INVESTIGATIONS OF HEALTH HAZARDS IN THE PAINTING TRADES

National Institute for Occupational Safety and Health
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Irving J. Selikoff, M.D.
Principal Investigator

OF THE CITY UNIVERSITY
OF NEW YORK

ENVIRONMENTAL SCIENCES LABORATORY
MOUNT SINAI SCHOOL OF MEDICINE OF THE CITY UNIVERSITY OF NEW YORK

FINAL REPORT

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Irving J. Selikoff, M.D.
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I. Introduction

A. Background

In 1972, workers in a number of painting industry crafts, and their union leaders, became increasingly concerned with the possibility that important health hazards existed in their work. This apprehension was derived from a number of fragmentary experiences. For example, statistical data from the Workmen's Compensation Board in New York indicated that the highest rate for serious accidents in that state was found among general painters and consideration was given to the possibility that exposure to solvents and other neurotoxic chemicals in a "scaffold trade" was responsible for this. Changing paint technology had resulted in the introduction in the prior decade of a wide variety of new paints, including epoxies, of incompletely appreciated and incompletely studied human toxicity, and dust exposures had become more varied and potentially more worrisome, e.g. sandblasting in metal painting and shipyard work and asbestos exposure in dry wall construction. The latter industry had only begun after World War II, and had rapidly expanded.

The Executive Council of the International Brotherhood of Painters and Allied Trades consulted with scientists of our Laboratory, providing detailed descriptions of trade conditions and proffering their evaluation of potentially hazardous agents in their work. This review and evaluation was facilitated by the establishment of a special health hazard committee of the Brotherhood, by its General President (Mr. S. Frank Raftery).

Review of the scientific literature (Dr. Kingsley Kay) quickly revealed that changing paint technology had indeed resulted in the introduction of a very wide variety of new chemical exposures in the painting trades, some potentially quite serious. It further revealed that few if any studies had been undertaken to determine what the health effects might be under conditions of use of these materials in the construction industry, but that the projections of toxicity were based largely on toxicological evaluation including animal studies. Further, it quickly became evident that it would be most difficult to predict what toxic effects might be present under trade conditions, since there were virtually no data concerning environmental levels, conditions and intensity of worker exposures, or recorded occupational disease effects. Preliminary field examination of trade conditions made by Dr. Kay with Painters' Union groups showed that symptoms were common among men on the job and were, in some cases, potentially serious and that management personnel, too, had little information concerning possible health effects of materials that had been introduced and were then in use.

Two areas of particular Painters' Union concern were looked at in somewhat more detail, by review with industry experts. First, inquiry revealed that vinyl chloride monomer could be present in "vinyl" paints; other monomers were unstudied. Second, consultation with industry representatives indicated that the majority of spackle and taping compounds then widely used in the painting and decorating industry (dry wall construction; taping, spackling in general painting, etc.) and that the use of these materials was associated with airborne concentrations of asbestos fibers sometimes well above Asbestos Standard levels. Further discussions of the general problem were afforded by the invitation of the National Association of Paint and Coating Manufacturers to Dr. I.J. Selikoff, to meet with their Board. It was there found that materials manufacturers were equally interested in determining whether or not there was potential health hazard with the use of particular materials, so that appropriate corrective measures could be considered.

The scattered data were further critically considered with two other groups. A joint National Health Hazard Committee had been organized by the Painters' Union and employer groups, on the national scale. Discussions with the members of this Joint Committee were valuable. Similarly, consultation with NIOSH, including its then Director, Dr. Marcus M. Key, provided useful guidance.

Thus, in 1973, information had been obtained from a variety of sources, scientific, labor, industry, government, that significant health hazards might exist in the painting trades, that few data were available concerning such hazards, and that their evaluation required careful study.

B. Previous status of the problem

It was something of a paradox to find a paucity of study of occupational hazards in a trade involving a very large number of men, using a wide variety of chemicals, some individually known to have important toxic potential, in a key industry in our country. In particular, there had been few investigations in the past two decades, despite the significant changes in trade conditions during that time.

1. Absence of recent investigations -- This was particularly disadvantageous in planning an investigation, since few guidelines were available regarding study design and focus. This absence of guidance underlay the wide exploration that had to be considered.

2. Changing technology -- Again, relatively few leads were available apart from toxicology studies of chemicals involved. For example, lead had largely been replaced by titanium after World War II and paints now in use were virtually unknown 20, and in some instances, 10 years before. It has been mentioned that dry wall construction is a post-World War II phenomenon, the fledging industry being established 1946-1950, and growing, in 1973, to the employment of 50-75,000 workers. Sandblasting techniques have progressed (with, frequently, opportunity for intensive silica exposure) and even "wallpapers" and adhesives were hardly the same as they had been in the recent past. This change in technology had been widespread and pervasive.

3. Suspect agents -- With this changing technology had come the introduction of a wide variety of materials containing agents known in other circumstances to be potentially toxic. These included exotic chemicals, fungicides, antioxidants, curing agents, new solvents, accelerators, monomers and polymers, amorphous silica, asbestos, a variety of chromates, etc. Little

was known of the potential effect of these agents in the particular circumstances in which they were used in the painting trades.

4. Suggestions of health hazards -- Scattered experiences, here and there, had indicated that, in at least some instances, adverse health effects were being noted. Painters overcome by epoxies used in confined spaces, the accident rates commented on above, the finding of extraordinary lung burdens of asbestos in taping and dry wall construction workers, etc., had been recorded.

5. Exposed populations -- All these factors were of increased concern because of the very large number of workers involved. Although the International Brotherhood of Painters and Allied Trades only includes some 225,000 members, other workers in the same trades, but not members of the Brotherhood, raise the total number involved to approximately 400,000. In addition, many workers are engaged in doing the same kind of work as "maintenance personnel" in large industrial concerns, shipyards, refineries, chemical plants, commercial installations and other facilities.

C. Plan of pilot study

The above experience provides a background for our approach to this pilot study of health hazards in the painting trades.

1. Multiple variables -- Exposures in the painting trades are totally unlike those which might obtain, let us say, in a factory production facility, with one or a few agents, repetitively used, at constant levels, fixed in time and place. On the contrary, exposures are extremely varied, sometimes from day to day and surely from year to year, differing among the different crafts, and, possibly in different parts of the country.

a. Many trades -- The appellation of the Painters' Union, "International Brotherhood of Painters and Allied Trades" tells the story. In addition to general painters, there are dry wall construction workers and tapers, wall paper hangers, iron painters (lead paints!), wood finishers, paint factory workers, sandblasters, scenic artists, glaziers, floor tile sanders, carpet layers and others. In some places, the crafts are distinct. In others, men cross craft lines, occasionally within the same local unions. Within each craft, exposures are varied; for example, men in the glazier firms may put porcelain panels in place; these can be backed with asbestos cement sheets which require abrasive sanding and manipulation, with consequent asbestos exposure.

b. Wide geographical distribution -- Workers in these trades are found in all parts of our country. Yet their exposures are not necessarily the same in each! For example, taping compounds suitable for Florida and the Southwest are not necessarily adequate for Minnesota and the North Central States, because of different climatic conditions.

c. Multiple target organs -- It was clear from preliminary review of potential hazards, that if disease was to be found, it was not necessarily to be discovered in a single organ system -- pulmonary disease, or skin disease, or kidney disease, or liver disease, or central nervous system disease -- but, rather, more than one system was likely to be affected among the trades in general, and even within particular groups.

d. Paucity of preliminary data -- This has been commented upon above.

2. Examination perspectives -- It was therefore considered necessary to envisage a pilot study which would allow for evaluation of clinical effects, some known or suspected and others unsuspected, among workers in different parts of the country, and in a variety of trades. It was considered that this could best be accomplished by field surveys in a number of cities of different size, rather than hospital or clinic based study. Further, the examination schedule had to include a wide review of occupational history, analysis of symptoms by an equally wide system review including skin, lung, nervous system, pulmonary, cardiac, gastrointestinal, locomotor, etc. Examinations had to be equally broad, to range from pulmonary function study to clinical examination of the abdomen for hepatosplenomegaly, from standard biochemical tests, to special tests for lead exposure, from chest x-rays and associated physical examination, to central nervous system study, from a vascular test such as the Allan test to study of skin changes and upper respiratory allergy, from consideration of possible occupational carcinogenesis (CEA and sputum cytology) to special blood tests for trace metals.

In the event, examinations were made of over 1,000 workers in the crafts concerned (painters, iron painters, sandblasters, paper hangers, dry wall construction workers and tapers, paint factory workers, titanium plant workers, wood finishers, floor tile sanders). Parenthetically, we were most interested in obtaining information concerning the health status of titanium workers; this had never been done before and and this information would provide a broad background for evaluation of the possible toxic effects of titanium among painters in general. Too, the titanium workers provided a useful control for painters in general, since they were not exposed to the painting chemicals. Similarly, by including sufficiently large groups of men working in the different crafts, all within the same Union and in the same socio-economic stratum, we would have the opportunity of being able to analyze the experiences of "control groups" some exposed to particular agents to which others were not exposed. This was an important perspective, and our analyses have been made with awareness of the importance of such control groups.

The examinations were made in Los Angeles, New York, Toledo, Paterson, New Jersey, St. Louis, and represented a broad spectrum of workers in these trades, in the United States.

II. Clinical findings

A. Introduction

In preparation for the clinical field examinations, several steps were necessary in order to get as complete information as possible on the health hazards to be considered in the various trades (painters, tapers, floor tile workers, sandblasters, etc.). Information was also sought on the recent developments in the trade, according to the new technology applied, and on the approximate time of significant changes (such as use of epoxy resins, etc.). An extensive search of the available literature was undertaken. This information was essential in order to plan the study, defining the critical problems which had to be looked into.

Several main areas of interest were defined as being the most important health hazards in the trade:

- organic solvents (thinners, etc.)
- pigments (mainly lead, but also cadmium and titanium compounds)
- silica dust (sandblasters and metal painters)
- asbestos dust (tapers, etc.)
- resins and plastics (epoxy resins, PVD, etc.)

An examination form was designed with adverse health effects of these hazards specifically considered, with emphasis on a complete occupational history, and a thorough inquiry into possible symptoms and/or physical signs related to these agents.

Relevant laboratory tests were selected following the same guidelines and comprised:

- complete blood counts, including platelets and differential WBC (for aromatic solvents and lead)
- SMA 12 (especially in order to detect abnormal liver tests and also for evaluations of the general health status).
- Lead in blood and urine samples, titanium in blood and urine samples, cadmium in urine.
- Chest x-ray films (silica and asbestos exposure).
- Pulmonary function tests (spirometry and flow volume curves).
- Carcinogenic embrionic antigen, in order to detect elevated values, which may indicate a suspicion of malignancy.
- Sputum cytology, in order to detect atypic or definitely abnormal cells, which may contribute to an early diagnosis of carcinoma of the bronchus.

Analysis of the occupational histories showed that although most of the examined workers had had various kinds of exposure during employment, their predominant type of work (and exposure) allowed a grouping into several main categories:

- painters and paint manufacturers
- tapers
- floor tile workers
- sandblsters and metal painters
- titanium dioxide manufacturers
- others (glaziers, etc.)

Clinical field examinations were conducted in three different geographic areas:

The first included 408 members of the Painters' Union who were examined at the time of the "23rd General Convention of the International Brotherhood of Painters and Allied Trades" in Los Angeles, California, September 8-12, 1974.

This first examination provided a wealth of detailed information on the historical development of the various trades in the Painters' Union. It also provided a much deeper insight into the actual working conditions and health hazards of the workers. Several areas of concern were defined, while others, such as the lead and cadmium problem were found to be of minor importance, at least for the present time and for the group examined.

The preliminary analysis of the results of this examination was reported in a progress report ....

The following two field examinations were conducted in Toledo, Ohio, (85 workers) and St. Louis, Missouri (506 workers).

In St. Louis a group of 207 titanium dioxide manufacturers was included. The assessment of their health status was thought to be particularly important since at the time of the examination no clinical survey of such a workers' population was known to have ever been undertaken.

B. Painters and other trades

1. Central nervous system

a. Findings

The analysis of the results of these three clinical field examinations revealed the high prevalence of acute symptoms of overexposure to organic solvents (thinners) in Painters (Table B1).

Central nervous system effects of these volatile solvents were manifest, during spray-painting, but also when brush-painting in enclosed areas, without adequate ventilation. Symptoms such as dizziness, light headedness, unusual exhilaration, loss of balance, feeling "drunk" or "high" were very often reported: the overall prevalence for all examined painters was 71%. The variations in prevalence in the three groups of painters, according to location (Table B2) were small; a slightly higher prevalence was noted for the Toledo Group. Digestive symptoms, such as nausea, vomiting and loss of appetite accompanied the prenarcotic symptoms in almost one-third of the cases. Loss of consciousness was found to have occurred during work with marked solvent exposure in 4% of all painters.

When comparing the prevalence of acute symptoms of overexposure to solvents (prenarcotic symptoms) in painters, with the prevalence of such symptoms in the other trades, it is obvious that only in the metal painters, who are frequently spray-painting, these symptoms occur with similar frequency.

The paint manufacturers, although solvents enter the composition of the products, are protected to a considerable extent since most of the operations are enclosed and/or do not require the permanent presence of the worker.

The tables in this section are labeled "B- " and follow the end of this section (Page 30).

Since many of the examined painters reported that work with epoxy paints was particularly associated with adverse effects, the prevalence of prenarcotic symptoms in painters with epoxy exposure was compared with that in painters who reported to have never had such an exposure. Prenarcotic symptoms were reported by 84 percent of workers who had used epoxy paints, as compared with 60 percent of those who had never used such compounds.

In order to add an objective parameter to the assessment of central nervous system effects of solvents, a reaction time test was added to the battery of tests used in the group examined in St. Louis.

According to the classical literature, reaction time stabilizes at about 16-18 years of age and stays relatively constant up to the seventies.

Therefore, when considering a "normal" population, with an average age distribution, the reaction time curve is expected to be flat, and the mean value is expected to be 190 milliseconds. There were **272** painters who were given a reaction test. The mean value for all the age groups (less than 39, 40 to 49, 50 to 59, and over 60) were higher than normal; the highest mean values were found in the two younger age groups (less than 39 years - mean 204 milliseconds, 40-49 years - mean 199 milliseconds).

An attempt to interprete these preliminary data would have to consider several possibilities:

- longer reaction time in younger painters may reflect a higher degree of susceptibility to central nervous system effects due to organic solvents in younger persons.

- longer reaction time in younger painters may also be related to higher exposure levels to more potent solvents (like those used for epoxy paints) in the younger group.

The preliminary results of this very simple test are such as to warrang further studies on solvent induced central nervous effects in painters. Even slight or moderate lengthening of reaction time and other subtle changes in central nervous system function may lead to a significant increase in accident proneness ~~and in the actual rate of~~ accidents.

b. Feasibility Study for Extended Neurological Examination

Existing methods for identifying industrial neurotoxins depend on the prior occurrence of overt clinical changes in exposed workers which significantly affect their job performance or other activities of daily living, and may, in addition, presage undesirable long range effects on their health and longevity. It is therefore essential to develop new procedures to detect or predict relevant neurological deficits due to industrial materials before they produce gross, possibly irreversible, behavioral signs and symptoms.

The results of behavioral studies reported in the literature, supported by our own observations of a number of workers exposed to volatile toxins, suggest that the principal neurotoxic effects of these solvents is a reduction in information handling capacity of the individual, and therefore that the most sensitive tests of neurological deficits will be those which examine his ability to acquire, process and generate information.

Methods of Examination

How selected?

A total of 18 male painters, ranging in age from 24-68, all of whom had a history of heavy exposure to volatile agents used in the painting trade (lacquers, epoxies, alcohols and cleaners) were examined in the course of this survey. Most of these individuals complained of functional disturbances associated with the use of paint materials; others admitted to symptoms on direct questioning. Most of the subjects drank beer, some regularly, a few in large quantities; a few admitted to the use of other intoxicants; one reported several courses of electroconvulsive therapy and one had had equine encephelitis in childhood. In the interview, the workers were asked to describe exposure symptoms in detail with respect to onset, quality, duration and remedy; and to explicate other neurologic symptoms not immediately associated with exposure. Current neurological status was determined in each by a complete clinical examination undertaken by a senior neurologist. A four channel EEG recording was made on seven workers in this group during patterned visual stimulation. The data were stored on magnetic tape for processing in our laboratory at MSSM.

Selection

Observations

The most common symptoms reported to be associated with ongoing exposure were: light-headedness (8); headache, usually bifrontal or diffuse, often diffuse, often pounding (7); "dizziness" (7); disturbance in balance (5); feeling "high", not always pleasantly so (5); silly or other unusual behavior (5); lack of concentration, fear of accident and careless work (4); confusion (4); loss of consciousness (4); paresthesias (4); and slurred speech (1).

Most subjects experienced relief of overt symptoms after 10-30 minutes in fresh air. In several cases, the full effects of the intoxication were not felt during the exposure but appeared to develop, or were first noticed, only after the workers left the painting environment. Three cases of prolonged unconsciousness were reported to have occurred in this way. Often, the original symptoms persisted for hours after the exposures or deficits became apparent when they performed new tasks. The most significant of these deficits were associated with driving a car after work. All subjects with acute symptoms reported some difficulty in driving. There were several reports of accidents or near misses, and one patient stated that he drove home after symptomatic exposure and had no recollection of doing so. Detailed questioning suggested that the basic problem was not as much due to slowed reaction time or faulty judgement as it was a failure to observe cars or pedestrians in their peripheral view, causing "surprises" when they encroached upon or intersected the path of the worker's car. A number of industrial accidents or near accidents were also reported by this group which usually occurred in situations requiring control of a number of factors, e.g., a sprayer-hose while balancing on a scaffold. Other interexposure behavior changes commonly reported by these workers are well known to behavioral toxicologists. They included nervousness, irritability, uncertainty, and changes in sleep patterns.

No significant neurological deficits were found in cranial nerve functions, gait, power, coordination or reflexes. One patient had a moderately coarse tremor of both upper extremities and another was observed to have a few myoclonic jerking movements. Two older patients had diminished vibratory sensibility in the lower extremities. Only minor mental deficits were found in four of the patients; the most common of these (3) was a positive Face-Hand Test. The four channels of EEG data were derived from six posterior electrodes symmetrically distributed on the two sides. Normal alpha rhythms were present in all subjects and in two there was a large amount of 18-22 wave/sec. beta as well. No unusual potentials were observed, but it was noted that the three oldest subjects in this group (ages 58, 60 and 61) showed many more state changes than did the younger ones under the same conditions of recording. In these three, frequent, and sometimes long periods of drowsiness and light sleep patterns were seen. The differences proved stable in a few repeat examinations at different times of day; the second tracing being recorded at the end of a regular work day. The EEG samples were too restricted to justify more elaborate computational analyses of the data.

A periodic patterned visual stimulus was employed at a number of different rates, singly, and in pairs separated by variable time intervals, to study visual evoked potentials. No reliable differences in the averaged responses were apparent on inspection, but a firm judgement awaits the computation of variances.

The clinical and electrophysiological data described above suggest that the most productive measures of neurological deficits in workers exposed to volatile toxins are likely to be those dealing with acquisition and utilization of sensory data. Eye movement tracking and sequential scanning strategies generated in response to visual display may be among the most sensitive of these tests.

Further, we have the impression in these pilot studies that the principal long range neurotoxic effects of these materials (or the effects of exposure on middle-aged workers) are related to premature aging. The most sensitive and reliable objective test for this is of course quantitative study of sleep EEGs.

2. Respiratory tract

a. Acute symptoms

Irritation of the upper airways and conjunctiva and tracheobronchial irritation were also reported with significant frequency. (45 percent and 38 percent respectively of all examined painters). When compared with (Table B3) the prevalence of such symptoms in other workers (tile floor workers, tapers, sandblasters and metal painters) no significant differences were found. This finding may reflect the presence of irritative particulate matter, which is known to be widespread in the last mentioned trades.

Upper airways and tracheobronchial irritative symptoms were more common in epoxy exposed painters than in painters without epoxy exposure (Table B4); tracheobronchial irritation was twice as common in epoxy exposed workers (52 percent of those examined).

Skin changes, including allergic skin reactions and chronic dermatitis, were also more frequent in workers with epoxy exposure.

b. Chronic bronchitis

Chronic bronchitis (according to the MRC criteria) was found in 30% of all examined workers. The highest prevalence was found (Table B5) in sandblasters (38%), immediately followed by the paint manufacturers (33%) and painters (28%) and floor tile workers (29%). The highest prevalence of chronic bronchitis in sandblasters is probably due to their complex occupational exposure to both inorganic particulate matter, mainly silica, and various solvents. The finding of chronic bronchitis in almost one-third of the examined painters was given special attention, and a detailed analysis, taking into account the smoking habits and location was undertaken (Table B6).

Several interesting facts emerged from this analysis. While it was expected that the current smokers would have the highest prevalence of chronic bronchitis, it was rather striking to find between 11 and 14% of workers who had never smoked to have such a problem. Also, the prevalence of chronic bronchitis in persons who had always smoked only a pipe and/or cigar (20 and 24%) was unexpected.

Another finding which resulted from the analysis of the three groups examined (according to location) was that the St. Louis group had a higher prevalence of chronic bronchitis in smokers, as well as in ex-smokers and nonsmokers.

The possibile explanation of these findings was thought to be the occupational exposure to irritant fumes and dusts which most probably contribute to the development of chronic bronchitis in non-smokers, pipe and/or cigar smokers and to the persistence of chronic bronchitis symptoms in ex-smokers, even after many years of non-smoking.

c. Pulmonary function tests

The pulmonary function tests (Table B7-B9) showed a rather impressive prevalence of obstructive ventilatory dysfunction in painters. More than half of the smokers and ex-smokers had obstructive respiratory dysfunction, but, 48 percent of painters who had smoked only a cigar and/or pipe had similar findings. The most striking fact was that 26 percent of non-smokers also had obstructive ventilatory dysfunction.

The results of the pulmonary function tests correlated very well with the diagnosis of chronic bronchitis (MRC questionnaire). Again, the St. Louis Group showed the highest prevalence of obstructive ventilatory dysfunction.

Since chronic bronchitis and obstructive ventilatory dysfunction were found with significant prevalence in painters who had either never smoked or had smoked only cigars and/or pipes, a contributory factor of occupational origin, related to the irritative effects of many solvents and dusts seems very likely. The probability of such a casual relationship was strengthened when an analysis of the pulmonary function tests in workers with a history of irritative symptoms of the upper airways and tracheobronchial tree was undertaken. More than 80 percent of painters who had repeatedly experienced irritative symptoms during their work, developed obstructive ventilatory dysfunction, non-smokers, cigar and/or pipe smokers included.

Restrictive ventilatory dysfunction was a much less frequent finding, even in the trades with known asbestos exposure, such as tapers (Table B10).

3. Chest x-ray findings

The chest x-ray films were read according to the International Classification ILO U/C.

The finding of small rounded opacities, typical for silicosis in 8 percent of the sandblasters, showed that the hazard of developing silicosis in this trade is significant, although much of the work is done outdoors, and most workers have an intermittent type of work schedule (Table B11).

The prevalence of chest x-ray changes in the painters and paint manufacturers was less expected: parenchymal changes were present in 14 percent of all examined painters (5 percent had small rounded opacities and 9 percent had small irregular opacities), and pleural changes (pleural thickening and/or calcification) in 10 percent of painters. The overall prevalence of chest x-ray abnormalities was 20 percent. These findings indicate that painters have a quite prominent hazard of developing pneumoconiosis.

The detailed analysis of the occupational histories in the examined painters revealed that 57 had had indirect asbestos exposure (working in areas where asbestos was handled by other workers), 41 had had indirect silica exposure and 16 had had both indirect asbestos and silica exposure.
The possibility of other varieties of particulate matter contributing to the development of a "mixed dust" pneumoconiosis deserves special attention and further research is needed in this area.

Painters are exposed to a variety of solvents, some of which are known to be toxic for the liver, while the hepatotoxicity of others has still to be evaluated.

4. Liver

Special attention was given to the liver size and firmness, and to other possible pathological changes of the liver and spleen during the physical examination.

Laboratory workup of the examined workers included liver function tests - bilirubin, alkaline phosphatase, SGPT, SGOT and LDH.

The analysis of data revealed that an enlarged liver was not a frequent finding (between 4 and 6 percent of examined workers in the Toledo, Los Angeles and St. Louis Groups). Most of the liver function tests mentioned showed abnormalities to be present in a rather low percentage of those examined (less than 10 percent), but alkaline phosphatase elevation was more frequent a finding in the St. Louis Group, where the prevalence of this abnormality rose to 38 percent of examined painters (Table B12). Various factors have to be considered in an attempt to explain these findings. Careful histories of drinking habits had shown that there was a significant alcohol intake in 28 percent of St. Louis painters, while in the Toledo and Los Angeles Groups, this was the case in only 21 percent of examined painters. Age distribution may also contribute to a higher prevalence of increased alkaline phosphatase levels, when the older age groups are better represented. This factor did not seem to play a significant role in the differences observed between the St. Louis Group and the other two groups.

Since several other occupationally induced abnormalities were more frequently found in the St. Louis Group, it is possible that higher exposure levels to potentially toxic substances, and more frequent such episodes may have contributed to the much higher prevalence of increased alkaline phosphatase. The prevalence of liver dysfunction in painters and the etiologic contribution of some of the chemical handled by these workers deserves further attention and should be studied in depth (Table B13).

5. Hematological results

Complete blood cell counts were undertaken in order to detect any possible toxic effects on the bone marrow and/or blood cells, since the use of aromatic hydrocarbon solvents (benzen, toluene with significant benzene admixture, styrene with possible benzene content) was known to have occurred, at least in the past. Slight anemia (hemoglobin less than 14gm/100ml) was detected in 11 percent of the painters, and slightly reduced white blood counts (less than 5000/mm^3) in 10 percent of cases (Table B14). While these abnormalities were more frequently found with painters than with the other trades, the differences were not impressive, and the fact that only minor abnormalities were detected seemed to indicate that toxic effects on the bone marrow were not a prominent problem in the population studied.

No evidence for recent increased lead or cadmium absorption or toxicity was found. Lead blood levels and urinary cadmium were found within normal limits. Neverthless, there were several metal painters who had experienced lead poisoning in the past, in some of them lead colic had been diagnosed.

An attempt was also made to estimate blood and urine titanium levels, but no levels exceeding the detectable limits of the method used (0.5mg/l in urine; 0.25mg/l in blood) were found.

6. History of malignancy

A history of malignancy (all sites) was elicited in 33 (4 percent) of the examined population (Table 15), in most (23) of the cases a cancer of the skin had been successfully treated, but there were also 3 persons who had undergone surgery for bronchial carcinoma and 3 for carcinoma of the larynx. One additional case of cancer of the lung was diagnosed during the survey.

7. Special laboratory tests

Two non-invasive techniques for early detection of malignancies are under investigation by our laboratory for possible use as screening tools in high risk occupational groups. Serum carcinoembryonic antigen (CEA) and sputum cytopathology were included in the protocol of this field survey.

a. Sputum cytopathology

Cytopathologic changes seen in cells exfoliated from the bronchial linings may reflect a wide variety of conditions including early lung malignancies. Cells from a coughed sputum specimen are staged according to five categories of criteria of cellular abnormality which are felt to reflect progressively more severe alteration, the most serious and severe being cancer. The stages of mild, moderate and marked atypia are thought to be "pre-malignant" in the sense that they are in the chain of progressive changes which may result in cancer. Most persons with mild or moderate atypia do not develop a lung neoplasm. However, they may have a greater risk than others with normal sputum cytopathology. Individuals with marked atypia do not have cancer, but may be developing it, and they are known to have a high probability of developing a malignancy if some type of intervention is not instituted; i.e. stopping cigarette smoking, or ceasing a hazardous exposure such as Uranium mining. The three stages of atypia are felt to be reversible, while the stage of carcinoma is not.

The atypias may also be the result of acute infection, (thus multiple specimens are more reliable) or chronic respiratory irritation such as the inhalation of irritant dusts, fumes or vapors. The possible combined effect of cigarette smoking and occupational exposure to irritants on sputum cytopathology is not known.

In this pilot study, one aerosol induced sputum specimen was collected at the time of examination. All individuals over the age of 40 were included. Other investigators have shown a low yield of adequate specimens from those under 40 as well as from all nonsmokers. Using the above protocol, which included nonsmokers, 88% of the specimens collected contained adequate material for evaluation of cytopathology.

Table B16 summarizes the sputa cytopathologic findings. Other investigators have reported that cigarette smoking is the most potent inducer of atypical sputa. As can be seen in this table, no significant difference exists between current smokers and ex and nonsmokers, until the severe category is reached. This suggests that the occupational exposure of the nonsmokers may be responsible for the high percentage (54%) of nonsmokers with atypical sputa

These typical sputa findings may be the end result of the previously described frequent upper respiratory and tracheobronchial symptoms experienced on the job by the tradesmen examined. The atypical sputa findings also correlate well with the findings of an increased number of nonsmokers with a history of chronic bronchitis. The combined finding of increased respiratory symptoms, chronic bronchitis, and atypical sputum cytopathology suggests strongly that the occupational exposure to irritants experienced by men in the painting trades may significantly contribute to the appearance of mild and moderately atypical pulmonary cells. It is not known whether these minimal changes will progress to the more serious stages. Long term, prospective follow-up of these men is needed to answer this question.

As can be seen in Table B16, one previously undiagnosed case of lung cancer was detected using sputum cytopathology. That man is currently under therapy. The second case, from a more recent survey, has not had a tumor localized yet. His cytology was "highly suspicious but not conclusive for malignancy." He is being followed closely by his personal physician. In addition, one individual with a previously known laryngeal carcinoma was examined during the survey and he also had malignant cells in his sputa, however, he was not included in Table B16 because his tumor was already known.

b. Carcinoembryonic antigen

CEA was first described in 1965 and was thought to be specific for entodermally derived tumors. However, further studies have described elevated CEA titers in many other malignancies and also in some non-malignant inflammatory disorders. It is now felt that CEA represents a non-specific cell-phase marker, most likely reflecting cell pathology and degree of cell differentiation. CEA titers below 10ng/ml. are not diagnostic for malignancy; however, individuals in the range 2.6-10ng/ml. are more likely to have an illness than those with titers below 2.6ng/ml. Heavy cigarette smoking, emphysema, excessive alcohol consumption and liver function abnormality are the most common findings in this group. CEA titers above 20ng/ml. are almost always associated with malignancy, or severe medical illness requiring hospitalization. Whether individuals with elevated CEA titers, but without clinically evident malignancies, have a higher risk of developing a malignancy than those with lower CEA titers has not been adequately evaluated.

Serum specimens were collected from 943 tradesmen and were analyzed blind at the Hoffmann-La Roche Inc., Nutley, New Jersey facility under the direction of Dr. Hans Hansen using a modified radioimmunoassay with a sensitivity of ± .5ng/ml.

We have compared the results (Tables B17,B18,B19) with normal individuals who had no known toxic occupational exposures or active medical illnesses. Smoking history was kept constant because of the well known effect of cigarette smoking on CEA titers in the range of 2.6-10ng/ml. The effect of smoking can be seen by comparing the different normal groups. In the non-smokers, 97% had CEA titers below 2.6ng/ml., while only 80% of the current smokers had similar titers.

All trades examined had CEA distributions significantly different from the normals. Any additive effect of occupational exposures and cigarette smoking would not be present in the nonsmoking group, yet they were significantly different from the normals, suggesting an external work environment effect. The distribution of the nonsmoking tradesmen is identical to the smoking normals.

Other investigators have shown that CEA titers are often correlated with abnormal liver function tests and alcohol consumption. The previously discussed increased number of painting tradesmen with abnormal liver function tests correlates well with the finding of elevated CEA titers. Both of these findings suggest that these workers' occupational exposures not only cause acute symptoms, but are reflected in liver function abnormality as well as CEA titers. The significance of the CEA titers in the 2.6-10ng/ml. range is not known, however, prospective follow-up on this group is planned to learn whether the elevated titer group has a different morbidity and mortality experience than the normal CEA

It is interesting to note that combining the sputum cytopathology results with the CEA titers gives more information on possible pulmonary malignancy than either test alone. All of the individuals with markedly atypical sputa had elevated CEA titers. The one positive cytology had a CEA of 345ng/ml. The "suggestive but not conclusive" individual had a normal CEA. This individual will be followed closely to watch for a rising CEA titer. Only 39% of the moderate atypias had elevated CEA titers. Whether these men have a higher risk than the individuals with only one of the tests abnormal will only be answered by prospective follow-up. It will be interesting to see if the moderate atypias with abnormal CEA's will soon progress to markedly atypical sputa. All of these men have been advised to stop smoking and remain under close medical observation. Our laboratory plans to continue to follow these men every six months with CEA and sputum cytology.

Table B1

Acute symptoms of overexposure in painters

Symptoms	Actively employed (337) No.	Actively employed (337) %	Previously employed (148) No.	Previously employed (148) %	Total (485) No.	Total (485) %
Upper respiratory and conjunctival irritation	159	47%	59	40%	218	45%
Tracheo-bronchial irritation	140	42%	42	28%	182	38%
Prenarcotic symptoms	243	72%	101	68%	344	71%
Digestive symptoms	72	21%	23	16%	95	20%

Table B2

Acute symptoms of overexposure in 485 painters (three locations)

Symptoms	Los Angeles (218)		Toledo (78)		St. Louis (189)		Total (485)	
	No.	%	No.	%	No.	%	No.	%
Upper respiratory and conjunctival irritation	109	50%	26	33%	83	44%	218	45%
Tracheo-bronchial irritation	93	43%	26	33%	64	34%	182	38%
Prenarcotic symptoms	152	70%	64	82%	128	68%	343	71%
Digestive symptoms	46	21%	16	21%	33	17%	95	20%
Loss of consciousness	7	3.2%	3	4%	9	5%	19	4%

Table B3

Acute symptoms of overexposure

Symptoms	Painters (485)		Paint manufacturers (30)		Tile floor (31)		Tapers (116)		Metal painters and sandblasters (77)	
	No.	%	No.	%	No.	%	No.	%	No.	%
Upper respiratory and conjunctival irritation	218	45%	10	33%	12	39%	31	27%	47	61%
Tracheo-bronchial irritation	182	38%	13	43%	10	32%	27	23%	40	52%
Prenarcotic symptoms	343	71%	11	37%	15	48%	6	5%	63	82%
Digestive symptoms	95	20%	2	7%	1	3%	1	1%	22	29%

Table B4

Acute symptoms in epoxy exposed painters

Symptoms	Epoxy exposed (225) No.	Epoxy exposed (225) %	Non-epoxy exposed (260) No.	Non-epoxy exposed (260) %
Upper respiratory and conjunctival irritation	128	57%	90	35%
Tracheo-bronchial irritation	116	52%	66	25%
Prenarcotic symptoms	188	84%	156	60%
Digestive symptoms	58	26%	37	14%
Skin changes	48	21%	13	5%

Table B5

Chronic bronchitis (by history - MRC)

	Total number examined	Chronic bronchitis	
		No.	%
Painters	485	135	28%
Paint manufacturers	30	10	33%
Tapers	116	20	17%
Floor tile workers	31	9	29%
Sandblasters	77	29	38%
TiO_2	206	45	22%

Table B6

Chronic bronchitis in painters

	Total number examined	Chronic bronchitis	Smokers		Ex-smokers		Pipe and/or cigars		Non-smokers	
			Total No.	Chronic bronchitis	Total No.	Chronic bronchitis	Total No.	Chronic bronchitis	Total No.	Chronic bronchitis
Los Angeles	218	56 (26%)	77	40 (52%)	89	8 (9%)	15	3 (20%)	37	5 (14%)
Toledo	77	18 (23%)	45	16 (36%)	15	0	3	0	14	2 (14%)
St. Louis	179	62 (35%)	72	40 (56%)	62	15 (24%)	17	4 (24%)	28	3 (11%)
Totals	474	136 (28%)	194	96 (49.5%)	166	23 (14%)	35	7 (20%)	79	10 (13%)

Table B7

Respiratory function impairment in painters (smokers)

	Total number tested	Restrictive dysfunction	Obstructive dysfunction
Los Angeles	76	9 (12%)	33 (43%)
Toledo	43	2 (5%)	23 (53%)
St. Louis	70	6 (8%)	38 (54%)

Table B8

Respiratory function impairment in painters

Ex-smokers

	Total number tested	Restrictive dysfunction		Obstructive dysfunction	
Los Angeles	88	4	5%	43	49%
Toledo	13	1	8%	7	54%
St. Louis	52	2	4%	27	52%

Table B9

Respiratory function impairment in painters (non-smokers) (cigar and/or pipe smokers)

	Never smoked					Pipe and/or cigar only				
	Total No. tested	Restrictive ventilatory impairment		Obstructive ventilatory impairment		Total No. tested	Restrictive ventilatory impairment		Obstructive ventilatory impairment	
Los Angeles	35	1	3%	11	31%	15	1	7%	5	33%
Toledo	13	--		--		3	--		2	66%
St. Louis	25	1	4%	8	32%	15	--		9	60%
Total	73	2	3%	19	26%	33	1	3%	16	48%

Table B10

Respiratory function impairment in workers with mineral dust exposure

	Number	Sandblasters Total number (73)		Number	Tapers Total number (106)	
		Restrictive impairment	Obstructive impairment		Restrictive impairment	Obstructive impairment
Smokers	41	3 (7%)	23 (56%)	51	2 (4%)	19 (37%)
Ex-smokers	25	3 (12%)	13 (52%)	33	1 (3%)	12 (36%)
Non-smokers	7	0	1 (14%)	22	2 (9%)	6 (27%)

Table B11

Chest X-ray changes

Exposure	Total number workers examined	Small rounded opacities (p, q or r; 1/0 or more)		Small irregular opacities (s or t; 1/0 or more)		Pleural thickening and/or calcification		Total parenchymal and/or pleural changes	
		No.	%	No.	%	No.	%	No.	%
Silica (sandblasters)	76	6	8%	1	1%	7	9%	12	16%
Asbestos (tapers)	113	7	6%	3	3%	2	2%	11	10%
Painters and paint manufacturers	524	26	5%	48	9%*	52	10%*	105	20%*

* There were 57 workers with indirect asbestos exposure, 41 with indirect silica exposure and 16 with both indirect asbestos and silica exposure.

Table B12

Liver function tests in Painters

	Los Angeles Total number tested (218)		Toledo Total number tested (78)		St. Louis Total number tested (207)	
	No.	%	No.	%	No.	%
Bilirubin > 1.0mg.	9 (1)*	4%	3	4%	7	3%
Alkaline phosphatase	33 (5)*	15%	6 (2)*	8%	78 (21)*	38%
SGPT	8 (2)*	4%	0		2	1%
SGOT	11 (4)*	5%	8 (5)*	10%	14 (5)*	7%
LDH	3	1%	0		9 (2)*	4%
Enlarged liver	13 (7)*	6%	3 (3)*	4%	10 (4)*	5%
Significant alcohol intake	46	21%	16	21%	57	28%

* Abnormalities in persons with significant alcohol intake

Table B13

Liver function tests in workers of various painting trades

	Painters and Paint manufacturers (503)		Titanium dioxide workers (207)		Tapers (116)		Sandblasters and metal painters (77)	
	No.	%	No.	%	No.	%	No.	%
Bilirubin >1.1	19 (1)*	4%	8 (3)*	4%	5	4%	1 (1)*	1%
Alkaline phosphatase >85	117 (28)*	23%	35 (20)*	17%	27 (9)*	23%	14 (13)*	18%
SGPT >52	10 (2)*	2%	4 (2)*	2%	9 (2)*	8%	1	1%
SGOT >40	33 (14)*	7%	5 (3)*	2%	9 (2)*	8%	5 (5)*	6%
LDH >225	12 (2)*	2%	5 (1)*	2%	7 (4)*	6%	1 (1)*	1%

* Number of cases with significant alcohol intake

Table B14

Hematologic changes

Trade	Total number examined	Hemoglobin <14gm/100ml		WBC $<5000/mm^3$	
		No.	%	No.	%
Painters	465	50	11%	47	10%
Paint manufacturers	26	2	8%	1	4%
Tapers	114	8	7%	2	2%

Table B15

Malignancies (by history)

	Total number examined	Skin	Lung	Larynx	G.I.	Other
Painters and paint manufacturers	507	18 (4%)	4 (0.8%)	2 (0.4%)	2 (0.4%)	0
Tapers	116	1 (0.9%)	-	-	-	1 (0.9%)
Others	156	4 (2.6%)	-	1 (0.6%)	-	
Total	779	23 (3%)	4 (0.5%)	3 (0.4%)	2 (0.3%)	1 (0.1%)

Table B16

Sputum cytology and smoking

Smoking history	Number tested	Normal	Mild atypia	Moderate atypia	Marked atypia	Malignant
Smokers	296	91 (31%)	181 (61%)	20 (7%)	3 (1%)	1 (0.3%)
Ex-smokers	203	89 (44%)	99 (49%)	13 (6.5%)	1 (0.5%)	1 (0.5%)
Non-smokers	93	43 (46%)	44 (47%)	6 (7%)	0	0

Table B17

CEA Assay

Current Smokers

Trade	Number	0-2.5ng/ml.	2.6-5ng/ml.	5.1-10ng/ml.	10.1+ng/ml.
Painters	202	86 (43%)	85 (42%)	26 (13%)	5 (2%)
Tapers	52	28 (54%)	18 (34.5%)	5 (9.5%)	1 (2%)
Silica exposed	46	21 (46%)	18 (39%)	5 (11%)	1 (2%)
TiO_2	82	32 (39%)	41 (50%)	7 (8.5%)	2 (1.5%)
Other	38	17 (45%)	16 (42%)	5 (13%)	0
Normals	620	502 (81%)	93 (15%)	19 (3.3%)	6 (0.7%)

Table B18

CEA

Ex-Smokers

Trade	Number	0-2.5ng/ml.	2.6-5ng/ml.	5.1-10ng/ml.	10.1+ng/ml
Painters	164	108 (66%)	41 (25%)	14 (9%)	1 (0.6%)
Tapers	37	28 (76%)	7 (19%)	2 (5%)	0
Silica exposed	24	19 (79%)	5 (21%)	0	0
TiO_2	72	52 (72%)	19 (26%)	1 (1%)	0
Other	26	20 (77%)	5 (19%)	1 (4%)	0
Normals	235	219 (93%)	12 (5.2%)	2 (0.9%)	2 (0.9%)

Table B19

CEA Assay

Non-Smokers

Trade	Number	0-2.5ng/ml.	2.6-5ng/ml.	5.1-10ng/ml.	10.1+ng/ml.
Painters	115	88 (77%)	20 (17%)	6 (5%)	1 (1%)
Tapers	20	16 (80%)	2 (10%)	2 (10%)	0
Silica exposed	8	5 (63%)	1 (12%)	2 (25%)	0
TiO_2	37	30 (81%)	7 (19%)	0	0
Other	20	15 (75%)	4 (20%)	1 (5%)	0
Normals	892	865 (97%)	25 (2.8%)	2 (0.2%)	0

C. Dry wall taping

Dry wall construction began to replace masonry to a significant degree after World War II. At the present time, it has assumed a dominant role in the construction industry. Taping of joints in such dry wall construction is required, and consists of applying and finishing spackle material at wallborad joints. We have found that these work procedures may be associated with significant asbestos exposure.

Tapers spend approximately 5-10% of their working time sanding dried spackle material, and thus experience intermittent asbestos exposure. Significant exposure may also occur among painters who occasionally apply or sand spackle and taping compounds. It is estimated that some 75,000 construction workers are currently employed in this work in the U.S. Moreover, carpenters, electricians and plumbers may work in the vicinity of wallboard finishing.

The purpose of the following study is to describe the work procedures and to focus attention on the health hazards from asbestos exposure in dry wall construction.

1. Work procedures:

a. Mixing:

Prior to applying the spackle at the wallboard joints the compound is prepared in the following way:

Half of a 25 lb. bag of dry wall taping compound is poured into a 5 gallon bucket containing about 2 gallons of water. The mixture is stirred until it has obtained a certain degree of consistency, whereupon the remainder of the dry mix is added. The mixture is again stirred. This operation takes the taping worker approximately 60-90 seconds.

Until Pre-Mix material came into use in 1965, the mixing procedure was commonly performed three times a day.

The tables in this section are labeled "C- " and follow the end of this section (Page 53).

b. Sanding:

After the taping compound has been applied at the wallboard joint and has dried, the surface is sanded. Two different sanding techniques are used.

Pole sanding:

This is the most common technique presently used. An experienced taper is able to sand the joints of a 20x9 foot wall in approximately 3 minutes. The apparatus used consists of a 5 foot pole with a steel base to which a piece of sandpaper is attached. After a room has been pole sanded, it is usually finished with hand sanding, before the second coat of taping compound is applied. This is usually not sanded.

Hand sanding:

This technique is preferred to pole sanding by some tapers, since this procedure allows better visual and manual control over the operation. Hand sanding is also required in those situations in which the space relationships prevent the taper from using the pole sanding technique.

2. Analysis of materials

Analysis of taping compounds and spackle materials indicates that a number of biologically active minerals are to be found. The compounds that have been analyzed by x-ray powder diffraction, optical and electron microscopy include both dry wall and paste types. Of 15 industrial dry wall taping and spacklin compounds, 13 contained asbestos. (Table C1) In addition, 3 compounds contained two different types of asbestos, namely chrysotile and tremolite. Quartz and other silicates were also present in some of the compounds. Thus, the results of our analyses show that most of the taping compounds used in the U.S. Construction Industry contain asbestos on the order of 4-5%. One compound contained 10-15% chrysotile and 8-12% tremolite.

3. Dust concentration

We have further done a series of dust counts and found that fiber counts are considerable. Air samples taken in the breathing zone of dry wall tapers show that fiber concentrations several times the maximum level permitted by the United States Government regulations are released during sanding of wall board taping compounds. It should also be emphasized that the asbestos fiber concentrations generated by sanding are similar to those measured in the work environment of asbestos insulation workers. Tables C 2 and C 3 show fiber concentrations experienced during pole and hand sanding of dry wall taping compounds. Pole sanding is the most common technique of sanding at the present time. During pole sanding, 7 of the 10 samples exceeded the threshold limit value of 5 fibers ml greater than 5μm. Hand sanding, which is preferred by some tapers, generated fiber concentrations close to or in excess of the threshold limit value.

During mixing of dry wall taping comounds, fiber counts 7 to 10 times the threshold limit value were observed. (Table C4) The background data indicate that there are detectable fiber concentrations in adjacent rooms during the mixing procedure.

It is of considerable importance to emphasize that the fiber concentrations measured by standard procedures using optical microscopy, are in fact unreliable indices of the actual asbestos posure. Electron microscopic analyses show a large number of fibers too fine to be seen by optical microscopy. The disease inducing potential of these fibers, many of which are shorter than 5μm in length and 300Å in width is readily recognized. It may be then that optical fiber counts grossly underestimate the dust levels which actually obtain.

The presence of particularly small fibers in this asbestos trade is also suggested by an analysis in our laboratory of the lung of a taping worker who died of lung cancer. Innumerable fibers were present. However, virtually none were seen by optical microscopy. These were only visible with the electron microscope. Whether the small fiber size encountered in this trade is a consequence of the original material used, or the work procedures, remains to be further elucidated.

4. X-ray abnormalities

We have undertaken a preliminary survey of taping workers in the United States in order to determine whether significant asbestos disease exists among them. The taping workers were members of the Local Unions 1974 and 1976 of Dry Wall Taper and Pointers in New York, N.Y. and Hopelawn, N.J. respectively.

The age distribution of the examined taping workers as presented in Table C5 The duration of exposure, summarized in Table C6, shows that 79% of the workers had ten years of exposure or more. The x-ray abnormalities among 108 tapers are summarized in Tables C7 and C8. Each film was read by 5 physicians and a consensus reading was thereafter obtained. The x-ray abnormalities were classifed according to the ILO/UC International Classification of Radiographs of Pneumoconioses. 48 abnormal films were found among the examined, i.e. in 44.4%.

Pleural fibrosis characteristic for asbestosis was found in 12 cases, of which 4 had pleural disease as the only indication of asbestos exposure. In summary, 48.1% of the examined tapers had abnormalities on their chest x-ray.

In comparison with roentgenographic changes observed in asbestos insulation workers (Table C9) a similarity in morbidity pattern is discernible, especially in the group with 10-19 years of exposure. The difference in total number of examined individuals may account for some of the differences and will be studied further.

Included in the description of clinical findings of Painters and other trades are data on individuals classified as "tapers". For the most part, these are individuals who had performed some taping of wallboard and spackling compounds for at least two years or longer. Thus, individuals were included who performed dry wall taping only occasionally. As can be seen, the disease manifestations of such occasional exposure is considerably less than that of full-time tapers and differs little from other general painters who also had indirect and some direct exposure to asbestos in general painting activities.

In conclusion, we have found that full-time taping workers in dry wall construction do have risk of exposure to asbestos and perhaps to other minerals as well. Our preliminary findings suggest that asbestos disease is an important hazard in this trade, which has not been previously investigated. We recommend that appropriate measures be taken as soon as possible in order to prevent further asbestos exposure.

Table C 1

Mineral content of fifteen industrial dry wall taping and spackling compounds

Study Number

Mineral Phase	1	2	3	4	5	6	7	8	9	10	11	12	13	14	1
Chrysotile	6-8%	5-10%		6-8%	5-10%	5-12%	5-10%	5-10%	5-10%	5-10%		5-8%	10-15%		8-
Tremolite	4-7%											4-7%	8-12%	2-3%	
Talc		++	+									++	++	++	+
Quartz	+	++	+	++	++	++					++			++	
Mica	++	++		+	++	++	++	++	++	++		++	++	++	++
Kaolinite		+			+	++	+	+	+	+		+		+	
Calcite	+						++		+	++	++	++	++	++	+
Dolomite	++						++	++		++	+				
Plaster of Paris (bassanite)		++	++	++	++	++			++						

Major constituents are indicated by ++; minor constituents by +; weight percentages of asbestos minerals (chrysotile and tremolite) were determined by step-scanning x-ray diffraction.

Table C 2

PERSONAL SAMPLES - DRY WALL TAPING COMPOUNDS

Operation	Concentration (fibers/ml)
Pole Sanding of Dry Wall Taping Compounds	1.2
" " " " " " "	10.6
" " " " " " "	11.6
" " " " " " "	8.6
" " " " " " "	15.4
" " " " " " "	18.3
" " " " " " "	2.4
" " " " " " "	19.3
" " " " " " "	2.6
" " " " " " "	8.9

Background to Pole Sanding	Distance	Concentration (fibers/ml)
Air sampling in same room	8 ft.	2.5
" " " " "	8 ft.	19.8
" " " " "	10 ft.	3.5
Air sampling in adjacent room	21 ft	0.7
" " " " "	25 ft.	8.8

Table C 3

PERSONAL SAMPLES - DRY WALL TAPING OPERATIONS

Operation	Concentration (fibers/ml)
Hand sanding of dry wall taping compounds	
" " " " " " "	1.3
" " " " " " "	4.8
" " " " " " "	5.0
" " " " " " "	4.7
" " " " " " "	2.9
" " " " " " "	6.0
" " " " " " "	1.3
" " " " " " "	5.8
" " " " " " "	6.5
" " " " " " "	16.9
" " " " " " "	3.2

Background to hand sanding	Distance	Concentration (fibers/ml)
Air sampling in same room	8 ft.	2.1
" " " " "	8 ft.	2.5
Air sampling in adjacent room	15 ft.	7.1
" " " " "	15 ft.	1.5

Table C 4

PERSONAL SAMPLES - DRY WALL TAPING COMPOUND

Operation	Concentration (fibers/ml)
Mixing of dry wall taping compounds	59.0
" " " " " "	35.4

Background to dry mixing	Distance	Time Lapse	Concentration (fibers/ml)
Air sampling in same room	10 ft.	0 min.	3.8
" " " " "	20 ft.	0 min.	13.1
" " " " "	9 ft.	15 min.	0.5
Air sampling in adjacent room	16 ft.	0 min.	2.1
" " " " "	35 ft.	0 min.	3.1

Operation	Distance from Sanding	Time Lapse (After Sanding)	Concentration (fibers/ml)
Sweeping floors after sanding	0-10 ft.	35 min.	26.4
" " " "	50 ft.	15 min.	41.4

Table C 5

Age distribution of 114 drywall tapers

Duration of exposure (in years)	Total workers	21-30 years	31-40 years	41-50 years	51-60 years	61+ years
0-4	6	3	2	1	-	-
5-9	18	9	5	3	1	-
10-14	29	9	13	6	1	-
15-19	25	-	8	11	6	-
20-24	28	-	4	19	3	2
25+	8	-	-	-	4	4
TOTAL	114	21	32	40	15	6
		(18.4%)	(28.1%)	(35.1%)	(13.2%)	(5.2%)

Table C 6

Duration of exposure of 114 drywall tapers

Duration of exposure (in years)	0-4	5-9	10-14	15-19	20-24	25+	Total
Number of workers examined	6 (5%)	18 (16%)	29 (25%)	25 (22%)	28 (25%)	8 (7%)	114

Table C 7

X-ray abnormalities among 108 Tapers (drywall construction)

Years from onset of exposure	No.	X-ray abnormality (ILO/UC Classification)						
		0/0	0/1	1/0	1/1	1/2	2/1	2/2
0-4	4	1	1	0	2	0		
5-9	15	5	2	1	7	0		
10-14	25	13	2	6	3	1		
15-19	28	9	8	7	3	1		
20-24	28	6	10	6	4	1		1
25-29	8	1	2	1	4	0		
	108			21	23	3		1

Total abnormal x-rays (parenchymal changes): 44.4%

Table C 8

PLEURAL FIBROSIS AMONG 108 DRY WALL TAPERS

Duration from onset of exposure (years)	X-ray abnormality >1/0 (ILO/UC) + pleural	Number of tapers with pleural disease only
10-14	1	1
15-19	3	0
20-24	3	3
25+	1	0
Total	8	4

Table C 9

Roentgenographic changes among asbestos insulation and drywall taping workers

Duration from onset (in years)	X-ray abnormality (grade) 0		1		2		3	
	No.	%	No.	%	No.	%	No.	%
0-9								
(346) Insulators	310	89.6	36	10.4	0	0	0	0
(19) Tapers	9	47.4	10	52.6	0	0	0	0
10-19								
(379) Insulators	212	55.9	158	41.7	9	2.4	0	0
(53) Tapers	32	60.4	21	39.6	0	0	0	0
20-29								
(57) Insulators	11	19.3	35	61.4	7	12.3	4	7.0
(36) Tapers	19	52.8	16	44.4	1	2.8	0	0

D. Titanium Workers

1. Health status of titanium workers

The International Brotherhood of Painters and Allied Trades includes among its members workers who are involved in the manufacture of titanium pigment. With the assistance of the union, we examined 207 of such workers. This group was of interest in our pilot study because it allows the isolation of a group exposed to only one chemical that is in paint, titanium dioxide. In recent years, titanium dioxide (TiO_2) has largely replaced white lead and zinc pigments in the manufacture of interior and exterior white paint products. The titanium pigment is thought to be of low toxicity. In the manufacturing process itself and the application of paints containing titanium pigment exposure to the agent may occur. If pigment manufacturers with the highest exposure to this compound are found to be unaffected by it, then its safety can be reasonably assumed for painters and others.

The examination of this group of pigment workers was important in another way to our study of painting trades workers. This workforce has little exposure to the other compounds which are in paints-- solvents, fungicides, pigments, etc. The results observed in these studies of titanium workers can serve as an internal control for the laboratory and questionnaire results observed among general painters.

[illegible]

2. Titanium pigment manufacture; description of the process

Titanium dioxide pigments are currently produced by two processes, the classical sulfate process and the recently introduced chloride process. The process used in the plant we studied was the sulfate process. In this process, the ilmenite ore is dried, milled and then digested in 16-ton loads with sulfuric acid and water. When the temperature reaches about 160°C, a violent exothermic reaction occurs, releasing various fumes of sulfur oxides, and the mixture is converted to a porous cake. The case is then extracted with water. Next the sulfate solution goes to clarification tanks, crystallization and concentration. Precipitation tanks then receive the clarified solution, where the crystal structure is reformed and the solution is transformed to a white pulp. The precipitate is allowed to settle and anhydrous titanium dioxide settles out of solution. This goes through a series of filters and is washed, leaving pure titanium hydrate. This is then fed into calciners, huge rotating tub-like ovens. This requires a half a day where the white pulp dries to a powder and extreme heat develops its crystalline form and quality of opacity. During this process water and sulfur trioxide, SO_3, are driven off. The pigment is then ground to a uniform size of about 1 μ. During the entire process the workers are exposed to TiO_2 dust, various sulfur oxides and sulfuric acid fume and mist.

3. Examination of workers

Workers for examination were recruited from among the workforce at a titanium pigment plant in St. Louis, Missouri. Those with 20 or more years of experience in the plant were encouraged by the union to come in for physical examination. Two-~~hundred and~~ seven workers, or 27% of the workforce of 759 men were examined, including 52% of those with more than 20 years of service in the plant. One hundred and twenty-nine of 254 (51%) with 20-29 years of experience, and 35 of 63 workers in the plant (56%) with more than 30 years of experience, were examined. The examination was identical to that given the general painters in our survey, and included the questionnaire, physical examination, laboratory tests, pulmonary function examination, and x-rays that have been done in the rest of the country. Especially stressed in this part of the examination was the history of acute respiratory symptoms, history of cancer, and, for comparison with the general painters, history of central nervous system symptoms.

An attempt was made to grade each worker's exposure to titanium dioxide dust, sulfur dioxides, sulfates, and/or sulfuric acid. All jobs in the plant were given an exposure rating of 1 to 3 (see Table 1).* The basis for these scores was published information about the process and the particular job classification of the worker. Each worker was given an index number for his total exposure to sulfuric acid and TiO_2, derived by multiplying the years of exposure at each exposure level by the weight of that exposure level. The job categories were then added together for each individual worker. Three categories were derived based on an index of "low" (less than, or equal to 40), "moderate" (41-69) and "high" (greater than or equal to 70).

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*The tables in this section are labeled "D- " and follow the end of this section. (page 71)

Spirometric measurements were again made on the Systems Research Laboratory's predictive pulmonary screener, as with other examinations. Flow-volume curves were obtained, using a Vertek 3500 Fleish pneumotachygraph, recording both flow and integrated volume signals on a Hudson X-Y plotter. Each instrument was calibrated in the laboratory before and after the survey and in the field against other instruments, as well as by using a calibrating syringe and known normal subjects. Workers were standing, and nose clips were placed. Unless the first flow-volume curve was normal, the best of at least three forced expirations was used. Spirometry was completed in 197 subjects. In 25 subjects, only the FVC and the FEV$_1$ were obtained. The predicted values of Morris et. al. were used for forced vital capacity (FVC) and mid-maximal expiratory flow (MMEFR). Other criteria for abnormality are indicated in the appropriate tables.

Chest x-rays (14" x 17" PA ~~films) were~~ taken on 201 ~~of the~~ 207 workers who were examined. The chest x-rays were read by a panel of five experienced physicians, and the results coded according to the ILO/UC pneumoconiosis classification. A consensus reading was used for statistical analysis. Standard laboratory tests ("12-channel" and "6-channel") were run on all workers enrolled in the examination. These tests were performed by the same laboratory which performed those for the general painters. A carcinoma-embryonic antigen (CEA) test was performed by the Hoffman-LaRoche Research Laboratories. The results were analyzed in relationship to smoking and inter-group comparisons were performed between the painters and the titanium workers. Sputum cytology was studied in the laboratory of Dr. Eugene G. Saccomanno.

4. Clinical Findings - Results

Age

The mean age of all 207 workers was 51, range 28-66.

Smoking.

There were 91 smokers (44%), 72 ex-smokers (35%), and 38 people who had never smoked (18%). There was no difference in the prevalence of nonsmokers among the age groups (18% age less than 50 years, 19% age greater than 50 years) (Table 4). Similarly, there was virtually no difference in smoking habits among the groups with different durations of exposure (Table 2). This is especially true for the distribution of nonsmokers among these categories.

a. Pulmonary function and x-ray findings

Changes in pulmonary function in relation to age, duration of exposure, smoking, and exposure indices were analyzed (Tables 2, 3). When the population was subdivided by smoking categories, no further significant differences in prevalence of airflow impairment with either longer periods of work or age categories under 50 and over 50 were seen. Forty-one percent of those who had never smoked were found to have airflow impairment. Of the nonsmokers over 50 years of age, 19% had airflow impairment. There were higher percentages among current smokers, and intermediate percentages among former smokers, an example of multi-factor interaction. A significant progression in the prevalence of airflow impairment existed for nonsmokers in Grade 1 exposure as compared to Grade 3 exposure to TiO_2 dust ($0.01<p<0.05$). For Grade 2 versus Grade 3 exposure, the significance level was $p = 0.1$. Similarly, prevalence of airflow obstruction among nonsmokers significantly increased when grades 1 and 3 and Grades 2 and 3 3xposures to sulfur oxide fumes were compared ($p = 0.01$ in both cases). No significant differences in the prevalence of airflow impairment were found among the smokers and ex-smokers for any of the exposure indices. (Tables 5 -6).

b. Symptoms

Our questionnaire included detailed questions on the occurrence of acute upper and lower respiratory irritation. Table 7 shows the prevalence of histories of such symptoms. Of note is the fact that 76% of the population had experienced some form of lower respiratory irritation. (70%, cough; 41%, shortness of breath; 30% , the feeling of chest tightness; 12%, audible wheezing upon exposure to fumes, primarily those of the digestion reaction, or sulfuric acid mist). Nausea and/or vomiting commonly accompanied more severe exposures, as did dizziness and/or headache.

The prevalence of airflow abnormalities among the 197 titanium workers who had undergone pulmonary function tests was examined in relationship to their history of acute symptoms from the irritants present in the workplace. Smoking habits were considered in this analysis. There was no significant difference in the prevalence of airflow impairment among the smoking categories in which no symptoms of acute respiratory irritation were found. In the group with histories of lower respiratory symptoms due to the exposure to irritants, the prevalence of airflow abnormalities was significantly different between smokers and ex-smokers ($X^2 = 6.29$, $0.02>p>0.01$). This statistical difference, as well as those between the other smoking categories, is similar to that which was found in the entire population.

Within the smoking categories significantly different levels of prevalence of impaired airflow occurred between those with and without acute respiratory symptoms only for the ex-smokers ($X^2 = 4.58$, $0.05>p>0.02$). However, the numbers in many of these categories are small and the statistical significance expressed may have little biological meaning (Table 8).

The prevalence of restriction of pulmonary volume was analyzed in relationship to the duration of exposure to the plant environment. Since the prevalence numbers of this abnormality are small, smoking habits could not be considered. Diminished vital capacity unassociated with flow abnormalities occurred in 6% of the population (11 out of 179). "Mixed" abnormalities were more common occurring in 18/197 cases (9%) (Table 9). The more pronounced examples of these abnormalities were present in workers with severe obstructive pulmonary disease.

The prevalence of chronic bronchitis (cough and sputum, fulfilling the MRC criteria) among the 206 titanium workers from whom this history was obtained, was analyzed in relationship to the occurrence of acute symptoms from irritants and smoking habits (Table D-10). Smokers have a significantly greater prevalence of bronchitis than nonsmokers or former smokers (who do not differ from each other significantly in this respect. However, there is no significant difference in prevalence of chronic bronchitis within the smoking categories in relationship to the occurrence of lower respiratory symptoms due to irritant exposure. Although the number of smokers and former smokers in the bronchitis categories are very small, it seems that smoking had a more pronounced effect upon bronchitis prevalence (cough and sputum) than did a history of symptoms due to irritant exposure. Such a history of symptoms did not significantly change the incidence of bronchitis among the different smoking categories.

Of the 201 workers x-rayed, 28 (14%) had abnormal films (Table 11-13). In only one case was the degree of fibrosis greater than the ILO/UC category 1.

Table 12 compares the radiographic findings by length of exposure to the combined titanium dioxide/sulfur fumes. While the group with more than 30 years exposure tended to have a higher prevalence of abnormal x-rays, these numbers were small and did not reach the $p < 0.05$ level of statistical significance. The x-rays were also analyzed in respect to the workers' smoking histories. In Table 13 , the effect of cigarette smoking upon x-ray results can be seen. Current smokers tended to have higher prevalence of abnormal x-rays than ex-smokers or non-smokers. The $p < 0.05$ level of significance again was not quite reached ($X^2 = 3.37$, $0.1 > p > 0.05$. The single case with a more advanced pneumoconiosis (Grade 2/1) had no history of asbestos or silica exposure.

The relationship of diminished pulmonary volume to x-ray abnormalities is shown in Table 14. The numbers are small, but no clear pattern of restrictive disease in relationship to x-ray findings can be seen. It should be remembered that the majority of those workers with diminished vital capacities had flow abnormalities as well, representing people with, primarily, chronic obstructive pulmonary disease.

c: Other findings.

Other acute symptoms

Of interest in respect to a comparison with the general painters in the fact that few central nervous system symptoms were found among any of the titanium workers. Exposure to central nervous system toxic substances (solvents and vapors) occurred in two plant operations -- painting, and bonding of rubber to pipes. In restoof the operations, a few workers reported dizziness, lightheadedness and/or headache and nausea and vomiting along with the respiratory symptoms which occurred on exposure to the digestion fumes. (Table 7,).

Anemia

Since the general painters are exposed to numerous solvents, one of which may be benzene-containing toluol, a packed cell volume was performed on all painters. The titanium group serves as an internal control in this study. The prevalence of anemia among the titanium workers as measured by packed cell volume (<42%) was 12% (26/207).

Sputum cytology and history of malignancy

Only one of the titanium workers had malignant cells category 4a (inconclusive) in th sputum cytology test,. In another section of this report, the distribution of the cytological abnormalities in relation to smoking habits is compared with the distribution of abnormalities found in the rest of the population examined.

A history of malignancy was obtained from two of the workers, one who previously had had a skin cancer removed, and the other who had had a bladder cancer removed.

Liver Function Tests

The prevalence of specific liver function test abnormalities was analyzed in relationship to duration of exposure to the titanium pigment operation. There was no significant variation in the prevalence of these abnormalities with length of exposure to the process (Table 15). In addition, there was no significant difference in the prevalence of these abnormalities in this population as compared to a population of 400 controls (Table 16). The abnormal liver function tests did, however, vary significantly with the history of alcohol consumption obtained from the worker (Table 17, 18). Since no significant differences in drinking habits among the groups with different work exposures were found, the variations among work group categories (duration of exposure) are most likely due to the distribution of alcohol intake among them.

Dermatitis and skin irritation

Irritation of the skin and/or burns from sulfur oxides and sulfuric acid had occurred in 10% of those examined. Four workers who worked with amine compounds used in one part of the pigment manufacturing process also reported skin irritation. Other problems occurred sporadically among the workforce and are listed in Table 19.

5. Significance and discussion

The physiological history of TiO_2 is one of inertness. In the process of the preparation of TiO_2, workers are exposed to high concentrations of the dust. Since its particles vary in size between 0.1-1.0μ, they easily reach the alveoli.

A review by Sroka described an intense fibrous pulmonary reaction and x-ray findings that could not be differentiated from the early stages of silicosis. However, the prevalence and incidence of such observations was not recorded. Elo et. al. studied two biopsy cases and one autopsy case in three workers from a TiO_2 processing plant in Finland. The two biopsy cases had experienced nine years of exposure to TiO_2 and complained of a productive cough and dypsnea, aggravated by dust in the plant. Both were smokers. The autopsy was performed on a 38 year old man with a history of old tuberculosis, who had drowned. He had worked in the titanium industry for about 9 years. Studies showed that significant amounts of TiO_2 may accumulate in the alveolar epithelium and in neighboring connective tissue. Electron microscopic examination demonstrated that TiO_2 particles were phagocytized by the alveolar macrophages and accumulated within their lysosomes, possibly resulting in lysosomal breakage and release of lysosomal hydrolases. In addition, a greenish color of the pulmonary surfaces was seen in places where large accumulations of pigment were found, as well as slight increases in connective tissue in the pleura, sub-pleura, and alveolar septa. There were some bronchiolar hyperplastic changes. However, no evidence of a specific lesion being produced by TiO_2 was reported. Other metals may have been present, but these were not analysed.

Lahmann and Zerget found no evidence from the histologic study of organs from animals fed TiO_2 for periods of up to 16 months, of any tissue change or of appreciable quantities of titanium in the tissues, and they concluded that titanium is not absorbed from the gastro-intestinal tract. Ereaux reported that the oral administration of large quantities of titanium salts mixed with the diet did not have any toxic effect, but actually caused an improvement in health. Vernetti-Blina studied the chronic toxicity of TiO_2 administered by mouth, by subcutaneous injection and by inhalation in varying dosages over periods of 1-2 months. The results of the administration by mouth and subcutaneous injection were essentially negative, but while inhalation of the dust for 8 hours a day for 30 days caused no appreciable disturbance of health, there were some pulmonary lesions. The lungs showed an increase in the connective tissue of the stroma and medium bronchi; the peribronchial glands showed some hyperplasia. Christie et. al. studied rats which inhaled TiO_2 dust for approximately 8 hours per day, five days per week for as long as 13 months. They showed retention of the dust in the lungs, and that the particles were phagocytosed and carried to the sump area in the subpleural areas and in the regions of the alveolar ducts. Here the pigment remained intracellular within the macrophages with some slight lymphoid proliferation. There were small focal areas of emphysema in relationship to the larger deposits of dust. There was no evidence of any specific lesion.

Exposure to sulfur dioxide is not limited to operations where it is used. Its prominent biologic effect in man, severe irritation, is produced by the conversion of sulfur dioxide to sulfurous acid on contact with moist mucus membranes.

Chronic exposure to sulfur dioxide is widespread in industry; however, the toxicity to the respiratory system is not well known. In most industrial situations, as at the titanium pigment plant, the exposure is to a mixture of sulfur dioxide, sulfuric acid aerosol, particulates, sulfates, and other oxides of sulfur.

The workers examined in this study were exposed to both particulate TiO_2 and to sulfur oxides and sulfates. There was significant prevalence of obstructive airway disease. It was not possible to determine the relative contribution of the titanium and sulfur oxides, since most workers had exposure to both. In the population as a whole and especially those who had never smoked, a significant amount of airflow impairment was found (49% and 41% respectively). Where exposure levels could be separated, the workers who had higher exposures to either titanium dust of sulfuric acid or sulfur oxide fumes, in the nonsmoking category did show significant prevalence of airflow impairment. The level of exposure as judged by the history of any episodes of acute symptoms, however, did not have an effect upon the incidence of airflow impairment among nonsmokers. The influence of this exposure level (a history of high exposure reflected in pulmonary symptoms) did not affect the incidence of chronic bronchitis between the smoking categories. The radiologic data indicate that the titanium dioxide dust is probably not altogether biologically inert. However, its fibrogenous properties appear to be less than other pneumoconiotic dusts and the radiologic disease less severe. The relationship of smoking and the chest x-ray abnormalities suggest a co-factor role for TiO_2 and cigarette smoke, since this combination tended to produce more severely scarred lungs on the x-rays. A small prevalence of restricted pulmonary disease was found (6%) and a higher prevalence of "mixed" restriction and obstruction (9%) was found.

Table D-1

Job Classification	Exposure	TiO_2	H_2SO_4
Dry Milling		3	1
Digestion		3	3
Clarification		2	2
Crystalization		2	2
Washing		2	2
Precipitation		2	3
Dry Calcination		3	1
Washing and Drying		1	3
Wet Milling-Micronizer		2	2
Grinding and Packing		3	1
Acid Manufacture		1	3
Warehouse		2	1
General Plant Exposure		2	2

Table D-2

Prevalence of airflow impairment** among titanium pigment workers according to duration of exposure and smoking habits

Duration of exposure (years)	Smokers	Ex-smokers*	Non-smokers	Total
< 20 mean age: 44	9/19 (47%)	3/9 (33%)	3/5 (60%)	15/33 (45%)
20-29 mean age: 51	31/53 (58%)	21/50 (42%)	10/26 (38%)	62/129 (48%)
≥ 30 mean age: 58	9/16 (56%)	6/13 (46%)	2/6 (35%)	17/35 (49%)
	49/88 (56%)	24/72 (33%)	15/37 (41%)	94/197 (48%)

* Ceased smoking more than 2 years earlier

** Airflow impairment: MMF $\leq .79$ predicted, FEV/FVC ≤ 74 %, $FEF_{50}/FVC \leq .69$

Smokers vs. Non-smokers: $p < .1$

Smokers vs. Ex-smokers: $p < .01$

Differences in prevalences of airflow impairment with duration of work: $p > .1$

Table D-3

Prevalence of airway obstruction in titanium pigment makers by age

Age	No.	Prevalence of airflow impairment*
< 50	76	27 (36%)
		$p < .005$
50-59	100	57 (57%)
		$p < .75$
> 60	21	13 (62%)

* Abnormal pulmonary flow parameters:

$FEV_1/FVC \leq .79$ predicted

$MMF \leq .79$ predicted

$FEF_{50}/FVC \leq .69$

Table D - 4

Prevalence of airway obstruction in titanium pigment makers in relation to smoking habits and age

	No.	Age < 50 years	Age ≥ 50 years
Current smokers	88 (45%)	39/75 (52%)	49/111 (44%)
Ex-smokers*	72 (36%)	23/77 (30%)	49/131 (37%)
Non-smokers	37 (19%)	14/76 (18%)	23/121 (19%)

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*Ex-smoker: smoking discontinued ≥ two years earlier

Table D-5

Prevalence of impaired pulmonary function in relation to TiO_2 dust exposure and smoking

Exposure category	I	II	III
Smokers	19/37 (51%)	25/40* (65%)	7/11 (64%)
Ex-smokers	8/20 (40%)	20/45 (44%)	2/5 (40%)
Non-smokers	4/9 (44%)	11/27 (41%)*	0/3 (0%)*
	31/66 (47%)	57/112 (51%)	9/19 (47%)

I $\leq$ 40 dose years.

II 41-69 dose years.

III $\geq$ 70 dose years.

* prevalence of airflow impairment for non-smokers grade I vs. grade II $.01 < p. < .05$; grade II vs. grade III $p = .01$

Table D-6

Prevalence of impaired pulmonary function in relation to sulfur oxides, exposure and smoking

Exposure category	I	II	III
Smokers	23/40 (59%)	20/32 (58%)	9/15 (60%)
Ex-smokers	9/19 (41%)	18/47 (65%)	3/6 (50%)
Non-smokers	7/17 (41%)	8/18 (44%)*	0/3 (0%)*
	39/76 (51%)	46/97 (47%)	12/24 (50%)

*Prevalence of airflow obstruction among non-smokers significantly different between exposure level I & II; (p = .01), & II & III (p = .01)

Table D-7

Prevalence of acute respiratory irritation due to exposures encountered in titanium pigment manufacture among 207 workers

Eye irritation	119	(57%)
Nose irritation	71	(34%)
Throat irritation	58	(28%)
Chest symptoms (total)	157	(76%)
Cough	146	(70%)
Chest tightness	62	(30%)
Shortness of breath	85	(41%)
Wheeze	25	(12%)
Associated nausea and/or vomiting	30	(16%)
Associated dizziness and/or headache	9	(4%)

Table D-8

Prevalence of airflow abnormalities* among 197** titanium pigment manufacturers in relation to the presence of acute symptoms from irritating gases, or fumes and smoking habits

	No history of acute lower-respiratory symptoms			History of acute lower respiratory symptoms		
	Nl. Air-flow	Impaired airflow	Sub-total	Nl. Air-flow	Impaired airflow	Sub-total
Smokers 88	11	9	20	32	36	68
Ex-smokers 72	8	11	19	37	16	53
Non-smokers 37	6	3	9	18	10	28

Prevalence of impaired airflow: Smokers with acute symptoms vs. ex-smokers with acute symptoms: $X = 6.29$, $0.02 > p > 0.01$

Prevalence of impaired airway flow in ex-smokers without symptoms vs. ex-smokers with symptoms: $X = 4.58$ $.05 > p > .02$

*Airflow abnormalities: $FEV_1/FVC = 74\%$ or $FEF\ 50\%/VC \leq 0.69$ $MMEFR \leq 70\%$ predicted; (if FVC is $> 80\%$ predicted)

**10 of the workers did not receive pulmonary function testing

Table D-9

Prevalence of "restriction" of lung volume among 197 titanium pigment manufacturers

		FVC $< 80\%$		FVC $< 60\%$		
Total years	N	Pure*	Mixed**	Pure	Mixed	Total
< 20	33	0/33 (0%)	1/33 (3%)	0/33 (0%)	0/33 (0%)	1/33 (3%)
20-29	129	5/129 (4%)	10/129 (8%)	2/129 (2%)	0/129 (0%)	17/129 (13%)
≥ 30	35	4/35 (11%)	5/35 (14%)	0/35 (0%)	2/35 (6%)	11/35 (31%)
Total	197	9/197 (4%)	16/197 (8%)	2/197 (1%)	2/197 (1%)	29/197 (15%)

*pure: FVC less than predicted; no flow abnormalities.

**mixed: FVC less than predicted; flow abnormalities standardized for vital capacity present ($FEV_1/FVC < 80\%$, or $< 60\%$ $FEF_{50}/VC < .69$

Table D-10

Prevalence of cough and sputum (chronic bronchitis) among 206* titanium pigment manufacturers in relation to history of acute irritant symptoms and smoking

		No acute symptoms			History of acute symptoms			Total	
		No bronchitis	Bronchitis		No bronchitis	Bronchitis		No bronchitis	Bronchitis
Smokers	(91)	16	6 (27%)	22	40	29 (42%)	67	56	35 (38%)
Ex-smokers	(77)	18	1 (5%)	19	52	6 (10%)	58	70	7 (9%)
Non-smokers	(38)	9	0 (0%)	9	26	3 (10%)	29	35	3 (8%)
								161/206	45/206 (22%)

Prevalence of bronchitis among:
Smokers with symptoms vs. ex-smokers with symptoms $p < 0.01$
Smokers with symptoms vs. non-smokers with symptoms $p < .01$

No other comparisons significant

Table D-11

ILO/UC Pneumoconiosis Classification of TiO_2 Exposed Workers X-rays

Total	0/0-0/1	1/0	1/1	1/2	2/1
201	173 (86%)	19 (9.5%)	8 (4%)	0	1 (0.5%)

table D-12

Abnormal Chest X-rays and Length of Exposure

length of exposure	total number examined	number with abnormal x-rays
0 - 9 years	7	1 (14.3%)
10 - 19 years	17	2 (11.8%)
20 - 29 years	134	17 (12.7%)
30+ years	43	8 (18.6%)

table D-13

Abnormal Chest X-rays and Smoking History

smoking history	total	number with abnormal x-rays
Non-smokers	37	2 (5.4%)
Former smokers	75	10 (13.3%)
Current smokers	89	16 (18%)

Table D-14

Relation of x-ray changes to "restriction of lung volumes in 201 titanium pigment manufacturers

		Normal lung volume (FVC)	Diminished lung volume (FVC)	
Normal		43	10 (16%)	57
dirty		4		
1/0	8/11	5	2 (29%)	11
1/1		2	1 (33%)	
1/2		0	0	
2/1		1	0	

Diminished lung volume:
FVC < 80% predicted

Table D-15

Prevalence of alterations in liver function tests in 207 titanium workers in relation to length of exposure

Length of exposure (Years)	No.	Total bilirubin > 1.1	SGOT > 40	SGPT > 53	LDH >52	Alkaline Phosphatase > 85
< 20	35	0	0	0	1 3%	4 11%
20-29	135	8 6%	4 2%	3 2%	4 2%	27 20%
≥ 30	37	0	1 3%	1 2%	0	4 11%
Total	207	8 4% (3*)	5 2% (3*)	4 2% (2*)	5 2% (1*)	35 17% (20*)

No significant variation with length of exposure process.

No significant difference from 400 controls.

*Indicates number of men in whom alcohol intake may be a significant factor

Table D-16

Liver function abnormalities in 400 ambulatory patients with no history of liver disease or vinyl chloride exposure*

Bilirubin > 1.1	SGOT > 40	Alkaline Phosphatase > 86
23/400 (5.7%)	11/400 (3.7%)	56/400 (13.7%)

* Observed primarily in annual health maintenance surveys, Chemical studies were made in the same laboratory.

Table D-17

Abnormal liver functions in 206* titanium workers in relation to drinking habits

	Total	Abnormal liver functions
Alcohol intake		
** < 2+	118	19 (16%)
≥ 2+	88	27 (31%)
Total	206	46

X^2 6.18; $0.02<p<0.01$

*1 person: no history of alcohol intake available.

**Alcohol intake ≥ 2+: ≥ 24 cans beer/week and/or ≥ 1 quart liquor/week

Table D-18

Distribution of drinking habits among 206 titanium pigment workers by the duration of their exposure to titanium

Alcohol intake		< 2	≥ 2
Duration of work in titanium manufacture	Total		
< 20	35	20 (57%)	15 (43%)
20-29	135	75 (55%)	60 (45%)
≥ 30	36	23 (64%)	13 (36%)
Total	206	118 (57%)	88 (43%)

No significant difference in drinking habits among the groups with different length of work in the titanium pigment manufacturing process.

Table D-19

Skin Problems in 207 Titanium Pigment Workers

Irritation and burns from sulfuric acid or sulfur oxide fumes	21	(10%)
Irritation from amines*	4	(2%)
Drying from solvents	5	(2%)
Antimony rash	1	(0.5%)
Fibrous glass rash	2	(1%)
Cement dermatitis	2	(1%)
Burn from steam	1	(0.5%)
Other dermatitis	14	(7%)

*Trimethanolamine and methyl isopropanolamine are used in the titanium process.

III. Perspectives for Mortality Studies.

Two mortality studies are possible within the International Brotherhood of Painters and Allied Trades. One could include every member of the Brotherhood at any year of choice from 1967 onward. These records are currently computerized and available at the offices of the Brotherhood in Washington. For each member, active or retired, who dies, a death certificate is also on file and available for extraction and follow-up. This group would include more than 200,000 individuals, most identifiable by craft on the union records. The current breakdown of the active members of the brotherhood is provided in Table 1. This delineation by trade would allow subgroups to be separately identified in a mortality study conducted for the purpose of identifying hazards within specified painting trades activities.

A second group suitable for study would include the approximately 10,000 members of the Brotherhood in District Council 9, New York City. Here, also, records are computerized and available on any member who joined locals within the Council after 1960. A list of the membership in 1960 has been obtained and reviewed with a view to ascertaining the difficulty of tracing all individuals listed thereon. As similar records are maintained by the International, this review would also apply to the prospects of tracing individuals in the larger cohort available through the International Office. Appendix IV is a single sheet from these records to illustrate the information available.

An evaluation was made of the feasibility of tracing a painter cohort. Table 2 lists the status of 793 individuals of Local 51 (N. Y.) who were active members of the local on December 31, 1960. Of the 615 individuals who had been members of the union for five or more years, information was unavailable on vital status on only 32. Thus, from available records, the status of 95% of a defined cohort can be determined and hand tracing should increase this figure to at least 98%.

Table 1

Active Membership 12-1-75
IBPAT

Painters	135,430
Glaziers	22,228
Sign Painters	9,709
Carpet and Linoleum	7,328
Specialty	7,612
Industrial	3,877
Paint Makers	7,157
Dry Wall	2,945
Civil Service	3,215
Scenic Artists	1,196
Total	200,697

Table 2

Status of Members of Local 51 (NY) IBPAT

Active members, 12-31-60	793
Members with less than 5 years seniority, 12-31-60	178
Active members, 6-16-75	365
Deceased, 1961-1975	218
Suspended or out, 1961-1975 (more than 5 years seniority)	32

IV. Industrial Hygiene Evaluation

A. Materials and Work Practices

Appendix I is a list of various compounds in different paint materials according to the type of paint used (i.e. water-based emulsion, oil-based, epoxy, etc.). The exposure of workers to these various pigments, solvents, fillers, and other materials is delineated in Appendix II, where various work activities are described in different painting trades, and the materials listed, along with exposures to workers. It should be noted that while the spectrum of exposures in a given trade is extremely complex, the overlapping of different painting activites, for example taping, spackle sanding, and painting, leads to an even greater multiplicity of exposures than indicated on the tables. When one considers in addition the effects of activities of other construction crafts (asbestos workers, welders, etc.), the problem is even compounded further. Appendix III provides information on the toxicity and carcinogenicity of some of the compounds to which painters may be exposed, as well as analytical techniques for the sampling and analysis of these materials. The list is limited; for many of these materials virtually no information is available.

B. Industrial Hygiene Survey Criteria

Two of the significant findings in the pilot clinical survey were the demonstration of widespread central nervous system effects and manifestations of exposures to mixed dusts. Thus, exposure to organic solvents and to various particulate aerosols should receive emphasis in any industrial hygiene survey. Concern for pigments also exists, as several of these may have potential carcinogenic effects. Finally, analysis of the work environments of new "exotic" pigments, for example urethanes and epoxies, is wanting. Table 1 lists the materials to be evaluated in any industrial hygiene survey according to painting trade.

Three phases are required in an industrial hygiene survey of painting trades. Phase one would include a further review of the industry. This would be accomplished by contact with industry sources and the International Brotherhood of Painters and Allied Trades. A comprehensive literature review should also be conducted, and sampling and analytical procedures determined for materials not included in Appendix III. Following this definition of the painting trades, sites will be selected at which walk-through surveys will be conducted to determine the scope of painting activities and exposures present. The sites chosen would be representative of the various painting trades (general painting, steel painting, taping, sandblasting, etc.). The full scope of potential hazards should be evaluated in full-scale industrial hygiene surveys of several workplaces of each type of painting activity.

V. Summary and Recommendations

The pilot study results detailed in the foregoing report clearly demonstrate the existence of significant health hazards in the painting trades. Some are potentially serious, and require urgent, detailed study, with appropriate corrective measures defined and instituted.

Nevertheless, it is equally clear that these hazards are specific and identifiable, making it unwise to broadly coat the entire industry with a broad "hazardous" brush. Thus, we found no evidence of an important continuing lead problem among painters. There was neither clinical nor laboratory evidence that such a hazard is likely to be significant. Skin diseases were by no means as common as might have been anticipated; apparently, industry concerned with this possibility had been effective in eliminating or preventing wide use of materials likely to have such effects. It was good to find, too, that individuals very heavily exposed ~~to titanium had comparatively~~ few abnormalities which might be attributed to this metal, at least in clinical studies (long-term mortality evaluation remains to be accomplished). Finally, although our experiences were insufficient to warrant any firm opinion, it is likely that even sandblasting, if undertaken with appropriate precautions, need not necessarily result in widespread, serious hazard. In part, these encouraging findings must remain tentative until they are confirmed by wider experience. Nevertheless, they do point to the need to evaluate the effects of each agent, material, trade and craft separately, to seek specific information and to avoid blanket indictment.

On the other hand, in a number of areas, unexpectedly potentially serious problems were encountered. Central nervous system effects were widespread and troublesome. The fact that they were also poorly understood and inadequately studied increases the seriousness with which they should now be considered. Asbestos exposure was found to be common; the results of this exposure are not now known, but apprehension is surely warranted. Little is known about the carcinogenic or mutagenic potential of the many new chemicals now used in these trades; the painter's environment remains largely unexplored and unevaluated. Similarly, long-term toxicological effects of the materials being used is little known; most of the animal toxicity studies have been short-term, acute investigations.

Recognizing the limited data base available for consideration, the following recommendations are made. The list as a whole must be considered tentative, since with this limitation, other hazards may not yet have been identified and the recommendations of necessity therefore incomplete. With further experience, the relative weight given to the various recommendations might change. Nevertheless, for the present, the results of our studies would suggest that the following problems should be rapidly and effectively addressed:

1. Control of asbestos hazards

   Control of asbestos exposure in the painting trades is urgently required. We have found widespread use of asbestos-containing materials, with the asbestos content of such materials often unknown to either the employer or the worker. In other instances, the asbestos is known to be present but its disease potential has been unappreciated. Examples have included spackle compounds used by general painters, taping and joint compounds used in dry wall construction, addition of asbestos to sealant compounds in factory manufacture, indirect exposure to asbestos materials used by other trades in the construction industry, demolition, maintenance, repair and waste disposal, and the continued use, in some areas, of surfacing materials containing asbestos in their formulations. Both industry and union groups, together with NIOSH, can identify safe working practices, and substitutes where these are feasible. The results of the mistakes of the past will be with us for many years to come; it would be unwise to add to this burden.

2. Work practices for sandblasting

There has been much useful input from industry, labor and from NIOSH scientists to work practices and standards needed for the control of silicosis hazards resulting from sandblasting. It is known that incompletely controlled use of the procedure can result in rapidly progressive, fatal silicosis. Our findings equally suggest, however, that well controlled use sharply decreases the hazard. Effective cooperation among the parties concerned in defining the most appropriate and safest work practices, and the measures needed for surveillance and control, can largely eliminate serious hazards from this trade.

3. Measurement and analysis of occupational environments in the painting trades

This is a matter of great urgency. There is virtually nothing known of many of the working conditions under which literally hundreds of thousands of men in these trades are employed in the United States. A variety of solvents are known to be toxic at specified levels; what these levels are in the painter's workplace have not been studied. Dusts have been little characterized and levels remain unmeasured, in large part. Even the nature of the chemicals present is generally unrecorded. It is unlikely that appropriate control of occupational health hazards in the painting trades will be achieved without much more information concerning the environment in which the men work.

4. Materials analysis

Concomitant with and, in a sense, part of the environmental evaluation recommended in (3) above would be analysis of the materials utilized in the trades. Here, cooperation among industry (manufacturers and compounders), labor, NIOSH and others would be important. Likely, much could be accomplished by review of available recorded data. In some instances, laboratory analytical programs would have to be undertaken.

5. Central nervous system effects

This is a major area, requiring urgent study. It is recommended that this be approached in terms of clinical neurology, behavioral toxicology and, in some instances, necessary laboratory studies. Investigations should include both short-term and long-term effects. As an extension, relation to accidents among workers should be considered, including accidents which might occur hours after exposure.

6. Lung disease in painters

There is good reason to suspect that "mixed dust pneumoconiosis" is common among painters, as might be expected from our limited knowledge concerning their working conditions (silica, mica, talc, other silicates, asbestos, diatomaceous earth, titanium, etc.). It is not known whether other inhaled agents (chemicals, gases, fumes) have additional influence, modifying or accelerating the dust effects. The frequency of abnormal x-ray and pulmonary function findings indicate that this problem requires rapid investigation. As an important first step, analyses should be made of the histological findings and inorganic dust burden of the lungs of painters. The necessary histopathological and mineralogical analytical techniques are now well established and appropriate studies would rapidly give us the necessary information for evaluation of the hazard which may exist, and guide necessary corrective measures.

7. Mortality studies

Recent analysis by Guidotti and Goldsmith of the results of three very large scale studies of cancer incidence in a number of occupations (U.S. National Center for Health Statistics (death certificate data;),death certificate data of the Registrar General of Great Britain and morbidity data of the Social Security Administration) has demonstrated that construction painters were the only group with significantly higher than expected deaths of lung cancer in each of the three studies and had overall cancer rates higher than expected. While these studies had methodological difficulties and constraints (such as utilization of last occupation as recorded on the death certificate, for example) the data, taken in context with our findings, warrant urgent investigation of the mortality experience of workers in the painting trades. The techniques for this are well established (as, a retrospective-prospective cohort approach) and such studies should be mounted as soon as possible. The results would provide information concerning whether or not cancer hazards exist and, if they do, their nature and probable source. It is not known, for example, whether the leukemia incidence among painters is increased or not, in association with benzene contamination of many of the solvents used by these workers. Too, the possibility of multiple factor interactions with unexpected and unanticipated changes in cancer incidence is a matter of considerable interest in these complex trades.

8. Carcinogenic potential of chemical exposures

Some of the chemicals used in formulation of paints and other materials utilized in these trades are known to be carcinogenic, in animal studies. It is difficult to evaluate the hazard associated with them as a result of environmental exposures, because of the absence of data (see 3) above. However, this aspect of the problem may turn out to be of lesser concern. The carcinogenic potential for most of the chemicals used in these trades has simply not been studied, certainly not with currently acceptable laboratory techniques. It would be impossible to tackle all agents with equal dispatch and resources. Rather, concomitant with materials analysis

(4), priorities should be established for such testing. Included in such priorities evaluation could be such factors as number of workers exposed, level of exposure, suspected carcinogenic potential of the chemical configuration, known toxicity, known mutagenicity, etc. Here, the special expertise of carcinogenesis testing laboratories could be recruited.

9. Mutagenic potential of chemical exposures

   It would be well to consider the mutagenic potential of the various compounds, including those considered in (8) above, utilizing newer bacterial mutagenesis test systems, and similar approaches.

   Consideration should also be given to the possibility of appropriate epidemiological studies aimed at obtaining information concerning teratogenic and mutagenic effects of the chemicals being considered.

10. Accidents

    Appropriate detailed studies of the etiological factors involved in the high incidence of accidents in the painting trades should be considered. This would include a detailed investigation of the incidence of both fatal and non-fatal accidents, relating to type of work, as well as evaluation of the possible influence of chemical exposures. Since neurotoxic and anesthetic-like effects are sometimes seen, the matter is of considerable interest. Data are not available, for example, to evaluate the pertinence of suggestions that painters tend to have higher rates of automobile accidents driving home from work, as the result of their toxic on-the-job exposures.

11. Long-term toxicological investigations

    Most toxicological studies concerned with the investigation of chemicals used in the painting trades, have been classical short-term toxic effect studies, especially with regard to the central nervous system and lung.

These should now be extended with long-term toxicological investigations.

12. Educational programs

In some instances, toxic agents are already known and the procedures necessary for their control have been identified. It would be advantageous to look to the development of appropriate educational programs directed to the worker at the job site as well as supervision and management, detailing what is known of these problems and the measures needed. It is likely that union-industry cooperation in such educational programs would be particularly valuable, together with the participation of appropriate government agencies. As new information becomes available concerning other agents and trade practices, the existence of effective educational channels and programs would facilitate the necessary educational and control measures needed for the new problems, as well.

Appendix 1

Materials used in paint products

Acrylic Coatings

Substituted polyacrylic and acrylate resins such as methyl methacrylate
Plasticizing resins
Pigments (if enamel)
Solvents - acetone
  methyl ethyl ketone
  ethyl acetate
  butyl lactate
  ethylene dichloride
  toluol
  xylol

Ref: 1, 6

Alkyd Coatings

Resin
  (Polybasic acid [phthalic anhydride, fumaric acid, maleic anhydride, succinic acid]
  (Polyhydric alcohols [glycerol, pentaerythritol, glycols, sorbitol]
  (Drying oils

Rosin

Solvents, including coal tar hydrocarbons
  turpentine
  mineral spirits
  ketones
  esters
  alcohols

Ref: 1, 6

Amino Resin Coatings

Resin: urea and formaldehyde condensate (or)
  melamine and formaldehyde condensate

Solvents, including coal tar hydrocarbons
  aliphatic hydrocarbons
  ketones
  esters
  alcohols

Ref: 1, 6

Anti Aglae Paint

Antialgae Agents

Copper oxide	7% metallic copper
Copper soap	
Arsenic oxide	7% arsenic
Copper sulfate	pentahydrate
Mercury soap	0.3% mercury

2,2 - Dihydroxy-5,5-dichlorodiphenyl methane
or 2,2 - Methylene Bis (4-Chlorophenol)

Ref: 8, 12

Antifouling Paint

Binder: rosin and synthetic resins such as ester gum
chlorinated rubber
phenolics, including phenol-formaldehyde

Pigment: copper and mercury
Solvent: aromatic hydrocarbons
mineral spirits

Ref: 1, 6

Bituminous Coating

1. Coal tar
   Solvent - aromatic hydrocarbons
   coal tar solvents
   Bitumastic pitches

2. Asphalt
   Solvent - aliphatic solvents such as mineral spirits
   Bitumastic pitches
   Gilsonite
   Drying oils
   Resins
   Pigment

3. Emulsion
   Bitumastic pitches
   Clay
   Water

Ref: 1, 6

Bleach

Oxalic acid
    12-16 oz. crystals/gal H_2O
    Rinse with 3 oz. Borax/gal. H_2O

Sodium hydroxide and hydrogen peroxide (1:2)

Laundry bleach

    1/2 pt/gal H_2O
    Neutralize with H_2O and sand

Permanganate - Bi Sulfite

    1 oz. sodium permanganate/gal H_2O
    Rinse with 3 oz. sodium bisulfite/gal H_2O

Ref: 1

Catalyzed bitumen-epoxy coatings

coal tar pitches
asphalt pitches
epoxy resins
polyamine and polyamid catalysts

Power stirring following addition of catalyst

Ref: 6

Catalyzed epoxy coating

Epichlorohydrin
Bisphenol A
Catalyst - Polyamine
    Amine
    Polyamid
Solvent - ketone/aromatic hydrocarbon mixture

Ref: 6

Catalyzed Phenolic Coatings

Substituted phenol
Formaldehyde
Catalyst (Polyamines
Resins (such as epoxy)
Fatty acids
Thinner - ketone/aromatic blends

Ref: 6

Chlorinated Rubber Paint

Chlorinated rubber resin
Resin modifiers such as alkyd resin
Plasticizers
Stabilizers
Antimony oxide, if flame retardant
Pigments, including phthalocyanine blue and green

Solvents, including coal tar hydrocarbons
aliphatic hydrocarbons
ketones
esters
alcohols
aromatic hydrocarbons

Ref: 1, 6

Chlorosulfonated polyethylene (Hypalon) coatings

Polyethylene
Chlorine
Sulfur Dioxide
Resins, such as phenolic
Lead-containing alkaline catalyst
Thinner - ketones and/or aromatics

Ref: 1, 6

Emulsion paint - GENERAL FORMULA AND CONSTITUENTS

	%
opaque pigment	20
extinder pigment	15
pigment dispersant (phosphates, silicates)	0.3
protective colloid	1.2
latex	40
plasticizer*	--
modifier*	--
preservative	0.5
antirust agent*	0.1
pH buffer	0.1
fungicide*	0.5
coalescing agent (volatile plastizer)	2.0
defoamer	0.5
thickener*	0.5
freeze-thaw stabilizer*	2.5
H_2O	16.8

*optional in some formulations

Paint pigments

- White pigments
  - Rutile titanium dioxide
  - Anatase titanium dioxide
  - 50% Rutile titanium dioxide - calcium sulfate
  - ainc sulfide
  - 30% Rutile titanium Dioxide - calsium sulfate
  - lithopone
  - antimony dioxide
  - zinc oxide
  - 35% leaded zinc oxide
  - basic carbonate white lead
  - basic sulfate white lead
  - basic silicate white lead

- White extenders
  - silica
  - barytes
  - whiting
  - wollastonite
  - mica
  - clay
  - diatomaceous silica
  - lucite

- Green
  - chromium oxide
  - pigment green B
  - phthalocyanines

- Blue
  - phthalocyanines

Black
- Mineral black
- iron oxide
- graphite

Yellow ochre
- umber
- sienna
- lead chromate
- zinc chromate
- iron oxide
- benzidine yellow
- hansa yellow
- cadmium yellow

Red
- Red lead
- cadmium red
- toluidine
- iron oxide
- chlorinated paras
- naphthols
- para red

Thickener - Emulsions
- Methyl cellulose
- Hydroxyethyl cellulose
- SB → Alkali soluble proteins
- Acrylic → Acrylate salts
- Casein
- Cellulosics
- Carboxymethyl cellulose - Nonspecific thickener
  Usually 1-2% by weight of solids

Driers
- 4% Ca
- 6% Co
- 6% Mn
- 6% Zr
- 24% Pb

Usually furnished already dispersed in water

Coalescing agents
- Carbitol
- Carbitol acetate
- Hexylene glycol
- Butyl cellusolve acetate
- Butyl carbitol acetate

Fungicides
- Phenyl mercuric salts
- Copper & guinolate
- Tetrachlorophenol
- Zinc oxide
- Tri-N-butyl tin

Orange
- Benzidine orange
- Dinitraniline

Freeze thaw inhibitors

- non-ionic emulsifiers
- N-coco beta amino butyric acid
- polyalkylene plyamines
- polysubstituted phenates
- modified glyceryl monoricinoleate
- urea
- thieurea
- barium compounds

Buffer

- ammonia

Emulsion Masonry Paint

	gal.
carboxymethyl cellulose (4400) 3% solution	20.0
polypropylene glycol (1200)	.3
wetting agent	.1
lecithin-water dispersible	.3
ethylene glycol	2.0
titanium dioxide - rutile	7.1
calcium silicate	2.9
fungicide	0.5
water	25.0
carbitol	1.8
polyvinyl acetate copolymer	38.0
	100.0

Styrene-Butadiene Emulsion

White Wall Finish

	Gal.
Titanium Dioxide - Rutile	6.4
Clay	5.6
Talc	3.2
Casein solution (10% casein)	15.6
Lecithin (H_2O dispersible)	.6
Morpholine	.1
Defoamer	.1
H_2O	14.4
(Styrene-Buthdiene Latex (48% solids)	41.6
(Ethylene Glycol	2.0
(H_2O	10.3

May contain: A long oil Soya Alkyd Plus
Driers (3016/100 gal.)

Polyvinyl acetate emulsions.

Accoustical tile finish.

	gal.
Titanium dioxide (anatnse)	1.55
Calcium carbonate	29.00
Talc	3.80
PVA latex (copolymer 55% NVM)	22.00
Hydroxyethyl cellulose 300 cp (10% solution)	4.03
Dispersant - 25% solution	1.65
Ethylene glycol	.75
Defoamer	.25
Water	35.47
	100.00

Cedar shake coating	gal.
Titanium dioxide-rutile semi-chalking	5.00
Calcium carbonate	3.30
Talc	3.15
Lecithin	.38
Dispersant	.25
Carbitol acetate	.25
PVA latex (copolymer 55% solids)	39.16
Water	41.50
Hydroxyethyl cellulose 2% solution 250 cp	6.(X)
	100.00

Oil/Water Emulsion

	Parts
Linseed oil	70.0
Water	30.0
Emulsifier (lipophilic--dissolved in linseed oil	.14
Emulsifier (hydrophilic--dissolved in water	.06
Thickener	.06
Ammonia hydroxide added to adjust pH	100.26

Oil Emulsion Exterior Paint

	lb.	gal.
Water	205.0	24.6
Wetting agent	4.0	0.5
Pottassium polyphosphate	5.0	0.4
Ethylene glycol	10.0	1.1
Titanium dioxide-rutile	175.0	5.0
anatase	50.0	1.6
Talc	50.0	2.1
Zinc oxide	25.0	2.8
Fungicide	2.0	0.2
Defoamer	2.0	0.2
Wetting agent	16.0	2.1
Carboxy ethyl cellulose 15000cp 2.5% solution	130.0	16.7
Emulsified linseed oil (60% solids)	340.0	41.6
Lead drier 23%	7.0	0.8
cobalt 6%	2.0	0.2
manganese 6%	2.0	0.2
	1115.0	100.0

Casein

	lb.
Whiting	50
Clay	20
Dextrin	2
Casein	12
Lime	15
Trisodium Phosphate	1
Bichloride of mercury	1 oz.

10 lb. blended ingredient/1 gallon H_2O

Acrylic Emulsion

A typical acrylic exterior emulsion paint formulation is as follows:

Acrylic Exterior Emulsion Paint	gal.
Wetting agent	2.0
Defoamer	.2
Water	6.0
Titanium dioxide-rutile	6.0
anatose	1.2
Mica-water ground	1.2
Calcium carbonate	3.8
Hydroxyethyl cellulose (4000 cp) 2% solution	6.0
Ethylene glycol	2.6
Acrylic latex (46% NVM)	68.9
Preservative	1.0
Defoamer	.2
Water	1.0
Ammonia Hydroxide (28%)	.1
	100.0

Typical Polyvinyl Acetate Emulsion Primer:

	gal.
Diethylene glycol monoethyl ether	3.0
4000 cp methylcellulose	.6
Water	4.9
Preservative sodium salt o-phenylphenol	.1
Dispersing agent	.1
Wetting agent	.3
Polypropylene glycol (1200)	.3
Dibutyl phthalate	2.1
Clay	1.7
Titanium dioxide-rutile	2.9
Water	28.0
Polyvinyl acetate homopolymer	6.0
	100.0

Gray Baking Primer
(water soluble)

	gal.
Titanium dioxide-rutile semi-chalking	3.5
Barytes	3.3
Strontium chromate	1.2
Potassium tripolyphosphate	.2
"Nonionic dispersant"	.2
Tungoil-fumaric acid adduct vehicle	58.8
Carbon black dispersion	1.5
Emulsifier	.1
Manganese drier (5%)	.4
Defoamer	.1
Cellosolve	2.6
Water	28.1
	100

Baking Red Primer
(emulsion type)

	gal.
Red iron oxide	1.7
China clay	3.3
Barytes	3.9
Tamol 731 (25%)	.4
Ben-a-gel	.1
"Nonionic dispersant" (20% ethyl alcohol)	.2
Styrene-butadiene latex (40% solids - baking type)	74.4
Nonionic wetting agent (50%)	.7
Manganese drier 5%	.7
Methylcellulose 15cps. (5%)	2.0
Water	12.6
	100

Ref: 2

Epoxy Resin Paint

Resin - epichlorohydrin and bisphenol A
Drying oil of fatty acid (linseed oil)
Solvents, including coal tar hydrocarbons
    aliphatic hydrocarbons
    ketones
    esters
    alcohols
    aromatic hydrocarbons

Ref: 1, 6

Flock

Flock-rayon
    cotton
    nylon

Adhesive - water soluble glue
    natural or synthetic enamel
    lacquer
    rubber-based

Ref: 1

Fluorescent Paint

Fluorescent dye
Polyacrylic laquers
Pigments: 1 or 2 of following - zinc sulfide
    cadmium sulfide
    strontium sulfide
    calcium sulfide

Solvents: ketones
    lacquer thinner

Ref: 6

Metallic Paint

Pigment: Aluminum flakes
Copper flakes
Zinc flakes
Tin flakes

Vehicle: Oleoresinous & synthetic varnishes
Long oil varnish (aluminum paint)
Short oil varnish (aluminum enamel)
Nitrocellulose lacquer
Bronzing lacquer
Asphalt & tar-based varnishes

Ref: 1, 6

Nitrocellulose coatings

Nitrocellulose
Resins, such as Alkyds and Polyacrylics
Acrylics
Phenolics
U/F
Vinyl
Amino
Polystyrene

Plasticizers - Castor oil
Linseed oil
Rapeseed oil
Tricresyl Phosphate
Dibutyl Phthalate

Solvent & Thinners: Acetone, benzine, MEK, ethyl acetate amyl acetate, zylene, butyl alcohol, methyl amyl ketone, ethyl lactate, butyl propionate

Pigments

Ref: 1

Oleoresinous Coatings - Representative Formulations

Resins: Congo copals
Kauri copals
Boca resins
Damar
Sandarac
Rosin

Vehicles: Tung
Linseed (metallic driers incorporated during curing)
Soybean
Perilla
Oiticicia
Fish oils

Solvents: Turpentine
Mineral spirits Aliphatic hydrocarbons

Driers: Organic salts of Co, Pb,Mg,Ca,Zn, (2-5% of formulation)
only Co,Mg are active driers

Flatteners: Zinc stearate
Aluminum stearate
Silica Aerogel

Pigments: Opaque white fillers & extenders - Titanium dioxide
Zinc oxide
Lithopone
Zinc sulfide

Inert transparent - Calsium carbonate (whiting)
Barium sulfate (Paryte)
Al&Mg silicate (asbestine)
Silica
Al stearate

Opaque color pigments - Umbers
Siennas
Chrome yellow
Ultramarine blue
Prussian blue

Oil Base Exterior Paint

White pigments
- white lead
- lead sulfate
- zinc oxide
- lithopone
- titanium dioxide - anatase
  - rutile
  - brookite

Extenders Titanium calcium
- calcium carbonate
- silica
- calcium sulfate
- barium sulfate
- magnesium silicate
- aluminum silicate
- mica

Colored pigments
- sienna
- umber
- ochre
- oxides - zinc oxide
  - basic leaded zinc oxide
- ultramarine blue
- iron blues - prussian, chinese, malori
- chrome oxide
- lamp black
- vine black
- carbon black
- drop black

ref: 1, 6, 8, 12

Paint and varnish remover - solvent type

1/2 gallon benzol
1 quart acetone
1 quart 188 or 190 proof denatured grain alcohol
1/2 lb. paraffin wax

Methylene chloride may be solvent if nonflammable mixture is desired.

Other stripping agents

Phenols
Cresols
Organic acid catalyst

Ref: 1, 12

Plastisol Coatings

Polyvinyl Chloride
Heat & Light Stabilizers
Plasticizers: nondrying
- chemical - phthalic anhydride
  - sebacic anhydride
  - phosphoric acid esters
- resinous - alkyds esterified with ethylene glycol or glycerol alcohol

Ref: 1

Polyester coatings

Polyester resin
Styrene
Methyl ethyl ketone peroxide catalyst
Accelerator (cobalt naphthenate)
Fibrous glass
Solvent - ketones

Ref: 6

Polyurethane Coatings

1. Two-component polyol-cured formulation
   partially polymerized polyisocyanate-polyol fraction
   polyol fraction containing pigment

2. One-package moisture-cured formulation
   diisocyanate (prepolymerized with polyol)
   moisture from atmosphere

3. Oil-modified formulations
   diisocyanate completely reacted with polyalcohol esters of drying oils

Solvents - ethyl acetate
butyl acetate
ketones (MEK, MIBK)
alcohols
aliphatic hydrocarbons

Ref: 1, 6

Silicone and Silicone Alkyd Paints

Silicone resins, may contain metallic pigment

Solvents, including coal tar hydrocarbons
aliphatic hydrocarbons
ketones
esters
alcohols
aromatic hydrocarbons

Ref: 1, 6

Shellac

Lac
Alcohol solvent - special denatured alcohol #1
100 gallons ethyl alcohol
5 gallons methyl alcohol
proprietary denatured alcohol
100 gallons 190 proof
2 gallons denatured wood alcohol
1 gallon ethyl acetate
1 gallon petroleum hydrocarbon (aviation gas)

Ref: 1

Special Effects Coatings

Wrinkle finish

1. synthetic enamel (usually alkyd)
2. oleoresinous varnish

Hammered Effect

aluminum powder
toluol, xylol
vehicle: varnish
  long-oil alkyds
  short-oil alkyds
  resin-modified short-oil alkyds
  styrenated alkyds
  nitrocellulose utilizing silicone additives

Multi-Color Emulsion

water
plastic sacs containing lacquer enamel

Ref: 1

Stains

Water stains

Water soluble dyes
Alcohol or other solvent
Water

Oil stains

Solvent: Benzol
  Naphtha
  Tüluol
  Turpentine
oil solution, linseed or varnish
oil soluble dyes
pigments, ground in oil

Non-grain raising stains (NGR)

Solvent: Alcohol
  Glycol
  Tüluol
  Acetone or other ketones

Dyes

Spirit stains

Alcohol
Aniline Dyes

Ref: 1

Vinyl and Vinyl Copolymers

Vinyl copolymer resins (such as polyvinyl chloride-polyvinyl acetate) with or without short-oil alkyds (or) modified epoxy resins
Short-oil alkyds modified with semi-drying or non-drying oils
Acrylic resins
Pigments

Solvents include: ketones
esters
chlorinated hydrocarbons

Ref: 6

Wood fillers

Paste fillers

12 parts (by measure)	Boiled linseed oil
6	Drier
1	Turpentine

Silex (silica) for desired consistency
linseed oil may be replaced by alkyd drying vehicle thinned with VM & P Naphtha

Putty sticks

Wood dough

White lead putty

4 lb. white lead paste
1 lb. whiting
1 t. Drier

Caulking

Water putty

Ref: 1

Zinc Primers and Coaters

1. Three-package system (post-cure)
   water solution of sodium, potassium or lithium glassy silicates
   finely divided zinc dust (sometimes containing lead dust)
   acidic curing solution
2. Two-package system (self-cure)
   alcohol or cellosolve ester solution of silicate esters,
   such as tetraethyl silicate
   finely divided zinc dust

Ref: 6

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Appendix 2

Paint products and their methods of application

Industrial Painting

ype of Paint	Volatile Organic Constituents	Other Volatiles Constituents	Method of Application	Clean-up and/or Diluents	Use
Nitrocellulose Lacquer/enamels ref:1,6, 16	Ketones Aromatics Esters Alcohols		Spray	Lacquer Solvent (blend of esters, ketones,	metal products furniture
Acrylic Lacquer/enamel ref: 1, 4, 16	Ketones Esters Chlorinated Hcs Lacquer thinner		Brush Spray Dip Flow Tumble Roll	Lacquer Solvent (blend of esters, ketones,	Automobiles Over polished metal Appliances
Fluorescent ref: 6	Ketones Lacquer thinner		Brush Spray Dip	Aromatics	Toys Tools Signs
Silicone & Silicone Alkyd ref: 6	Aromatics		Dip Spray (heat cure)	Toluene Xylene Enamel thinner	Mufflers
Polyester ref: 6	Ketones		Spray (asbestos or fiberglass may be added to air stream Brush/Roll	MEK MIBK	Metal Products
Emulsion Baking Primer Red ref: 2	Ethyl Alcohol	Water	Spray Dip		Automobiles Appliances
Gray ref: 2	Cellosolve	Water	Spray Dip		Automobiles Appliances

Industrial Painting

Type of [illegible]	Volatile Organic Constituents	Other Volatiles Constituents	Method of Application	Clean-up and/or Diluents	
Casein ref: 2, 4		Water	Roll		Board
Acrylates ref: 4			Roll		Board
Styrene-Buthadiene ref: 2, 4	Ethylene Glycol	Water	Roll		Board
Chlorosulfonated Polyethylene Hypalon^R ref: 4	ketone/aromatic mix		Roll	Toluene Xylene (preferably with MEK,MIBK)	Board
Shellac ref: 16					
ALKYD ref: 1	Mineral Spirits Turpentine			Mineral spirits turpentine painters naphtha (aromatics may be added to mineral spirits)	Automobiles furniture baking enamel farm equipmen
Amino U/F, F/M ref: 1, 4					appliances

Industrial Painting

Type of Paint	Volatile Organic Constituents	Other Volatiles Constituents	Method of Application	Clean-up and/or Diluents	[illegible]
Polyvinyl Acetate Emulsions Acoustical Tile ref: 1, 2	Ethylene Glycol	Water	Then baked		Acoustical ti
Cedar Shakes ref: 2		Water	Spray Dip		Cedar shakes
Catalyzed Bitumen-Epoxy Coal tar epoxy ref: 16	System may be thinned with toluene, xylene blend of above with ketones (MIBK,MEK)		Brush Trowel Spray	Toluene Xylene Blend of above with ketones (MEK,MIBK)	
Vinyl and vinyl acrylic primer (such as vinyl buty ref: 4	Aromatic naphthas ketones		Spray hot spray roll	ketones (MIBK,MEK) aromatics esters	Tin plate black iron
Catalyzed epoxy ref: 6	ketone/aromatic mix		Brush Spray Roll	ketone mixture (MIBK,MEK) aromatics	
Oil base ref: 16			Brush Spray Roll	Mineral spirits Naphtha Turpentine	
Thermosetting stoving enamel ref: 4	Butanol		Spray		Appliances

Industrial Painting

Type of Paint	Volatile Organic Constituents	Other Volatiles Constituents	Method of Application	Clean-up and/or Diluents	Uses
Conversion finishes ref: 1			Spray		Completion of Nitrocellulose Oleoresinous
Polystyrene ref: 1					Metal finishes Baking enamels
Epoxy ref: 1, 4, 16	Mineral spirits Aromatics		Flow Brush Spray Dip Roll		Zinc Tin Aluminum Wood Some Plastics
Urethane ref: 1	Esters) Ketones)Moisture cured type	isocyanate			
Shellac ref: 1, 4, 16					Undercoating Wire coating
Plastisols ref: 1			Dip Spray Roll Mold		Dishwashers Metal coating
Anti Algae Paints					

Industrial Painting

Type of Paint	Volatile Organic Constituents	Other Volatiles Constituents	Method of Application	Clean-up and/or Diluents	Uses
Special effects wrinkle finish ref: 1			dip spray		metal plastic composition wppd
Special effect hammered finish ref: 1	xylol toluol				portable radi car heaters cash register vacuum sweepe
Special effects flock ref: 1, 16					jewelry boxes musical instr cases
Polyfluorines ref: 6					coatings for chemical vesse

INDUSTRIAL PAINTING

Type of Paint	Volatile Solvent Constituents	Other Volatile Constituents	Method of Application	Clean-up and/or Diluents	Uses
Anti Fouling ref: 1	Aromatics Mineral spirits depends on resin		Brush Roll Spray	Mineral spirits	Immersed surface

Method of Application	Type of Coating	Representative Constituents
ipping ref: 4	Paint	well pigmented short to medium drying oil aliphatic and aromatic HC solvents
low coating ref: 4	Paint	pigments with minimum tendency to settle medium length oils medium to short oil alkyd resins with or without U/F, M/F resins higher up and slow evaporating solvents than dip
		wetting agents antifoaming agents - complex organic amines amonium salt silicone derivatives water, as diluent.
oller ref: 4	Paint	cellulose or spirit based bitumin alcohol soluble nitrocellulose spirit soluble resins ~~plasticizers, pigment~~ waxes or silicones
pray (booth) ref: 4	Lacquer	nitrocellulose cellulose acetate cellulose ethers
pray ref: 4	Paint (stoving finish)	xylol is principle solvent also. butyl alcohol lower boiling point solvents
t spray ref: 4	Lacquer	high polymiric chain molecules strong solvent. butyl acetate MIBK methyl cellosolve xylol butyl alcohol

Method of Application	Type of Coating	Representative Constituents
Hot spray ref: 4	Paint/varnish	low molecular weight polymers phenolics weak solvents lead driers
airless spray ref: 4	Paint/Lacquer	low boiling solvents small amount high boiling solvent mixture may be highly pigmented.
Spray ref: 4	2-Component system	types of coatings: polyester polyurethane acid catalyzed U/F or M/F resin epoxies PVB self-etch primers

Type of Paint	Volatile organic Constituents	Other Volatiles	Method of Application	Clean-up Diluents	[illegible]
Catalyzed Bitumen-Epoxy Coal tar Epoxies Asphalt Epoxy ref: 6	System may be thinned with toluene xylene blend of above with ketones (MEK, MIBK)		Brush Trowel Spray	Toluene Xylene Blend of above with ketones (MEK MIBK)	Tank coatings Dams, Piers Barge/Ship bottom Buried steel
Bituminous coating (cold-applied) Coal tar ref: 6	Mineral Spirits Aromatics Coal tar solvents		Brush Spray	Aromatics (toluene, xylene) Aromatic-naphthenic coal tar solvent	Protect buri steel and concrete Tanks
Asphalt ref: 6, 16	Aliphatic solvents such as mineral spirits		Brush Spray	Painters Naphtha Mineral Spirits	Roofs Above-ground protection Industrial coating in corrosive env
Phenolic Varnish and Paint ref: 6	Mineral Spirits Aromatics		Brush Spray Roll	Mineral Spirits Enamel thinner with toluene and xylene	Marine coatin Resistant to corrosive f
Epoxy Ester ref: 6, 16	Mineral Spirits Aromatics		Brush Spray Roll	Mineral Spirits Enamel thinner with toluene and xylene	Industrial maintenanc Resistant to corrosive environmen
Alkyd Paint and Varnish ref: 6	Mineral Spirits Turpentine		Brush Spray Roll	Mineral Spirits Painters Naphtha Turpentine (Aromatic may be added to min. spir)	Protect steel from corrosi Widely applie to metal and wood
Zinc Primers and Coaters 3-Package System ref: 6	Aromatics		Spray	Water (clean-up) No thinners	Corrosion prevention Tank interior Stationary an mobile marine equipment

STRUCTURAL PAINTING

Type of Paint	Volatile organic Constituents	Other Volatiles	Method of Application	Clean-up Diluents	Uses
Self Cure			Spray	Isopropyl Alcohol (clean-up) No thinners	As above
Organic ref: 6			Brush Spray Roll	Depends on binder	As above
Chlorosulfonated Polyethylene Hypalon^{R} ref: 1, 16			Brush Spray Roll	Toluene, Xylene (Preferably with addition of MEK, MIBK)	Chemical ar atmospher exposed w
Catalyzed Epoxy ref: 6, 16	Ketone/Aromatic mix		Brush Spray Roll	Ketone mixture (MEK or MIBK) Aromatics	Heavy duty coatings f industrial plants Water tanks
Catalyzed Phenolic ref: 6	Ketone/Aromatic mix		Brush Spray	MIBK, MEK 50:50 Toluene/Xylene with MEK, MIBK	Heavy duty coatings Concrete Tank interi
Vinyl and Vinyl Acrylic (such as vinyl butyral) ref: 6	Ketones		Spray Hot spray Roll	Ketones (MIBK, MEK) Aromatic Hydrocarbon esters	Marine equipment H_2O tank interiors
Fluorescent ref: 6	Ketones Lacquer thinner		Brush Spray	Aromatics	

STRUCTURAL PAINTING

Type of Paint	Volatile organic Constituents	Other Volatiles	Method of Application	Clean-up Diluents	
Chlorinated Rubber primer used if applied to steel ref: 6	Aromatics		Brush	Toluene Xylene Aromatic-type enamel thinners	H_2O tank interior Steel Wood Concrete
Oil Base (including red lead) ref: 1, 6, 16			Brush Spray Roll	Mineral Spirits Naphtha Turpentine	Steel prime
Conversion Finishes ref: 1					Competitor o Nitrocellul Oleoresinou
Polystyrene ref: 1					Ship bottom
Silicone ref: 1	Aromatics		Spray	Toluene Xylene Enamel thinner	Masonry

RESIDENTIAL PAINTING

Type of Paint	Organic Solvents	Other Volatiles	Method of Application	Thinners and/or for clean-up purposes	Specific Uses
Oleoresinous varnish/enamel ref: 6, 16	turpentine petroleum solvents		spray brush roll	mineral spirits painters naphtha	exterior primer house paint
Alkyd ref: 16	mineral spirits turpentine			mineral spirits painters naphtha turpentine	exterior
vinyl ref: 6, 16	ketones esters chlorinated hydrocarbons		brush roll spray	ketones aromatics esters	exterior interior primer and sea masonry
polystyrene					concrete floor
chlorinated rubber ref: 1, 6	aromatics		Brush	toluene xylene aromatic enamel thinners	concrete masonry
silicone ref: 1, 6	aromatics		spray	toluene xylene enamel thinner	masonry
Epoxy ref: 1, 16			Brush spray roll		wood masonry aluminum

RESIDENTIAL PAINTING

Type of Paint	Organic Solvents	Other Volatiles	Method of Application	Thinners and/or for clean-up purposes	Specific U
Polyurethane ref: 6		isocyanote		esters lacquer thinner MEK, MIBK	Masonry floo
Shellac ref: 16	Alcohols				Wood
Acrylic emulsion ref: 6, 16				water	exterior woo
Epoxy ref: 16	Mineral spirits Aromatics				
Casein ref: 1, 2		Water	Brush		Masonry
Styrene-Buthadiene ref: 1					interior wal masonry
Oil/water emulsions ref: 2	Glycols	water	roll brush spray	MEK	Exterior Interior

RESIDENTIAL PAINTING

Type of Paint	Organic Solvents	Other Volatiles	Method of Application	Thinners and/or for clean-up purposes	Specific U
Nitrocellulose lacquer/enamel ref: 1, 6, 16			spray brush	lacquer thinner	exterior
Metallic pigmented paint ref: 1, 6			brush spray roll		pipes water tanks fences basements gutters
Paint and varnish remover-solvent type ref: 16	benzol acetone denatured grain alcohol methylene chloride			alcohol naphtha mineral spirits	exterior pri (Al
penetrating oil ref: 1	turpentine paint thinner pentachlorophenol				exterior
anti algae paints					

RESIDENTIAL PAINTING

Type of Paint	Organic Solvents	Other Volatiles	Method of Application	Thinners and/or for clean-up purposes	Specific U
SPECIAL EFFECTS Texture wall paint emulsion or oil-based ref: 1, 2			brush roll		interior decoratio
SPECIAL EFFECTS Multicolor emulsion ref: 1, 2		water	spray		
Stains Water ref: 2, 6	alcohol	water			
oil ref: 1	benzol naphtha toluol turpentine		spray dip		furniture
NGR ref: 1	alcohol glycol tuluol acetone or other ketones		brush spray dip		
spirit ref: 1	alcohol				furniture

SIGN PAINTING

Type of Paint	Volatile Organic Constituents	Other Volatile Constituents	Method of Application	Clean-up and/or Thinners	Specific Uses
Oleoresinous enamel ref: 16	Turpentine Petroleum Solvents		Brush Spray		
Acrylic ref: 16	Ketones Esters Chlorinated HCS		Brush Spray	Laquer Solvent	
Vinyl ref: 16	Aromatic Naphthas		Brush Spray	Ketones (MIBK,MEK) Aromatics Esters	
Epoxy ref: 16			Brush Spray		

SIGN PAINTERS

Sign Painters

Sign Painters' Exposures:	Cleansing solutions - reported by tradesmen caustic soda carbon tetrachloride Particulates from brushing surfaces Dry pigment powder

ref: 16

FLOOR FINISHING

Type of Surface Coating or Material	Volatile Solvent Constituents	Other Volatile Constituents	Method of Application	Clean-up and/or thinners	Specific Uses
Stains Oil ref: 1	Benzene Naptha Tolluene Turpentine		Brush Rub		
Spirit ref: 1	Alcohol		Brush Rub		
Paste Wood Filler ref: 1	Turpentine VM&P Naphtha				
Paint/Varnish Remover - Solvent Type ref: 1	Benzol Acetone Denatured Grain Alcohol Methyene Chloride			Alcohol Naptha Mineral Spirits	
Polish ref: 1	Turpentine				
Bleach ref: 1		Oxalic Acid Sodium Hydrox Laundry Bleach			
Wood Sealers/Penetrating Oils ref: 1			Rub		

FLOOR FINISHING

Type of Surface Coating or Material	Volatile Solvent Constituents	Other Volatile Constituents	Method of Appliance	Clean-up and/or thinners	Specific Uses
Oleoresinous varnish ref: 1	Turpentine Petroleum solvents		Brush	Mineral Spirits Painters Naphtha	

FLOOR FINISHERS AND TILE LAYERS

Floor Finishers

Floor Finishers Exposures:

Various paints/lacquers
Pigmented and non-pigmented floor finishing particulates
Wood dust
Silica
Organic solvents
Bleaches

ref: 1, 16

Tile Layers

Tile Layers Exposures:

Bitumen/latex emulsion
Rubber latex-acrylonitrile blend
Polychloroprene (rubber)
Polychloroprene with synthetic resins in hydrocarbon solvent
Modified polyurethane
Polyvinyl acetate and filler
Polyvinyl acetate
Synthetic resin/rubber latex with inorganic fillers (linoleum)
Particulates, including asbestos
Plastics, including polyvinyl chloride

ref: 11, 13, 16

DRYWALL TAPERS, PAPER HANGERS, AND SCENERY DESIGNERS

Drywall Tapers

Drywall Tapers Exposures: Various particulates, including asbestos

Paper Hangers

Paper Hangers Exposures: Synthetics, including vinyl and modified polyurethane
Flock

ref: 1, 11, 16

Scenery Designers

Scenery Designers Exposures: Various lacquers and enamels
Adhesives
Fibrous glass
Various dusts, including wood, paint

ref: 16

Sand Blasters

Sand Blasters' Exposures:	Various particulates, including lead-pigmented paint, silica, chromium-pigmented paint

GLAZIERS AND GLASS MANUFACTURERS

Glaziers

Glaziers' Exposures:

Window-patching adhesive - polyvinyl acetate copolymer with plasticizers
Silica

ref: 9, 11

Glass Manufacturers

Glass Manufacturers' Exposures:

Raw Materials
Sand - almost pure quartz
Flux - soda ash
    salt cake
    limestone or lime
Lead oxide
Pearl ash (potassium carbonate)
Saltpetre
Borax
Boric acid
Arsenic trioxide
Feldspar
Fluorspar
Metallic oxides
Carbonates, nitrates
Cullet

Finishing
Abrasives
Hydrofluoric acid

Other
Heat
UV light

ref: 9, 10

Appendix 3

Toxicity and Sampling Methods

A. Organic Solvents

B. Pigments

inorganic pigments
organic pigments

C. All Other Compounds Investigated

A. Organic Solvents

SAMPLING AND ANALYSIS OF ORGANIC VAPORS

Sampling and Analysis

Method applies for the following compounds:

Compound	TLV (ppm)
acetone	1000
benzene	25 (skin)
carbon tetrachloride	10 (skin)
ethylene dichloride	200
Methyl Ethyl Ketone	200
toluene	
xylene	

Sample Collection

type: breathing zone

apparatus: 7 cm long glass charcoal tube, both ends sealed; 6 mm O.D. 4 mm. I.D. containing 2 sections of 20/40 mesh activated charcoal separated by 2 mm portion of urethane foam; calibrated sampling pump

flow rate: 1 lpm or less

sampling time: see Table I

Sample Analysis

type: GC-FI

apparatus: wet chemistry; GC-FI

procedure: desorb with carbon disulfide, analyze with GC-FI

Range and Sensitivity:

see Table I

Precision

10.5% RSD

Interferences

1. water vapor
2. two or more solvents: one may displace other from charcoal due to differing polarities

Effect of Storage

1. tubes should be capped immediately after sampling
2. more volatile compounds will migrate until equilibrium reached (~3% in back-up section)

Table I*

Solvent	Method Classification+	Detection limit mg/sample**	Sample time at 1 lpm Min (min)	Sample time at 1 lpm Max (min)	Temp °C	Mol. Wt.
Acetone	D	----	.5	7.7	60	58.1
Benzene	A	0.01	0.5	55	9	78.1
Carbon tetrachloride	A	0.20	10	60	60	154.0
Ethylene dichloride	D	0.05	1	12	90	99.0
Methyl Ethyl Ketone	B	0.01	0.5	13	80	72.1
Toluene	B	0.01	0.5	22	120	92.1
Xylene	A	0.02	0.5	31	100	106

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* Adapted from NIOSH Manual of Analytical Methods - Organic Solvents

+ NIOSH Classification of Analytical Methods: A-Recommended; B-Accepted; D-Operational.

** Lower limit in mg/sample for compound at 16 x 1 attenuation on GC fitted with 10:1 splitter; value can be lowered by reducing attenuation or eliminating splitter.

Advantages and Disadvantages

1. sampling device small
2. minimal interference
3. analysis is quick
4. multiple solvents analyzed in one sample
5. overloading and breakthrough possible

1. NIOSH Manual of Analytical Methods.

Sampling and Analysis[2]

Method applies for the following compounds:

Compound	TLV (ppm)
n-amyl acetate	100
n-butyl acetate	150
n-butyl alcohol	100
cyclohexane	300
ethyl acetate	400
heptane	500
hexane	500
isoamyl acetate	100
isobutyl acetate	150
isobutyl alcohol	100
isopropyl acetate	250
methyl acetate	200
Methyl Ethyl Ketone	200

Sample Collection

type: breathing zone

apparatus: 7 cm long glass charcoal tube, both ends sealed, 6 mm O.D., 4 mm I.D. containing 2 sections of 20/40 mesh activated charcoal separated by 2 mm portion of urethane foam; calibrated sampling pump

flow rate: 1 lpm or less

sampling time: 10 min at 1 lpm

Sample Analysis

type: GC-FI

apparatus: wet chemistry, GC-FI

procedure: desorb with carbon disulfide, analyze with GC-FI

Range and Sensitivity

not determined

Prescision

10.5% RSD

Interferences

not stated

Effect of Storage

not stated

Advantages and Disadvantages

not stated

[2] NIOSH Manual of Analytical Methods

Sampling and Analysis[4] - Methyl Iso Amyl Ketone

TLV = 100 ppm

Sampling Method

no information given

Sample Analysis

Gas chromatography with infrared or mass spec analysis

No other information given

4. Patty: Industrial Hygiene and Toxicology, p. 1724.

Sampling and Analysis - Ethyl Alcohol [1,2]

TLV = 1000 ppm

Sample Collection

type: not stated

apparatus: two gas washing bottles containing distilled H_2O in series, calibrated sampling pump

flow rate: 0.12 cfm

sampling time: not stated

Sample Analysis

type: wet chemistry titration

apparatus: wet chemistry

procedure: 1. ethyl alcohol oxidized to acetic acid by 0.1 N chromic acid

2. excess chromic acid determined by titration with 0.1 N sodium thiosulfate; 1 ml sodium thiosulfate = 1.15 mg ethyl alcohol

Range and Sensitivity

10 ppm/cf

Precision

not stated

Interferences

Any chemical oxidized by 0.1 N chromic acid to acetic acid

Effect of Storage

not stated

Advantages and Disadvantages

not stated

1. Browning: Toxicity and Metabolism of Industrial Solvents, p. 325.
2. AIHA Analytical Abstract.

Sampling and Analysis - Methyl Alcohol [1,2]

TLV = 200 ppm

Sample Collection

type: not stated

apparatus: midget impinger containing H_2O or 5% $KMnO_4$

flow rate: not stated

sampling time: not stated

Sample Analysis

type: colorimetric

apparatus: wet chemistry, standards, photometer

procedure: 1. H_2 sample oxidized to formaldehyde which is detected with Schiff's reagent

2. photometric comparison with standards

Range and Sensitivity

up to 250 ppm; 40 ppm sensitivity for 10-litre sample

Precision

colorimetry not linear at 30 ppm; linear 100-250 ppm; 92% collection efficiency at 200 ppm

Interferences

aldehydes do not interfere

Effect of Storage

not stated

Advantages and Disadvantages

not stated

1. Jacobs, pp. 614-617.
2. Browning: Toxicity and Metabolism of Industrial Solvents, p. 312.

SECTION 2

Toxicity of Solvents included in Section 1

The Acetates[1,2]

All of the acetates are eye, nose and throat irritants and may cause slight anesthesia in high concentrations. In addition, the repeated inhalation of methyl acetate vapors may eventually cause blindness. Methyl, iso and n-amyl acetate are cumulative poisons. Repeated long-term inhalation of iso and n-amyl acetates may result in injury to the respiratory tract, liver, kidneys and digestive system; in high concentrations they cause irrigation and narcosis.

Amyl acetate is primarily an irritant and, to some extent, a narcotic. It appears that the chemical does not cause systemic injury. Work reported in Browning states that even high concentrations are not immediately fatal to animals; in addition, amyl ac etate does not cause dermatitis , although it does dry the skin.

Symptoms of loss of appetite, a sensation of fullness, eructations and dyspepsia were described in men using lacquer in which amyl acetate was the chief consitituent. There is a report of industrial injury when a worker had attacks of vertigo, headache, sudden blindness in both eyes and an unsteady gait. He developed bilateral optic atrophy a central scotoma in one eye and narrowing of the visual field in the other. It was thought that these symptoms resulted in the formation of methyl alcohol and formaldehyde during the metabolism of methyl acetate.

1. NPIRI: Raw Materials Data Handbook
2. Browning: Toxicity and Metabolism of Industrial Solvents, pp.522-542.

ALCOHOLS [1,2]

In terms of the cumulative injury to the internal organs of workers, the effects of alcohols tend not to be severe. However, some of them have been known to have a cumulative effect on the central nervous system, for example, methyl alcohol. All the alcohols are weaker narcotics than the hydrocarbons; their toxicity increases with higher molecular weight.

Cases of acute poisoning are rare from inhalation of methyl alcohol. but they have occurred. The symptoms of two patients who drank methyl alcohol were vomiting, epigastric pain, irrational behaviour, failure of vision and semi coma. Neither ever recovered full vision; one man was blinded.

In the case of n-butyl accohol, if the dosage is high enough, it acts as a narcotic. Symptoms of restlessness, irritation of mucous membrane, ataxia, prostration, narcoses, and death will result. Rabbits given 1 to 1.5 g/Kg orally showed slight paralysis after 20 to 30 minutes.

Eye irritation will result from exposure to n-butyl and isobutyl alcohol. In addition, n-butyl will produce an inflammation of cornea with burning, lacrimation, photophobia and blurring of vision at concentrations of 200 ppm or above. Both alcohols will produce vacuolar formations in superficial layers of cornea. Although methyl alcohol is usually thought to produce its effect on the eyes through ingestion, there have been instances of eye injury through inhalation or absorption through the skin. Methyl alcohol causes injury primarily by affecting the retina and optic nerve.

Loss of visual acuity, field of vision, depth perception, eye condensation, poor distance judgement follow intoxication with ethyl alcohol. Cases of industrial poisoning from repeated exposure to the vapour of methyl alcohol have usually been manifested by conjunctivitis, headache, giddiness, insomnia, gastrointestinal disturbances and failure of vision. The main industrial hazard of ethyl alcohol is that workers may drink it. However, inhalation of the vapours of ethyl alcohol can have similar effects to ingestion.

Dermatitis of fingers and hands in the form of fissured exzema occurs on prolonged contact with n-butyl alcohol. Serious dermatitis also occurs with isobutyl alcohol.

1. Browning: Toxicity and Metabolism of Industrial Solvents.
2. NAPIM: Raw Material Data Handbook.

ALIPHATIC HYDROCARBONS[1]

The higher alkanes, n-nonane, n-decane, n-undecane, n-dodecane, n-tetradecane, tridecane, n-pentadecane, 2,6-dimethyl undecane, and 2 methyl decane are liquid paraffin hydrocarbons. Repeated or prolonged skin contact will dry and defat the skin, resulting in irritation and dermatitis. Contact of liquid hydrocarbons with lung tissue will lead to pneumonitis, pulmonary edema, and hemorrhage.

Patty: Industrial Hygiene and Toxicology, p. 1196.

Benzene

Benzene is toxic by all routes of entry. Although skin absorption is believed to be poor[1] errythema, blistering and dermatitis may result from skin contact with the liquid.

Symptoms of chronic benzene exposure were described by Helmur[2], including headaches, fatigue, cutaneous hemorrhages, nervousness, sleeplessness, shortness of breath and palpitations. The work place atmosphere was estimated to contain 5,320 p.p.m. benzene.

Various blood alterations result from benzene exposure. Many cases of leukemia associated with benzene exposure are reported in the literature, several involving persons employed in the printing trades. One rotogravure pressman developed fatal aplastic anemia four years after freedom from benzene exposure. His work history included twenty five years of association with ink solvents and thinners containing 10-35% benzene by volume.[3] Other cases are described in the Criteria Document.

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[1] "Occupational Exposure to Benzene, criteria for a recommended standard," U.S. Department of HEW, 1974, work reported pp. 25

[2] as above, pp.29-30

[3] Smith: "Benzal Fatality After Four Years Freedom From Exposure", N.Y. Industrial Bulletin, August 1943, pp.329

THE GLYCOLS

Industrially, the chief hazard of ethylene glycol is ingestion, which is fatal. It presents negligible hazards to health in industrial handling at normal temperatures. However, prolonged breathing of mists or heated vapors or sustained and extensive skin contact may affect the central nervous system and cause kidney injury.

Ethylene glycol is low in oral toxicity, is not significantly irritating to eyes or nose and is not readily absorbed through the skin. In addition, its vapor pressure is low enough so that toxic concentrations will not occur at room temperatures.

No cases of injury to human eyes have been reported.

Although the hazards from inhalation are low, there is a hazard where the material is being handled hot or where agitation or other mechanical operations may create a fog or mist in the air. At these levels, however, the chemical has a disagreeable odor and causes eye irritation.

2-ethoxyethanol is the least toxic of all ethers. No evidence of injury from industrial use, in the form of cellosolve, has been noted.

Brown: Toxicology and Metabolism of Industrial Solvents, p. 522, 602.

TRIMETHYLBENZENES AND OTHER ALKYL AROMATICS

Isopropylbenzene (cumene) is a skin irritant that can be absorbed slowly form intact skin. It has a narcotic effect but no injurious haemopoietic effect. No injuries from industrial use have been reported; however, experiments with animals have shown that there may be possibilities for injury to the liver and kidney.

Hyperaemia and congestion of the lungs, liver and kidneys have been reported by Browning.

The narcotic effect of isopropylbenzene manifests itself much more slowly than that of benzene, but once it happens, it is much more persistent.

The effects of 1,3,5 trimethylbenzone (mesitylene) and 1,2,4 trimethylbenzene (pseudocumene) are considered to be the same since most of the work with these chemicals has been with the paint thinner "Fleet X", of which they are components.

Absorption takes place easily by vapor inhalation but can also occur through the gastrointestinal tract and probably, although slowly, through the intact skin.

These two chemicals are ~~central nervous system depressants,~~ respiratory irritants and perhaps haemopoietic depressants.

Symptoms of intoxication include blood and bone marrow disturbances where the coagulation process may be impaired; respiratory disturbances of the asthmatic type and nuerological disturbances, such as headache, fatigue and drowsiness.

Pulmonary edema, pneumonitis and haemorrhage will result from contact of liquid aromatic hydrocarbons with lung tissue. Repeated or prolonged contact with the skin will cause dermatitis due to their dehydrating and defatting action.

Basically, p-ethyl toluene has the same effects and causes symptoms similar to the other chemicals.

1. Browning: Toxicity and Metabolism of Industrial Solvents, pp. 95, 113, 117.
2. Patty: Industrial Hygiene and Toxicology, p. 1220.

TOLUENE

Toluene is toxic by all routes of entry, but percutaneous absorption is too slow to produce systemic poisoning; dermatitis may result.[1]

When placed in rabbit's eyes, toluene caused a slight transient conjunctival irritation, but no corneal damage was detectable. Two patients who sustained eye splashes of toluene suffered transient eye disturbances, but healing was complete within 48 hours.[2]

Patty reports, acute and chronic human exposures to 50-800 ppm for 8 hours without white blood count alterations. Specific exposures and reactions follow:

Exposure	Effect
200	Mild fatigue, weakness, confusion, paresthesias of skin, moderate insomnia, restlessness.
300	Same as above, but more pronounced.
400	Mental confusion also noted.
600	Extreme fatigue, mental confusion, exhilaration, nausea, headache and dizziness after 3 hours; after 8 hours, fatigue, mental confusion, nausea and dizziness more pronounced; pupils dilated, decreased coordination.

Chronic exposures to the same concentrations 15 times in three months did show blood changes.[3]

Carpenter and co-workers, also exposed humans to toulene for 7 to 8 hours with the following results:

Exposure (ppm)	Effect
200	Mild eye and throat irritation, slight exhilaration.
400	Slight eye irritation with lacrimation, nausea, hilarity.
600	Lassitude, hilarity, verbosity (loss of appetite , listlessness as after effects).
800	Transistory headaches, vision problems, feeling of drunkedness, nausea, verbosity.

The case of a man addicted to pure toluene is reported in the Criteria Document.[5] After 14 years of addiction, permanent cerebral atrophy resulted. Exposure was through oral inhalation of the vapor from a soaked rag.

Average workroom concentrations of 67ppm resulted in changes in serum glycoproteins, seromucoid and haptoglobulins production in exposed women, according to polish work included in the Criteria Document.[6] The investigators speculated that these changes were evidence of early alterations in liver function.

Rotogravure workers exposed to toluene concentrations of 200ppm. were examined by Forni etal.[7] No statistically significant chromosome changes were found.

Additional epidemeological and animal toxicity data are found in the Criteria Document.[8]

3 - Patty, Frank, Industrial Hygiene and Toxicology, p.1227.

1 - Gleason, etal., Clinical Toxicology of Commercial Products.

2 - Grant, Toxicology of the Eye, p.1027.

4 - Carpenter, C.P. etal., "Studies on the Inhalation of 1,3-Butadrene; with a Comparison of its Narcotic Effect with Benzol, Tolual and Styrene and a note by the Human", Journal of Industrial Hygiene and Toxicology, 26:69 - 78, 1944.

5 - "Occupational Exposure to Toluene, Criteria for a recommended standard,"[1] U. S. Department of HEW, 1973, p.24.

6 - As above, pp.25-26.

7 - Forni, A., etal., "Chromosome Studies in Workers Exposed to Benzene or Toluene or Both". Archives Environmental Health, 22:373-78, 1971.

8 - "Occupational Exposure to Toluene", pp.16-35.

Xylene

The majority of human volunteers exposed to 200ppm xylene vapor for 3-5 minutes developed eye, nose and throat irritation.[1] Exposure to 0.6-1.9 mg/m^3 (about 0.14-0.44 ppm) is the lowest odor threshold reported in the Criteria Document.[2] Marked inhibition of the electrical activity of the human cortex has been reported at 0.05 ppm[3]. Several cases of occupational exposure to toluene are discussed in the Criteria Document; however, multiple solvent exposures make it difficult to attribute the results to xylene specifically.[4]

Accidental splashes of xylene in the human eye have caused transient superficial damage, with rapid recovery. Xylene vapor induces the formation of vacuoles in the corneas of cats; recovery is complet within a day following exposure.[5]

Patty reports chronic animal exposure.[6] Rats and rabbits placed in atmospheres containing 690 ppm for 8 hrs/day, 6 days/week for 130 days showed no significant blood changes. An exposure of rabbits to 1150 ppm, 8 hrs/day, 6days/week for 55 days caused decreased red blodd cells, decreased leukocytes and increased platelets.

Fetal malformations and mortalities have been associated with xylene exposure in chicks and rats.[7]

Skin irritation from xylene is more severe than from benzene or toluene.[8]

1. Nelson, K.W. et al, Sensory Response to Certain Industrial Solvents, Industrial Hygiene and Toxicology, 25:282-85, 1943
2. Occupational Exposure to Xylene, Criteria for a Recommended Standard, U.S. Dept. of HEW, 1975 pp. 20-21
3. as above, pp. 20
4. as above, pp.21-29
5. Grant: Toxicology of the Eye pp. 1089-90
6. Patty: Industrial Hygiene and Toxicology pp. 1234
7. Criteria Document pp.36-37
8. Patty: pp.1234

Sampling and Analysis- Xylene

TLV-100ppm

Sample Collection : as stated previously

Sample Analysis: as stated previously

Range and sensitivity: lower limit < 10µg/sample

Precision: 60-200 ppm RSD9.0%; 5 ppm RSD13%

Interferences: 1) water vapor 2) any compound having approximately the same retention time.

Effect of storage: as stated previously

Advant/Disadvant: as stated previously

2. Occupational Exposure to Xylene, Criteria for a Recommended Standard, U.S. Department of HEW, 1975, pp.82-83

* the first sampling test is the Standard for Organic Sampling.

Penetrating oil - exterior

- Paraffin
- Zinc stearate
- Turpentine/paint thinner
- Pentachlorophenol
- Boiled linseed oil
- Burnt Sienna in oil
- Raw umber in oil
- Indial iron oxide in oil

Ref: 1

Phenolic Coatings

Resin: phenolformaldehyde
phenolfurfurae
Drying oil
Solvents, including coal tar hydrocarbons
aliphatic hydrocarbons
ketones
esters
alcohols
aromatic hydrocarbons

Ref: 1, 6

Polishes

Wax

1 lb. softened (melted) wax
1/2 pt. warm turpentine
1 t. ammonia

Liquid polishes

Silicones

Polishing abrasives

- Boron carbide
- Aluminum oxide
- Flint
- Garnet
- Emery
- Rottenstone
- Pumice
- Tripoli
- Diatomaceous earth

Ref: 1

SECTION 3

Toxicity, Sampling and Analysis of Additional
Organic
Solvents

METHYLENE CHLORIDE

Methylene chloride causes dizziness, nausea, tingling or numbness of the extremities, sense of fullness in the head, sense of heat, stupor or dullness, lethargy and drunkenness in excessive exposures. Very high concentrations may produce rapid unconsciousness.

Mice exposed to 11,000 ppm for 8 hours survive; the mouse LC_{50} is about 15,000 ppm.

Various experimental animals exposed to 5,000 ppm 7 hrs/day, 5 days/wk, for 6 months showed no abnormal pathology; at 10,000 ppm slight narcosis was observed.

Methylene Chloride is mildly irritating to the skin; eye splashes are painful and irritating, but not injurious.

One human fatality is reported.[1]

It has recently been shown that methylene chloride is metabolized to carbon monoxide in humans.[2]

[1] Patty: Industrial Hygiene and Toxicology, pp 1257-58

[2] Stewart et al.: "Formation of Carbon Monoxide Following Exposure to Dichloromethane (Methylene Chloride), Dept. of Environmental Medecine, Medical College of Wisconsin.

Sampling and Analysis - Methylene Chloride

TLV = 500 ppm

No specific methodology is included here, however Patty notes several procedures, including GC - mass spec and GC - IR, are applicable (p.1246)

NAPHTHAS

Naphthas are cumulative toxins. Repeated inhalation of vapors or sustained skin contact may cause mild anemia; in high concentrations inebriation and narcosis result.[1]

[1] Grant: Toxicology of the Eye.

Sampling and Analysis - Naphtha

TLV = 100 ppm

Naphthas are combinations of various petroleum distillates. GC analysis must be used to determine composition.

Range and Sensitivity

0.1-10 ugll; 0.1 ugll

Precision

< 5% RSD

Interferences

1) sulfur dioxide, removed by adding 1% acitone to reagent before use.

2) O_3

3) PAN

4) other nitrogen oxides

5) strong oxidizing or reducing agents

Effect of Storage

3-4% loss of color/day

Advantages and Disadvantages

direct ~~coloration of absorbing material.~~

1. NIOSH Manual of Analytical Methods

B. Pigments

Section 1

Inorganic Pigments

BARITE (lead sulfate)

For the toxicity of Barite, refer to "Lead".

Sampling and Analysis - Barite (lead sulfate)[1]

Sample Collection

type: bulk sample of white pigment

apparatus: not stated

flow rate: not pertinent

sampling time: not pertinent

Sample Analysis

type: wet chemistry

appartus: wet chemistry

procedure: 1) add ammonium hydroxide to HCl-insoluble fraction of $BaSO_4$ analysis until faint precipitant forms, add 2ml concentrated HCl

2) dilute to 200 ml, pass rapid current of H_2S through clear solution until lead sulfite precipitates, wait 15 minutes

3) filter, wash filtrate with H_2S, boil filter and prepipitate ~~in 50 ml dilute HNO_3 until lead~~ sulfide dissolves, filter and wash, add 10 ml H_2SO_4 to filtrate

4) evaporate until copious H_2SO_4 fume evolves, cool, add 75 ml distilled H_2O and 75 ml ethyl alcohol (95%), let it stand 1 hr.

5) filter, wash with alcohol and distilled H_2O, dry and ignite at 600° C, weigh as $PbSO_4$

$$\%Pb = \frac{\text{weight } PbSO_4 \times 0.6833 \times 100}{\text{original sample wt. (g)}}$$

Range and Sensitivity

method designed for analysis of white pigments

No additional information given.

[1] Melcher, Frank J.: Standard Methods of Chemical Analysis, D. Van Nostrand Co. Inc., Princeton, 1963 pp 1692-3

Blacks: Carbon, Animal, Vegetable, Lamp

Carbon black particulates have been the focus of several studies. One investigated the incidence of cancer in the manufacture of carbon black; the mortality sutdy cohort included 1,085 employees. No excess deaths were found.[1] Another stimulated by work showing that carcinogenic compounds could be extracted from carbon black, tested the carcinogenic activity of extracts on the skin of mice. Six squamous carcinomas were produced in 212 animals; 126 animals survived the duration of the experiment.[2]

Carbon black is used as a pigment in eye cosmetics. After two years of exposure, black pigmentation of the upper tarsal border has been reported. Although there is lymphatic infoltration and the pigment is surrounded by macrophages in these cases, no symptoms are evident.[3]

No reference to toxic properties of animal, vegetable, of lamp blacks was found.

1. Ingalls: Incidence of Cancer in the Carbon Black Industry, Cancer, p. 662, 1950.
2. von Hamm and Mallette: Studies on the Toxicity and Skin Effects of Compounds in the Rubber and Plastics Industries, Ind Hyg and Occup Med, 6:237-42, 1952
3. Grant: Toxicology of the Eye, p.229.

Sampling and Analysis - Carbon Blacks[1]

TLV =3.5 mg/m^3

Sample Collection

type: breating zone

apparatus: preweighed 0.8 µm 35 mm filter and holder, calibrated sampling pump

flow rate: 2 lpm

sampling rate: variable

Sample Analysis

type: weighing, sizing

apparatus: immersion oil, optical microscope, porton graticule, balance

procedure: 1. weigh sample and compare to tare weight

2. render filter transparent and count and size enough particles to obtain significant count in each size range

Range and Sensitivity: limited to lower limit of the optics used, no upper bound.

Precision: dependent upon the counter; $\pm\sqrt{n}$, where n=number counted in each size range

No additional information given.

1. NIOSH: The Industrial Environment-Its Evaluation and Control, pp.145-53.

BLANC FIXE (BARIUM SULFATE)

Blanc Fixe rarely causes intestinal disturbances. When injected into the anterior chamber of the eyes of rabbits, it attracts many leukocytes, causes hyperemia of the iris, dilation of the peri limbal vessels and clouding and vascularization of the cornea. The particles later become encapsulated with fibrim and endothelial cells. [1]

---

[1] - Grant, Toxicology of the Eye p. 173

Sampling and Analysis - Blanc Fixe (Barium Sulfate)[1]

TLV

Sampling Collection

type: bulk sample of white pigments

apparatus: not stated

flow rate: not applicable

sampling time: not applicable

Sample Analysis

type: wet chemistry

aparatus: wet chemistry

procedure: use residue from

TiO_2 analysis

1) transfer residue to platinum crucible
2) add 10x weight anhydrous sodium carbonate
3) filter and wash insol carbonate
4) acidify with HCl; boil, filter, wash with H_2O
5) add Methyl red indicator, adjust pH to alkalinity with ammonium hydroxide
6) add 4ml HCl (1:1) and dilute to 200ml
7) boil, add excess hot H_2SO_4
8) digest on steam bath
9) ignite in oxidizing atmosphere; weigh as B_aSO_4

$$\% \; B_aSO_4 \quad \frac{\text{wt of precipitate X 100}}{\text{wt of original sample}}$$

Range and Sensitivity: method designed for analysis of white pigments

Precision: not stated

Interferences: not stated

Effect of Storage: not stated

Advan/Disadvan: not stated

1 Meleher, Frank J., ed., Standard Methods of Chemical Analysis,
D. Van Nostrand Co., Inc., Princeton 1963, Vol.2B - p.1691

Cadmium-Mercury Red

Cadmium-mercury red is essentially a cadmium pigment. Medium-pure Cd-Hg red is formulated as[1]:

78% cadmium sulfide
22% mercuric sulfide

It is highly toxic, especially by inhalation of the fume and may be fatal.[2]

No poisonings were found in a plant having cadmium sulfide fume concentrations of 1.0-6.6 mg/m^3 and dust concentrations of up to 31 mg/m^3.[3]

Cases of acute cadmium poisonings have shown proliferative lesions of the alveoli and cuboidal metaplasia of the alveolar sacs, with thickening and edema of the septa.[4]

Emphysema, fibrosis and degeneration of the kidneys, alteration in liver function and bone marrow changes have been associated with chronic cadmium poisoning. Evidence exists that the first signs of chronic exposure may occur after a long latent period following cessation of exposure and that the disease if progressive.[5]

1. Snell, Foster D. and Leslie S. Ettre, eds, Encyclopedia of Industrial Chemical Analysis, Interscience, 1973, Vol. 17 pp. 184-5
2. Condensed Chemical Dictionary, 8th Ed.
3. Browning: Toxicity of Industrial Metals, pp.90
4. Browning: pp. 86-87
5. Browning: pp.88-89

Sampling and Analysis-Cadmium Sulfide as Cd[1]

TLV=

Sample Collection

type:atomic absorption

apparatus: wet chemistry.AA spectrophotometer , standards procedure:
1) samples are ashed with HNO_3 and solubilized in an acid solution
2) samples and standards aspirated into the AA, flame fed by C_2h_2, analysed at 2288A.

Range and Sensitivity: 0.04-5 µg/ml; 0.04 µg/ml

Precision: 2% RSD (analytical)

Interferences: none

Effect of Storage: not stated

A[illegible]: [illegible]

NIOSH Manual of Analytical Methods

Calcium Carbonate

Calcium carbonate is generally regarded as an inert dust.[1] It exhibits no significant oral toxicity.[2] The powder produces no toxic effect when applied to the surface of the rabbit eye.[3]

1. Drinker and Hatch: Industrial Dust, p. 56.
2. Gleason: Clinical Toxicology of Commercial Products.
3. Grant: Toxicology of the Eye, p. 215

Sampling and Analysis - Calcium Carbonate[1]

TLV=

Sample Collection

type: buld sample of white pigment

apparatus: not stated

flow rate: not pertinent

sampling time: not pertinent

Sample Analysis

type: colorimetric

apparatus: wet chemistry

procedure: 1. filtrate from $PbSO_4$ analysis neutralized with ammonium hydroxide or HCl, add 10 ml excess ammonium hydroxide, pass H_2S into solution for 10 min

2. let settle, filter, add bromine water, boil to remove bromine, dilute to 200 ml, neutralize with ammonium hydroxide and add 5 ml excess

3. slowly add 16 saturated ammonium oxalate, boil 5 min and keep warm 1 hr., filter and wash precipitate with hot H_2O

4. wash precipitate into beaker with hot H_2O and 40 ml H_2SO_4, save filter

5. dilute solution to 200 ml, heat to 90°C, titrate with 0.1N potassium permanganate, place paper in solution and titrate to end

% as $CaCO_3$ = $\frac{[illegible]}{\text{weight of sample}}$

Range and Sensitivity: method designed for analysis of white pigment

Precision: not stated

Interferences: not stated

Effect of Storage: not stated

Advantages/Disadvantages: not stated

1. Meleher: Standard Methods of Chemical Analysis, pp. 1693-94.

CHROME ORANGE

Chrome orange has the same effects as lead chromate.[1] See "Chrome Yellow".

Gleason: Clinical Toxicology of Commercial Products

Sampling and Analysis - Chrome Orange

TLV =
See bichromates

CHROME YELLOW

Chrome yellow is essentially lead chromate, but lighter shades may contain lead sulfate.[1] The compound has a lower acute toxicity than soluble lead salts. The acute reaction is that of chromate poisoning; signs of chronic lead poisoning may appear days or weeks after exposure.[2]

Chromate dermatitis, sensitization, ulcers and potential of lung carcinoma are described in the section on "Chromic Acid". Also, see "Lead".

1. Meheler: Standard Methods of Chemical Analysis, 1968
2. Gleason: Clinical Toxicology of Commercial Products

Sampling and Analysis - Chrome Yellow

TLV =

See bichromates

Clay

Clay used as an extender pigment is natural hydrous Aluminum Silicate.[1] Inhalation of powdered aluminum by [illegible] gave no evidence of injurious effects at a concentration of 1.57 mg/ft^3. Exposure lasted 8 hrs./day for one year. A progressive accumulation of the substance in the lung was noted. Similar findings resulted from doses given by injection.[2]

1. Meleher: Standard Methods of Chemical Analysis pp.1801
2. Browning: Toxicity of Industrial Metals, pp.7

[illegible]

[illegible] barium sulfide and zinc oxide. [illegible] kidneys, and muscular paralysis.[1]

A further discussion of the toxicity of zinc oxide and barium sulfide is found under the specific compound.

1. Merck Index, 8th Ed., pp. 324, 1130, 129.

Sampling and Analysis - Lithopone as Ba, Zn

TLV =

Total barium sulfate determination is outlined under Barium Sulfate; procedures for zinc are found under Zinc Oxide. A procedure for total barium follows.

Sampling and Analysis - Barium[1]

Sample Collection

type: breathing zone

apparatus: 0.8µm 35mm cellulose membrane filter and holder; calibrated sampling pump.

flow rate: not stated

sampling time: not stated

Sample Analysis

type: atomic absorption

apparatus: wet chemistry; AA spectrophotometer; standards

procedure: 1. samples are asked with HNO_3 and solubilized in an acid solutio

2. samples and standards aspirated into AA, flame-fed by N_2O-C_2H_2, analyzed at 5536 Å

Range and Sensitivity

0.1 - 50 µg/ml; 0.1 µg/ml

Precision

2% RSD (analytical)

Interferences

alkali salts, prevented by 1000µg/ml KCl in samples and standards

Effect of Storage

not stated

[illegible]

not stated

1. MIC -- Method of Analytical [illegible]

Lithol Red

Lithol red may be composed of either a barium salt or calcium salt. Barium Browning states that barium salts are highly toxic to humans by ingestion and to animals by intravenous injection; however, the only known effect of barium exposure in industrial workers is baritosis, a benign pneumoconiosis. The radiographic changes in this disease results from disposited dust in the lung and are characterized by evenly distributed circumscribed nodules, larger and more thickly deposited than those resulting from silicosis. The nodulation generally disappears following cessation of exposure. One researcher reported that chronic bronchial and peribronchial irritation may result.[1]

Calcium

No information specific to calcium salts was found.

---

1 - Browning *Toxicology of Industrial Metals* p. 55-56

[illegible]

[illegible]

Sample Collection: no information given

Sample Analysis:

type: spectoral

apparatus: optical emission spectograph

procedure: elemental spectra of the sample is determined and compared with pigment spectra, found in the Encyclopedia of Spectroscopy

No other information is given.

---

1 - Welcher, Standard Methods of Chemical Analysis, p.1683.

[illegible]

[illegible] Hatch considered magnesium carbonate to be an inert dust.[2]

Although [illegible] reported [illegible] in the pol[illegible] trades complained of discolored sputum, no evidence of damage exists.[3]

1. Browning: Toxicity of Industrial Metals, pp. 180
2. Drinker and Hatch: Industrial Dust, pp. 56
3. Browning: pp. 183

[illegible]

Titanium dioxide is generally [illegible] absorption through ingestion has proved [illegible] a pound of [illegible] TiO_2 did not create disease and [illegible] eliminated [illegible].

Browning assesses the hazard of developing titaniosis, a fibrotitic lung disease, as very slight.[2]

Nonspecific lesions were found in the lungs of rats exposed to concentrations of TiO_2 ranging from 10 to 328 mppcf. Ashed lung tissue showed greater than 10% titanium content. Exposures lasted up to 10 months, 5 day/week.[3] Long term, low level exposures to SO_2 diminished the lung clearance capacity of rats exposed to TiO_2.[4] Risto et al found slightly increased connective tissue in the lungs of humans exposed to TiO_2, particulate size 0.1-1.0 um. The TiO_2 was phagocytized and accumulated within lysomes.[1]

1. Gleason et al, Clinical Toxicology of Commercial Products.
2. Browning, Toxicity of Industrial Metals.
3. Christie et al, "Pulmonary Effects of Inhalation of Titanium Dioxide by Rats," Industrial Hygiene Journal, Jan-Feb 1963, pp 42-46.
4. Ferin, J. and L. J. Leach, "The Effect of SO_2 on Lung Clearance of TiO_2 Particles in Rats, AIHA Journal, June 1973, pp 260-63.

Sampling and Analysis - Titanium Dioxide[1]

TLV = "nuisance particulate." When toxic impurities are not present <1% also <5-7 um in diameter.

Sample Collection

type: bulk sample of white pigment

[illegible]

[illegible]

[illegible]

[illegible]

[illegible]

[illegible]

procedure: 1) sample dissolved in hot H_2SO_4 - ammonium sulfate, diluted,

acidity adjusted

2) Solution passed through Jones reductor, caught in excess

of ferric sulfate

3) Ferrous sulfate titrated with 0.1 N potassium permanganate

Range and Sensitivity: method designed for analysis of white pigments

Precision: not stated

Interferences: arsenic, iron, chromium

Effect of Storage: not stated

Advantages/Disadvantages: not stated

1. Snell, Foster Dee and Leslie S. Ettre, eds., Encyclopedia of Industrial Chemical Analysis, Interscience, New York, 1973, 17:167.

Sampling and Analysis - Titanium Dioxide[2]

TLV =

Sample Collection

type: bulk sample of white pigment

apparatus: not stated

flow rate: not applicable

sampling time: not applicable

Sample Analysis

[illegible]

[illegible]

procedure: 1) [illegible]

2) [illegible]

3) [illegible]

[illegible]

[illegible]

[illegible]

[illegible]

Precision: not stated

Interferences: not stated

Effect of Storage: not stated

Advantages/ Disadvantages: not stated

2. Welcher, Frank J. ed., Standard Methods of Chemical Analysis; D. Van Nostrand Co., Inc., Princeton, 1963, Vol. 2B, p. 1391

[illegible]

[illegible] Browning, [illegible] employed 2 - 35 years in the manufacture of zinc oxide. Zinc absorption and excretion was high, as evidenced by blood, urine and fecal analysis.[1]

Zinc oxide is 98-99% pure.[2]

1. Browning: Toxicity of Industrial Metals.
2. Welcher: Standard Methods of Chemical Analysis, Vol. 2B, p. 1381.

Sampling and Analysis - Zinc Oxide as Zn[1]

TLV = 5 ppm (fume)

Sample Collection

type: breathing zone

apparatus: 0.8 µm 35mm cellulose membrane filter and holder; calibrated sampling pump

flow rate: not stated

Sample Analysis

type: atomic absorption

apparatus: wet chemistry; AA spectrophotometer; standards

procedure: 1. samples are ashed with HNO_3 and solubilized in an acid solution

2. samples and standards aspirated into AA, flame-fed by C_2H_2, analyzed at 2139 Å

Range and Sensitivity

0.025-2 µg/ml; 0.025 µg/ml

Precision

[illegible]

Interferences

[illegible]

[illegible]

Health Risk:

Zinc sulfide [illegible] therapeutically sprayed into the eyes.[1] It is insoluble in water.[2]

In gastric acid it is toxic, ingestion will result in the development of H_2S in the stomach. The soluble sulfides are strong local irritants to the skin and mucous membranes.[3]

1. Grant: Toxicology of the Eye, p. 1099.
2. Merck Index, p. 1130.
3. Gleason: Clinical Toxicology of Commercial Products.

Sampling and Analysis - Zinc Sulfide

TLV =

see Zinc Oxide

Section 2

Organic Pigments

[illegible][1]

[illegible]

TLV -

Sample Collection: No information given

Sample Analysis

type: spectral

apparatus: optical emission spectrograph

procedure: elemental spectra of the sample is determined and compared with pigment spectra, found in the Encyclopedia of Spectroscopy

No additional information given.

1. Welcher: Standard Methods of Chemical Analysis, p. 1683.

[illegible]

[illegible]

Hansa Yellow

Chemically, [illegible] coupled with o-chloroacetoacetanilide.[1]

[1] [illegible] Standard Methods of Chemical Analysis, p. [illegible]

Sampling and Analysis - Hansa Yellow

See Standard Sampling and Analysis

Madder Lakes

Madder lakes are made by oxidizing anthracene to anthraquinone, the sulfonic acid of which is then fused with caustic soda and potassium chlorate; the meld is run into hot water and alizarin precipitated with HCl. It is of low toxicity.[1]

[1] Condensed Chemical Dictionary, 8th. ed.

Para Red

Para Red is a diazotized p-nitroaniline coupled with β-naphthol.[1]

[1] Welcher: Standard Methods of Chemical Analysis, p. 1033.

Sampling and Analysis - Para Red

See Standard Sampling and Analysis

Benzidine Yellow

Benzidine yellow is one of a family of organic azo pigments prepared by coupling the tetrazonium salt of 3,3-dichlorobenzidine with acetoacetarylides. It is probably toxic.[1]

[1] Condensed Chemical Dictionary, 8th ed.

Sampling and Analysis - Benzidine Yellow

See Standard Sampling and Analysis

C. [illegible] Organisms Investigated

[illegible][1]

The only [illegible] effects [illegible] direct [illegible] of the acid on the skin, eyes, [illegible] exposed teeth. Patty [illegible] from 80% [illegible] severe burns; from 50 - 80%, moderate to severe burns, and below 50%, relatively mild injury.

The LD_{50} for rats has been shown to be 3.3 g/kg. The LC_{50} for 1-hour exposure in guinea pigs and mice is about 5000 ppm. Symptoms of irritation of the eyes and respiratory tract occurred above 100 ppm.

Except for local effects, no evidence of cumulative toxicity has been found.

1. Patty, Industrial Hygiene and Toxicology, pp. 1779-80.

Sampling and Analysis - Acetic Acid[1]

TLV = 10 ppm

Sample Collection

type: not stated

apparatus: gas washing bottle containing measured volume of 0.01 N sodium hydroxide

flow rate: not stated

sampling time: not stated

Sample Analysis

type: wet chemistry titration

apparatus: wet chemistry

procedure: excess alkali titrated to colorless endpoint, phenolphthalein as indicator

[illegible]: [illegible] ppm

[illegible]

[illegible]

1. [illegible]

Ammonia

Ammonia is both a local and systemic toxin.

A compilation of case histories of human ammonia poisonings revealed the following symptoms:[1]

1. varying degrees of acute inflammation of the respiratory tract
2. chemical burns of the skin and respiratory tract
3. residual bronchitis
4. laryngitis, tracheitis, bronchopneumonia and pulmonary edema on autopsy; also kidney congestion and early hemorrhagic nephritis
5. respiratory irritation, hoarseness and tightness in the throat
6. productive cough, sometimes with blood-stained sputum
7. moist rales in the lungs
8. cyanosis, intense dyspnea
9. decreased maximum breathing capacity and diffusing capacity, hypoxemia
10. vomiting
11. severe gastritis
12. residual vision impairment, permanent eye lesions

In general, atmospheric concentrations were not determined.

Controlled experimental human exposure to 500-550ppm resulted in decreased blood pressure and increased non-protein nitrogen. There was no change in several other serum indicators.[2]

Hindfelt and Siesjo have studied the cerebral effects of acute ammonia intoxication in the rat. Their findings show no significant changes in the cerebral-spinal fluid pH, although lactate concentrations increase. Lactate also accumulates in the intracellular spaces, but a slight decrease in pH was noted. Energy balance in the brain tissue was not affected, despite decreased phosphocreatine and increased cytoplasmic NADH/NAD ratio. Intraperitoneal doses in these experiments were 0.56, 0.62 and 0.78 mmoles/100g; the ammonia compound used was ammonium acetate.[3]

Significant increase of potassium in the red blood cells of rats given 7.9 mmoles ammonium acetate/ intraperitoneally, was shown by [illegible] Francavilla.[4] No change was noted in sodium [illegible] potassium concentrations correlated with [illegible]

Silverman [illegible] exposed to 500 ppm [illegible] changes were noted; [illegible]

Irritation of [illegible][6]

Patty reports that [illegible]. Irritation increases with concentration; chemical burns may result from 3% or greater ammonia content.[7]

1. US Dept of HEW: Occupational Exposure to Ammonia, criteria for a recommended standard, 1974, pp. 26-33.
2. Schmidt and Vallencourt: Changes in Blood Following Exposure to Gaseous Ammonia, Science, 108:555-56, 1948.
3. Hindfelt and Siejo: Cerebral Effects of Acute Ammonia Intoxication, Scand. Jnl. Clin. and Lab. Invest., 28: 353-373.
4. Albano and Francavilla: Intracellular Potassium Concentration During Ammonia Intoxication, Gastroenterology, 61,893-97, 1971.
5. Silverman et al.: Physiological Response to Ammonia in Low Concentrations, Jnl. Indus. Hyg. and Tox., 31;74-78, 1949.
6. Criteria Document, pp. 33-37.
7. Patty: Industrial Hygiene and Toxicology, pp. 859-62.

Sampling and Analysis - Ammonia[1]

TLV = 25ppm

Sample Collection

type: breathing zone

apparatus: midget impinger containing 10 ml 0.1N H_2SO_4, calibrated personal sampling pump

flow rate: 1 lpm

sampling time: 5 min.

Sample Analysis

type: colorimetric

apparatus: wet chemistry, spectrophotometer, standard curve

procedure: 1. Nessler reagent is added to 25 ml of sample (additional adsorbing solution may be necessary to obtain required volume) [illegible] min., color intensity [illegible] spectrophotometrically at [illegible] nm and compare [illegible] standard curve.

Range and Sensitivity: not sufficient at low concentration.

Precision: at 5 ppm, [illegible]; at [illegible] ppm, [illegible].

Interferences: volatile amines, aldehydes, acetone, alcohols, ammonium salts and formaldehyde

Effects of Storage: ship in tightly closed containers; analyze within one week.

Advantages/Disadvantages: relatively inexpensive equipment.

ANTIMONY[1]

Autopsies following acute oval animal poisonings reveal fatty degeneration of the centrolobular regions of the liver, with injection at the periphery. Degenerative changes in the kidney are also apparent, but to a lesser degree than in the liver. Subcutaneous and intravenous administration produce similar effects.

The urine and blood of rabbits fed 15mg tartrate per kg contained increased non-protein nitrogen. Jaundice was also noticed; fatty degeneration of liver and parenchymal neurosis was found in some animals. Animals fed 2-6mg/kg had normal amounts of non-protein nitrogen.

Antimony trioxide and pentoxide were tolerated by rats in doses up to 4mg daily; the pentoxide was less toxic. Injury to the heart resulted to rats fed 1g/kg antimony metal, although normal growth was maintained. Two scientists report hemoporefic effects from antimony. The intraperitoneal LD of antimony metal is set at 10mg/kg.

Guinea pigs exposed to 45mg SbO_3/kg for two hours daily for seven weeks and later 3hr/day showed extensive interstitial pneumonitis. The dosage corresponded to 1.6mg retained per day. Rats exposed to SbO_3 for 14 months developed chronic lipoid pneumonia.

Antimony trisulfide dust exposure to rabbits 7hrs/day, 5da/wk for six weeks at a concentration of 3.07mg/m^3 produced venous congestion in the lungs and areas of focal hemorrhage. Inflammation resulted from exposures to 27.8mg/m^3. Dogs' lungs were not significantly affected by exposures of 5.32 and 5.55mg/m^3. Some animals exposed to antimony compounds developed flabby dilated myocardium tissue; microscopic evidence indicated parenchymatous degeneration. Humans given therapeutic antimony treatments have developed cardiac injury.

[illegible] exposed through inhalation.

In [illegible] experience, [illegible] workers in stereotype departments using lead and antimony plates experienced toxic systemic effects including abdominal cramps, diarrhea, vomiting, dizziness, tenderness and tingling of the nerves, severe headaches and prostration. Upper respiratory irritation and pneumonitis occurred among antimony smelters.

Thirty-five of 75 abrasive grinding wheel manufacturers using antimony displayed abnormal electroencephlograms; antimony concentrations in the plant varied from 0.5° to 5.5mg/m^3.

Antimony is generally recognized as a skin irritant, although few cases have been reported.

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1 - Browning, Ethel Toxicity of Industrial Metals, Butterworth, London. pp 25-37

[illegible]

TLV = 0.5 mg/m^3

Sample Collection

type: not stated

apparatus: filter paper, holder and calibrated sampling pump

flow rate: appropriate to equipment used

sampling time: not stated

Sample Analysis

type: colorimetric, spectrophotometric

apparatus: wet chemistry, colorimeter of spectrophotometer

procedure: trivalent antimony reacts with KI to form iodoantimonite,

Range and sensitivity: 5-500 ug/sample; 1.0 ug/ml can be determined.

Precision: ± 2% in 5-500 ug/sample range

Interferences: bismuth; thallium, tungsten, sulfite, and oxidents must be absent

Effect of storage: none stated

1 - AIHA Analytical Guide "Antimony".

ASBESTOS

The toxicology of asbestos is well documented in the literature. Although asbestosis was first described in the early nineteenth century, only recently has the magnitude of its toxic effects on workers been understood.

In 1917 Pancoast et al. reported roentonographic changes in workers exposed to asbestos. Ferruginous bodies were discovered in spectrum samples of asbestos workers in 1930[2]; much later, Selikoff and Hammond reviewed 1,975 New York City hospital autopsies to determine the extent of ferruginous body contamination of persons from the general population.[3] Nine hundred and forty two showed ferruginous bodies. Of the 129 males employed in shipyard or construction work, 70 percent of the tissue samples contained ferruginous bodies.

Epidemiological work shows that the effects of asbestos exposure are more wide ranging than asbestosis. Selikoff et al., Elmes and Simpson and Newhouse have demonstrated excess of lung cancers among asbestos insulation fabricators and installers.[4]

Broncogenic cancer, mesotheliomas of the pleura and peritoneum and digestive tract cancers are associated with asbestos exposure. Selikoff found excess deaths due to cancers of the lung, pleura perinoetm, stomach and colon and rectum in a cohort of New York - New Jersey asbestos workers; smoking histories were considered.[5] Asbestosis was also a cause of death.

Asbestos-related disease is not confined to the work place; Dr. Henry Anderson of the Mount Sinai School of Medicine showed X-ray evidence of asbestosis in 37% of a cohort of families of asbestos workers. Champion [illegible] of mesothelioma in a family member and another in a [illegible].

1. [illegible], "A [illegible] of [illegible] Inhalation on the Lungs, Trans. [illegible], 1917.

2. Lynch, K.M., and W.A. Smith, "Asbestos Bodies in Sputum and Lung", JAMA, 95: 659-61, 1930.

3. Selikoff, I.J., and C.E. Hammond, "Asbestos Bodies in New York Cit Population in two Periods of Time", Inter. Conf. of Pneumoconiosis, Johannesburg, pp. 40-57, 1969.

4. "Occupational Exposure to Asbestos, criteria for a recommended standard", US Dept. of HEW, 1972.

5. "First Annual Report, "Environmental Sciences Laboratory, Mt. Sinai School of Medicine of the Cit University of New York, 1974, p. 4.

6. Dr. Henry Anderson, personal communication.

7. Chambin, P., "Two Cases of Malignant Mesothelioma after Exposure to Asbestos", Am. Rev. of Resp. Dis., 103: 821-6, 1971.

Sampling and Analysis - Asbestos[1]

TLV = 2 fibers > 5 um/cc TWA; 10.0 um/cc peak

Sample Collection

type: breathing zone

apparatus: 37 mm Millipore filter mounted on open face holder;

calibrated sampling pump

flow rate: 1.0 lpm

sample time: 15 min to 8 hr.

Sample Analysis

type: microscopic

apparatus: phase contrast microscope, 400-450 x

procedure: count particles > 5 um in 20 randomly selected fields.

Range and Sensitivity: not stated

Precision: ± 10% of counts

Interferences: grains, [illegible] present in fields of 20 or more fibers.

Effect of Storage: count samples within two days.

[illegible]

1. "Occupational Exposure to Asbestos, criteria for a recommended standard", US Dept. of HEW, 1972.

BICHROMATES

Ammonium, potassium and sodium bichromates are irritants, sensitizers and contact allergens.[1] All are associated with pulmonary irritation and perforation of the nasal septum. Bronchogenic carcinoma is a serious hazard. "Chrome sores" may be formed on the skin.[2] The tolerance for ammonium bichromate is listed at 0.1 mg/m^3.[3]

1. Adams: Occupational Contact Dermatitis, pp. 20, 41.
2. Merck Index, 8th Ed., pp. 67, 258, 854, 958.
3. Condensed Chemical Dictionary, p. 51.

Sampling and Analysis - Bichromates [1]

TLV =

Sample Collection

type: not stated

apparatus: filter paper and holder; calibrated sampling pump.

flow rate: 1 cfm

sample time: not stated

Sample Analysis

type: spectral

apparatus: wet chemistry; UV spectrophotometer; standards

procedure: 1. sample is dissolved from filter and KI added as oxidizing agent
2. UV absorbance of I-KI complex is read at 289 or 352 mu
and compared with standards

No other pertinent information given.

(This method may be ineffective in determining potassium bichromate.)

1. AIHA Analytical Abstracts.

Toxicity

There has been no injury to the eye reported from local application. However, cases of poisoning from systemic absorption have occurred, sometimes resulting in death.[1] Boric acid is toxic by mouth in high doses.[2]

1. Grant, Toxicology of the Eye, p. 191.

2. Condensed Chemical Dictionary, p. 56.

Sampling and Analysis - Boric Acid

TLV =

No sampling and analysis method was found; titration techniques might be applied.

[illegible]

[illegible] one of the most important [illegible] contact allergens in [illegible] industry.[1] The soap is [illegible].[2]

The LD_{50} of metallic cobalt powder is 10-20mg/kg when given intraperitoneally; cobalt soap also has a high LD_{50}.[3] In animals, soluble salts injected into the trachea cause acute lung irritation, edema and hemorrhage. Human inhalation effects to cobalt are difficult to assess, as other metals are usually present.[4]

1. Adams: Occupational Contact Dermatitis, p. 20.
2. Welcher: Standard Methods of Chemical Analysis, p. 53, vol 2a.
3. Browning: Toxicity of Industrial Metals, p. 113.
4. Browning: pp. 117-18.

Sampling and Analysis - Cobalt Soap, as Co[1]

TLV:

Sample Collection

type: breathing zone

apparatus: 0.8 µm 35 mm cellulose membrane filter and holder, calibrated sampling pump

flow rate: not stated

sampling time: not stated

Sample Analysis

type: atomic absorption

apparatus: wet chemistry, AA spectrophotometer, standards

procedure: 1. samples are ashed with HNO_3 and solubilized in acid solution
2. samples and standards are aspirated into AA, flame fed by C_2H_2, analyzed at 2407Å

Range and Sensitivity: 0.15-8 µg/ml; 0.15 µg/ml.

Precision: 3% RSD (analytical)

Interferences: none

No other information given

1. NIOSH Manual of Analytical Methods.

Crystalline Silica[1]

Silica causes silicosis, a nodular pulmonary fibrosis. The progression of silicotic lesions is related to the degree and duration of exposure and the [illegible] [illegible] in [illegible].

Silica exposure occurs in mixed dust exposures, such as coal and iron mining, in addition to work with materials having high silica content, e.g. granite.

Fibrogenic response in other organs has been demonstrated in animals.

1. US Dept of HEW: Occupational Exposure to Crystalline Silica, criteria for a recommended standard, 1974.

Sampling and Analysis - Crystalline Silica[1]

TLV = 50$\mu g/m^3$, TWA

Sample Collection

type: breathing zone

apparatus: o.8µm 37mm PVC membrane filter and holder, 10mm nylon cyclone, calibrated sampling pump

flow rate: not stated

sampling time: sufficient to collect up to 5 mg total dust

Sample Analysis

type: X-ray diffraction

apparatus: wet chemistry, X-ray diffraction unit, fluorite standard, calibration curve

procedure: 1. ash the sample, prepare the residue for analyis as described

2. scan 2θ-range corresponding to d 4.5 to 2.3 Å , presence of crystalline forms determined by occurance of diffraction peaks

3. free silica to fluorite intensity ratios determined, mass of silica determined from calibration curve

Precision and Sensitivity: [illegible] $\mu g/cm^2$; [illegible] µg/species (total dust loading not to exceed [illegible] mg/cm^2)

Precision: ±5 µg

Interferences: [illegible] have similar diffraction peaks, removed by [illegible] secondary peak

2. [illegible] and feldspars, removed by analysis at sec-
ondary peak

3. [illegible]

4. X-ray fluorescence from iron in sample, reduce by using
diffracted beam monochromator

Effect of Storage: not stated

Advantages/Disadvantages: 1. sensitive, rapid analysis
2. limited sample size
3. high degree of technical skill needed.

1. US Dept. of HEW: Occupational Exposure to Crystalline Silica, criteria for a recommended standard, 1974

[illegible]

Gilsonite [illegible] (similar to asphalt): the dust is [illegible][1] [illegible] photosensitizer.[2,3]

1. Grant, Toxicology of the Eye, p. 522.
2. Condensed Chemical Dictionary, p. 416.
3. Merck Index, 8th ed., p. 1032.

Sampling and Analysis: Gilsonite

No method found in the literature.

[illegible] [illegible] of [illegible] lead exposure to humans is well documented in the literature. [illegible] absorption through ingestion can be clinical [illegible] lead poisoning, manifested by general weakness, anorexia, irritability, pains in the muscles and joints, tremors and encephalopathy.[1] In the past, cases of wrist-drop and foot-drop due to weakness of the extensor muscles, were often seen in occupational exposures; this is rarely seen today.[2]

Lead causes anemia by interference with heme synthesis; intestinal colic, nephropathy and arterio sclerosis are also attributed to lead absorption.[3]

Epidemiological studies of occupational exposure have examined the effects of lead on the circulatory system. Dingwall - Fordyce and Lane found excess deaths due to cerebrovascular diseases among workers in an accumulator factory. Lane found excess total deaths in a survey of a storage battery cohort; most of the excess was attributed to vascular lesions of the central nervous system.[5] A study by Cramer and Dahling failed to show increased evidence of hypertension in workers with high urinary corproporphin levels, when compared with other workers with lower levels in an accumulator factory.[6] A further discussion of epidemiological studies of lead exposure is found in the Criteria Document.[7]

Sub- clinical manifestations of lead exposure is also being studied. Activity of amino levu'inic acid dehydrogenase has been investigated by Hernberg and Nikkanen; they found inhibition of the enzyme among urban dwellers having blood leads of 5 to 30ug/100ml.[8] Workers without clinical manifestations of neurologic lesions were found to have a minimal loss of peripheral nerve function; the work, done by Catton et al, evaluated lead accumulator workers.[9]

Browning reports evidence that lead is absorbed by the fetus.[10] Canadian work shows a correlation between lead exposure and learning disabilities in children.

Several studies of print shops document the airborne concentrations of lead from typesetting and remelt operations; type material is 80-90% lead.[12, 13, 14]

1. Mayers, M.R., "Occupational Health - Hazards of the Work Environment", Williams and Wilkin's Co., Baltimore, 1969, pp. 69-70.
2. Browning, Ethel, "Toxicity of Industrial Metals", Buttersworth, London, 1961, pp. 153-157.
3. Browning, pp. 153-59.
4. Dingwell - Fordyce and R.E. Lane, "A Follow-up Study of Lead Workers", Brit. J. Indus. Med., 30: 313-15, 1963.
5. Lane, R.E., "Health Control in Inorganic Lead Industries - A Follow-up Study of Exposed Workers", Arch. Env. Hlth., 8: 243-50, 1964.
6. Cramier., Kim, and Lennart Dahlberg, "Incidence of Hypertension among Lead Workers, a Follow-up Study Based on Regular Control over Twenty Years", Brit. J. Indus. Med., 23: 101-104, 1966.
7. "Occupational Exposure to Inorganic Lead, criteria for a recommended standard", US Dept of HEW, 1972.
8. Hernberg, A. and J. Nikkanen, "Enzyme Inhibition by Lead Under Normal Urban Conditions", the Lancet, January 10, 1970, pp. 63-64.
9. Catton, M.J. et al., "Sub-clinical Neuropathy in Lead Workers", Brit. Med. Jnl, April 11, 1970, pp. 80-82.
10. Browning, pp. 152-3.
11. Parkinson, David.
12. Ruf, Harold W., "Studies of the Lead Hazards in Certain Phases of Printing", J. I. Indus. Hyg. and Tox., 22: 445-463, 1940.
13. [illegible], John [illegible] et al., "Lead in the Printing Industry", Jnl. Indus. Hyg. and Tox., 30: 643-45, [illegible].
14. [illegible] Alfred [illegible] and George [illegible] "[illegible] Exposures at the [illegible] Printing Office", Jnl. Indus. Hyg. and Tox., 25: 445-50, 1943.

Sampling and Analysis - Lead[1]

TLV (inorganic compounds, fumes, and dust) - .15 mg/M

Sample Collection

type - breathing zone

apparatus - 0.45 μ cellulose membrane filter, mounted in 2- or 3-piece filter cassettes; calibrated pump

flow rate - 2 lpm

sampling time - 50 min. or more

Sample Analysis

type - dithizone colorimetry

apparatus - wet chemistry; spectrophotometer

procedure - ashed filter is subjected to dithizone method of analysis

No other information given

1. Occupational Exposure to Inorganic Lead - Criteria for a Recommended Standard. U.S. Dept. of HEW, 1972.

Sampling and Analysis - Lead[2]

TLV -

Sample Collection

type -

apparatus - 0.8 or 0.45 μ cellulose membrane filter, mounted in 2-or 3-piece filter cassette; calibrated sampling pump

flow rate - 2 lpm

sampling time - 50 min. or more

Sample Analysis

type - atomic absorption

apparatus - wet chemistry, atomic absorption spectroscope unit with standard curve

procedure - sample is ashed, organic matrix destroyed and solubilized lead analyzed by AA at 2833 Å

Range

up to 40 μg/ml

Sensitivity

0.1 μg/ml

Precision

not completely determined

Interferences

none known

Effect of Storage

sample may be dislodged during shipping; prevent by placing clean filter on top of sample filter

Advantages/Disadvantages

1. simple and fast
2. inexpensive equipment

2. NIOSH Manual of Analytical Methods

Lead Soap

Lead soap is [illegible] and is [illegible].[1] It is absorbed through [illegible] in appreciable amounts.[2]

1. Merck Index, Op. Cit., p. 610.
2. Patty: Industrial Hygiene and Toxicology, p. 1[illegible]2.

Sampling and Analysis - Lead Soap

[illegible]

See Lead

[illegible]

[illegible]

[illegible] through the skin [illegible]

[illegible] exposure, but may develop suddenly. Headaches, psychomotor instability, speech difficulty and tremors of the limbs are symptoms characteristic of manganese poisoning. Conflicting evidence of blood changes due to manganese exist.[3]

1. Merck Index Eighth Edition pp.643
2. Browning: Toxicity of Industrial Metals PP. 186-7
3 as above pp. 190-94

[illegible]

[illegible] is reported.[2]

[illegible][3]

[illegible] liquid.[1]

1. Merck Index pp. 771-2

2. Gleason: Clinical Toxicology of Commercial Products pp.130-31

3. Grant: Toxicology of the Eye pp. 774

4. Patty: Industrial Hygiene and Toxicology pp. 1810

Sampling and Analysis - Oxalic Acid[1]

TLV= 200 ppm

Sample Collection

type: not stated

apparatus: glass washing bottle containing absorbing medium, calibrated sampling pump. No additional information given.

Sample Analysis

type: colorimetric

apparatus: wet chemistry

procedure: 1) dilute sample to 100ml. with CO_2-free H_2O, add 25 ml. 2N sulfuric acid, heat to 70 F.

2) Titrate with 0.1 N potassium permanganate until faint pink persists 1 min. (1 ml. 0.1N potassium permanganate= 0.006302 g dihydrate or 0.004501 anhydrous oxalic acid.)

No additional information given.

1. Jacobs, Poisons, Hazards, Solvents pp. 89, 504

[illegible]

Lac is the natural resin [illegible] solvents used for [illegible] methyl alcohol, [illegible] and turpentine. Inhalation of shellac is [illegible] these solvents. [illegible] the solvent is something as simple as methyl alcohol the effect is minimal, but when it is an oil, there is a greater risk due to the mixture of fatty acid compounds. The fatty acids are known to have deleterious effects on animal tissues.

A case study was done in 1932 on a 59-year-old man who owned a furniture factory. He originaly complained of chronic tiredness and weakness for years, increasing dyspnea for nine months and weight loss. As time passed, he was found to have diffuse lung density, inspiratory rales and enlargement of the heart, along with fever and increasingly severe dyspnea; he ultimately died. Upon autopsy, he was found to have had pneumonia, pleuritis, hyperplasia of the lymph nodes, nephrosclerosis and arteriosclerosis. The fatty acids in the shellac caused necrosis of the lung tissue.

Hirsch and Russell: Chronic Exudations And Indurative Pneumonia Due to Inhalation of Shellac.

[illegible]

[illegible] There is irritation of nasal tissues like sneezing, frequently.

Patty: Industrial Hygiene and Toxicology, p. 86[illegible].

Sampling and Analysis - Sodium Hydroxide [1]

TLV = 2 ml/m^3

Sample Collection

type: none stated

apparatus: bubbler containing standard sulfuric acid containing methyl red; calibrated sampling pump.

flow rate: none stated

sampling time: sufficient to color solution

Sample Analysis

type: colorimetric

apparatus: wet chemistry

procedure: calculate sodium hydroxide concentration in air needed to cause color change in volume sampled

No additional information given.

1. Patty: Industrial Hygiene and Toxicology, p. 867.

Sulfuric Acid

Sulfuric acid is corrosive to all body tissues. [illegible] with dilute concentrations results in dermatitis; necrosis of skin tissues is also possible.[1]

Eye splashes cause serious, often permanent, ocular damage. A fine spray in the air creates stinging and burning of the eyes, although the rapid dilution by tearing may prevent significant injury.[2] Sulfuric acid mist removes tooth enamel in three regions where contact occurs.[3]

Increased pulmonary resistance is a reflex action against inhalation of sulfuric acid. Inhalation of high concentrations produces sneezing and coughing; if pexposure is prolonged, bronchitic symptoms, rhinorrhea, lacrimation and epitaxis result. Some evidence supports the development of emphysema and digestive distrubances following long-term exposure.[4]

A study of sulfuric acid aerosols (mean particle size 1.0 µm) in concentrations of 0.35-5.0 mg/m^3 to humans showed respiratory responses at the lowest exposur, while subjective response of discomfort by all subjects occured at 3.0 µg/m^3. A further discussion of sulfuric acid is found in the Criteria Document.

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1. Merck Index, 8th ed., p. 1008.
2. Grant: Toxicology of the Eye, p. 959.
3. US Dept HEW: Occupational Exposure to Sulfuric Acid, criteria for a recommended standard, 1974, work reported pp. 20-21.
4. as above, pp. 23-24.
5. Amdur et al.: Inhalation of Sulfuric Acid Mist by Human Subjects, Arch Ind Hyg Occup Med, 6: 305-12, 1952.

Sampling and Analysis - Sulfuric Acid[1]

TLV = 1mg/m^3

Sample Collection

type: breathing zone

apparatus: 0.8 µm cellulose membrane filter and holder, calibrated sampling pump

[illegible]

[illegible]

[illegible]

[illegible]

Apparatus: [illegible]

procedure: 1. filter dampened with distilled water, pH adjusted [illegible] with perchloric acid, [illegible] isopropyl alcohol [illegible]

2. Titrate with 0.005 M perchlorate with thorin as indicator, endpoint is change from yellow to pink

Range and Sensitivity: at least 0.5 mg/sample; 0.1 mg/m^3 in a 100 l sample.

Precision: at 1 mg/m^3, 10 % with RSD of 4%; at 10 mg/m^3, 10% with RSD of [illegible].

Interferences: 1. soluble particulate sulfates

2. metal ion interference, eliminated by utilizing ion exchange resins

3. phosphate ions, if greater concentration than H_2SO_4, removed by precipitation with $MgCO_3$

4. sulfite ions, if greater concentration than H_2SO_4, removed by titration with standard iodine

Effect of Storage: ship so as not to damage samples

Advantages/Disadvantages: 1. sample collection is easy, stable, conveniently shipped

2. analysis rapid and simple

3. sulfate salts interfere

1. US Dept HEW: Occupational Exposure to Sulfuric Acid, criteria for a recommended standard, 1974

[illegible]

[illegible]

Lilis et al. report numbness, tingling, of the fingers, increased sensitivity to cold, pain in the joints of the fingers and hands. There have also been symptoms resembling Raynaud's syndrome. Several workers had suffered from gastritis and gastrointestinal bleeding.

Pulmonary function examinations seem to indicate that there is no injury from inhalation.[1]

On initial exposure, giddiness has been reported. After repeated exposure, headache, irritability, dominuition of memory, insomnia, general asthenia, tingling, weight loss and decrease of physical force are exhibited.[2]

It has been discovered recently that angiosarcoma of the liver, a very rare type of tumor, develops in relatively high frequency in workers exposed to gaseous vinyl chloride.[3]

1. Lilis et al.: Prevalence of Disease Among Vinyl Chloride and Polyvinyl Chloride Workers, Toxicity of Vinyl Chloride and Poly vinyl Chloride, NY Academy of Sciences.
2 Miller: Changes in Pulmonary Function in Workers Exposed to Vinyl Chloride and Polyvinyl Chloride, as above.
3. Popper and Thomas: Alteration of Liver and Spleen Among Workers Exposed to Vinyl Chloride, as above.

Sampling and Analysis - Vinyl Chloride[1]

TLV-1ppm

Sample Collection

type: breathing zone

apparatus: charcoal tube, calibrated sampling pump

flow rate: 50 ml/min

sampling time: 100 min (max)

Sample Analysis

type: GC

apparatus: [illegible] GC-FID [illegible]

[illegible] is desorbed with CS_2, [illegible] this, and [illegible]

[illegible]

[illegible]: 0.2-[illegible] [illegible]; [illegible]

[illegible] GC

Precision: unknown

Interferences: 1. water vapor

2. compounds with the same retention time

Effects of Storage: [illegible] on dry ice to prevent migration of VC during transit; freeze until analyzed in the laboratory.

Advantages/Disadvantages:
1. sampling unit is small
2. no liquids are necessary
3. overloading may occur
4. precision limited by the pressure drop which can be maintained.

1. NIOSH: Manual of Analytical Methods

Appendix 4

LISTING OF EMPLOYEE MASTER CARDS AS OF 6/16/75

S.#	LOCAL	EMPLOYEE NAME	SOC.SEC.	DATE OF BIRTH	DATE OF ENTRY	CLASS	G H I	H I P	EFF. DATE PENS	DATE RET.	D I S	DATE DEATH	OUTS	OUTS DATE	STA
09	0051	S SHERMAN	068077401	11992	110936	44				3/57					
03	0051	F ADAMSKY	103032627	80292	052347	44			269	11/68	R	10/69			
15	0051	I PERLCUTTER	100056040	41592	120144	44				7/65					
12	0051	A J PEREMES	066016094	91293	031750	44			570	5/70	R				
374	0051	L MELLIS	236141586	81593	081354	00	0						SU	5/57	
930	0051	J RYAN	103011098	12093	081056	22	0								
951	0051	P FERSTOT	130096241	90293	032657	00	0						SU	3/62	
758	0051	A PHILLIPS	131019831	72793	072242	08						12/67			
711	0051	WESSELIUS	083010682	92293	041441	44				10/62					
3638	0051	G LEEFIS	064163164	50593	020146	33							OU		
4064	0051	P SARANDIS	125035385	32593	032946	00									
2628	0051	C SOVINSKI	052038565	10693	052923	99				3/59		10/67			
2726	0051	D WEADE	086099816	90293	101226	44				10/58		9/65			
2051	0051	S BUSCH	083038256	40893	042025	99				4/59					
2536	0051	A PRONCHAK	113035585	121293	052025	99				8/59					
7862	0051	L J WEIDEMAN	092107371	111593	080036	08							SU	4/61	
5792	0051	DANIELSON	206103992	112793	122351	08						4/63			
02486	0051	I NYGREN	052096191	91393	052735	99				4/59		5/67			
02189	0051	ROSENBERG	092032371	110793	091635	44				8/64		10/72			
02027	0051	BILELIS	090094421	12193	111436	44				11/64		7/74			
02772	0051	CARP	090100789	122193	093035	44				1/59					
05812	0051	H GERTLER	102149334	101494	022454	00						7/70	SU	5/62	
05747	0051	A ZOLA	117129463	31494	033151	00							OU		
05775	0051	H LENSSENS	134247051	53194	052352	00							OU		
[illegible]	0051	D WEBER	116099829	40994	100256	22	0								
07007	0051	V THULIN	053149768	81994	040750	00	0						SU	5/60	
02930	0051	J R YOUNG	107075696	31794	110945	08						12/63			
02125	0051	C DENGES	090012431	123194	052246	00									
02217	0051	GONZALEZ	095165527	62494	062245	55							OU		
02390	0051	L C LALONDE	124103800	40294	010045	00	0						OU	10/57	
02488	0051	J O CONNELL	077076338	31794	052746	00	0						CC	10/60	
03173	0051	C HANSON	083102440	21794	092746	00	0						OU	6/67	
03266	0051	J HUTCHINGS	119105421	102094	071147	00									
03348	0051	L C SCHWEIZER	055018647	102494	020648	33							OU		
03354	0051	I WINOKUR	053013681	20194	022348	00									
03470	0051	C T FOTOPULOS	114036196	60394	060748	00									
	0051	L NACHTIGAL	066073832	52894	082048	00	0						SU	3/56	
			109091203	100494	031725	99				3/61		5/64			
				102994	081936	08									