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according to appropriate protocols. The discussion in Chapter IV-A indicated that most experimental protocols have been deficient in design or analysis or both. Applying strict evaluative criteria to such protocols has reduced the number of chemicals of interest to relatively few, which have consistently been reported at levels greater than control values. These chemicals are listed in Table IV-39A. While a much longer list of chemical compounds was presented in the interim draft version of the public health section of this report (February 13, 1983), the draft list was based on a preliminary and less stringent evaluation of leaching data. The assessment of potential public health risks in the ERD is circumscribed by SRI's current analysis of the water quality experiments. Subsequent testing may warrant consideration of additional substances.

Other chemicals of potential interest are those that have been demonstrated to permeate plastic or metal pipe from the outside, which could contaminate domestic drinking water supplies. These substances are treated separately elsewhere in this chapter.

b. Exposure Assessment

An exposure assessment is a method for estimating quantities of a substance that humans are likely to ingest, inhale, or absorb through the skin. Necessarily based on multiple assumptions and approximations, an exposure assessment attempts to predict patterns of frequency and magnitude of human contact with the chemical(s) of interest.

In conducting the present analysis, the exposure assessments were not comprehensive, but consisted of identifying approximate concentrations of chemicals to be expected in drinking water. The results of such assessments were presented in Chapter IV-A.

c. Evaluation of Animal and Human Literature

Relevant epidemiologic, medical, and toxicological papers were identified by a review of the administrative record, a computerized

TABLE IV-30A

CHEMICALS OF INTEREST FOR PUBLIC HEALTH ANALYSIS*

CPVC Pipe

Dichloromethane
Chloroform
Carbon tetrachloride
Trichloroethylene
Tetrachloroethylene
Toluene
Dibutyl tin
Tetrahydrofuran
Dimethylformamide
Cyclohexanone
Methyl ethyl ketone

Polybutylene Pipe

Tetrakis [methylene (3,5-di-t-butyl-4-hydroxyhydrocinnamate] and fragments thereof

PVC Pipe

Dichloromethane
Chloroform
Carbon tetrachloride
Dimethyl tin bis-isooctylthioglycolate
Trichloroethylene
Tetrahydrofuran
Dimethylformamide
Cyclohexanone
Toluene
Methyl ethyl ketone

Table IV-30A (concluded)

Copper Pipe	
Cadmium	Tin
Nickel	Lead
Copper	Zinc

Galvanized Iron Pipe	
Cadmium	Iron
Nickel	Zinc
Copper	Lead
Selenium	

*Refer to chapter IV-A for documentation.

literature search (MEDLINE, TOXLINE and RTECS) supplemented by a manual search, and by reference to standard texts. Most of the materials reviewed were secondary sources.

d. Identification of Data Gaps

In performing the tasks described above, important deficiencies in the data were identified. Data gaps with respect to the identities of leachates and their probable range of concentrations in water (exposure assessment) were described in Chapter IV-A. Inadequacies in medical and toxicologic literature are described in a later section of this chapter.

e. Selection of Potentially Hazardous Chemicals

From the list of chemicals of interest, several were chosen for further analysis on the basis of both toxicologic properties and probable human exposure via the water supply. Other chemicals were not chosen for such an evaluation because they were judged to pose minimal risks of chronic toxicity at predicted concentrations. Chemicals not considered for further analysis on this basis include iron, selenium, and toluene (Exhibit IV-1).

f. Assessment of Likelihood and Magnitude of Risks

As noted earlier, there is considerable uncertainty in estimating risks of chronic disease from low doses of chemicals. There are multiple sources of such uncertainty: (1) the identities and concentrations of chemical leachates over time in drinking water; (2) quantities of tap water actually consumed by individuals (exposure); (3) the nature of toxic effects of chemical ingestion (where the medical or toxicologic literature has significant gaps or where the studied routes of exposure are not oral); (4) the validity of extrapolating the probability of toxic reactions from high doses to low doses and from animals to humans; and (5) potential interactions among chemicals in drinking water that may result in additive, synergistic, potentiating, or antagonistic effects. How each of these areas of uncertainty has been handled is discussed briefly in the following

Exhibit IV-1

JUSTIFICATION FOR CHEMICALS CONSIDERED TO POSE NEGLIGIBLE RISK

(1) Iron. The leaching studies reviewed earlier indicate that typical iron concentrations in water standing in galvanized steel pipe are less than 1 ppm (1 mg/liter). (See Tables IV-19, IV-20, and IV-26.) Even in the NSF study (1980), using pH 5 water--unrealistically low for California--the iron concentration in early samples would, if ingested by humans, contribute only a fraction of the daily dietary intake of this essential nutrient. The Recommended Daily Allowance for iron ranges from 10 mg for infants to 18 mg for teenage males and for females in their child-bearing years (Food and Nutrition Board, 1979). Furthermore, people have adequate homeostatic mechanisms to deal with this metal.

(2) Selenium. While selenium is toxic at high doses, it is an essential trace element. The recommended adequate and safe daily dietary intake ranges from 0.01 to 0.06 mg for infants to 0.05 to 0.2 mg for adults (Food and Nutrition Board, 1979). The NSF study (1980) (Table 4-26), using water at an unrealistic pH, found that levels in water standing in galvanized steel pipes were typically less than the MCL of 0.01 mg/liter (40 CFR 141.11[b]). Even if the concentration of selenium were consistently 0.01 mg/liter over the long term, this amount would constitute a small fraction of the daily dietary intake of this mineral (NAS, 1980).

(3) Toluene. Unlike iron and selenium, toluene plays no essential role in nutrition. It is clearly toxic to humans at high doses (Committee on Alkyl Benzene Derivatives, 1981). The concentrations of toluene detected in sampling protocols reviewed in Chapter IV-A were consistently less than 1 ppb (1 ug/liter). Such concentrations are 5 orders of magnitude (100,000 times) lower than the EPA ambient water quality criterion for toluene (14.3 mg/liter), which was established to protect human health against toxic effects not only from drinking water, but also from eating contaminated fish (45 Fed. Reg. 79340; November 28, 1980). More recently, EPA's Office of Drinking Water has calculated a chronic "Suggested No Adverse Response Level" (SNARL) of 343 ppb for toluene (EPA, 1982). SRI has recalculated this chronic SNARL to be 34.5 ppb, still nearly two orders of magnitude greater than the concentrations described in Chapter IV-A. EPA's Science Advisory Board has completed an extensive review of toluene's toxicologic properties and concluded that this substance cannot be currently considered mutagenic, carcinogenic, or teratogenic (McClellan, 1982). However, toluene is being tested for carcinogenicity under the auspices of the National Toxicology Program and may warrant more extensive discussion if that bioassay should prove to be positive.

section on risk assessment. For the chemicals reviewed in this chapter, the analysis has followed the following sequence:

(1) Is there an applicable state or federal standard pertaining to the presence of the chemical of interest in the water supply? If so, the expected range of concentrations in the water supply has been compared with the relevant standard, assuming the latter has been calculated from chronic toxicity data.

(2) If there is no applicable standard, is there a "Suggested No Adverse Response Level" (SNARL) for chronic effects calculated by the National Academy of Sciences or the U.S. Environmental Protection Agency's Office of Drinking Water? If so, the expected range of concentrations in the water supply has been compared with such SNARL(s).

(3) If there is neither standard nor SNARL, are the data adequate to calculate a chronic SNARL? If so, such calculations have been done. If not, the risk assessment is qualitative only, except for solvent cements and carcinogens.

(4) In the case of the four solvent cements for which no chronic SNARLs or quantitative risk estimates have been calculated, a procedure devised by the Department of Health Services, based on occupational exposure limits, has been followed. In this procedure occupational exposure standards are used to calculate proposed acceptable concentrations and proposed maximum short-term acceptable concentrations. These values are compared to solvent concentrations in water carried by plastic pipes in order to estimate health risk.

(5) For recognized animal carcinogens, results of low-dose risk estimates calculated by EPA's Carcinogen Assessment Group using the multistage model of Crump and Watson (1979) are presented. A range of plausible risks were calculated under assumptions of different leachate concentrations.

3. Risk Assessment: Health Effects of Leachates

The risks of toxicity to consumers exposed to leachates in potable water depend on several factors:

(1) The toxic effects of such chemical leachates
(2) The doses at which such effects have been observed
(3) The concentrations of such leachates that have been found or can reasonably be expected to be found in drinking water, at different times in the life of the pipes
(4) The ratio of (2) and (3)
(5) Whether the toxic effects of interest are reversible or irreversible
(6) Whether the effects of exposure are likely to be cumulative, and if so, the cumulative dose.

(7) Whether the toxic effects are thought to be governed by a threshold.

Risk estimation concerning potential health effects from exposure to chemicals has traditionally involved different methods for dealing with carcinogens and noncarcinogens, principally because carcinogenesis is postulated, at least for genotoxic agents, to be a nonthreshold process (NAS, 1977, 1980). For certain chemicals, mutagenesis or other genetic damage may also be considered a nonthreshold process, but genetic toxicology has not yet evolved to a point where human risks of genetic damage and potential disease can be predicted from the results of *in vitro* or *in vivo* assays positive for effects on DNA (Streisinger, 1983).

Noncarcinogenic effects are generally considered to display threshold behavior. Thus, the usual approach to assessing risks of chronic health effects from exposure to a chemical not known to be carcinogenic is to ascertain a "no-observed-effect-level" of long-term exposure and to apply an appropriate safety or uncertainty factor (NAS, 1980). Such calculations essentially involve linear extrapolation to levels of exposures below those where no adverse effects have been observed. Suggested-no-adverse-response-levels ("SNARLs") are the result of such estimates, and normal consumption of drinking water contaminated up to the SNARL should not result in toxicity to humans; however, total safety cannot be vouchsafed (NAS, 1980). Criticisms of such an approach have focused primarily on the choice of dose levels and on small sample sizes (so that the pertinent study may have inadequate statistical power to detect an effect). Another problem encountered with this approach is that there may be no adequate data from which to calculate a chronic SNARL. (See, e.g., evaluations for cyclohexanone, dimethylformamide, methyl ethyl ketone, and tetrahydrofuran.)

While this approach has potential shortcomings, SRI has utilized it for substances not known to be carcinogens because it does incorporate substantial safety factors and because it is utilized by regulatory agencies with responsibilities for protecting public health from contaminants in drinking water (e.g., EPA, California Department of Health Services) and by

the National Academy of Sciences in its reports mandated by the Safe Drinking Water Act (see, e.g., NAS, 1977, 1980, 1982).

Risk estimation for carcinogens in drinking water is more complex. Neither the National Academy of Sciences nor EPA calculates SNARLs for substances demonstrated to be carcinogenic in animals or humans. The basis for this is that the toxicity of carcinogens may involve effects that are irreversible and self-propagating after the exposure has ceased. Furthermore, as noted earlier, carcinogenesis due to exogenous agents has been, and for regulatory purposes still is, considered to be a nonthreshold process. Therefore, to estimate incremental cancer risks from daily exposure to such agents, the general approach is to calculate plausible estimates of upper lifetime risks of 10^{-5}, 10^{-6}, and 10^{-7}. In other words, exposure levels are calculated that would correspond to an increase in an individual's lifetime risk of 10^{-5} (one in one hundred thousand), 10^{-6} (one in one million), and 10^{-7} (one in ten million). In general, such calculations are made using mathematical models that are based principally on the results of animal bioassays, although epidemiologic data are preferable when available. The models currently in use have been extensively reviewed by the California Department of Health Services (October 1982).

The scope and budget for this project did not permit an independent reevaluation of the risks posed by carcinogens in drinking water. In general, the calculations performed by EPA in developing its water quality criteria have been relied upon. These estimates were formulated using the linearized multistage model (Crump and Watson, 1979). For some carcinogens, notably those whose carcinogenicity is probably modulated by activated metabolites, this model may overstate risk (Hoel et al., 1983). However, regulatory agencies dealing with such a potentially serious outcome as cancer generally prefer to err on the side of conservatism. This preference in part explains the widespread use of the multistage model. The National Drinking Water Advisory Council has advocated that this model be used to set recommended MCLs for carcinogens (Neal, undated).

In evaluating the risks posed by carcinogens, there is always a question of what constitutes a significant incremental risk of cancer. Federal agencies have in the past considered an individual risk level of 10^{-6} as a measure of acceptability of risk. This corresponds to one additional case of cancer occurring in a population of one million people. The California Department of Health Services has also recently proposed a 10^{-6} level as one measure of determining significant risk (December 1982). The choice of such a risk level is precautionary in nature, and is based on both the large degree of uncertainty involved in quantitative risk assessment and on the possibility of synergistic (and potentiating) interactions among carcinogens (and other chemicals not carcinogenic in themselves). Where the potentially exposed population is very large, even a risk level of 10^{-6} may lead to additional cases of cancer. At the federal level, the use of 10^{-6} as a target risk level may be changing. The National Drinking Water Advisory Council has recommended the use of 10^{-5} for at least some carcinogens, as has EPA in some cases (Neal, undated; Marshall, 1982). Although not formulated in official policy statements, California regulatory agencies have used a 10^{-6} target risk level for the general population.*

California's population in 1980 was about 24 million. In the early 21st century, it will probably exceed 30 million. If the projections of Section II-C hold true, by 2010, more than 8 million people could be drinking from plastic pipe. At that time, an average lifetime risk of 10^{-6}--or an annual risk of about 1.5×10^{-8}--would imply that one additional case of cancer might be expected about every 8 years in California. Some population groups (for example, people who move into newly

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*The choice of 10^{-5}, 10^{-6}, or 10^{-7} as a target risk level could have a significant impact on the judgment as to the acceptability of pipes that may leach small quantities of carcinogens. Of these alternatives 10^{-5} is least protective and 10^{-7} most protective of public health. Historically, however, 10^{-6} has been most commonly used by regulatory agencies. It should be recognized that these numbers can take on an unwarranted talismanic significance, whereas in actuality they represent crude approximations of plausible risk limits.

plumbed houses in infancy) may be at higher risk, whereas others would be at lower risk.

a. Sources of Uncertainty in Risk Assessment

As noted earlier, there are several sources of uncertainty in assessing the risk of low level contamination of drinking water by plumbing pipe. The following paragraphs discuss the principal sources of uncertainty that are relevant to this document.

i. Leachate Concentrations Over Time--Critical variables in risk assessment are the identities and quantities of chemicals that people are likely to ingest over time. As is evident in Chapter IV-A, such information is not readily available from earlier sampling protocols, most of which have suffered from significant methodologic flaws. One can make predictions about the leaching behavior of certain substances; for example, for chemicals whose leaching into water is equilibrium-limited rather than diffusion-limited, concentrations should decline exponentially with increasing numbers of equilibrium dwell-time flushings. For other substances whose leaching behavior may be diffusion-limited or even more complex (see Chapter IV-A), such predictions cannot be made. In the absence of better data, such substances can be treated in at least two ways. One is a worst-case analysis, in which an estimated (high) concentration of the substance in the pipe is presumed to completely leach from the pipe at a constant rate for 20 years. Alternatively, one can assume a faster leaching rate and calculate the potential health risks for a shorter, but higher, level of exposure. Both of these approaches have been used here. Better, long-term leaching data would facilitate this analysis.

ii. Exposure--One does not know the quantities of tap water actually consumed by individuals; thus, simplifying assumptions have been necessary. For purposes of risk calculations, SRI has assumed that all of an individual's daily fluid intake has come from tap water. For adults, this quantity is assumed to be 2 liters/day and for infants and young children, 1 liter/day. One further assumption has been that oral ingestion is the only

significant source of human exposure to chemical leachates. Potential absorption of some chemicals present at ppb quantities through intact skin (e.g., in hand-washing or bathing) has been historically assumed to be insignificant. (The validity of such an assumption may be altered by the results of an ongoing EPA investigation of this issue [Maibach, 1983].)

iii. The Nature of Chemical Effects--Risk assessment is subject to generic uncertainties due to an incomplete data base. For example, some chemical leachates have not been studied for carcinogenicity (e.g., methyl ethyl ketone) or the investigations for carcinogenicity have been inadequate (e.g., dimethyl tin bis isooctylthioglycolate). In cases like these, the chemicals of interest have been treated as if they do not possess the capability of causing cancer. A substance may have been found to cause tumors in animals only when administered in a form or route different from that to be expected in drinking water (e.g., intratesticular injection of zinc in roosters). Unless the route of exposure is oral (including gavage) or by inhalation, no quantitative cancer risk assessment has been undertaken. It should be noted that chemicals demonstrated to be carcinogenic (or toxic in some other respect) by one route of exposure may or may not possess this capability if exposure takes place by another route (Tobin et al., 1982; Theiss, 1982).

iv. Low-Dose and Interspecies Extrapolation--Extrapolating the occurrence of toxic effects from high to low doses and from animals to humans are fundamental concepts in toxicology. These procedures assume a similarity of biological effects (at low doses and in humans) that has generated substantial controversy, particularly with respect to risk estimates for carcinogens acting through "epigenetic" mechanisms (see, e.g., Stott et al., 1981; Hoel et al., 1983; California Department of Health Services, October 1982; Munro and Krewski, 1981; NIEHS, 1976). It is beyond the scope of this document to present an extensive discussion of these issues. Suffice it to say that such extrapolations are necessary for quantitative risk assessment, even though there are major uncertainties involved in performing such calculations.

v. Chemical Interations--Chemical leachates may interact with each other and with other chemicals in drinking water or food in ways that may result in additive, synergistic, potentiating, or antagonistic effects on toxicity. Examples of all of these types of interactions are known to occur but, in general, such information is not available for the variety of interactions that chemical leachates might take part in. The area of greatest potential concern in this respect is carcinogenesis. In the absence of information to the contrary, SRI has treated all such effects (where quantifiable) as if they were additive (California Department of Health Services, October 1982).

b. Background Information--Health Effects

i. Effects on Genes and Chromosomes--Chemicals that can affect the structure of genetic material are classified as mutagens (causing mutations--changes in DNA sequences) or clastogens (causing disruption of chromosomal architecture). Genetic effects may be passed on to an affected cell's progeny, and may result in cancer (non-germ cell mutations) or reduced fertility or birth defects (affected cells are germ cells). Many, but not all, chemicals shown to cause mutations in short-term in vitro assays are recognized carcinogens (McCann and Ames, 1977; Ames, 1979; Rosenkranz and Poirier, 1979). As noted previously, recent work indicates that in some cases, changing a single DNA base-pair (the basic building block of genes) may be sufficient to cause cancer (Anonymous, 1982). Others believe that, since many tumors display disturbed chromosomal structures, clastogenic effects are more important in the development of cancer (Cairns, 1981). In any case, damage to genetic material is likely to be deleterious. However, the state-of-the-art of genetic toxicology is not yet at a stage where short-term tests for genotoxicity can be used to quantitatively predict human genetic risk (Streisinger, 1983; Bartsch et al., 1982).

ii. Cancer--Of the chronic diseases at issue in this Environmental Review, cancer seems to be the source of greatest concern. One out of four persons in California will develop cancer, which is the second leading cause

of death in California and the United States. Cancer is not one disease, but rather a group of diseases of multiple causes, all characterized by an uncontrolled proliferation of cells which can lead to the death of the host. Despite billions of dollars spent on cancer research during the past decade, the causal and developmental mechanisms of cancer remain obscure.

The development of cancer in a particular individual depends on both internal and external factors. The former include genetic constitution, general health, age, sex, race, nutrition, hormones and competence of the immune system. External factors include exposure to carcinogens--cancer-causing agents--either voluntarily (e.g., cigarette smoking) or involuntarily (e.g., exposures in the workplace or general environment). The rest of this section will focus on the role of carcinogens in cancer causation. It should be borne in mind that the following discussion is an extreme simplication of a highly complex topic.

It has been estimated that up to 90 percent of all cancer is caused by "environmental" agents (Higgenson, 1976). The majority of cancer cases has been attributed to so-called voluntary "lifestyle" factors, such as cigarette smoking, diet, exposure to sunlight, and consumption of alcohol (Doll and Peto, 1980; Miller, 1981). Such estimates are controversial (Epstein, 1981), but even assuming that only a few percent of cancers are attributable to environmental contaminants, several thousand cancer cases per year in California may be attributable to such contaminants. Furthermore, dividing causes into lifestyle and nonlifestyle categories creates the appearance of greater certainty about the development of cancer than currently exists: people are exposed to many carcinogens and "promoters" (see below) in ambient air and water and the workplace, as well as through such "lifestyle" factors as smoking and high-fat diets.

There are several sources of evidence for identification of carcinogens: human studies, animal models, short-term tests, and structure activity analyses. (The latter two sources are considered supportive evidence only, and cannot prove or disprove mammalian carcinogenicity.) Although epidemiologic investigations of human populations exposed to

putative carcinogens provide the most direct evidence of carcinogenicity, a variety of usually unavoidable structural problems limits the utility of such studies. Such problems include small study population sizes, multiple potentially confounding exposures, fragmentary or nonexistent documentation of exposure to the agent(s) in question, difficulty in identifying an appropriate control or reference population, and an inability to detect increases in risk less than 50-100 percent. When good epidemiologic data are available, they clearly represent the best evidence. However, most commercial chemicals found to be carcinogenic in animals have not been and probably cannot be investigated by epidemiologic methods, because of the methodological and statistical limitations of such methods (Karstadt, 1981).

Evidence of a substance's ability to cause cancer in animals represents the next best level of evidence that such substance poses a risk of cancer to humans. All known human carcinogens, with the possible exception of arsenic, have been reported to be carcinogenic in one or more animal species (Tomatis, 1979; IARC, 1982b). For reasons noted above, the reverse cannot be demonstrated or disproved. Carcinogenicity can generally be demonstrated in more than one animal species under adequate experimental protocols (Ames et al., 1981). The strong correlations between studies of human and animal carcinogens have led every federal and California agency concerned with regulation of carcinogens, as well as national and international expert committees, to regard animal carcinogens as potential human carcinogens.

An in-depth exposition of current theories about causes and mechanisms of carcinogenesis is beyond the scope of this document: there are several recent articles and books on the subject (Farber, 1981; Yuspa and Harris, 1982; Berenblum, 1979; California Department of Health Services, 1982; Stott et al., 1981; Weisburger and Williams, 1981; Sontag, 1981; OTA, 1981; Becker, 1981).

The evolution of cancer is a gradual process thought to occur in multiple stages, some of which are irreversible and others probably reversible (Farber, 1981; Berenblum, 1979; Peto, 1977). The irreversible change involves an alteration of DNA, designated "initiation," which can be

passed on to the cell's progeny. Initiated cells may then be subject to the action of "promoters" (also known as "modifiers" or "late stage carcinogens"), which may eventually result in the development of a tumor, although the mechanisms of action are not known. While some substances are known to have only promoter activity (e.g., phenobarbital), others may act as initiators as well as promoters and are known as "complete carcinogens." However, this well-accepted theory cannot explain the apparent complete carcinogenicity of chemicals that do not bind to or directly change DNA.

Whether a chemical or its metabolites can bind to and alter DNA--i.e., whether it is genotoxic--has been proposed as a basis for classification of carcinogens (Weisburger and Williams, 1981). Dividing carcinogens into genotoxic versus epigenetic or nongenotoxic categories may have major implications for risk assessment and regulation (Kolbye, 1982). Promoters have been designated to act by unknown epigenetic mechanisms, though there is evidence that they can affect DNA indirectly (Marx, 1983; Parke, 1982). There is by no means, however, a consensus among scientists and regulators about the appropriateness of such a classification scheme for regulatory purposes and, considering that this bifurcated approach has not been officially adopted by any agency, it would be premature to suggest that DCHD subscribe to this policy (see, e.g., Toddhunter, 1983; Weinstein, 1983, Albert, 1983).

iii. Effects on Reproduction--Exposure to chemical agents has been associated with a variety of adverse reproductive outcomes in humans, including reduced fertility, miscarriages, stillbirths, birth defects, low-birth-weight infants, and effects on later physical and mental development of children who initially appear normal (Council on Environmental Quality, 1981b). Most of the medical literature concerning chemical effects on reproduction has focused on birth defects rather than other types of reproductive impairment. Other outcomes have not been subject to as much study as the development of birth defects and will be given less emphasis here. The overall influence of identifiable causative factors in human birth defects has been estimated to include genetic damage (about 23-35 percent) and environmental agents of all kinds, including

chemicals, radiation, drugs, alcohol, infections and so forth (about 7-11 percent). The remainder (about two-thirds) have no identified cause (Wilson, 1973).

Substances capable of causing physical defects in the embryo or fetus are called teratogens. Most chemicals identified as human teratogens have involved situations where there has been high-level maternal exposure to the agent(s) in question, usually in a medical, occupational or "lifestyle" (smoking, consumption of alcohol) setting. This does not mean that such effects (or other reproductive impairments) do not occur at lower doses or in other contexts. Rather, these have been situations when there has been well-documented exposure to the agents in question and the agents have been potent enough to be detected by epidemiologic methods. For chemicals in the general environment, documentation of such relatively "pure" exposures to putative teratogens is not available. In addition, the timing of exposure may affect the outcome as well. Table IV-30B lists possible effects according to the time of parental exposure.

Because of the difficulties in identifying teratogens by epidemiologic methods, there has been considerable safety testing in animal studies. It cannot be assumed that a positive or negative teratogenic response in animals will produce a similar response in humans. Interspecies variability in physiology, placental structure, gestational sequence, background incidence and susceptibility to chemical substances needs to be taken into account (U.S. Food and Drug Administration, 1980). Nevertheless, many recognized human teratogens have been reported to have similar effects in at least one animal species. Other adverse reproductive outcomes, such as spontaneous abortions and reduced fertility, have also been shown to occur in animal models (Council on Environmental Quality, 1981b). An adverse effect on reproduction in animals may therefore result in a similar effect in humans. However, because of the interspecies differences noted earlier, no single animal study can be said to be predictive of human outcomes. Because of these difficulties in extrapolating from animals to man, no methodology for low-dose risk assessment has been developed. Furthermore, with the exception of genetic damage to the germ cells, most adverse

Table IV-30B

POTENTIAL CHEMICAL EFFECTS ON REPRODUCTION AND PERINATAL DEVELOPMENT

Before Conception	During Pregnancy	After Birth
Menstrual disorders	Maternal	Abnormal development due to chemicals transmitted in breast milk or brought home on parents' workclothes.
Male potency and libido	Enhanced toxicity	
Reduced fertility	Toxemia	
Sterility	Miscarriage	
Germ cell mutation	Fetal	
	Death	
	Malformation	
	Functional deficit	
	Biochemical change	
	Growth retardation	
	Mutations	
	Cancer	

Source: Adapted from Sullivan and Barlow, 1979.

reproductive effects are thought to be governed by thresholds, so that very low doses (in the ppb range) would be unlikely to produce birth defects.

iv. Effects on the Nervous System--Chemicals capable of exerting toxic effects on the nervous system are known as neurotoxins. Such substances may affect the central nervous system (CNS), the peripheral nerves, or both. It is common for different regions of the nervous system and different cell types to display selective vulnerability to various neurotoxins. The effect may vary with the dose absorbed--for example, a single exposure to a high concentration of hexane produces giddiness, mild euphoria and narcosis, while repeated exposure to lower levels may produce toxic effects principally on the peripheral nerves (Spencer et al., 1980).

Neurotoxic effects of chemicals are usually characterized by a steep dose-response curve, the boundaries of which do not vary widely among individuals. Therefore persons exposed to low concentrations of neurotoxins are unlikely to develop "idiosyncratic" symptoms characteristic of damage seen at higher doses (Schaumberg et al., 1981). For this reason, where the drinking water concentration of chemical leachates is substantially lower than known neurotoxic levels, it is not anticipated that individuals will develop gross neurologic dysfunction.

However, subclinical disease of the nervous system may result from repeated exposure to low doses of neurotoxins, particularly to those capable of bioaccumulation. As an example of this phenomenon is the effect that chronic low level exposure to lead can have on the mental and behavioral development of children. Also to be considered is the possibility of potentiation of the effect of a subthreshold dose of a neurotoxin. For example, the weakly neurotoxic compound methyl ethyl ketone has been reported to potentiate the neurotoxic effect of n-hexane (Schaumberg et al., 1981). Such potentiating effects are of primary concern in the occupational setting.

4. Evaluation of Specific Leachates

Summaries of the toxic effects of selected chemical leachates are presented in this section according to the following format:

Exposure
Absorption and Metabolism
Acute Toxicity*
Chronic Toxicity
Effects on Genes and Chromosomes
Cancer
Effects on Reproduction
Evaluation

The concentrations in pipe water listed under "Exposure" are in some cases different from those listed in the prior draft of the public health section because of the more rigorous scrutiny applied to leaching studies since the distribution of the earlier draft. More extensive discussions of the toxicities of these substances can be found in Appendix D. The evaluation for each of the chemicals includes relevant drinking water standards, SNARLs or their equivalents for substances not known to be carcinogens or for certain metals, certain forms of which are carcinogens, and plausible limits for 10^{-5} to 10^{-7} incremental lifetime cancer risks for carcinogens. In each evaluation, there is a judgment as to whether the substance is likely to pose a significant risk to health, given current knowledge. Such assessments are subject to change in response to results of further testing. A summary evaluation for each type of pipe is presented at the end of Chapter IV-B.

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*Included only for substances of potential significance in occupational exposures: cyclohexanone, dimethylformamide, lead, methyl ethyl ketone, and tetrahydrofuran.

5. Substances Associate with CPVC and PVC Pipes

a. Dimethylformamide

Exposure--NIOSH (1980) estimates that 69,000 workers in 25 major industries are exposed annually to dimethylformamide (DMF). OSHA (1981) has set a permissible workplace exposure limit of 10 ppm (8-hour time-weighted average), but workplace concentrations ranging from less than 10 ppm to more than 200 ppm have been reported. The exposures of plumbers to DMF while installing plastic pipe have ranged from "undetectable" to less than 0.5 ppm. Consumers may be exposed to DMF as contained in solvents, degreasers, and adhesives (NIOSH, 1981). In a study of organic solvents, DMF ranked twelfth out of 34 solvents on a consumer exposure index (1979). DMF has been detected in effluent from industrial sources and domestic sewage treatment plants. DMF is a contituent of adhesives for CPVC plastic pipe and has been found to leach into water carried by such pipe. Concentrations reported in Chapter IV-A ranged from "nondetectable" to 7.1 ppm.

Absorption and Metabolism--Dimethylformamide is effectively absorbed by the lungs, skin and gastrointestinal tract. When human subjects were exposed to 8 ppm DMF in air for 6 hours on 5 consecutive days, most of the absorbed dose was eliminated within 24 hours. DMF is metabolized by humans to N-methyl formamide (NMF) and formamide, two suspected teratogens (Maham, 1977; NIOSH, 1981). The presence of NMF in the urine is a sensitive indicator of exposure to DMF, even to concentrations lower than 10 ppm, the OSHA permissible exposure limit (Krivanek et al., 1978; Maxfield et al., 1975). Alcohol consumption can retard metabolism of DMF, and DMF can inhibit alcohol metabolism. Alcohol intolerance (a reaction similar to that induced by the drug "Antabuse") has been noted in workers exposed to DMF (Chivers, 1978). The interaction of DMF and alcohol indicates that they are probably metabolized by the same enzymes.

Acute Toxicity--Dimethylformamide is moderately irritating to the eyes, skin, and respiratory tract. Repeated contact with the liquid may defat the skin and cause dermatitis (Proctor and Hughes, 1978). Exposure to high

concentrations can produce dizziness and headache (Wink, 1972). DMF is toxic to the liver and highly irritating to the gastrointestinal tract (Massman, 1956). Single doses administered to laboratory rats and hamsters have produced liver damage (Ungar et al., 1976; Mathew et al., 1980) and functional changes in the central nervous system (Weiss and Orzel, 1967).

Chronic Toxicity--Workers exposed to atmospheric concentrations of DMF of 20-35 ppm for 32 weeks complained of nausea, vomiting and abdominal pain; liver enlargement was detected in some cases (Proctor and Hughes, 1978). Several other cases of gastrointestinal disorders due to DMF exposure have been reported. No epidemiologic investigations of DMF-exposed populations have been reported.

DMF has produced liver and kidney damage and changes in cardiac function when administered to laboratory animals via inhalation, skin application, or oral intubation. Dogs, rabbits, guinea pigs, rats, and mice were exposed to 23 ppm DMF for 5 1/2 hours followed by a 1/2 hour exposure to 426 ppm for 58 weekdays. Functional effects on the liver, pancreas, spleen, kidneys, adrenals, and thymus glands of all animals were seen. Degenerative changes in the heart and cardiovascular function were seen in dogs (Clayton et al., 1963).

Effects on Genes and Chromosomes--No effects on genes and chromosomes in humans have been reported. DMF has been studied in several test systems designed to detect damage to genes and chromosomes. No damage was reported in nine out of ten tests. Recently DMF was tested in five additional tests under the National Toxicology Program; results were not available for review as this report was written.

Cancer--There have been no studies of the cancer-causing potential of DMF in humans. One study in rats reported increased incidence of liver tumors, but the study was poorly designed and results were not statistically significant. A 2-year study designed to detect the effect of long-term inhalation of DMF in rats and mice is currently being conducted, but results will not be available for at least a year.

Many studies with tumor cell cultures have shown an "anticancer" effect; one test showed the ability of DMF to transform normal cells to malignant cells.

Effects on Reproduction--One undocumented report of increased incidence of abortion in Soviet female workers exposed to DMF was reviewed, but no careful investigations of the possible effects of DMF on human reproduction have been conducted.

DMF has been shown to cross the placenta to the fetal blood circulation in rats. DMF is metabolized in humans, rats, and dogs to two compounds that have caused malformations in offspring of female laboratory animals exposed during the gestation period. Results of animal tests are conflicting. In the majority of studies, DMF has not caused gross malformations, but some decrease in implantations, depression of fetal weights, and variations in development have been seen. The available data indicate that DMF does not cause serious birth defects, but most investigations have not been conducted with sufficiently large numbers of animals to permit certain conclusions.

Evaluation--There is no federal drinking water standard applicable to DMF. There are no adequate chronic toxicity data in animals or humans from which a chronic snarl can be calculated (NAS, 1982). In the absence of such data, no recommendation can be made for a concentration of DMF in drinking water that will ensure long-term safety. However, the California Department of Health Services has proposed both long- and short-term maximum acceptable concentrations of DMF in drinking water, derived from the OSHA permissible exposure limit (California Department of Health Services, 1980). The procedure, which is outlined below, results in the derivation of proposed maximum acceptable concentrations (PMACs) based on several absorption estimates and proposed short-term acceptable concentrations (PSTMACs).

PROCEDURE FOR DERIVING PROPOSED MAXIMUM ACCEPTABLE CONCENTRATONS

1. Current or recommended PEL (TWA) (mg/m^3)

multiplied by

2. Volume of air inhaled by worker per work day (mg/day)

multiplied by

3. Work week (assume 40 hours) or 5 days

multiplied by

4. Retention factor

equals

5. Amount of substance absorbed by worker per week (mg)

This, divided by

6. Weight of worker (assume 70 kg)

equals

7. Dose on body weight basis (mg/kg)

8. Seven day week.

equals

9. Daily dose on body weight basis (mg/kg)

multiplied by

10. Child or newborn body weight

divided by

11. Safety factor (100)

equals

12. Proposed maximum dose for child or newborn (mg)

The dose, divided by

13. Volume of water consumed per day (assume one liter)

equals

14. Proposed maximum acceptable concentration in water (mg/l)

CALCULATIONS FOR PELs (PMACs) FOR PLASTIC PIPE ADHESIVE SOLVENTS IN TAP WATER

	MEK	THF	CYCLO	DMF
1. OSHA PEL mg/m^3	590	590	200	30
2. x 9.6 m^3/d	566.4	566.4	1920	288
3. x 5 d/w	28320	28320	9600	1440
4. x 0.5 R (Retention)	14160	14160	4800	720
6. . 70 kg	202.3	202.3	68.5	10.3
8. . 7 d/w	28.9	28.9	9.8	1.5
10. x 25 kg	722.4	722.4	244.9	36.7
11. . 100 Safety Factor	7.22	7.22	2.44	0.367
13. . 0.2 L/d	36	36	12	1.8
14. PMAC (R = 0.5) mg/L	36	36	12	1.8
(R = 0.7) mg/L	50	50	17	2.5
(R = 0.3) mg/L	22	22	7	1.1
15. PSTMAC (R = 0.5) mg/L	360	360	120	3.6

In order to account for potential additive effects, the Department proposed using a formula analogous to the ACGIH (1982) method for calculating workplace threshold limit values (TLVs) for mixed exposures:

$$\frac{C_1}{TLV_1} + \frac{C_2}{TLV_2} + \frac{C_3}{TLV_3} = 1$$

where C = concentration of the compound in air and TLV = the respective threshold limit value. If the sum is less than or equal to one, the exposure is considered acceptable; if the sum is greater than one, the exposure is considered excessive. The same procedure can be followed substituting concentrations in water and PMACs for airborne concentrations and TLVs.

The procedure developed by the Department of Health Services is one of several semi-quantitative models that could be used to provide a rough estimate of risk in the absence of appropriate data from human or animal studies. The procedure has been followed in this report because it embodies several features that should be included in such a model: an indicator of toxicity (TLV or PEL), absorption estimates (R), a means by which acceptable concentrations of contaminants in water can be calculated for individuals of different weights with different daily water intakes, and a safety factor. The results produced by the model are probably conservative, which is appropriate given the lack of chronic toxicity data.

Using this method, the acceptable limits for DMF can be calculated as described below:

PMAC (R = 0.5) mg/L	1.8
(R = 0.7) mg/L	2.5
(R = 0.3) mg/L	1.1
PSTMAC (R = 0.5) mg/L	3.6,

where R = the portion of the inhaled dose that is absorbed.

The data on leaching of DMF from plastic piping as presented in Chapter IV-A vary widely with test protocols. The static test with a 2-week dwell period showed a low concentration of DMF, with nondetectable levels in subsequent static tests (see Table IV-6). If these data were used to predict DMF exposure, concentrations in water after adequate flushing would be well below the calculated PMACs and PSTMAc. If, however, values from kinetic tests were used as predictors, concentrations higher than the PMAC and PSTMAC would result and a significant risk to public health might be incurred (see Table IV-9). It is impossible on the basis of the information available to decide which data are more appropriate for purposes of risk estimation. It is hoped that additional testing will resolve the dilemma.

b. Tetrahydrofuran

Exposure--NIOSH (1980) estimates that approximately 95,000 workers are exposed annually to tetrahydrofuran (THF). Both the current OSHA Permissible Exposure Limit and ACGIH Threshold Limit Value are an 8-hour time-weighted average of 200 ppm (OSHA, 1981; ACGIH, 1982). THF has good warning properties; its odor is detectable at 25-50 ppm, well below the recommendations for workplace exposures (du Pont, 1977). Tetrahydrofuran is a constituent of adhesives for use with plastic pipe. Concentrations ranging from trace amounts to 79 ppm were detected in the work environment of plumbers installing plastic pipe (Halts, 1980). Tetrahydrofuran has also been identified as a leachate from CPVC plastic pipe: concentrations reported in Chapter IV-A range from 2.7 ppm to 375 ppm.

Absorption and Metabolism--No information on the metabolism of THF was available. Because the compound is so volatile, most of an inhaled dose would be expected to be eliminated in the expired air.

Acute Toxicity--THF is a mild irritant of the eyes skin and mucus membranes; repeated contact with the skin may cause dermatitis. Exposure to concentrations above 200 ppm may produce nausea, dizziness, and headache but these symptoms are readily reversible in fresh air (AIHI, 1959). Two reports of injuries due to exposure to THF were reviewed but, in both cases,

individuals had been exposed to other compounds and no causal connections with THF could be established. Gosselin et al. (1976) estimate that the lethal oral dose for humans is 50-500 mg/kg.

In laboratory animals, concentrations above 3,000 ppm produced upper respiratory tract irritation after exposures of 8 hours per day for 20 days. Concentrations of approximately 60,000 ppm were required to induce narcosis (sleep) in cats, rabbits, rats, and mice.

Chronic Toxicity--No chronic toxicity in humans or animals attributable to THF exposure has been reported. Dogs exposed to atmospheric concentrations of 336 ppm or 2,100 ppm for 6 hours per day, 5 days per week over a 12-week period exhibited decreased blood pressure, but no other effects were observed (du Pont, 1977). A prechronic (90-day) test of THF has recently been completed under the NCI/NTP carcinogenesis bioassay program. Results were requested but have not yet been received.

Effects on Genes and Chromosomes--No tests for effects on genes and chromosomes have been reported.

Cancer--No studies of cancer in humans populations exposed to THF have been reported. Although a prechronic test was conducted under the National Toxicology Program bioassay program, no chronic test has been scheduled. The rationale for this decision has not been published, but it is probable that results of the prechronic test did not suggest cancer-causing potential (Juodeika, 1983).

Effects on Reproduction--No effects of THF on human or animal reproduction have been reported.

Evaluation--There is no federal drinking water standard applicable to THF. There are no adequate chronic toxicity data in animals or humans from which a chronic snarl can be calculated (NAS, 1982). In the absence of such data, no recommendation can be made for a concentration of THF in drinking water that will ensure long-term safety. However, the California Department

of Health Services has proposed both long- and short-term maximum acceptable concentrations of THF in drinking water, derived from OSHA permissible exposure limit (California Department of Health Services, 1980). The procedure which is outlined in the evaluation for DMF, results in the derivation of proposed maximum acceptable concentrations (PMACs) based on several absorption estimates and proposed short-term acceptable concentrations (PSTMACs).

In order to account for potential additive effects, the Department proposed using a formula analogous to the ACGIH (1982) method for calculating workplace threshold limit values (TLVs) for mixed exposures:

$$\frac{C_1}{TLV_1} + \frac{C_2}{TLV_2} + \frac{C_3}{TLV_3} = 1$$

where C = concentration of the compound in air and TLV = the respective threshold limit value. If the sum is less than or equal to one, the exposure is considered acceptable; if the sum is greater than one, the exposure is considered excessive. The same procedure can be followed substituting concentrations in water and PMACs for airborne concentrations and TLVs.

The procedure developed by the Department of Health Services is one of several semi-quantitative model that could be used to provide a rough estimate of risk in the absence of appropriate data from human or animal studies. The procedure has been followed in this report because it embodies several features that should be included in such a model: an indicator of toxicity (TLV or PEL), absorption estimates (R), a means by which acceptable concentrations of contaminants in water can be calculated for individuals of different weights with different daily water intakes, and a safety factor. The results produced by the model are probably conservative, which is appropriate given the lack of chronic toxicity data.

Using this method, the acceptable limits for THF can be calculated as described below:

PMAC (R = 0.5) mg/L	36
(R = 0.7) mg/L	50
(R = 0.3) mg/L	22
PSTMAC (R = 0.5) mg/L	360,

where R = the portion of the inhaled dose that is absorbed.

The data on leachability of THF from plastic piping as presented in Chapter IV-A (see Tables IV-6 and IV-9) indicate that all concentrations in water were below the PSTMAC, and only concentrations found in a static test after a 2-week dwell time exceeded any of the PMAC values. These results suggest that adequate flushing procedures prior to use would reduce THF concentrations in water from plastic plumbing pipe to levels that would probably not pose a significant risk to public health. Preliminary calculations using the leaching data in Chapter IV-A also suggest that excessive mixed exposures to the three solvents with similar biologic effects (MEK, THF, and cyclohexanone) would be improbable if adequate flushing were performed.

c. Cyclohexanone

Exposure--NIOSH (1980) estimates that approximately 10,000 workers are exposed annually to cyclohexanone. The current ACGIH-recommended workplace TLV is an 8-hour time-weighted average exposure of 25 ppm; the OSHA permissible exposure limit is an 8-hour time-weighted average concentration of 50 ppm. No cyclohexanone was detected in breathing-zone samples of plumbers installing plastic pipe (California Department of Health Services, 1980). Cyclohexanone is a component of cements for CPVC plastic pipe and leaches into the water carried through these systems. Concentrations reported in Chapter IV-A range from 0.2 ppm to 13.0 ppm.

Absorption and Metabolism--Inhaled cyclohexanone may be excreted unchanged in the expired breath or reduced to cyclohexanol and glucuronidated in the liver. Cyclohexanol glucuronide is excreted in the urine; other metabolites may be eliminated in the feces (Greener et al., 1982).

Acute Toxicity--Cyclohexanone is considered to be moderately toxic by dermal, oral, and inhalation exposure. It has mild narcotic properties (Sax, 1979). At atmospheric concentrations equal to and greater than 75 ppm, it is irritating to the eyes and respiratory tract. Repeated skin contact may cause contact dermatitis, but absorption through the skin is not significant (Proctor and Hughes, 1978). Gupta et al. (1979) performed a series of acute toxicity tests with mice, rats, and guinea pigs. Dying animals exhibited signs of irritation of the intestines and other internal organs. Repeated doses produced cumulative effects in mice as indicated by a reduction in the dose required to cause death. The lowest median lethal oral dose of cyclohexanone reported for rats is 1,620 mg/kg; the lowest concentration reported to be lethal to rats is 2,000 ppm/4 hours (NIOSH, 1981).

Chronic Toxicity--No effects in humans or animals due to long-term exposure to cyclohexanone have been reported.

Effects on Genes and Chromosomes--No effects on genes and chromosomes were seen in five tests sponsored by the National Institute for Occupational Safety and Health (McGregor, 1980). One investigator reported positive (mutagenic) results in two tests in bacteria, but details of the assays were not provided.

Cancer--No studies of the cancer-causing potential of cyclohexanone in humans or laboratory animals have been completed. Cyclohexanone is currently being tested in a carcinogenesis bioassy under the direction of the National Toxicology Program. No results were available at the time of this writing.

Effects on Reproduction--No data on the effects of cyclohexanone on reproduction have been reported.

Evaluation--There is no federal drinking water standard applicable to cyclohexanone. There are no adequate chronic toxicity data in animals or humans from which a chronic snarl can be calculated (NAS, 1982). In the absence of such data, no recommendation can be made for a concentration of cyclohexanone in drinking water that will ensure long-term safety. However, the California Department of Health Services has proposed both long- and short-term maximum acceptable concentrations of cyclohexanone in drinking water, derived from OSHA permissible exposure limit (California Department of Health Services, 1980). The procedure which is outlined in the evaluation for DMF, results in the derivation of proposed maximum acceptable concentrations (PMACs) based on several absorption estimates and proposed short-term acceptable concentrations (PSTMACs).

In order to account for potential additive effects, the Department proposed using a formula analogous to the ACGIH (1982) method for calculating workplace threshold limit values (TLVs) for mixed exposures:

$$\frac{C_1}{TLV_1} + \frac{C_2}{TLV_2} + \frac{C_3}{TLV_3} = 1$$

where C = concentration of the compound in air and TLV = the respective threshold limit value. If the sum is less than or equal to one, the exposure is considered acceptable; if the sum is greater than one, the exposure is considered excessive. The same procedure can be followed substituting concentrations in water and PMACs for airborne concentrations and TLVs.

The procedure developed by the Department of Health Services is one of several semi-quantitative model that could be used to provide a rough estimate of risk in the absence of appropriate data from human or animal studies. The procedure has been followed in this report because it embodies several features that should be included in such a model: an indicator of

toxicity (TLV or PEL), absorption estimates (R), a means by which acceptable concentrations of contaminants in water can be calculated for individuals of different weights with different daily water intakes, and a safety factor. The results produced by the model are probably conservative, which is appropriate given the lack of chronic toxicity data.

Using this method, the acceptable limits for cyclohexanone can be calculated to yield the following results:

PMAC (R = 0.5) mg/L	12
(R = 0.7) mg/L	17
(R = 0.3) mg/L	7
PSTMAC (R = 0.5) mg/L	120,

where R = the portion of the inhaled dose that is absorbed.

In the case of cyclohexanone, SRI has chosen to calculate PMACs and PSTMACs using the ACGIH TLV. This value is one-half the OSHA PEL and reflects current thought regarding the toxicity of the compound. Using the TLV of 25 ppm (100 mg/m^3) rather than the OSHA PEL of 50 ppm (500 mg/m^3) the values derived are as follows:

PMAC (R = 0.5) mg/L	6.0
(R = 0.7) mg/L	8.5
(R = 0.3) mg/L	3.5
PSTMAC (R = 0.5) mg/L	60.0

Using these more conservative values and examining the data on leachability of cyclohexanone presented in Chapter IV-A (see Tables IV-6 and IV-9), one still finds that no concentrations in water exceeded the PSTMAC and only concentrations found in a static test after a 2-week dwell time exceeded the lowest PMAC (3.5 mg/L).

The data on leachability of cyclohexanone from plastic pipe in Chapter IV-A indicate that all concentrations in water found after static or kinetic

tests were below the PMACs and PSTMAC as calculated above. Adequate flushing procedures prior to use should thus reduce cyclohexanone concentrations in water from plastic pipe systems to levels that would not pose a significant risk to public health. Preliminary calculations using the leaching data in Chapter IV-A also suggest that excessive mixed exposures to the three solvents with similar biologic effects (MEK, THF, and cyclohexanone) would be improbable if adequate flushing were performed.

d. Methyl Ethyl Ketone

Exposure--NIOSH (1980) estimates that approximately 2.5 million U.S. workers are exposed annually to methyl ethyl ketone (MEK). The current ACGIH TLV and OSHA PEL are set at 200 ppm (8-hour time-weighted average) to prevent irritation (OSHA, 1981; ACGIH, 1982). The compound has good warning properties; its odor can be detected at 25 ppm, well below the maximum recommended workplace concentrations. MEK is a constituent of adhesives for plastic piping and has been detected in the air at plumbing installations in concentrations ranging from trace amounts to 34 ppm (California Department of Health Services, 1980). MEK leaches from CPVC plastic plumbing pipe: concentrations reported in Chapter IV-A range from 0.2 to 115 ppm.

Absorption and Metabolism--MEK is effectively absorbed by any route of administration and is readily eliminated unchanged in the breath, or in the urine in unchanged or metabolized form (Tado et al., 1972). Urinary metabolites are 2-butanol, 2-butanol glucuronide, 3-hydroxy-2-butanone, and 2,3-butanediol (Di Vincenzo et al., 1976). MEK may affect the metabolism of other compounds by stimulating the activity of liver enzymes (Traiger et al., 1975).

Acute Toxicity--MEK is slightly irritating to the nose and throat at concentrations of 100 ppm. Short-term exposure to 300 ppm was described as objectionable, and mild headache and throat irritation occurred. In sufficiently high concentrations, MEK can cause central nervous system depression and narcosis; in guinea pigs, inhalation of 10,000 ppm for 5 hours was required to cause narcosis (Proctor and Hughes, 1978).

Chronic Toxicity--The most significant chronic effect of MEK is the potentiation of the neurological effects of other solvents. MEK can increase the damage to peripheral nerves caused by methyl butyl ketone and n-hexane. Animals exposed to methyl ethyl ketone only have shown no signs of neurologic injury. Sprague-Dawley rats exposed by inhalation of 800 ppm MEK for 6 hours per day, 5 days per week for 4 weeks had increased liver weights, indicating possible liver damage. No significant toxic effects were seen in Fischer-344 rats exposed to 1,250, 2,500, or 5,000 ppm MEK for 5 days per week over a 90-day period (CIIT, 1981).

Effects on Genes and Chromosomes--No studies of the effects of MEK on genes or chromosomes were reported.

Cancer--No carcinogenesis bioassays of MEK have been reported. The Chemical Industry Institute of Toxicology (1981) had planned liftime inhalation bioassays in rats and mice but cancelled plans when results of a 90-day study showed no significant toxic effects.

Effects on Reproduction--In two studies conducted in pregnant rats, no statistically significant increase in major malformations was seen in offspring. No effect on the number of implantations or early embryonic deaths was seen. At the 3,000 ppm exposure level, there was a significant increase in the number of offspring with skeletal variations and delayed ossification of the skull. These two phenomena occur spontaneously in control groups. They are not considered true teratogenic effects, but increases in occurrence may reflect toxicity of the compound.

Evaluation--There is no federal drinking water standard applicable to MEK. EPA has calculated 1-day and 10-day SNARLs for MEK of 7.5 mg/L and 0.75 mg/L, respectively. There are no adequate chronic toxicity data in animals or humans from which a chronic snarl can be calculated (NAS, 1982). In the absence of such data, no recommendation can be made for a concentration of MEK in drinking water that will ensure long-term safety. However, the California Department of Health Services has proposed both long- and short-term maximum acceptable concentrations of MEK in drinking

water, derived from OSHA permissible exposure limit (California Department of Health Services, 1980). The procedure which is outlined in the evaluation for DMF, results in the derivation of proposed maximum acceptable concentrations (PMACs) based on several absorption estimates and proposed short-term acceptable concentrations (PSTMACs).

In order to account for potential additive effects, the Department proposed using a formula analogous to the ACGIH (1982) method for calculating workplace threshold limit values (TLVs) for mixed exposures:

$$\frac{C_1}{TLV_1} + \frac{C_2}{TLV_2} + \frac{C_3}{TLV_3} = 1$$

where C = concentration of the compound in air and TLV = the respective threshold limit value. If the sum is less than or equal to one, the exposure is considered acceptable; if the sum is greater than one, the exposure is considered excessive. The same procedure can be followed substituting concentrations in water and PMACs for airborne concentrations and TLVs.

The procedure developed by the Department of Health Services is one of several semi-quantitative model that could be used to provide a rough estimate of risk in the absence of appropriate data from human or animal studies. The procedure has been followed in this report because it embodies several features that should be included in such a model: an indicator of toxicity (TLV or PEL), absorption estimates (R), a means by which acceptable concentrations of contaminants in water can be calculated for individuals of different weights with different daily water intakes, and a safety factor. The results produced by the model are probably conservative, which is appropriate given the lack of chronic toxicity data.

Using this method, the acceptable limits for MEK can be calculated as described in the evaluation for DMF. The final values derived are as follows:

PMAC (R = 0.5) mg/L 36
(R = 0.7) mg/L 50
(R = 0.3) mg/L 22
PSTMAC (R = 0.5) mg/L 360,

where R = the portion of the inhaled dose that is absorbed.

The data on leachability of MEK from CPVC pipe presented in Chapter IV-A (see Tables IV-6 and IV-9) indicate that all concentrations in water were below the PSTMAC, and only the concentrations found in a static test after a 2-week dwell time exceeded any of the PMAC values. These results suggest that adequate flushing procedures prior to use would reduce MEK concentrations in water from plastic pipe to levels that would probably not pose a significant risk to public health. Preliminary calculations using the leaching data in Chapter IV-A also suggest that excessive mixed exposures to the three solvents with similar biologic effects (MEK, THF, and cyclohexanone) would be improbable if adequate flushing were performed.

e. Carbon Tetrachloride

Exposure--In Chapter IV-A, carbon tetrachloride was reported to have consistently been detected in CPVC and PVC pipe within a concentration range of nondetectible to 10 ppb. This chemical has been found in many raw water and finished water sources throughout the United States in concentrations of up to 5 ppb (IARC, 1979; NAS, 1977). It is not usually found in surface waters in California, and is infrequently detected in groundwater sources (Spath, 1983).

Absorption and Metabolism--Carbon tetrachloride is rapidly absorbed from the gastrointestinal tract, the lungs, or through injured skin, and is distributed to the liver, fatty tissues, brain, kidney, blood, and bone marrow. Absorption from the GI tract is augmented by the presence of fats and alcohol. Carbon tetrachloride is excreted principally through the lungs unchanged (about 85 percent of absorbed dose) and as carbon dioxide (10 percent) and other metabolites, which (in rabbits) include chloroform and

hexachloroethane (NAS, 1977). Highly reactive free-radical intermediates are thought to be responsible for carbon tetrachloride's toxicity. Such reactive metabolites can bind irreversibly, primarily to proteins and lipids in the liver, and may do the same in other tissues (IARC, 1979).

Chronic Toxicity--Chronic exposures to carbon tetrachloride causes liver and kidney damage in humans and animals. Symptoms in humans include nausea, vomiting, headache, drowsiness, and fatigue (NAS, 1977).

Effects on Genes and Chromosomes--Carbon tetrachloride was reportedly negative for mutagenic activity in several bacterial assays (IARC, 1979, 1982b; NAS, 1980). It is possible that the negative results may be due to inadequate experimental protocols. One report cited by IARC (1979) indicated that carbon tetrachloride could react with DNA of rodent cells under certain conditions (Rocchi et al., 1973). The International Agency for Research on Cancer considers that there is inadequate evidence of carbon tetrachloride's activity in short-term assays (1982b).

Cancer--In 1979 the International Agency for Research on Cancer reviewed the 11 bioassays involving oral, inhalational, intratracheal, subcutaneous, and intrarectal administration of carbon tetrachloride in several species (rats, mice, hamsters, trout). It was found to be carcinogenic to rats and mice, producing liver tumors in several strains of both species. In one experiment involving subcutaneous injection of carbon tetrachloride to rats, it produced mammary tumors (IARC, 1979).

There is no conclusive epidemiological evidence of cancer in humans exposed to carbon tetrachloride. However, there are several reports of liver cancer following carbon tetrachloride poisoning, and of an increased incidence of several type of malignancy at different sites in persons occupationally exposed to carbon tetrachloride (IARC, 1979, 1982b).

Effects on Reproduction--Carbon tetrachloride has been shown to be fetotoxic and fetolethal, but probably not teratogenic in rats and mice.

Evaluation--The health effect of primary concern for carbon tetrachloride is cancer. There are no MCLs for carbon tetrachloride, although the EPA has recently listed a potential range of recommended MCLs for carbon tetrachloride of 5 to 500 ppb (47 Fed. Reg. 9357, March 4, 1982). This range is not based on potential cancer risk estimates presented for the water quality criteria for carbon tetrachloride (45 Fed. Reg. 79327, November 28, 1980). The upper limit cancer risk estimates (based on consumption of 2 liters of water per day and consumption of 6.5 grams fish and shellfish) are as follows:

Criteria (ppb)	Risk Level
4.0	10^{-5}
0.40	10^{-6}
0.04	10^{-7}

Subtracting the exposure attributable to fish consumption (approximately 6 percent), these values become:

Criteria (µg/liter)	Risk Level
4.3	10^{-5}
0.43	10^{-6}
0.043	10^{-7}

According to the analysis of leaching data in Chapter IV-A, carbon tetrachloride was found at concentrations in the range of approximately 1 to 10 ppb. Since the multistage model is linear at low doses, lifetime risks corresponding to consumption of water containing these concentrations can be calculated:

Concentration (ppb)	Lifetime Risk
1	2.4×10^{-6}
10	2.4×10^{-5}

These lifetime risks are based on a daily exposure to these concentrations for 70 years.

For a shorter exposure period, the risks may decrease proportionately. If, for example, all the carbon tetrachloride in the pipes were to leach out at either of these concentrations in one year, the above risks are divided by 70:*

Concentration (ppb)	Risk for 1-Year Exposure
1	3.4×10^{-8}
10	3.4×10^{-7}

If an individual move into five new homes plumbed with CPVC pipe during his or her liftime, these would change to the following:

Concentration (ppb)	Risk Level
1	1.7×10^{-7}
10	1.7×10^{-6}

An alternative way to look at potential risks is to assume that the residual concentration of carbon tetrachloride in CPVC pipe leaches in its entirety into drinking water. Such risk calculations were made using the following assumptions:

Residual concentration in CPVC pipe = 50 ppm
Quantity of pipe = 20 lb
Quantity of water used per person from faucets = 20 gal/day (not necessarily for drinking)
Number of persons per family (national average) = 2 3/4

$$\frac{50 \text{ ppm} \times 20 \text{ lb} \times 454 \text{ g/lb}}{20 \text{ gal/person/day} \times 2\ 3/4 \text{ persons} \times 365 \text{ days} \times 3.78 \text{ liters/gal}} =$$

0.45 gm/76,000 liters = 5.9 ppb, if leaching occurs over one year.

*This procedure is obviously arbitrary, but is the one most commonly used for regulatory purposes (Thorslund, 1983).

If the exposure occurs over one year, the risk estimate is approximately 2.0×10^{-7}. The result is the same if one assumes a 20 year leaching period and 20 years of exposure. Assuming that an individual may move into five new houses during his or her lifetime, the risk estimate becomes approximately 1×10^{-6}. Under the above assumptions, the plausible risk limit from exposure to carbon tetrachloride alone from CPVC pipe is at the commonly accepted threshold of regulatory significance.

f. Perchloroethylene

Exposure--NIOSH (1980) estimates that 1.6 million workers are exposed annually to perchloroethylene ("PERC," tetrachloroethylene). The OSHA Permissible Exposure Limit (PEL) for PERC is 100 ppm (8-hour time-weighted average concentration); the ACGIH recommends a workplace TLV (8-hour time-weighted average concentration) of 50 ppm (OSHA, 1981; ACGIH, 1982). Perchloroethylene may be formed in water as a result of chlorination. It has been detected in numerous domestic water supplies and industrial effluent (IARC, 1979). Perchloroethylene has been identified as a leachate in CPVC plastic plumbing pipe; concentrations reported in Chapter IV-A range from 1 to 10 ppb.

Absorption and Metabolism--Perchloroethylene is absorbed through the lungs, skin, and gastrointestinal (GI) tract; the presence of fats enhances GI absorption. Most of an inhaled dose of radiolabelled perchloroethylene is eliminated unchanged or as CO_2 in the expired breath. The remainder is metabolized slowly; elimination may require more than 7 days. The major urinary metabolite is trichloroacetic acid (Monster et al., 1979). Ethylene oxide, a suspected carcinogen, may be a metabolic intermediate (Henschler and Bonse, 1977).

Chronic Toxicity--Chronic exposure to perchloroethylene has caused impaired memory and other symptoms of central nervous system damage, abdominal pain, and damage to the peripheral nerves (IARC, 1979). Repeated exposure to PERC by inhalation has produced liver damage in rats, rabbits,

and guinea pigs (Proctor and Hughes, 1978). Oral doses have produced liver and kidney damage in dogs and mice (Klaasen and Plaa, 1966, 1967).

Effects on Genes and Chromosomes--Conflicting results have been obtained in tests designed to detect damage to genes and chromosomes. IARC (1982b) considers that there is inadequate evidence of the genotoxic activity of perchloroethylene in short-term tests. PERC may be a very weak mutagen.

Cancer--Two epidemiologic investigations of dry cleaners occupationally exposed to PERC have been conducted. Both studies found excess deaths from various cancers in the exposed populations, but problems involving possible mixed exposures and incomplete follow-up present problems in interpretation of results. IARC (1982b) considers results to be inconclusive.

Perchloroethylene has been tested in three animal studies for carcinogenic potential. Sprague-Dawley rats exposed for 12 months by inhalation to 300 or 600 ppm perchloroethylene in air showed no increased tumor incidence over treated controls. Negative results were also obtained in a skin-painting study with mice (Van Duuren et al., 1979). In an NCI bioassay in which perchloroethylene was administered by gavage no increased tumor incidence was observed in rats, but hepatocellular carcinoma incidence was significantly increased in mice (NCI, 1977). IARC (1979) considers that the NCI bioassay results provide "limited evidence" of the carcinogenicity of perchloroethylene.

Effects on Reproduction--No teratogenic effects in offspring or other adverse reproductive outcomes were seen when pregnant rats and mice were exposed to perchloroethylene on Days 6-15 of gestation. Offspring of female rats exposed to 100 ppm or 900 ppm perchloroethylene during the gestation period were examined in a series of behavioral tests. No significant differences were noted between offspring of animals in the 100 ppm group and unexposed controls. Behavior of offspring from the 900 ppm group varied from controls but not in a consistent treatment-related manner. In a third experiment, female rats were exposed by inhalation to 1,000 ppm

perchloroethylene throughout pregnancy. Maternal liver weights were increased, fetal body weights were decreased, and there were variations in soft and skeletal tissues, all indicating toxicity, but not teratogenic effects.

Evaluation--The health effect of primary concern for perchloroethylene is cancer. The State Department of Health Services maintains an action level for this chemical of 4 ppb. There are no MCLs for perchloroethylene, although the EPA has recently listed a potential range of recommended MCLs of 5 to 500 ppb (47 Fed. Reg. 9357, March 4, 1982). This range is not based on potential cancer risk estimates presented for the water quality criteria for perchloroethylene (45 Fed. Reg. 79340, November 28, 1980). The upper limit cancer risk estimates (based on consumption of 2 liters of water per day and consumption of 6.5 grams of fish and shellfish) are as follows:

Criteria (ppb)	Risk Level
8.0	10^{-5}
0.80	10^{-6}
0.08	10^{-7}

Subtracting the exposure attributable to fish and shellfish consumption (approximately 9 percent), these values become:

Criteria (ppb)	Risk Level
8.8	10^{-5}
0.88	10^{-6}
0.09	10^{-7}

According to the analysis of the leaching data in Chapter IV-A, perchloroethylene was found at concentrations ranging from approximately 1 to 10 ppb. Since the multistage model is linear at low doses, lifetime

risks corresponding to consumption of water containing concentrations can be calculated:

Concentration (ppb)	Lifetime Risk
1	1.1×10^{-6}
10	1.1×10^{-5}

These lifetime risks are based on a daily exposure to these concentrations for 70 years. For a shorter exposure period, the risks may decrease proportionately. If, for example, all the perchloroethylene in the pipes were to leach out at either of these concentrations in one year, the above risks can be divided by 70:*

Concentration (ppb)	Risk for 1-Year Exposure
1	1.6×10^{-8}
10	1.6×10^{-7}.

If an individual moves into five new homes plumbed with CPVC pipe during his or her lifetime, these would change to the following:

Concentration (ppb)	Risk Level
1	8×10^{-8}
10	8×10^{-7}.

An alternative way to look at potential risks is to assume that the residual concentration of perchloride in CPVC pipe leaches in its entirety into drinking water. Such risk calculations were made using the following assumptions:

*This procedure is obviously arbitrary, but is the one most commonly used for regulatory purposes (Thorslund, 1983).

Residual concentration in CPVC pipe = 50 ppm
Quantity of pipe = 20 lb
Quantity of water used per person from faucets = 20 gal/day
(not necessarily for drinking)
Number of persons per family (national average) = 2 3/4

$$\frac{50 \text{ ppm} \times 20 \text{ lb} \times 454 \text{ g/lb}}{20 \text{ gal/person/day} \times 2\ 3/4 \text{ persons} \times 365 \text{ days} \times 3.78 \text{ liters/gal}} =$$

0.45 gm/76,000 liters = 5.9 ppb if leaching occurs over one year.

If the exposure occurs over one year, the risk estimate is approximately 9.6×10^{-8}. The result is the same if one assumes a 20-year leaching period and 20 years of exposure. Assuming that an individual may move into five new houses during his or her lifetime, the risk estimate becomes approximately 4.8×10^{-7}. Under the above assumptions, the plausible risk limit from exposure to perchloroethylene alone from CPVC pipe is somewhat lower than the commonly accepted threshold of regulatory significance.

g. Trichloroethylene

Exposure--NIOSH (1980) estimates that approximately 2.8 million workers are exposed annually to trichloroethylene. OSHA has set a permissible workplace exposure limit of 100 ppm (8-hour time-weighted average concentration); the ACGIH recommends a TLV of 50 ppm (8-hour time-weighted average) (OSHA, 1981; ACGIH, 1982). Trichloroethylene has been detected in finished drinking water supplies in concentrations of 0 to 0.5 ppb and in tap, lake, spring, and subterranean waters at 80 to 105 nanograms per liter (IARC, 1979). Trichloroethylene has been detected as a leachate from CPVC plastic pipe: concentrations reported in Chapter IV-A range from 1 to 10 ppb.

Absorption and Metabolism--Trichloroethylene is rapidly absorbed by the lungs; about 45 percent of an inhaled dose is excreted unchanged in the

expired breath (IARC, 1979). The portion that is not exhaled is metabolized by the liver to trichloroethanol, trichloroacetic acid, trichloroethanol glucuronide, and chloral hydrate (Monster et al., 1976; Muller et al., 1974; Cole et al., 1975). Alcohol intolerance, a reaction similar to that seen in persons taking Antabuse, has been seen in exposed workers (Proctor and Hughes, 1977). This reaction suggests that trichloroethylene and alcohol are metabolized by the same enzymes. Several investigators have suggested that a reactive epoxide is formed when trichloroethylene is metabolized and that this epoxide, rather than the parent compound or other metabolites, is a carcinogen (Van Duuren and Banerjee, 1976; Henschler and Bonse, 1978).

Chronic Toxicity--Chronic exposure to trichloroethylene produces damage to the central nervous system and the liver. Double vision, changes in color perception, and loss of coordination and sense of smell have been reported. Trichloroethylene can penetrate the skin; repeated contact can cause dermatitis. Repeated immersion of the hands in the liquid reportedly caused paralysis of the fingers (Proctor and Hughes, 1978).

Effects on Genes and Chromosomes--Results of tests for mutagenicity and chromosomal aberrations are conflicting. However, positive results were seen in a sufficiently large number of tests to suggest that trichloroethylene is at least a weak mutagen that can adversely affect genes and chromosomes.

Cancer--Four epidemiological studies have failed to show an association between exposure to trichloroethylene and increased numbers of deaths from cancer. However, in at least two studies, sample sizes were too small and follow-up periods too short for result to be considered conclusive. The International Agency for Research on Cancer considers that the evidence for the carcinogenicity of trichloroethylene is limited (IARC, 1982b).

Three long-term studies of the cancer-causing potential of trichloroethylene have been performed with laboratory animals. In an inhalation study in which rats, mice, and hamsters were exposed to 0, 100, or 500 ppm trichloroethylene in air, a twofold increase in malignant

lymphomas was seen in the two dosed groups (Henschler et al., 1980). In an NCI bioassay, a significant increase in hepatocellular carcinoma incidence was seen in male and female mice, but not in rats administered high doses (549-2,339 mg/kg) by gastric intubation. Because the trichloroethylene used in the original bioassay was contaminated with epichlorohydrin (a carcinogen), a second bioassay using highly purified trichloroethylene was undertaken in 1980. Results reportedly corroborate the findings of the first bioassay (Juodeika, 1983).

Effects on Reproduction--Three tests of the effects of trichloroethylene on reproduction have been reported. No gross malformations were seen in the offspring of pregnant rats or mice exposed by inhalation during the organ-forming period of gestation. In one group of rabbits, an excess of hydrocephalus occurred (Beliles, 1982). No significant maternal toxicity, embryotoxicity, or postnatal effects were seen in female rats or offspring exposed during gestation to 1,800 ppm trichloroethylene (Dorfmueller et al., 1979).

Evaluation--The health effect of primary concern for trichloroethylene is cancer. The State Department of Health Services' action level for this chemical is 5 ppb. There are no MCLs for trichloroethylene, although the EPA has recently listed a potential range of recommended MCLs for trichloroethylene of 5 to 500 ppb (47 Fed. Reg. 9357, March 4, 1982). This range is not based on potential cancer risk estimates presented for the water quality criteria for trichloroethylene (45 Fed. Reg. 79341, November 28, 1980). The upper limit cancer risk estimates (based on consumption of 2 liters of water per day and consumption of 6.5 grams of fish and shellfish) are as follows:

Criteria (ppb)	Risk Level
27	10^{-5}
2.7	10^{-6}
0.27	10^{-7}

Subtracting the exposure attributable to fish and shellfish consumption (approximately 6 percent), these values become:

Criteria (ppb)	Risk Level
27.8	10^{-5}
2.8	10^{-6}
0.28	10^{-7}

According to the analysis of leaching data in Chapter IV-A, trichloroethylene was found at concentrations in the range of approximately 1 to 10 ppb. Since the multistage model is linear at low doses, lifetime risks corresponding to consumption of water containing these concentrations can be easily calculated:

Concentration (ppb)	Lifetime Risk
1	3.6×10^{-7}
10	3.6×10^{-6}.

These lifetime risks are based on a daily exposure to these concentrations for 70 years. For a shorter exposure period, the risks may decrease proportionately. If, for example, all the trichloroethylene in the pipes were to leach out at either of these concentrations in one year, the above risks are divided by 70:*

Concentration (ppb)	Risk for 1-Year Exposure
1	5.1×10^{-9}
10	5.1×10^{-8}

*This procedure is obviously arbitrary, but is the one most commonly used for regulatory purposes (Thorslund, 1983).

If an individual moves into five new homes plumbed with CPVC pipe during his or her lifetime, these would change to the following:

Concentration (ppb)	Risk Level
1	2.5×10^{-8}
10	2.5×10^{-7}.

An alternative way to look at potential risks is to assume that the residual concentration of trichloroethylene in CPVC pipe leaches in its entirety into drinking water. Such risk calculations were made using the following assumptions:

Residual concentration in CPVC pipe = 50 ppm
Quantity of pipe = 20 lb
Quantity of water used per person from faucets = 20 gal/day (not necessarily for drinking)
Number of persons per family (national average) = 2 3/4

$$\frac{50 \text{ ppm} \times 20 \text{ lb} \times 454 \text{ g/lb}}{20 \text{ gal/person/day} \times 2\ 3/4 \text{ persons} \times 365 \text{ days} \times 3.78 \text{ liters/gal}} =$$

0.45 gm/76,000 liters = 5.9 ppb if leaching occurs over one year.

If the exposure occurs over one year, the risk is approximately 3.0×10^{-8}. The result is the same if one assumes a 20-year leaching period and 20 years of exposure. Assuming that an individual may move into five new houses during his or her lifetime, the risk estimate becomes approximately 1.5×10^{-7}. Under the above assumptions, the plausible risk limit from exposure to trichloroethylene alone from CPVC pipe is below the commonly accepted threshold of regulatory significance.

h. Dichloromethane

Exposure--NIOSH (1980) estimates that approximately 2 million persons per year are occupationally exposed to dichloromethane (DCM). OSHA (1981)

has set a Permissible Exposure Limit (PEL) of 500 ppm; the ACGIH TLV is 100 ppm (ACGIH, 1982).

Dichloromethane is formed during the chlorination of water. It has been found in 1 percent of raw and 8 percent of finished water supplies tested; the mean concentration in several samples of finished water was 1 mg/liter. Dichloromethane was found in 9 of 10 domestic water supplies at a mean concentration of 1.6 mg/liter (IARC, 1979). DCM is approved for use by the FDA in food-contact materials and is also permitted as a residue in coffee, hops, and various spices (USFDA, 1977). DCM has been identified as a leachate from CPVC plastic plumbing pipe; concentrations reported in Chapter IV-A range from "nondetectable" to 10 ppb.

Absorption and Metabolism--Dichloromethane is absorbed by the lungs, skin, and gastrointestinal tract. Both degree of absorption and metabolic pathway appear to vary with the magnitude of the dose. The major metabolites of dichloromethane are formaldehyde and carbon monoxide.

Chronic Toxicity--Long-term exposure to dichloromethane has caused damage to the liver, heart, and central nervous system. Two epidemiologic investigations found no increased deaths from cancer, heart disease, or any other cause. Liver damage has been seen in mice after a single lethal dose and after chronic inhalation of 5,000 ppm dichloromethane (IARC, 1979).

Effects on Genes and Chromosomes--Conflicting results have been obtained in several tests designed to detect adverse effects on genes and chromosomes. Dichlormethane was mutagenic in the bacterial test most commonly used as a screen to predict carcinogenicity (Simmon et al., 1977).

Cancer--Dichloromethane has been tested in three assays designed to detect cancer-causing potential. In the first study, an increase in tumors in male mice occurred but was not statistically significant. In the second study, rats and hamsters were exposed to dichloromethane by inhalation for 2 years. A significant increase in benign tumors in female rats and high-dose male rats was seen; lymphosarcomas were increased in female hamsters.

Dichloromethane is currently being tested in an NTP-sponsored 2-year inhalation study; results will not be available for at least 1 year. In an NTP bioassay recently completed, the compound produced significant increases of liver cancers in mice; a decision whether to regard neoplastic nodules in rats as indicative of carcinogenic potential is pending.

Effects on Reproduction--Three studies of the effects of dichloromethane on reproduction in mice and rats have been reported; no effects on litter size, resorptions, or fetal development were seen.

Evaluation

The health effect of greatest potential significance is the recent finding of carcinogenicity. In the NTP bioassay, there were reportedly significant increases in hepatocellular carcinomas in mice of both sexes and neoplastic liver nodules in both male and female rats. Dichloromethane should be regarded as a potential carcinogen on the basis of this bioassay. However, the results of the bioassay were not received in sufficient time to permit a quantitative estimate of potential risks to humans.

j. Organic Tin Compounds

Exposure--Organic tin compounds are used as stabilizers in PVC and CPVC pipe. The specific compounds reportedly used as stabilizers include dimethyl and dibutyl tin bis isooctylthioglycolates. Both dimethyl and dibutyl tin were found in standing water samples from CPVC pipe as indicated in Table IV-15. Apparent contaminants of butyl tin and trimethyl tin were also found in quantities below one part per billion. All these substances were analyzed as chloride, rather than isooctylthioglycolate, derivatives. With the exception of dibutyl tin dichloride, the organotin compounds were no longer detectable after 14 days (sensitivity of analysis was 0.01 ppb).

Absorption and Metabolism--Dialkyl and trialkyl tins may be absorbed from the gastrointestinal tract, although the fraction absorbed differs among species. Most organotin compounds are poorly absorbed from the GI

tract, except trimethyl-, triethyl-, and dimethyl tin (Kimbrough, 1976). Alkyl tin compounds are distributed to the liver and, in the case of trialkyl tins, to the central nervous sytem. After injection in animals, dibutyl tin concentrates in the liver, with smaller amounts in the kidney. Dibutyl tin is excreted unchanged in the bile (Barnes and Stoner, 1959).

Chronic Toxicity--There are few published data on chronic toxicity of organic tin compounds. Two studies by Seinen et al., (1977a,b) on weanling rodents indicate that dibutyl tin dichloride (but not dimethyl tin dichloride) can reversibly cause atrophy of the thymus and other immune system tissues at concentrations as low as 20 ppm in the diet for 2 weeks.

Effects on Genes and Chromosomes--Dibutyl tin dichloride was positive in one mammalian cell test for mutagens, but negative in a bacterial assay (Li et al., 1982). No information was available for dimethyl tin.

Cancer--Dibutyl tin diacetate caused a dose-related trend in the incidence of liver tumors in mice, but there was no significant increase in tumors in rats or mice in a NTP bioassay (NTP, 1979).

Dimethyl tin bis isooctylthioglycolate (75 percent) and monomethyl tin (25 percent) were tested for chronic toxicity in a protocol inadequate for carcinogenicity testing (Mosinger, undated). No tumors were reported in test animals. This evidence is inadequate to assess the carcinogenicity of dimethyl tin bis isooctylthioglycolate.

Effects on Reproduction--No information was available for dibutyl tin bis isooctylthioglycolate.

Dimethyl tin bis isooctylthioglycolate--one inadequate study was submitted in the record (Mosinger, undated). This study was negative for teratogenesis, but only 1 male and 5 female animals were treated.

Evaluation--There is no drinking water standard for organic tin compounds. There are no adequate data from which to calculate a chronic

SNARL. However, in view of the leaching data reviewed in Chapter IV-A, these substances are unlikely to pose a significant risk to health. Trimethyl tin, clearly the most toxic of these compounds, was not detectable at 0.01 ppb sensitivity after 3 days and 2 flushes of the pipe. Butyl tin, probably the least toxic, was present initially at 0.63 ppb, which declined to nondetectable levels after 10 days and 4 flushes of the pipe. Dimethyl tin, present initially at 0.61 ppb, reached nondetectable levels after 10 days and 5 flushes. After 2 weeks of pipe use, there is not likely to be exposure to these substances.

Dibutyl tin, present initially at a concentration of 2.6 ppb, was still present after 21 days at a level of 0.12 ppb. The lowest experimental level of a toxic effect due to dibutyl tin observed was depression of immune function in weanling rats given 20 ppm in the diet for 2 weeks (Seinen et al., 1977a). It was formerly thought that 40 ppm in the diet represented a no-observable-effect level (NOEL) for dibutyl tin dichloride (DBTC), based on a 90-day feeding study (Gaunt et al., 1968). However, immune function had not been assessed. The work of Seinen et al. showed that the dietary NOEL for DBTC in rats must be less than 20 ppm (1977a). Without specification of a NOEL, however, no chronic SNARL can be calculated for DBTC.

However, the atrophic effect on the thymus caused by DBTC was reported to be completely reversible after cessation of exposure (Seinen et al., 1977a). Thus, assuming that pipes will be flushed several times prior to occupancy, it is likely that occupants will be exposed only to DBTC, and then in concentrations below one part per billion for a relatively short period of time. This also assumes, however, that the concentration of DBTC in the water continues to decline as is evidenced in Table IV-15, and that there is no significant breakdown of the pipe in the future which might allow further leaching of this stabilizer into the drinking water.

The above evaluation assumes that none of these organic tin compounds is a carcinogen, although none has been adequately tested for this property.

5. Substances Associated with Polybutylene Pipe: Irganox

a. Exposure

No workplace exposure limits or recommendations for Irganox have been issued. As described in Chapter IV-A, Irganox and Irganox derivatives have been identified as leachates from polybutylene piping systems at concentrations up to 50 ppb and 11 ppm, respectively.

b. Absorption and Metabolism

In studies in rats with radiolabelled Irganox, no measurable radioactivity was found in the urine, expired air, blood, liver, or kidney. The only significant activity (80-84 percent) was found in the feces. These results suggest that most of the Irganox passes through the gastrointestinal tract without being absorbed or metabolized (Ciba-Geigy, 1982).

c. Chronic Toxicity

Irganox was fed to rats in dietary concentrations of 1,000, 3,000, and 10,000 ppm for 104 weeks. No observable effects were seen at any concentrations. The "no-observable-effect-level" was estimated to be 446-547 mg/kg/day.

d. Effects on Genes and Chromosomes

Irganox did not adversely affect genes or chromosomes in a series of tests in mice, bacteria, and cell cultures.

e. Cancer

When Irganox was fed to Sprague Dawley rats in dietary concentrations of 1,000, 3,000, or 10,000 ppm in the diet for 104 weeks, no significant increases in tumor incidence were seen at any concentration. No

treatment-related increases in tumors were seen in mice fed Irganox in the diet at levels of 100, 300, or 1,000 ppm.

f. Effects on Reproduction

Two studies designed to detect adverse effects on reproduction have been conducted with Irganox. Pregnant mice and rats were given oral doses of 150 mg/kg body weight daily from Day 6 through Day 15 of gestation. No adverse effects were seen in rat pups. At the highest dose level in mice, slight retardation in development, as indicated by an increase in incomplete ossification of ribs, was seen in offspring (Ciba-Geigy, 1982).

g. Evaluation

There is no drinking water standard applicable to Irganox. Neither EPA nor the NAS has published a chronic SNARL for this compound. There are no appropriate human data from which a SNARL can be calculated. However, the chronic animal study described earlier, the no-observed-adverse-effect level was estimated to be approximately 500 mg/kg. A chronic SNARL can be calculated as follows, assuming a safety factor of 1,000 and assuming that total exposure to this substance is through drinking water:

$$(500\text{mg/kg} \times 10\text{ kg})/(1{,}000 \times 1\text{ liter}) = 5.0\text{ mg/liter} = 5{,}000\text{ ppb}$$

where 10 kg represents the weight of a child, 1,000 represents a safety factor, and 1 liter the daily consumption of fluid by a child weighing 10 kg.

It appears that the concentration of Irganox detected in standing water in new polybutylene pipe--i.e., 50 ppb, would not pose a significant chronic hazard. This assumes that Irganox is not a carcinogen and does not possess significant reproductive toxicity.

The toxicologic significance of the Irganox derivatives detected in one 5-day PB leaching study at about 11 ppm is unknown. One cannot infer from the study the magnitude of the concentrations of these derivatives over

time. A preliminary analysis of the structures of these compounds indicated that they are probably of minor toxicologic significance. No empirical data on the toxicity of these derivatives, however, could be located. At this stage, given the lack of information about leaching kinetics and toxicity, no estimate of risk for such compounds has been attempted.

7. Substances Associated with Metal PipeLead

a. Lead

Exposure--In California, lead solder used to join copper or galvanized metal pipes is probably the commonest source of lead in drinking water. Lead levels decrease with pipe age, but according to data presented in Chapter IV-A, water lead concentrations in newly plumbed homes may exceed the current MCL of 50 ppb. In national surveys, lead has been found in raw and finished waters at concentrations ranging from nondetectable to 140 ppb (NAS, 1977). Of nearly 2,600 samples of tap water from 969 water systems, the average lead concentration was 13.1 ppb, the maximum of 64 ppb, and in only 1.4 percent of samples did lead exceed 50 ppb.

Higher concentrations occur in areas with soft water, such as Seattle, or where lead service pipes are used. Using the average figure of 13 ppb, the NAS noted that lead intake from drinking water is 10 percent or less of that from the diet. The daily dietary intake of lead in food has been estimated to be 300 mg for men and about 100-150 mg for women and children (NAS, 1977). In urban areas, airborne lead may substantially contribute to lead absorption. Other sources of exposure, particularly for children, include lead in dirt and paint chips.

Absorption and Metabolism--Absorption of lead from the gastrointestinal tract varies with the age of the individual, the chemical form of the lead, and the dietary levels of iron, calcium, fats, and proteins. Children absorb a much higher percentage of dietary lead (about 40 percent) than do adults (about 8-10 percent) (Hammond and Beliles, 1980; IARC, 1980). Lead is rapidly transferred to bone, a cumulative process that occurs throughout

life. Lead in other tissues rises during childhood and adolescence, and reaches a steady state by early adulthood (Hammond and Beliles, 1980). Adults excrete lead primarily in the urine, but also in feces, sweat, and breast milk, and through deposition in the hair and nails. In infants the principal route of excretion is gastrointestinal.

Chronic Toxicity--Low level lead exposure can lead to toxic effects on the nervous system, the kidney, the blood-forming system, and the gastrointestinal tract, with a variety of systemic symptoms. The hematologic and CNS effects of lead are considered most critical. Recent investigations using psychometric and behavioral tests have indicated that subtle effects may take place in children with blood lead levels of 50 µg/100 ml blood. Reports cited by the National Academy of Sciences (1977, 1982) suggest that tap-water lead may raise blood lead concentrations to a range where CNS effects may occur. However, an analysis reported by the EPA indicates that blood lead levels increase as the cube root of water lead concentrations (EPA, 1980c).

Effects on Genes and Chromosomes--There is mixed evidence regarding lead's potential genetic effects in various short-term assays. Some compounds, e.g., lead chloride and lead acetate, are negative in some tests and positive in others. Multiple studies of chromosomes of people occupationally exposed to lead have produced contradictory results. The International Agency for Research on Cancer considers evidence of lead's activity in short-term tests to be inadequate (IARC, 1982b).

Cancer--Some lead salts (lead acetate, lead subacetate, and lead phosphate) are clearly carcinogenic in animals. However, the nature of the animal experiments is such that potential human risks cannot be calculated (NAS, 1982). Epidemiologic studies of lead-exposed workers have been judged inadequate evidence of lead's carcinogenicity in humans (IARC, 1982b).

Effects on Reproduction--Lead has clearly been implicated as fetotoxic, fetolethal, and teratogenic in animals. Menstrual disorders, impaired fertility, miscarriages, and stillbirths have been reported in women

suffering from lead intoxication. The offspring of pregnancies in which there was maternal lead poisoning have had retarded intrauterine and postnatal growth, as well as neurological damage (Gerber et al., 1980). Sperm abnormalities have been reported in men occupationally exposed to lead who have had substantially elevated blood lead levels.

Evaluation--The current MCL for lead is 0.050 mg/liter (50 ppb) (40 CFR 141.11[b]). Recent work indicates that this level may not protect children from neurological effects. On the basis of potential CNS and hematologic effects on the fetus and growing children, the National Academy of Sciences concluded that, "The present limit of 50 µg/liter may not, in view of other sources of environmental exposure, provide a sufficient margin of safety...Although further studies will be necessary to arrive at a reasonable limit, it is suggested that the limit be lowered...(but because of the limitations of current data) this committee cannot now suggest a lower lead standard" (NAS, 1982).

To the extent that the sampling data cited in Chapter IV-A of this report represent levels found in households with new copper pipe joined with lead solder, there may be a significant risk of neurologic damage to fetuses, infants, and young children, particularly in view of other sources of exposure. Lead leachates from older plumbing systems (> 2 years) are well below the current MCL and are unlikely to pose a significant risk. In the current state of knowledge, the extent of the risk to this population cannot be quantitatively assessed. More reliable leaching data from new copper systems are needed. The use of alternative solders should also be explored. If tin/antimony or tin/silver solder were used instead, however, lead leachate concentrations and the associated risks would be substantially lower and probably negligible.

b. Copper

Exposure--In Chapter IV-A, different concentrations of copper leachates were reported in experiments of variable correspondence to realistic conditions. In in-service pipes, the copper concentration was reported to

be 250 ppb in standing water, while in new pipes, an initial leachate concentration of 2,200 ppb declined to 320 ppb by Day 21. Corrosive water produced leachates in laboratory studies of up to 1,610 ppb after a year. Copper was not detected in new galvanized pipe except using corrosive water, which generated maximum concentrations of 40 ppb. Other studies showed that concentrations are typically likely to be substantially less than 1 ppm, particularly in running samples. Under unusual conditions, e.g., prolonged standing or extremes of pH, the concentrations can exceed 1 ppm.

Copper is an essential nutrient, and is found in many foods. For example, pepper contains 53 ppm and oysters up to 1,500 ppm of copper (Venugopal and Luckey, 1978; Doull et al., 1980). The average adult dietary intake is 4 to 5 mg/day (Butler and Daniel, 1973).

Absorption and Metabolism--About 30 percent of dietary copper is absorbed from the gastrointestinal tract: the fraction absorbed depends on the chemical form (salt or metal complex, water solubility) and on the presence of other substances--leucine increases absorption, while various ions of molybdenum, sulfur, iron and zinc decrease absorption (Venugopal and Luckey, 1978). Copper is transported throughout the body, and is found in high concentrations in liver, brain, kidney, and muscles. A 70-kg adult body contains between 80 and 150 mg copper. Fetal copper concentrations are ten times those of adults (Venugopal and Luckey, 1978).

Copper is excreted principally in bile along with unabsorbed dietary copper. Small amounts are excreted in urine and perspiration. Balanced against biliary excretion, net copper absorption in the GI tract is about 5 percent. (Venugopal and Luckey, 1978).

Control of copper absorption, distribution, and excretion is achieved primarily by the liver, but also by carrier proteins in blood (albumin and ceruloplasmin), and cells of the intestinal lining. Hereditary defects in copper metabolism produce diseases characterized by progressive damage to the liver, CNS, and other organs. These include Indian childhood cirrhosis and Mencke's kinky hair syndrome, which are generally fatal in childhood;

persons with Wilson's disease can typically live a normal life when treated with penicillamine (Scheinberg, 1981; Vaughan et al., 1979; Lefkowitch et al, 1982).

Chronic Toxicity--With the exception of persons with the hereditary disorders (Wilson's disease, Mencke's syndrome, Indian childhood cirrhosis) noted above, chronic copper poisoning from exessive ingestion is rare, and is not thought to result in disease in normal individuals (Gosselin et al., 1976; Doull et al, 1980; NAS, 1977). Tissue levels of copper do not increase with age (in adults), although blood levels do (Doull et al, 1980). The significance of the increase is unknown.

Effects on Genes and Chromosomes--Evidence of copper's effect on genetic material is inadequate, although copper may have mutagenic potential.

Cancer--Copper salts injected into roosters' testes cause testicular tumors. Copper has not been thoroughly tested by other routes in animals. Epidemiologic evidence is inconclusive. Overall, evidence for carcinogenicity is weak.

Effects on Reproduction--There is little evidence that excess copper has any effect on reproduction in animals or humans (EPA, 1980d).

Evaluation--The current ambient water quality criterion with respect to human health effects is 1 mg/liter (1 ppm). This level was set only with respect to undesirable taste and odor qualities, however, since the EPA believed that health data were inadequate to specify a protective level (45 Fed. Reg. 79331; November 28, 1980). Under typical conditions of use, copper in drinking water from copper pipes contributes a fraction of normal dietary intake.

As noted, copper is an essential nutrient for which people have evolved adequate homeostatic mechanisms that handle occasional excesses and deficiencies. While the metal can be toxic at high doses, SRI concurs with

the assessment of the National Academy of Sciences that, "the potential for toxicity (from copper in drinking water at observed levels) is virtually nonexistent for humans" (NAS, 1977).

c. Zinc

Exposure--Data presented in Chapter IV-A indicate that typical concentrations of zinc are higher in galvanized steel pipe than in copper pipe and are less than 1 ppm in standing water in 8-year-old galvanized systems and up to 7 ppm in static tests of new pipes (Tables 4-19, 4-20, and 4-21). Zinc is commonly found in drinking water, particularly in areas of soft, acidic water. In water supplies of low pH, zinc concentrations have exceeded 5 mg/l (NAS, 1977). Excluding some industrial operations, the principal source of zinc exposure for the general population is food. Zinc is present in all animal and plant tissues, and is found in high concentrations in meat, fish, dairy products, grains, nuts, and legumes. Average daily intake of this metal is between 8 and 15 mg/day (Venugopal and Luckey, 1978). Recommended Daily Dietary Allowances for zinc are 3 to 5 mg for infants, 10 mg for children 1 to 10 years, and 15 mg for older children and adults (National Research Council, 1979).

Absorption and Metabolism--Zinc is an essential nutrient, required for DNA and protein synthesis and for the activity of numerous intracellular enzymes. Absorption of zinc from the GI tract is variable, depending on the amount in the diet, but averages about 50 percent. A diet high in calcium, phosphate, and copper can decrease zinc absorption, as can some chelating agents.

Zinc is distributed to all tissues, with high concentrations in muscle, skin, bone, liver, kidney, pancreas, eye, and the male reproductive system. Excretion occurs principally in feces, with contributions from unabsorbed dietary zinc, bile, pancreatic, and other GI secretions. Lesser amounts are excreted in urine, sweat, and breast milk (Venugopal and Luckey, 1978). Zinc absorption, metabolism and excretion are governed by an efficient homeostatic mechanism.

Chronic Toxicity--Repeated low-dose ingestion of zinc is essential to sustain life, and an effective homeostatic mechanism virtually assures that temporary exposures to concentrations moderately greater than what is necessary will not result in toxicity. Concentrations of up to 0.25 percent (or 2,500 ppm) in the diet have not caused toxicity in rats. Above this dietary level one finds growth retardation, anemia and abnormal bone formation (Hammond and Beliles, 1980). There has been a report of zinc poisoning in two adults from extended consumption of water from galvanized pipes with zinc concentrations of 40 mg/liter. Symptoms consisted of nausea, loss of appetite, muscular pain and stiffness, and irritability (NAS, 1977).

Effects on Genes and Chromosomes--The National Academy of Sciences (1977) concluded that there are no data to suggest that zinc is mutagenic in animals or humans. The NIOSH Registry of Toxic Effects of Chemical Substances (1981) reports no positive results for zinc in tests of mutagenesis.

Cancer--Zinc injected into the testicles of rats and roosters has produced testicular tumors, an effect ascribed in part to hormonal factors and the high levels of zinc already present in the testes (Hammond and Beliles, 1980). Zinc has not been found to be carcinogenic by other routes of exposure (Sunderman, 1971).

Effects on Reproduction--Zinc at 4000 ppm (0.4 percent) in the diet of pregnant rats was reported to cause increased resorption and fetal death (Schlicker and Cox, 1968). Zinc injection or feeding at doses ranging from 15 to 50 mg/rat per day reportedly resulted in infertility and reduced testicular size (Venugopal and Luckey, 1978).

Evaluation--There are no federal drinking water standards for zinc. EPA has recommended an ambient water criterion for this metal of 5 mg/liter (5 ppm), based on considerations of taste and odor qualities (45 Fed. Reg. 79341; November 28, 1980). While zinc does produce chronic toxic effects at high doses (e.g., several thousand ppm in the diet), it is unlikely to

present a significant risk of chronic toxicity in drinking water from either copper or galvanized iron pipe. It is essential for human nutrition, and homeostatic controls have evolved to regulate absorption and excretion. Zinc levels in drinking water are a small fraction of those in food. In general, zinc deficiency is a more serious health problem than zinc toxicity. The National Academy of Sciences recently concluded that, "[t]he possibility of detrimental health effects arising from zinc consumed in food and drinking water is extremely remote" (1980).

d. Tin

Exposure--The leaching studies reviewed in Chapter IV-A report tin concentrations only for new copper pipe (Tables IV-26 and IV-28). Neither study can be viewed as establishing realistic leaching conditions. In the NSF study (1980), the highest reported concentration of tin (in static samples in pH 11 water) was 1,000 ppb or 1 mg/liter after 6 hours, which declined to a maximum of 300 ppb after 24 hours. The study of Dunnigan and Blumenkranz (1983), involving a 3-month static sample in a new house in which 95/5 tin/antimony solder was used, reported a concentration of 19,800 ppb or 19.8 ppm.

Since tin is rarely included in large-scale surveys of water systems, there is little basis for comparison. The concentrations reported in Chapter IV-A, however, are substantially above those noted by The National Academy of Sciences (1977). Tin has been found in public water supplies at concentrations up to 2.2 ppb and in natural sources at concentrations up to 30 ppb. The principal sources of tin exposure, however, are canned foods and drinks, which generally contain less than 100 mg/kg (100 ppm), but have been reported to contain in excess of 1,000 mg/kg (1,000 ppm) after extended storage in unlacquered cans or if the product has been stored in an open can for several days (NAS, 1977). Some foods naturally contain tin, (e.g., asparagus and dried peas contain about 9 ppm), with tin content dependent on the concentration of tin in the soil. Stannous chloride is used extensively in processing fruits and vegatables (Venugopal and Luckey, 1978). Some toothpastes also contain tin in the form of stannous fluoride.

There are varying estimates of the average daily intake of tin, ranging from 1 mg to 30 mg (NAS, 1977).

Absorption and Metabolism--Tin is poorly absorbed from the GI tract, and hence most of what is ingested is excreted in feces (Venugopal and Luckey, 1978). Absorbed tin can be found mainly in the liver, lungs, and kidneys with trace quantities in other tissues (NAS, 1977; Hammond and Beliles, 1980).

Chronic Toxicity--There are a variety of chronic effects from oral ingestion of tin salts when high doses (at least 0.3Δ of the diet) are used. Deleterious effects on growth and development, the liver, the blood, the GI system and the male reproductive system have been reported. With lifetime consumption of tin at 5 ppm in drinking water, mice and rats reportedly had mild liver and kidney changes (Venugopal and Luckey, 1978). Whether the latter finding can be extrapolated to a human context is questionable, however, since this level of consumption is approximately the average human daily exposure in food.

Effects Genes and Chromosomes--No information was available.

Cancer--While Venugopal and Luckey report that orally ingested tin salts are carcinogenic, the accuracy of this observation is in doubt (Venugopal and Luckey, 1978; Furst and Radding, 1979). A recent bioassay of stannous chloride conducted under the auspices of the National Toxicology Program was negative (NTP, 1982).

Effects on Reproduction--No information was available.

Evaluation--There is no drinking water standard for tin in the United States or in most other countries. There is no water quality criterion for tin. This is due in part to the lack of a reliable rapid method for determining the low concentrations in water (NAS, 1977). Of greater significance are: (1) the relatively marginal contribution of waterborne tin to the average daily intake in food; (2) inorganic tin's poor

gastrointestinal absorption and its consequent low oral toxicity. Furthermore, there is evidence that tin is an essential micronutrient, at least in animals (NAS, 1977). While tin concentrations in static testing of new pipe may reach as much as 20 ppm, these are unlikely to pose a hazard, particularly in copper pipe, where passivation would cause tin levels (e.g., if tin/antimony solder were used) to decline.

e. Cadium

Exposure--In the sampling data described in Chapter IV-A, the highest concentration of cadmium reported was in new galvanized pipe at 3 ppb (Table IV-20). All other values reported in a variety of tests were less than 1 ppb. These values are consistent with the trace amounts found in surveys reported by the National Academy of Sciences (1977). The greatest contribution is in food. Cadmium is found in trace amounts in plants and marine organisms. Common sources of exposure are shellfish, liver and kidneys, wheat, rice, leafy vegetables, and cigarette smoke. The average dietary intake of cadmium has been estimated to range from 40 to 215 µg/day (Venugopal and Luckey, 1978; Friberg et al., 1974; NAS, 1977). Breast milk and cow's milk are important sources of exposure for infants. Based on a national community water supply survey, the contribution of drinking water to the average daily adult intake is thought to be about 3 to 4 µg/day (Ryan et al., 1982).

Absorption and Metabolism--Cadmium is poorly absorbed from the gastrointestinal tract: thus, most ingested cadmium is excreted in feces. The percentage absorbed varies from 0.5 to 12 percent in various animal species (Hammond and Beliles, 1980). Limited human evidence indicates that about 5 to 7 percent is absorbed. (Rahola et al., 1972; NAS, 1980) Younger animals absorb a greater fraction of ingested cadmium than do older ones. Cadmium absorption is enhanced by a dietary deficiency of calcium, vitamin D, iron, zinc, copper or protein. Inhaled cadmium is more efficiently absorbed: 50 percent or more present in cigarette smoke or metal fumes is absorbed (Ryan et al., 1982).

Absorbed cadmium is widely distributed throughout the body, concentrating preferentially in the kidney and liver. The metal accumulates in the body (at least up to age 50), with a biologic half-life estimated to range from several months to 47 years (Ryan et al., 1982). At birth the body burden of cadmium has been estimated to be 1 nanogram, which increases to 15-50 mg by age 50 (NAS, 1977). Excretion of absorbed cadmium is thought to occur principally in the urine, although other routes have not been well investigated (Hammond and Beliles, 1980). As noted above, cadmium appears in breast milk.

Chronic Toxicity--Chronic human exposure to cadmium has resulted in damage to the kidneys, lungs, bones, and cardiovascular system (NAS, 1980; Klaasen, 1980).

Effects on Genes and Chromosomes--There is conflicting evidence as to whether or not cadmium has genotoxic effects in short-term assays and whether it can cause chromosomal aberrations in people exposed to cadmium. The International Agency for Research on Cancer regards existing data as inadequate (IARC, 1982b).

Cancer--Cadmium compounds injected in rodents are carcinogenic. One feeding study involving 50 ppm of cadmium chloride in rats' diet did not cause an increased incidence of tumors. Epidemiologic evidence suggests that occupational cadmium exposure may increase the risk of lung cancer and prostate cancer. However, the evidence here is conflicting (IARC, 1982b).

Effects on Reproduction--Various cadmium compounds administered by injection to animals have been demonstrated to be fetotoxic, fetolethal, and teratogenic. Cadmium in drinking water at 10 ppm has been reported to be teratogenic to mice. Acute exposure of experimental animals to levels of cadmium far in excess of typical human intake can cause damage to male and femal gonads. Such effects have not been reported in humans.

Evaluation--The current MCL for cadmium is 0.010 mg/liter or 10 ppb (40 CFR 141.11[b]). The National Academy of Sciences has calculated a chronic

SNARL for cadmium of 0.005 mg/liter (NAS, 1980). The NAS utilized 70 kg as the average weight of persons exposed. Using a young child's weight instead of 70 kg, and assuming consumption of one rather than two liters per day, this SNARL can be adjusted as follows:

0.005 mg/liter x 10kg/70kg x 2 liter/1 liter = .0014 mg/liter

According to the sampling data summarized in Chapter IV-A, typical cadmium concentrations are less than both the NAS's and SRI's adjusted SNARLs. Even the extreme value of 3 ppb in new galvanized pipe (Table IV-20) is lower than the NAS's chronic SNARL and the MCL. In view of these low values and the observation that cadmium consumption in drinking water is but a fraction of that in food, it appears that under typical use conditions, cadmium in drinking water does not present a significant risk to human health.

Antimony

Exposure--Antimony was not extensively tested for in the sampling protocols in the administrative record. However, it could leach into water if tin/antimony solder rather than lead were used to join metal pipes, although the theoretical likelihood of such leaching is small (Herrera et al., 1982). Very limited evidence would appear to support the hypothesis of minimal leaching. (Dunnigan and Blumenkranz, 1982) There is no current drinking water standard for antimony.

Antimony exposure occurs principally in industry and by administration of some pharmaceutical products. Average human daily intake from all sources has been estimated to be 100 µg (NAS, 1980).

Absorption and Metabolism--Antimony metal occurs in +3 (trivalent) and +5 (pentavalent) oxidation states, which are distributed and metabolized differently. Trivalent antimony has a greater affinity for red blood cells than pentavalent compounds, which are found at higher levels in plasma. Antimony is poorly absorbed from the GI tract, and tends to cause vomiting. Thus, when given medicinally, antimony is administered by injection or

intravenously. Intravenously administered antimony concentrates in the liver, thyroid, and heart. Trivalent antimony is excreted primarily in feces, while the pentavalent form is excreted principally in the urine (NAS, 1980).

Chronic Toxicity--Chronically administered antimony compounds have been reported to cause anemia, heary muscle degeneration, and other symptoms in animals. The lowest no-observed-effect-level has been reported to be 0.0025 mg/kg for guinea pigs fed antimony for 6 months (Arzamastsev, 1964). In industrial contexts, diseases of the lungs, skin, and GI tract have been reported (Hammond and Beliles, 1980).

Effects on Genes and Chromosomes--Evidence is inadequate to assess potential genetic effect of antimony.

Cancer--One drinking water study using mice was negative at 5 ppm, but this study was inadequate by current standards. Epidemiologic evidence of occupational cancer due to antimony is inconclusive.

Effects on Reproduction--Antimony metal has been reported to cause decreased fertility in one study involving rats. Female antimony workers in the USSR reportedly had a variety of gynecologic problems and a greater incidence of miscarriage and premature delivery than a control group (NAS, 1980). There is insufficient evidence to assess potential reproductive effects of antimony.

Evaluation--There are no U.S. drinking water standards for antimony. A chronic SNARL can be calculated from the lowest no-observed-effect level of 0.0025 mg/kg in guinea pigs fed antimony trichloride for 6 months noted above:

$$\frac{0.0025 \text{ mg/kg} \times 70 \text{ kg}}{1000 \times 2 \text{ liters/day}} = 8.75 \times 10^{-5} \text{ mg/liter/day},$$

which is far below the estimated average daily intake of 100 µg/day from all

sources. The usefulness of this SNARL as a guideline is therefore questionable. The Soviet drinking water standard is 0.05 mg/liter, or 50 ppb.

In view of the low probability of antimony leaching from soldered joints, the probable marginal contribution of waterborne antimony to the total daily intake of this metal, and the poor absorption of such compounds from the GI tract, it is unlikely that the antimony component of antimony/tin solder would pose a significant risk of toxicity.

7. Summary Evaluation of Substances Associated with Plastic and Metal Pipe

a. Polybutylene Pipe

From the perspective of public health, PB pipe appears initially to be more acceptable than CPVC in part because no solvents are used in joining the pipe and in part because no carcinogens have been detected as leachates. While leaching studies have not been as adequate, or thorough the results indicate that only Irganox and Irganox derivatives are present at levels above control levels. Irganox itself seems relatively nontoxic, principally because it does not appear to be absorbed. However, smaller Irganox derivatives, with unknown toxicity, are also present initially in concentrations of parts per million. The structures of these substances (shown in Chapter IV-A) do not provoke a high degree of toxicologic suspicion. The leaching kinetics of such substances are unknown. Should subsequent leaching studies demonstrate that they may be present in drinking water under conditions of initial occupancy, it would be desirable to conduct at least minimal short-term tests on these substances--e.g., Ames tests--before passing judgment on the acceptability of PB pipe.

If better-designed, more sensitive leaching studies confirm the results of earlier studies, and if Irganox derivatives are not likely to be present by the time a dwelling is occupied or such derivatives are found to be

negative for genotoxicity, PB pipe would probably represent, from the standpoint of public health, an acceptable alternative to metal pipe for use in plumbing.

b. CPVC Pipe

From the standpoint of public health, the most significant concern about CPVC leachates is that several of them are recognized carcinogens. Individual low-dose risk calculations for carbon tetrachloride, perchloroethylene, and trichloroethylene (based on estimates developed by EPA's Carcinogen Assessment Group using the linearized multistage model) have been presented in the individual evaluations for these substances.

The individual risks from these substances can be summed to give a cumulative risk:

1) Assume 1 ppb concentration:

	Risk
Lifetime exposure	3.9×10^{-6}
1-year exposure	5.6×10^{-8}
Five 1-year exposures	2.8×10^{-7}

2) Assume 10 ppb concentration:

	Risk
Lifetime exposure	3.9×10^{-5}
1-year exposure	5.6×10^{-7}
Five 1-year exposures	2.8×10^{-6}

3) Assume 50 ppm residual concentrations of carbon tetrachloride, perchloroethylene, and trichloroethylene in CPVC pipe, and other leaching parameters described in the individual evaluations for these substances:

	Risk
1-year exposure	3.2×10^{-7}
Five 1-year exposures	1.6×10^{-6}

Thus, within the limitations of the linearized multistage model and of the assumptions noted here and in the separate evaluations for these substances, the estimate of the cumulative incremental lifetime risk from exposure to these chlorinated organic compounds is slightly above the commonly accepted threshold of regulatory significance.

A more sophisticated analysis for calculating the cumulative risk is as follows:

Risk (above background) = $1 - \exp(-q_1d_1 - q_2d_2 - q_3d_3)$, where q = carcinogenic potency factor calculated by EPA and d = concentration of the chemicals of interest

$q_1 = 0.08275\ (mg/kg/day)^{-1}$ carbon tetrachloride
$q_2 = 0.039776\ (mg/kg/day)^{-1}$ perchloroethylene
$q_3 = 0.0126\ (mg/kg/day)^{-1}$ trichloroethylene

Several scenarios are presented below.

1) $d_1 = d_2 = d_3 = 1$ ppb
for lifetime exposure, risk = 3.86×10^{-6}
for 1-year exposure, risk = 5.5×10^{-8}
for five 1-year exposures, risk = 2.75×10^{-7}

2) $d_1 = d_2 = d_3 = 10$ ppb
for lifetime exposure, risk = 3.86×10^{-5}
for 1-year exposure, risk = 5.5×10^{-7}
for five 1-year exposures, risk = 2.75×10^{-6}

3) $d_1 = d_2 = d_3 = 5.9$ ppb (assuming 50 ppm residual concentrations of these compounds in CPVC pipe)
for 1-year exposure, risk = 3.3×10^{-7}
for five 1-year exposures, risk = 1.65×10^{-6}

Not surprisingly, the potential risk estimates are in close agreement with simple addition of calculated risk.

It should be noted that these calculations do not take background levels of such substances in water supplies into account. For households supplied from surface water, the background levels of these compounds are generally nondetectable. These chlorinated organic chemicals have been

detected in wells supplying drinking water in industrialized areas (particularly Los Angeles), but the average concentration for all of these chemicals combined is about 1 ppb (Spath, 1983). In any given instance, background groundwater contamination of drinking water supplies may augment the risk noted above. Generally the increased risk appears to be minor, but occasionally it may be significant.

Furthermore, the potential risk estimate does not take into account the recent finding that dichloromethane is carcinogenic. (No risk estimate was made for the latter substance because the raw data on tumor incidence were not available from the National Toxicology Program.) In addition, if there is any synergistic interaction among these chemicals, the cumulative risk may be underestimated by adding the upper risk limits of individual chemicals. Finally, because of the inadequacies of existing leaching data, it is unclear that these are the only carcinogens that may leach into drinking water from CPVC pipe or that the reported leachate concentrations are representative of values that would be obtained in real life situations.

On the other hand, the cumulative risk estimates (for these three chemicals) may be too high. For example, the carcinogenicity of perchloroethylene and carbon tetrachloride may be a high-dose phenomenon dependent upon cytotoxicity and saturation of primary metabolic pathways; if this is so, calculations made using the multistage model may overstate risk (see Stott et al., 1981; Hoel et al., 1983). Furthermore, calculations based on residual concentrations of chlorinated organic contaminants may have been greater than they are in reality. For example, there is evidence that residual concentrations of carbon tetrachloride in CPVC pipe are 25 ppm or less, not 50 ppm (Desrosiers and Dunnigan, 1982). Additional leaching studies may indicate that these concentrations or other potential carcinogens do not persist long enough to present a significant risk to occupants of buildings plumbed with CPVC pipe.

In summary, the current state of knowledge would indicate that, under the assumptions noted and within the limitations of the risk estimation model, the potential cumulative incremental cancer risk posed by leachates

from CPVC pipe appears to be slightly in excess of 10^{-6}, a commonly accepted threshold of regulatory concern. This result, however, is based on specific assumptions that are subject to revision at the direction of DHCD and on the basis of further studies, which will permit more meaningful conclusions to be drawn.

For the solvent cements we have calculated proposed maximum allowable concentrations according to a procedure proposed by the California Department of Health Services. This procedure is probably conservative, but should not be viewed as a fully adequate substitute for (unavailable) chronic toxicity data. Ongoing NTP bioassays for DMF and cyclohexanone will provide important information. Of the four solvents found in plastic pipe cements, DMF presents the greatest noncarcinogenic health risk; further testing is needed to clarify the effects of DMF on the liver and reproduction.

c. Copper and Galvanized Steel Pipe

In view of the results of the leaching data and the current state of knowledge about the toxicity of leachates from both copper and galvanized steel pipes, there appears to be little likelihood of any significant health risk from either of these kinds of pipe, with the possible exception of lead leachates from lead/tin solder. While it is clear that in homes with plumbing older than 2 years, the concentrations of lead are below the current MCL, new homes may contain water lead levels in excess of 50 ppb. If, as has been suggested by the EPA, blood lead levels correlate with the cube root of drinking water lead concentrations, then concentrations below 50 ppb probably do not present a significant risk to the mental development of infants and young children, who are at highest risk for lead toxicity. However, the National Academy of Sciences (1982) has indicated that the 50 ppb level may not be protective, considering that children are also exposed to lead in food air, and other environmental sources.

There is not enough information on the kinetics of lead leaching in newly plumbed systems to make a judgment about potential risks. It will be

important to quantify the time-dependence of lead leaching from new plumbing. At the same time, it would be desirable to investigate alternative solders that could reduce the risks of lead exposure, such as tin/antimony and tin/silver solder. Since lead solder leaching is a problem in other parts of the country as well, the EPA has recently begun to explore potential impacts of using these alternatives (Lassovsky, 1983). The cumulative nature of lead intoxication indicates that alternative solders be seriously considered if leaching studies demonstrate persistent concentrations of lead above the MCL.

Finally, additional studies should also investigate the possibility of persistent leaching due to cutting oils, such as organometallics. Most components of cutting oils, while potentially toxic, are likely to be rinsed out during pre-occupancy flushings. The ingredients of metal fluxes would not be anticipated to pose significant risks of toxicity, although soldering may create substances of potential toxicologic concern.

8. Through-Permeation of Pipes

Pilot studies reported by Elliot (1982) and Ikesaki (1983) indicate that several low-molecular-weight organic solvents may permeate PB, PVC, and polyethylene pipes and thereby contaminate drinking water. In addition, gasoline was found to permeate PB and PE pipes (as well as the control copper pipe), but not PVC pipe. Some of the low-molecular-weight chlorinated organic compounds caused the PVC pipe to lose its structural integrity.

The significance of these findings from a public health standpoint cannot be evaluated at this time. Several compounds tested are recognized carcinogens that were found to permeate pipe and contaminate water at concentrations in excess of levels corresponding to upper bound incremental lifetime cancer risks of 10^{-6}. If similarly elevated concentrations of carcinogens were to be found in drinking water under more realistic experimental conditions, this would clearly present a significant risk to public health.

Such an assessment would require investigation of the extent of permeability of such pipes at lower soil concentrations representative of, e.g., a gasoline spill, residential structure fumigation, or offsite migration from a hazardous waste site or chemical storage tank. In the absence of such data, it can be said only that contamination of drinking water through permeation of plumbing pipe may present a potentially serious problem. The magnitude of the problem is, in SRI's estimation, likely to prove greater for distribution pipes than for service lines.

C. Worker Safety and Health

1. Introduction and Scope

The potential for harmful effects on those who work with pipes or piping materials must have a prominent place in any consideration of environmental impact. The major area of concern is the effect(s) on those who work most directly with the piping system--the plumbers and pipefitters who install and repair plumbing systems. For a variety of reasons--principally the difficulty of clearly defining the extent of exposure and the relatively small impact of any change of use patterns--the effects on those who work in industries remote from the actual installation will not be discussed in detail here.

2. Potential Occupational Health and Safety Impacts of Pipe Materials

Figures IV-6 and IV-7 are simplified schematics of the flow of metal and plastic pipes (and associated materials) through the economy from extraction of the raw materials to final disposal of the pipe at the end of its useful life. Obviously, the complexities of the actual distribution system cannot be fairly portrayed in this section of this assessment. (See Section III for a more complete discussion of the pathways followed by piping systems in the economy.) Further, even at this simplified level, the effects on occupational health and safety in areas far removed from the sale and installation of pipes cannot be accurately determined, but are probably negligible. The marginal increase or decrease in health effects in such industries as the extraction of coal, gas, or oil due to increased use of plastic pipe can be discussed but cannot be forecast. One can imagine a decrease in the demand for coal due to decreases in orders for cast iron pipe, accompanied by an increased demand for oil and natural gas as raw materials (and energy sources) in the production of the petrochemicals used as feedstocks for the production of the resins used to formulate plastics.

Similarly, increased use of plastics would imply an increased demand for the solvents used to make cements. However, the increased (potential) exposures to workers in this industry might be balanced (overall) by the decreased exposure of workers in the companies producing solder and flux for use in copper pipes.

The occupational hazards associated with physical distribution of pipe and joining materials are unlikely to be measurably affected by the materials being distributed. One could suppose, for instance, that increased storage of the (flammable) cements might lead to an increase in fires in warehouses. However, one could also suppose that substitution of plastic pipe could lead to a decrease in the incidence of lifting injuries due to handling metal pipe or boxes of solder.

Thus, we concentrate on the installation, repair, and disposal activities at the bottom of Figures IV-6 and IV-7, paying principal attention to the plumbing trade. The effects that any change in the current patterns of use of pipe and associated materials will have on the expectation of injury or illness among plumbers will be of greatest interest.

3. Health and Safety Aspects of the Plumbing Trade--Effects of Pipe Materials

> Plumber: A workman who installs and repairs pipes and plumbing. [Middle English "plumber," from Old French "plommier," from Late Latin "plumbarius," lead worker, from Latin "plumbum," lead.] American Heritage Dictionary of the English Language, Houghton-Mifflin Company, Boston, Mass., 1981.

As can be seen from the definition above, the work of the plumber has traditionally involved work with one of the oldest systemic toxicants known--lead. The National Institute for Occupational Safety and Health (NIOSH, 1975) has stated in regard to plumbing, heating, and air conditioning contractors: "...[they] represent one of the oldest professional trades and one of the most hazardous occupations known to man.

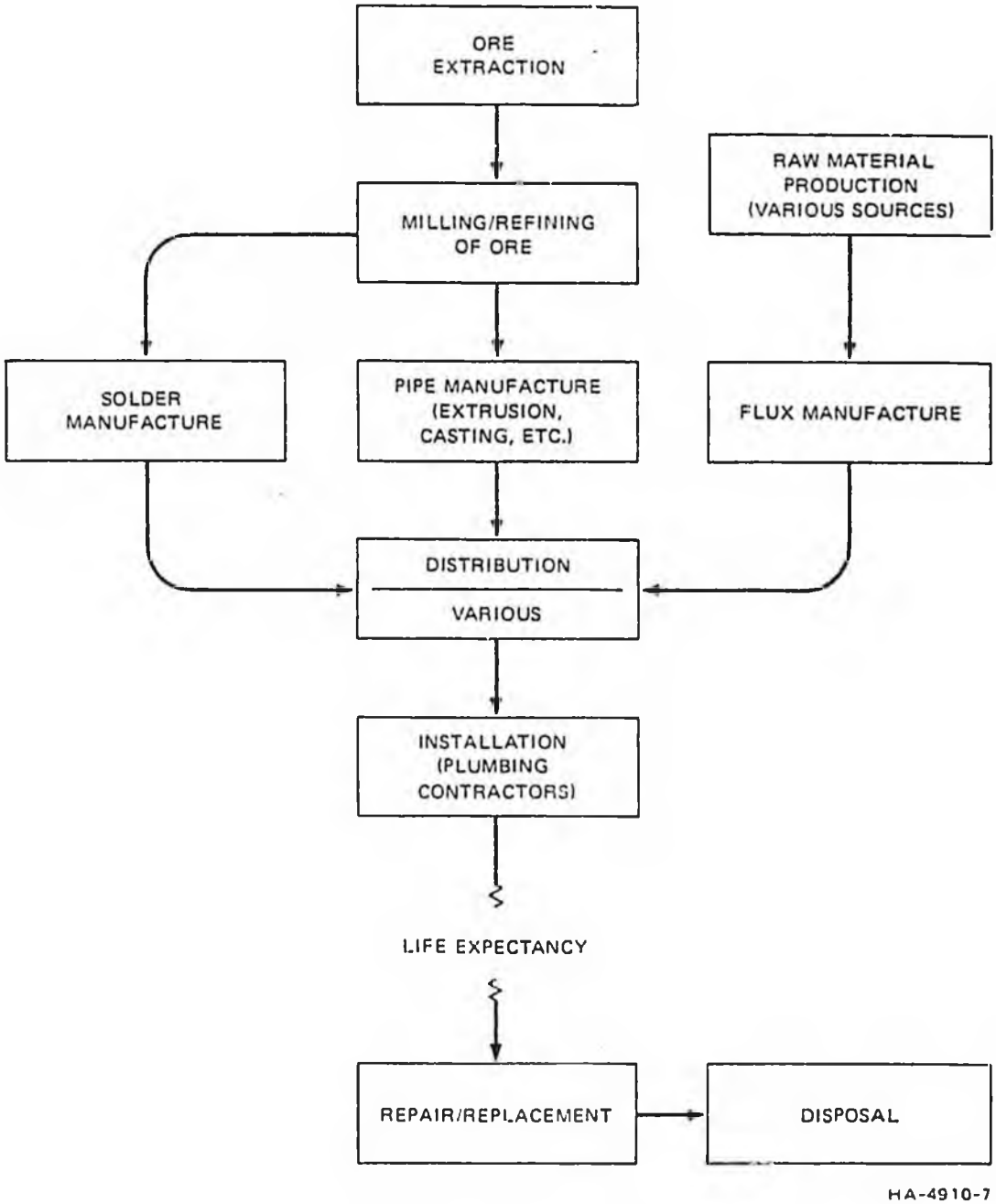

FIGURE IV-6 SIMPLIFIED SCHEME OF DISTRIBUTION OF METAL PIPE — OPPORTUNITIES FOR OCCUPATIONAL EXPOSURES

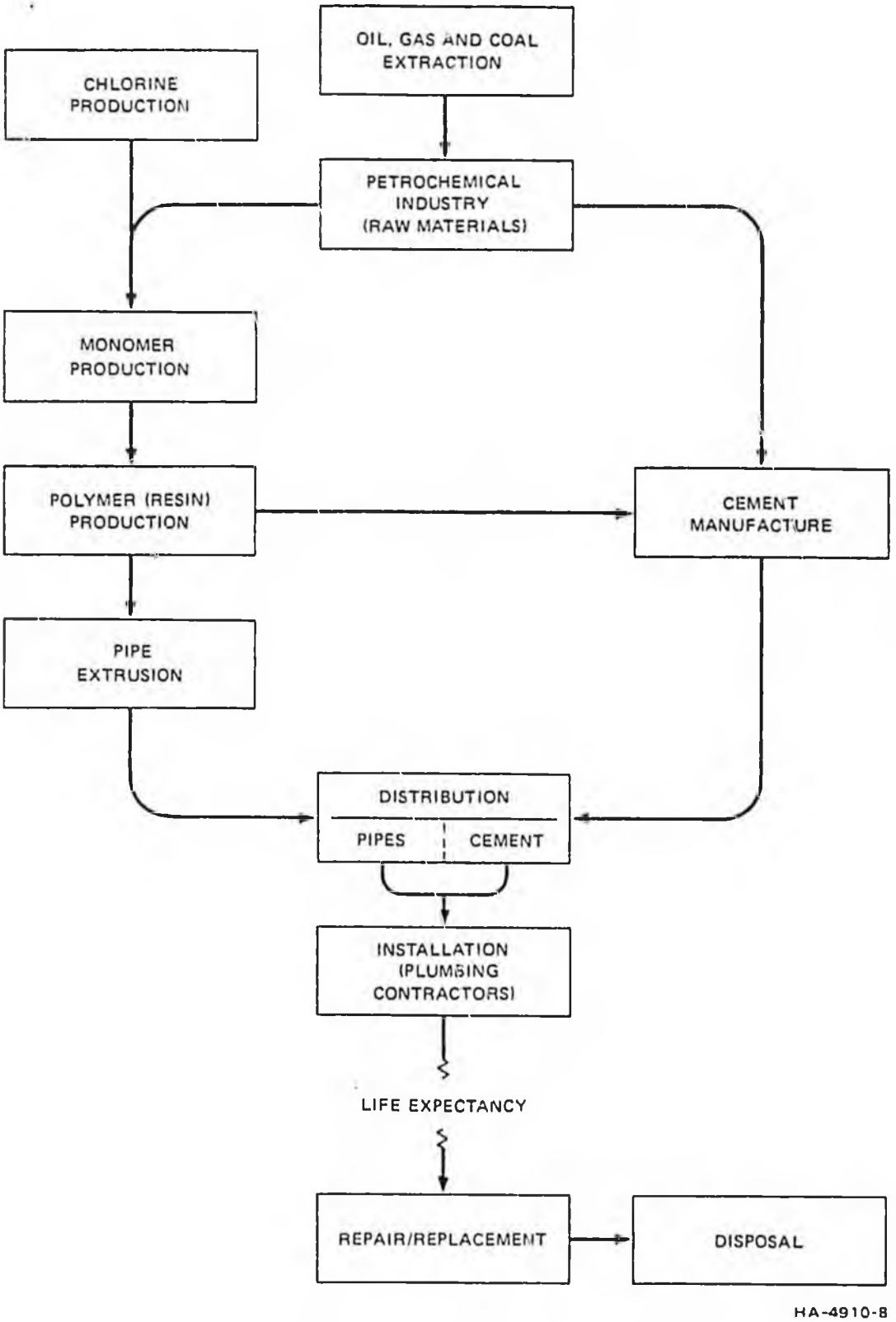

FIGURE IV-7 SIMPLIFIED SCHEME OF DISTRIBUTION OF PLASTIC PIPE — OPPORTUNITIES FOR OCCUPATIONAL EXPOSURES

The history of occupational injuries such as cave-in of excavations and illnesses from lead poisoning and dusts were some of the first to be documented in the written history of man..." Thus, concern about the health and safety of plumbers is not new--the current concern regarding health and safety aspects of plastic piping systems is simply the latest of many regarding potentially hazardous aspects of the plumbing trade. Thus, substitution of plastic pipe for metal pipe should not be viewed as the superimposition of a potentially hazardous material on a background of relatively low hazard. Rather, the existing piping systems in current use have substantial hazards, and the substitution must be viewed as an exchange of risk. The purpose of this section will be to discuss the nature of the competing risks involved, and to assess the relative advantage of substituting one for the other.

4. Function of the Plumber

The plumber's task is to provide piping and fixtures that will convey water and gas from the street mains into the building and deliver it to appropriate fixtures, and then to provide drain and vent piping to convey liquid wastes from the building to the street sewer main, and to vent gaseous waste from the system. Piping is usually laid in trenches to reach the building from the street, and then installed throughout the building by various means.

Before discussing the specifics of operations carried out to install plumbing, we will describe the materials used. One may consider four different services for which plumbing might be used in residential buildings: hot water, cold water, wastes, and gas (for fuel). (Specialty services such as distilled or chilled water are relatively uncommon.) Gas lines are not covered by the proposed action, and so will not be considered here. The other three services may make use of plastic piping. In California, plastic is currently most commonly used in drain, waste, and vent DWV piping.

5. Plumbing Materials

The most common materials used for piping in current plumbing practice are the various plastics, cast iron, galvanized steel, and copper. In past years, a variety of other materials (including lead, bronze, and red brass) were used in addition to the current materials. Although some of these are still in use for the restoration of antique systems, it is now rare for plumbers to install (for instance) the lead pipes that were the cause of extensive lead exposure in past years.

The use of plastic pipe--the subject of this discussion--is not new. According to Babbit (1960), plastic pipe was available for use in the 1940s. The most common plastic pipe material used in the early years was polyethylene. No information on actual extent of use of plastic pipe in plumbing systems has been found, but we have obtained some information on the use of plastic conduit (for electrical wiring). In 1968, 9% of the conduit installed was plastic, while in 1980, 54% was plastic. Although this information is not definitive for the use of plastic pipe, it is indicative of acceptance of the plastics in construction materials. For the purposes of this report, it has been assumed that plastic piping was used sparingly until the mid-1960s, but with increasing frequency thereafter; it is probable that something on the order of half of the linear feet of plumbing materials used today is plastic. To discuss the materials used in a rational fashion, we have chosen to break down the discussion into two sections--water supply piping and DWV piping.

6. Exposures in Plumbing

a. Cutting

The only chemical exposures likely during pipe cutting are those to cutting oils in cutting and threading steel or iron pipe. Sawing is unlikely to produce respirable dust, since the hand sawing of metals is a

slow-speed operation that will usually not produce fine dust. Of particular interest to this project is the probable lack of excessive exposure to plastic dust. The tubing cutter (see Figure IV-8) will turn off a cut "thread" of plastic, if any cutting scraps at all are seen. More commonly, it acts as a rolling knife, forcing through the plastic or copper pipe being cut, with no residual. In some cases (see Table IV-31) measurable dust concentrations have been found. Preliminary results from an as yet unpublished NIOSH study indicate that both respirable and total dust levels may be elevated when PVC pipe is cut with power tools, as would be expected.

b. Installation

It is in installation that the major potential for occupational exposure to chemicals occurs for plumbers. The installation will make use of all of the pipe joing methods previously listed, and the plumber may thus be exposed to solder and flux fumes when installing copper pipe, solvent

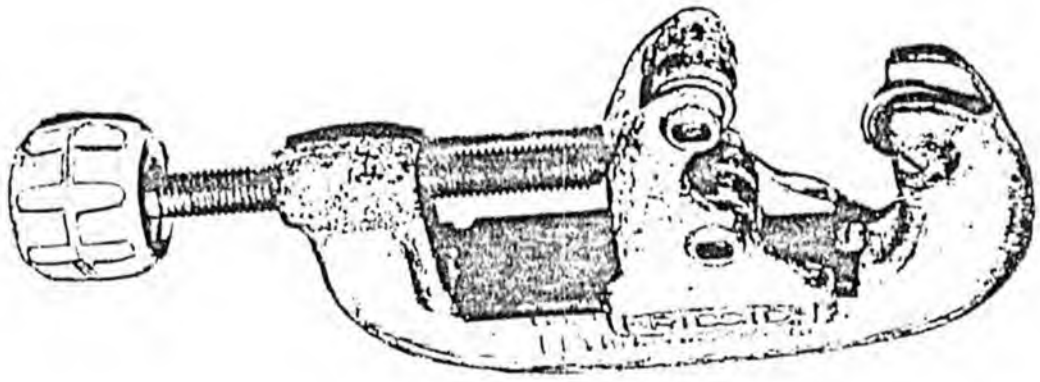

FIGURE IV-8 TUBING CUTTER

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Table IV-31

EXPOSURE DATA ON VCM, AN, AND ABS DUST

Plastic Pipe Operations

Exposure to AN:

Operation	8-Hour TWA (ppm)	Short-Term (ppm)
Prefabrication	0.00021	0.00028 0.00030
Roughing	0.0005	0.002
Topping Off	0.0005	0.002
Finishing	0.0003	0.00048 0.00059

Exposure to ABS dust:

Prefabrication	0.13 mg/M^3

PVC Plastic Pipe Operations

Exposure to VCM:

Roughing	0.007 0.003	0.047 0.010

Source: CDHS (1980a).

vapors when installing plastic pipe (except polybutylene and polyethylene), and lead fumes in the (rare) installation of bell and spigot or wiped joints. There will be, of course, ample opportunity for dermal exposure to all of the above.

c. New Construction

New construction may be categorized by two important variables affecting the potential chemical exposures of plumbers: the size of the job and the end use of the building under construction (commercial or residential). The size of the job, whether commercial or residential, is important because of the pace of the work. On a large enough job, the general contractor will have an on-site staff (superintendent and staff) who are charged with the responsibility for completing the work on-time and on-budget. While this is also true on small jobs, the economic penalties for late or noncompletion of a large job are substantial, and there will be constant visits from the general contractor's staff to assess the performance of the subcontractors (including the plumbing contractor) and to push them to complete the work as soon as possible. On a small job, there will be relatively greater flexibility, and the working plumbing foreman will have much greater latitude to determine the pace of work.

The end use of the building is important because it may (depending on the local jurisdiction) define the allowable plumbing fixtures and pipe type. In some localities, for instance, ABS is allowed for DWV in residential but not in commercial construction. There will usually be special code requirements for medical facilities, schools, and other institutional buildings.

Generally speaking, the plumbing contractor will begin work coincidentally with the final stages of framing of the building, but prior to the installation of electrical wiring or sheathing/subflooring. It is necessary to have the framing in place so that the fixtures can be placed properly in relation to floors and walls, but the plumbers need access to

the joists and studs to drill holes for running pipes, and then need clear access to the pipe, fixtures, and fittings to make joints. Since wiring and piping will often run side-by-side, it is wise to avoid having the wiring in place while the plumbers are working, so that their torches (soldering copper pipe, for instance) will not accidentally melt the insulation on the wires.

Thus, plumbers will usually be working within the open framework of the building, and there will usually be substantial natural ventilation to remove any airborne contaminants. There may be times, however, when the plumbers are working in effectively enclosed spaces. If the building has a basement, then the plumbers may not get into the building until the subflooring has been installed on the first floor; they will then have to work close to the underside of the flooring. (In industrial construction, or in large commercial buildings of poured concrete, this will always be true, regardless of the level.) Second, even within a partially framed building, the plumbers will often be working in small spaces where air exchange is minimal. In addition, plumbing requires close attention to detail--particularly in making good joints. Thus, a plumber installing plastic or copper pipe will sometimes work with his nose close to the pipe (<u>especially</u> when working in enclosed spaces) in order to verify the soundness of joints. (See Figure IV-9 for typical soldering--although the piping is a specialized type--the Sovent system.)

As noted before, there may be prefabrication of substantial components of the plumbing, which will reduce the potential exposures of the installing plumbers. Perhaps more important, there will be specialization in large jobs that may act to substantially increase the exposures of some plumbers while reducing that of others. On a site where plastic is used, the journeyman or advanced apprentice usually doing the actual installation would have relatively intimate and constant contact with the cement throughout the day. This exposure would result not only from the vapor exposure as he cemented the joints, but also from the residual cement on his hands (or gloves) and clothing. A large job with the aforementioned time and cost pressures would accentuate this behavior, and it would not be

uncommon for a plumber to end the day with his coveralls coated with the cement. Installation of copper under the same conditions would also lead to potentially massive exposure to solder fumes. The exposure time-course could be characterized as relatively frequent peaks and valleys, superimposed on a background varying from "clean" to "dirty."

d. Remodeling/Replacement

Replacement of old plumbing with new (as in the remodelling of an old home or building and installation of new fixtures) involves a different set of exposure conditions. First, instead of the (relatively) open and unobstructed working conditions of new construction, the plumber will be faced with fitting pipe into existing wall and crawl spaces after the removal of old pipe. This implies much more enclosed conditions (with more potential for exposure to any airborne materials of concern), but also a slower pace of work (feet per hour). Such work is frequently done by a single plumber (almost always a journeyman), and thus the specialization mentioned above is not found. The exposure profile might be characterized by exaggerated peaks and valleys, with the valleys relatively prolonged.

7. Exposure Potential by Operation

The potentials for substantial exposures in the various plumbing operations are summarized in Table IV-32. Each operation that offers opportunities for exposure is discussed below.

a. Cutting Galvanized Steel Pipe

The major exposure here will be dermal exposure to any cutting oils used. When the work of cutting and threading the pipe is being performed by one plumber--usually a new apprentice working under instruction from an older apprentice or journeyman--there may be relatively constant exposure.