577	1.6	0	79
C.I. 21108-PIGMENT YELLOW 83	5567-15-7	M2074	1.6	0	79
C.I. SOLVENT ORANGE 57	64147-43-9	M2081	1.6	0	79
C.I. SOLVENT YELLOW 48	61725-88-0	M2087	1.6	0	79
C.I. 12315-PIGMENT RED 22	6448-95-9	M2111	1.6	0	79
C.I. 42040-BASIC GREEN 1	633-03-4	M2187	1.6	0	79
C.I. 51319-PIGMENT VIOLET 23	6358-30-1	M2223	1.6	0	79

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MALEIC-MODIFIED PENTAERYTHRITOL ESTER	--	M4048	1.6	0	79
BENTONITE	1302-78-9	90340	1.6	8	71
CONTACT CLEANER-ELECTRICAL	--	20064	1.6	79	0
LEAD CARBONATE	598-63-0	80252	1.6	0	77
BUTANOL	71-36-3	13850	1.6	38	39
ETHANOL, 2-BUTOXY-	111-76-2	29930	1.5	0	76
ETHYL ETHER	60-29-7	32590	1.5	0	76
MENTHOL	89-78-1	44440	1.5	0	76
FABRIC FINISHER	--	92340	1.5	76	0
CALCIUM SULFATE	7778-18-9	80244	1.5	11	64
INORGANIC LEAD COMPOUNDS	--	91160	1.5	75	0
DEGREASER	--	92220	1.5	75	0
COPPER	7440-50-8	20115	1.5	0	73
C.I. 11270-BASIC ORANGE 2	532-82-1	M2097	1.5	0	73
SODIUM NITRATE	7631-99-4	69220	1.5	27	46
GLUE-GUM	--	90925	1.4	70	0
TALL OIL FATTY ACID	61790-12-3	M0700	1.4	0	69
C.I. 41000-BASIC YELLOW 2	2465-27-2	M1444	1.4	0	69
CALCIUM OXIDE	1305-78-8	15755	1.4	34	35
TARS	--	71135	1.4	68	0
ETHYLENE OXIDE	75-21-8	32550	1.4	0	67
SULFAMIC ACID	5329-14-6	82834	1.4	28	39
GLUE-ANIMAL	--	M0607	1.3	0	66
C.I. 22610-DIRECT BLUE 6	2602-46-2	M1416	1.3	0	66
C.I. 28160-DIRECT RED 81	2610-11-9	M2171	1.3	0	66
C.I. 56200-SOLVENT YELLOW 44	2478-20-8	M4220	1.3	0	66
BELT DRESSING	--	80040	1.3	66	0
RUST INHIBITOR	--	92855	1.3	66	0
SOAP	--	68766	1.3	63	2
C.I. 42535:2-PIGMENT VIOLET 3	1325-82-2	M0934	1.3	0	63
C.I. 42600:1-PIGMENT BLUE 14	1325-88-8	M2201	1.3	0	63
DECOMPOSITION PRODUCTS-RESIN, PHENOLIC	--	22755	1.3	63	0
WAXES	--	M0599	1.3	0	62
ETHOXYLATED ALCOHOL	--	M0888	1.3	0	62
MAGNESIUM SULFATE	7487-88-9	43410	1.3	1	61
CALCIUM CHLORIDE	10043-52-4	15720	1.3	28	34
DIISOPROPYLAMINE	108-18-9	25850	1.2	0	60
SODIUM METASILICATE	6834-92-0	80079	1.2	0	60
FILLER COMPOUND	--	80022	1.2	60	0
LARD OILS	8016-28-2	M1047	1.2	0	59
C.I. 21000-BASIC BROWN 1	10114-58-6	84046	1.2	0	58
HEXANE	110-54-3	36955	1.2	16	42
NITRILOTRIETHANOL, 2,2',2"-	102-71-6	M0260	1.1	0	56
SODIUM SULFITE	7757-83-7	69470	1.1	18	38
ALUMINUM STEARATE	637-12-7	80037	1.1	0	54

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GASOLINES	--	M0541	1.1	0	54
C.I. 30235-DIRECT BLACK 38	1937-37-7	M1410	1.1	0	54
C.I. 40001-DIRECT YELLOW 6	1325-38-8	M2181	1.1	0	54
PHTHALIC ACID, DIBUTYL ESTER	84-74-2	80248	1.1	8	46
INK, PRINTING	--	92760	1.1	54	0
IRON OXIDE, BROWN	--	81739	1.1	0	53
SODIUM PHOSPHATE, TRI	7601-54-9	74795	1.1	18	35
CALCIUM ROSINATE	--	M0819	1.1	0	52
C.I. 42000-BASIC GREEN 4	569-64-2	84620	1.0	0	51
C.I. 15575-ACID ORANGE 8	5850-86-2	M2124	1.0	0	51
C.I. 29155-DIRECT ORANGE 29	6420-40-2	M2176	1.0	0	51
SODIUM NITRITE	7632-00-0	69230	1.0	15	36
SILICIC ACID, DISODIUM SALT	1344-09-8	68657	1.0	26	24
GASOLINE-LEADED	--	90880	1.0	49	0
ANTIMONY TRIOXIDE	1309-64-4	M2263	1.0	0	48
ZINC	7440-66-6	77115	1.0	0	47
HYDROXYETHYLCELLULOSE	9004-62-0	82127	1.0	0	47
SILICIC ACID, CALCIUM SALT	10101-39-0	83497	.9	0	46
OZONE	10028-15-6	81651	.9	46	0
HYDROGENATED CASTOR OIL	--	M1137	.9	0	45
NAPHTHENIC OILS	--	M4649	.9	0	45
VARNISH	--	92960	.9	41	4
OIL, OTHER	--	52142	.9	45	0
WATER SOFTENER	--	76613	.9	45	0
OIL, HYDRAULIC	--	92500	.9	45	0
PINE OIL	8002-09-3	80061	.9	0	44
TETRACHLOROETHYLENE	127-18-4	54790	.9	20	24
LAYOUT FLUID	--	42405	.9	44	0
C.I. 12310-PIGMENT RED 2	6041-94-7	M2110	.9	0	43
NAPHTHOLIC DYE	--	M5057	.9	0	43
LEAD OXIDES	--	42685	.9	43	0
TIN OXIDES	--	73253	.9	43	0
C.I. 42510-BASIC VIOLET 14	632-99-5	84118	.9	0	42
C.I. 21010-BASIC BROWN 4	5421-66-9	M1443	.9	0	42
ZINC CHLORIDE	7646-85-7	77150	.9	26	16
POTASSIUM IODIDE	7681-11-0	81683	.9	42	0
COOLANT	--	92175	.9	42	0
PHENOLIC RESINS	--	80183	.8	0	41
TALLOW	61789-97-7	82009	.8	0	41
BLOWN ASPHALT	--	M1559	.8	0	41
DODECYL PHENOL	27193-86-8	T0375	.8	0	41
DODECYL BENZENE SULFONIC ACID	27176-87-0	81668	.8	0	40
DETERGENT	--	92255	.8	40	0
DIPROPYLENE GLYCOL	25265-71-8	81390	.8	0	39
LEAD MOLYBDATE	10190-55-3	83299	.8	0	39

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TITANIUM SULFATE	13693-11-3	83481	.8	0	39
C.I. 30145-DIRECT BROWN 95	16071-86-6	M1412	.8	0	39
C.I. 30295-DIRECT GREEN 6	4335-09-5	M1414	.8	0	39
C.I. 50240-BASIC RED 2	477-73-6	M1441	.8	0	39
C.I. 42555-BASIC VIOLET 3	548-62-9	M1517	.8	0	39
C.I. 21095-PIGMENT YELLOW 14	5468-75-7	M2152	.8	0	39
C.I. 23790-DIRECT BLUE 25	2150-54-1	M2159	.8	0	39
C.I. 29060-DIRECT YELLOW 34	6420-33-3	M2175	.8	0	39
C.I. 42535-BASIC VIOLET 1	8004-87-3	M2194	.8	0	39
C.I. 22250-DIRECT YELLOW 1	6472-91-9	M3100	.8	0	39
C.I. DIRECT BLUE 262	--	M4408	.8	0	39
ALUMINUM	--	M0661	.8	31	8
ETHYLENE, CHLORO-, POLYMER	--	M1774	.8	32	6
PAINT THINNER	--	92650	.8	38	0
EPOXY RESINS	61788-97-4	90740	.8	0	37
GLUCOSE	50-99-7	81836	.8	2	35
CARBON DIOXIDE	124-38-9	17367	.8	6	31
BENZENE	71-43-2	09070	.8	33	4
PASTE	--	53975	.8	37	0
PENTAERYTHRITOL	115-77-5	54185	.7	0	36
MAGNESIUM OXIDE	1309-48-4	80298	.7	3	33
ENAMEL	--	28855	.7	36	0
EMULSIFIER	--	92320	.7	36	0
POTASSIUM OXIDES	--	80065	.7	0	35
IRON OXIDE, RED	--	M1463	.7	0	35
TALL-OIL SOAPS, POTASSIUM	61790-44-1	M1951	.7	0	35
ZINC OXIDE	1314-13-2	77190	.7	0	34
AMMONIUM CHLORIDE	12125-02-9	05270	.7	1	33
WOOD FILLER-PLASTIC WOOD TYPE	--	76623	.7	34	0
PROPENENITRILE, POLYMER WITH 1,3-BUTADIENE AND ETHENYLBENZENE, 2-	9003-56-9	B0079	.7	0	33
PETROLEUM DISTILLATES 149-232C	--	M2800	.7	0	33
ALIPHATIC HYDROCARBONS 149-232C	--	M2806	.7	0	33
SODIUM PENTACHLOROPHENATE	131-52-2	69270	.7	0	32
NAPHTHENIC MINERAL OIL	--	M0920	.7	0	32
DIMETHYLDIALLYL AMMONIUM CHLORIDE HOMOPOLYMER	26062-79-3	M2831	.7	0	32
KAOLIN	1332-58-7	41775	.6	0	31
PROTEINS	--	M0534	.6	0	31
IRON SILICATE	12673-39-1	M1606	.6	0	31
FERRIC ALUMINUM SILICATE	39457-32-4	M2683	.6	0	31
CITRIC ACID	77-92-9	19680	.6	13	18
MAGNESIUM CARBONATE	546-93-0	80144	.6	23	8
AEROSOL CLEANER	--	04065	.6	31	0
NITRIC ACID	7697-37-2	50742	.6	31	0
NITROGEN	7727-37-9	50865	.6	31	0
POLYPROPYLENE	9003-07-0	60275	.6	31	0

Agent Name	CAS #	NIOSH Hazard Number	Percent of Group Exposed	Actual # Exposures to Agent	Trade-Name Ingredient Exposures
SWEEPING COMPOUND	--	80136	.6	31	0
HEXYLENE GLYCOL	107-41-5	37630	.6	0	30
PETROLATUM	8009-03-8	80109	.6	0	30
IRON	7439-89-6	91095	.6	2	28
AMIDES	--	82780	.6	30	0
SODIUM PETROLEUM SULFONATE	8030-73-7	M1542	.6	0	29
SAND	--	67915	.6	29	0
BOILER WATER TREATMENT CHEMICAL	--	80087	.6	29	0
ALGICIDE	--	80755	.6	29	0
PAINT	--	92630	.6	29	0
POTASSIUM HYDROXIDE	1310-58-3	60440	.6	0	28
GUM ARABIC	9000-01-5	80049	.6	0	28
ALKYD RESINS	--	M0598	.6	0	28
DICHLOROBENZENE, ORTHO-	95-50-1	24003	.6	28	0
ERUSTICATOR	--	92860	.6	28	0
ETHANOLAMINE	141-43-5	04980	.5	0	27
IRON OXIDE, YELLOW	1309-37-1	80990	.5	0	27
ROSIN ESTER, FUMARIC	--	M0755	.5	0	27
PENTAERYTHRITOL RESIN ESTER	--	M0997	.5	0	27
MODIFIED STARCH	--	M1923	.5	0	27
C.I. 42595-BASIC BLUE 7	2390-60-5	M2198	.5	0	27
C.I. 74160-PIGMENT GREEN 36	14302-13-7	M2254	.5	0	27
C.I. 77400-PIGMENT METAL 2	7440-50-8	M2276	.5	0	27
C.I. 77491-PIGMENT BROWN 7	1345-27-3	M2279	.5	0	27
SULFONAMIDE-FORMALDEHYDE RESIN	--	M2424	.5	0	27
ALIPHATIC HYDROCARBONS 15.5-149C	--	M2805	.5	0	27
C.I. 12360-PIGMENT RED 31	6448-96-0	M3063	.5	0	27
C.I. 77847-PIGMENT WHITE 8	1314-96-1	M3168	.5	0	27
CHLORINE	7782-50-5	18040	.5	23	4
LIGNIN	9005-53-2	80293	.5	27	0
COTTON	--	94040	.5	27	0
LITHIUM HYDROXIDE	1310-65-2	81879	.5	0	26
PHENYL-ALPHA-NAPHTHYLAMINE, N-	90-30-2	82955	.5	0	26
SODIUM SOAP	8046-71-7	83329	.5	0	26
LEAD NAPHTHENATE	61790-14-5	83453	.5	0	26
POLYETHOXYLATED ALCOHOL	--	M2317	.5	0	26
BRASS, OXIDES OF	--	13025	.5	26	0
SODIUM THIOSULFATE	7772-98-7	80153	.5	26	0
AMMONIUM PHOSPHATE, DIBASIC	7783-28-0	M0155	.5	26	0
HEXACHLOROETHANE	67-72-1	17385	.5	0	25
FUMARIC ACID	110-17-8	33940	.5	0	25
ETHYLENEDIAMINETETRAACETIC ACID TETRASODIUM SALT	64-02-8	80223	.5	0	25
CALCIUM ALUMINATE	11104-48-6	81752	.5	0	25
DIMETHYLAMINOETHANOL	108-01-0	83678	.5	0	25
NABAM	142-59-6	84470	.5	0	25

Agent Name	CAS #	NIOSH Hazard Number	Percent of Group Exposed	Actual # Exposures to Agent	Trade-Name Ingredient Exposures
NAPHTHA	8030-30-6	M0628	.5	0	25
TALL OIL ALKYD RESIN	--	M0698	.5	0	25
CALCIUM FERRITE	11113-52-3	M2864	.5	0	25
SODIUM TRIPOLYPHOSPHATE	7758-29-4	80076	.5	8	17
ACETIC ACID ETHENYL ESTER, POLYMER WITH ETHENE	24937-78-8	M0970	.5	16	9
POTASSIUM DICHROMATE(VI)	7778-50-9	80064	.5	25	0
AMMONIUM PHOSPHATES	--	80259	.5	25	0
GLUE-EPOXY BASE	--	90920	.5	25	0
ISOBUTANE	75-28-5	81851	.5	0	24
SODIUM ABIETATE	14351-66-7	M0657	.5	0	24
C.I. DIRECT YELLOW 127	12222-68-3	M2040	.5	0	24
C.I. 17755-ACID RED 137	6222-63-5	M2141	.5	0	24
SODIUM LINEAR ALKYLBENZENE SULFONATE	--	M2370	.5	0	24
ETHYLENE, TRICHLORO-	79-01-6	73790	.5	24	0
RESINS, NATURAL	--	92820	.5	24	0
BLACK LIQUOR	--	A1167	.5	24	0
PRODUCTS OF COMBUSTION-LIGNIN	9005-53-2	A1568	.5	24	0
STYRENE	100-42-5	70130	.5	0	23
POTASSIUM PHOSPHATE, TRIBASIC	7778-53-2	83451	.5	0	23
C.I. 50415:1-SOLVENT BLACK 7	8005-02-5	M1407	.5	0	23
CALCIUM HYPOCHLORITE	7778-54-3	15746	.5	3	20
MICA	12001-26-2	48535	.5	11	12
WATERPROOFING AGENT	--	80029	.5	23	0
SODIUM LIGNOSULFONATE	8061-51-6	81991	.4	0	22
SODIUM XYLENESULFONATE	1300-72-7	82789	.4	0	22
OCTADECYLAMINE	124-30-1	83376	.4	0	22
TALLOW OIL	--	M1859	.4	0	22
SLATE	--	68763	.4	22	0
DRAIN OPENER	--	80019	.4	22	0
COCONUT DIETHANOLAMIDE	8040-31-1	M0779	.4	0	21
AMMONIUM STEARATE	1002-89-7	82889	.4	9	12
STEEL, STAINLESS, OXIDES OF	--	69717	.4	21	0
LITHIUM CHLORIDE	7447-41-8	84472	.4	21	0
ACRYLAMIDE	79-06-1	03540	.4	0	20
RONNEL	299-84-3	67525	.4	0	20
SODIUM TRICHLOROPHENATE	1320-79-2	80075	.4	0	20
SODIUM MYRISTYL POLYETHER SULFATE	--	M0853	.4	0	20
OXIRANE, METHYL-, POLYMER WITH OXIRANE	9003-11-6	M3966	.4	0	20
BIS(DIMETHYLAMINOMETHYL) CYCLOHEXANONE, 2,6-	--	M5200	.4	0	20
DECOMPOSITION PRODUCTS-OIL, GENERAL	--	22737	.4	20	0
BIOCIDE	--	92080	.4	20	0
GLYCOLIC ACID	79-14-1	35260	.4	0	19
TEREPHTHALIC ACID	100-21-0	82013	.4	0	19
CALCIUM LIGNOSULFONATE	8061-52-7	83024	.4	0	19
POLYBUTENE	9003-29-6	M0521	.4	0	19

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CHLORINATED PARAFFIN WAX	--	M0601	.4	0	19
POTASSIUM ROSIN SOAP	--	M1952	.4	0	19
CALCIUM SOAP OF ACETIC ACID	--	M2560	.4	0	19
POLYETHYLENE GLYCOL DIOLEATE	9005-07-6	M3961	.4	0	19
TALLOW, SODIUM SALT	8052-48-0	M4897	.4	0	19
SODIUM FORMATE	141-53-7	81986	.4	4	15
DECOMPOSITION PRODUCTS-GENERAL	--	22734	.4	19	0
POTASSIUM PERMANGANATE	7722-64-7	60490	.4	19	0
GLASS CLEANER	--	76622	.4	19	0
WATER TREATMENT COMPOUND	--	80009	.4	19	0
INORGANIC HYPOCHLORITES	--	91080	.4	19	0
CATALYST	--	92135	.4	19	0
DIOXANE, 1,4-	123-91-1	25145	.4	0	18
NAPHTHALENE	91-20-3	49600	.4	0	18
SULFUR	7704-34-9	70845	.4	0	18
VANILLIN	121-33-5	M0609	.4	0	18
COBALT DRIER	--	M0642	.4	0	18
C.I. 50420-ACID BLACK 2	8005-03-6	M1423	.4	0	18
PROPENOIC ACID, 2-METHYL-, HOMOPOLYMER, SODIUM SALT, 2-	54193-36-1	M3905	.4	2	16
AIR FRESHENER	--	80016	.4	18	0
PIPE JOINT SEALER	--	80033	.4	18	0
ASPHALT VOLATILES	--	80636	.4	18	0
HYDROCARBONS	--	91070	.3	0	17
STODDARD SOLVENT	8052-41-3	69855	.3	2	15
SODIUM SULFIDE	1313-82-2	69460	.3	13	4
DECOMPOSITION PRODUCTS-AMMONIUM CHLORIDE	12125-02-9	A1525	.3	17	0
BUTYL STEARATE	123-95-5	80587	.3	0	16
PHOSPHORIC ACID, TRITOLYL ESTER	1330-78-5	82880	.3	0	16
DI(TERT-NONYL) POLYSULFIDE	--	83925	.3	0	16
PHENOLIC MODIFIED PENTA ESTER OF RESIN	--	M0641	.3	0	16
SORBITAN FATTY ACID ESTERS	--	M0707	.3	0	16
SULFURIZED LARD OIL	61790-49-6	M0799	.3	0	16
DIMETHYL BENZYL AMMONIUM CHLORIDE	1875-92-9	M0983	.3	0	16
MIDCONTINENT NEUTRAL BASE OIL, SOLVENT-TREATED	--	M1209	.3	0	16
CALCIUM SOAP OF HYDROFOL ACID	--	M2543	.3	0	16
ACRYLIC EMULSION RESIN	--	M2569	.3	0	16
SULFITE LIQUOR	--	M3284	.3	0	16
HYDROGENATED TALLOW FATTY ACID	--	M3360	.3	0	16
SODIUM HEXAMETAPHOSPHATE	10124-56-8	69055	.3	12	4
AMMONIUM SULFAMATE	7773-06-0	06190	.3	16	0
SALTS	--	67855	.3	16	0
INDICATOR	--	80011	.3	16	0
ANTIMONY DIOXIDE	--	82890	.3	16	0
FREONS	--	90860	.3	16	0
INSECTICIDE	--	92545	.3	16	0

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THINNER	--	92940	.3	16	0
INORGANIC CHROMATES	--	M0063	.3	16	0
ALKYLDIMETHYLBENZYLAMMONIUM CHLORIDE	8039-63-2	04280	.3	0	15
METHYL SALICYLATE	119-36-8	48320	.3	0	15
GLUCONIC ACID	526-95-4	80287	.3	0	15
ALGIN	9005-38-3	80439	.3	0	15
METHYLOLACRYLAMIDE	924-42-5	81901	.3	0	15
NITROTOLUENE, ORTHO-	88-72-2	81910	.3	0	15
SODIUM LAURYL SULFATE	151-21-3	81990	.3	0	15
DIRECT DYES	--	M1424	.3	0	15
LEAD OLEATE	1120-46-3	M1691	.3	0	15
SEAL OIL	--	M1924	.3	0	15
C.I. 77520-PIGMENT GREEN 15	7758-97-6	M2282	.3	0	15
C.I. BASIC YELLOW 13	12217-50-4	M3077	.3	0	15
LECITHIN, HYDROXYLATED	8029-76-3	M3305	.3	0	15
C.I. BASIC GREEN 3	61901-57-3	M3336	.3	0	15
DIETHYLENETRIAMINE-MODIFIED UREA-FORMALDEHYDE RESIN	--	M3431	.3	0	15
BORATED DEXTRIN ADHESIVE SOLIDS	--	M4166	.3	0	15
DIMETHYL DITALLOW AMMONIUM BENTONITE	--	M4247	.3	0	15
FUMARIC ACID-ROSIN ADDUCT	--	M4419	.3	0	15
IRON OXIDES	--	40297	.3	15	0
INK SOLVENT	--	92530	.3	15	0
OILS, ESSENTIAL	--	80096	.3	0	14
CARNAUBA WAX	8015-86-9	80365	.3	0	14
CANDELILLA WAX	8006-44-8	82931	.3	0	14
POTASSIUM COCONUT OIL SOAP	--	M1948	.3	0	14
SOYBEAN PROTEIN	--	80495	.3	9	5
CADMIUM OXIDES	--	15630	.3	14	0
PRODUCTS OF COMBUSTION-FUEL OIL	--	60712	.3	14	0
TURPENTINE	8006-64-2	74990	.3	14	0
RUBBERS, NATURAL	--	67537	.3	0	13
AROMATIC HYDROCARBONS	--	80027	.3	0	13
OLEYL AMINE	112-90-3	83831	.3	0	13
PHTHALIC ALKYD	--	M0874	.3	0	13
SULFATED LARD OIL	--	M1018	.3	0	13
AROMATIC HYDROCARBONS 149-232C	--	M2808	.3	0	13
CELLULOSE, CARBOXYMETHYL ETHER	9000-11-7	80349	.3	3	10
STEEL, STAINLESS	12597-68-1	69715	.3	13	0
BUFFER	--	80116	.3	13	0
POLYPROPYLENE GLYCOL ETHER	--	80187	.2	0	12
SODIUM SILICOFLUORIDE	16893-85-9	84233	.2	0	12
COBALT OXIDES	--	84328	.2	0	12
PHOSPHORIC ACID ESTERS	--	M0378	.2	0	12
AZO COMPOUNDS	--	M0487	.2	0	12
FISH FATTY ACID	--	M1172	.2	0	12

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C.I. 29190-DIRECT RED 26	--	M1420	.2	0	12
C.I. 40002-DIRECT ORANGE 15	1325-35-5	M1421	.2	0	12
OILS	--	M1460	.2	0	12
TALL OIL SOAPS	--	M1858	.2	0	12
C.I. 16265-ACID RED 187	6717-29-9	M2139	.2	0	12
C.I. 19555-DIRECT YELLOW 28	8005-72-9	M2150	.2	0	12
C.I. 24890-DIRECT YELLOW 4	3051-11-4	M2163	.2	0	12
C.I. 40006-DIRECT YELLOW 6:1	1325-42-4	M2182	.2	0	12
SULFATED SPERM OIL	--	M2304	.2	0	12
C.I. 29156-DIRECT ORANGE 102	6420-41-3	M3154	.2	0	12
ANISIDINE DERIVATIVES	--	M4153	.2	0	12
SULFONATED NAPHTHALENE FORMALDEHYDE CONDENSATE, SODIUM SALT	--	M4910	.2	0	12
STILBENE DYE	--	M5026	.2	0	12
C.I. 53186-SOLUBILISED SULFUR BLACK 1	--	T0365	.2	0	12
POTASSIUM FERROCYANIDE	13943-58-3	80199	.2	9	3
HELIUM	7440-59-7	35925	.2	12	0
IODINE	7553-56-2	40030	.2	12	0
OIL, FUEL NO. 2	--	52132	.2	12	0
SILVER OXIDES	--	68748	.2	12	0
SULFUR DIOXIDE	7446-09-5	70860	.2	12	0
CHROMIC POTASSIUM SULFATE, DODECAHYDRATE	7788-99-0	82893	.2	12	0
MANGANESE HYDROXIDE	12626-88-9	82900	.2	12	0
INORGANIC THIOSULFATES	--	M0214	.2	12	0
PHTHALIC ANHYDRIDE	85-44-9	59230	.2	0	11
MONTMORILLONITE	1318-93-0	80570	.2	0	11
CETYL ALCOHOL	36653-82-4	80588	.2	0	11
AMMONIUM BROMIDE	12124-97-9	81702	.2	0	11
BUTYLENE OXIDE	26249-20-7	82164	.2	0	11
DIPROPYLENE GLYCOL MONOMETHYL ETHER	34590-94-8	84537	.2	0	11
LINSEED ALKYD	--	M0679	.2	0	11
POLY(OXY(METHYLSILYLENE))	9004-73-3	M1657	.2	0	11
OXOALCOHOLS	--	M1674	.2	0	11
POLY(OXY-1,2-ETHANEDIYL), ALPHA-TRIDECYL-OMEGA-HYDROXY-	24938-91-8	M2586	.2	0	11
ETHOXYLATED CASTOR OIL	--	M2673	.2	0	11
TRIPROPYLENE GLYCOL MONOMETHYL ETHER	25498-49-1	M4318	.2	0	11
ETHANONE, 2-BROMO-1-(4-HYDROXYPHENYL)-	--	T0809	.2	0	11
QUARTZ	14808-60-7	66495	.2	4	7
TRIBUTYL PHOSPHATE	126-73-8	73730	.2	7	4
ANTIMONY OXIDES	--	07328	.2	11	0
DIETHYLENE GLYCOL	111-46-6	25544	.2	11	0
FOUNTAIN SOLUTION	--	33895	.2	11	0
NATURAL GAS	8006-14-2	50195	.2	11	0
PRODUCTS OF COMBUSTION-DIESEL	--	80017	.2	11	0
MANGANESE SULFATE	7785-87-7	81680	.2	11	0
SODIUM HYDROSULFITE	7775-14-6	84077	.2	11	0

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INORGANIC TITANIUM COMPOUNDS	--	M0106	.2	11	0
LINOLEIC ACID, 9,12-	60-33-3	43040	.2	0	10
SORBITOL	50-70-4	80530	.2	0	10
POTASSIUM BIFLUORIDE	7789-29-9	81945	.2	0	10
RICINOLEIC ACID	141-22-0	83687	.2	0	10
TRI-N-BUTYLAMINE	102-82-9	83937	.2	0	10
CUMENE HYDROPEROXIDE	80-15-9	A1346	.2	0	10
POTASSIUM PENTABORATE	11128-29-3	M0768	.2	0	10
TETRAETHYLENE GLYCOL 2-(2-ETHYL HEXOATE)	18268-70-7	M1519	.2	0	10
POLYETHYLENEIMINE	26913-06-4	M2379	.2	0	10
POLYETHYLENEGLYCOL DIMETHACRYLATE	25721-76-0	M2476	.2	0	10
BISPHENOL A FUMARATE RESIN	26810-78-6	M2686	.2	0	10
STYRENE POLYMER	--	M3828	.2	0	10
POTASSIUM CHROMATE	7789-00-6	60370	.2	9	1
ALUMINUM OXIDES	--	04608	.2	10	0
FLOOR WAX	--	33305	.2	10	0
PIGMENTS	--	59465	.2	10	0
OFFSET PRINTING COMPOUND	--	80176	.2	10	0
GASOLINE-UNLEADED	--	90885	.2	10	0
POLISH	--	92720	.2	10	0
PRIMER	--	92750	.2	10	0
ACRYLIC ACID	79-10-7	03570	.2	0	9
ACRYLONITRILE	107-13-1	03800	.2	0	9
ETHYL ACRYLATE	140-88-5	31490	.2	0	9
LEAD	7439-92-1	42490	.2	0	9
METHYL ACETATE	79-20-9	46410	.2	0	9
NONYLPHENOXYPOLYETHOXYETHANOL-IODINE COMPLEX	11096-42-7	80310	.2	0	9
ALUMINUM SILICATE	1327-36-2	80625	.2	0	9
DIPROPYLENE GLYCOL DIBENZOATE	27138-31-4	80828	.2	0	9
PYROGALLIC ACID	87-66-1	81963	.2	0	9
ETHYLHEXYL ACRYLATE, 2-	103-11-7	82082	.2	0	9
POLYETHOXY POLYPROPOXY POLYETHOXY-IODINE COMPLEX	--	82105	.2	0	9
LATEX, ACRYLIC	--	83425	.2	0	9
CROTONIC ACID	3724-65-0	84287	.2	0	9
SODIUM SESQUICARBONATE	533-96-0	M0653	.2	0	9
PALE OIL	--	M0780	.2	0	9
BOILED LINSEED OIL	--	M0894	.2	0	9
METHYL TALLOWATE	--	M1351	.2	0	9
LATEX, RUBBER	--	M1626	.2	0	9
CHLOROETHYL)TRIMETHYLAMMONIUM CHLORIDE, (2-	999-81-5	M4610	.2	0	9
POLYVINYL TRIMETHYLBENZYLAMMONIUM CHLORIDE	9017-80-5	M4754	.2	0	9
BORIC ACID	10043-35-3	12960	.2	9	0
OXALIC ACID	144-62-7	52480	.2	9	0
RUBBER, OTHER	--	67539	.2	9	0
ROOFING CEMENT	--	80067	.2	9	0

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PRINTING CHEMICAL	--	80097	.2	9	0
DECOMPOSITION PRODUCTS-POLYMER, POLYPROPYLENE	9003-07-0	80610	.2	9	0
POTASSIUM FERRICYANIDE	13746-66-2	81080	.2	9	0
THIOUREA	62-56-6	82272	.2	9	0
GLYOXAL	107-22-2	84477	.2	9	0
PHOTOGRAPHIC CHEMICAL	--	92670	.2	9	0
PHENOL	108-95-2	55460	.2	0	8
POTASSIUM CARBONATE	584-08-7	60350	.2	0	8
PROPYLENE OXIDE	75-56-9	63550	.2	0	8
SODIUM CITRATE	68-04-2	68905	.2	0	8
POLYETHERS	--	80010	.2	0	8
SOYA ALKYD RESINS	--	80121	.2	0	8
POTASSIUM NITRATE	7757-79-1	80350	.2	0	8
BONE MEAL	--	80371	.2	0	8
SORBITAN MONOSTEARATE	1338-41-6	81999	.2	0	8
SOYA FATTY ACID	--	M1173	.2	0	8
MEDIUM OIL ALKYD RESIN	--	M1202	.2	0	8
POLY(OXY-1,2-ETHANEDIYL), ALPHA-(1-OXO-9-OCTADECENYL)-OMEGA-HYDROXY-,(Z)-	9004-96-0	M1221	.2	0	8
CADMIUM OXIDE	1306-19-0	M1554	.2	0	8
MEDIUM OIL SOYA ALKYD RESIN	--	M1844	.2	0	8
CHROMIC OXIDE	1308-38-9	M2273	.2	0	8
METHYL TRIMETHOXY SILANE	--	M3881	.2	0	8
AMINOETHYLETHANOLAMINE-FATTY ACIDS CONDENSATE	--	M4815	.2	0	8
POLYMETHYLSILOXANE	--	T0436	.2	0	8
BARIUM CHLORIDE	10361-37-2	08650	.2	8	0
COPPER OXIDES	--	20170	.2	8	0
FIBERGLASS	--	33245	.2	8	0
HYDROGEN PEROXIDE	7722-84-1	38605	.2	8	0
METHANE	74-82-8	45655	.2	8	0
SODIUM CHLORATE	7775-09-9	68870	.2	8	0
DEOXIDIZER	--	80006	.2	8	0
SOIL	--	80032	.2	8	0
ROLL CLEANER, PRINTING	--	80066	.2	8	0
OILS, FUEL (GENERAL)	--	80123	.2	8	0
PUMICE	1332-09-8	92780	.2	8	0
METAL ELEMENTS	--	M0031	.2	8	0
EPOXIDES	--	M0309	.2	8	0
IONIZING RADIATION	--	P0412	.2	8	0
ETHYLBENZENE	100-41-4	31830	.1	0	7
IRON OXIDE (FE3O4)	1317-61-9	70131	.1	0	7
SPERMACETI	8002-23-1	80531	.1	0	7
MAGNESIUM ALUMINUM SILICATE	1327-43-1	83046	.1	0	7
CRESOL, 2,6-DI-TERT-BUTYL-, P-	128-37-0	83383	.1	0	7
BENZOTHIAZOLETHIOL, 2-	149-30-4	84030	.1	0	7
C.I. 42750:1-PIGMENT BLUE 19	58569-23-6	M0648	.1	0	7

Agent Name	CAS #	NIOSH Hazard Number	Percent of Group Exposed	Actual # Exposures to Agent	Trade-Name Ingredient Exposures
SOYBEAN OIL	8001-22-7	M0682	.1	0	7
TUNG OIL	8001-20-5	M0708	.1	0	7
LINSEED ISOPHTHALIC ALKYD RESIN	--	M0918	.1	0	7
SAFFLOWER OIL ALKYD	--	M0925	.1	0	7
C.I. 77811-PIGMENT WHITE 27	7631-86-9	M0930	.1	0	7
SERICITE	12174-53-7	M0959	.1	0	7
ALIPHATIC PETROLEUM DISTILLATES	--	M1014	.1	0	7
COBALT OCTANOATE	6700-85-2	M2523	.1	0	7
CALCIUM MANGANESE OCTOATE	--	M2578	.1	0	7
PETROLEUM DISTILLATES 232-343C	--	M2801	.1	0	7
C6-C9 AROMATIC HYDROCARBONS	--	M2824	.1	0	7
DIMETHYL DIOCTADECYL AMMONIUM BENTONITE	--	M4244	.1	0	7
C.I. 27885-DIRECT VIOLET 9	6227-14-1	M4517	.1	0	7
PHENOLPHTHALEIN	77-09-8	56240	.1	7	0
ETCHING COMPOUND	--	80026	.1	7	0
BROMCRESOL GREEN	76-60-8	81325	.1	7	0
BROMOTHYMOL BLUE	--	81736	.1	7	0
CHLORPHENOL RED	4430-20-0	81772	.1	7	0
CRESOL PURPLE	2303-01-7	81783	.1	7	0
SODIUM PHOSPHATE, DIBASIC	7558-79-4	83514	.1	7	0
GLUE-RUBBER BASE	--	90930	.1	7	0
PHOTOGRAPHIC DEVELOPER	--	92675	.1	7	0
RETARDER	--	92830	.1	7	0
DECOMPOSITION PRODUCTS-CELLULOSE	9004-34-6	A1410	.1	7	0
UREA, POLYMER WITH FORMALDEHYDE	9011-05-6	M3046	.1	7	0
BENZOIC ACID	65-85-0	10210	.1	0	6
CHROMIC ACID	7738-94-5	19360	.1	0	6
MAGNESIUM CHLORIDE	7786-30-3	43360	.1	0	6
PETROLEUM SULFONATE	--	80059	.1	0	6
DIGLYCOL STEARATE	106-11-6	80402	.1	0	6
BUTOXYETHYL ACETATE, 2-	112-07-2	81350	.1	0	6
POTASSIUM PERSULFATE	7727-21-1	81953	.1	0	6
BUTYLATED HYDROXYTOLUENES	--	82226	.1	0	6
POLYMETHACRYLATE RESIN	--	82797	.1	0	6
TRIETHANOLAMINE OLEATE	2717-15-9	83030	.1	0	6
SULFONATED OIL	--	83872	.1	0	6
CYCLOALKANES	--	90690	.1	0	6
PHOSPHORUS-CONTAINING ORGANIC COMPOUNDS	--	M0482	.1	0	6
MARBLE	--	M0841	.1	0	6
CELLULOSE ETHYL HYDROXYETHYL ETHER	9004-58-4	M0858	.1	0	6
ISOPHTHALIC ALKYD RESIN	--	M0906	.1	0	6
MANGANESE DRIER	--	M0921	.1	0	6
COCONUT ACID	--	M1144	.1	0	6
STYRENE-BUTADIENE RESIN	--	M2365	.1	0	6
NAPHTHALENE SULFONATE	25155-19-5	M3861	.1	0	6

Agent Name	CAS #	NIOSH Hazard Number	Percent of Group Exposed	Actual # Exposures to Agent	Trade-Name Ingredient Exposures
DIBUTYLTIN DI-2-ETHYLHEXOATE	2781-10-4	M3953	.1	0	6
BUTYLATED UREA-FORMALDEHYDE RESIN	--	M4296	.1	0	6
HEXAMETHYLOLMELAMINE	--	M5166	.1	0	6
DECOMPOSITION PRODUCTS-OIL, LUBE	--	22739	.1	6	0
MAGNESIUM	7439-95-4	43320	.1	6	0
SODIUM FLUORIDE	7681-49-4	84425	.1	6	0
SODIUM DODECYLBENZENESULFONATE	25155-30-0	69000	.1	0	5
TRIMETHYLAMINE	75-50-3	82039	.1	0	5
SODIUM DICHROMATE(VI)	10588-01-9	M0073	.1	0	5
AMMONIUM PHOSPHATE, MONOBASIC	7722-76-1	M0156	.1	0	5
MINERAL GREASE	--	M0929	.1	0	5
ROCK WOOL	--	M1268	.1	0	5
BINDER ABRASIVE WHEEL	--	92070	.1	5	0
POLYVINYL RESINS	--	M0526	.1	5	0
CYCLOHEXANOL	108-93-0	21560	.1	0	4
DIETHYLENETRIAMINE	111-40-0	25210	.1	0	4
THIADIAZINE-2-THIONE, TETRAHYDRO-3,5-DIMETHYL-, 2H-1,3,5-	533-74-4	26940	.1	0	4
HYDRAZINE	302-01-2	38110	.1	0	4
CALCIUM PHOSPHATES	--	80246	.1	0	4
ETHYLENEDIAMINETETRAACETIC ACID	60-00-4	80283	.1	0	4
MAGNESIUM SILICATE	1343-88-0	80299	.1	0	4
STEARYL ALCOHOL	112-92-5	80705	.1	0	4
SODIUM PHOSPHATE, TRI, CHLORINATED	--	80860	.1	0	4
AZOBISFORMAMIDE	123-77-3	81713	.1	0	4
CRESOL RED	1733-12-6	81784	.1	0	4
LITHIUM GREASE	--	81878	.1	0	4
C.I. 77955-PIGMENT YELLOW 36	37300-23-5	82134	.1	0	4
ETHYLENEDIAMINETETRAACETIC ACID, SODIUM SALT	7379-28-4	82806	.1	0	4
GLYCERYL MONOSTEARATE	31566-31-1	82942	.1	0	4
SORBITOL MONOSTEARATE	26836-47-5	83357	.1	0	4
OCTANOIC ACID, ZINC SALT	557-09-5	83596	.1	0	4
DIBUTYLTIN DIACETATE	1067-33-0	83741	.1	0	4
SODIUM BORATES	--	M0079	.1	0	4
LEAD MONOXIDE	1317-36-8	M0125	.1	0	4
RUBBERS	--	M0529	.1	0	4
C.I. 21090-PIGMENT YELLOW 12	6358-85-6	M0644	.1	0	4
OXIDIZED PETROLATUM	--	M0662	.1	0	4
FORMALDEHYDE-TOLUENE SULFONAMIDE RESIN	9008-60-0	M0791	.1	0	4
LEAD CHROMATE, LEAD MOLYBDATE, LEAD SULFATE COMPLEX	--	M0814	.1	0	4
LONG OIL-FISH OIL ALKYD RESIN	--	M1703	.1	0	4
SODIUM SOAP OF WOOL FAT	--	M1789	.1	0	4
MEDIUM LINSEED OIL ALKYD RESIN	--	M1846	.1	0	4
C.I. DIRECT BLUE 80	12222-00-3	M2038	.1	0	4
C.I. 12335-PIGMENT RED 8	6410-30-6	M2112	.1	0	4
C.I. 21120-PIGMENT RED 38	6358-87-8	M2154	.1	0	4

Agent Name	CAS #	NIOSH Hazard Number	Percent of Group Exposed	Actual # Exposures to Agent	Trade-Name Ingredient Exposures
C.I. 45170:2-PIGMENT VIOLET 1	1326-03-0	M2211	.1	0	4
C.I. 61100-DISPERSE VIOLET 1	128-95-0	M2230	.1	0	4
ALKYLPHENOXYPOLY(ETHYLENEOXY)ETHANOL	--	M2394	.1	0	4
ACRYLAMIDE-SODIUM ACRYLATE COPOLYMER	25085-02-3	M3175	.1	0	4
ACRYLIC COPOLYMERS	--	M3807	.1	0	4
C.I. 15585:1-PIGMENT RED 53:1	5160-02-1	M3849	.1	0	4
ETHANOL, 2-ETHOXY-, ACETATE	111-15-9	M4063	.1	0	4
POLYETHYLENE GLYCOL DECANOATE	--	M4369	.1	0	4
METHYLENEBIS(NAPHTHALENE SULFONATE)	--	M4960	.1	0	4
MONOAZO DYE	--	M5022	.1	0	4
INK OIL 535	8002-05-9	T0284	.1	0	4
FORMALDEHYDE-AMINOTRIAZINE-SULFONAMIDE RESIN	--	T0469	.1	0	4
CARBON MONOXIDE	630-08-0	17460	.1	4	0
INDUSTRIAL WASTE	--	39995	.1	4	0
INORGANIC IODIDES	--	40025	.1	4	0
NITROGEN OXIDES	--	50875	.1	4	0
TONER	--	73495	.1	4	0
BOWL CLEANER	--	80086	.1	4	0
PRODUCTS ACID CORE SOLDER	--	80128	.1	4	0
SILVER NITRATE	7761-88-8	80142	.1	4	0
GALLIC ACID	149-91-7	84280	.1	4	0
ANTIFREEZE	--	92040	.1	4	0
DUPLICATOR FLUID	--	92280	.1	4	0
INORGANIC NITRATES	--	M0180	.1	4	0
ACETIC ACID ESTERS	--	M0367	.1	4	0
PROPYLPARABEN	94-13-3	80496	.1	0	3
TRIETHYLENETETRAMINE	112-24-3	82037	.1	0	3
GUAR GUM	9000-30-0	83111	.1	0	3
LITHIUM STEARATE	4485-12-5	83508	.1	0	3
OILS, PETROLEUM-DERIVED	--	M0542	.1	0	3
GREEN SOAP	8026-70-8	M0620	.1	0	3
ANTIMONY DITHIOCARBAMATE	64216-11-1	M1109	.1	0	3
C.I. 44045-BASIC BLUE 26	2580-56-5	M2206	.1	0	3
C.I. 50415-SOLVENT BLACK 5	11099-03-9	M2221	.1	0	3
ROSIN MALEIC ADDUCT	--	M3840	.1	0	3
C.I. 14710-ACID RED 4	5858-39-9	M4104	.1	0	3
C.I. DIRECT YELLOW 117	12217-74-2	M4255	.1	0	3
HYDROGENATED PETROLEUM BASE OIL	--	M4262	.1	0	3
C.I. 30110-DIRECT BROWN 1:2	--	M4499	.1	0	3
SULFONATED POLYSTYRENE	--	M4801	.1	0	3
CARBON TETRACHLORIDE	56-23-5	17490	.1	3	0
CHLOROFORM	67-66-3	18500	.1	3	0
COAL	--	19767	.1	3	0
OIL, QUENCHING	--	52144	.1	3	0
SEWAGE, SANITARY	--	68508	.1	3	0

Agent Name	CAS #	NIOSH Hazard Number	Percent of Group Exposed	Actual # Exposures to Agent	Trade-Name Ingredient Exposures
WHITENING AGENT	--	80003	.1	3	0
INORGANIC SULFUR ANION COMPOUNDS	--	82008	.1	3	0
MELAMINE FORMALDEHYDE	13236-84-5	82796	.1	3	0
ALUMINUM SULFIDE	1302-81-4	83263	.1	3	0
ACIDS	--	90050	.1	3	0
BRAKE FLUID	--	92120	.1	3	0
COPY MACHINE FLUID	--	92180	.1	3	0
RUBBER, BUTADIENE-STYRENE	61789-96-6	13455	.0	0	2
CAMPHOR	76-22-2	15800	.0	0	2
CINEOLE	470-82-6	19540	.0	0	2
DICHLOROTETRAFLUOROETHANE	1320-37-2	24425	.0	0	2
ISOPRENE	78-79-5	40940	.0	0	2
NICKEL NITRATE	13138-45-9	50480	.0	0	2
PHTHALIC ACID, BENZYL BUTYL ESTER	85-68-7	80249	.0	0	2
TERPINEOL, ALPHA-	98-55-5	80268	.0	0	2
MANGANESE LINOLEATE	646-00-4	80300	.0	0	2
TERPINEOL, BETA-	138-87-4	83076	.0	0	2
TRIMETHYL-1,3-PENTANEDIOL MONOISOBUTYRATE, 2,2,4-	25265-77-4	83626	.0	0	2
TRIBUTOXYETHYL PHOSPHATE	78-51-3	83871	.0	0	2
TERPINOLENE	586-62-9	84513	.0	0	2
GYPSUM	13397-24-5	90980	.0	0	2
TALLOW SOAP	--	M0652	.0	0	2
ALUMINUM PASTE	--	M0692	.0	0	2
LIMONENE	138-86-3	M1342	.0	0	2
ANETHOLE	104-46-1	M1381	.0	0	2
SYNTHETIC WAX	--	M1812	.0	0	2
WAX EMULSION	--	M1818	.0	0	2
C.I. 12085-PIGMENT RED 4	2814-77-9	M2108	.0	0	2
SODIUM NAPHTHALENE BETA-SULFONATE- FORMALDEHYDE CONDENSATE	29321-75-3	M2454	.0	0	2
ETHOXYBUTANOL, 2-	--	M2911	.0	0	2
COBALT LINOLEATE	14666-96-7	M2950	.0	0	2
FENCHYL ALCOHOL	--	M3922	.0	0	2
ISOPROPYLTOLUENE	25155-15-1	M3923	.0	0	2
ALLYLANISOLE, PARA-	--	M3924	.0	0	2
TERPENE ALCOHOLS	--	M4411	.0	0	2
SODIUM SALT OF CARBOXYLIC ACIDS	--	T0393	.0	0	2
DIISOBUTYLENE-MALEIC ANHYDRIDE POLYMER SODIUM SALT	37199-81-8	T1168	.0	0	2
ARGON	7440-37-1	07485	.0	2	0
FERRIC CHLORIDE	7705-08-0	33160	.0	2	0
TOLUIDINE, ORTHO-	95-53-4	73470	.0	2	0
METHANE, CHLORODIFLUORO-	75-45-6	80048	.0	2	0
DEVELOPER	--	80095	.0	2	0
GLUTAMIC ACID	56-86-0	80420	.0	2	0
SODIUM PHTHALATE	15968-01-1	82855	.0	2	0
BRASS, CU, ZN, PB GROUP	12597-71-6	90400	.0	2	0

Agent Name	CAS #	NIOSH Hazard Number	Percent of Group Exposed	Actual # Exposures to Agent	Trade-Name Ingredient Exposures
MORPHOLINE	110-91-8	48910	.0	0	1
SODIUM CHROMATE(VI)	7775-11-3	68900	.0	0	1
ZINC SULFATE	7733-02-0	77220	.0	0	1
SODIUM BICARBONATE (1:1)	144-55-8	80069	.0	0	1
SODIUM BISULFATE	7681-38-1	80071	.0	0	1
PIPERONYL BUTOXIDE	51-03-6	80218	.0	0	1
BENZOTRIAZOLE, 1,2,3-	95-14-7	82113	.0	0	1
SODIUM SULFOSUCCINATE	20526-58-3	82124	.0	0	1
PYRETHRINS	--	82156	.0	0	1
ORGANIC SULFATES	--	82250	.0	0	1
ISOOCTYL(2,4-DICHLOROPHENOXY) ACETATE	25168-26-7	82947	.0	0	1
CUPROUS CHLORIDE	7758-89-6	83435	.0	0	1
ALKYL SULFONATE, LINEAR	--	83455	.0	0	1
CYCLOHEXYLAMINE	108-91-8	84526	.0	0	1
NAPHTHA-HIGH FLASH OR HEAVY	--	M0629	.0	0	1
MONURON TRICHLOROACETATE	140-41-0	M1843	.0	0	1
C5-C9 HYDROCARBONS	--	M2813	.0	0	1
C5-C9 ALIPHATIC HYDROCARBONS	--	M2819	.0	0	1
SODIUM PHOSPHONATE	13933-52-3	M3399	.0	0	1
DISODIUM N-(2-HYDROXYETHYL) -IMINODIACETATE	--	M5097	.0	0	1
FUMARIC ACID-ROSIN ADDUCT, SODIUM SALT	--	T0773	.0	0	1
INORGANIC MANGANESE COMPOUNDS	--	44025	.0	1	0
PHOTOGRAPHIC PLATE CLEANER	--	59173	.0	1	0
SALICYLIC ACID	69-72-7	67680	.0	1	0
INORGANIC SILICATES	--	68655	.0	1	0
CORRECTION FLUID	--	80018	.0	1	0
CURING AGENT	--	80020	.0	1	0
AJAX	--	80039	.0	1	0
AMMONIUM SULFATE	7783-20-2	80158	.0	1	0
INHIBITOR	--	80256	.0	1	0
POTASSIUM PHOSPHATE, DIBASIC	7758-11-4	82861	.0	1	0
DEFOLIANT	--	92210	.0	1	0
INORGANIC IRON COMPOUNDS	--	M0099	.0	1	0
POTASSIUM PHOSPHATE, MONOBASIC	7778-77-0	M0132	.0	1	0
PLASTICS	--	M0522	.0	1	0

TABLE VI-8

IARC SUSPECTED HUMAN OR ANIMAL CARCINOGENS:
PRODUCTION WORKERS

Percent of Group Exposed	Actual # Exposures to Agent	Trade-Name Ingredient Exposures	NIOSH Hazard Number	CAS #	Agent Name	Comment
.1	3	0	17490	5-62-3	CARBON TETRACHLORIDE	IARC: ANIMAL CARCINOGEN
.1	0	4	38110	30-20-1	HYDRAZINE	IARC: ANIMAL CARCINOGEN
.2	9	0	82272	6-25-6	THIOUREA	IARC: ANIMAL CARCINOGEN
.4	0	18	25145	12-39-1	DIOXANE, 1,4-	IARC: ANIMAL CARCINOGEN
.8	33	4	09070	7-14-3	BENZENE	IARC: SUSPECTED CARCINOGEN FOR MAN
2.5	0	122	26880	7-77-8	DIMETHYL SULFATE	IARC: ANIMAL CARCINOGEN
3.3	0	161	M2160	7-25-7	C.I. 23850-DIRECT BLUE 14	IARC: ANIMAL CARCINOGEN
5.0	248	0	90310	133-22-1	ASBESTOS	IARC: SUSPECTED CARCINOGEN FOR MAN

Extracted from 889 agents in NOHS data base for SIC codes 2621, 2631, and 2661.

TABLE VI-9

IARC SUSPECTED HUMAN OR ANIMAL CARCINOGENS OR OSHA CLASS I OR II CARCINOGENS OBSERVED FOR SPECIFIC OCCUPATIONS IN THE PULP AND PAPER INDUSTRY*

	BENZENE	C. I. 23850-DIRECT BLUE 14	C. I. 22610-DIRECT BLUE 6	C. I. 30145-DIRECT BROWN 95	C. I. 30235-DIRECT BLACK 38	ASBESTOS	THIOUREA	TRICHLOROETHYLENE	TETRACHLOROETHYLENE	HYDRAZINE	DIMETHYL SULFATE	1,4-DIOXANE	CHLOROFORM	CARBON TETRACHLORIDE	HEXACHLOROETHANE	WORKER CLASS**	TOTAL NUMBER OF AGENTS PER OCCUPATION
MACHINE OPERATIVES, MISCELLANEOUS SPECIFIED							2621		2621	2621		2661	2631	2631	2621 2631	2621 2631		P	8
ENGINEERING AND SCIENCE TECHNICIANS, N.E.C.		2621									2621		2631	2631	2631	2631	2621	P	7
PLUMBERS AND PIPE FITTERS	2631				2621								2631	2631	2631	2631		P	6
CHECKERS, EXAMINERS, AND INSPECTORS; MANUFACTURING			2621			2621				2621		2661				2621	2621 2661	P	6
NOT SPECIFIED LABORERS												2661	2631	2631	2631	2631	2631	P	6
MACHINE OPERATIVES, NOT SPECIFIED												2661	2631	2631	2631	2631		P	5
MISCELLANEOUS OPERATIVES												2661	2631	2631	2631	2631		P	5
CHEMISTS								2621	2621	2621						2621		N	4
MIXING OPERATIVES												2661	2621		2621			P	3
NOT SPECIFIED OPERATIVES							2621					2661				2621		P	3
RESEARCH WORKERS, NOT SPECIFIED		2621	2621														2621	N	3
ELECTRICIANS	2631							2621										P	2

TABLE VI-9 (CONTINUED)

IARC SUSPECTED HUMAN OR ANIMAL CARCINOGENS OR OSHA CLASS I OR II CARCINOGENS OBSERVED
FOR SPECIFIC OCCUPATIONS IN THE PULP AND PAPER INDUSTRY*

	BENZENE	C. I. 23850-DIRECT BLUE 14	C. I. 22610-DIRECT BLUE 6	C. I. 30145-DIRECT BROWN 95	C. I. 30235-DIRECT BLACK 38	ASBESTOS	THIOUREA	TRICHLOROETHYLENE	TETRACHLOROETHYLENE	HYDRAZINE	DIMETHYL SULFATE	1,4-DIOXANE	CHLOROFORM	CARBON TETRACHLORIDE	HEXACHLOROETHANE	WORKER CLASS**	TOTAL NUMBER OF AGENTS PER OCCUPATION
OCCUPATIONS:																	
FOREMEN, N.E.C.									2621	2621						P	2
MILLWRIGHTS	2631								2621							P	2
STATIONARY ENGINEERS									2621						2631	P	2
JANITORS AND SEXTONS									2621		2661					P	2
ENGINEERS, N.E.C.		2621													2621	M	2
HEAVY EQUIPMENT MECHANICS, INCL. DIESEL									2621						2631	M	2
CRANEMEN, DERRICKMEN, AND HOISTMEN											2661					P	1
MACHINISTS	2631															P	1
MISCELLANEOUS MECHANICS AND REPAIRMEN									2621							P	1
PRESSMEN AND PLATE PRINTERS, PRINTING									2631							P	1
ASBESTOS AND INSULATION WORKERS											2621					P	1
CUTTING OPERATIVES, N.E.C.											2661					P	1
GRADERS AND SORTERS, MANUFACTURING											2661					P	1

TABLE VI-9 (CONTINUED)

IARC SUSPECTED HUMAN OR ANIMAL CARCINOGENS OR OSHA CLASS I OR II CARCINOGENS OBSERVED FOR SPECIFIC OCCUPATIONS IN THE PULP AND PAPER INDUSTRY*

OCCUPATIONS:	BENZENE	C. I. 23850-DIRECT BLUE 14	C. I. 22610-DIRECT BLUE 6	C. I. 30145-DIRECT BROWN 95	C. I. 30235-DIRECT BLACK 38	ASBESTOS	THIOUREA	TRICHLOROETHYLENE	TETRACHLOROETHYLENE	HYDRAZINE	DIMETHYL SULFATE	1,4-DIOXANE	CHLOROFORM	CARBON TETRACHLORIDE	HEXACHLOROETHANE	WORKER CLASS**	TOTAL NUMBER OF AGENTS PER OCCUPATION
PACKERS AND WRAPPERS, EXCEPT MEAT AND PRODUCE								2661								P	1
GRINDING MACHINE OPERATIVES								2661								P	1
SAWYERS								2661								P	1
STATIONARY FIREMEN							2621									P	1
WELDERS AND FLAME-CUTTERS						2631										P	1
WINDING OPERATIVES, N.E.C.								2661								P	1
FORK LIFT AND TOW MOTOR OPERATIVES								2661								P	1
FREIGHT AND MATERIAL HANDLERS								2661								P	1
REGISTERED NURSES															2621	N	1
DUPLICATING MACHINE OPERATORS							2621									N	1
SECRETARIES, N.E.C.							2621 2661									N	1
STENOGRAPHERS							2621									N	1
COMPOSITORS AND TYPESETTERS							2621									N	1

* Extracted from 889 agents in NOHS data base for SIC codes 2621, 2631, and 2661.
**P = Production worker; N = Nonproduction worker; M = Miscellaneous worker

TABLE VI-10

HAZARDS DERIVED FROM LITERATURE REVIEW
AND EXTRACTED FROM NOHS DATA BASE:
PRODUCTION WORKERS

Percent of Group Exposed	Actual # Exposures to Agent	Trade-Name Ingredient Exposures	NIOSH Hazard Number	CAS #	Agent Name
48.8	2404	0	P0610	0-00-0	CONTINUOUS NOISE
20.5	1007	0	P0615	807-72-0	POTENTIAL CONTINUOUS NOISE
9.2	378	76	91115	8008-20-6	KEROSENE
7.7	277	0	04620	10043-01-3	ALUMINUM SULFATE (ALUM)
7.2	126	226	A1358	1346-36-7	TITANIUM OXIDE
6.8	218	119	69070	131-07-3	SODIUM HUDROXIDE
5.2	1	257	81663	775-78-2	SODIUM SULFATE
5.0	248	0	90310	133-22-1	ASBESTOS *
4.1	12	189	80164	8050-09-7	ROSIN
3.9	92	98	70870	766-49-3	SULFURIC ACID
3.3	43	121	33640	5-00-0	FORMALDEHYDE
3.2	8	149	69060	768-15-2	SODIUM HYPOCHLORITE
3.1	125	26	71055	1480-79-6	TALC
1.9	0	94	80216	800-22-6	TALL OIL
1.4	34	35	15755	130-57-8	CALCIUM OXIDE
.7	3	33	80298	1309-48-4	MAGNESIUM OXIDE
.7	0	34	77190	131-41-3	ZINC OXIDE
.6	0	31	41775	1332-58-7	KAOLIN
.5	23	4	18040	778-25-0	CHLORINE
.5	27	0	94040	0-00-0	COTTON
.4	0	20	03540	7-90-6	ACRYLAMIDE
.4	0	18	70845	770043-4	SULFUR
.3	13	4	69460	131-28-2	SODIUM SULFIDE
.3	14	0	74990	800-66-4	TURPENTINE
.2	12	0	70860	744-60-9	SULFUR DIOXIDE
.2	8	0	68870	775-50-9	SODIUM CHLORATE
.1	7	0	M3046	901-10-5	UREA, POLYMER WITH FORMALDEHYDE
.1	4	0	17460	63-00-8	CARBON MONOXIDE
.1	0	4	38110	302-01-2	HYDRAZINE

* IARC: SUSPECTED CARCINOGEN FOR MAN

TABLE VI-11

HAZARDS DERIVED FROM LITERATURE REVIEW
AND EXTRACTED FROM NOHS DATA BASE:
NONPRODUCTION WORKERS

Percent of Group Exposed	Actual # Exposures to Agent	Trade-Name Ingredient Exposures	NIOSH Hazard Number	CAS #	Agent Name
7.3	0	38	A1358	1346-36-7	TITANIUM OXIDE
5.6	14	15	69070	131-07-3	SODIUM HYDROXIDE
4.2	18	4	70870	766-49-3	SULFURIC ACID
2.1	0	11	69090	768-15-2	SODIUM HYPOCHLORITE
1.7	2	7	04620	10043-01-3	ALUMINUM SULFATE (ALUM)
1.3	7	0	P0610	0-00-0	CONTINUOUS NOISE
1.2	6	0	P0615	807-72-0	POTENTIAL CONTINUOUS NOISE
1.0	3	2	33640	5-00-0	FORMALDEHYDE
.8	0	4	03540	7-90-6	ACRYLAMIDE
.8	0	4	71055	1480-79-6	TALC
.6	3	0	74990	800-66-4	TURPENTINE
.6	0	3	M1529	5-00-0	FORMALIN
.6	0	3	81663	775-78-2	SODIUM SULFATE
.4	0	2	80216	800-22-6	TALL OIL
.2	1	0	17460	63-00-8	CARBON MONOXIDE
.2	0	1	18040	778-25-0	CHLORINE
.2	0	1	70845	770-43-4	SULFUR
.2	0	1	41775	1332-58-7	KAOLIN

TABLE VI-12

HAZARDS DERIVED FROM LITERATURE REVIEW
AND EXTRACTED FROM NOHS DATA BASE:
MISCELLANEOUS WORKERS

Percent of Group Exposed	Actual # Exposures to Agent	Trade-Name Ingredient Exposures	NIOSH Hazard Number	CAS #	Agent Name
33.3	78	0	P0610	0-00-0	CONTINUOUS NOISE
33.3	18	60	A1358	1346-36-7	TITANIUM OXIDE
22.6	0	53	80164	8050-09-7	ROSIN
21.4	0	50	33640	5-00-0	FORMALDEHYDE
21.4	0	50	80216	800-22-6	TALL OIL
11.5	27	0	70870	766-49-3	SULFURIC ACID
10.3	24	0	69070	131-07-3	SODIUM HYDROXIDE
8.5	20	0	04620	10043-01-3	ALUMINUM SULFATE (ALUM)
5.6	13	0	P0615	807-72-0	POTENTIAL CONTINUOUS NOISE
5.6	9	4	18040	778-25-0	CHLORINE
3.8	9	0	94040	0-00-0	COTTON
3.4	8	0	69460	131-38-2	SODIUM SULFIDE
3.4	8	0	70860	744-60-9	SULFUR DIOXIDE
3.4	8	0	74990	800-66-4	TURPENTINE
3.4	8	0	91115	8008-20-6	KEROSENE
3.0	0	7	70845	770-43-4	SULFUR
1.7	1	3	15755	130-57-8	CALCIUM OXIDE
1.7	0	4	03540	7-90-6	ACRYLAMIDE
1.7	0	4	41775	1332-58-7	KAOLIN
1.3	3	0	M3046	901-10-5	UREA, POLYMER WITH FORMALDEHYDE
1.3	0	3	77190	131-41-3	ZINC OXIDE
.9	0	2	90310	133-22-1	ASBESTOS *
.4	0	1	81663	775-78-2	SODIUM SULFATE

*IARC: SUSPECTED CARCINOGEN IN MAN

TABLE VI-13

INJURY AND ILLNESS INCIDENCE RATES FOR WORKERS IN PULP AND PAPER MILLS *

(per 100 full-time workers)

SIC Classification	1972	1973	1974	1975	1976	1977	1978
26--Paper and allied products**							
Total Cases	16.0	15.8	15.1	13.3	13.7	13.6	13.5
Lost Workday Cases	4.1	4.3	4.4	4.1	4.7	5.0	5.7
Lost Workdays	76.4	87.1	85.8	85.6	94.8	101.6	103.3
2611--Pulp mills							
Total Cases	13.5	17.6	18.7	16.5	14.0	13.1	11.1
Lost Workday Cases	2.2	3.1	3.1	3.2	3.0	3.3	3.5
Lost Workdays	60.1	115.0	72.1	95.4	74.4	87.4	82.7
2621--Paper mills							
Total Cases	12.2	12.9	11.8	10.1	11.3	10.6	10.3
Lost Workday Cases	3.0	3.0	3.3	3.2	3.8	4.1	4.6
Lost Workdays	69.4	90.3	78.9	80.5	96.2	107.0	106.5
2631--Paperboard mills							
Total Cases	16.8	16.2	14.7	13.6	12.6	13.6	12.3
Lost Workday Cases	3.9	4.1	4.0	3.7	3.9	5.0	4.9
Lost Workdays	95.8	111.6	93.4	97.2	90.7	111.5	106.7
2661--Building paper and building board mills							
Total Cases	17.1	17.6	17.8	13.0	16.2	13.6	13.3
Lost Workday Cases	3.5	4.0	4.2	4.2	6.2	5.8	5.7
Lost Workdays	102.5	128.6	128.2	136.3	182.5	144.7	106.7

*Incidence rates represent the number of injuries and illness or lost workdays and were calculated as (N/EH) X 200,000, where

N = number of injuries and illnesses or lost workdays
EH = total hours worked by all employees during calendar year
200,000 = base for 100 full-time equivalent workers (working 40 h/wk, 50 wk/yr).

TABLE VI-13 (CONTINUED)

INJURY AND ILLNESS INCIDENCE RATES FOR WORKERS IN PULP AND PAPER MILLS

(per 100 full-time workers)

**SIC 26 includes the following industries in addition to 2611, 2621, 2631, and 2661: 2641-2649, miscellaneous converted paper products; and 2651-2655, paperboard containers and boxes.

Adapted from references NO2499, NO2500, NO2764, NO2766

TABLE VI-14

RANKING OF 2-DIGIT SIC MANUFATURING INDUSTRIES
BASED ON NUMBER OF FATALITIES AND ILLNESS AND INJURY
INCIDENCE RATES FOR 1976

Manufacturing Industry	Fatality Rank*		Injury and Illness Rank**	
Lumber and wood products (SIC 24)	1	(280)	1	(167)
Food and kindred products (SIC 20)	2	(170)	2	(124)
Primary metal industries (SIC 33)	3	(130)	3	(115)
Fabricated metal products (SIC 34)	5	(80)	6	(110)
Machinery, except electrical (SIC 35)	5	(80)	10	(71)
Transportation equipment (SIC 37)	5	(80)	9	(74)
Stone, clay, and glass products (SIC 32)	7	(70)	4	(114)
Chemicals and allied products (SIC 28)	8	(60)	16	(51)
Paper and allied products (SIC 26)	9	(50)	7	(95)
Electric and electronic equipment (SIC 36)	10	(30)	17	(45)
Textile mill products (SIC 22)	12.5	(20)	15	(56)
Printing and publishing (SIC 27)	12.5	(20)	18	(40)
Petroleum and coal products (SIC 29)	12.5	(20)	12.5	(62)
Rubber and plastics products (SIC 30)	12.5	(20)	5	(113)
Apparel and other textile products (SIC 23)	17	(10)	20	(31)
Furniture and fixtures (SIC 25)	17	(10)	8	(94)
Leather and leather products (SIC 31)	17	(10)	11	(69)
Instruments and related products (SIC 38)	17	(10)	19	(37)
Miscellaneous industries (SIC 39)	17	(10)	14	(59)
Tobacco manufactures (SIC 21)	20	(5)	12.5	(62)

*Numbers in parentheses are unpublished estimated number of fatalities, (telephone communication, Mr. Steven Newell, Bureau of Labor Statistics, October 10, 1980)

**Numbers in parentheses are lost workday illness and injury incidence rates per 100 full-time employees, adapted from reference N02499

TABLE VI-15

OSHA TESTS FOR HAZARDOUS SUBSTANCES IN PULP AND PAPER MILLS
FROM JULY 1972 THROUGH APRIL 1980

Agent	Test Method	Total No. of Sites	Total No. of Employees Affected	No. of Sites Exceeding OSHA Standard	No. of Employees at Sites Exceeding OSHA Standard
		SIC 2611 - Pulp Mills			
Physical and Dust Hazards					
Dust (asbestos)	Air	2	55	--	--
" (nuisance)	"	4	33	--	--
" (nuisance)	Direct	1	8	--	--
" (wood)	Air	1	1	--	--
Improper illumination	Direct	2	380	1	100
Noise (continuous)	"	17	748	11	689
" (intermittent)	"	20	207	13	98
Total		47	1,432	25	887
Chemical Hazards					
Calcium oxide	Air	1	1	--	--
Carbon dioxide	Direct	1	30	--	--
Carbon monoxide	"	3	56	--	--
Chlorine	"	10	204	1	3
Chromium	Air	1	1	--	--
Copper	"	1	3	--	--
Ethyl mercaptan	"	1	1	--	--
Formaldehyde	Direct	1	3	--	--
Hydrogen sulfide	"	3	14	--	--
Iron oxide	Air	2	4	--	--
Manganese	"	2	4	--	--
Mercury	Direct	1	18	--	--
Methyl mercaptan	Air	1	1	--	--
Nickel	"	1	1	--	--
Nitric oxide	Direct	1	3	--	--
Sulfur dioxide	"	3	7	--	--
" "	Air	2	2	2	2
Turpentine	"	1	1	--	--
Xylene	Direct	1	3	--	--
Total		37	357	3	5

TABLE VI-15 (CONTINUED)

OSHA TESTS FOR HAZARDOUS SUBSTANCES IN PULP AND PAPER MILLS
FROM JULY 1972 THROUGH APRIL 1980

Agent	Test Method	Total No. of Sites	Total No. of Employees Affected	No. of Sites Exceeding OSHA Standard	No. of Employees at Sites Exceeding OSHA Standard
		SIC 2621 - Paper Mills			
Physical and Dust Hazards					
Dust (asbestos)	Air	24	497	3	24
" (cotton)	"	1	1	1	1
" (nuisance; other)	"	58	349	2	2
" (silica)	"	26	109	7	20
" (wood)	"	10	173	--	--
Electrical shock	Direct	12	1,010	4	806
Heat stress (humid)	"	4	80	--	--
Noise (continuous)	"	212	3,851	108	2,156
" (intermittent)	"	114	4,349	69	3,509
" (impact)	"	1	12	--	--
" (ultrasonic)	"	1	8	1	8
Pressure (high)	"	13	60	5	32
" (low)	"	2	46	1	40
Total		478	10,545	201	6,598
Chemical Hazards					
Acetone	Air	2	11	1	1
Acrolein	"	1	1	--	--
Acrylonitrile	"	2	7	--	--
"	Direct	2	10	1	5
Ammonia	"	7	1,036	--	--
"	Air	6	26	1	5
Antimony	"	2	3	1	2
Benzene	"	3	10	--	--
Butyl alcohol	"	1	4	--	--
Cadmium	"	1	1	--	--
Calcium carbonate	"	1	5	--	--

TABLE VI-15 (CONTINUED)

OSHA TESTS FOR HAZARDOUS SUBSTANCES IN PULP AND PAPER MILLS
FROM JULY 1972 THROUGH APRIL 1980

Agent	Test Method	Total No. of Sites	Total No. of Employees Affected	No. of Sites Exceeding OSHA Standard	No. of Employees at Sites Exceeding OSHA Standard
Calcium oxide	"	10	36	1	4
Carbon monoxide	Direct	31	423	1	3
Chlorine	"	36	3,407	--	--
"	Air	11	115	--	--
Chlorine dioxide	"	2	14	--	--
" "	Direct	4	51	--	--
Chloroform	Air	1	22	--	--
Chromium	"	5	9	--	--
Coal tar	"	2	13	1	12
Copper	"	1	1	--	--
Ethyl acetate	"	3	6	--	--
Ethyl alcohol	"	1	3	--	--
Ethyl cellosolve	"	3	6	--	--
Ethyl mercaptan	"	1	2	--	--
Formaldehyde	"	3	16	--	--
"	Direct	5	65	--	--
Hydrogen chloride	"	1	9	--	--
Hydrogen sulfide	"	20	265	2	11
" "	Air	3	48	--	--
Iron oxide	"	3	4	--	--
Isopropyl alcohol	"	2	16	--	--
Lead	"	9	13	1	1
Manganese	"	1	2	--	--
Mercury	"	1	3	--	--
"	Direct	1	3	--	--
Methyl cellosolve	Air	1	22	--	--
Methyl ethyl ketone	"	5	47	1	1
" " "	Direct	1	1	--	--
Methyl isobutyl ketone	"	2	13	--	--
" " "	Air	2	42	--	--
Methyl mercaptan	"	1	2	--	--
" "	Direct	2	103	1	3
Methyl chloroform	Air	3	11	--	--
Methylene chloride	"	1	22	--	--
Molybdenum	"	1	2	--	--
Nickel	"	3	4	--	--
Ozone	"	2	3	--	--
"	Direct	3	8	--	--
Phosphoric acid	Air	3	12	--	--

TABLE VI-15 (CONTINUED)

OSHA TESTS FOR HAZARDOUS SUBSTANCES IN PULP AND PAPER MILLS
FROM JULY 1972 THROUGH APRIL 1980

Agent	Test Method	Total No. of Sites	Total No. of Employees Affected	No. of Sites Exceeding OSHA Standard	No. of Employees at Sites Exceeding OSHA Standard
Sodium hydroxide	"	1	2	--	--
Styrene monomer	"	1	2	--	--
" "	Direct	1	2	--	--
Sulfur dioxide	Direct	14	169	--	--
" "	Air	7	37	4	25
Sulfuric acid	"	4	7	--	--
Tin (inorganic)	"	1	1	--	--
" (organic)	"	1	1	--	--
Toluene	"	7	61	1	1
"	Direct	3	34	--	--
Toluene diisocyanate	Air	1	5	--	--
Vanadium	"	1	2	--	--
Vinyl chloride	"	1	2	--	--
" "	Direct	3	17	--	--
Xylene	"	1	2	--	--
"	Air	6	14	1	1
Zinc oxide	"	1	2	--	--
Other chemicals	Air,direct	7	23	--	--
Total		279	6,341	18	75

SIC 2631 - Paperboard Mills

Physical and Dust Hazards

Dust (silica)	Air	3	27	--	--
" (asbestos)	"	1	40	--	--
" (nuisance; other)	"	3	13	--	--
Electrical shock	Direct	5	99	3	48
Noise (continuous)	"	138	7,038	105	6,757
" (intermittent)	"	13	241	7	46
" (impact; other)	"	3	32	2	27
Pressure (high)	Direct	2	10	1	1
Total		168	7,500	118	6,879

TABLE VI-15 (CONTINUED)

OSHA TESTS FOR HAZARDOUS SUBSTANCES IN PULP AND PAPER MILLS
FROM JULY 1972 THROUGH APRIL 1980

Agent	Test Method	Total No. of Sites	Total No. of Employees Affected	No. of Sites Exceeding OSHA Standard	No. of Employees at Sites Exceeding OSHA Standard
Chemical Hazards					
Ammonia	Direct	1	4	--	--
Calcium oxide	Air	1	7	--	--
Carbon monoxide	Direct	19	189	1	80
Chlorine	"	5	52	1	12
Chlorine dioxide	"	2	18	2	18
Ethyl acetate	Air	2	8	--	--
Formaldehyde	"	1	4	--	--
Hexane	"	1	2	--	--
Hydrogen sulfide	"	1	12	--	--
" "	Direct	7	34	--	--
Isopropyl acetate	Air	1	2	--	--
Isopropyl alcohol	"	1	2	--	--
Methyl chloroform	"	1	6	--	--
Methyl ethyl ketone	"	1	2	--	--
N-propyl acetate	"	1	2	--	--
Sodium hydroxide	"	4	52	--	--
Sulfur dioxide	Direct	3	32	1	12
Sulfuric acid	"	3	18	2	12
" "	Air	1	40	--	--
Toluene	"	1	2	--	--
Toluene diisocyanate	"	2	8	--	--
Total		59	496	7	134

TABLE VI-15 (CONTINUED)

OSHA TESTS FOR HAZARDOUS SUBSTANCES IN PULP AND PAPER MILLS
FROM JULY 1972 THROUGH APRIL 1980

Agent	Test Method	Total No. of Sites	Total No. of Employees Affected	No. of Sites Exceeding OSHA Standard	No. of Employees at Sites Exceeding OSHA Standard
SIC 2661-Building Paper and Building Board Mills					
Physical and Dust Hazards					
Dust (asbestos)	Air	19	470	4	14
" (nuisance; other)	"	15	211	4	18
" (nuisance; other)	Direct	3	29	--	--
" (silica)	Air	3	36	1	2
" (talc)	"	1	12	--	--
" (wood)	"	4	23	1	7
" (wood)	Direct	1	3	--	--
Electrical shock	"	2	260	--	--
Heat stress (humid)	"	1	25	--	--
Noise (continuous)	"	42	361	16	145
" (intermittent)	"	12	213	4	44
Pressure (low)	"	1	2	--	--
Other hazards	"	1	4	--	--
Total		105	1,649	30	230
Chemical Hazards					
Acrylamide	Air	1	6	--	--
Acrylonitrile	Direct	1	4	--	--
Benzene	"	1	1	--	--
"	Air	1	8	--	--
Benzopyrene	"	1	1	--	--
Butyl cellosolve	"	1	1	--	--
Carbon monoxide	Direct	6	211	--	--
Chromium	Air	1	2	--	--
Coal tar	"	3	11	--	--

TABLE VI-15 (CONTINUED)

OSHA TESTS FOR HAZARDOUS SUBSTANCES IN PULP AND PAPER MILLS
FROM JULY 1972 THROUGH APRIL 1980

Agent	Test Method	Total No. of Sites	Total No. of Employees Affected	No. of Sites Exceeding OSHA Standard	No. of Employees at Sites Exceeding OSHA Standard
Formaldehyde	"	1	100	--	--
Hexane	"	1	6	--	--
Isobutyl acetate	"	1	3	--	--
Lead	"	1	1	--	--
Mercaptobenzene	"	1	1	--	--
Methyl ethyl ketone	"	3	12	--	--
" " "	Direct	1	6	1	6
Nickel	Air	1	2	--	--
Nitric oxide	Direct	1	1	--	--
Ozone	"	1	1	--	--
Pentachlorophenol	Air	1	6	--	--
Petroleum distillates	"	1	6	--	--
Sodium hydroxide	"	1	1	--	--
Toluene	"	5	23	--	--
"	Direct	1	6	--	--
Vinyl chloride	Air	1	4	--	--
Xylene	"	3	7	--	--
Other chemicals	"	2	5	--	--
Total		43	436	1	6

Adapted from reference N03126

TABLE VI-16

SAMPLING AND ANALYTICAL METHODS
FOR CHEMICAL HAZARDS IN PULP AND PAPER MILLS

Substance	Exposure Points/Uses	Sampling Method*	Analytical Method*	Reference
Asbestos	Blend chest; present in felt used on paper machines (filler)	Cellulose membrane filter	Fiber counting by Light Microscopy (239): positive identification of asbestos content by x-ray diffraction	N03294
Calcium oxide	Pulping liquor preparation and recovery; lime kiln operation	Cellulose membrane filter (S205)	Ashed with nitric and perchloric acids; dissolved in hydrochloric acid; atomic absorption spectrophotometry	N03279
Carbon black	Blend chest (pigment addition; paper finishing)	Polyvinyl chloride membrane filter (S262)	Gravimetric analysis	N03280
Carbon monoxide	Liquor recovery units and furnaces (burner effluent)	Aluminized 5-layer gas sampling bag (S340)	Infrared absorption spectrophotometry; ecolyzer (112)	N03299; N03290
Chlorine	Bleaching operations	Fritted bubbler containing methyl orange	Colorimetric spectrophotometry (209)	N03270
Diatoemaceous earth (Diatomite)	Blend chest (filler)	Cellulose membrane filter	Gravimetric; analysis for SiO_2 content	N03282; N03288
Formaldehyde	Blend chest (melamine- and urea-formaldehyde resin; wet-strength additive); beater (refiner additive)	Midget bubbler containing buffered Girard T reagent (S327)	Polarography	N03298
Hydrazine	Liquor recovery furnace (corrosion inhibitor)	Midget bubbler containing hydrochloric acid (S237)	Alkalinization and reaction with p-dimethylamino-benzaldehyde; colorimetric spectrophotometry	N03281
"	"	Adsorption on sulfuric acid-coated silica gel	Water elution, reaction with 2-furaldehyde; gas chromatography (248, proposed)	N03271
Hydrogen sulfide	Alkaline pulp digesters; brown stock washers; black liquor storage and oxidation (alkaline pulping effluent)	Absorption in alkaline cadmium hydroxide to form cadmium sulfide (CdS) (S4)	Spectrophotometric measurement of methylene blue formed from CdS reaction with N,N-dimethyl-p-phenylene-diamine (126)	N03275

TABLE VI-16 (CONTINUED)

SAMPLING AND ANALYTICAL METHODS
FOR CHEMICAL HAZARDS IN PULP AND PAPER MILLS

Substance	Exposure Points/Uses	Sampling Method*	Analytical Method*	Reference
Magnesium oxide	Sulfite pulping recovery processes (pulping raw material)	Cellulose membrane filter (S369)	Ashed with nitric acid; atomic absorption spectrophotometry	N03284
Polynuclear aromatic hydrocarbons				
(a) general	Sulfate recovery furnace (adsorbed on particles on electrostatic precipitator)	Glass fiber filter	Extraction with benzene; gas chromatographic separation; ultraviolet spectrophotometry (183)	N03272
(b) Benzo(a)-pyrene	"	Glass fiber filter	Extraction with benzene; thin-layer chromatographic separation; spectro-photofluorometry (186)	N03273
Polychlorinated biphenyls	Paper recycling (used as dye carrier in carbonless paper)	Florisil adsorption	Desorption with hexane; perchlorination; gas chromatography (253)	N03277
Resin Monomers				
Butadiene	Resins used in paper sizing and coatings; monomers may be present if stripping of resins not done	Charcoal adsorption (S91)	Desorption; gas chromatography	N03274
Styrene		Charcoal adsorption (S30)	" "	N03295
Acrylamide		Impinger	Differential pulse	N02929
Epichlorohydrin		Charcoal adsorption	Desorption; chromatography	N03296
Silica	Pulp stock (filler)	Cellulose acetate membrane filter (S315)	X-ray diffraction; redeposition on silver membrane filter (259)	N03282; N03288
Sodium hydroxide	Alkaline pulping liquor; extraction during chlorine bleaching	Polytetrafluoroethylene filter (S381)	Hydrochloric acid extraction; titration with sodium hydroxide under nitrogen atmosphere	N03286
Sulfates Sulfites Sulfur dioxide Sulfuric acid	Various points in acid and alkaline pulping processes; magnesium bisulfite: adsorption tower and red liquor recovery	Cellulose ester membrane filter (particulates) followed by cellulose filter containing potassium hydroxide (sulfur dioxide)	Contents of each filter extracted with deionized water; anion-exchange chromatography (268, proposed)	N03289; N03283

TABLE VI-16 (CONTINUED)

SAMPLING AND ANALYTICAL METHODS
FOR CHEMICAL HAZARDS IN PULP AND PAPER MILLS

Substance	Exposure Points/Uses	Sampling Method*	Analytical Method*	Reference
Talc	Filler in printing paper and liner board	Cellulose membrane filter	Gravimetric; particle counting by light microscopy	NO3300
Titanium dioxide	Blend chest (pigment, filler)	"	Gravimetric	NO3300
Turpentine	Black-liquor recovery furnace (alkaline pulping byproduct)	Charcoal adsorption (S88)	Desorption with carbon disulfide; gas chromatography	NO3276
Zinc oxide	Blend chest (pigment; paper finishing) nitrile and polyvinyl chloride) (S316)	Membrane filter (copolymer of acrylo- (222)	Extraction with isopropyl alcohol; X-ray diffraction	NO3278; NO3287

*Numbers in parentheses indicate Method Number, where available.

TABLE VI-17

HUMAN AND ANIMAL TOXICITY DATA FOR HAZARDS IN PULP AND PAPER MILLS

Hazard	CAS Number	Toxicity Data*	Major Human Effects**
Physical and Dust Hazards			
Noise	--	--	Gradual loss of hearing ability
Hot, humid environments	--	--	Heat exhaustion, heat stroke, and hyperpyrexia, heat syncope, heat cramps, heat rash
Wood dust	--	--	Adenocarcinoma of nasal cavity and sinuses
Cotton dust	--	inh-hmn TCLo 10,000 mg/m^3	Byssinosis which results in chest tightness, breathlessness; mill fever, symptoms of malaise, cough, fever, chills, bronchitis
Talc (fibrous)	14807-96-6	skn-hmn 300 ug/3 d (I)	Mild irritant; danger of pneumoconiosis after chronic inhalation
Talc (nonasbestos) less than 1% crystalline silica	--	None	
Asbestos	1332-21-4	ihl-hmn TCLo 1.2 fibers/cc/19 y (C) ihl-rat TCLo 12 mg/m^3/13 wk ipr-rat TDLo 280 mg/kg ipl-rat TDLo 100 mg/kg CARCINOGENIC DETERMINATION: HUMAN POSITIVE	Pulmonary fibrosis and carcinoma; the common fibrosis is mechanical, with human body sealing off each individual fiber, eventually leading to reduced respiratory ability; cardiac failure
Chemical Hazards			
RAW MATERIALS			
Magnesium oxide	1309-48-4	ihl-hmn TCLo 400 mg/m^3 TFX:UNS itr-ham TDLo 500mg/kg/30 wk (I) TFX:NEO	Toxic by inhalation, fumes can cause irritation to eyes, nose, influenza-like symptoms known as metal fume fever
Sulfuric acid	7664-93-9	eye-rbt 1,380 ug SEV orl-rat LD50 2,140 mg/kg ihl-gpg LC50 18 mg/m^3	Tissue burns, pulmonary edema; concentrated sulfuric is extremely destructive to all tissues; can produce thermal burns, corneal damage may be permanent; esophageal and gastric stenosis may occur

TABLE VI-17 (CONTINUED)

HUMAN AND ANIMAL TOXICITY DATA FOR HAZARDS IN PULP AND PAPER MILLS

Hazard	CAS Number	Toxicity Data*	Major Human Effects**
		SULFURIC ACID, mist	
		ihl-hmn TCLo 3mg/m3/24 wk ihl-rat LCLo 726 mg/m3/7 h ihl-mus LCLo 571 mg/m3/210 mo ihl-gpg LCLo 196 mg/m3/ h	
Sodium sulfate	7757-82-6	ivn-mus LDLo 1,220 mg/kg ivn-rbt LDLo 4,470 mg/kg	Not absorbed from the gastrointestinal tract; used as cathartic
Sodium sulfide	1313-82-2	orl-hmn LDLo 50 mg/kg	Irritation, skin corrosion
Sulfur	7704-34-9	eye-hmn 10.7 mg/m3 orl-hmn LDLo 500 mg/kg	Nuisance dust, eye and mucous membrane irritant; no permanent effects reported
Sodium hydroxide	1310-73-2	orl-rat LDLo 500 mg/kg skn-rbt 50 mg/24 h SEV eye-rbt 1% SEV	Markedly corrosive action to all body tissue; burns and ulceration; dust and mist cause extreme pulmonary irritation
Calcium oxide	1305-78-8	--	Severe irritant; may produce both a thermal and caustic burn; permanent eye damage to cornea; ulceration, bronchitis, and pneumonia
PULP BLEACHING AGENTS			
Chlorine	7782-50-5	ihl-hmn LCLo 1,268 mg/m3/30 mo ihl-rat LC50 864 mg/m3/1 h ihl-mus LC50 404 mg/m3/1 h ihl-mam LCLo 1,475 mg/m3/5 mo	Reacts with body fluids causing corneal and skin burns; a known asphyxiant causing pulmonary edema
Chlorine dioxide	10049-04-4	ihl-rat LCLo 1,403 mg/m3/15 mo	Respiratory irritant, lacrimator; causes running nose, purulent bronchitis and disseminated bronchopneumonia

TABLE VI-17 (CONTINUED)

HUMAN AND ANIMAL TOXICITY DATA FOR HAZARDS IN PULP AND PAPER MILLS

Hazard	CAS Number	Toxicity Data*	Major Human Effects**
Sodium hypochlorite	7681-52-9	--	Bronchial irritation, pulmonary edema due to corrosion of mucous membranes; gastric perforation, corneal damage and esophageal strictures
Sodium chlorate	7775-09-9	skn-rbt 500 mg/24 h MLD eye-rbt 10 mg/MLD orl-hmn LDLo 50 mg/kg orl-wmn TDLo 800 mg/kg TFX:RBC unk-hmn LDLo 214 mg/kg unk-chd LD50 185 mg/kg orl-rat LD50 1,200 mg/kg ipr-mus LD50 596 mg/kg orl-cat LDLo 1,350 mg/kg orl-rbt LDLo 8,000 mg/kg	Acute poisoning causes nausea, vomiting, and diarrhea; causes irritation, methemoglobinemia, hemolytic anemia and tubular neurosis of kidneys
PAPERMAKING ADDITIVES			
Aluminium Sulfate	10043-01-3	ipr-mus LD50 270 mg/kg	Pharmaceutically mild astringent and antiseptic for the skin; concentrated solutions produce gingival necrosis and hemorrhagic gastroenteritis; incoordination, clonic contractions; evidence of nephritis
Kerosene	8008-20-6	skin-rbt 400 mg/24 h MOD skin-gpg 400 mg/24 h MOD orl-hmn LDLo 500 mg/kg orl-rat LDLo 28 gm/kg ipr-rat LDLo 10700 mg/kg itr-rat LDLo 800 mg/kg orl-rbt LD50 28 gm/kg ipr-rbt LD50 6600 mg/kg ivn-rbt LD50 180 mg/kg orl-gpg LD50 20 gm/kg	Defatting action on skin can cause irritation and infection; orally, central nervous system depression, intestinal irritation, and sometimes hemorrhages in visceral organs (spleen, kidney, liver); aspiration in lungs leads to fulminating hemorrhagic and often fatal bronchopneumonia
Rosin	8050-09-7	ihl-rat 220 mg/m3	Toxicity is probably low, scanty data available; heating of rosin produces vapors which irritate eyes, nose, throat and causes sensitization of the respiratory tract

TABLE VI-17 (CONTINUED)

HUMAN AND ANIMAL TOXICITY DATA FOR HAZARDS IN PULP AND PAPER MILLS

Hazard	CAS Number	Toxicity Data*	Major Human Effects**
Vanillin	121-33-5	orl-hmn LDLo 500 mg/kg orl-rat LD50 1580 mg/kg ipr-rat LD50 1160 mg/kg scu-rat LD50 15000 mg/kg ipr-mus LD50 475 mg/kg ivn-dog LDLo 1320 mg/kg orl-rbt LDLo 300 mg/kg orl-gpg LD50 1400 mg/kg ipr-gpg LD50 1190 mg/kg	Moderately toxic; probable oral lethal dose for humans is 0.5-5 g/kg
Titanium dioxide	13463-67-7	skn-hmn 300 ug/3 d (I) MLD ims-rat TDLo 300 mg/kg/84 wk (I)	Coughing, profuse lacrimation; can be considered a nuisance dust; virtually inert and not toxic to humans; possible carcinogen
Acrylamide	79-06-1	skn-rbt 50 mg/3 d MLD eye-rbt 10 mg/30 sec eye-rbt 100 mg/24 h SEV orl-rat LD50 170 mg/kg orl-mus LD50 170 mg/kg ipr-mus LD50 170 mg/kg orl-rbt LDLo 126 mg/kg skn-rbt LDLo 1,000 mg/kg orl-gpg LDLo 252 mg/kg	Can be absorbed through unbroken skin; causes central nervous system neuropathy; causes fatigue, contact dermatitis, muscular atrophy
Formaldehyde	50-00-0	skn-hmn 150 ug/3 d (I) MLD skn-rbt 540 mg open MLD eye-rbt 750 ug SEV orl-wmn LDLo 36 mg/kg ihl-hmn TCLo 17 mg/m^3/30 mo orl-hmn LDLo 500 mg/kg orl-rat LD50 800 mg/kg ihl-rat LCLo 312 mg/m^3/4 h scu-rat LD50 420 mg/kg scu-rat TDLo 1,300 mg/kg/65 wk (I) ivn-rat LD50 87 mg/kg ihl-mus LCLo 900 mg/gm^3/2 h ipr-mus LDLo 16 mg/kg scu-mus LD50 300 mg/kg scu-dog LDLo 550 mg/kg ihl-cat LCLo 820 mg/m^3/8 h skn-rbt LD50 270 mg/kg scu-rbt LDLo 240 mg/kg orl-gpg LD50 260 mg/kg	Severe irritant to skin, eyes, mucous membranes; ingestion can cause violent vomiting, vertigo, coma, or death

TABLE VI-17 (CONTINUED)

HUMAN AND ANIMAL TOXICITY DATA ON OF HAZARDS IN PULP AND PAPER MILLS

Hazard	CAS Number	Toxicity Data*	Major Human Effects**
Melamine formaldehyde (amino resin)	9003-08-1	--	Eczematous reaction (dermatitis); irritant to eyes, skin, and mucous membranes; when heated, decomposition occurs and toxic cyanide fumes are released
Urea formaldehyde (amino resin)	9011-05-6	--	Formaldehyde in resin causes irritation or burning of eyes and bronchial pathways
Zinc oxide (fume)	1314-13-2	skn-rbt 500 mg/24 h MOD eye-rbt 500 mg/24 h MOD orl-hmn LDLo 500 mg/kg ihl-hmn TCLo 600 mg/m3	"Metal fume fever"; fever, chills, vomiting, tightness in chest, cough; possibly pneumonia from fresh fumes
Silica (diatomaceous earth)	60676-86-0	orl-rat LD50 3,160 mg/kg ipr-rat LDLo 400 mg/kg itr-rat LDLo 120 mg/kg ivn-mus LDLo 9 mg/kg ipr-mus LDLo 40 mg/kg ivn-cat LDLo 15 mg/kg ivn-rbt LDLo 35 mg/kg ipr gpg LDLo 120 mg/kg	Silicas are inert in the digestive tract, chronic inhalation of silica dusts causes pneumoconiosis (known as silicosis); fibrotic changes and development of nodules in lungs causes shortness of breath; pure diatomaceous earth may produce a benign pneumoconiosis; cristobalite and quartz may produce pulmonary fibrosis; symptoms include cough, dyspnea, no fever or other systemic reaction
Silica (crystalline-cristobalite)	14464-46-1	ihl-hmn TCLo 400 particles/cc/4 y (I) ihl-hmn TCLo 16 mppcf/8 h/17.9 y (I) ipl-rat TDLo 90 mg/kg itr-rat LDLo 200 mg/kg	
Silica (crystalline-quartz)	14808-60-7	ihl-hmn 16 mppcf/8 h/17.9 y (I) ihl-hmn LCLo 300 ug/m3/10 y (I) ipr-rat TDLo 90 mg/kg ipr-rat TDLO 450 mg/kg/4 wk (I) ivn-rat LDLo 90 mg/kg ivn-rat TDLo 90 mg/kg ipl-rat TDLo 90 mg/kg itr-rat LDLo 200 mg/kg imp-rat TDLo 900 mg/kg ivn-mus LDLo 40 mg/kg imp-mus TDLo 4,000 mg/kg ivn-dog LDLo 20 mg/kg ipl-ham TDLo 83 mg/kg	
Carbon black	7440-44-0	ivn-mus LD50 440 mg/kg	Nuisance dust; conjunctivitis, epithelial hyperplasia of cornea, inflammation of eyelids and mucous membranes

TABLE VI-17 (CONTINUED)

HUMAN AND ANIMAL TOXICITY DATA FOR HAZARDS IN PULP AND PAPER MILLS

Hazard	CAS Number	Toxicity Data*	Major Human Effects**
CONTAMINANTS OR BYPRODUCTS			
Hydrazine	302-01-2	ihl-rat LC50 760 mg/m3/4 h ipr-rat LD50 76 mg/kg orl-mus TDLo 410 mg/kg/26 wk (C) TFX:CAR ihl-mus LD50 336 mg/m3/4 h ipr-mus LD50 163 mg/kg ipr-mus TDLo 400 mg/kg/5 wk (I) TFX:CAR unk-mus LD50 200 mg/kg skn-rbt LD50 90 mg/kg skn-rbt LD50 91 mg/kg ivn-rbt LD50 20 mg/kg	Violent poison, strong irritant to the skin and eyes; highly toxic by skin absorption, inhalation or ingestion
Turpentine	8006-64-2	eye-hmn 990 mg/m3 orl-hmn LDLo 500 mg/kg ihl-hmn TCLo 990 mg/m3 TFX:IRR ihl-mus LD50 600 ug/kg skn-mus TDLo 240 gm/kg/20 wk (I) TFX:NEO ivn-mus LD50 1,180 ug/kg	Inhalation can cause kidney irritation; 1 tbsp has caused death in children, 4 to 6 oz. in adults. Nausea, diarrhea, central nervous system depression, conjuctivitis, dizziness and tachypnea
Tall oil	8002-26-4	orl-rat LD_{50} 7,600 mg/kg*** orl-mus LD_{50} 4,600 mg/kg*** orl-gpg LD_{50} 4,600 mg/kg***	Mixture of oleic, linoleic and rosin acids, all very low in toxicity; a mild allergen
Dimethyl sulfoxide	67-68-5	skn-rbt 10 mg/24 h open MLD eye-rbt 100 mg	Primary skin irritant; corneal opacity has been produced in experimental animals; releases histamine; hemolytic to red blood cells
Polychlorinated biphenyls			
Aroclor 1242	53469-21-9	ihl-hmn TCLo 10 mg/m3 orl-rat LD50 4,250 mg/kg	Chloracne, liver damage, nausea, vomiting, birth defects, anorexia and weakness; exposure to high concentrations may lead to coma and death
Aroclor 1254	27323-18-8	orl-rat LD50 1,295 mg/kg orl-rat TDLo 1,220 mg/kg/35 wk (C) ivn-rat LD50 358 mg/kg orl-mus TDLo 17 gm/kg/48 wk (C) skn-mus TDLo 4,000 ug/kg ipr-mus LD50 2,840 mg/kg CARCINOGENIC DETERMINATION: ANIMAL POSITIVE	

TABLE VI-17 (CONTINUED)

HUMAN AND ANIMAL TOXICITY DATA FOR HAZARDS IN PULP AND PAPER MILLS

Hazard	CAS Number	Toxicity Data*	Major Human Effects**
EFFLUENTS			
Sulfur dioxide	7446-09-5	ihl-hmn LCLo 1,066 mg/m^3/1 mo ihl-hmn TCLo 8 mg/m^3/5 d ihl-man TCLo 11 mg/m^3/1 mo ihl-rat LCLo 2,665 mg/m^3 ihl-mus LCLo 15,988 mg/m^3/5 h ihl-frg LCLo 3 mg/m^3/15 mo ihl-mam LCLo 7,994 mg/m^3/5 mo	Mainly irritant; causes ulceration of cornea, coughing, lacrimatio dyspnea; pulmonary edema and pneumonitis; sulfurous acid formed on moist body parts
Hydrogen sulfide	7783-06-4	ihl-hmn LCLo 850 mg/m^3/30 mo ihl-rat LC50 629 mg/m^3 ihl-mus LC50 594 mg/m^3/1 h ihl-gpg LCLo 1 mg/m^3/8 h ihl-mam LCLo 1,134 mg/m^3/5 mo	Rapid and powerful systemic poison; asphyxiant due to paralysis of respiratory system; headache, digestive disturbances, corneal bullae, general debility
Methyl mercaptan	74-93-1	scu-mus LD50 2.4 mg/kg ihl-rat LC50 1,351 mg/m^3	Highly toxic by inhalation; irritating to skin and eyes; pulmonary edema, possibly neural and hepatic damage, anemia, respiratory paralysis and death
Dimethyl sufide	75-18-3	orl-rat LD50 535 mg/kg ihl-rat LC50 104,023 mg/m^3 ipr-mus LD50 8,000 mg/kg	Causes softening and irritation of skin; orally, produces irritant and corrosive action as it releases hydrogen sulfide and free alkali
Dimethyl disulfide	524-92-0	ihl-rat LC50 3,154 mg/m^3	With human body's inherently large capacity for detoxifying sulfides, concentrations of gas mixtures more important than lengths of exposure
Carbon monoxide	630-08-0	ihl-man LDLo 4,661 mg/m^3/30 mo ihl-man TCLo 757 mg/m^3/45 mo ihl-rat LD50 2,105 mg/m^3/4 h ihl-mus LD50 2,848 mg/m^3/4 h ihl-mus TCLo 350 mg/m^3/(12-18 d preg) ihl-dog LCLo 4,661 mg/m^3/46 mo ihl-cat LCLo 10,486 mg/m^3/35 h ihl-rbt LCLo 4,661 mg/m^3 ihl-gpg LC50 6,662 mg/m^3/4 h ihl-mam LCLo 5,826 mg/m^3/5 mo	No direct chemical poison; symptoms of tissue anoxia and formation of carboxyhemoglobin which prevents oxygen transport

*Source: N00893
**Sources: N00596, N00600, N00601, N00825, N00872, N00891, N00896, N00916, N00917, N00919, N02897
***Source: N03217

TABLE VI-17 (CONTINUED)

HUMAN AND ANIMAL TOXICITY DATA FOR HAZARDS IN PULP AND PAPER MILLS

Abbreviations:

ihl: inhilation	hmn: human	C: continuous	h: hours
ipr: intraperitoneal	mus: mouse	MLD: mild (irritation)	d: days
ipl: intrapleural	rbt: rabbit	MOD: moderate (")	wk: wk
skn: skin	gpg: guinea pig	SEV: Severe (")	mo: months
ivn: intravenous	mam: mammal	TFX: toxic effects	y: years
orl: oral	frg: frog	NEO: neonatol	
unk: unreported	ham: hamster	UNS: unspecified	
ims: intramuscular	wmn: woman	LC: lethal concentration	
scu: subcutaneous	chd: child	LD: lethal dose	
imp: implant		TC: toxic concentration	
itr: intratracheal		TD: toxic dose	
		IRR: irritant	
		CAR: carcinogen	

TABLE VI-18

NOISE LEVELS IN PULP AND PAPER MANUFACTURING

Location	Noise Levels (dBC)	Octave Band Analysis (dB)		
		300-600 Hz	600-1200 Hz	1200-2400 Hz
Drum barker	98	88	89	78
Slasher-gang saws	98	91	96	88
Wood chippers	108	100	100	99
Pulp grinder	105	95	96	94
Evaporators	93	85	84	85
Evaporator pumps	96	88	86	87
Jordan Refiners				
range	102-111	96-103	96-104	94-99
average	107	99	99	97
Wet-End, Fourdrinier				
range	99-106	92-94	91-92	87-90
average	102	93	91	89
Hoods floor level	97	88	85	83
10 ft above floor	99	92	92	83
15-18 ft above floor	108	91	93	97
Fourdrinier, opposite calendar				
range	98-101	87-88	84-88	81-89
average	99	88	86	84

Adapted from reference N02692