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CHLOROBENZENE

Health Advisory
Office of Drinking Water
U.S. Environmental Protection Agency

I. INTRODUCTION

The Health Advisory (HA) Program, sponsored by the Office of Drinking Water (ODW), provides information on the health effects, analytical methodology and treatment technology that would be useful in dealing with the contamination of drinking water. Health Advisories describe nonregulatory concentrations of drinking water contaminants at which adverse health effects would not be anticipated to occur over specific exposure durations. Health Advisories contain a margin of safety to protect sensitive members of the population.

Health Advisories serve as informal technical guidance to assist Federal, State and local officials responsible for protecting public health when emergency spills or contamination situations occur. They are not to be construed as legally enforceable Federal standards. The HAs are subject to change as new information becomes available.

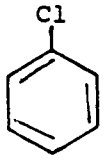
Health Advisories are developed for One-day, Ten-day, Longer-term (approximately 7 years, or 10% of an individual's lifetime) and Lifetime exposures based on data describing noncarcinogenic end points of toxicity. Health Advisories do not quantitatively incorporate any potential carcinogenic risk from such exposure. For those substances that are known or probable human carcinogens, according to the Agency classification scheme (Group A or B), Lifetime HAs are not recommended. The chemical concentration values for Group A or B carcinogens are correlated with carcinogenic risk estimates by employing a cancer potency (unit risk) value together with assumptions for lifetime exposure and the consumption of drinking water. The cancer unit risk is usually derived from the linear multistage model with 95% upper confidence limits. This provides a low-dose estimate of cancer risk to humans that is considered unlikely to pose a carcinogenic risk in excess of the stated values. Excess cancer risk estimates may also be calculated using the One-hit, Weibull, Logit or Probit models. There is no current understanding of the biological mechanisms involved in cancer to suggest that any one of these models is able to predict risk more accurately than another. Because each model is based on differing assumptions, the estimates that are derived can differ by several orders of magnitude.

This Health Advisory (HA) is based on information presented in the Office of Drinking Water's draft Health Effects Criteria Document (CD) for Chlorobenzene (U.S. EPA, 1985a). The HA and CD formats are similar for easy reference. Individuals desiring further information on the toxicological data base or rationale for risk characterization should consult the CD. The CD is available for review at each EPA Regional Office of Drinking Water counterpart (e.g., Water Supply Branch or Drinking Water Branch), or for a fee from the National Technical Information Service, U.S. Department of Commerce, 5285 Port Royal Rd., Springfield, VA 22161, PB #86-117769/AS. The toll-free number is (800) 336-4700; in the Washington, D.C. area: (703) 487-4650.

II. GENERAL INFORMATION AND PROPOERTIES

CAS No. 108-90-7

Structural Formula



Synonyms

- Monochlorobenzene, benzene chloride, chlorobenzol, phenyl chloride.

Uses

- Production of chloronitrobenzene and diphenyl ether; rubber intermediates; solvent in adhesives, paints, waxes, polishes; and inert solvent.

Properties: (Irish, 1963)

Chemical Formula	C_6H_5Cl
Molecular Weight	112.6
Physical State (room temp.)	Colorless, neutral liquid
Boiling Point	132°C
Melting Point	-45°C
Density	--
Vapor Pressure	11.8 mm Hg (at 25°C)
Specific Gravity	1.106 (at 25°C)
Water Solubility	500 mg/L (at 20°C)
Oil/Water Coefficient	918 (Sato and Nakajima, 1979)
Log Octanol/Water Partition Coefficient	2.84 (Leo et al., 1971)
Odor/Taste Threshold (water)	0.41-1.5 ug/L (Tarkhova, 1965) 10-20 ug/L (Varshavskaya, 1968)
Odor Threshold (water)	50 ug/L (Amoore and Hautala, 1983)
Odor Threshold (medium unknown)	0.21 mg/L (A.D. Little, 1968)
Conversion Factor (air)	1 ppm = 4.7 mg/m^3

Occurrence

- There are no natural sources of chlorobenzene.

- Chlorobenzene production in 1984 was 256 million lbs (USITC, 1985). The majority of releases result from chlorobenzene's use as a solvent. Due to chlorobenzene's volatility, most of its environmental releases occur to air. Chlorobenzene is released to water and the ground during the disposal of waste solvent. Because chlorobenzene is used in metal cleaning operations, releases occur in industrial areas nationwide (U.S. EPA, 1987).

- Chlorobenzene released to the atmosphere is expected to degrade slowly by free radical oxidation. Chlorobenzene released to surface water is expected to partition rapidly to air where it also is expected to degrade. Chlorobenzene has been shown to be relatively resistant to biodegradation. Based on limited studies, EPA estimates the half-life of chlorobenzene in soil to be several months. When released to the ground, chlorobenzene is expected to bind to soil and to migrate slowly to ground water. Chlorobenzene has been reported to bioaccumulate in fish, aquatic invertebrates and algae. In higher organisms, chlorobenzene has been shown to be metabolized to other compounds (U.S. EPA, 1979).

- Chlorobenzene rarely occurs as an environmental contaminant. Federal surveys of drinking waters derived from surface water have not reported the presence of chlorobenzene. A few groundwater systems have been found with chlorobenzene levels in the low ppb range. No information of the occurrence of chlorobenzene in food has been identified. Chlorobenzene has been identified as a contaminant of air at very low levels (less than 1 ppb) in urban and suburban areas. Even with the low levels of chlorobenzene in air, inhalation is probably the major route of environmental exposure (U.S. EPA, 1983).

III. PHARMACOKINETICS

Absorption

- No data are available which demonstrate the percentage of the dose absorbed following oral exposure. Based upon what is known about the high lipid solubility of chlorobenzene along with absorption characteristics of benzene and the smaller chlorinated ethanes and ethylenes which are also highly lipid soluble, it will be assumed, for the purpose of the development of Health Advisories, that 100% of any orally administered dose is absorbed, while 60% of a dose inhaled over a period of one to several hours is absorbed and retained (Astrand, 1975; Dallas et al., 1983).

Distribution

- Sullivan et al. (1983) studied the distribution of ^{14}C-chlorobenzene in male Sprague-Dawley rats following single or multiple 8-hour inhalation exposures at 100, 400 or 700 ppm (460, 1,880 or 3,290

mg/m^3). The highest concentrations were found in the fat (epididymal and perirenal). The kidneys and liver also showed significant amounts. The amounts found in these tissues were proportional to dose except for adipose tissue which showed greatly exaggerated accumulation with dose when compared to the other tissues. The ^{14}C burden of adipose tissue increased with increasing exposure concentrations. In addition, there also was a tendency for multiply-exposed rats to exhibit higher tissue burdens than rats exposed only once.

Metabolism

- The metabolic transformation of chlorobenzene has been studied in several mammalian species, including the human (Williams et al., 1975). While absolute quantities and ratios differ between species, the principal metabolites for each species are p-chlorophenol, p-chlorocatechol and p-chlorophenyl-mercapturic acid.

- Because of its lipophilicity (log P =2.96), chlorobenzene tends to bioaccumulate in adipose tissue as exposure continues (Sullivan, et al., 1983). Upon termination of exposure, the chemical would be expected to be released from the fat stores and become available for metabolic activation and potential continuation of induction of toxicity.

Excretion

- The chlorophenol metabolite is excreted as the ethereal sulfate or the glucuronide (Spencer and Williams, 1950; Azouz, et al, 1953). Other excretion products include the chlorophenyl mercapturic acid, 4-chlorocatechol, and to a lesser degree in some species, phenol and hydroquinone (Williams et al., 1975; Sullivan et al., 1983).

- When the metabolic pathways for chlorobenzene biotransformation become saturated, increasing amounts of the chemical are exhaled unchanged (Sullivan et al., 1983). In rats exposed to 100 ppm (470 mg/m^3) (a dose which did not saturate metabolic pathways) in air for 8 hours, 5% was excreted via inhalation and 95% in the urine. Repeated dosing (8 hr/day for 4 or 5 days) at 700 ppm (a dose that does saturate metabolic pathways) results in 32% being exhaled and 68% excreted in the urine.

IV. HEALTH EFFECTS

Humans

- The only information available on the effects of chlorobenzene in the human comes from case reports of poisonings or occupational exposures. No data on actual exposure concentrations are presented in any of these reports.

- Inhalation exposure to chlorobenzene has been observed to result in signs of central nervous depression (sedation and narcosis) as well

as irritation of the eye and respiratory tract (Rozenbaum et al., 1947; Girard et al., 1969; Smirnova and Granik, 1970).

- Rozenbaum et al. (1947) also noted thrombocytopenia and leukopenia in some of the workers described in their study. The question arises as to whether this effect was induced by the chlorobenzene or some contaminant.

- Cardiac effects such as chest pain, bradycardia and ECG irregularities and toxemia of pregnancy have been noted in individuals exposed to chemicals used in the production of chlorobenzene (Dunaeveskii, 1972; Petrova and Vishnevskii, 1972). Chlorobenzene cannot be identified as the causative agent since these workers were exposed to mixtures of substances over varying periods of time.

Animals

Short-term Exposure

- Reported oral LD_{50}'s in adult animals range from 2.8 to 3.4 g/kg (Irish, 1963; Vecerek et al., 1976). Reported inhalation LC_{50}'s range from 0.05 (guinea pig) to 20 mg/l (mouse-2 hour exposure) (Rozenbaum et al., 1947; Lecca-Radu, 1959).

- In rats, single subcutaneous doses greater than 5 g/kg produced hyperexcitability and muscle spasms, followed by CNS depression and death (Rozenbaum, et al, 1947; von Oettingen, 1955).

- Chlorobenzene causes necrosis of the liver and interferes with porphyrin metabolism (Rimington and Ziegler, 1963; Khanin, 1969; Knapp et al., 1971). Oral doses of 1140 mg/kg/day administered to rats for 5 days resulted in increases in urinary excretion of coproporphyrin III, uroporphyrin and porphobilinogen (Rimington and Ziegler, 1963). Delta-aminolevulinic acid levels also were increased as were liver protoporphyrin and uroporphyrin.

- Kidneys of rabbits receiving 2 to 20 doses of chlorobenzene at 0.9 mg/kg by injection over a two-week period showed swelling of the tubular and glomerular epithelia (Rozenbaum et al., 1947).

- Chlorobenzene has been shown to produce alterations in bile duct-pancreatic flow (a phenomenon of unexplained significance)(Yang et al., 1979), and blood dyscrasias such as leukopenia and lymphocytosis (Cameron et al. 1937; Rozenbaum et al., 1947; Zub, 1979). As noted for the human, there is a question as to whether these hematopoietic effects resulted from chlorobenzene or a contaminant.

- Administration of chlorobenzene in corn oil by gavage for 14 consecutive days to male and female F344/N rats and $B6C3F_1$ mice was ineffective in rats and mice at doses of 500 mg/kg/day. Rats were also given 1,000 and 2,000 mg/kg/day doses, which were fatal. Survival, body weights and necropsies data were obtained. Histopathology was not performed.

Long-term Exposure

- Adolescent dogs (6/sex/group) were exposed to chlorobenzene vapors at target levels of 0, 0.78, 1.57 or 2.08 mg/l air for 6 hr/day, 5 days/week for 6 months (Monsanto, 1980). Significant changes included a decrease in absolute adrenal weights in males at the mid- and high-dose levels, an increase in liver:body weight ratio in females at the mid- and high-doses, a sex-independent, dose-related increased incidence in emesis and an increase in the frequency of abnormal stools in treated females. The NOAEL is 0.78 mg/L.

- Oral administration of chlorobenzene by capsule at doses of 0, 27.25, 54.5 or 272.5 mg/kg/day to male and female beagle dogs daily, 5 days/week, until sacrifice at 93 days resulted in observable effects (mortality, lesions, various toxic effects) only at the high dose (Knapp et al., 1971; Hazelton, 1967a). The NOAEL is 54.5 mg/kg/day.

- Oral dosing of rats at levels of 14.4, 144 or 288 mg/kg/day, 5 days/week for 6 months yielded significant increases in liver and kidney weights and histopathological changes in the livers of mid- and high-dose animals (Irish, 1963). No changes were observed at the low dose. The NOAEL is 14.4 mg/kg/day.

- Male and female rats were fed chlorobenzene in their diets at levels equal to 12.5, 50, 100 or 150 mg/kg/day for 90 to 99 days (Knapp, et al., 1971; Hazelton, 1967b). Males showed retarded growth at the highest dose. At the mid- and high dose levels, significant increases in liver and kidney weights were noted. The two lowest dose produced no adverse effects. The NOAEL is 50 mg/kg/day.

- In subchronic (90 or 91 day) studies in which both sexes of rats and mice received chlorobenzene in corn oil by gavage five times weekly with 0, 60, 125, 250, 500 or 750 mg/kg/day (NTP, 1985; Battelle, 1978a,b). Rats and mice showed depressed body weight gain at the highest three doses. In rats, polyuria and porphyria were noted at the two highest doses. Histopathology was noted in the liver, kidney and lymphoid tissue in both species at the three highest doses. Liver and liver/body weights were increased in male mice and female rats at doses above 60 mg/kg. The NOAEL is 60 mg/kg/day.

- The only chronic exposure study available on chlorobenzene is the NTP gavage bioassay in rats and mice (NTP, 1985). On five days/week, both sexes of rats and female mice received 60 or 120 mg chlorobenzene/kg day in corn oil; male mice received 30 or 60 mg/kg/day. Significant changes included equivocal mild to minimal liver necrosis in the rats and a decrease in the survival rate for low dose male mice, but not high dose male mice. The NOAEL is 60 mg/kg/day.

Reproductive Effects

- There are no available data on reproductive effects of chlorobenzene.

Developmental Effects

- John et al. (1984) and Hayes et al. (1982) have reported the results of a two-phase teratology study in which pregnant rats and rabbits were exposed via inhalation to 0, 75, 210 or 590 ppm chlorobenzene, 6 hr/day, during the period of major organogenesis (days 6 through 15 for rats;days 6 through 18 for rabbits). In the rats, maternal toxicity (decreased body weight gain) was observed at the highest dose. No teratological changes were observed in fetuses from rats exposed at any dose. Rabbits showed maternal toxicity (statistically significant increase in relative and absolute liver weights) at the mid and high doses. Again, no structural malformations were noted in the fetuses. However, since the control group exhibited malformations at levels higher than historically noted, the rabbit study was repeated, using doses of 0, 10, 30, 75 or 590 ppm. In this study, no significant changes in rates and types of malformations were observed.

Mutagenicity

- Chlorobenzene has been shown to cause mitotic disturbances in Allium cepa (Ostergen and Levan, 1943) and reverse mutations in Streptococcus antibioticus (Koshkinova, 1968) and Aspergillus nidulans (Prasad and Pramer, 1968;Prasad, 1970).

- Chlorobenzene was not mutagenic in the Ames Salmonella assay or in E. coli, either with or without metabolic activation (Monsanto, 1976a; Dupont, 1977;Merck, 1978; Simmon et al., 1979).

- Chlorobenzene did not induce specific locus forward mutations in mouse lymphoma L5178Y cells, either with or without activation (Monsanto, 1976b).

- Chlorobenzene did induce reciprocal recombination in the yeast Saccharomyces cerevisiae strain D3 in the presence of the metabolic activation system (Simmon et al. 1979).

Carcinogenicity

- Chlorobenzene has been tested for carcinogenic potential in rats and mice in the NTP Bioassay Program (NTP, 1985). The report of these studies states that the chemical produced a statistically significant increase in the incidence of neoplastic nodules of the liver in high dose (120 mg/kg/day) male rats. Incidences of neoplastic nodules in male rats were 2/50 in untreated controls, 2/50 in vehicle controls, 4/49 in low dose and 8/49 in high dose. However, there were also hepatocellular carcinomas in two vehicle control male rats, and combining these with the neoplastic nodule data results in an increase in high dose males of borderline significance ($P = 0.048$) by one statistical test (life table) of the three used (also incidental tumor test and Fischer's exact test) by the NTP. No increased incidence was observed in numbers of hepatocellular carcinomas in male rats or of neoplastic nodules or hepatocellular carcinomas in female rats or mice of either sex.

V. QUANTIFICATION OF TOXICOLOGICAL EFFECTS

Health Advisories (HAs) are generally determined for One-day, Ten-day, Longer-term (approximately 7 years) and Lifetime exposures if adequate data are available that identify a sensitive noncarcinogenic end point of toxicity. The HAs for noncarcinogenic toxicants are derived using the following formula:

$$HA = \frac{(NOAEL \text{ or } LOAEL) \times (BW)}{(UF) \times (\_\_\_ L/day)} = \_\_\_ mg/L \ (\_\_\_ ug/L)$$

where:

NOAEL or LOAEL = No- or Lowest-Observed-Adverse-Effect-Level in mg/kg bw/day.

BW = assumed body weight of a child (10 kg) or an adult (70 kg).

UF = uncertainty factor (10, 100 or 1,000), in accordance with NAS/ODW guidelines.

___ L/day = assumed daily water consumption of a child (1 L/day) or an adult (2 L/day).

One-day and Ten-day Health Advisories

No satisfactory data are available from which to calculate One-day and Ten-day HAs for the 10 kg child. The 14-day studies in rats and mice by the NTP (1985) are not selected because of inadequate assessment of toxicity in these studies. It is recommended that, for this duration of exposure, the Longer-term HA for a 10-kg child be applied. Therefore, the One-day and Ten-day Health Advisories are 4.3 mg/L (4,300 ug/L).

Longer-term Health Advisory

Subchronic studies were conducted in which both rats and mice were dosed by gavage five times weekly with chlorobenzene at 0, 60, 125, 250, 500 or 750 mg/kg in corn oil (10 animals/species/sex/dose level) (NTP, 1985; Battelle, 1978a,b). Deaths were found with the three highest doses in mice and the two highest doses in rats. Food consumption did not vary among the groups in mice, but it was lower in the two highest dose rat groups. Body weight gain was affected in both species, with significant changes observed in mice and rats at the three highest doses. No clinically significant chlorobenzene-related changes were observed in any of the hematological parameters measured in either species. None of the clinical chemistry parameters measured in mice were changed. However, in the rats, alkaline phosphatase and GGPT levels were slightly elevated at 500 and 750 mg/kg. Urinalyses of the controls and two highest dose groups revealed a dose-dependent polyuria with concomitant decreases in specific gravity and creatinine concentration. At the two highest doses, urinary coproporphyrin excretion was increased in rats. In mice, this increase was observed only in females at 250 and 500 mg/kg. Liver and liver: body weights were increased significantly in female mice at 250 and 500 mg/kg and in male mice at 125 and 250 mg/kg. Both male and female rats at 250 and

500 mg/kg and females at 125 mg/kg showed these increases. Absolute and organ/body weights for spleen were decreased in all treated groups of male rats but with no clear dose response. Mice and rats at the three highest doses (250, 500 and 750 mg/kg) all exhibited significant histopathological changes including hepatic necrosis, nephrosis, myeloid depletion, lymphoid depletion and lymphoid necrosis. The 60 mg/kg/day NOAEL with 5 days/week treatment of rats and mice in the NTP (1985) study is equivalent to the 54.5 mg/kg/day, 5 days/week NOAEL in dogs and the 50 mg/kg/day, 7 days/week NOAEL in the Hazelton (1967a,b) studies.

From the NTP (1985) data, a NOAEL of 60 mg/kg/day was identified.

A Longer-term Health Advisory is calculated as follows:

For the 10-kg child:

$$\text{Longer-term HA} = \frac{(60\ \text{mg/kg/day})\ (10\ \text{kg})\ (5)}{(100)\ (1\ \text{L/day})\ \ (7)} = 4.3\ \text{mg/L}\ (4{,}300\ \text{ug/L})$$

where:

60 mg/kg/day = NOAEL, based upon absence of various effects at higher doses in rats and mice.

10 kg = assumed body weight of a child.

5/7 = conversion of 5 day/week exposure to 7 day/week exposure.

100 = uncertainty factor, chosen in accordance with NAS/ODW guidelines for use with a NOAEL from an animal study.

1 L/day = assumed daily water consumption of a child.

For the 70-kg adult:

$$\text{Longer-term HA} = \frac{(60\ \text{mg/kg/day})\ (70\ \text{kg})\ (5)}{(100)\ (2\ \text{L/day})\ \ \ (7)} = 15.0\ \text{mg/L}\ (15{,}000\ \text{ug/L})$$

where:

60 mg/kg/day = NOAEL, based upon absence of various effects at higher doses in rats and mice.

70 kg = assumed body weight of an adult.

5/7 = conversion of 5 day/week exposure to 7 day/week exposure.

100 = uncertainty factor, chosen in accordance with NAS/ODW guidelines for use with a NOAEL from an animal study.

2 L/day = assumed daily water consumption of an adult.

Lifetime Health Advisory

The Lifetime HA represents that portion of an individual's total exposure that is attributed to drinking water and is considered protective of noncarcinogenic adverse health effects over a lifetime exposure. The Lifetime HA is derived in a three step process. Step 1 determines the Reference Dose (RfD), formerly called the Acceptable Daily Intake (ADI). The RfD is an estimate of a daily exposure to the human population that is likely to be without appreciable risk of deleterious effects over a lifetime, and is derived from the NOAEL (or LOAEL), identified from a chronic (or subchronic) study, divided by an uncertainty factor(s). From the RfD, a Drinking Water Equivalent Level (DWEL) can be determined (Step 2). A DWEL is a medium-specific (i.e., drinking water) lifetime exposure level, assuming 100% exposure from that medium, at which adverse, noncarcinogenic health effects would not be expected to occur. The DWEL is derived from the multiplication of the RfD by the assumed body weight of an adult and divided by the assumed daily water consumption of an adult. The Lifetime HA is determined in Step 3 by factoring in other sources of exposure, the relative source contribution (RSC). The RSC from drinking water is based on actual exposure data or, if data are not available, a value of 20% is assumed for synthetic organic chemicals and a value of 10% is assumed for inorganic chemicals. If the contaminant is classified as a Group A or B carcinogen, according to the Agency's classification scheme of carcinogenic potential (U.S. EPA, 1986), then caution should be exercised in assessing the risks associated with lifetime exposure to this chemical.

The data base used for the derivation of the Longer-term Health Advisories also is selected for deriving the Lifetime Health Advisory in that more toxicologic endpoints and species were assessed in the subchronic studies compared to the NTP (1985) carcinogenicity bioassay.

The Lifetime Health Advisory is calculated as follows:

Step 1: Determination of the Reference Dose (RfD)

$$RfD = \frac{(60\ mg/kg/day)(5)}{1,000)\quad(7)} = 0.043\ mg/kg/day$$

where:

60 mg/kg/day = NOAEL based upon absence of various effects at higher doses.

5/7 = conversion of 5 day/week exposure to 7 day/week exposure.

1,000 = uncertainty factor, chosen in accordance with NAS/ODW guidelines for use with a NOAEL from an animal study of less-than-lifetime duration.

Step 2: Determination of the Drinking Water Equivalent Level (DWEL)

$$DWEL = \frac{(0.043\ mg/kg/day)\ (70\ kg)}{(2\ L/day)} = 1.51\ mg/L\ (1,510\ ug/L)$$

where:

0.043 mg/kg/day = RfD.

70 kg = assumed body weight of an adult.

2 L/day = assumed daily water consumption of an adult.

Step 3: Determination of the Lifetime Health Advisory

Lifetime HA = 1.5 mg/L x 20% = 0.3 mg/L (300 ug/L)

It is important to note that the taste and odor threshold in water has been identified at levels ranging from 0.41 to 1.5 ug/L (Tarkhova, 1965) to 10 to 20 ug/L (Varshavskaya, 1967). All of the Health Advisories derived in this document have been developed on the basis of toxicity, not on the aesthetic characteristics of the water quality. Any guidance developed on a site-specific basis may, however, require one to consider the aesthetic, in addition to the toxic, consequences following exposure to chlorobenzene in the drinking water.

Evaluation of Carcinogenic Potential

- The EPA Carcinogenic Assessment Group (CAG) did not derive a carcinogenic potency factor or range of risk estimates for chlorobenzene (U.S. EPA, 1985b).

- EPA has classified chlorobenzene as to its carcinogenic potential, using the weight of evidence classification scheme in its risk assessment guidelines for carcinogens (U.S. EPA, 1986). The Agency has placed the chemical in Group D: Inadequate evidence. EPA's Carcinogen Assessment Group has not derived a carcinogenicity potency factor (q_1^*) or a range of excess lifetime cancer risk estimates.

VI. OTHER CRITERIA, GUIDANCE AND STANDARDS

- EPA (1980) proposed ambient water quality criteria for chlorobenzene, one based upon available toxicity data (488 ug/L) and one based upon organoleptic effects (20 ug/L). These criteria were derived for the 70 kg adult, assumed to drink 2 liters of water per day and eat 6.5 g of contaminated fish and seafood per day. The toxicity-based criteria were calculated using the 14.4 mg/kg/day NOAEL from the study by Irish et al. (1963) and an uncertainty factor of 1,000.

- ACGIH (1982) has adopted a TLV of 75 ppm (350 mg/m^3) for chlorobenzene in the workplace.

- On the basis of a 1983 draft of the NTP report (NTP, 1985), the National Academy of Sciences performed a quantitative risk assessment to estimate excess lifetime cancer risk (NAS, 1983). The upper 95% confidence limit estimate of that risk was 2.13×10^{-7} per ug/L of drinking water. This corresponds to a drinking water concentration of 2.35 ug/L being equivalent to a 1 in a million excess risk.

Assumptions were defined for a 70-kg adult, drinking 2 liters of water per day.

- WHO (1984) recommended a guideline for chlorobenzene of 3 ug/L based upon avoidance of taste and odor problems.

- The U.S. EPA Office of Drinking Water proposed an RMCL of 0.06 mg/L (U.S. EPA, 1985c).

VII. ANALYTICAL METHODS

- Analysis of chlorobenzene is by the purge-and-trap gas chromatographic procedure used for the determination of volatile organohalides in drinking water (U.S. EPA, 1984a). This method calls for the bubbling of an inert gas through the sample and trapping chlorobenzene on an adsorbant material. The adsorbant material is heated to drive off the chlorobenzene onto a gas chromatographic column. This method is applicable to the measurement of chlorobenzene over a concentration range of 0.05 to 1500 ug/L. Confirmatory analysis for chlorobenzene is by mass spectrometry (U.S. EPA, 1985d). The detection limit for confirmation by mass spectometry is 0.3 ug/L.

VIII. TREATMENT TECHNOLOGIES

- Treatment techniques which are effective in removing chlorobenzene from drinking water include adsorption on granular activated carbon (GAC) or powdered activated carbon (PAC). Aeration, reverse osmosis and boiling also are capable of removing chlorobenzene.

- Dobbs and Cohen (1980) developed adsorption isotherms for a number of organic chemicals, including chlorobenzene. They found that Filtrasorb ® 300 carbon had a capacity of 91 mg of chlorobenzene per gram of carbon at an equilibrium concentration of 1.0 mg/L and 9.3 mg/g at a concentration of 100 ug/L.

- PAC gave inconsistent removal rates when it was added to well water containing several contaminants including chlorobenzene (U.S. EPA, 1985b).

- Conventional coagulation filtration treatment does not appear to be effective in chlorobenzene removal. Limited data collected at Water Factory 21 indicated that there was an 18.2% removal of chlorobenzene when only filtration was used (U.S. EPA, 1985b). Another study of conventional treatment practices found them to be completely ineffectiv in chlorobenzene removal (Love et al., 1983).

- The Henry's Law Constant for chlorobenzene is 145 atm at 20°C (U.S. EPA, 1985b). This indicates that the chemical might be amenable to removal by aeration. In a bench-scale study, a diffused air aerator reduced the chlorobenzene in a 97 ug/L solution by 90% using a 15:1 air-to-water ratio (Love et al., 1983).

- Air stripping is an effective, simple and relatively inexpensive process for removing chlorobenzene and other volatile organics from water. However, the use of this process then transfers the contaminant directly into the air stream. When considering use of air stripping as a treatment process, it is suggested that careful consideration be given to the overall environmental occurrence, fate, route of exposure and various hazards associated with the chemical.

- Degradation with ozone is ineffective as a method for removing chlorobenzene (*U.S. EPA, 1985d*).

- Reverse osmosis appears to have the potential for use in chlorobenzene removal. A laboratory study reviewed by EPA reported successful decontamination with 97 to 100% of the chlorobenzene removed.

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