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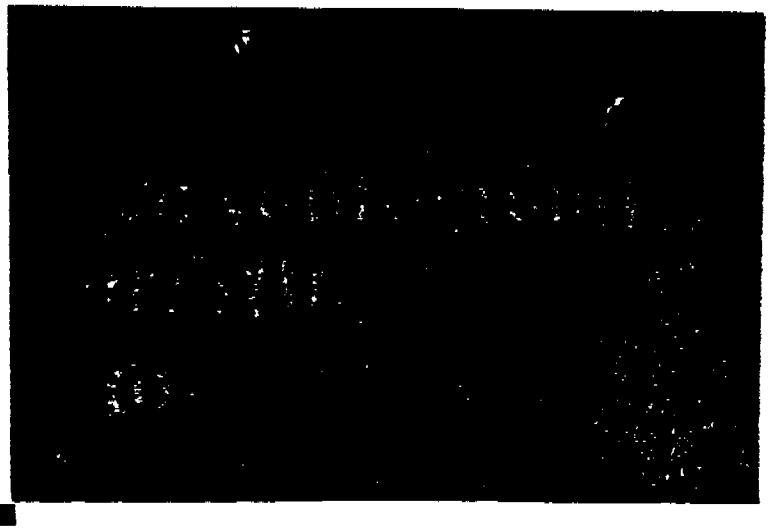


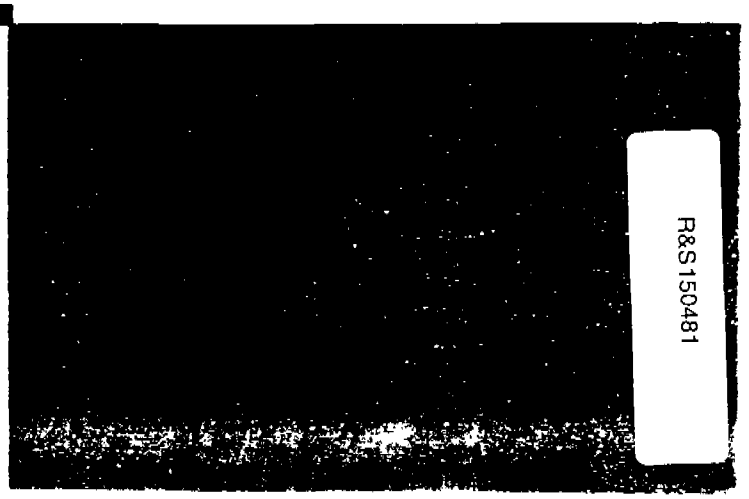
VINYL CHLORIDE

Draft
For Public Comment

U.S. DEPARTMENT OF HEALTH & HUMAN SERVICES
Public Health Service
Agency for Toxic Substances and Disease Registry

Comment Period Ends:

February 18, 1992



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TOXICOLOGICAL PROFILE FOR VINYL CHLORIDE

Prepared by:

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Under Contract No. 205-88-0608

Prepared for:

U.S. DEPARTMENT OF HEALTH AND HUMAN SERVICES
Public Health Service
Agency for Toxic Substances and Disease Registry

October 1991

R&S150482

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Vinyl chloride is metabolized in the liver in a multistep process. The intermediary metabolites of vinyl chloride, 2-chloroethylene oxide and 2-chloroacetaldehyde, have been suggested to be responsible for some of the adverse effects produced by vinyl chloride. Thus, activation of the enzyme system responsible for production of these toxic metabolites would be expected to increase the toxicity of vinyl chloride exposures. 2-Chloroethylene oxide is formed by action of the mixed function oxidase system associated with cytochrome P-450. The barbiturate, phenobarbital, and the organochlorine pesticide, Aroclor 1254, increased mixed function oxidase activity and have been shown to greatly increase the hepatotoxicity of vinyl chloride (Conolly and Jaeger et al. 1979; Conolly et al. 1978; Jaeger et al. 1974, 1977; Jedrychowski et al. 1985; Reynolds et al. 1975a, 1975b). Thus, persons taking barbiturates or who might be exposed to organochlorine pesticides that are known to induce microsomal enzymes (such as Aroclor 1254) would be expected to be at increased risk for developing vinyl chloride-induced hepatotoxicity.

An alternative pathway for vinyl chloride metabolism in the liver involves the enzymes, alcohol dehydrogenase and aldehyde dehydrogenase. Vinyl chloride metabolized by these enzymes is not metabolized to 2-chloroethylene oxide, the toxic intermediary metabolite. Persons consuming sufficient amounts of alcohol inhibit this enzyme system (Hefner et al. 1975b; Hultmark et al. 1979). Therefore, these persons have increased levels of the toxic metabolite, 2-chloroethylene oxide. Radike et al. (1981) demonstrated that ethanol-consuming rats exposed to vinyl chloride had an increased incidence of cancer and an earlier death rate than animals exposed to vinyl chloride in the absence of ethanol.

Some persons consume the agent, Antabuse, to curb the desire for alcohol. In its role as a therap utic agent, Antabuse blocks aldehyde dehydrogenase and causes a build-up of acetaldehyde, which is emetic, in the body when alcohol is consumed. If persons taking Antabuse are exposed to vinyl chloride, the alternative metabolic pathway for vinyl chloride metabolism will be blocked, causing more vinyl chloride to be metabolized to the toxic metabolite, 2-chloroethylene oxide. Thus, these persons may be at increased risk for hepatotoxicity, cancer, and early death.

Very high levels of vinyl chloride have been demonstrated to cause cardiac arrhythmias in dogs (Carr et al. 1949; Oster et al. 1946). Persons with a propensity to develop cardiac arrhythmias due to heart disease or damage may be at an increased risk of having heart beat irregularities when exposed to high concentrations of vinyl chloride.

Vinyl chloride has been shown to cause decreased circulation in the hands and fingers of some persons. Persons with impaired circulation due to some other cause such as connective tissue disorders, systemic sclerosis, hyperviscosity of the blood, or use of vibrating tools, may experience more severe impairment of the circulation.

Work by Black et al. (1983, 1986) has shown that persons with the HLA allele HLA-DR5 may have an increased likelihood of developing vinyl chloride disease, and those with the alleles HLA-DR3 and B8 may have an increased severity of the disease.

2.8 ADEQUACY OF THE DATABASE

Section 104(i)(5) of CERCLA directs the Administrator of ATSDR (in consultation with the Administrator of EPA and agencies and programs of the Public Health Service) to assess whether adequate information on the health effects of vinyl chloride is available. Where adequate information is not available, ATSDR, in conjunction with the National Toxicology Program (NTP), is required to assure the initiation of a program

of research designed to determine the health effects (and techniques for developing methods to determine such health effects) of vinyl chloride.

The following categories of possible data needs have been identified by a joint team of scientists from ATSDR, NTP, and EPA. They are defined as substance-specific informational needs that if met would reduce or eliminate the uncertainties of human health assessment. This definition should not be interpreted to mean that all data needs discussed in this section must be filled. In the future, the identified data needs will be evaluated and prioritized, and a substance-specific research agenda will be proposed.

2.8.1 Existing Information on Health Effects of Vinyl Chloride

The existing data on health effects of inhalation, oral, and dermal exposure of humans and animals to vinyl chloride are summarized in Figure 2-4. The purpose of this figure is to illustrate the existing information concerning the health effects of vinyl chloride. Each dot in the figure indicates that one or more studies provide information associated with that particular effect. The dot does not imply anything about the quality of the study or studies. Gaps in this figure should not be interpreted as "data needs" information (i.e., data gaps that must necessarily be filled).

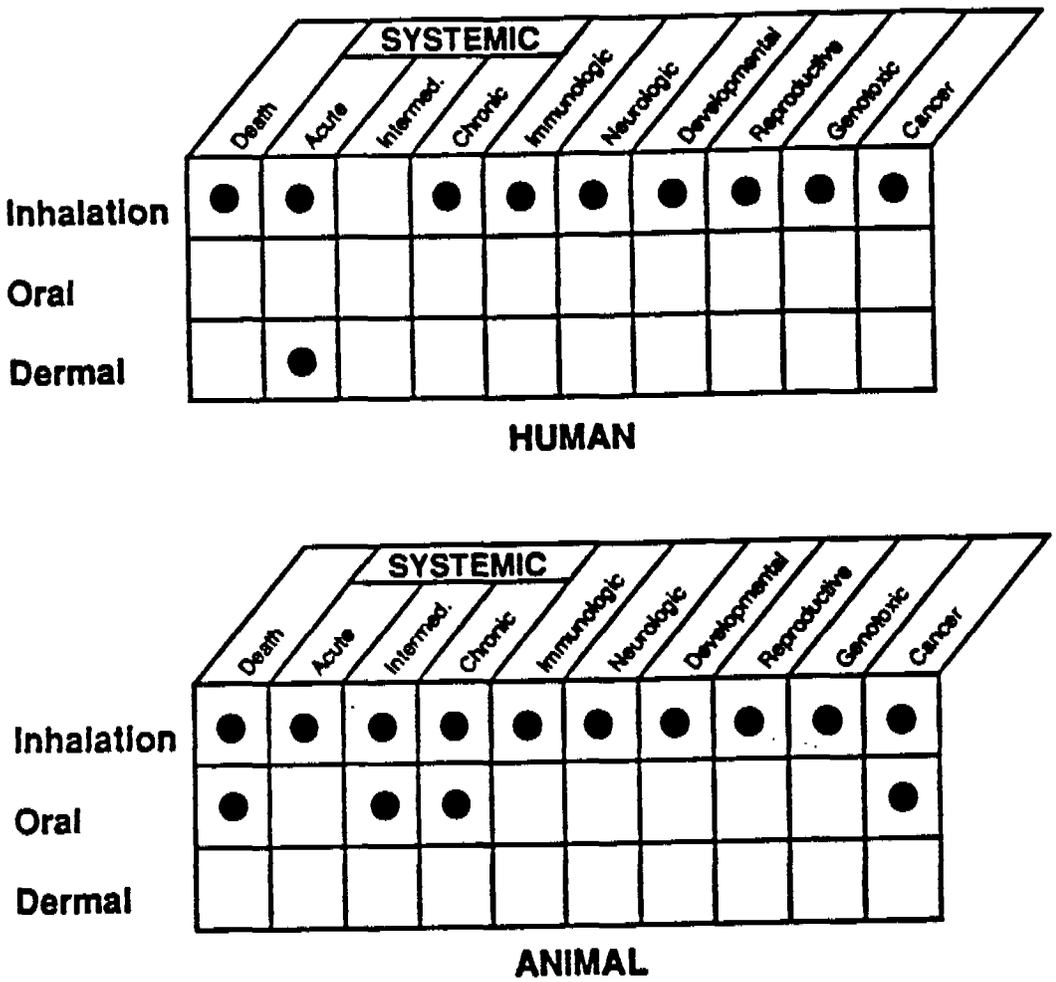
Virtually all of the literature regarding health effects in humans comes from studies of workers exposed to vinyl chloride during the production of PVC. Case reports and cohort studies describe some acute health effects and a wide range of long-term health effects. The predominant mode of exposure in these studies is via inhalation. These studies are limited by the lack of reliable data on individual exposure levels. No studies were found regarding the health effects of oral exposure. One case report examined the effects of dermal exposure to liquid vinyl chloride, but exposure by this route is not expected to contribute significantly to producing adverse health effects because of the limited absorption of vinyl chloride through the skin.

A large number of studies examining the health effects of inhaled vinyl chloride by animals were reviewed. As can be seen in Figure 2-4, no information is available on the acute systemic, immunologic, neurologic, developmental, reproductive, or genotoxic effects of exposure of animals by the oral route. No information is available regarding the health effects of exposure by the dermal route, but toxicokinetic studies indicate that this route is not an important means of exposure.

2.8.2 Identification of Data Needs

Acute-Duration Exposure. *Populations in areas that contain hazardous waste sites may be exposed to* vinyl chloride for brief periods. Exposure most likely would occur by inhalation, but relatively brief oral and dermal exposures are also possible. There are acute inhalation exposure data in humans and animals that indicate that the central nervous system is a major target organ of vinyl chloride toxicity. Symptoms of central nervous system depression ranging from dizziness and drowsiness to loss of consciousness have been observed in humans and animals as a result of brief exposure to very high levels of vinyl chloride. A threshold for central nervous system effects appears to be approximately 8,000 ppm. Extremely high concentrations of vinyl chloride produce death and respiratory irritation in humans and animals by the inhalation route. Based on studies in animals, the threshold for these effects appears to be in the range of 100,000–400,000 ppm. Extremely high concentrations of vinyl chloride produce cardiac arrhythmias in dogs exposed by the inhalation route. No threshold was reported for these effects; acute inhalation studies examining the incidence of cardiac irregularities at a variety of lower doses may be helpful in determining the threshold for this effect. Pharmacokinetic data indicate that similar end points might be expected if sufficiently high doses could be consumed by the oral route. However, the solubility characteristics of vinyl

FIGURE 2-4. Existing Information on Health Effects of Vinyl Chloride



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chloride in aqueous media (1,100-2,763 mg/L at 25°C) (Cowfer and Magistro 1983; EPA 1985b) indicate that achieving concentrations of vinyl chloride in excess of 5,000 ppm may be extremely difficult. Animal studies indicate that an adverse effect on the fetus is the most sensitive end point observed following brief inhalation exposures to vinyl chloride. Concentrations as low as 500 ppm were observed to cause adverse effects on developing fetuses. Studies examining the developmental, neurological, and systemic effects of the highest doses achievable in drinking water would be helpful for determining whether any effects would occur when vinyl chloride-contaminated groundwater or food products are consumed. One report described severe frostbite with second degree burns on the hands of a man resulting from the rapid evaporation of spilled liquid vinyl chloride. Toxicokinetic studies indicate that absorption of vinyl chloride vapor by the dermal route is insignificant; thus, studies examining the effects of acute-duration dermal exposure do not seem warranted.

A report was located regarding hepatic and respiratory effects observed 18 months following a single 1-hour inhalation exposure to vinyl chloride. However, limitations in the study diminished its reliability. Because of the implications of chronic effects from acute exposure, confirmation of these results in another study would be valuable.

Intermediate-Duration Exposure. No studies in humans specifically address intermediate-duration effects by any route. Most epidemiological studies of occupationally exposed persons have concentrated on persons who have been employed over several years. A study with reliable quantification of exposure levels that examined the effects experienced by vinyl chloride workers in their 1st year of exposure would be helpful for predicting the effects that might be observed in populations exposed to hazardous waste sites for similar periods of time. There is a large database describing the effects of intermediate-duration inhalation exposures in animals. Animals exposed to vinyl chloride vapor for more than 2 weeks and less than a year have experienced effects on the liver, kidneys, lungs, and blood. Data were sufficient to determine an intermediate-duration inhalation MRL based on liver effects. However, the MRL was based on a LOAEL and a no-effect level in animals would be more suitable for MRL derivation. Extremely limited information was available regarding oral intermediate-duration effects. One chronic study presented interim sacrifice data that identified relative weight and histopathological changes in the liver. However, only a single-dose group was compared to controls, precluding determination of the dose-response of the effects observed. Thus, no MRL for oral intermediate-duration exposures could be determined. Additional studies examining the effects of oral exposure to vinyl chloride would be helpful for predicting effects that might be observed in humans consuming contaminated drinking water or foods over a similar period of time. As noted above, absorption of vinyl chloride vapor through the skin is not expected to be significant; thus, additional dermal exposure studies do not seem warranted.

Chronic-Duration Exposure and Cancer. A large number of studies of workers exposed to vinyl chloride have identified a wide range of target organs that may be affected by chronic-duration inhalation of vinyl chloride. The target organs include the liver, lungs, blood, immune system, cardiovascular system, skin, bones, nervous system, and the reproductive organs. These studies are severely limited in that individual exposure levels have not been documented. In general, studies in animals provide supportive evidence for these effects and give indications of the exposure levels that may be associated with them.

No information was available regarding chronic-duration oral exposure in humans. However, studies in animals indicate that the liver, blood, and skin are target organs for oral exposure to vinyl chloride. A chronic-duration oral MRL was calculated based on hepatic toxicity. This value was based on a LOAEL. A no-effect level in animals would be more suitable for MRL determination.

No information was available regarding effects of chronic-duration dermal exposure in humans or animals, but absorption of vinyl chloride through the skin is not expected to be significant. Thus, dermal exposure studies do not seem warranted.

There is sufficient evidence to indicate that vinyl chloride is carcinogenic to humans and animals exposed via inhalation and in animals exposed via the oral route. The mechanism for carcinogenicity appears to be associated with the formation of reactive intermediates.

Genotoxicity. There are substantial data on both clastogenesis and DNA alkylation in humans exposed to vinyl chloride that indicate that this chemical acts as a potent genotoxicant. These findings are supported by both animal studies and in vitro studies that show positive genotoxicity in a variety of microbial organisms, cultured cell lines, and isolated nucleic acid assays. There are also data that support the premise that it is the chloroethylene oxide metabolite that is ultimately responsible for the direct action on the DNA, and that the mechanism involves alkylation and subsequent base-pair substitution. There are studies that indicate that the clastogenic effects of vinyl chloride exposure in humans are reversible; additional studies on the fate of the alkylated DNA in humans after an exposure-free interval would be useful for predicting the mechanisms involved in the latency period for carcinogenesis. In addition, further work elucidating the genotoxic role of chloroacetaldehyde would be useful to explain the inconsistent findings of the effects of this metabolite on clastogenesis and the induction of carcinogenesis.

Reproductive Toxicity. Data from a number of epidemiological studies provide suggestive evidence of adverse effects on male and female reproductive function. Sexual impotence and decreased androgen levels were found in men exposed occupationally to vinyl chloride. In women exposed to vinyl chloride, menstrual disturbances and an increased incidence of elevated blood pressure and edema during pregnancy (preeclampsia) were observed. Although no reports of two-generation reproduction studies in animals were located, two studies examining a range of toxic effects in rats indicate that vinyl chloride is toxic to the testes. A two-generation reproduction study in animals would be helpful to assess whether adverse effects on the rate of conception could be correlated with damage to the male reproductive organs or whether disturbances in female menstrual activity could be verified. Also, animal models of preeclampsia could be tested to determine the mechanism by which vinyl chloride might cause this effect. Well-designed and well-conducted epidemiological studies examining such changes would also be helpful. No data are available on the possible reproductive toxicity resulting from oral exposure to vinyl chloride. Oral studies that use drinking water as the vehicle of administration would be particularly useful because contaminated groundwater is a potentially significant source of human exposure.

Developmental Toxicity. The epidemiological studies that have addressed developmental toxicity in offspring of humans who have been exposed to vinyl chloride are controversial. Although some of these purport to show a significant association between birth defects and vinyl chloride exposure, their design and analysis have been severely criticized. At this time, there are insufficient human data to provide a definitive answer to this question. A well-designed and well-conducted epidemiological study examining potential developmental end points would be helpful. There are data showing that vinyl chloride is a developmental toxicant in animals when exposure is by inhalation. Continuous low-level exposure appears to be the most toxic, but the studies that indicated this are flawed. Additional studies examining exposure to low levels of vinyl chloride throughout gestation would be helpful in settling this issue. This issue is particularly important because women living in the vicinity of hazardous waste sites have the potential to be exposed to low levels of vinyl chloride on a continuous basis. Also, studies in animals suggest that offspring exposed in utero may experience adverse effects after birth, although these studies are also flawed. Epidemiological studies designed to look at this end point would be helpful. There are no data for oral exposures. Because of this

deficiency, oral studies examining a range of developmental end points would be useful in assessing the possibility of these effects in humans.

Immunotoxicity. Studies of workers occupationally exposed to vinyl chloride suggest that the immune system may be activated by vinyl chloride. Some data suggest that reactive intermediates may bind to proteins in the body, sufficiently altering them so that they become antigenic. In some instances, an autoimmune-like syndrome develops. The likelihood of this may be associated with the possession by individuals of specific genetic determinants (HLA alleles). Because of the low incidence of the autoimmune response in humans, the immunotoxicity may be best further studied in one of the strains of mice known to have a propensity for developing autoimmune diseases. Also, additional epidemiological studies examining the immune response of exposed populations may be helpful.

Neurotoxicity. A number of studies in humans and animals demonstrate that vinyl chloride is a central nervous system depressant following brief high-level inhalation exposures. Two studies in animals have also found degenerative effects in central nervous system tissue following chronic inhalation exposure to high levels of vinyl chloride. It is unknown whether these degenerative changes might also occur at lower doses; thus, a study examining the effects of a range of lower doses would be informative. In addition, relatively recent studies present suggestive evidence that vinyl chloride may also produce peripheral nerve damage in humans exposed chronically via inhalation. Animal studies examining histopathological and electrophysiological end points in peripheral nerves would be helpful for assessing what doses may be associated with this effect. Epidemiological studies examining exposed populations for subclinical peripheral nerve damage would be helpful. Oral exposure studies in animals would be beneficial for assessing the likelihood that populations exposed to contaminated water might develop symptoms of neurotoxicity.

Epidemiological and Human Dosimetry Studies. Virtually all of the data on effects in humans following inhalation exposure to vinyl chloride come from epidemiological studies of workers exposed during the production of PVC. These studies are limited by the absence of information on individual exposure levels. Also, in North America and Western Europe, only limited numbers of females have been studied.

For the most part, studies examining the carcinogenic potential of vinyl chloride have been adequate to distinguish an increased incidence of the rare cancer, angiosarcoma. However, many studies have used cohorts that are too small to detect smaller increases in other types of cancer (respiratory, central nervous system, lymphatic, or hematopoietic). Epidemiological studies designed to investigate reproductive and developmental effects of vinyl chloride have not been useful, in part because of a poor choice of statistical analysis, inadequate controls, or failure to take into account other chemical exposures. Additional cohort studies of these end points would be useful for examining these effects in humans.

Clastogenic effects have been used as a dosimeter for exposures to radioactive substances, and work has been done to use this approach for chemical exposures as well. More data on quantified exposures and well-controlled cytogenetic studies would be useful in developing a method for monitoring populations living near hazardous waste sites.

In addition, as noted above, well-designed and well-conducted epidemiological studies examining the incidence of peripheral neuropathies, developmental toxicity (birth defects, miscarriages, delayed prenatal development, postnatal hepatotoxicity), reproductive toxicity (male infertility, menstrual irregularity, preeclampsia in pregnant women), and immune reactivity in exposed populations would also be helpful.

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Biomarkers of Exposure and Effect. Several potential biomarkers for exposure to vinyl chloride have been identified. Vinyl chloride measured in expired air is an adequate indicator of recent, moderate- to high-level exposure. However, for low-level exposures or exposures that occur over 1–2 hours prior to the time of measurement, this biomarker is not useful. Thiodiglycolic acid, a major urinary metabolite of vinyl chloride, has been used to monitor workers occupationally exposed to vinyl chloride. However, this biomarker is rapidly excreted, and, therefore, the period of its utility is limited. Also, thiodiglycolic acid is not specific for vinyl chloride; it may also be produced as a result of the metabolism of 1,1 dichloroethene, ethylene oxide, or 2,2-dichloroethylether.

The DNA adducts 1,N^6-ethenoadenosine and 3,N^4-ethenocytidine, may be used to indicate vinyl chloride exposure, although studies correlating the levels of these adducts with exposure levels are still lacking. These products remain in the body longer than free vinyl chloride or thiodiglycolic acid, thereby increasing the period after exposure that a potential exposure may be detected. However, the presence of these adducts cannot indicate how long it has been since exposure occurred. In addition, these adducts are formed as the result of binding of the intermediary metabolites with nucleic acids, and other compounds producing the same intermediary metabolites will also produce these adducts. For example, these adducts have been identified as a result of exposure to vinyl bromide, ethyl carbamate, acrylonitrile, 2-cyanoethylene, and 1,2-dichloroethane. Studies attempting to identify a metabolite more specific to vinyl chloride may be helpful in developing a biomarker that may be used to facilitate future medical surveillance, which can lead to early detection and possible treatment.

The central nervous system depression associated with brief high-level exposures is easily determined by observation. The hepatic changes that may develop during longer-term exposures are difficult to detect by standard biochemical liver function tests. In contrast, tests of clearance such as the indocyanine clearance test or measurement of serum bile acid levels are more specific and sensitive indicators of vinyl chloride-induced liver damage. Angiosarcoma of the liver is a rare tumor type that has been shown to result from vinyl chloride exposure. However, other agents are known to cause angiosarcoma of the liver, such as arsenic and Thorotrast®. The cyanosis and blanching of the fingers in response to exposure to the cold may be an early indicator for the development of vinyl chloride disease. However, other conditions also known to cause these symptoms include connective tissue disorders, mechanical arterial obstruction, hyperviscosity of the blood, and exposure to drugs, chemicals, or vibrating tools. Finally, measurement of chromosomal aberrations may indicate the genotoxic effects of vinyl chloride. However, these aberrations do not specifically indicate vinyl chloride-induced damage. Also, DNA adducts may signal the potential to develop genotoxic effects. Further work identifying the correlation between the adducts and genotoxic effects would be useful.

Absorption, Distribution, Metabolism, and Excretion. There are few data on humans for all toxicokinetic parameters across all exposure routes. Additional studies examining the toxicokinetics of inhalation and oral exposure in humans would be beneficial. There are a number of animal studies describing the absorption, distribution, metabolism, and excretion of vinyl chloride administered via the oral and inhalation routes, but few describing the toxicokinetics of vinyl chloride administered via the dermal route. One study in rats found an extremely limited absorption of vinyl chloride vapor across the skin. If there is negligible absorption resulting from dermal exposure, then additional studies examining toxicokinetics from dermal exposures are not warranted; another study verifying the limited nature of dermal absorption, however, would be reassuring. Furthermore, the intermediary metabolites of vinyl chloride appear to be responsible for many of the toxic effects observed. Therefore, information regarding differences in the metabolic pattern according to sex, age, nutritional status, and species and correlations to differences in health effects would also be useful.

C mparative Toxicokinetics. The absorption, distribution, metabolism, and excretion have been studied in animals, but information on toxicokinetics in humans is extremely limited. Human and animal data indicate that similar target organs (liver, central nervous system) for the toxic effects of vinyl chloride exist, suggesting some similarities of kinetics. Limited information is available regarding interspecies differences in kinetics. Most toxicokinetic studies have been conducted using rats, but one study in primates indicates that metabolism may saturate at lower concentrations in primates than rats. This may suggest a lower saturation point in humans also. Additional studies measuring the toxicokinetics in humans could provide valuable data as the roles of individual metabolites in the disease processes associated with vinyl chloride exposure are elucidated.

2.8.3 On-going Studies

On-going studies regarding the health effects of vinyl chloride were reported in the Federal Research in Progress File (FEDRIP 1990) database and SCISearch (1990). Table 2-5 presents a summary of on-going studies that address the health effects of vinyl chloride.

Recently, EPA made improvements in methods for measuring volatile organic chemicals. The major change is the use of smaller sample volumes allowed by increased use of capillary gas chromatographic columns. Capillary columns provide better resolution, minimum detection limits, and less column bleed than packed columns (Reding 1987).

Vinyl chloride has been measured in sediment using GC/ECD with sensitivity in the low-ppb range. Accuracy and precision data were not given (Wang et al. 1985). No information on analysis of vinyl chloride in soil was located. GC/HSD of headspace gases is the EPA-recommended method for solid matrices with sensitivity in the sub-ppb range. Accuracy (101.9%) is good and precision (11.4%) is adequate (EPA 1982d). Vinyl chloride levels in food have been determined using GC/FID. Headspace analysis is a common method of preparation of foods with sensitivity in the low-ppb range (IARC 1978).

6.3 ADEQUACY OF THE DATABASE

Section 104(i)(5) of CERCLA directs the Administrator of ATSDR (in consultation with the Administrator of EPA and agencies and programs of the Public Health Service) to assess whether adequate information on the health effects of vinyl chloride is available. Where adequate information is not available, ATSDR, in conjunction with NTP, is required to assure the initiation of a program of research designed to determine the health effects (and techniques for developing methods to determine such health effects) of vinyl chloride.

The following categories of possible data needs have been identified by a joint team of scientists from ATSDR, NTP, and EPA. They are defined as substance-specific informational needs that if met would reduce or eliminate the uncertainties of human health assessment. This definition should not be interpreted to mean that all data needs discussed in this section must be filled. In the future, the identified data needs will be evaluated and prioritized, and a substance-specific research agenda will be proposed.

6.3.1 Identification of Data Needs

Methods for Determining Biomarkers of Exposure and Effect. Methods are available for measuring vinyl chloride and/or its metabolite, thiodiglycolic acid, in breath, urine, blood, and tissue. These methods are sensitive for measuring levels at which health effects might occur, and for measuring higher background levels that might be found in specific populations known to be exposed to elevated levels of vinyl chloride (e.g., workers in the plastics industry and individuals living in the vicinity of hazardous waste sites). Measurement of urinary thiodiglycolic acid can be used as an indicator of vinyl chloride intake as long as individual variability in metabolism due to such factors as liver disease, use of drugs, and alcohol intake can be accounted for. Exposure to vinyl chloride at concentrations below 1–5 ppm could be masked by background metabolic levels of thiodiglycolic acid within normal limits. Also, the formation of thiodiglycolic acid is not unique to vinyl chloride exposure. The methods are generally reliable, although increased precision for most methods would increase reliability. Background levels for the general population are ill defined. Further research on the relationship between low-level exposure and levels of vinyl chloride in biological media would be helpful in assessing the risks and health effects of chronic, low-level exposure.

Existing methods are sensitive for measuring levels of vinyl chloride and its metabolite, thiodiglycolic acid, in individuals affected by exposure to very high levels of vinyl chloride. Also, methods are available to detect DNA adducts produced by the reaction of vinyl chloride metabolites with DNA. These DNA adducts are specific indicators of vinyl chloride's genotoxic potential. These methods, however, are not sufficiently sensitive to determine the genotoxic effects resulting from low-level exposure. Correlations between levels detected in biological tissues and fluids, and specific observed effects for lower levels of exposure, are not

established. Additional research in this area would allow better assessment of existing methods and would help in defining areas in which improvements are needed.

Methods for Determining Parent Compounds and Degradation Products in Environmental Media. Existing methods for determining vinyl chloride in air and water, the media of most concern for human exposure, are sensitive, reproducible, and reliable for measuring background levels in the environment. Research investigating the relationship between levels measured in air and water and observed health effects could increase our confidence in existing methods and/or indicate where improvements are needed. Methods specifically regarding the analysis of vinyl chloride in soils were not located. EPA does, however, have sensitive and reliable methods for determining the concentration of vinyl chloride in soil matrices, which include contaminated soils.

6.3.2 On-going Studies

No on-going studies concerning methods for measuring and determining vinyl chloride in biological and environmental samples were located.

The Environmental Health Laboratory Sciences Division of the Center for Environmental Health and Injury Control, Centers for Disease Control, is developing methods for the analysis of vinyl chloride and other volatile organic compounds in blood. These methods use purge and trap methodology and magnetic sector mass spectrometry which gives detection limits in the low parts per trillion range.

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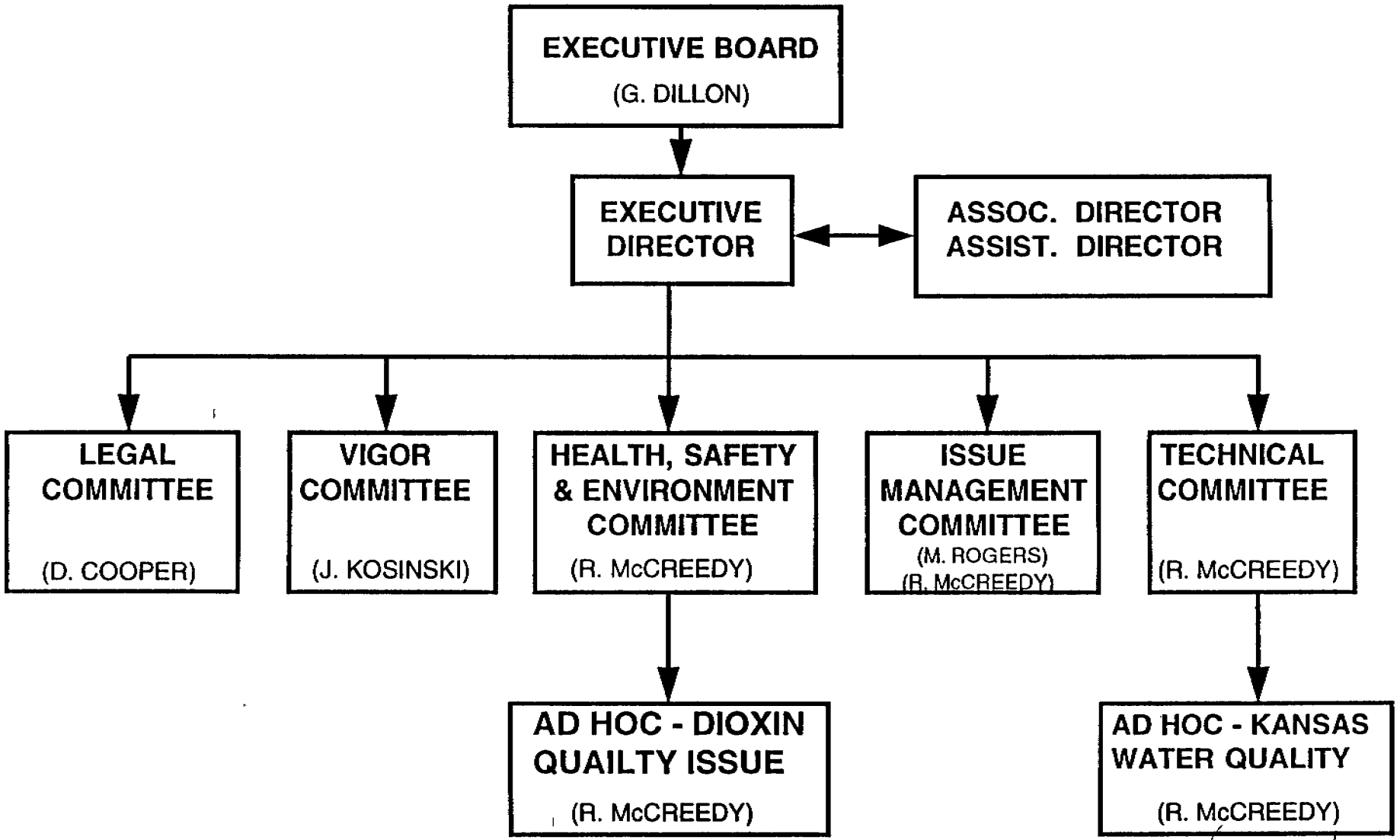

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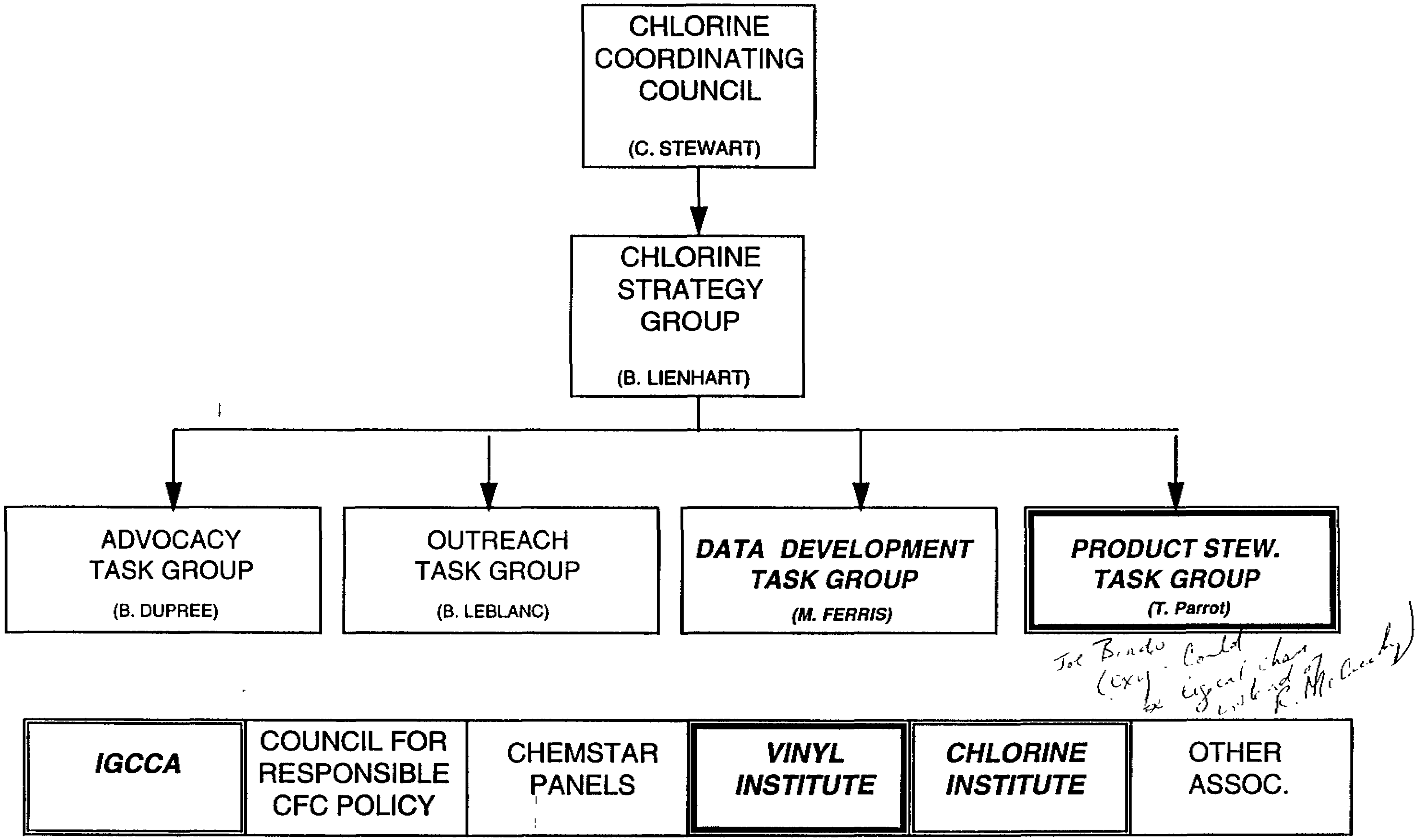

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