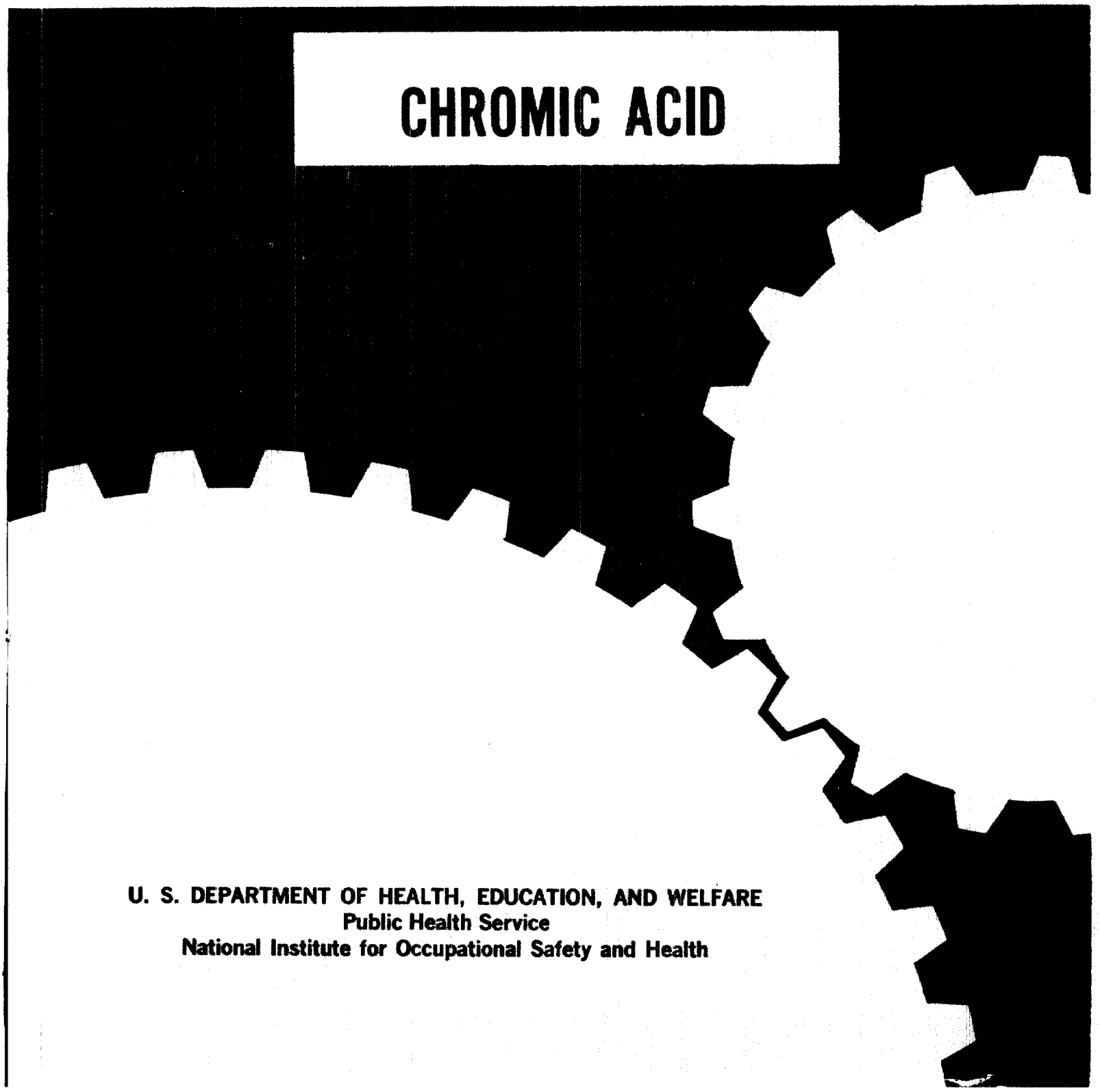
criteria for a recommended standard . . . .

OCCUPATIONAL EXPOSURE TO



U. S. DEPARTMENT OF HEALTH, EDUCATION, AND WELFARE
Public Health Service
National Institute for Occupational Safety and Health

criteria for a recommended standard . . . .

OCCUPATIONAL EXPOSURE TO CHROMIC ACID

U. S. DEPARTMENT OF HEALTH, EDUCATION, AND WELFARE
Public Health Service
National Institute for Occupational Safety and Health

1973

PREFACE

The Occupational Safety and Health Act of 1970 emphasizes the need for standards to protect the health of workers exposed to an ever-increasing number of potential hazards at their workplace. To provide relevant data from which valid criteria and effective standards can be deduced, the National Institute for Occupational Safety and Health has projected a formal system of research, with priorities determined on the basis of specified indices.

It is intended to present successive reports as research and epidemiologic studies are completed and sampling and analytic methods are developed. Criteria and standards will be reviewed periodically to ensure continuing protection of the worker.

I am pleased to acknowledge the contributions to this report on chromic acid by members of my staff, by Robert B. O'Connor, M.D., NIOSH consultant in occupational medicine, and by Edwin C. Hyatt, NIOSH consultant on respiratory protection. Valuable and constructive comments were presented by the Review Consultants on Chromic Acid and by the ad hoc committees of the Industrial Medical Association and of the American Academy of Industrial Hygiene. The NIOSH recommendations for standards are not necessarily a consensus of all the consultants and professional societies that reviewed this criteria document on chromic acid. Lists of the NIOSH Review Committee members and of the Review Consultants appear on the following pages.

Marcus M. Key, M.D.
Director, National Institute for
Occupational Safety and Health

The Office of Research and Standards Development, National Institute for Occupational Safety and Health, had primary responsibility for development of the criteria and recommended standard for chromic acid. George D. Clayton and Associates developed the basic information for consideration by NIOSH staff and consultants under contract No HSM-99-72-34. Bryan D. Hardin had NIOSH program responsibility and served as criteria manager.

REVIEW COMMITTEE
NATIONAL INSTITUTE FOR OCCUPATIONAL SAFETY AND HEALTH

Martin T. Abell
Division of Laboratories and
Criteria Development

George A. Carson, Ph.D.
Division of Laboratories and
Criteria Development

Jane A. Lee, R.N.
Division of Technical Services

James B. Lucas, M.D.
Division of Technical Services

Frank L. Mitchell, D.O.
Office of Research and
Standards Development

William L. Wagner
Division of Technical Services

Joseph K. Wagoner, S.D. Hyg.
Director, Division of Field Studies and
Clinical Investigations

Ex Officio:

Charles H. Powell, Sc.D.
Assistant Institute Director
for Research and Standards
Development

NIOSH REVIEW CONSULTANTS ON CHROMIC ACID

P. Leslie Bidstrup, M.D.
London, SW1X 9DG, England

Richard I. Chamberlin
Consulting Engineer
77 Massachusetts Avenue
Cambridge, Massachusetts 02139

Warren S. Ferguson
Director, Occupational Health and Product Safety
Allied Chemical Corporation
Morristown, New Jersey 07960

Jan Lieben, M.D.
Medical Director, American Viscose Division
FMC Corporation
Philadelphia, Pennsylvania 19103

T.F. Mancuso, M.D.
Research Professor of Occupational Health
Graduate School of Public Health
University of Pittsburgh
Pittsburgh, Pennsylvania 15232

James O. Pierce, Sc.D.
Director, Environmental Trace Substances Center
University of Missouri
Columbia, Missouri 65201

CRITERIA DOCUMENT: RECOMMENDATIONS FOR AN OCCUPATIONAL EXPOSURE STANDARD FOR CHROMIC ACID

Table of Contents

I. RECOMMENDATIONS FOR A CHROMIC ACID STANDARD

The National Institute for Occupational Safety and Health recommends that worker exposure to chromic acid (chromium trioxide) in the workplace be controlled by compliance with the following sections. The standard is designed to protect the health and safety of workers for an 8-hour day, 40-hour week over a working lifetime. Compliance with the standard should prevent adverse effects of occupational exposure to chromic acid. The standard is measurable by techniques that are valid, reproducible, and available. Sufficient technology exists to permit compliance with the recommended standard. The standard will be subject to review and revision as necessary.

"Chromic acid" is defined to mean chromium trioxide (chromium (VI) oxide, or chromic acid anhydride) and aqueous solutions thereof. "Occupational exposure to chromic acid" is defined as exposure above half the recommended workroom environmental standard.

Section 1 - Environmental (Workplace air)

(a) Concentration: Occupational exposure to chromic acid shall be controlled so that no worker is exposed either to:

(1) A concentration of chromic acid greater than 0.05 milligram as chromium trioxide per cubic meter of air determined as a time-weighted average exposure for an 8-hour workday, 40-hour work week; or

(2) A ceiling concentration in excess of 0.1 milligram as chromium trioxide per cubic meter as determined by a sampling time of fifteen (15) minutes.

(b) Sampling and Analysis: Procedures for sampling and analysis of air samples shall be as provided in Appendices I and II, or by any method shown to be equivalent in precision, accuracy, and sensitivity to the methods specified.

Section 2 - Medical

Medical surveillance shall be made available as outlined below for all workers occupationally exposed to chromic acid. Maintenance personnel periodically exposed during routine maintenance or emergency repair operations shall also be offered medical surveillance.

(a) Preplacement and annual medical examinations shall include:

(1) A work history to elicit information on all past exposures to chromic acid and other hexavalent chromium compounds.

(2) A medical history to elicit information on conditions indicating the inadvisability of further exposure to chromic acid, eg, skin or pulmonary sensitization, or a skin or mucous membrane condition that may promote response to chromic acid.

(3) Thorough examination of the skin for evidence of dermatitis or chromic ulcers and of the membranes of the upper respiratory tract for irritation, bleeding, ulcerations or perforations.

(4) An evaluation of the advisability of the worker's using negative- or positive-pressure respirators.

(b) Preplacement examinations shall include 14" x 17" chest X-rays. Thereafter, X-ray examinations shall be offered at 5-year intervals and annually after age 40.

(c) All workers with symptoms of skin or upper respiratory tract irritation shall be offered medical examinations at the time the symptoms first occur.

(d) Proper medical management shall be provided for workers adversely affected by exposure to chromic acid.

(e) Initial annual examinations shall be offered within 6 months of the promulgation of a standard incorporating these recommendations.

(f) The medical representatives of the Secretary of Health, Education, and Welfare, of the Secretary of Labor, and of the employer shall have access to all medical records. Physicians designated and authorized by any employee or former employee shall have access to his medical records.

(g) Medical records shall be maintained for persons employed one or more years with exposure to chromic acid. X-rays for the five years preceding termination of employment and all medical records with pertinent supporting documents shall be maintained at least 20 years after the individual's employment is terminated.

Section 3 - Labeling(Posting)

(a) All storage containers of chromic acid shall bear the following label in addition to or in combination with labels required by other statutes, regulations or ordinances.

CHROMIUM TRIOXIDE

(CHROMIC ACID)

DANGER! STRONG OXIDANT

CONTACT WITH OTHER MATERIAL MAY CAUSE FIRE

MAY CAUSE DELAYED BURNS OR EXTERNAL ULCERS

Keep container closed.

Do not get in eyes, on skin, on clothing.

Do not breathe dust or mist from solutions.

In case of contact, immediately flush skin or eyes with plenty of water for at least 15 minutes.

For eyes, get medical attention immediately.

Wash clothing before reuse.

Use fresh clothing daily. Take hot showers after work, using plenty of soap.

(b) The following warning sign shall be affixed in a readily visible location at or near entrances to areas in which there is occupational exposure to chromic acid.

WARNING!

Chromic Acid Area

Unauthorized Persons

KEEP OUT

Section 4 - Personal Protective Equipment and Clothing

Subsection (a) shall apply whenever a variance from the standard recommended in Section 1 is granted under provisions of the Occupational Safety and Health Act, or in the interim period during the application for a variance. Until the limits of exposure to chromic acid prescribed in paragraph (a) of Section 1 are met, an employer must utilize, as provided in subsection (a) of this Section, a program of respiratory protection for every worker exposed.

(a) Respiratory Protection: Engineering controls shall be used to maintain chromic acid concentrations below the prescribed limits. Appropriate respirators shall be provided and used when a variance has been granted to allow respirators as a means of control of exposure in routine operations and while the application for variance is pending. Administrative controls may also be used to reduce exposure. Respirators shall also be provided and used for nonroutine operations (occasional brief concentrations above the time weighted average or ceiling and for emergencies); however, for these instances a variance is not required but the requirements set forth below continue to apply. Appropriate respirators as described in Table I-1 shall only be used pursuant to the following requirements:

(1) For the purpose of determining the class of respirator to be used, the employer shall measure the atmospheric concentration of chromic acid in the workplace when the initial application for variance is made and thereafter whenever process, worksite, climate, or control changes occur which are likely to affect

the chromic acid concentration. The employer shall ensure that no worker is being exposed to chromic acid in excess of the standard because of improper respirator selection or fit.

(2) A respiratory protective program meeting the general requirements outlined in section 3.5 of American National Standard Practices for Respiratory Protection Z88.2-1969 shall be established and enforced by the employer.

Table I-1

Multiples of TWA Limit for 8 hour day	Respirator Type
less than 10x	Half-mask respirator with replaceable high efficiency or dust, fume, and mist filter.
less than 100x	Full facepiece respirator with replaceable high efficiency filter.
greater than 100x	Type C (positive pressure) supplied-air respirator.

(3) The employer shall provide respirators in accordance with Table I-1 and shall ensure that the employee uses the respirator provided.

(4) Respiratory protective devices described in Table I-1 shall be those approved either under the following regulations or under 30 CFR 11, published in the Federal Register March 25, 1972.

(A) Filter-type dust, fume, and mist respirator--30 CFR 14 (Bureau of Mines Schedule 21B)

(B) Supplied-air respirator--30 CFR 12 (Bureau of Mines Schedule 19B)

(5) A respirator specified for use in higher concentrations of chromic acid may be used in atmospheres of lower concentrations.

(6) Employees shall be given instruction on the use of respirators assigned to them, on cleaning respirators, and on testing for leakage.

(b) Protective Clothing:

(1) Coveralls or other full body protective clothing shall be worn in areas where there is occupational exposure to chromic acid. Protective clothing shall be changed at the end of each workday.

(2) Protective gloves, aprons, and footwear which are impervious to chromic acid shall be worn at operations where chromic acid spills and splashes may contact the skin.

(3) Unless eye protection is afforded by a respirator hood or facepiece, protective goggles or face shields impervious to chromic acid shall be worn at operations where chromic acid splashes may contact the eyes.

(4) All protective equipment shall be maintained in a clean and satisfactory working condition.

Section 5 - Informing Employees of Hazards from Chromic Acid

At the beginning of employment in a chromic acid area, employees exposed to chromic acid shall be advised of the hazards of exposure and the relevant symptoms. Proper conditions for safe use and precautions to minimize exposure shall be provided and explained to the employee. Instruction shall include, as a minimum, all information in Appendix III which is applicable to the material to which there is exposure. This information shall be posted in the work area and kept on file and readily accessible to the worker at all places of employment where chromic acid is manufactured or used in unit processes and operations.

A continuing educational program shall be instituted to ensure that all workers have current knowledge of job hazards, proper maintenance procedures and cleanup methods, and that they know how to correctly use respiratory protective devices and protective clothing.

Information as required shall be recorded on U.S. Department of Labor Form OSHA-20 "Material Safety Data Sheet" or a similar form approved by the Occupational Safety and Health Administration, U.S. Department of Labor.

Section 6 - Work Practices

(a) Control of Airborne Chromic Acid: Chromic acid shall be controlled at sources of dispersion by means of effective and properly maintained methods such as fully enclosed operations, local exhaust ventilation, and/or the use of surface active or other agents which reduce mist formation. Other methods may be used if they are shown to effectively control atmospheric levels of chromic acid within the limits of the recommended standard.

(b) General Housekeeping:

(1) Spills shall be cleaned up promptly. Equipment shall be kept in good repair and free of leaks.

(2) No dry sweeping shall be performed. Wet methods or dry vacuuming shall be used as appropriate.

(3) Work clothes contaminated with chromic acid shall be changed immediately. Outer garments shall be changed daily.

(4) Protective gloves, aprons, footwear, and goggles contaminated with chromic acid shall be cleaned with water. When the inside of such protective equipment is contaminated, the equipment shall be removed immediately and discarded, or decontaminated before being reused.

(5) Skin contaminated with chromic acid shall be washed immediately and thoroughly with water. Eyes contaminated with chromic acid shall be washed immediately with copious amounts of water, after which the worker shall be referred immediately to a physician and, if necessary, an eye specialist.

(c) Chromic Acid Anhydride Work Practices: Those persons working directly with chromic acid anhydride, with unsealed containers of the anhydride, or with the anhydride in other than fully enclosed operations shall adhere to the following work practices.

(1) All protective devices and clothing specified below are required.

(A) Dust respirator meeting at least the minimum requirements of Section 4(a).

(B) Protection for the head, neck, and face against airborne particles of chromic acid anhydride, eg, a broad-brimmed hat, such as a full-brimmed hard hat, or respirator hood.

(C) Face shield or goggles, if eye and face protection is not provided by the respirator hood or facepiece.

(D) Coveralls or other full body protective clothing.

(E) Impermeable gauntlets, shoes, and apron.

(2) Protective devices and clothing shall be removed and the arms, hands, and face thoroughly washed:

(A) After working with the anhydride; and

(B) At 30-minute intervals when working with the anhydride for extended periods of time.

Section 7 - Sanitation Facilities

(a) Emergency shower facilities shall be available in the workplace for the removal of chromic acid.

(b) Hand washing and emergency eye washing facilities shall be provided in the work area. Instructions shall be posted for proper use of the eye washing facilities and for obtaining medical assistance.

(c) Food storage, preparation, and eating should be prohibited in areas where there is exposure to chromic acid.

Section 8 - Monitoring and Recordkeeping Requirements

Workroom areas shall not be considered to have chromic acid exposure if environmental levels, as determined on the basis of an industrial hygiene survey or by the judgment of a compliance officer, do not exceed half of the environmental standard. Records of these surveys, including the basis for concluding that air levels are below half of the environmental standard, shall be maintained. Requirements set forth below apply to areas in which there is chromic acid exposure.

Employers shall maintain records of environmental exposures to chromic acid based upon the following sampling and recording schedules:

(a) The first environmental sampling shall be completed within 6 months of the promulgation of a standard incorporating these recommendations.

(b) Environmental samples shall be taken within 30 days after installation of a new process or process changes.

(c) Samples shall be collected at least quarterly in accordance with Appendix I for the evaluation of the work environment with respect to the recommended standard.

(d) Samples shall be collected and evaluated in accordance with Appendix I for determination of time-weighted values and ceiling values.

(e) For work areas in which either the time-weighted average or the ceiling concentration of chromic acid exceeds the standard, monitoring and recordkeeping shall be repeated on a weekly basis until three consecutive sampling periods have demonstrated that environmental levels meet the standard.

(f) Records of all sampling (and of medical examinations) shall be maintained for at least 20 years. Records shall indicate the type of personal protective devices, if any, in use at the time of sampling. Records shall be maintained so that they can be classified by employees. Each employee shall be able to obtain information on his own environmental exposure.

II. INTRODUCTION

This report presents the criteria and the recommended standard based thereon which were prepared to meet the need for preventing occupational disease arising from exposure to chromic acid. The criteria document fulfills the responsibility of the Secretary of Health, Education, and Welfare, under Section 20(a)(3) of the Occupational Safety and Health Act of 1970 to "....develop criteria dealing with toxic materials and harmful physical agents and substances which will describe....exposure levels at which no employee will suffer impaired health or functional capacities or diminished life expectancy as a result of his work experience."

The National Institute for Occupational Safety and Health (NIOSH), after a review of data and consultation with others, formalized a system for the development of criteria upon which standards can be established to protect the health of workers from exposure to hazardous chemical and physical agents.

These criteria for a standard for chromic acid are in a continuing series of criteria developed by NIOSH. The proposed standard applies only to the processing, manufacture, and use of chromic acid in products as applicable under the Occupational Safety and Health Act of 1970.

The standard was not designed for the population-at-large, and any extrapolation beyond general occupational exposures is not warranted. It is intended to (1) protect against injury from chromic acid, (2) be measurable by techniques that are valid, reproducible,

and available to industry and official agencies, and (3) be attainable with existing technology.

III. BIOLOGIC EFFECTS OF EXPOSURE

Extent of Exposure

The chief source of chromium trioxide is from chromite ore obtained primarily from South Africa, Rhodesia, and the USSR, with minor amounts from Turkey, the Philippines, Cuba, Finland, Canada, India, and the United States. [1] Bourne and Yee [2] in 1950 reported the approximate analysis of chromite ores from Rhodesia and Transvaal, respectively, as: chromic oxide, 51.1 and 45.6%; iron oxide, 11.4 and 25.8%; aluminum oxide, 15.2 and 14.3%; silicon dioxide, 4.8 and 1.4%; and magnesium oxide, 12.7 and 11.8%.

According to Gafafer, [3] chromium trioxide is produced by roasting chromite ore with soda ash and lime to form sodium chromate, which is converted to sodium dichromate by acidification and crystallization. The sodium dichromate is then treated with sulfuric acid, and the temperature is raised above 197 C. [4] A molten reaction mixture results with the heavier chromic acid anhydride settling out. The anhydride reacts with water to form chromic acid and dichromic acid.

In 1969, approximately 25 thousand short tons of chromium trioxide were produced in the United States, of which ninety percent was used in metal treatment, such as chrome plating, copper stripping, and aluminum anodizing. [4] Other uses included catalysts, refractory purposes, organic synthesis, photography, and exports. Chromic acid is a strong oxidizing agent and concentrated solutions attack most common metals. [5] It is not combustible, but may ignite readily

oxidizable substances. [5] Significant physical properties [6] of chromium trioxide are presented in Table X-1.

NIOSH estimates that 15,000 people are potentially exposed to chromic acid mist.

Historical Reports

One of the first reports of injury to workers in this country from exposure to chromium compounds was in 1884 by MacKenzie. [7] He reported that factory workers employed in the chambers where bichromate was made invariably developed perforation of the nasal septum, generally within a few days of exposure. The characteristic development of septal perforation was described in detail. Although destruction of the cartilage was reported to be very extensive in many cases, the external appearance of the nose was said to be unchanged. Other effects reported included ulceration of the turbinates and nasal pharynx, and inflammation of the lower respiratory tract. Perforation of the tympanic membrane was also reported, due either to passage of bichromates through the Eustachian tubes or to direct external contact.

Reporting on 12 cases in two plating plants, Blair [8] in 1928 described four electroplaters who experienced symptoms of a bad cold with coryza, sneezing, watery discharge from the eyes and nose, and itching and burning of the nose, especially when they left the plant and came in contact with outdoor air. Of these four men, one had a perforated nasal septum, one a large unilateral ulcer on the septum, and two had marked congestion of the nasal mucosa with hyperemia,

swelling, mucoid discharge, and small ulcers. In the remaining 8 cases reported, workers who removed objects from the plating tanks complained little of nasal symptoms, but had ulcerative lesions of the hands and fingers. These "chrome holes" reportedly occurred only at the site of a preexisting scratch, cut, or break in the skin. No environmental levels of chromic acid were given, but the installation of an efficient ventilation system was reported to be sufficient to prevent nasal symptoms. Long sleeved rubber gloves were used to protect the hands and 5% sodium hyposulfite was reported useful to neutralize chromic acid on the hands.

Bloomfield and Blum [9] in 1928 reported a study of workers engaged in chromium plating and gave data on concentrations of chromic acid to which they were exposed. Of 19 workers exposed to chromic acid mists, 17 had symptoms including perforated septa, ulcerated septa, inflamed mucosa, nosebleed, and chrome holes.

Lehmann [10] in 1932 reported the first cases of lung cancer in workers employed in a chromate plant in Germany. The lung cancer occurred in two of "several hundred" workers. The first report in the United States on the incidence of cancer of the respiratory system among chromate workers was that by Machle and Gregorius in 1948. [11] In this mortality study of employees from seven chromate plants with mixed exposures to trivalent and hexavalent chromium compounds, the crude death rate (ie, the death rate not adjusted for age) for lung cancer was 25 times greater than normal. This investigation was

followed by others establishing an increased risk for lung cancer in workers in chromate plants. [3,12-16]

Effects on Humans

Chromium is a naturally occurring trace element found in human tissues. Imbus et al [17] reported normal levels of 2.65 μg/100 g of blood and 3.77 μg/liter of urine. Schroeder et al [18] reported the normal level in adult tissue in the United States to be 2.3 μg/g of ash in the kidney and 1.6 μg/g ash in the liver. Levels were higher in persons from other countries. The element was found in relatively high concentrations in all tissues of newborns (51.8 μg/g ash and 17.9 μg/g ash in the kidney and liver, respectively) but the concentration fell during the first two decades of life and was stable thereafter, except in the lungs. [18,19] In the lungs, the chromium level was reported to be 85.2 μg/g ash in infants. This decreased to a low of 6.8 μg/g ash during the second decade, after which the reported level gradually rose to 38.0 μg/g ash in the 70-80 year age group. [18] Chromium affects glucose and lipid metabolism in animals [19,20] as well as in man, [18,19] and is an essential micronutrient in mice and rats. [19]

In workers occupationally exposed to mixed chromites and chromates in the chromate producing industry, the U.S. Public Health Service [3] reported median blood chromium levels of 0.004 and 0.006 mg/100 ml blood for white and black workers, respectively. No overall mean or median was reported. Median urine levels of 0.043 and 0.071 mg/liter, respectively, were reported for white and black workers.

Among similarly exposed production and maintenance workers, Mancuso [21] reported the average urinary level was 5.1 µg/100 ml. Office workers surveyed in his study were reported to have average urinary chromium levels of 1.0 µg/100 ml. Not unexpectedly, urinary levels were higher among men exposed to soluble than those exposed to insoluble chromium compounds. Elevated levels of blood and urinary chromium were found to persist for several years after occupational exposure ceased. [21]

Chromium compounds can be allergens and are encountered in many occupations. [22,23] In one study [24] of dermatitis in an automobile assembly plant, 24.3% of those with dermatitis were found to be sensitive to chromate apparently carried over on the surface of nuts, bolts, and screws from a chromate dip. Dermatitis has been reported in the chromate producing industry [3,21] as has pulmonary sensitization. [21,23] Reports [7-9,25-27] of exposure to chromic acid, however, indicate problems not of sensitization, but rather of direct corrosive action. While there is no evidence that chromic acid workers under current conditions of industrial exposure have any appreciable risk of skin or pulmonary sensitization, these responses have been reported in workers exposed to other hexavalent chromium compounds. Though evidently rare, skin or pulmonary sensitization from exposure to chromic acid should be considered a possibility.

Careful review of the literature reveals no known cases of death caused by acute exposures. The atmospheric concentration immediately hazardous to life is not known. Acute pulmonary complications in two

workers exposed to massive amounts of chromic acid mist were reported in 1950 by Meyers. [28] The exposure levels of chromic acid were not measured by the investigator. One worker was exposed to the chromic acid mist for approximately four days while concentrating chromic acid by boiling the acid in large vats. The first symptoms were coughing and wheezing, followed by severe frontal headaches, dyspnea, pain on deep inspiration, fever, and loss of weight. After six months the worker had improved with respect to weight and cough but still had chest pains on deep inspiration. A bronchoscopic examination 6 months after exposure "revealed that the tracheal mucosa and the mucosa of the entire tracheobronchial tree was hyperemic and somewhat edematous." Eleven months after exposure, the worker still had complaints of infrequent chills, cough, and mild pains located in the anterior part of the chest.

The second worker, though working at the same operation, was exposed for only one day. He stated that he had no immediate ill effects from inhalation of the mist, but during the following three or four days hoarseness developed, with a cough productive of whitish mucoid sputum. A chest X-ray, hematologic studies, and urinalysis produced no abnormal results, but during the following three months the patient became anorexic and noted a gradual loss of 20 to 25 pounds. He was admitted to the hospital 4 1/2 months after exposure because of sharp pain at the right upper abdominal quadrant, cough, and severe, sharp chest pain, precipitated and aggravated by his chronic cough. This cough was productive of heavy, thick, greenish

mucoid sputum. He had had pneumonia on three occasions. On admission, he had a pleural friction rub. After 15 days in the hospital on a 3,500-calorie diet, he was discharged. At that time the chest was clear; the friction rub could not be palpated, nor was it heard. Approximately six months after exposure an X-ray indicated some emphysematous changes, and bronchoscopy seven months after exposure showed mucous pearl secretions. Eleven months after exposure, a persistent cough productive of greenish mucoid sputum remained.

Exposure to chromic acid in electroplating operations has been reported to cause a variety of adverse effects. These include lacrimation, [8,25] inflammation of the conjunctiva, [29] nasal itch and soreness, [8,25,27] epistaxis, [9,25,27] ulceration [8,9,25,27,29,30] and perforation [8,9,25,27,30] of the nasal septum, congestion of the nasal mucosa [8,9,25] and turbinates, [25] chronic asthmatic bronchitis, [29] dermatitis [30] and ulceration of the skin, [8,9,27,30] inflammation of the laryngeal mucosa, [29] cutaneous discoloration, [8] and dental erosion. [27] According to some reports, [8,9] ulceration of the skin occurs only when the plating solution comes in direct contact with the skin at the site of a pre-existing break in the skin.

Reviewing the effects of exposure to chromic acid mist from anodizing solutions, Zvaifler [26] considered the effects distinctly different from those in chrome plating operations, although others [30] report the effects are identical. Zvaifler [26] described the

cases he observed (over 100 cases) as ranging from superficial greyish ulceration of the anterior nasal mucosa with small spots of bleeding, to general involvement of all the nasal mucosa with superficial scabbing and crusting and a general dry appearance of the nose, sometimes extending to the posterior pharynx.

Pascale et al [31] in 1952 reported five cases of hepatic injury apparently due to exposure to chromic acid from plating baths. A person, who had been employed five years at a chromium plating factory, was hospitalized with jaundice and was found to be excreting significant amounts of chromium. Her lungs and cardiovascular system were normal. A liver biopsy showed histological changes resembling those found in toxic hepatitis. To investigate the possibility that the liver damage was of occupational origin, eight fellow workers were screened for urinary chromium excretion. Four of these were found to be excreting significant amounts and were examined in more detail. In three workers who had been exposed to chromic acid mists for 1 to 4 years, liver biopsies and a series of twelve hepatic tests showed mild to moderate abnormalities. No liver biopsy was taken from the fifth worker, who had been removed from further exposure because of nasal ulceration after 6 months at the plating bath. Only one of his liver function tests indicated a borderline abnormality. The urinary excretion of chromium (2.8 and 2.9 mg/24 hours) by the two workers employed four years was greater than the excretion (1.48 mg/24 hours) by the worker employed five years who suffered the greatest liver damage. The lowest urinary chromium excretion (0.184 mg/24 hours) was

measured in the fifth worker, the individual with least exposure. All five exhibited some signs of damage to the nasal mucosa. This plus the levels of urinary excretion suggests that exposures were significant, but no environmental data were reported.

Epidemiologic Studies

No epidemiologic data are available on the incidence of pulmonary cancer in workers exposed only to chromic acid. The epidemiologic data that are available pertain to workers in the chromate-producing industry. These workers were subject to mixed exposures, and these data have only indirect and limited application to chromic acid exposures.

The first report of lung cancer from exposure to chromium was given in 1932 by Lehmann. [10] He reported two cases of workers with lung cancer out of several hundred workers who had been employed in a chromate plant in Germany. No information was given on the length of exposure or on the nature and airborne concentration of the exposure to chromium compounds. Lehmann did not consider these two cases to be occupationally related.

Machle and Gregorius [11] gave the first report on the incidence of cancer of the respiratory system in the chromate industry in the United States. The workers had been exposed to chromite ore and a mixture of trivalent and hexavalent chromium compounds. Available records from seven chromate plants for the preceding 10-15 years (1933-1948) were studied. Of the 193 deaths in all plants, 66 (34.2%) were due to cancer of any type or at any site, a rate over twice that

for a control industrial group. This increase was attributable to an excessive proportion of deaths from cancer of the respiratory system. Lung cancers comprised 60% of all cancers as compared to an expected rate of 9%. In five of the seven plants, (no deaths due to lung cancer were recorded in two plants) lung cancer rates varied from 13 to 31 times normal. The mean duration of exposure prior to onset was 14.5 years.

One plant (plant C in Machle and Gregorius [11]) with no cancer deaths was small and no deaths from any cause were seen among its workers during the period covered. The second was one of two plants (D1 and D2 [11]) in the study owned by a single company. In plant D2 there were 33 deaths in 1,853 male-years (a term used by the authors to indicate that only males were included in the group studied) of exposure. Four of the 33 deaths (12.1%) were cancer deaths; none were cancer of the respiratory system. In contrast, in plant D1 there were 29 deaths in 2,491 male-years of exposure, of which five were due to lung cancer. These five deaths represented 17.4% of all deaths, or 71.4% (5 of 7) of all cancer deaths.

The worker populations of plants D1 and D2 were comparable with respect to age distribution, exposure history, color, and geographic location. The two plants differed significantly in the incidence of nasal irritation and septal perforation. These complaints occurred in 53.4% of the workers in plant D1, but only in 29.6% of workers in D2. The authors considered this a difference in degree only and stated that perforations may occur without associated high rates for lung

cancer. There was a distinct difference in the compounds processed by the two plants. Plant D1 produced sodium bichromate from chromite ore. Plant D2, in which there was no lung cancer, produced chromic acid and basic chromic sulfate from the sodium bichromate. The authors concluded that the experience in these plants suggested the monochromates may be responsible for the lung cancer.

Baetjer [32] in 1950 reviewed the literature reporting lung cancer cases attributable to chromate exposure. At that time, 122 cases of respiratory cancer had been reported. Of these, 109 worked in the chromate-producing industry, 11 in the chrome pigment industry, and 2 in other industries. Sixty-three cases occurred in Germany, 57 in the United States, and 1 each in Switzerland and England. The average duration of employment in the German chromate-producing group was 22 years. The average for the United States cases was 16 years, and the average in the German pigment cases was 12 years.

Although the best available data had been used, the Machle and Gregorius report [11] had been limited since in it the cause of death often was based on clinical findings alone. Furthermore, it did not include any workers who left the industry prior to developing lung cancer, and the control groups were not comparable in all respects to the chromate group. To overcome these objections, Baetjer [16] in 1950 reported an investigation of the records of two hospitals in Baltimore, where a chromate-producing plant was located. In the records from 1925 to 1946 of one hospital, there were 198 cases of men with lung cancer which was histopathologically confirmed with biopsy

or autopsy material. In the second hospital's records, from 1930 to 1948, there were 92 such cases. Two control groups were chosen from the records of the first hospital. One of these (226 cases) was selected from all men admitted for 10 days or more, excluding those admitted for traumatic injuries or psychiatric illness. This group, like the lung cancer group, chose to come or was referred to the hospital for more or less serious illness. The second control group (177 cases) consisted of males with cholelithiasis, chosen because this disease, like lung cancer, presents diagnostic problems. From the records of the second hospital, only the first control group was selected (499 cases). All control groups were selected to have the same age and yearly distribution as the cancer cases, and no case was included in more than one group.

The number of chromate workers among the lung cancer patients was compared to the number in the control groups. None of the patients in the control groups had a reported exposure to chromium compounds. Seven (3.5%) of the 198 lung cancer victims at the first hospital and 3 (3.3%) of the 92 at the second hospital were or had been chromate workers. Statistical analysis indicated that the percentage of lung cancer patients who had been chromate workers was significantly higher than expected on the basis of the control groups. The percentage of lung cancer patients who were employed at the chromate-producing plant was compared with the percentage of the employed male population of Baltimore who were employed at the plant. Statistical analysis again indicated that the percentage of chromate

workers in the lung cancer series was significantly higher than the percentage of chromate workers in the employed male population of Baltimore. This study therefore confirmed the earlier conclusions of Machle and Gregorius [11] that the number of deaths due to cancer of the lungs and bronchi was greater in the chromate-producing industry than was normally expected.

Mancuso and Hueper in 1951 [12] reported on a study of occupational cancer in workers in a chromate plant. The workers were exposed to a mixture of trivalent and hexavalent chromium compounds including chromic acid. Of 33 deaths from all causes, nine (27.2%) were from all types of cancers. Six of these (18.2% of all deaths) were from cancer of the respiratory system. The mean latent period was 10.6 years. In comparison, out of 2,931 deaths in Lake County, Ohio, in which the plant was located, 34 were due to lung cancer. The ratio of lung cancer to total deaths in the chromate plant was 17 times that of Lake County.

This was followed with a report by Mancuso [21] on the clinical and toxicologic aspects of 97 workers examined in a chromate-production plant: 63% showed perforations of the nasal septum or ulcers of the mucosa, 87% had chronic rhinitis, 42% had chronic pharyngitis, 10% had hoarseness, and 12% had polyps or cysts. Thirty-seven percent of the 97 examined had some involvement of the nose, throat, and sinuses.

A total of 17.5% of those given gastrointestinal X-ray examinations had evidence or ulcers, gastritis, or gastrointestinal tumor.

In comparison, X-ray examinations of a group of cement workers showed that 4 of 41 (9.8%) had similar evidence. The author stated "workers of a chromate factory seem to have an excess liability to inflammatory and ulcerative conditions of the gastrointestinal tract.caused by the ingestion of chromates." Any relevance of these data to workers exposed only to chromic acid would, however, be speculative. Neither the sex of the workers nor the years of work in the industry by departments was defined. In the nine production departments of this plant, air concentrations of total chromium expressed as chromium trioxide, based on weighted average 8-hour exposures, ranged from 0.17 to 3.12 with a median value of 1.0 mg/cu m. [2]

Baetjer et al [33] in 1959 reported on the analysis for soluble and insoluble chromium of lung tissue from 16 men who had worked 1.5 to 42 years in a chromate plant. Eleven of these were lung cancer victims. The range of concentrations for exposures to both soluble and insoluble chromium for the workers was stated to be great. The concentrations of soluble (water soluble and acid soluble) chromium in the lungs ranged from 0.54 to 42.4 mg/10 g lung tissue ash. For acid insoluble chromium the concentrations ranged from 0.0 to 148.2 mg/10 g lung tissue ash. One worker who had been out of the industry for 23 years (whose exposure to chromium compounds was estimated as "heavy" during his two years in the industry) had 3.3 mg soluble chromium/10 g lung tissue ash. In this same worker, the urine contained no chromium; in 100 g of blood, analysis showed 5.4 µg in the cells and 2.0 µg in the plasma. No correlation was found between the presence

or absence of lung cancer and the concentration either of soluble or of insoluble chromium. The concentration varied greatly from one lobe to another and even in different areas of the same lobe. The bronchogenic cancers often contained little or no chromium.

The Division of Occupational Health, Public Health Service, published a report in 1953 of a study on the health of 897 workers in the chromate-producing industry. [3] Approximately 1,800 samples were collected throughout the industry for the purpose of defining the atmospheric environment. The great majority of these were air samples, but material and settled dust samples were also collected.

It was found that the milling, roasting, and leaching processes generated dusts containing chromite ore, soda ash, roast, residue, and sodium chromate. Sodium bichromate and sodium sulfate were usually found associated only with the neutralizing, treating, and concentrating operations. An appreciable portion of the total chromium was present in an acid soluble-water insoluble state, indicating the presence of a form or forms of chromium which were dissimilar from either insoluble chromite ore or water soluble, hexavalent chromium. Roasting, leaching, neutralizing, and treating operations had the largest proportion of acid soluble-water insoluble chromium. However, settled dust samples from many areas not associated with roast or residue processes had high percentages of acid soluble-water insoluble chromium.

From the six chromate-producing plants, employing about 935 persons, 897 males were medically examined. [3] The study produced no

data to show that exposure to the chrome compounds affected the rate of dental caries. Some of the workers, however, developed a yellowish discoloration of the teeth and tongue. A higher percentage of the chromate workers experienced gingivitis and periodontitis. Pulmonary markings suggestive of fibrosis were not significant among chromate workers but bilateral hilar enlargements were observed. No correlation could be established between prevalence of heart disease and years in the chromate industry. White and red blood cells and casts in urine appeared more frequently than is usually observed in the average industrial population. These findings tended to increase with increasing years of exposure in the chromate-producing industry.

Perforation of the nasal septum was found in 509, or 56.7% of these chromate workers. It was reported that septal perforation sometimes occurred after less than six months of exposure. Other workers with years of heavy exposure did not experience perforation, apparently because of prophylactic measures. The use in combination of a mask, petrolatum in the nostrils, and nasal douching was judged to be the most effective protection. [3] The authors concluded that the prevalence of nasal perforation was not a valid index of the prevalence of pulmonary carcinoma.

Ten of the 897 chromate workers examined were diagnosed as having bronchogenic carcinoma (3 of the 10 had been diagnosed before the survey). [3] The mean age of these 10 workers was 54.5 years and the mean exposure to chromate 22.8 years. A survey of a comparison group from a chest X-ray survey in Boston showed 20.8 lung cancer

cases per 100,000 people, whereas the rate for bronchogenic cancer among chromate workers was 1,115 per 100,000 or 54 times that of the control group. Comparing the morbidity and mortality experience of male members of sick benefit associations in seven chromate producing plants, cancer of the respiratory system was found to be 29 times the rate for all males in the United States.

Taylor in 1966 [13] reported on a study of a group of chromate workers followed over a period of 24 years (1937-1960) using Old-Age and Survivors Disability Insurance records. The workers were exposed to multiple trivalent and hexavalent chromium compounds. A total of 1212 chromate workers were included in the study. For respiratory cancer 8.344 deaths were expected, 71 deaths were observed; for all other cancers 23.894 deaths were expected, 32 observed; for respiratory diseases 7.843 deaths were expected, 19 observed. Expected deaths from the selected causes were determined from the age-cause specific mortality rates for the U.S. civilian male population. No data were presented on levels of worker exposure to chromates.

All of the preceding reports [3,11-13,16,32] of lung cancer in the United States chromate-producing industry have varying degrees of overlap. At the time of the earliest report, [11] there were seven chromate plants in the United States, all of which were included in that study. The later reports used different methods, but included all or some of the same plant populations and cases reported by the other studies. Therefore, these reports do not all represent different worker populations.

Bidstrup [14] in 1949 interviewed and X-rayed 724 chromate workers in Great Britain and discovered one case of lung cancer. Bidstrup and Case [15] reported a follow-up study of the remaining 723 workers, conducted almost six years after the first study. In the follow-up, it was found that 217 workers had left the industry and were lost to the follow-up. A total of 59 men were known to have died, 12 of these by lung cancer. This compared to 3.3 expected lung cancer deaths, or an incidence of 360% of expected. The difference was statistically significant, but as the authors pointed out, by the time all the men at risk have lived their life span, the lung cancer increase probably will be found to be very much higher. The possibility that the increase was due to nonoccupational factors such as diagnostic bias, place of residence, social class, or smoking habits was examined and discarded. It was not possible to form an opinion about the identity of the occupational carcinogen.

The chromate workers in the above studies [3,11-16,32] had exposures to a mixture of trivalent and hexavalent chromium compounds of which chromic acid was only a minor part. The workers were exposed to chromite ore, chromite-chromate intermediates and chromates as well as trace metals and minerals associated with the processing of the chromite ore. These studies suggest that exposure to the roasted chromite ore complex may be important as a causative agent of the lung cancer observed in chromate workers.

In the literature, there is no direct evidence that exposure to chromic acid per se at the measured concentrations and under the

conditions of industrial exposure has led to cancer. However, no study of this nature has been undertaken. More definitive data are needed on this subject.

Bloomfield and Blum [9] in 1928 reported on a study of health hazards in chromium plating. In the study 19 workers were examined who had worked from one week to three years with exposure to chromic acid in the chromium plating room. Of the 19 workers examined, 17 had inflamed mucosa, 11 nosebleed, 6 chrome holes, 4 ulcerated septa, and 3 had perforated septa.

In 1930 the Inspectorate of Factories, London, issued a report on the examination of 223 workers engaged in chromium plating. [30] Ninety-five (42.6%) had dermatitis or scars of old ulcers, 116 (52%) had either perforation or ulceration of the septum or devitalization of the mucous membrane. Ulceration of the nasal mucous membrane was seen as early as two weeks after exposure. No data were given on the atmospheric concentration of chromic acid to which the examined workers were exposed.

In 1944 Zvaifler [26] reported on the study of over 100 cases of workers exposed to chromic acid mists from a 5.0% chromic acid solution used in anodizing operations. Atmospheric concentrations of chromic acid were not given. Neither the length of time of exposure nor the sex of the workers was given in the cases discussed. The author stated that cases of chromic acid poisoning from anodizing operations (using a 5% solution of chromic acid) are quite different

from those resulting from chromium plating and are largely limited to the nasal mucosa though skin rashes are common.

Vigliani and Zurlo in 1954 [29] reported on a study of exposures to a variety of agents in the working environment and their effects on the workers for validating safe exposure levels. In a group of 150 workers exposed to chromates and chromic acid from electrolysis baths and during the production of chromic acid anhydride and alkali chromates, the investigators reported an ulcer of the nasal septum, inflammation of the larynx and ocular conjunctiva and chronic asthmatic bronchitis among the workers, one cancer of the nasal septum and one lung cancer. The average concentration of chromates at the time of the investigation was stated to range from 0.11 to 0.15 mg/cu m.

In 1965 Kleinfeld and Rosso [25] reported on a study of nine cases of workers with injury to the nasal septa from exposure to chromic acid while engaged at chromium plating. Atmospheric concentrations of chromic acid ranged from 0.18 to 1.4 mg/cu m. The plating tanks were not provided with local exhaust ventilation. General room ventilation was provided through the use of room fans and opened windows. Four of the nine workers examined with exposure times ranging from 2 to 12 months had perforated nasal septa, three workers with exposure times ranging from 1 to 10 months had ulcerated nasal septa, and two workers with exposure times of 0.5 and 9 months, respectively, had moderate injection of the nasal septa. The air

sampling was done at the breathing zone level near where the worker stood.

In 1972 Gomes [27] made a study of the incidence of cutaneous-mucous lesions in workers exposed to chromic acid in the State of Sao Paulo, Brazil. He found that only 50% of the industries used exhaust protection and that the threshold limit for workers in electroplating with hot chromic acid was frequently surpassed. Clinical examination of the 303 workers exposed to chromic acid revealed that 24% had perforated nasal septa and 38.4% ulceration of the same. Together, these lesions of the nasal septum affected more than 50% of the workers. More than 50% of the workers examined showed ulcerous scars not only on the hands, but also on forearms, arms, and feet. Ulcerous scars on the feet were due to working without boots and the wearing of Japanese type sandals.

Animal Toxicity

In order to study in animals the reported cancer hazard due to chromium, Hueper [34] attempted to identify a species and tissue sensitive to the carcinogenic action of chromium or its compounds. Chromium and chromite ore were introduced in a powdered form suspended in two different vehicles (lanolin, gelatin) by various routes (in the femur, intrapleural, intraperitoneal, intravenous, intramuscular, intranasal sinus) into mice, rats, guinea pigs, rabbits, and dogs. Results were equivocal at best as to evidence supporting a carcinogenic action of metallic chromium and chromite ore. Only in

rats were tumors observed which might have been causally related to the chromium deposits.

Subsequently, Hueper [35] implanted chromite ore roast mixed with sheep fat into the thigh muscle tissue and into the pleural cavity of male rats. Of the 25 rats with pleural implants 2 developed squamous-cell carcinomas coexisting with sarcomas of the lung and 2 developed tumors (one of which was benign) remote from the site of implantation. Of the 31 rats receiving implants in the thigh muscle, 3 had fibrosarcomas of the thigh and ten developed tumors (four of which were benign) remote from the site of implantation. Two series of 15 female rats each were implanted with sheep fat only into the pleural cavity and into the thigh, respectively, as controls. Of those with pleural implants, one developed a benign tumor at the site and three developed tumors (2 benign) remote from the site of implant. In the series with thigh implants, three developed tumors (one benign) remote from the implant. This suggested to the investigator that the chromite ore roast contained carcinogenic material, possibly the water insoluble-acid soluble chromium compounds present.

Hueper and Payne [36] implanted finely pulverized calcium chromate, sintered calcium chromate, sintered chromium trioxide and barium chromate mixed with sheep fat into the pleural cavity and into the thigh muscle of rats. Of 20 male and 15 female rats in each series, rats implanted with calcium chromate developed 8 thigh tumors and 21 pleural tumors; rats implanted with sintered calcium chromate developed 8 thigh tumors and 17 pleural tumors; rats implanted with

sintered chromium trioxide (some of which had been changed to lower valency during sintering at 2000 F for one hour) developed 15 thigh tumors and 14 pleural tumors. The rats implanted with the barium chromate (low degree of solubility) did not develop any tumors either in the thigh muscle or the pleural cavity. With one exception, all tumors were sarcomas, usually of the spindle cell- or fibro-sarcomatous type. Two groups of 20 male and 15 female rats were implanted with pellets of sheep fat only into the plural cavity and into the thigh, respectively. No tumors were observed in any of these control animals. The data suggested to the investigators that several chromates with medium solubility produce cancer when introduced into the tissues of rats in the form of a depot assuring prolonged exposure to chromium in rather small amounts.

Payne [37] injected calcium chromate, sintered calcium chromate, and sintered chromium trioxide in a tricaprylin vehicle subcutaneously into the nape of the necks of mice. Only one tumor was observed in 52 mice injected with calcium chromate. No tumors were seen in the groups of 52 mice each which received sintered calcium chromate, sintered chromiun trioxide, or in the control group. When calcium chromate and sintered calcium chromate in sheep fat were implanted intramuscularly in mice, nine tumors were seen in mice implanted with the sintered calcium chromate, and only one in those receiving calcium chromate. The tumors observed were of the same type as those reported earlier by Hueper and Payne. [36] The author concluded that the carcinogenic action of chromium was dependent on the solubility of the

compound and the amount present, stating that if hexavalent chromium in the form of chromate ion is available in too large a dose, acute effects result, but that a smaller dose can result in malignancy.

These results and conclusions were corroborated by Roe and Carter [38] who injected rats intramuscularly with calcium chromate in arachis oil. Twenty once-weekly injections were given. The first two injections contained 5.0 mg of calcium chromate, but signs of severe local inflammation developed, so the dosage in the last 18 injections was 0.5 mg. Of 24 test rats, 11 developed spindle cell sarcomas and seven developed pleomorphic sarcomas at the injection site. No tumors were seen in 16 controls.

Laskin et al in 1969 [39] reported studies of selected chromium compounds in a cholesterol carrier using an intrabronchial implantation technique. Compounds under investigation included chromic chromate, chromic oxide, chromium trioxide, calcium chromate, and process residue. Pellets were prepared from molten mixtures of materials dispersed in equal quantities of cholesterol carrier. These studies included materials of differing solubilities and valences, and have involved over 500 rats that were under observation for periods of up to 136 weeks.

Lung cancers that closely simulate lung cancer in man were found in these studies. With the calcium chromate, eight cancers were found in an exposed group of 100 animals. Six of these were squamous cell carcinomas. In all the experimental groups except the one exposed to chromium trioxide, there was evidence of atypical squamous metaplasia

carcinomas. In all the experimental groups except the one exposed to

of the bronchus. In the 100 rats implanted with chromium trioxide, two tumors were observed, both hepato-cell carcinomas.

Since these studies implicated calcium chromate as a lung carcinogen, inhalation studies using this compound were begun. [40] The study is not yet completed, but preliminary results suggest a carcinogenic action in rats after chronic exposure to aerosols at a concentration of 2.0 mg/cu m. These results may be significant for the human experience in the chromate-producing industry. As noted by this researcher, [40] calcium chromate exists in the residue step to the extent of 3% in no-lime roasts and at significantly higher levels when lime is used.

Correlation of Exposure and Effect

Only five studies are available which report both the effects in man of chromic acid exposure and atmospheric levels of chromic acid. [9,25-27,29,41] All these reported the atmospheric levels as measured at the time of the study. Consequently, all share a common weakness, in that effects were reported which were cumulative effects of past exposures to chromic acid concentrations which may have been different from the levels reported. Nevertheless, limited correlations can be drawn.

In the study by Bloomfield and Blum, [9] six plating plants were surveyed and the atmospheric concentration of chromic acid was determined in each, based on a total of 39 air samples. Using these data and the occupational histories of the workers, the investigators estimated the amount of chromic acid to which some workers were

exposed daily during the time employed in the plating room. When the worker had been employed only a short time, "the estimated degree of exposure was more than an approximation" in the authors' opinion, since the ventilation system in use at the time of the survey had been in use throughout the individual's employment.

Exposures were estimated for 23 workers who were given physical examinations. Four of these were controls with no known exposure to chromic acid. Estimated exposures for the remaining 19 ranged from 0.12 to 5.6 mg/cu m. Six platers were exposed to chromic acid estimated at a level of 0.12 mg/cu m. Employment had ranged from one week to seven months. All had inflamed mucosa and three had nosebleed. The exposures in the past may have been different from those observed at the time of the study, but the data do indicate that distinct injury to the nasal tissues can result after relatively short exposures. Some of these six platers were exposed such a short time that their experience strongly suggests that, assuming an accurate estimate was made, a concentration of 0.12 mg/cu m can cause inflammation of the nasal mucosa and nosebleed. This was the conclusion of the authors, [9] who stated that continuous daily exposure to concentrations greater than 0.1 mg/cu m is likely to cause definite injury to the nasal tissues.

Kleinfeld and Rosso [25] studied a group of chromium plating workers exposed to airborne chromic acid levels ranging from 0.18 to 1.4 mg/cu m. The exposure period varied from 2 weeks to 12 months. Each of the workers studied had either perforated or ulcerated septum

or injection of the septum. These data again indicate that lengthy exposures are not necessary for adverse effects to be manifest, since septal perforation was reported after as little as two months exposure. The data do not suggest a safe exposure level.

The report by Vigliani and Zurlo [29] did not detail the frequency with which effects were observed, but reported on a three year observation of approximately 150 workers during which average concentrations were 0.11-0.15 mg/cu m. Exposures were to chromic acid mist and anhydride, and to alkali chromates. Health disturbances reported included ulceration of the nasal septum, inflammation of the laryngeal mucosa and conjunctiva, and chronic asthmatic bronchitis. Two cases of cancer were also observed, one a cancer of the nasal septum and one lung cancer. Vigliani and Zurlo recommended that the standard for chromic acid be reduced from 0.1 mg/cu m to 0.05 mg/cu m.

Gomes [27] reported the experience of electroplaters in the State of Sao Paulo, Brazil. The exposures of 81 platers were determined as representative of the exposures of 303 platers examined clinically. Air concentrations were determined using a universal testing kit with syringe-type pump and filter paper. Unfortunately, a direct correlation between those exposed to a given concentration and those free of symptoms cannot be made, but the results are nevertheless indicative of the level at which effects are observed. A total of 43.2% of the workers were exposed to atmospheric concentrations of 0.1 mg/cu m or less, yet only 37.6% of the workers examined were free of nasal ulceration and perforation. The incidence

of cutaneous lesions due to direct skin contact was greater due to improper use of or failure to use protective equipment. The author made no inferences from his data as to a safe exposure level. The percentage of the workers exposed to levels at or below 0.1 mg/cu m and the percentage free of respiratory symptoms are nearly the same, suggesting that threshold effects occur at or below this level.

In the reports by Zvaifler [26] and Gresh [41] of chromic acid exposures in an anodizing plant, a direct correlation again is not possible. However, in this case, a better estimate of changing conditions is possible. Nasal irritation and ulceration were observed in workers, and atmospheric concentrations were reported to range from 0.42-1.2 mg/cu m. After the ventilation system was improved, atmospheric levels in two samples were 0.09 and 0.10 mg/cu m. Four weeks later nasal irritation persisted, although none had worsened. After further revision of the ventilation system, atmospheric concentrations were reduced to negligible levels and the employees' nasal irritation cleared up within four weeks. Had exposure at 0.09-0.10 mg/cu m continued longer than four weeks the irritation may have cleared up eventually, but these data suggest again that effects are seen at 0.1 mg/cu m.

Because these reports all fail to give long-term environmental data, the effects observed cannot be directly related to the environmental data reported. Nevertheless, the five papers consistently illustrate that adverse effects can result after relatively short periods of employment and therefore short periods of exposure to

chromic acid. The papers also consistently indicate that nasal irritation does occur at atmospheric concentrations of 0.1 mg/cu m and may occur at lower levels.

IV. ENVIRONMENTAL DATA AND ENGINEERING CONTROLS

Measurements of atmospheric concentrations of chromic acid around industrial operations before and after controls were instituted attest to the marked effect of controls in lowering the airborne levels of this contaminant. In 1928, Bloomfield and Blum [9] studied six plating plants with varying degrees of ventilation control and changing operating conditions. In one plant, concentrations were as high as 6.9 mg of chromic acid per cubic meter of air with no ventilation in use while plating with a current density of 300 amperes per square foot. However, at the same current density but with ventilation operating at an air velocity of 1700 feet per minute at the face of the slot, there was no detectable chromic acid in the workers' breathing zone. In order to insure a reasonable safety factor, they recommended a lateral slot-type exhaust system operating at an air velocity of 2,000 fpm at the face of the slot, drawing air no more than 18 inches laterally. They also recommended that the exhaust slots be flush with the top of the tank and that the plating solution be at least 8 inches below the top of the tanks to allow ample time for the mist to be directed to the exhaust slot.

Sampling in the approximate breathing zone of workers plating at a tank measuring 20 x 4 feet with a current density of 150-200 amperes/square foot of surface, Riley and Goldman [42] reported an atmospheric concentration of 3.68 mg chromic acid/cu m with good general ventilation but no local exhaust ventilation. With the local exhaust (two slots, one on each of the long sides of the tank)

operating at a face velocity of 600 feet per minute (approximately 37.5 cubic feet/minute/square foot of tank surface), the atmospheric concentration was reduced to 1.12 mg/cu m. When the face velocity was 1800 fpm (approximately 112.5 cfm/sq ft), the atmospheric level was further reduced to 0.034 mg/cu m. Reiterating and confirming the earlier recommendations of Bloomfield and Blum, [9] the authors considered the capacity of the system a better criterion than face velocity, and recommended a cross-draft exhaust system operating with a control velocity of 100 fpm.

In 1944, Gresh [41] measured the effectiveness of ventilation in controlling chromic acid mist at an anodizing tank in which the solution was constantly agitated by an air line through the solution. Although equipped with an enclosed hood, ventilation at a rate of 148 cfm/sq ft of tank area reduced the atmospheric concentrations of chromic acid only to 0.09 mg/cu m. When a lateral exhaust system was installed, ventilation at 134 cfm/sq ft produced negligible atmospheric concentrations of chromic acid. The exhaust stacks on the roof of the plant were intended to be sufficiently high to permit dissipation of the mist, but nasal irritation of workers in other areas persisted. After moisture collectors were added to the ventilation system, tests indicated no emissions of chromic acid to the atmosphere from the exhaust stacks. After four weeks, the nasal irritation of the workers subsided. On this basis, the author recommended the inclusion of moisture collectors in ventilation systems to completely prevent escape of the chromic acid mist.

Kleinfeld and Rosso [25] in measuring atmospheric chromic acid concentrations at a chromium plating operation found levels ranging from 0.18 to 1.40 mg/cu m. The tanks had no exhaust ventilation. General room ventilation was provided by fans and opened windows. The ventilation rate was not reported, but installation of a local exhaust system reduced atmospheric concentrations of chromic acid to levels ranging from 0.003 to 0.009 mg/cu m.

Minimum design specifications for local exhaust ventilation of open surface tanks have been promulgated under the Occupational Safety and Health Act of 1970. Both the latest recommendations from the American National Standards Institute Z9.1-1971 [43] and the current OSHA regulations (29 CFR 1910.94, published in the Federal Register, vol 37, dated October 18, 1972, which were based on ANSI Z9.2-1960) call for ventilation at a control velocity of 150 fpm, with ventilation rates up to 375 cfm/sq ft, depending on tank size and hood type.

Control strategies beyond ventilation include the use of surface-active agents [44] and plastic chips. [44,45] By reducing the surface tension of the plating solution, the surface-active substances help to retard mist formation and carryover by the hydrogen bubbles generated during plating. Hama et al [44] reported values for chromic acid mist in the workers' breathing zone ranging from 0.002 to 0.06 mg/cu m in three plants in which only a fluorocarbon surface-active agent was in use. In a fourth plant in which ventilation was in use at a rate of 90 cfm/sq ft (the ventilation rate recommended at the

time for the tank was 175 cfm/sq ft), chromic acid was not detected in one sample and was 0.002 mg/cu m in a second sample. In the discharge stacks of three plants using both local exhaust ventilation and the fluorocarbon surface-active agent, chromic acid concentrations ranged from not detected to 0.02 mg/cu m.

Molos [45] sampled for chromic acid in a plant using floating plastic chips as a control measure. Samples were collected 8 inches above the level of the plating solution, the same level as the lateral exhaust slots. Chromic acid concentrations were 4.5-5.0 mg/cu m with ventilation off, and 0.02-0.05 mg/cu m with the ventilation system operating at 170 cfm/sq ft. In the workers' breathing zone, the concentration was 16.0-17.0 mg/cu m with only plastic chips in use. No breathing zone samples were reported for this plant with both plastic chips and local exhaust ventilation in use, but it was stated that "All tests, which were made under conditions where plastic chips were used as well as exhaust ventilation, showed no chromic acid concentration whatever in the workers' breathing zone." Molos concluded that a floating baffle of plastic chips is effective in reducing chromic acid mist and that they result in substantial conservation of chromic acid. Nevertheless, he considered local exhaust ventilation as necessary to ensure healthful working conditions, even when plastic chips are used.

V. DEVELOPMENT OF STANDARD

Basis for Previous Standards

The first standard for chromic acid (chromium trioxide) was published by the United States of America Standards Institute (now the American National Standards Institute) in 1943. [46] That standard specified a maximum allowable concentration of 1.0 milligram as chromic acid anhydride in 10 cubic meters of air for exposures not exceeding a total of 8 hours daily. This standard apparently was based on the 1928 report by Bloomfield and Blum. [9] Although there has been confusion about the meaning of some maximum allowable concentrations, whether a time-weighted average or ceiling was intended, the intent in this case apparently was for a ceiling of 1.0 mg/10 cubic meters.

In Manual of Industrial Hygiene and Medical Service in War Industries, published in 1943, [47] the USPHS listed the ANSI MAC of 0.1 mg/cu m for chromic acid.

In 1947 the American Conference of Governmental Industrial Hygienists adopted a Threshold Limit Value for chromic acid and chromates of 0.1 mg/cu m, which has remained unchanged since. The reports by the U.S. Public Health Service, [3] Bloomfield and Blum, [9] Machle and Gregorius, [11] Mancuso and Hueper, [12] Bidstrup, [14] Mancuso, [21] Kleinfeld and Rosso, [25] Vigliani and Zurlo, [29] and Baetjer [32] were considered when documentation for the TLVs was published in 1971. [48] The documentation states "A review of the present status of the suitability of the TLV between TLV subcommittee

members and industrial hygiene representatives of the chromate industry 10 years after improved controls had been in operation revealed that (1) the TLV for chromic acid mist was satisfactory in preventing nasal perforation; (2) contained a safety factor of three or four; and (3) that the limit was probably satisfactory for the prevention of lung cancer, as no new cases had appeared during the ten-year period; but (4) that the ten-year period was probably too short to be certain of its validity in this respect." [48] Data, however, were not presented to support points 1, 2, and 3.

The American Industrial Hygiene Association's Hygienic Guide on Chromic Acid, published in 1956, recommends a Maximum Acceptable Concentration (8 hours) of 0.1 mg/cu m for chromic acid. [49] Data from the same reports considered by the American Conference of Governmental Industrial Hygienists were used as the basis for the value.

The present Federal standard for chromic acid is a ceiling concentration not to be exceeded during any 8-hour period of 1.0 mg/10 cu m, (29 CFR 1910.93, published in the Federal Register, vol 37, dated October 18, 1972) based on the American National Standard Z37.7-1971 (year 1971 is in error, it should be 1943).

Basis for Recommended Environmental Standard

Industrial exposure to mixed chromite and chromate compounds has been shown to cause ulceration of the skin, [3,7,21] dermatitis, [3,22,24] ulceration and perforation of the nasal septum, [3,7,21,29] inflamed mucosa, [3,29] irritation of the conjunctiva, [3,7,29] and

cancer of the lung. [3,11-16,32] Other effects [21] reported as a result of mixed exposures include nasal mucosal polyps, chromitotic pneumoconiosis, chronic rhinitis, sinusitis, mucosal polyps and hydrops of nasal sinuses, inflammatory and ulcerative conditions of the gastrointestinal tract, and, often, an imbalanced ratio of the formed elements of the blood as well as lengthened bleeding time.

Occupational exposure to chromic acid has been shown to cause ulceration of the skin, [8,9,27,30] ulceration and perforation of the nasal septum, [8,9,25-27,30,31] inflamed or bleeding nasal mucosa, [8,9,25,26,28,31] and ulceration or congestion of the turbinates. [25,26] Erosion and discoloration of the teeth has been attributed to chromic acid exposure [27] as has discoloration of the skin. [8] Apparent liver damage has been reported, [31] but other reports have indicated there was no evidence either of hepatic or of renal damage after acute [28] and chronic [25] exposure. An increased incidence of lung cancer has not been found reported from exposure to chromic acid alone.

In one epidemiologic study [11] of seven chromate plants, it is suggested that the carcinogen is a monochromate found in the processing of the chromite ore. In that study, the crude death rate (ie, the death rate not corrected for age) from cancer of the lung was 25 times higher than normal, but all observed lung cancer deaths were confined to five of the seven plants. One plant was quite small and there were no deaths among its employees during the nine years surveyed. There were no lung cancer deaths in another plant which was one of two

plants in the study owned by a single company. The worker populations of the two plants were "similar with respect to age distribution, exposure history, color, geographic location, and were not greatly different in size." There was, however, an obvious difference in exposure, since one plant produced sodium bichromate from chromite ore, while the second plant produced chromic acid and basic chromic sulfate from the sodium bichromate. The incidence of death by lung cancer was 18 times normal in the plant producing sodium bichromate, while there were no lung cancer deaths in the plant processing the bichromate. Monochromates were suggested as the etiologic agent on the basis that the lung cancer was widely distributed in the first plant among all occupations entailing exposure to monochromates.

Thus, there is ample evidence that workers with mixed exposure in the chromate-producing industry have been at increased risk of lung cancer. [3,11-16,32] Unfortunately, no epidemiological study of workers exposed only to chromic acid has been undertaken. There is reason to suspect other chromium compounds as the carcinogens responsible for the increased lung cancer observed in chromate plants. The chromite ore itself has been suggested as the etiologic agent, [12] as have the monochromates, [11] and intermediate water insoluble-acid soluble compounds. [3] The animal studies by Hueper, [34,35] Payne, [37] Hueper and Payne, [36] and Roe and Carter [38] suggest that the etiologic agent is a moderately soluble chromate which can be slowly released from a tissue "reservoir" in amounts which are not sufficiently toxic to cause necrosis. Calcium chromate has been

implicated as a lung carcinogen by Laskin et al [39] and by Kuschner. [40] Hueper [50] has indicated the risk of cancer is negligible when chromic acid is used medicinally. This judgment was based in part on the "extreme rarity of such sequelae" to chronic ulcerative defects of the skin and nasal mucous membranes in workers having occupational contact with chromic acid mist and chromates. [50] Therefore, while there is no positive evidence that chromic acid in the workplace has contributed to an increase in cancer, neither is there definitive evidence that absolves chromic acid.

At least one report [31] has suggested that liver damage is a possible consequence of exposure to chromic acid. Other reports have indicated that neither hepatic nor renal involvement was observed after acute [28] and chronic [25] exposure. In the one report of liver damage, urinary excretion of chromium and the clinical findings of nasal ulceration or mucosal injection and hyperemia suggest significant exposures to chromic acid.

The 1928 report by Bloomfield and Blum [9] has served to a great extent as the basis for the previously recommended chromic acid standards of 0.1 mg/cu m. In that paper, the authors concluded that "Continuous daily exposure to concentrations of chromic acid greater than 1 milligram in 10 cubic meters of air is likely to cause definite injury to the nasal tissues of the operators." The lowest concentration to which chromium platers were estimated to have been exposed was 0.12 mg/cu m. Six platers were estimated to have been exposed to that level. One of these had been employed in the plating

room approximately one week and two approximately three weeks, yet all six platers suffered slightly (2 of 6) to markedly (4 of 6) inflamed mucosa. Three of these six, including the individual employed only one week, suffered nosebleeds. One plater who had been employed one year was estimated to be exposed to 2.8 mg/cu m at the time of the survey, but suffered no ill effects, apparently due to personal prophylactic measures. The mucous membranes can be protected, therefore, even against high concentrations of the mist. If the estimates were accurate, the experience of the six platers exposed to 0.12 mg/cu m demonstrates that adverse effects result fairly rapidly from exposures only slightly higher than 0.1 mg/cu m. Thus, the conclusion of the authors that damage is likely at concentrations above 0.1 mg/cu m seems less an endorsement of that as a safe exposure level, but rather an indication of the level at which adverse effects can be expected.

Zvaifler [26] and Gresh [41] in 1944 reported on over 100 cases observed in an anodizing plant. The majority of these involved superficial greyish ulceration of the nasal mucosa with engorgement of the vessels and small areas of bleeding in workers not directly associated with the anodizing tanks. Among those working directly at the tanks, the ulceration involved more of the septum, was deeper, and involved the turbinates and nasal septum as well as the mucosa. Atmospheric levels of chromic acid at the anodizing tank ranged from 0.42-1.2 mg/cu m. With increased ventilation, the atmospheric concentration at the tanks was reduced to 0.09-0.10 mg/cu m, but after

four weeks the worker's physical condition had not improved, although no worker's condition worsened. After a new ventilation system reduced atmospheric concentrations to negligible levels, the nasal irritation subsided.

After a three-year observation period in areas involving the preparation of chromic acid anhydride and alkali chromates, and use of electrolysis baths, Vigliani and Zurlo [29] reported inflammation and ulceration of the nasal mucosa, chronic asthmatic bronchitis, and inflammation of the conjunctiva in areas where air concentrations averaged 0.11-0.15 mg/cu m. These investigators concluded that the time-weighted average level of 0.1 mg/cu m should be lowered to 0.05 mg/cu m.

Kleinfeld and Rosso in 1965 [25] reported nine cases of nasal damage, ranging from moderate injection of the nasal septum and turbinates to septal perforation. Atmospheric levels at the time the workers were examined ranged from 0.18-1.4 mg/cu m. Atmospheric levels were determined during the summer and, according to the authors, levels probably would be higher in the winter. Nevertheless, these levels are indicative of the exposure of those employed only a short time. One person employed approximately two weeks had moderate injection of the septum and turbinates. Another employed one month had an ulcerated septum, while a perforated septum was observed in an individual employed only two months.

The most recent data available are those reported by Gomes. [27] For this survey of the electroplating industries, atmospheric levels

were determined for 81 workers to characterize the work environment. Of these 43.2% were exposed to atmospheric levels of 0.1 mg/cu m or less, but of the 303 workers who were examined clinically, only 37.6% were free of nasal ulceration and perforation, and only 13.3% were free of cutaneous lesions. The high incidence of cutaneous lesions reflects poor work practices and the low level of sanitary education pointed out by Gomes, since skin lesions apparently occur only on direct contact with the plating solution. [9,25,30] The percentage of workers with nasal ulceration or perforation is only slightly greater than the percentage of workers exposed to levels greater than 0.1 mg/cu m. The difference is not great enough to conclude that 0.1 mg/cu m definitely will result in damage to the nasal mucosa. It does demonstrate that 0.1 mg/cu m offers no margin of safety, since nasal ulceration and perforation apparently occur at this level.

Thus, there are reports [9,25,26,27,29,41] of nasal ulceration occurring at atmospheric concentrations only slightly above 0.1 mg/cu m. As a strong oxidizing agent, chromic acid can act in a short time, as evidenced by the short exposures necessary to cause ulceration or inflammation of the nasal mucosa. [9,25] Even very short exposures above 0.1 mg/cu m are likely to cause definite injury to the nasal tissues, so it is recommended that the current Federal standard of 0.1 mg/cu m as a ceiling concentration be retained.

The chronic effects reported, lung cancer [3,11-16,32] and liver damage, [31] have not been proved to be a result of exposure to chromic acid, but the possibility of a correlation cannot be rejected.

Without better data, it is not possible to establish with confidence what atmospheric concentration will protect against chronic effects if a correlation does exist. Nevertheless, because chronic effects are a possibility, it is recommended that the worker be afforded an additional factor of protection by supplementing the allowable ceiling of 0.1 mg/cu m with a time weighted average of 0.05 mg/cu m for an eight hour work day.

Special procedures are recommended any time the anhydride is being used, handled, or processed in other than fully enclosed operations. These recommendations include full protective clothing, respiratory protection, and eye protection. When handling the anhydride, contact with discrete particles is a distinct possibility, even if air sampling indicates an undetectably low atmospheric concentration. Because of its powerful oxidizing action, a single particle of chromic acid anhydride can cause ulceration of the skin or nasal mucosa, and severe damage to the eyes, so that these special precautions are necessary to adequately protect the worker.

Basis for Air Sampling and Analytical Methods

Two principal methods have been used to determine the concentration of chromic acid (Chromium VI) mist in air. Methods of collection have included absorption in distilled water and alkaline solutions, using impingers or sintered-glass bubblers, and filtration with absorbent paper. [3,48,51-53] Analytical methods have included titration of liberated iodine with standardized sodium thiosulfate solution, [51] colorimetry with hematoxylin [51] or s-diphenylcarba-

zide, [3,48,51,52,54] and field analysis by means of an impregnated filter paper, based on the colorimetric reaction between chromium and s-diphenylcarbazide and comparison with permanent standards.

Of the methods of collection, filtration offers the greatest collection efficiency and ease of collection of breathing zone samples. The AA type of membrane filter has a 0.8 micron pore size and provides a highly retentive matrix for particulates. The use of scrubbing liquids is inconvenient for personal breathing-zone sampling and is thus not recommended.

The iodide-thiosulfate method is subject to interferences from a wide variety of compounds with its nonspecific iodide reaction and the color definition is subject to a slight error. The hematoxylin method is suggested only as a check for very small amounts of chromium and is a visual colorimetric method. The use of the colorimetric field analysis technique involving a grab sample and visual analysis must be considered to be only semiquantitative, and useful only for that purpose.

The colorimetric diphenylcarbazide method does not react with trivalent chromium but produces a color with only the hexavalent form (present in chromic acid). However, cyanides, organic matter and other reducing agents, iron, copper, and molybdenum at concentrations above 200 ppm and vanadium above 4 ppm, interfere and must be separated or complexed before this method may be expected to provide chromic acid analytical data of an acceptable degree of accuracy and precision. [54]

The atomic absorption spectrophotometric method, applied directly, determines the total chromium and cannot make the desired distinction between the hexavalent chromium in chromic acid and the trivalent forms of chromium which may be present in the collected sample. Hence, it is necessary to separate the hexavalent from the trivalent chromium compounds by extracting the chelated complex of hexavalent chromium with ammonium pyrrolidine dithiocarbamate into methyl isobutyl ketone and then applying the atomic absorption spectrophotometric method to the extract for a specific determination of hexavalent chromium. [55] It is not subject to the interferences which affect the diphenylcarbazide method. The atomic absorption method is therefore recommended.

VI. REFERENCES

1. Mineral Facts and Problems, bulletin 650. US Dept Interior, Bureau of Mines, 1970

2. Bourne HG Jr, Yee HT: Occupational cancer in chromate plant--An occupational appraisal. Ind Med Surg 19:563-67, 1950

3. Gafafer WM (ed.): Health of Workers in Chromate Producing Industry, publication 192. US Dept Health, Education, and Welfare, Public Health Service, 1953, pp 131

4. Chemical Economics Handbook, Inorganic Chemicals C-Pi, 733.1020 A. Menlo Park, California, Stanford Research Institute, 1972

5. Chromic Acid, Chromium Trioxide--Chemical Safety Data Sheet SD-44. Washington, DC, Manufacturing Chemists' Association, 1952, pp 12

6. Physical constants of inorganic compounds, in Weast RC (ed.): Handbook of Chemistry and Physics--A Ready-Reference Book of Chemical and Physical Data, ed 52. Cleveland, Ohio, Chemical Rubber Company, 1971, p B-83

7. MacKenzie JN: Some observations on the toxic effects of chrome on the nose, throat and ear. JAMA 3:601-03, 1884

8. Blair J: Chrome ulcers--Report of twelve cases. JAMA 90:1927-28, 1928

9. Bloomfield JJ, Blum W: Health hazards in chromium plating. Pub Health Rep 43:2330-47, 1928

10. Lehmann KB: [Is there a reason for special concern in regard to the incidence of lung cancer in chromate workers? With comments on lung cancer in general.] Zentralbl Gewerbehyg 9:168-70, 1932 (Ger)

11. Machle W, Gregorius F: Cancer of the respiratory system in the United States chromate-producing industry. Pub Health Rep 63:1114-27, 1948

12. Mancuso TF, Hueper WC: Occupational cancer and other health hazards in a chromate plant: A medical appraisal--I. Lung cancers in chromate workers. Ind Med Surg 20:358-63, 1951

13. Taylor FH: The relationship of mortality and duration of employment as reflected by a cohort of chromate workers. Am J Pub Health 56:218-29, 1966

14. Bidstrup PL: Carcinoma of the lung in chromate workers. J Ind Med 8:302-05, 1951

15. Bidstrup PL, Case RAM: Carcinoma of the lung in workmen in bichromates-producing industry in Great Britain. J Ind Med 13:260-64, 1956

16. Baetjer AM: Pulmonary carcinoma in chromate workers--II. Incidence on basis of hospital records. Arch Ind Hyg Occup Med 2:505-16, 1950

17. Imbus HR, Cholak J, Miller LH, Sterling T: Boron, cadmium, chromium, and nickel in blood and urine--A survey of American working men. Arch Environ Health 6:286-95, 1963

18. Schroeder HA, Balassa JJ, Tipton IH: Abnormal trace metals in man--Chromium. J Chron Dis 15:941-64, 1962

19. Schroeder HA: Cadmium, chromium, and cardiovascular disease. Circulation 35:570-82, 1967

20. Schroeder HA: Serum cholesterol and glucose levels in rats fed refined and less refined sugars and chromium. J Nutrition 97:237-42, 1969

21. Mancuso TF: Occupational cancer and other health hazards in a chromate plant: A medical appraisal--II. Clinical and toxicologic aspects. Ind Med Surg 20:393-407, 1951

22. Samitz MH: Some dermatologic aspects of the chromate problem. Arch Ind Health 11:361-67, 1955

23. Barborik M: The problem of harmful exposures to chromium compounds. Ind Med 39:221-24, 1970

24. Newhouse ML: A cause of chromate dermatitis among assemblers in an automobile factory. J Ind Med 20:199-203, 1963

25. Kleinfeld M, Rosso A: Ulcerations of the nasal septum due to inhalation of chromic acid mist. Ind Med Surg 34:242-43, 1965

26. Zvaifler N: Chromic acid poisoning resulting from inhalation of mist developed from five per cent chromic acid solution--I. Medical aspects of chromic acid poisoning. J Ind Hyg Toxicol 26:124-26, 1944

27. Gomes ER: Incidence of chromium-induced lesions among electroplating workers in Brazil. Ind Med 41:21-25, 1972

28. Meyers JB: Acute pulmonary complications following inhalation of chromic acid mist--Preliminary observations of two patients

27. Gomes ER: Incidence of chromium-induced lesions among

who inhaled massive amounts of chromic acid. Arch Ind Hyg Occup Med 2:742-47, 1950

29. Vigliani EC, Zurlo N: [Observations of the Clinica del Lavoro with several maximum operating position concentrations (MAK) of industrial poison.] Arch Gewerbepathol Gewerbehyg 13:528-34, 1955 (Ger)

30. Medical Inspectorate of Factories: Chrome plating and anodic oxidation. J Ind Hyg 12:314-15, 1930

31. Pascale LR, Waldstein SS, Engbring G, Dubin A, Szanto PB: Chromium intoxication with special reference to hepatic injury. JAMA 149:1385-89, 1952

32. Baetjer AM: Pulmonary carcinoma in chromate workers--I. A review of the literature and report of cases. Arch Ind Hyg Occup Med 2:487-504, 1950

33. Baetjer AM, Damron C, Budacz V: The distribution and retention of chromium in men and animals. Arch Ind Health 20:136-50, 1959

34. Hueper WC: Experimental studies in metal cancerigenesis--VII. Tissue reactions to parenterally introduced powdered metallic chromium and chromite ore. J Nat Can Inst 16:447-69, 1955

35. Hueper WC: Experimental studies in metal cancerigenesis--X. Cancerigenic effects of chromite ore roast deposited in muscle tissue and pleural cavity of rats. Arch Ind Health 18:284-91, 1958

36. Hueper WC, Payne WW: Experimental cancers in rats produced by chromium compounds and their significance to industrial and public health. Am Ind Hyg Assoc J 20:274-80, 1959

37. Payne WW: Production of cancers in mice and rats by chromium compounds. Arch Ind Health 21:530-35, 1960

38. Roe FJC, Carter RL: Chromium carcinogenesis--Calcium chromate as a potent carcinogen for the subcutaneous tissues of the rat. J Cancer 23:172-76, 1969

39. Laskin S, Kuschner M, Drew RT: Studies in pulmonary carcinogenesis, in Hanna MG Jr, Nettesheim P, Gilbert JR (eds.): Inhalation Carcinogenesis. Proceedings of a Biology Div, Oak Ridge Nat Lab, Conference held in Gatlinburg, Tenn, Oct 8-11, 1969. US Atomic Energy Commission, Division of Technical Information, 1970, pp 321-51

40. Kuschner M: Studies in pulmonary carcinogenesis. National Cancer Institute contract no. PH43-NCI-66-962. Progress report, September 1, 1972

41. Gresh JT: Chromic acid poisoning from inhalation of mist developed from five per cent chromic acid solution--II. Engineering aspects of chromic acid poisoning from anodizing operations. J Ind Hyg Toxicol 26:127-30, 1944

42. Riley EC, Goldman FH: Control of chromic acid mists from plating tanks, P. Pub Health Rep 52:172-74, 1937

43. American National Standards: Practices for Ventilation and Operation of Open-Surface Tanks Z9.1-1971. New York, American National Standards Institute Inc, 1971, pp 36

44. Hama G, Fredrick W, Millage D, Brown H: Absolute control of chromic acid mist--Investigation of a new surface-active agent. Ind Hyg Quart 15:211-16, 1954

45. Molos JE: Use of plastic chips in the control of chromic acid mist. Ind Med 16:404-05, 1947

46. USA Standard: Allowable Concentration of Chromic Acid and Chromates Z37.7-1943. New York, United States of American Standards Institute, 1943, pp 7

47. Gafafer WM (ed.): Manual of Industrial Hygiene and Medical Service in War Industries. Philadelphia, WB Saunders Co, 1943, p 264

48. American Conference of Governmental Industrial Hygienists: Documentation of the Threshold Limit Values for Substances in the Workroom Air, ed 3. Cincinnati, Ohio, ACGIH 1971, pp 55-56

49. Chromic acid, Hygienic Guide Series. American Industrial Hygiene Association, 1956

50. Hueper WC: Carcinogenic hazards from arsenic and metal containing drugs, in Truhaut R (ed.): UICC Monograph Series. Potential Carcinogenic Hazards from Drugs. 7:90-91

51. Jacobs MB: The Analytical Toxicology of Industrial Inorganic Poisons. New York, Interscience Publishers, 1967, pp 396-402

52. Analytical Chemistry Committee: Analytical Abstracts, Chromates and Chromic Acid. American Industrial Hygiene Association, 1965

53. Ege JF Jr, Silverman L: Efficiency of filter paper and impingers for chromic acid mists in air. Ind Hyg Quart 8:12-13, 1947

54. Saltzman BE: Microdetermination of chromium with diphenylcarbazide by permanganate oxidation--Improved method of oxidation and color development. Anal Chem 24:1016-20, 1952

55. Brown E, Skougstad MW, Fishman MJ: Techniques of Water-Resources Investigations of the United States Geological Survey. Chapter A1, Methods for collection and analysis of water samples for dissolved minerals and gases. Dept of Interior, Laboratory Analysis, 1970, Book 5, pp 77-78

VII. APPENDIX I

AIR SAMPLING PRACTICES FOR CHROMIC ACID

General Requirements

Air concentrations shall be determined within the worker's breathing zone and shall meet the following criteria in order to evaluate conformance with the standard:

(a) Samples collected shall be representative of the individual worker's exposure.

(b) Sampling data sheets shall include:

(1) The date and time of sample collection

(2) Sampling duration

(3) Volumetric flowrate of sampling

(4) A description of the sampling location

(5) Other pertinent information

Breathing Zone Sampling

(a) Breathing zone samples shall be collected as near as practicable to the worker's face without interfering with his freedom of movement and shall characterize the exposure from each job or specific operation in each production area.

(b) A portable battery-operated personal sampling pump plus an unweighed 0.8 μ cellulose membrane filter (Type AA) mounted in either a 2- or 3-piece cassette shall be used to collect the sample.

(c) The sampler shall be operated at a flowrate of two liters per minute and samples taken for at least 10 minutes.

(d) A minimum of three samples shall be taken for each operation (more samples if the concentrations are close to the standard) and averaged on a time-weighted basis.

(e) A minimum of three blank filters carried in closed cassettes to the sampling site shall be provided to the analytical laboratory to determine the background correction which must be applied to the analytical results.

Calibration of Sampling Trains

Since the accuracy of an analysis can be no greater than the accuracy of the volume of air which is measured, the accurate calibration of a sampling pump is essential to the correct interpretation of the pump's indication. The frequency of calibration is dependent on the use, care, and handling to which the pump is subjected. In addition, pumps should be recalibrated if they have been subjected to misuse or if they have just been repaired or received from a manufacturer. If the pump receives hard usage, more frequent calibration may be necessary.

Ordinarily, pumps should be calibrated in the laboratory both before they are used in the field and after they have been used to collect a large number of field samples. The accuracy of calibration is dependent on the type of instrument used as a reference. The choice of calibration instrument will depend largely upon where the calibration is to be performed. For laboratory testing, a 1-liter burette or wet-test meter is recommended, although other standard calibrating instruments such as spirometer, Marriott's bottle, or dry-

gas meter can be used. The actual set-up will be the same for these instruments.

Instructions for calibration with the wet-test meter follow. If another calibration device is used, equivalent procedures should be followed.

(a) The calibration device used shall be in good working condition and shall have been calibrated against a spirometer (or other primary standard) upon procurement, after each repair, and at least annually.

(b) Calibration curves shall be established for each sampling pump and shall be used in adjusting the pumps prior to field use.

(c) The volumetric flowrate through the sampling system shall be spot checked and the proper adjustments made before and during each study to assure obtaining accurate airflow data.

(d) Flowmeter Calibration Test Method (see Figure 1)

(1) Apparatus

(A) Wet test meter (Arthur H. Thomas Co., Precision 5648-B10, or equivalent)

(B) Quick connector or by-pass valve

(C) In-line filter holder cassette with Type AA filter

(D) Tee

(E) Manometer (Dwyer No. 1215-36", U-tube or equivalent)

(F) Pump with rotameter (Mine Safety Appliances Co., Model G, or equivalent)

(G) Rubber or vinyl tubing

(H) Barometer

(I) Thermometer

(J) Stopwatch

(K) Small screw driver

(L) Graph paper

(2) Procedures

(A) Level wet test meter. Check the water level which should just touch the calibration point at the left side of the meter. If water level is low, add water 1 to 2 F warmer than room temperature to fill point. Run the meter for 30 minutes before calibration.

(B) Check the voltage of the pump battery with a voltmeter. For example, a reading of 7.0 volts for Mine Safety Appliances Company, Model G is required for calibration; and if reading is lower charge batteries until a reading of 7.0 volts is obtained.

(C) Mount the filter to be calibrated in the in-line filter holder.

(D) Assemble the calibration train as shown in Figure 1. Leave the quick connector disconnected.

(E) Turn the pump on, adjusting the rotameter with a screw driver to a reading of 10 (read middle of the float).

(F) Connect the wet test meter to the train. The pointer on the meter should run clockwise and a pressure drop of not more than 1.0 inch of water indicated. If the pressure drop is greater than 1.0 disconnect and check the system.

(G) Operate the system ten minutes before starting the calibration.

(H) Record the following on calibration data sheets:

(i) Wet test meter reading, start and finish

(ii) Elapsed time, start and finish

(iii) Pressure drop at manometer

(iv) Air temperature

(v) Barometric pressure

(vi) Serial number of pump and rotameter

(I) Adjust the rotameter reading to 9.0, 8.0, and 7.0, respectively, and repeat step (H) at each reading. Each point should run for 10 minutes or at least 0.5 cubic foot of air.

(J) Record the name of the person performing the calibration, the date, serial number of the wet test meter, and the numbers of the pump and flowmeter system being calibrated.

(K) Correct the wet test meter readings to standard conditions of pressure and temperature by means of the gas law equation.

(L) Use graph paper to record the actual airflow as the ordinate and the rotameter readings as the abscissa.

VIII. APPENDIX II

ANALYTICAL METHODS FOR CHROMIC ACID

Principle of the Method

(a) A known volume of air is drawn through a Type AA membrane filter to collect the chromic acid aerosol.

(b) The filter is placed in an individual plastic Petri dish and returned to the laboratory where the sample is leached from the filter with distilled water.

(c) The solution is filtered, if necessary.

(d) The hexavalent chromium is separated from trivalent chromium by chelation of the former with ammonium pyrrolidine dithiocarbamate (APDC) and extraction of the complex with methyl isobutyl ketone. [55]

(e) The extracted hexavalent chromium is analyzed by atomic absorption spectrophotometry.

(f) After correcting for the filter and reagent blank, the chromic acid concentration is calculated as milligrams chromium trioxide per cubic meter of air.

Range and Sensitivity

(a) The limit of detection of the method is 0.2 μg of chromic acid per filter sample or 0.01 mg chromic acid per cu m in a 20-liter air sample.

(b) The upper limit of the method may be varied, according to the sample requirements, by appropriate selection of the size aliquot portion taken for analysis and/or by changing the attenuation setting

of the spectrophotometer. The upper limit of the extraction procedure is 2.5 micrograms of hexavalent chromium [55] which would correspond to 0.25 mg of chromium trioxide per cu m in a nonaliquotted 20-liter air sample.

Interferences [55]

The method is essentially free from interference. The optimum pH for the extraction of the Cr-APDC complex by MIBK is 3.1. At this pH, however, manganese is partially extracted. The Mn-APDC complex is unstable and decomposes to a fine suspension of manganese oxides which clog the atomizer-burner. If the extract is not clear after standing overnight, it must be centrifuged. By adjusting the pH of the sample to 2.4 prior to chelation and extraction, less manganese is extracted and there is only a slight loss in the efficiency of the extraction of chromium.

Precision

(a) Single-operator precision of the method based on ten determinations of 5.0 µg of hexavalent chromium per liter in water solutions was reported to be 0.57 µg per liter. [55]

(b) No data are available currently on the accuracy of the overall analytical method.

Advantages and Disadvantages of the Method

(a) The method determines only hexavalent chromium in samples containing both trivalent and hexavalent forms of the element.

(b) The method is rapid and may be applied conveniently in all chemistry laboratories equipped with an atomic absorption spectrophotometer.

(c) The method is not affected by the presence of other common metallic elements with the exception of manganese; reduction of the pH of the sample solution from 3.1 to 2.4, prior to chelation and extraction of the hexavalent chromium, minimizes this effect.

Apparatus

(a) Filters, cellulose, membrane, 0.8 μ, Type AA

(b) Glassware, borosilicate, as required for the reagents Do not use polyethylene ware for chromium analyses

(c) Spectrophotometer, atomic absorption

Reagents

(a) Reagent grade chemicals shall be used in all tests.

(b) Water, demineralized (distilled or deionized), shall be used in preparing reagents or dilutions of samples.

(c) Ammonium pyrrolidine dithiocarbamate (APDC) solution: Dissolve 1.0 g APDC in demineralized water and dilute to 100 ml Prepare fresh daily.

(d) Bromphenol blue indicator solution: Dissolve 0.1 g bromphenol blue in 100 ml 50% ethanol.

(e) Standard solution I. 1.00 ml = 100 μg chromium: Dissolve 0.2829 g pure dried potassium dichromate in demineralized water and dilute to 1,000 ml.

(f) Standard solution II. 1.00 ml=10.0 µg chromium: Dilute 100 ml chromium standard solution I to 1,000 ml with demineralized water.

(g) Standard solution III. 1.00 ml = 0.10 µg chromium: Dilute 10.0 ml chromium standard solution II to 1,000 ml with demineralized water.

(h) Methyl isobutyl ketone (MIBK).

(i) Sodium hydroxide solution, 1M: Dissolve 40 g sodium hydroxide in demineralized water and dilute to 1 liter.

(j) Sulfuric acid, 0.12M: Slowly add 6.5 ml concentrated sulfuric acid (sp gr 1.84) to demineralized water and dilute to 1 liter.

(k) Wash acid - Add 50 ml of concentrated nitric acid to 150 ml of concentrated hydrochloric acid, mix and add to 200 ml of water. This solution may be used repeatedly to rinse glassware and should be stored in a borosilicate, glass-stoppered bottle.

Procedure

(a) Cleaning of glassware: Soak all glassware used for chromium analysis in detergent solution; rinse copiously with warm tap water; remove grease with alcoholic potassium hydroxide; rinse repeatedly with tap water to remove the residual caustic solution; rinse with the wash acid followed by repeated tap water and distilled water rinsings. Caution: Do not use chromic acid cleaning solution.

(b) Shipping of samples: Transfer each filter sample to an individual, covered, plastic Petri dish for transport to the laboratory.

(c) Analysis of Samples

(1) Transfer the filter containing the sample to a Griffin beaker and add sufficient demineralized water to cover the filter. Allow to stand for 10-15 minutes with occasional agitation.

(2) Decant the aqueous solution and demineralized water rinsings of the beaker into a 100-ml volumetric flask.

(3) Repeat steps (1) and (2) twice.

(4) Dilute the sample solution to volume and mix.

(5) Pipette a volume of the sample containing less than 2.5 µg of hexavalent chromium into a 200-ml volumetric flask and adjust the volume to approximately 100 ml.

(6) Prepare a set of 3 blanks, using filter papers carried through the leaching procedure described in Steps (1) through (5), and a set of hexavalent chromium standards; adjust the volume of each blank and each standard sample to approximately 100 ml.

(7) Add 2 drops of bromphenol blue indicator solution.

(8) Adjust the pH by addition of 1M sodium hydroxide solution by drops until a blue color persists. Add 0.12M sulfuric acid by drops until the blue color just disappears in both the standards and sample. Then add 2.0 ml 0.12M sulfuric acid in excess. The pH at this point should be 2.4.

(9) Add 5.0 ml APDC solution and mix. The pH should then be approximately 2.8.

(10) Add 10.0 ml MIBK and shake vigorously for 3 minutes.

(11) Allow the layers to separate and add demineralized water until the ketone layer is completely in the neck of the flask. The Cr-APDC complex is stable for at least 36 hours.

(12) Aspirate the ketone layer and measure the absorbance (or other scale reading) of the solution against the average blank using the 3578.7 A resonance line of chromium and the operating parameters of the atomic absorption spectrophotometer recommended by the manufacturer of the instrument for this determination. Repeat and average the duplicate results.

Calculations

(a) Prepare a plot of absorbance values (or other scale readings) of the series of standard samples whose concentrations are expressed as μg chromium trioxide per 10.0 ml of MIBK.

(b) Determine the μg chromium trioxide in the sample aliquot portion taken for analysis, using the standard curve prepared as described in Step (a).

(c) Calculate the μg chromium trioxide in the total filter sample and convert to milligrams.

(d) Divide the value obtained in Step (c) by the liters of air sampled and then multiply by 1000 to calculate the chromic acid

concentration in terms of milligrams chromium trioxide per cubic meter.

IX. APPENDIX III

MATERIAL SAFETY DATA SHEET

The following items of information which are applicable to a specific product or material containing chromic acid shall be provided in the appropriate section of the Material Safety Data Sheet or approved form. If a specific item of information is inapplicable (ie, flash point) initials "n.a." for not applicable shall be inserted.

(a) The product designation in the upper left hand corner of both front and back to facilitate filing and retrieval. Print in upper case letters in as large a print as possible.

(b) Section I. Source and Nomenclature.

(i) The name, address, and telephone number of the manufacturer or supplier of the product.

(ii) The trade name and synonyms for a mixture of chemicals, a basic structural material, or for a process material; and the trade name and synonyms, chemical name and synonyms, chemical family, and formula for a single chemical.

(c) Section II. Hazardous Ingredients

(i) Chemical or widely recognized common name of all hazardous ingredients.

(ii) The approximate percentage by weight or volume (indicate basis) which each hazardous ingredient of the mixture bears to the whole mixture. This may be indicated as a range or maximum amount, ie, 10-20% V; 10% max. W.

(iii) Basis for toxicity for each hazardous material such as established OSHA standard, in appropriate units and/or LD50, showing amount and mode of exposure and species, or LC50 showing concentration, duration of exposure, and species.

(d) Section III. Physical Data.

Physical properties of the total product including boiling point and melting point in degrees Fahrenheit; vapor pressure, in millimeters of mercury, vapor density of gas or vapor (air=1), solubility in water, in parts per hundred parts of water by weight; specific gravity (water=1); percent volatile, indicate if by weight or volume, at 70 degrees Fahrenheit; evaporation rate for liquids (indicate whether butyl acetate or ether=1); and appearance and odor.

(e) Section IV. Fire and Explosion Hazard Data.

Fire and explosion hazard data about a single chemical or a mixture of chemicals, including flash point, in degrees Fahrenheit; flammable limits, in percent by volume in air; suitable extinguishing media or agents; special fire fighting procedures; and unusal fire and explosion hazard information.

(f) Section V. Health Hazard Data.

Toxic level for total compound or mixture, relevant symptoms of exposure, skin and eye irritation properties, principal routes of absorption, effects of chronic (long-term) exposure, and emergency and first aid procedures.

(g) Section VI. Reactivity Data.

Chemical stability, incompatibility, hazardous decomposition products, and hazardous polymerization.

(h) Section VII. Spill or Leak Procedures.

Detailed procedures to be followed with emphasis on precautions to be taken in cleaning up and safe disposal of materials leaked or spilled. This includes proper labeling and disposal of containers containing residues, contaminated absorbants, etc.

(i) Section VIII. Special Protection Information.

Requirements for personal protective equipment, such as respirators, eye protection, and protective clothing, and ventilation such as local exhaust (at site of product use or application), general, or other special types.

(j) Section IX. Special Precautions.

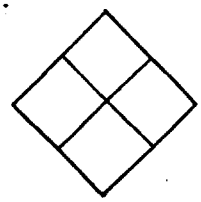
Any other general precautionary information such as personal protective equipment for exposure to the thermal decomposition products listed in Section VI, and to particulates formed by abrading a dry coating, such as by a power sanding disc.

(k) The signature of the responsible person filling out the data sheet, his address, and the date on which it is filled out.

PRODUCT DESIGNATION

MATERIAL SAFETY DATA SHEET

Form Approved
Budget Bureau No.
Approval Expires
Form No. OSHA



SECTION I SOURCE AND NOMENCLATURE	
MANUFACTURER'S NAME	EMERGENCY TELEPHONE NO.
ADDRESS (Number, Street, City, State, ZIP Code)	
TRADE NAME AND SYNONYMS	CHEMICAL FAMILY
CHEMICAL NAME AND SYNONYMS	FORMULA

SECTION II HAZARDOUS INGREDIENTS						
BASIC MATERIAL	APPROXIMATE OR MAXIMUM % WT. OR VOL.	ESTABLISHED OSHA STANDARD	LD 50		LC 50	
			ORAL	PERCUT.	SPECIES	CONC.

SECTION III PHYSICAL DATA			
BOILING POINT	°F.	VAPOR PRESSURE	mm Hg.
MELTING POINT	°F.	VAPOR DENSITY (Air=1)	
SPECIFIC GRAVITY (H_2O=1)		EVAPORATION RATE (_____=1)	
SOLUBILITY IN WATER	Pts/100 pts H_2O	VOLATILE % Vol.	% Wt.
APPEARANCE AND ODOR			

SECTION IV FIRE AND EXPLOSION HAZARD DATA		
FLASH POINT METHOD USED	FLAMMABLE (EXPLOSIVE) LIMITS	UPPER LOWER
EXTINGUISHING MEDIA		
SPECIAL FIRE FIGHTING PROCEDURES		
UNUSUAL FIRE AND EXPLOSION HAZARDS		

PRODUCT DESIGNATION	

SECTION V HEALTH HAZARD DATA	
TOXIC LEVEL	CARCINOGENIC
PRINCIPAL ROUTES OF ABSORPTION	SKIN AND EYE IRRITATION
RELEVANT SYMPTOMS OF EXPOSURE	
EFFECTS OF CHRONIC EXPOSURE	
EMERGENCY AND FIRST AID PROCEDURES	

SECTION VI REACTIVITY DATA
CONDITIONS CONTRIBUTING TO INSTABILITY
CONDITIONS CONTRIBUTING TO HAZARDOUS POLYMERIZATION
INCOMPATIBILITY (Materials to Avoid)
HAZARDOUS DECOMPOSITION PRODUCTS

SECTION VII SPILL OR LEAK PROCEDURES
STEPS TO BE TAKEN IN CASE MATERIAL IS RELEASED OR SPILLED
WASTE DISPOSAL METHOD

SECTION VIII SPECIAL PROTECTION INFORMATION	
VENTILATION REQUIREMENTS LOCAL EXHAUST	PROTECTIVE EQUIPMENT (Specify Types) EYE
MECHANICAL (General)	GLOVES
SPECIAL	RESPIRATOR
OTHER PROTECTIVE EQUIPMENT	

SECTION IX SPECIAL PRECAUTIONS
PRECAUTIONS TO BE TAKEN IN HANDLING AND STORAGE
OTHER PRECAUTIONS

Signature _____ Address

Date

TABLE X-1

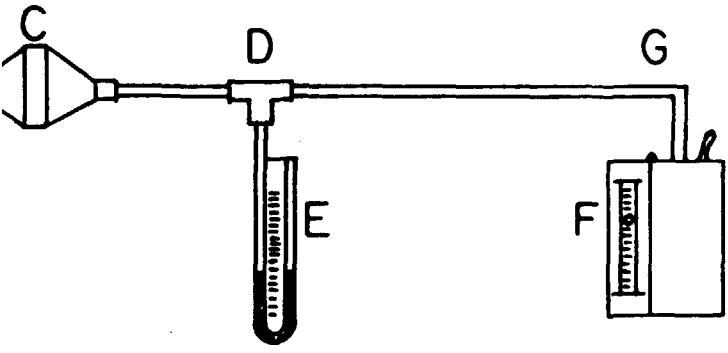
Physical Properties of Chromium Trioxide

Molecular Formula	CrO3
Formula Weight	99.99
Boiling Point	Decomposes
Melting Point	196 C
Density	2.70
Solubility	67.45 g per 100 g of water at 100 C; soluble in alcohol, ether, sulfuric acid, nitric acid

From reference number [6]

☆ U.S. GOVERNMENT PRINTING OFFICE: 1973— 759-560/1489

Figure 1. Calibration setup



for portable pumps with filters .