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TESTIMONY

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BEFORE

THE SUBCOMMITTEE ON HEALTH AND SAFETY

OF THE HOUSE SUBCOMMITTEE ON EDUCATION AND LABOR

ON THE WORKER'S RIGHT TO KNOW

AND THE WITHDRAWAL OF OSHA'S PROPOSED

RULE ON HAZARDS IDENTIFICATION

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C O N T E N T S

INTRODUCTION

Mr. Chairman, my name is Richard Lewis. I am an occupational health specialist on the staff of the International Chemical Workers Union (ICWU).

First, let me say ICWU greatly appreciates the opportunity to talk to you about efforts to reverse the pattern of occupational illness and injury in American workplaces. Public health, working in cooperation with government, has a proud tradition in this country of eradicating widespread disease. The track record has been brilliant in the area of infectious disease such as polio and measles. Unfortunately, the same type of progress has not yet been made in the area of occupational injury and illness. Data from the Bureau of Labor Statistics "Occupational Illness and Injury in U.S. Industry," indicates a total of 36,178 lost workday cases resulting in over half a million lost workdays in the Chemical and Allied Products Industry alone.[1] I want to emphasize these numbers are only for one year. We are talking about 4 1/2 to 5 million lost workdays due to injury and illness in the last decade in the Chemical and Allied Products Industry alone. No attempt has been made to quantify the ripple effect as chemicals move from manufacturers to downstream users, but the National Occupational Hazards Survey (NOHS) indicated that potential hazards exist downstream as well.[2] This represents a monumental problem from a public health point of view and obviously a tremendous amount of pain and suffering for workers.

Fighting for the prevention of occupational injury and illness has been a long hard struggle for public health advocates and workers. In the last ten years, under both Democratic and Republican administrations, the federal government has initiated a variety of programs under the Occupational Safety and Health Act aimed at the prevention of occupational illness and injury. Some of the most important programs include health standards regulating carcinogens and access to medical records, the New Directions program, and a policy favoring engineering controls over personal protective equipment as a means of worker protection. These tools for health and safety protection are a desperately needed beginning.

ICWU believes that identification of chemicals and hazard notification is a crucial next step in the development of a comprehensive health and safety program. Unfortunately, chemicals continue to directly threaten the well-being of workers with discouraging regularity by exploding, igniting, producing acid and alkali burns, intoxicating and suffocating workers in enclosed spaces, and producing long term chronic hazards such as cancer.

The Right to Know for the worker boils down to "what is it?" and "what will it do to me?"

In presentation of this testimony, ICWU decided it would be useful to demonstrate that workers are unable to obtain the needed information to determine the identity and hazards of the substances they are exposed to each day. We have done this in Section I of our testimony which relies on examples from our files to demonstrate this inadequate flow of health

and safety information to workers. However, it should be noted that I will cite only a few examples. I can tell you that we do not have a single local in which workers are fully advised of the identity and hazards of their exposures.

Section II of the testimony will demonstrate that workers are motivated and able to work with health and safety information and data when they are able to obtain it through requests to the company, NIOSH, OSHA, or the National Labor Relations Act.

In Section III of the testimony we will specifically respond to the Chemical Manufacturers Association (CMA) testimony given to this Subcommittee on May 27, 1981, and point out where that testimony contradicts scientific principles of occupational safety and health and indicate our experience at the local plant level.

I will turn now to the first major section of our testimony which describes what kind of information the worker needs to know.

I. WHAT THE WORKER NEEDS TO KNOW

1. Chemical Identity

The two questions: "what is it?" and "what will it do to me?" are interdependent. If a worker, an industrial hygienist, or a doctor does not know the identity of a substance, there is hardly any way he or she can determine what health risk may result from exposure. A worker can not take the simple step of referring to a toxicology text which many ICWU

locals now have in their libraries. A doctor, when presented with an ill worker, can not properly diagnose or treat an illness unless knowledge of the illness causing agent is readily available. One certain way to make the information readily available is to indicate the chemical identity and hazard of the chemical on the label.

The ICWU health and safety staff receives "what is it?" requests from workers on a weekly basis. The following examples are excerpted from the full text of letters in the ICWU health and safety files.

> No one in the plant knows what it is. The customer sent everything pre-measured and in the code of 001-005. How do I go about getting chemicals in the product?

The local member in this example is referring to the names of various chemicals in the product. The worker needs these names to see if the product is hazardous so that safe work controls can be utilized.

> Four members have been complaining of a rash on their arms and bodies. One member has been getting sick while in contact with this dust. The odor of this machine smells like a dead animal.
>
> I have asked the company on May 8 for a material status and manufacture name, but to date haven't received anything.
>
> Any information as soon as possible will be appreciated. We are going to start the line up today; 5-19-81.

This is a case of workers experiencing potentially occupational induced symptoms, yet all they know about the exposure is that barrels are marked "150#."

Many labels presently in use do not identify the chemicals present in the product. For example the health and safety staff received a letter

requesting specific health information on paint. As you can see by looking at the label in Appendix A-1, the specific chemical ingredients of the paint are not given; instead, broad categories of chemicals are listed. The use of a broad category such as aliphatic solvent presents both a safety and a health hazard. A safety hazard is present in that workers are unable to determine the flash point and consequently prevent a potential explosion resulting from use in an enclosed space.

A health hazard is also present. A category or class of chemicals contains individual chemicals which range from very toxic to relatively nontoxic. A worker who only knows he or she is exposed to aliphatic or aromatic solvents can't tell if the exposure is to ethyl acetate which may cause some irritation to the nose and throat,[3] or to xylene which causes irritation but at higher levels of concentration has caused dizziness and even death.[4]

Please note Appendix A-2 for the list of some twenty chemicals used as solvents in paints. Use of some of these solvents require stringent ventilation controls, others do not. This paint label does little to provide the health and safety information needed for adequate protection when exposure to toxic ingredients may be present.

ICWU has recent examples in their health and safety files of cases where workers have been exposed to known or suspect carcinogens that they were unable to identify. 4,4'-Methylenebis (2-Chloroaniline), commonly referred to as MOCA, is a curing agent used in epoxy resin systems. ICWU workers were exposed to MOCA in a nozzle cleaning operation in a Gulf And Western plant.

The carcinogenicity of MOCA is well established. The American Conference of Government Industrial Hygienists stated the following in 1976.

> Based on animal experiment, Cl-MDA (MOCA) is suspected of carcinogenic potential in man; worker exposure by all routes should be carefully controlled.[5]

In 1978 NIOSH published a Special Hazard Review on MOCA which included this statement in its conclusion.

> Results reported by five independent groups of investigators clearly demonstrate 4-4' Methylenebis (2-Chloroanaline), to be oncogenic (tumor forming) in the rat, mouse, and dog. Ingestion of daily doses of 4-4' Methylenebis (2-Chloroanaline) by mice and rats have resulted in the appearance of cancer of the liver, kidney, lungs, skin, and mammary glands.[6]

Reports of cancer findings on MOCA are also documented in the 1975 edition of Occupational Medicine, a well respected text by Dr. Carl Zenz.

> Although industrial experience from reported studies is minimal, the positive findings in two animal studies by three independent investigators firmly implicate 4-4'-Methylenebis (2-Chloroanaline) as a human carcinogen.[7]

MOCA is readily absorbed through the skin; therefore prevention of skin contact exposure to MOCA is an essential industrial hygiene control measure. Workers would vigorously adhere to protective measures if they knew exposure to a carcinogen was involved.

In the early 1970's NIOSH estimated 55,000 U.S. workers were potentially exposed to MOCA.[8] OSHA attempted to regulate MOCA along with 13 other carcinogens in 1973, but the final MOCA standard was remanded for procedural reasons by the Court of Appeals, Third Circuit

(Synthetic Organic Chemical Manufacturers Association v. Brennan 506 506 F2d 385, 1974). However, various states such as California continued to enforce the standard under State law.

The experience at the ICWU local was that workers were not notified of their exposure to MOCA. The company denied that MOCA exposure existed in the plant, yet MOCA appeared in a urine sample of an exposed worker and in an OSHA industrial hygiene sample of the nozzle cleaning solution.

In this case exposure to a known carcinogen was taking place. The carcinogenic properties of MOCA have been described in a number of well accepted and widely used occupational health references. There is no excuse to keep workers in the dark about the presence of MOCA in the plant nor about MOCA being a suspect human carcinogen.

ICWU has embarked on a major hazard notification computer program with the cooperation of NIOSH. Ideally, the computer program would be used to immediately notify exposed locals as soon as the scientific hazard information becomes available. However, the system is dependent on proper identification of the materials used in plants. When ICWU locals request lists of chemicals used by a company, they often receive useless trade name information.

A very recent ICWU example of exposure to acrylonitrile demonstrates that the use of trade names constitutes a road block to the meaningful flow of health and safety information to workers. Acrylonitrile is a metabolic asphyxiant with an action similar to cyanide,[9] but perhaps more serious because more frequent is the epidemiological evidence that shows that low level exposures may be associated with an excess of lung and colon cancer in workers.[10]

Workers at this local were only informed of the cancer causing properties of acrylonitrile when the local president had occasion to read an ICWU hazard alert during a labor management meeting. The alert included a list of acrylonitrile trade names, one of which a worker recognized as being present on a label in the plant. The ICWU hazard alert came out in 1981. A NIOSH Current Intelligence Bulletin published information on excess human cancer rates in acrylonitrile workers in 1977.[11]

The example of employees being exposed to trade name products containing carcinogens is fairly widespread according to the NOHS survey undertaken by NIOSH in 1972. The survey found that, of workers using trade name products (TRN) in the course of their employment, some 2% (5,638 workers) were potentially exposed to one of the fifteen chemicals that OSHA regulated as carcinogens.[12] OSHA has regulated only a small fraction of the chemicals currently documented as having substantial evidence of carcinogenicity;[13] so the 2% figure is below the actual figure for workers potentially exposed to trade name products containing carcinogens.

The previous set of examples demonstrate that workers exposed to hazardous substances are often unable to answer the basic question, "what is it?"

2. Hazard Notification

Once the identity of the chemical can be established the worker is still not in a position to assess the health and safety implications of a situation. To determine what the hazards are the worker must now turn to his or her own occupational health information resources, the union

health and safety department if the workers belong to a union, or the company.

The following are examples of information requests from locals where the chemical identity was known but hazard information was not known. Examples are excerpted from the full text of letters appearing in the ICWU health and safety files.

Workers often ask questions about specific control methods for hazardous chemicals.

> When there is suspect exposure above TLV should there be follow examination by the medical department?
>
> Question #4 - Has Thiotepa been determined to cause cancer in animals?
>
> Question #5 - What precautions has the company taken to protect its employees?

The now withdrawn OSHA labeling standard offered a reasonable and scientifically valid approach to the hazard determination process. The process attempted to set objective scientific criteria for hazard determination which are commonly used in the field of public health. Such a hazard determination process would insure a comprehensive set of hazard evaluations would be passed on to workers. A performance oriented approach, such as the one suggested by the CMA Guidelines in Exhibit 1 of their May 27 testimony, could result in as many different evaluation results as there are manufacturers to do the evaluating.

ICWU workers are being provided with inadequate hazard information by their employers. ICWU workers were notified by Lederle Laboratories of American Cyanamid in early 1978 that it was unsafe for women of child

bearing age to be exposed to two drugs that were being manufactured in the plant: methotrexate and diamox. The company instituted a policy barring women of "childbearing potential" from certain sections of the plant. In December of 1978 the company communicated to the union that they had determined a safe level of exposure for women of "childbearing potential" to methotrexate and that women would no longer be restricted from diamox production. The Physician's Desk Reference[14] a text used by doctors in prescribing drugs, links methotrexate with defective spermatogenesis a reproductive disorder in males, [redacted]. This information was not communicated by American Cyanamid to their workers prior to the Union's involvement.

A more widespread example of inadequate hazard evaluation and notification pertains to the chemical Toulene - 2,4-Diisocyanate. A NIOSH study conducted in 1972-1974 estimated that 50,000-100,000 U.S. workers were potentially exposed to Diisocyanates.[15] TDI exposures result in a number of adverse health effects one of which, sensitization, is particularly disabling. Sensitization to TDI denotes the tendency of some individuals to suffer a progressively more severe respiratory reaction when exposed to very low levels.

> Although the acute effects may be severe, their importance is overshadowed by respiratory sensitization in susceptible individuals; this has occurred after repeated exposure to levels of TDI as low as 0.02 ppm and below. The onset of symptoms of sensitization may be insidious, becoming progressively more pronounced with continued exposure over a period of days to months; initial symptoms are often nocturnal dyspnea and/or nocturnal cough with progression to asthmatic bronchitis. Often when the respiratory illness has become incapacitating and

> the worker has been hospitalized or otherwise removed from the worksite, a return to work causes an acute and severe asthmatic attack almost immediately or within a few hours.[16]

A sensitized worker may never be able to return to his or her workplace or any other workplace where TDI is present. Although the sensitization effects of TDI are documented in medical texts,[17] studies published in the American Industrial Hygiene Journal,[18] and NIOSH documents, sensitization warnings do not presently appear on labels of TDI used in a foaming operation of an ICWU shop. Because TDI can cause serious damage at extremely low levels it is important that yet unsensitized workers be aware of potential hazardous exposures so to avoid sensitization. More dramatically it is critical that sensitized workers are properly informed of TDI and its hazards so they can avoid debilitating acute respiratory reactions.

In summary, these previous examples demonstrate that workers are unable to obtain important information about what they are working with and what it can do to them. Examples are cited of how identification of trade name is not a useful health and safety practice and how a performance oriented hazard evaluation scheme fails to supply meaningful and accurate health and safety information. These examples stress that workers need both chemical identity and hazard information to protect themselves. ICWU wishes to emphasize that what public health advocates and workers want from a Right to Know standard is that labels and material safety data sheets reflect available scientific information. It makes little sense to ICWU to fund projects on the hazards of chemicals, e.g., the National Cancer Institute

Genetox study, and then fail to inform those who must work with these chemicals of findings of adverse effects.

II. USE OF RIGHT TO KNOW INFORMATION

There are several disciplines of public health professionals including occupational physicians, industrial hygienists, and epidemiologists* who would be better able to practice if hazardous workplace chemicals were properly identified. This testimony has mentioned previously the difficulties that the ICWU health and safety staff has in carrying out hazard alert notification when we cannot determine what exposures workers are experiencing. I am sure this committee will hear the testimony of public health scientists outlining the importance of obtaining exposure information, but we are here today to speak mainly for workers' need of Right to Know.

Although workers in ICWU locals receive professional support from the International, we serve 60,000 workers and have a limited health and safety staff. No union in America can fully service the health and safety needs of all its members. Much of the research must be done by members at the local level. ICWU has trained some 800 local leaders in hazard recognition and other health and safety practices over the last two years. Proper identification of chemicals would greatly enhance the ability of these workers to fully put this training to use. A Right to Know standard

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* Epidemiology is the science of examining the occurrence of disease in populations.

is even more essential to the approximately 75 million unorganized workers in this country who do not have a professional health and safety staff to turn to for assistance.

ICWU experience has shown that local members are highly motivated and able to collect occupational health information on the problems that occur in their shops as the following examples demonstrate.

> The reason for this letter is to open an avenue for questions concerning the health and well being of our people. Being new at this position, I am quite ignorant of the short and long term effects of many chemicals we work with regularly and am in need of any material available on the subject.
>
> Two chemicals I particularly want information on are Phosphorous Oxychloride and Ethylene Dichloride (EDC). I understand that there has been a change in status on EDC and would appreciate any update on this. Also, I have been personally affected by Phos. Oxy. in the past, yet in the few books I have on chemicals, I can find no reference to effects of this chemical nor any threshold limits. In short, just how harmful are these two.

> Last July I asked (name deleted) for some information on the Thiotepa that is manufactured in Department #721. At that time I asked (name deleted) how long were they going to run Thiotepa? He was not sure and would get back to me. Can you answer that question. How long does Thiotepa run take and how many times a year do we run Thiotepa?
>
> Question #2 - Are you monitoring for exposure levels? What are those levels?
>
> Question #3 - Does Thiotepa cause any toxic effects on the body? If it does what are they?
>
> Question #4 - Has Thiotepa been determined to cause cancer in animals?
>
> Question #5 - What precautions has the company taken to protect its employees?
>
> I would appreciate your answer as soon as possible. When we receive your answers we may at that time have additional question to ask.

In a few cases ICWU locals have gone several steps beyond basic hazard identification and carried out crude epidemiological surveys. For example, members of one local did the initial data collection on the association between exposure to the pesticide oryzalin and birth defects in exposed workers. This information sparked the interest of the New York State Health Department, NIOSH, and OSHA, who are still involved in the process of investigations.

Members of another local carried out a crude epidemiological survey. Eventually, with the help of a local independent occupational health expert from the Philadelphia Area Project on Occupational Safety and Health, the local determined that exposure to fluoride dust could be associated with the occurrence of illness in the plant.

> ...First of all I went through the pink slips, I placed them on the jobs, how long they worked in the drying room, when they got out and what reasons they got out for. Some of the employees I talked to had holes in their septum, bleeding noses, eye irritations, sore noses and many other symptoms. I took a few symptoms I know of to start with and left each employee as I talked to him, tell me how they felt when they worked in the drying room. This way if something was mentioned that another one had said to me I asked him if he felt this symptom and most of them added more to it. Starting out with five symptoms, I ended up with 17 different symptoms from perforated septums to one employee who had no problems but only worked in the area a short period of time.

Although the determination, persistance, and assessment skills of these workers prevailed in the above examples, we don't see any reason why there should be so many obstacles and barriers between workers and health hazard information.

Most manufacturers know the health hazard information on the chemicals they produce and they should be made to share such information. Health

hazard data must not be treated as trade secret information. Union health and safety professionals and workers should not have to ferret out health hazard information that is already known.

III. COMMENTS ON CMA TESTIMONY

The ICWU health and safety staff has had the opportunity to read the testimony offered by representatives of the Chemical Manufacturers Association to this Subcommittee. We will comment on that testimony at this time.

1. Chemical Industry Safety Record

The CMA testified that the chemical industry is the second safest industry in America according to National Safety Council Data.[19] This relative ranking should not obscure the magnitude of the health and safety problem in the chemical industry as documented by the Bureau of Labor Statistics.

The record of health and safety in the chemical industry clearly demonstrates that worker health is endangered. According to Bureau of Labor Statistics the rate of workplace injuries and illnesses in the Chemical and Allied Products Industry was 7.8 per 100 workers in 1978.[20] This figure represents a decrease of reported injuries and illnesses over the previous six years. The category of lost workday cases represents a subset of illnesses and injuries that are more serious than the previous category. The 1978 rate was 3.3 lost workday cases per 100 workers.[21] This rate has increased over the past six years. Although the rate of injury and illness is decreasing slightly, the rate for the more serious injury and illness category is increasing slightly.

ICWU suspects that the lost workday cases reflect the more chronic illness and injuries in the workplace. A Right to Know standard must deal with chronic hazards to be effective in reducing workplace disease. As mentioned earlier in this testimony, over one-half million workdays were lost in the Chemical and Allied Products Industry due to occupational injury and illness in 1978 alone.

These statistics in their present form show that there are serious health and safety problems in the chemical industry. However, there is reason to believe that these statistics are underestimates due to the inability of the reporting service to continuously pick up workers who have left work due to chronic illness and have not returned. There is also reason to believe that under-reporting of occupational illness does exist as a result of a lack of recognition of occupationally-related illness by doctors and members of society in general.[22]

An Interim Report To Congress On Occupational Diseases published in 1980 cited data indicating the magnitude of the lack of recognition of occupational disease.

> This survey suggests that of 1.8 million disability awards under state workers' compensation programs in 1978, approximately 30,000 or only 1.7 percent were for occupational diseases.[23]

We would like to call to the attention of the Subcommittee a recently released report entitled Occupational Safety and Health in the Chemical Industry issued by the Council on Economic Priorities (CEP). The report concluded that the severity of OSHA violations in the chemical industry "exceeds all other U.S. industries except mining."[24] The Council on Economic Priorities used records of OSHA inspections, citations, and

violations to measure the safety of industries. Evaluation of health and safety is a difficult exercise but the study clearly addresses health hazards in the industry.

2. Overlabeling

As we have demonstrated with numerous examples, proper chemical identification and hazard notification is far from the rule in ICWU shops. Our experience has shown us that many employers do not provide this information nor do they show respect for the worker's desire to obtain information on his or her daily exposures.

The CMA testimony contained references to their concern for "overlabeling" of workplace substances.

> Excessive, unwarranted hazard warnings would dull the awareness of workers for hazard communications that are soundly based.[25]

ICWU disagrees with this assessment and is confused as to why the CMA is speaking for workers in this instance. Workers are more concerned about benzene and other neurotoxic solvents dulling their senses than hazard warnings dulling their senses.

As to the technical complexity of the withdrawn standard, ICWU appreciates that some scientific information is difficult to understand. In addition, ICWU appreciates that large numbers of substances may require labeling as a result of a scientific evaluation process. However, public health and medical scientists have a long tradition of collecting, analyzing, and interpreting health hazard information and they have arrived at a workable consensus on the scientific principles that govern the process. Neither complexity nor the frequency of potential labeling justifies ignoring any valid scientific information.

3. The ANSI Standard

In regard to the CMA claim that the American National Standards Institute Standard for Precautionary Labeling of Hazardous Industrial Chemicals (hereinafter ANSI Standard) has been used effectively by the industry,[26] we disagree. The ANSI Standard is woefully inefficient in its approach to chronic health hazards as noted by the Committee on Government Operations 1976 report.

> Voluntary labeling guidelines developed by the chemical manufacturing industry are directed primarily to the avoidance of injury from single, accidental exposure and do not address the health hazards caused by chronic low-level exposure to toxic chemical substances.[27]

Moreover, the NOHS survey clearly suggests that the industry is not following its own recommendations in notifying downstream users of hazardous chemicals.[28]

4. General vs. Workplace Specific Hazard Determinations

CMA makes the argument that some chemicals with known hazardous effects need not be labeled as hazardous because actual foreseeable plant handling and use present no adverse health risk.[29] Our experience in industrial hygiene suggests this is not the prudent course. For example, it is expected that a worker running a chemical process through an enclosed system will not receive exposure. However, what if the system develops a slow leak or some other emergency arises? This is a situation in which hazard identification is needed. The normal foreseeable process sometimes breaks down. That is what accidents and emergencies are all about. Accidents involving toxic chemicals are life threatening and we believe workers should have the information to be prepared in the case of an accident.

We know from practical experience that some workers tend not to push for ventilation or equipment repair or even report malfunctioning equipment

because of job production pressures. All exposed workers should be aware of the potential consequences of such neglect.

The ICWU health and safety staff also rejects any contention that some hazardous substances should not be labeled as hazardous because they appear in a nonhazardous form. Public health protection calls for the labeling of any hazardous substance in all its forms, because physical states of substances often change intentionally or unintentionally in chemical production and use. An illustration of this change of physical state is the case of a hazardous powder that is shipped in a block form and therefore assumed to be safe. However, the substance in the block form may be worn down at the edges to powder by handling or may be crushed in a later manufacturing state.

A chemical producer does not and cannot know all of the downstream uses of a product, therefore, the prudent public health course is to label a known hazardous substance in all its forms so workers in all plants and in all manufacturing processes will be informed of potential hazards.

5. Are Chronic Hazard Determinations Beyond The Present State of Science?

CMA contends that OSHA's now withdrawn hazard identification scheme is beyond the present state of science[30] and outside of the area of scientific consensus.[31] CMA cited the inability of the present state of science to develop an objective definition of a reproductive hazard.[32] However, epidemiologists and clinicians routinely conduct studies and publish conclusions that determine the reproductive hazard of a substance. Modern occupational medicine texts contain such information.

For example, 1,2 Dibromo-3-chloropropane (DBCP) is described as follows in the Proctor and Hughes text.

> DBCP causes sterility in male workers and cancer in mice and rats... DBCP has caused oligospermia (scarcity of spermatozoa in semen) and aspermia (nonemission of semen) in male workers.[33]

Anesthetic gases such as halothane and ethrane which are used to put people to sleep in hospital operating rooms have been shown to be a reproductive hazard in workers and experimental animals. A 1973 study of female nurse-anesthetists in Michigan found a three fold excess of birth defects in mothers who continued to work in operating rooms during pregnancy compared to those who left the operating room during that period.[34] A NIOSH study of operating room personnel revealed an increased risk of miscarriage for women employed in operating rooms compared to non-operating room employees.[35] Results from animal studies indicate the reproductive hazards of halothane.

> Basford and Fink (1968) reported that when rats were exposed to 0.8 percent halothane for 24 hours on day 9 a significant increase in defects of the ribs and vertebrae were found in the offspring as compared to controls exposed to air and also starved during day 9.[36]

This is the sort of "objectively ascertainable end results in humans or other mammals"[37] information that the withdrawn standard called for to undergo examination by the criteria for a reproductive hazard. Since physicians routinely treat patients for chemically induced reproductive disorders and scientists are studying reproductive disorders, why can't workers be informed of their scientific conclusions?

6. Mislabeling

CMA also makes various claims that the evaluation scheme under the now withdrawn standard would have lead to mislabeling and labeling substances as carcinogenic based on "allegations" of carcinogenicity. These claims cannot be supported.

For example there is no support for the following claim.

> These statements from OSHA lead to the conclusion that, under the now-withdrawn proposal, almost any allegation of carcinogenicity for a substance would have required its being labeled as a carcinogen.[38]

Under the withdrawn standard, the determination of a health hazard can only be made through a series of evaluative steps common in scientific practice. None of these steps give any credibility to "allegations." These steps are published in Appendix B of the withdrawn standard on pages 4450 and 4451 of the January 16, 1981 Federal Register. These evaluative steps acknowledge the scientific credibility of well controlled scientific studies, statistically significant conclusions, case reports, expert opinion, and peer review journals. The withdrawn standard actually states on page 4434 that information of any lesser quality will not be utilized in hazard evaluation.

> The evaluation procedure does not consider anecdotal material because the Agency does not believe that such "data" warrant a positive hazard determination under this standard.[39] (Emphasis added)

A second example of an incorrect CMA claim concerns the following CMA statement:

> OSHA's specification proposal for determination of chronic hazards would have resulted in much mislabeling. For example, OSHA's proposal would have required inappropriate labeling as carcinogens of materials such as acetic acid (vinegar); boric acid (eyewash); fructose, lactose and maltose (natural sugars); and sodium chloride (table salt). These materials are so designated in the NIOSH Registry of Toxic Effects of Chemical Substances. (RTECS.)[40]

It is true that NIOSH RTECS must be consulted in the process of the required literature search as stated in Appendix A of the withdrawn standard. However, it is not true that the evaluator is bound to accept a

hazard description because it appears in the NIOSH RTECS. The hazard determination scheme for any health hazard, including cancer, is laid out in a series of evaluative steps in Appendix B on pages 4450 and 4451 as previously noted.

RTECS will only serve as a starting point to inform the hazard evaluator what studies must be consulted. RTECS is listed under the category "Materials To Be Reviewed." Once these materials are identified the evaluator must follow the scheme set out in Appendix B, a scheme which lists scientific criteria and does not necessarily accept judgemental statements that appear in studies in RTECS.

In review, chemical identification and hazard notification can be derived from sound industrial medical science. We reject the notion that such utilization of scientific information is beyond the present state of science. The ICWU health and safety staff confers with scientists and physicians on a weekly basis who use such terminology as reproductive hazard and carcinogen as the tools of their trade.

7. OSHA Rulemaking Proceedings

ICWU is concerned about future OSHA proceedings on the Right to Know. Dr. Smith of the CMA expressed a preference for OSHA to pursue "open, smaller, less-formal meetings,"[41] as opposed to formal OSHA public hearings mandated by the Administrative Procedures Act (APA). ICWU is not sure what an "open, smaller, less formal meeting" is, but we emphatically encourage OSHA to utilize APA rulemaking procedures if they are interested in developing a rulemaking record that reflects the interests of all affected parties.

CONCLUSION

We hope this information will be useful to the Subcommittee in evaluating the Right to Know. Basically we are here tody to state that workers should have access to health effects information that has been generated by various fields of public health science.

In closing, I would like to recount to you a set of events that transpired last summer in an ICWU local shop of a Conoco plant in Baltimore. The scientific and political arguments about Right to Know should never be viewed outside the context of the day to day health and safety danger that workers face. I opened this testimony by quoting a set of cold and impersonal government statistics, but will close with a different view of the problem.

Mark Edmunds, an employee of the Baltimore Conoco plant, is a member of ICWU. Early last summer Edmunds was assigned to clean a chemical tank that contained benzene residue. He refused the job because he believed it to be too dangerous.

On the morning of August 6, 1980, four day workers were hired to clean the chemical tank that contained the benzene residue. After a few hours work the men were leaving the tank for a second break from the harsh fumes. At this moment one worker, Linwood Bryant, age 40, fell unconscious to the floor of the chemical tank. Bryant was taken from the tank. Conoco lab technicians attempted to revive him. Linwood Bryant was pronounced dead at 2:45 p.m. that same afternoon. An autopsy confirmed that Bryant died of acute benzene poisoning.

I hope this one small example[42] demonstrates the seriousness of risks that workers are unknowingly exposed to in American workplaces.

I would be happy to answer any questions at this time.

REFERENCES

1. Occupational Injuries and Illnesses in the United States by Industry, 1978, U.S. Department of Labor, Bureau of Labor Statistics, Washington, August 1980, p. 29. (Hereinafter cited as 1978 BLS Statistics.)

2. "Hazards Identification; Notice of Proposed Rulemaking and Public Hearings," Occupational Safety and Health Administration, Federal Register, January 16, 1981, p. 4415. (Hereinafter cited as OSHA Proposed Hazards Identification Rule.)

3. N. H. Proctor and U. P. Hughes, Chemical Hazards of the Workplace, J. B. Lippincott Company, Philadelphia, 1978, p. 247. (Hereinafter cited as Proctor and Hughes.)

4. Proctor and Hughes, supra, p. 509.

5. A.C.G.I.H.: 4,4'-Methylene bis (2-chloroaniline). TLVs Threshold Limit Values for Chemical Substances and Physical Agents in the Workroom Environment with Intended Changes for 1976. p. 38. Cincinnati, 1976.

6. 4,4'-Methylene bis (2-Chloraniline) Special Hazard Review with Control Recommendations, NIOSH, 1978, p. 11. (Hereinafter cited as NIOSH MOCA Review)

7. C. Zenz, Occupational Medicine, Year Book Medical Publishers, Inc., 1975, p. 862. (Hereinafter cited as Occupational Medicine by Zenz.)

8. NIOSH MOCA Review, supra, p. 2.

9. Proctor and Hughes, p. 90.

10. Current Intelligence Bulletin 18: Acrylonitrile, NIOSH, 1977, p. 1.

11. Current Intelligence Bulletin 18: Acrylonitrile, NIOSH, 1977, p. 1.

12. OSHA Proposed Hazards Identification Rule, supra, p. 4416.

13. See Appendix B for EPA Carcinogen Assessment Group List of Chemicals Having Substantial Evidence of Carcinogenicity.

14. Physicians' Desk Reference, Medical Economics Company, Oradell, N.J., 1980, p. 984. See Appendix C.

15. Occupational Exposure To Diisocyanates, NIOSH, 1978, p. 18.

16. Proctor and Hughes, supra, p. 483.

17. Proctor and Hughes and Zenz Occupational Medicine, supra.

18. Elkins, H.B. et al: Massachusetts experience with toluene di-isocyanate. Am. Ind. Hyg. Assoc. J. 23:265, 1962. Hygienic Guide Series: Toluene diisocyanate. Am. Ind. Hyg. Assoc. J. 28:90, 1967.

19. "Statement of the Chemical Manufacturers Association for the Health and Safety Subcommittee of the House Education and Labor Committee Hearing on the Withdrawal of the Department of Labor's Proposed Standard on Hazards Identification," May 27, 1981, p. 3. (Hereinafter cited as CMA Statement.)

20. 1978 BLS Statistics, supra, p. 29.

21. 1978 BLS Statistics, supra, p. 29.

22. OSHA Proposed Hazards Identification Rule, supra, p. 4413, (statement of Dr. Thomas Mancuso).

23. Analysis done by ASPER, derivent from Hunt, A. Estimates of Compensated Occupational Disease Cases by Disease Type and Level of Disability, 1975 (unpublished) October 1977.

24. Occupational Safety and Health in the Chemical Industry, Council on Economic Priorities, New York City, 1981, p. 8.

    "Chemical companies are among the most hazardous; the severity of their violations exceeded those of all other U.S. industries with the exception of mining. In 1977 while all U.S. industry had 2.7 violations per inspection, the chemical industry had 7.7."

25. CMA Statement, supra, p. 7.

26. CMA Statement, supra, p. 2.

27. OSHA Proposed Hazards Identification Rule, supra, p. 4414.

28. OSHA Proposed Hazards Identification Rule, supra, p. 4415.

29. CMA Statement, supra, p. 5.

30. CMA Statement, supra, p. 6.

31. CMA Statement, supra, p. 5.

32. CMA Statement, supra, p. 6.

33. Proctor and Hughes, supra, p. 202.

34. Corbett, T.H. et al. "Incidence of cancer among Michigan nurse anesthetists." Anesthesiology 38: 260-263, 1973.

35. Ad Hoc Committee on the Effect of Trace Anesthetics, American Society of Anesthesiologists, Occupational Disease Among Operating Room Personnel, Anesthesiology, Vol. 41, October 1974, p. 321-340.

36. Basford, A.B. and Fink, B.R.: The teratogenicity of halothane in the rat. Anesthesiology 29: 1167-1173, 1968, from Catalog of Teratogenic Agents, T. Shepard, The Johns Hopkins University Press, Baltimore, 1980, p. 515.

37. OSHA Proposed Hazards Identification Rule, supra, p. 4429.

38. CMA Statement, supra, p. 12.

39. OSHA Proposed Hazards Identification Rule, supra, p. 4434.

40. "Testimony By Dr. Curtis Smith On Behalf of the Chemical Manufacturers Association Before The Health and Safety Subcommittee On Education and Labor Concerning Hazards Communications," May 27, 1981, p. 4. (Hereinafter cited as Statement by Dr. Smith of CMA.)

41. Statement by Dr. Smith of CMA, supra, p. 7.

42. See Appendix D. for newspaper account and pictures.

SILVER-BRITE® ALUMINUM PAINT — B59 S 11

SILVER-BRITE Aluminum Paint, B59 S 11, is a ready mixed, general purpose aluminum paint for use on exterior and interior metal surfaces as well as masonry. SILVER-BRITE Aluminum Paint can be applied by brush, roller or spray; however, roller application produces the best results.

SURFACE PREPARATION:
Surfaces to be painted must be clean, dry and free of oil, grease, rust and foreign material. Prime exposed metal surfaces as soon as possible after cleaning.

PRIMERS:
Iron and Steel: Under 200°F (93° C) KEM KROMIK METAL PRIMER, B50 N 2.

200 to 400°F (93 to 204° C), apply directly to Near-White Blast cleaned surface, SSPC-SP10-63T.

Galvanized Iron: Galvanized Iron Primer, B50 A 1.

Interior Masonry: Alkyd Wall Primer.

Exterior Masonry (Concrete Block): block filler.

Insulated Pipes & Duct Work: Latex Wall Primer.

Previously Painted: Spot prime with the appropriate primer and follow with one or two coats of SILVER-BRITE Aluminum Paint.

Application: Always apply to a cool surface. Do not shake with mechanical shaker or overly agitate. Follow recommended spreading rate closely. Allow first coat to dry overnight before applying second coat.

CHARACTERISTICS

REDUCTION:	
Brush or Roller:	Full body.
Conventional Spray:	Full body. May be reduced with up to 1 pint (.473 l) of Mineral Spirits, if needed. DeVilbiss JGA Gun, 78 or 704 Cap, FF Tip, Binks 18 or 19 Gun, 66 or 67 Cap, S Nozzle, 50 psi atomization pressure, 20 psi fluid pressure or equivalent equipment.
Airless Spray:	RUBBERSET® FLOMATIC® G-10 Gun, Graco "Golden" Gun, .010-.012 orifice, 2,000 psi fluid pressure or equivalent equipment.
Drying Time @ 77°F (25° C) & 50% RH:	To touch: 1-4 hours To Recoat: 24 hours
Gloss:	Bright Aluminum
Coverage:	690 sq. ft. per gallon (17 sq. m per l) @ 2.3 mils wet, 1.0 mil dry.
Heat Resistance:	Up to 400°F (204° C).

4/80

CAUTIONS
Contents are COMBUSTIBLE. Keep away from heat and open flame.
USE ONLY WITH ADEQUATE VENTILATION. Avoid breathing vapor and spray mist. Avoid contact with skin and eyes. Wash hands after using. Keep container closed when not in use. Do not transfer contents to other containers for storage. 0001 (9-7

DO NOT TAKE INTERNALLY
KEEP OUT OF THE REACH OF CHILDREN

PROFESSIONAL COATINGS
SILVER-BRITE
ALUMINUM PAINT

Pigment by Weight		18*
Aluminum Paste Type 2 Class B	18%	
Vehicle by Weight		82*
Petroleum Resin	19%	
Polymerized Linseed Oil	21%	
Aliphatic Solvent	38%	
Aromatic Solvent	3%	
Additives	1%	
	100%	100*

G—E 78 0001 (9-74

B59 S 11
NON-PHOTOCHEMICALLY REACTIVE

SHERWIN WILLIAMS
The Sherwin-Williams Company
Cleveland, Ohio 44101

Table 4. Estimated consumption of important solvents in paints and coatings.[4]

Pigments	Millions of pounds				
	1973	1974	1975	1976	1977
Aliphatic hydrocarbons	1603	1375	1280	1185	1120
Methyl ethyl ketone	380	342	319	345	357
Xylenes	544	435	363	325	300
Other aromatics	421	399	326	305	290
Toluene	326	250	268	260	255
Ethanol	200	210	200	200	200
Acetone	200	184	175	190	196
Glycol ethers and ether esters	180	194	176	190	195
n-Butanol	140	155	135	145	151
Isopropanol	121	125	115	120	125
Ethyl acetate	120	103	91	110	110
Methyl isobutyl ketone	122	125	103	105	105
Butyl acetates	93	86	80	84	86
Other ketones and esters	70	75	70	74	75
Ethylene glycol	57	52	48	54	56
Propyl acetates	60	55	50	53	54
Other alcohols	40	42	40	41	41
Other solvents	40	40	36	38	38
Propylene glycol	30	32	29	33	34
Methylene chloride	15	15	14	15	15
Other glycols	1	1	1	1	1

An Evaluation of Engineering Control Technology for Spray Painting, NIOSH, 1981, p. 12.

METHOTREXATE B
Tablets and Parenteral

WARNING
METHOTREXATE MUST BE USED ONLY BY PHYSICIANS EXPERIENCED IN ANTIMETABOLITE CHEMOTHERAPY.
BECAUSE OF THE POSSIBILITY OF FATAL OR SEVERE TOXIC REACTIONS THE PATIENT SHOULD BE FULLY INFORMED BY THE PHYSICIAN OF THE RISKS INVOLVED AND SHOULD BE UNDER HIS CONSTANT SUPERVISION.
DEATHS HAVE BEEN REPORTED WITH THE USE OF METHOTREXATE IN THE TREATMENT OF PSORIASIS.
IN THE TREATMENT OF PSORIASIS METHOTREXATE SHOULD BE RESTRICTED TO SEVERE, RECALCITRANT, DISABLING, PSORIASIS WHICH IS NOT ADEQUATELY RESPONSIVE TO OTHER FORMS OF THERAPY, BUT ONLY WHEN THE DIAGNOSIS HAS BEEN ESTABLISHED, AS BY BIOPSY AND/OR AFTER DERMATOLOGIC CONSULTATION.

1. Methotrexate may produce marked depression of bone marrow, anemia, leukopenia, thrombocytopenia and bleeding.
2. Methotrexate may be hepatotoxic, particularly at high dosage or with prolonged therapy. Liver atrophy, necrosis, cirrhosis, fatty changes, and periportal fibrosis have been reported. Since changes may occur without previous

Description: Methotrexate Lederle (formerly A-methopterin) is an antimetabolite used in the treatment of certain neoplastic diseases.
Methotrexate Tablets contain 2.5 mg of Methotrexate.
Methotrexate Sodium Parenteral is available both in 2.5 mg/ml and 25 mg/ml strengths each available in 2 ml vials.
Each 2.5 mg/ml (5.0 mg) vial contains per 2 ml: Methotrexate Sodium equivalent to 5 mg Methotrexate, 0.90% w/v of benzyl alcohol as a preservative, and the following Inactive Ingredients. Sodium Chloride 0.630% w/v and Water for Injection qs ad 100% V. Sodium Hydroxide to adjust pH to approx. 8.5.
Each 25 mg/ml (50 mg) vial contains per 2 ml: Methotrexate Sodium equivalent to 50 mg Methotrexate, 0.90% w/v of benzyl alcohol as a preservative, and the following Inactive Ingredients: Sodium Chloride 0.260% w/v and Water for Injection qs ad 100% V. Sodium Hydroxide to adjust pH to approx. 8.5.
If desired, the solution may be further diluted with a compatible medium such as Sodium Chloride Injection USP. Storage for 24 hours at a temperature of 21° to 25° C results in a product which is within 90% of label potency.
Each 20 mg vial of sterile cryodesiccated powder, for single use only, contains: Methotrexate Sodium equivalent to 20 mg Methotrexate. Contains no preservative, with Sodium Hydroxide as an inactive ingredient. Contains approximately 0.14 milliequivalents of sodium per vial. Reconstitute immediately prior to use with 20 ml of an appropriate sterile, preservative-free medium such as Sodium Chloride Injection, USP.
Action: Methotrexate has as its principal mechanism of action the competitive inhibi-

Continued on next page

Lederle—Cont.

tion of the enzyme folic acid reductase. Folic acid must be reduced to tetrahydrofolic acid by this enzyme in the process of DNA synthesis and cellular replication. Methotrexate inhibits the reduction of folic acid and interferes with tissue-cell reproduction.

Actively proliferating tissues such as malignant cells, bone marrow, fetal cells, dermal epithelium, buccal and intestinal mucosa and cells of the urinary bladder are in general more sensitive to this effect of Methotrexate. Cellular proliferation in malignant tissue is greater than in most normal tissue and thus Methotrexate may impair malignant growth without irreversible damage to normal tissues.

Orally administered Methotrexate is absorbed rapidly in most, but not all patients, and reaches peak serum levels in 1–2 hours. After parenteral injection, peak serum levels are seen in about one-half this period. Approximately one-half the absorbed Methotrexate is reversibly bound to serum protein, but exchanges with body fluids easily and diffuses into the body tissue cells.

Excretion of single daily doses occurs through the kidneys in amounts from 55% to 88% or higher within 24 hours. Repeated doses daily result in more sustained serum levels and some retention of Methotrexate over each 24 hour period which may result in accumulation of the drug within the tissues. The liver cells appear to retain certain amounts of the drug for prolonged periods even after a single therapeutic dose. Methotrexate is retained in the presence of impaired renal function and may increase rapidly in the serum and in the tissue cells under such conditions. Methotrexate does not penetrate the blood cerebrospinal fluid barrier in therapeutic amounts when given orally or parenterally. High concentrations of the drug when needed may be attained by direct intrathecal administration.

In psoriasis, the rate of production of epithelial cells in the skin is greatly increased over normal skin. This differential in reproductive rates is the basis for the use of Methotrexate to control the psoriatic process.

Indications:

Anti-neoplastic Chemotherapy

Methotrexate is indicated for the treatment of gestational choriocarcinoma, and in patients with chorioadenoma destruens and hydatidiform mole.

Methotrexate is indicated for the palliation of acute lymphocytic leukemia. It is also indicated in the treatment and prophylaxis of meningeal leukemia. Greatest effect has been observed in palliation of acute lymphoblastic (stem-cell) leukemias in children. In combination with other anticancer drugs or suitable agents Methotrexate may be used for induction of remission, but it is most commonly used, as described in the literature, in the maintenance of induced remissions.

Methotrexate may be used alone or in combination with other anticancer agents in the management of breast cancer, epidermoid cancers of the head and neck, and lung cancer, particularly squamous cell and small cell types.

Methotrexate is also effective in the treatment of the advanced stages (III and IV, Peters Staging System) of lymphosarcoma, particularly in those cases in children; and in advanced cases of mycosis fungoides.

Psoriasis Chemotherapy [See box warnings at top of Insert] Because of high risk attending its use, Methotrexate is only indicated in the symptomatic control of severe, recalcitrant, disabling psoriasis which is not adequately responsive to other forms of therapy, *but only when the diagnosis has been established, as by biopsy and/or after dermatologic consultation.*

Contraindications: Pregnant psoriatic patients should not receive Methotrexate. Psoriatic patients with severe renal or hepatic disorders should not receive Methotrexate.

Psoriatic patients with pre-existing blood dyscrasias, such as bone marrow hypoplasia, leukopenia, thrombocytopenia or anemia, should not receive Methotrexate.

Warnings: See Box Warnings

Precautions: Methotrexate has a high potential toxicity, usually dose-related. The physician should be familiar with the various characteristics of the drug and its established clinical usage. Patients undergoing therapy should be subject to appropriate supervision so that signs or symptoms of possible toxic effects or adverse reactions may be detected and evaluated with minimal delay. Pretreatment and periodic hematologic studies are essential to the use of Methotrexate in chemotherapy because of its common effect of hematopoietic suppression. This may occur abruptly and on apparent safe dosage, and any profound drop in blood-cell count indicates immediate stopping of the drug and appropriate therapy. In patients with malignant disease who have pre-exising bone marrow aplasia, leukopenia, thrombocytopenia or anemia, the drug should be used with caution, if at all.

Methotrexate is excreted principally by the kidneys. Its use in the presence of impaired renal function may result in accumulation of toxic amounts or even additional renal damage. The patient's renal status should be determined prior to and during Methotrexate therapy and proper caution exercised should significant renal impairment be disclosed. Drug dosage should be reduced or discontinued until renal function is improved or restored.

In general, the following laboratory tests are recommended as part of essential clinical evaluation and appropriate monitoring of patients chosen for or receiving Methotrexate therapy: complete hemogram; hematocrit; urinalysis; renal function tests; and liver function tests. A chest x-ray is also recommended. The purpose is to determine any existing organ dysfunction *or system impairment.* The tests should be performed prior to therapy, at appropriate periods during therapy and after termination of therapy. It may be useful or important to perform liver biopsy or bone marrow aspiration studies where high dose or long-term therapy is being followed.

Methotrexate is bound in part to serum albumin after absorption and toxicity may be increased because of displacement by certain drugs such as salicylates, sulfonamides, diphenylhydantoin, phenylbutazone, and some antibacterials as tetracycline, chloramphenicol and para-amino-benzoic acid. These drugs, especially salicylates, phenylbutazone, and sulfonamides, whether antibacterial, hypoglycemic or diuretic, should not be given concurrently until the significance of these findings is established.

Vitamin preparations containing folic acid or its derivatives may alter responses to Methotrexate.

Methotrexate should be used with extreme caution in the presence of infection, peptic ulcer, ulcerative colitis, debility, and in extreme youth and old age.

If profound leukopenia occurs during therapy, bacterial infection may occur or become a threat. Cessation of the drug and appropriate antibiotic therapy is usually indicated. In severe bone marrow depression, blood or platelet transfusions may be necessary.

Since it is reported that Methotrexate may have an immunosuppressive action, this factor must be taken into consideration in evaluating the use of the drug where immune responses in a patient may be important or essential.

In all instances where the use of Methotrexate is considered for chemotherapy, the physician must evaluate the need and usefulness of the drug against the risks of toxic effects or adverse reaction. Most such adverse reactions are reversible if detected early. When such effects or reactions do occur, the drug should be reduced in dosage or discontinued and appropriate corrective measures should be taken, according to the clinical judgment of the physician. Reinstitution of Methotrexate therapy should be carried out with caution, with adequate consideration of further need for the drug and alertness as to possible recurrence of toxicity.

Adverse Reactions: The most common adverse reactions include ulcerative stomatitis, leukopenia, nausea and abdominal distress. Others reported are malaise, undue fatigue, chills and fever, dizziness and decreased resistance to infection. In general, the incidence and severity of side effects are considered to be dose-related. Adverse reactions as reported for the various systems are as follows:

Skin: erythematous rashes, pruritus, urticaria, photosensitivity, depigmentation, alopecia, ecchymosis, telangiectasia, acne, furunculosis. Lesions of psoriasis may be aggravated by concomitant exposure to ultraviolet radiation.

Blood: bone marrow depression, leukopenia, thrombocytopenia, anemia, hypogammaglobulinemia, hemorrhage from various sites, aplastic anemia.

Alimentary System: gingivitis, pharyngitis, stomatitis, anorexia, vomiting, diarrhea, hematemesis, melena, gastrointestinal ulceration and bleeding, enteritis, hepatic toxicity resulting in acute liver atrophy, necrosis, fatty metamorphosis, periportal fibrosis, or hepatic cirrhosis.

Urogenital System: renal failure, azotemia, cystitis, hematuria, defective oogenesis or spermatogenesis, transient oligospermia, menstrual dysfunction; infertility, abortion, fetal defects, severe nephropathy.

Pulmonary System: Interstitial Pneumonitis. Deaths have been reported and chronic interstitial obstructive pulmonary disease has occasionally occurred.

Central Nervous System: Headaches, drowsiness, blurred vision. Aphasia, hemiparesis, paresis and convulsions have also occurred following administration of Methotrexate.

There have been reports of leucoencephalopathy following intravenous administration of Methotrexate to patients who have had cranio-spinal irradiation.

After the intrathecal use of Methotrexate, the central nervous system toxicity which may occur can be classified as follows: (1) chemical arachnoiditis manifested by such symptoms as headache, back pain, nuchal rigidity, and fever; (2) paresis, usually transient, manifested by paraplegia associated with involvement with one or more spinal nerve roots; (3) leucoencephalopathy manifested by confusion, irritability, somnolence, ataxia, dementia, and occasionally major convulsions.

Other reactions related to or attributed to the use of Methotrexate such as metabolic changes, precipitating diabetes; osteoporotic effects, abnormal tissue cell changes, and even sudden death have been reported.

Dosage and Administration:

Anti-neoplastic chemotherapy

Oral administration in tablet form is often preferred since absorption is rapid and effective serum levels are obtained. Methotrexate sodium parenteral may be given by intramuscular, intravenous, intraarterial or intrathecal route. Initial treatment is usually undertaken with the patient under hospital care.

*For conversion of mg/kg b.w. to mg/M^2 of body surface or the reverse, a ratio of 1:30 is given as a guide line. The conversion factor varies between 1:20 and 1:40 depending on age and body build.

Choriocarcinoma and similar trophoblastic diseases: Methotrexate is administered orally or intramuscularly in doses of 15 to 30 mg daily for a 5 day course. Such courses are usually repeated for 3 to 5 times as required, with rest

periods of one or more weeks interposed between courses, until any manifesting toxic symptoms subside. The effectiveness of therapy is ordinarily evaluated by 24 hour quantitative analysis of urinary chorionic gonadotropin hormone (CGH), which should return to normal or less than 50 IU/24 hr. usually after the 3rd or 4th course and usually be followed by a complete resolution of measurable lesions in 4 to 6 weeks. One to two courses of Methotrexate after normalization of CGH is usually recommended. Before each course of the drug careful clinical assessment is essential. Cyclic combination therapy of Methotrexate with other antitumor drugs has been reported as being useful. Since hydatidiform mole may precede or be followed by choriocarcinoma, prophylactic chemotherapy with Methotrexate has been recommended. Chorioadenoma destruens is considered to be an invasive form of hydatidiform mole. Methotrexate is administered in these disease states in doses similar to those recommended for choriocarcinoma.

Leukemia: acute lymphatic (lymphoblastic) leukemia in children and young adolescents is the most responsive to present day chemotherapy. In young adults and older patients, clinical remission is more difficult to obtain and early relapse is more common. In chronic lymphatic leukemia, the prognosis for adequate response is less encouraging.

Methotrexate alone or in combination with steroids was used initially for induction of remission of lymphoblastic leukemias. More recently corticosteroid therapy in combination with other antileukemic drugs or in cyclic combinations with Methotrexate included appear to produce rapid and effective remissions. When used for induction, Methotrexate in doses of 3.3 mg/M^2 in combination with prednisone 60 mg/M^2, given daily, produced remission in 50% of patients treated, usually within a period of 4 to 6 weeks. Methotrexate alone or in combination with other agents appears to be the drug of choice for securing maintenance of drug-induced remissions. When remission is achieved and supportive care has produced general clinical improvement, maintenance therapy is initiated, as follows: Methotrexate is administered 2 times weekly either by mouth or intramuscularly in doses of 30 mg/M^2. It has also been given in doses of 2.5 mg/kg intravenously every 14 days. If and when relapse does occur, reinduction of remission can again usually be obtained by repeating the initial induction regimen. Various experts have recently introduced a variety of dosage schedules for both induction and maintenance of remission with various combinations of alkylating and antifolic agents. Multiple drug therapy with several agents, including Methotrexate given concomitantly is gaining increasing support in both the acute and chronic forms of leukemia. The physician should familiarize himself with the new advances in antileukemic therapy.

Acute granulocytic leukemia is rare in children but common in adults. This form of leukemia responds poorly to chemotherapy and remissions are short with relapses common, and resistance to therapy develops rapidly.

Meningeal leukemia: Patients with leukemia are subject to leukemic invasion of the central nervous system. This may manifest characteristic signs or symptoms or may remain silent and be diagnosed only by examination of the cerebrospinal fluid which contains leukemic cells in such cases. Therefore, the CSF should be examined in all leukemic patients. Since passage of Methotrexate from blood serum to the cerebrospinal fluid is minimal, for adequate therapy the drug is administered intrathecally. It is now common practice because of the noted increased frequency of meningeal leukemia to administer Methotrexate intrathecally as prophylaxis in all cases of lymphocytic leukemia.

By intrathecal injection, the sodium salt of Methotrexate is administered in solution in doses of 12 mg per square meter of body surface or in an empirical dose of 15 mg. The solution is made in a strength of 1 mg per ml as stated above with an appropriate, sterile, preservative-free medium such as Sodium Chloride Injection, USP.

For the treatment of meningeal leukemia, Methotrexate is given at intervals of 2 to 5 days. Methotrexate is administered until the cell count of the cerebrospinal fluid returns to normal. At this point one additional dose is advisable.

For prophylaxis against meningeal leukemia, the dosage is the same as for treatment except for the intervals of administration.

On this subject, it is advisable for the physician to consult the medical literature.

Large doses may cause convulsions. Untoward side effects may occur with any given intrathecal injection and are commonly neurological in character. Methotrexate given by intrathecal route appears significantly in the systemic circulation and may cause systemic Methotrexate toxicity. Therefore systemic antileukemic therapy with the drug should be appropriately adjusted, reduced or discontinued. Focal luekemic involvement of the central nervous system may not respond to intrathecal chemotherapy and is best treated with radiotherapy.

Lymphomas: in Burkitt's Tumor, Stages I-II, Methotrexate has produced prolonged remissions in some cases. Recommended dosage is 10 to 25 mg per day orally for 4 to 8 days. In stage III, Methotrexate is commonly given concomitantly with other antitumor agents. Treatment in all stages usually consists of several courses of the drug interposed with 7 to 10 day rest periods. Lymphosarcomas in Stage III may respond to combined drug therapy with Methotrexate given in doses of 0.625 mg to 2.5 mg/kg daily. Hodgkin's Disease responds poorly to Methotrexate and to most types of chemotherapy.

Mycosis fungoides: therapy with Methotrexate appears to produce clinical remissions in one half of the cases treated. Dosage is usually 2.5 to 10 mg daily by mouth for weeks or months. Dose levels of drug and adjustment of dose regimen by reduction or cessation of drug are guided by patient response and hematologic monitoring. Methotrexate has also been given intramuscularly in doses of 50 mg once weekly or 25 mg 2 times weekly.

Psoriasis Chemotherapy

The patient should be fully informed of the risks involved and should be under constant supervision of the physician.

Assessment of renal function, liver function, and blood elements should be made by history, physical examination, and laboratory tests (such as CBC, urinalysis, serum creatinine, liver function studies, and liver biopsy if indicated) before beginning Methotrexate, periodically during Methotrexate therapy, and before reinstituting Methotrexate therapy after a rest period. Appropriate steps should be taken to avoid conception during and for at least eight weeks following Methotrexate therapy.

There are three commonly used general types of dosage schedules:

1) weekly oral or parenteral intermittent large doses
2) divided dose intermittent oral schedule over a 36 hour period
3) daily oral with a rest period

All schedules should be continually tailored to the individual patient. Dose schedules cited below pertain to an average 70 Kg adult. An initial test dose one week prior to initiation of therapy is recommended to detect any idiosyncrasy. A suggested dose range is 5–10 mg parenterally.

Recommended starting dose schedules:

1. Weekly single oral, IM or IV dose schedule: 10–25 mg per week until adequate response is achieved. With this dosage schedule, 50 mg per week should ordinarily not be exceeded.
2. Divided oral dose schedule: 2.5 mg at 12 hour intervals for three doses or at 8 hour intervals for four doses each week. With this dosage schedule, 30 mg per week should not be exceeded.
3. Daily oral dose schedule: 2.5 mg daily for five days followed by at least a two day rest period. With this dosage schedule, 6.25 mg per day should not be exceeded.

SPECIAL NOTE: Available data suggest that schedule 3 may carry an increased risk of serious liver pathology.

Dosages in each schedule may be gradually adjusted to achieve optimal clinical response, but not to exceed the maximum stated for each schedule.

Once optimal clinical response has been achieved, each dosage schedule should be reduced to the lowest possible amount of drug and to the longest possible rest period. The use of Methotrexate may permit the return to conventional topical therapy, which should be encouraged.

Antidote for Overdosage: Leucovorin (citrovorum factor) is a potent agent for neutralizing the immediate toxic effects of Methotrexate on the hematopoietic system. Where large doses or overdoses are given, Calcium Leucovorin may be administered by intravenous infusion in doses up to 75 mg within 12 hours, followed by 12 mg intramuscularly every 6 hours for 4 doses. Where average doses of Methotrexate appear to have an adverse effect, 2 to 4 ml (6 to 12 mg) of Calcium Leucovorin may be given intramuscularly every 6 hours for 4 doses. In general, where overdosage is suspected, the dose of Leucovorin should be equal to or higher than the offending dose of Methotrexate and should best be administered within the first hour. Use of Calcium Leucovorin after an hour delay is much less effective.

CAUTION: Pharmacist: Because of its potential to cause severe toxicity, Methotrexate therapy requires close supervision of the patient by the physician. Pharmacists should dispense no more than a seven (7) day supply of the drug at one time. Refill of such prescriptions should be by direct order (written or oral) of the physician only.

How Supplied:

2.5 mg Tablets—Bottles of 100.

2.5 mg per ml—2 ml Vials.

25 mg per ml—2 ml Vials.

20 mg Vial.

Military Depots

25 mg/ml—2 ml vial

NSN 6505-01-020-2367

EPA CARCINOGEN ASSESSMENT GROUP CARCINOGENS LIST REFERENCED IN CANCER POLICY CANDIDATES NOTICE

CHEMICALS HAVING SUBSTANTIAL EVIDENCE OF CARCINOGENICITY[1]

2-Acetylaminofluorene
Acrylonitrile
Aflatoxins[2]
Aldrin
4-Aminobiphenyl
Amitrole
Aramite
Arsenic and Arsenic Compounds
Asbestos —
Auramine and the manufacture of Auramine
Azaserine [3]
Benz(c)acridine
Benz(a)anthracene
Benzene
Benzidine
Benzo(a)pyrene
Benzo(b)fluoranthene
Benzo(j)fluoranthene [4]
Beryllium and Beryllium Compounds
N,N-Bis(2-Chloroethyl)-2-Naphthylamine (Chlornaphazine) [5]
Cadmium and Cadmium Compounds
Carbon Tetrachloride
Chlorambucil [3]
Chloroalkyl Ethers
Bis(2-chloroethyl) ether (BCEE)
Bis(chloromethyl) ether (BCME)
Chloromethyl methyl ether (CMME), technical grade
Chlordane
Chlorinated Ethanes
1,2-Dichloroethane [Ethylene Chloride; Ethylene Dichloride (EDC)]
Hexachloroethane
1,1,2,2-Tetrachloroethane
1,1,2-Trichloroethane [4]
Chlorobenzilate
Chloroform
Chromium Compounds, Hexavalent
Chrysene [4]
Citrus Red No. 2
Coal Tar and Soot
Coke Oven Emissions [Polycyclic Organic Matter (POM)]
Creosote
Cycasin
Cyclophosphamide [3]
Daunomycin [4]
DDT (Dichlorodiphenyltrichloroethane)
Diallate [4]
Dibenz(a,h)acridine
Dibenz(a,j)acridine
Dibenz(a,h)anthracene
7H-Dibenzo(c,g)carbazole
Dibenzo(a,e)pyrene
Dibenzo(a,h)pyrene
Dibenzo(a,i)pyrene
1,2-Dibromo-3-chloropropane (DBCP)
1,2-Dibromoethane [Ethylene Bromide, Ethylene Dibromide (EDB)]
3,3' Dichlorobenzidine (DCB)
Dieldrin
Diepoxybutane
1,2-Diethylhydrazine
Diethylstilbestrol (DES)
Dihydrosafrole
3,3'-Dimethoxybenzidine (*o*-Dianisidine)
p-Dimethylaminoazobenzene
7,12-Dimethylbenz(a)anthracene
3,3'-Dimethylbenzidine (*o*-Tolidine)
Dimethylcarbamoyl Chloride
1,1-Dimethylhydrazine
1,2-Dimethylhydrazine
Dimethyl Sulfate
2,4-Dinitrotoluene
1,4-Dioxane
1,2-Diphenylhydrazine
Epichlorohydrin
Ethylenebisdithiocarbamate (EBDC)
Ethyleneimine (Aziridine) [4]
Ethylene Oxide
Ethylenethiourea
Ethyl Methanesulfonate
Formaldehyde
Glycidaldehyde
Heptachlor
Hexachlorobenzene
Hexachlorobutadiene
Hexachlorocyclohexane (HCH)
Hydrazine
Indeno(1,2,3-cd)pyrene [4]
Iron Dextran [3] [4]
Isosafrole
Kepone (Chlordecone)
Lasiocarpine
Melphalan [3]
Methapyrilene [3]
3-Methylcholanthrene
4,4'-Methylenebis(2-Chloroaniline) (MOCA)
Methyl Iodide
Methyl Methanesulfonate
N-Methyl-N'-nitro-N-nitrosoguanidine
Methylthiouracil [3]
Mitomycin C [3]
Mustard Gas
1-Naphthylamine, technical grade
2-Naphthylamine
Nickel and Nickel Compounds
Nitrogen Mustard and its hydrochloride
Nitrogen Mustard N-oxide and its hydrochloride
5-Nitro-*o*-toluidine
4-Nitroquinoline-1-oxide
Nitrosamines
N-Nitrosodiethanolamine
N-Nitrosodiethylamine (DENA)
N-Nitrosodimethylamine (DMNA)
N-Nitrosodi-n-butylamine
N-Nitrosodi-n-propylamine
N-Nitrosomethylethylamine
N-Nitrosomethylvinylamine
N-Nitroso-N-Ethylurea (NEU)
N-Nitroso-N-Methylurea (NMU)
N-Nitroso-N-methylurethane
N-Nitrosomorpholine
N-Nitrosonornicotine

Nitrosamines (continued)
N-Nitrosopiperidine
N-Nitrosopyrrolidine
N-Nitrososarcosine
Pentachloronitrobenzene
Phenacetin [3]
Polychlorinated Biphenyls (PCBs) ✓
Pronamide
1,3-Propane Sultone
B-Propiolactone
Propylthiouracil[3]
Reserpine [3]
Saccharin [5]
Safrole [5]
Selenium Sulfide
Streptozotocin [1]
2,3,7,8-Tetrachlorodibenzo-*p*-dioxin (TCDD)
Tetrachloroethylene (Perchloroethylene)
Thioacetamide
Thiourea
o-Toluidine Hydrochloride
Toxaphene
Trichloroethylene
2,4,6-Trichlorophenol
Tris (1-aziridinyl)phosphine sulfide (Thio-TEPA)[3]
Tris(2,3-dibromopropyl)phosphate
Tris(2,3-dibromopropyl)phosphate
Trypan Blue, commercial grade
Uracil Mustard [3]
Urethane (Ethyl carbamate; ethyl ester of carbamic acid)
Vinyl Chloride
Vinylidene Chloride

[1] This is not a comprehensive list of *all* chemicals having substantial evidence of carcinogenicity. Other chemicals will be added. No attempt has been made to select chemicals based upon appropriateness for regulation by EPA. The list is intended to be a basis for selection by the various program offices according to their specific needs.
[2] Fungal toxin, not an industrially manufactured product.
[3] Used as a drug.
[4] Evaluated by IARC as not having sufficient evidence of carcinogenicity.
[5] Used as a food.

The News American
February 15, 1981

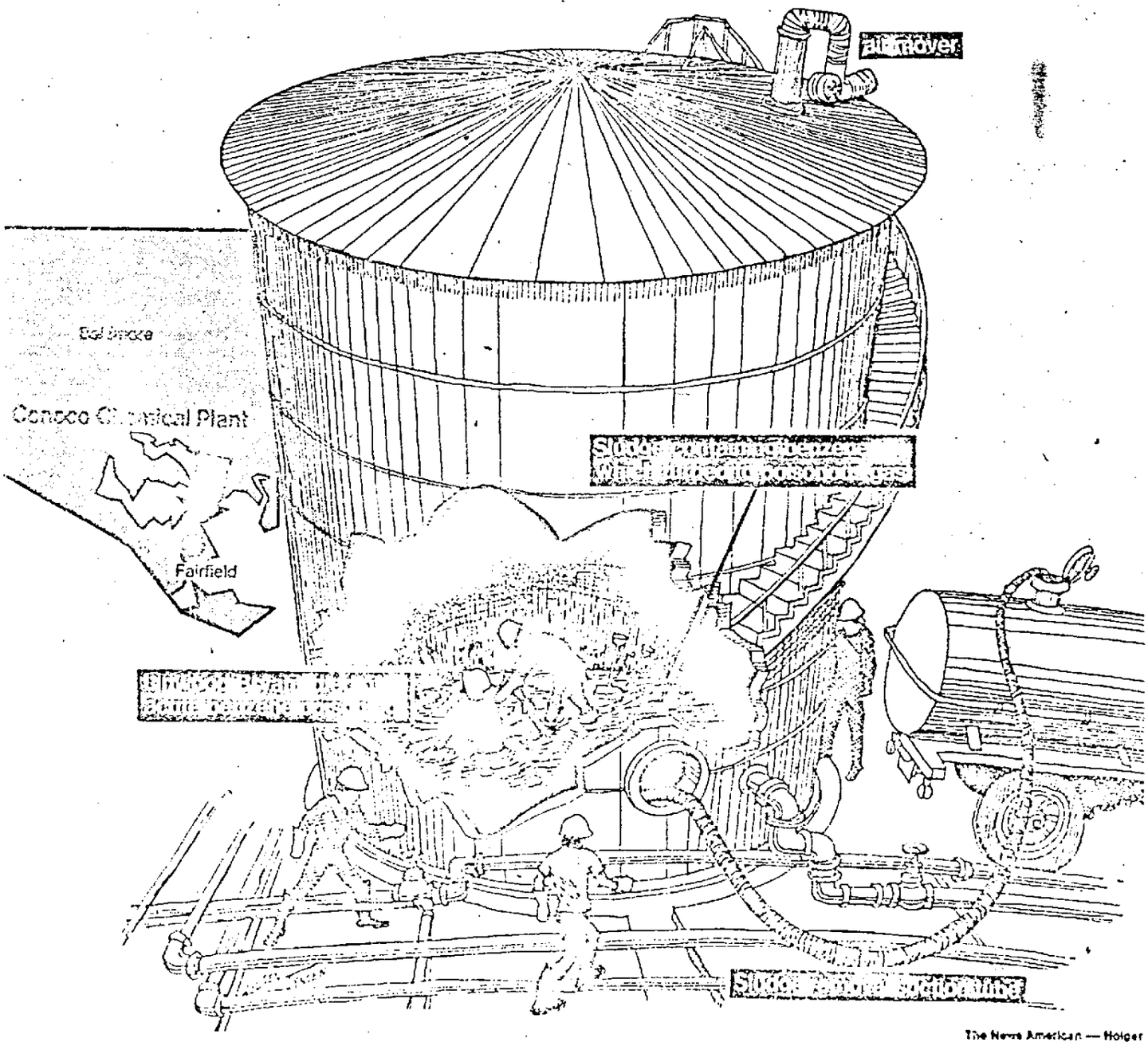
APPENDIX D

(H+S)(OSHA)(LS)

THE DISPOSABLE WORKERS

A NEWS AMERICAN SPECIAL REPORT

Why was an unskilled drifter hired to clean deadly chemicals that trained employees wouldn't touch? Why wasn't his family told of his death?



The News American — Holger

THE SCENE: Sketch shows how Linwood Bryant died at Conoco's Fairfield plant. Bryant is profiled on 10A.

SAL 000060949

The hospital's diagnosis

City Ambulance No. 9 took Bryant's three fellow workers to South Baltimore General Hospital.

Doctors at the emergency room examined them and determined that they had been exposed to noxious fumes. The official diagnosis: "acute hydrocarbon exposure."

Another ambulance carried Bryant to the hospital. He was pronounced dead there at 2:45 p.m. His body was sent to the state Medical Examiner's Office for an autopsy, where it was determined that he died from acute benzene poisoning.

No blood or urine tests were conducted on any of the three workers — an omission that later led MOSH consultant Dr. Ziem to send a letter criticizing South Baltimore General's handling of the case.

According to Dr. Ziem, without blood and urine samples doctors could not have determined whether the laborers had sustained damage to their bone marrow. And because benzene is a known cause of leukemia, a cancer that attracks bone marrow, those tests should have been performed, Dr. Ziem says.

"It's good medical practice to document it to see whether there was any effect on the bone marrow," says Dr. Ziem, who is a recognized expert on occupational disease. "And if there was, then one would want to watch them (those exposed) more closely."

But the chief of emergency services at South Baltimore General, Dr. Jean Thorne, says blood and urine tests were not performed on the three workers because doctors were not aware at that time that they had been exposed to benzene.

"These men came in and said they had been exposed to gas. But they had no idea what type of gas. They had no severe symptoms," Dr. Thorne says. "From a practical standpoint, we can't run all kinds of tests on everyone who comes in saying they were exposed to gases. We get those cases all the time. Sometimes companies will send us a roomful of workers who might have been exposed. In 98 percent of the cases, there's nothing wrong.

"We observed them (Reeves, Washington and Grant) for several hours to see if they had any latent symptoms before letting them go."

In the meantime, Joseph Pilkerton, manager of the Baltimore Peakload office and the man who hired Bryant and the other three workers, had notified MOSH of the accident.

When MOSH industrial hygienists LeClair and Jay Rupp checked Tank 152 a few hours after their 5:30 p.m. arrival at Conoco, the air blower on the tank's top hatch was not working. But Conoco company officials have told MOSH that it was on while the workers were inside.

Air samples taken by LeClair and Rupp inside the tank showed concentrations of benzene from 500 to 1,000 parts per million. The allowed level is 50 parts per million.

Safety officials say that with the level of benzene in the tank, the Norton 7500-30 respirators the workers were issued — which only filter air in the tank before it is inhaled — were virtually useless in the tank. More expensive respirators connected to an oxygen source outside the tank would have proably prevented Bryant's death, MOSH officers say.

Even when Rupp and LeClair took air samples with the fan working, tests indicated the benzene inside the tank atmosphere was at least double the level that the respirators could have safely handled.

And, say state safety investigators, neither Chesapeake nor Peakload officials sufficiently trained the four laborers in how to use the respirators.

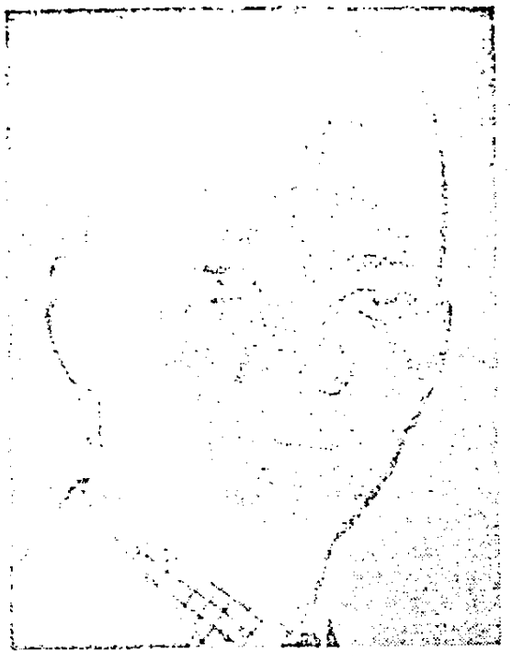
If the workers had received proper instructions, safety officials say, they would have known that smelling fumes while wearing the masks was an indication that they were being poisoned.

Whether the air sampling performed earlier that day by Conoco was legally sufficient is a matter of debate.

According to LeClair, current state standards require that a company take an air sample from inside a tank before workers enter. But the standards do not specifically state that air throughout the tank be tested, LeClair says.

The death of a day laborer

By Larry Lewis
and Joe Calderone
News American Staff



LINWOOD BRYANT: Shown in old photo.

On the morning of Aug. 6, 1990, Linwood Bryant, 40, was hired by a temporary labor agency to clean a south Baltimore chemical tank that contained benzene residue, a deadly organic chemical distilled from petroleum.

That afternoon, he was pronounced dead of chemical poisoning at South Baltimore General Hospital.

Bryant was an unemployed drifter — a temporary laborer who was staying at a city mission for the homeless. He had received virtually no training, enjoyed no union protection, was entitled to no benefits from his employer.

Until last week, his family didn't even know he was dead.

He had been hired by a labor subcontractor — the kind of company Baltimore industrial and manufacturing concerns are increasingly turning to for help in performing hazardous tasks that full-time employees cannot or will not perform.

The practice, say safety officials, exposes untrained, temporary workers to dangerous situations and has resulted in at least two deaths in recent months. It also permits manufacturing and industrial concerns to sidestep legal responsibility for accidents and injuries.

A *News American* inquiry uncovered these facts about Linwood Bryant's employment, the accident that led to his death, and the growing practice of hiring labor subcontractors for hazardous tasks:

● Bryant and his three co-workers were sent into the chemical tank despite the fact that employees of Conoco Inc., the owner of the tank, refused to do the work because they feared it was too dangerous.

See WORKERS, 10A

WORKERS from 1A

● Bryant, according to safety officials, was given inadequate equipment and training for the task.

● In violation of state and federal work laws, a 14-year-old youth was permitted to work beside Bryant in the chemical tank.

● Six months after he died, Bryant's relatives in New York still had not been notifed of his death either by the police or by the three companies that directly or indirectly employed him, even though *News American* reporters were able to locate the family after trying for only several hours.

● The three workers who survived the accident were not tested to determine the effects of the chemical exposure on their health, and did not receive medical examinations until more than a day after the accident.

● Maryland law permits companies that subcontract work to escape liability for accidents and deaths that occur on their properties, a major factor in the increasing use of subcontractors and temporary employees to do specialized and hazardous work.

As a temporary employee, Bryant received the minimum wage, $3.10 an hour. Like many day laborers, his working conditions are loosely regulated by state and federal agencies. No government agency keeps statistics on how many temporary laborers are injured or killed in Baltimore or in Maryland, or even on how many there are.

Despite increasing government regulation of safety and the workplace, there isn't even a precise definition of what a day laborer is. Anyone who calls himself a day laborer is one — like Linwood Bryant.

The practice of hiring subcontractors for specialized and sometimes dangerous work is increasing for many reasons, say occupational safety experts such as Dr. Grace Ziem, a physician and a consultant to the Maryland Occupational Safety and Health agency; Harvey Epstein, commissioner of Maryland's Division of Labor and Industry and president of a national group of state occupational safety agencies; and Dave Wilson, president of United Steelworkers of America Local 9477 and a local labor leader who specializes in safety issues.

Because unionized workers are increasingly reluctant to perform potentially hazardous jobs without special training and equipment, the experts say, some companies hire non-union workers for temporary work in an effort to save money.

Another reason companies sometimes hire labor subcontractors for hazardous work is that state law places responsibility for the health and safety of the workers on the firm that pays their salary — regardless of for whom the work actually is being done.

In fact, Maryland law does not require a company that hires a subcontractor to tell the subcontractor that a job might be dangerous or involve hazardous materials.

Although subcontractors sometimes bring expertise to the handling of such jobs, more frequently they do hazardous tasks as inexpensively and quickly as possible, often endangering workers, say Dr. Ziem and Wilson.

In another industrial death last July 24, involving a subcontractor, 20-year-old Mark Jones suffocated when he was lowered into a grain storage bin at Locust Point. The bin, owned by Indiana Grain Co., had not been tested for oxygen content before Jones entered, MOSH inspectors charge.

Jones worked for the Davco Corp., a Tennessee-based subcontractor hired by Indiana Grain to renovate its Baltimore grain storage facilities.

When he was killed, Jones was doing a job normally performed by Indiana Grain Co. employees, who were on strike at the time. MOSH officials have recommended that the Davco Corp. be fined $35,500.

Under Maryland law, Indiana Grain is not responsible for the death because the company, like Conoco, had hired a subcontractor.

Dr. Ziem says more and more companies are using temporary laborers as "disposable people" to perform hazardous tasks.

"When a company wants dirty work done, they call us," says Frank Dennison, a member of the office staff at Peakload Labor Inc., the company that hired Bryant. "We get the worst (jobs)."

Day laborers hired for jobs others won't take

"The law encourages subcontracting out of hazardous work," Dr. Ziem says. "If you're a large company and you subcontract a job out, first of all it's a way to get cheap labor, and if someone gets hurt, they can't file for workman's compensation against you. Unless the primary employer can be held responsible, we're going to see more of this."

According to Frank Morgan, chief of MOSH: "Temporary laborers are usually not the type of people who can demand safety and health conditions be met.

"Economically, most of the temporary people desperately need the money to live on a day-to-day basis, so they're not about to refuse a job, even if it might be hazardous. And they're not organized so they have no union speaking in their behalf. Most of the time, they don't have the knowledge or education to be aware of hazards.

"They're not the type of workers who are about to demand changes. It's a situation that's ripe for exploitation of the workers."

The Rev. Wade Dryden, director of the Helping Up Mission where Bryant spent his last night, says that the day laborers "are used and used and used. There's always somebody else to take their place.

"They get the dirty jobs union men won't do. Union men get $10 an hour. These men get the minimum wage."

Why Bryant died

The story of Linwood Bryant and Tank 152 boils down to this:

Conoco used the tank to store chemicals. When the company's own employees refused to clean the tank — fearing the fumes that had accumulated inside — Conoco turned to a subcontractor, Chesapeake Environmental Inc., which went to Peakload Labor for the workers to do the job.

Peakload hired Linwood Bryant, two other men and a 14-year-old to go into the tank with respirator gas masks. Two of the workers were wearing shorts.

Within two hours, Linwood Bryant was dead, according to an autopsy, of acute benzene chemical poisoning.

What follows are the events that led to Linwood Bryant's death and the aftermath, reconstructed from interviews with his coworkers, workers at the Conoco chemical plant, safety and MOSH officials, his employers, police and family members:

Deadly benzene

According to Mark Edmunds, a Conoco employee for seven years and a union official, Tank 152 had been used to store Nalkylene, a form of the chemical benzene that is the active ingredient in soap detergents.

It is also very dangerous and is known to cause cancer and damage the body's central nervous system.

The tank, which sits amid a 73-acre field of tanks and connecting pipes, is about 30 feet high and 25 feet in diameter and is painted yellow.

Because the form of Nalkylene stored in Tank 152 would not decompose naturally, the company stopped adding it several years ago.

Since then, Edmunds says, the part of the plant where Tank 152 sits has been shut down. Because it is isolated from chemical spills around the plant, tanks have been stored in Tank 152.

Sometime in early 1984, Conoco decided to clean Tank 152. Although the company will not discuss the tank or the accident that occurred there, Edmunds says he and two summer workers refused to clean it because he believed the fumes inside were too dangerous.

"My supervisor told me there was three or four inches left in the bottom of the tank," Edmunds says. "But it was still knee-deep in there. And you could smell the fumes as soon as you get near the tank."

Edmunds say he reported to his superiors that the job was too dangerous, and he was given another assignment for the day.

Then, about a month before the accident, Conoco made an attempt to clear the tank of gas. The tank was flooding with hot water and steam, drained and again filled with steam — a procedure that continued for the next 18 days.

And about 12 days before the accident, Conoco installed a fan-like "air mover" to the top hatch of the tank in an attempt to draw fresh air into it before workers went inside.

Conoco had gotten Chesapeake Environmental, a firm with an office at 8128 Pulaski Highway that specializes in environmental cleanup, to undertake the cleanup. On Aug. 5, four workers from Anybody Anytime Inc., another temporary labor agency, worked cleaning the tank.

According to MOSH officials, three of the same workers and another temporary employee returned the next morning but were dismissed shortly after they began work because George Crowley of Chesapeake says they appeared drunk — symptoms identical to those of benzene poisoning.

Crowley then called Peakload to get another cleanup crew.

On both mornings, Conoco had tested the air inside Tank 152 and determined that there were no toxic fumes present and that the oxygen was sufficient.

But, safety inspectors say, the tests were inadequate because the air sampled was within the area where the fan had created only a narrow column of air flowing from the bottom hatch to the top manhole — without circulating air to any other areas.

Most of the atmosphere inside the tank away from the entrance holes remained stagnant and heavily laced with benzene, state safety officials say.

A thorough test of the air inside the tank would likely have revealed that it could kill workers wearing the kind of respirators issued to the four workers, says Ron LeClair, a state industrial hygienist with MOSH.

And, according to LeClair, no further tests of the air were done while the men were working in the sludge — an activity LeClair says would have increased the level of benzene in the tank's atmosphere.

Inside the tank, Bryant and the three other workers — Milton Grant, 21, Bernard Washington, 20, Raymond Reeves, 14 — pushed the sludge with shovels and sponge mops, attempting to get it to a pump that would push it through a hose to a truck outside.

While in the tank, two or three of the day laborers reported that they complained of headaches and dizziness, MOSH investigators say.

Crowley told MOSH investigators that the workers did not tell him of such problems.

After taking a break, the men re-entered the tank, but again complained of fumes and were attempting to get out when Bryant slipped and fell into the sludge, Reeves and Grant say. They and Crowley began pulling Bryant's unconscious body from the tank porthole.

Two Conoco workers ran toward the tank and Edmunds followed them.

"He was covered with that sludge," Edmunds says. "We had to wipe it away from his eyes, nose, mouth and ears. His eyes were open, but he was completely limp."