March 31, 1987

n-HEXANE

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.

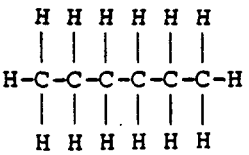
Health Advisories serve as informal technical guidance to assist Federal, State and local officials responsible for protecting public health when emergency spills or contamination situations occur. They are not to be construed as legally enforceable Federal standards. The HAs are subject to change as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

II. GENERAL INFORMATION AND PROPERTIES

CAS No. 110-54-3

Structural Formula



Synonyms

- Esani, Heksan, Hexahen (NIOSH, 1978)

Uses

- Hexane is used commercially as a solvent in glues, varnishes, cements and inks (NIOSH, 1977).
- Hexane also is used in the seed oil industry to extract the natural oils from various seeds, including soybeans and cotton seeds.

Properties (Windholz, 1983)

Chemical Formula	$CH_3(CH_2)_4CH_3$
Molecular Weight	86.18
Physical State	Liquid
Boiling Point	68.7°C
Melting Point	--
Density	--
Specific Gravity	0.655 at 25°C
Vapor Pressure	150 mm at 25°C
Water Solubility	23 mg/liter
Log Octanol/Water Partition Coefficient	--
Taste Threshold	--
Odor Threshold	--
Conversion Factor	1 ppm = 2.78 mg/m^3

Occurrence

- Hexane has not been included in Federal and State surveys of drinking water and no other information on the occurrence of hexane has been located.

III. PHARMACOKINETICS

Absorption

- Bus et al. (1983) studied the absorption of n-hexane in rats following a single 6-hour inhalational exposure to 1,000 (2,780 mg/m^3), 3,000 (8,340 mg/m^3) or 10,000 (27,800 mg/m^3) ppm c^{14}-n-hexane. Total

radioactivity collected in the various excreta fractions was approximately 98% of the total administered dose levels.

Distribution

° No information was found in the available literature on distribution of n-Hexane.

Metabolism

° DiVincenzo et al. (1976) studied the metabolism of n-hexane in guinea pigs. The chemical was dissolved in corn oil and injected intraperitoneally in a single dose of 450 mg/kg body weight in male guinea pigs. Blood samples were collected 1, 2, 4, 6, 8, 12 and 16 hours after the dose was administered. The two major metabolites of n-hexane in the serum were identified as 2,5-hexanedione and 5-hydroxy-2-hexanone; however, they were not quantified.

° Studies in shoe factory workers show that n-hexane is metabolized to 2-hexanol, 2,5-hexanedione, 2,5-dimethylfuran and O-valerolactone (Perbillini et al., 1980).

° Baker and Rickert (1981) studied metabolism of n-hexane in the Fischer-344 rat following inhalation of n-hexane. Male F-344 rats were exposed to 500 (1,390 mg/m^3), 1,000 (2,780 mg/m^3), 3,000 (8,340 mg/m^3) or 10,000 (27,800 mg/m^3) ppm n-hexane in the air. n-Hexane and its metabolites, methyl-n-butyl ketone (MBK), 2,5-dimethylfuran (DMFU), 2,5-hexanedione (2,5-HD), 2-hexanol and 1-hexanol, were quantified by GC/MS in several tissues at time intervals during and following a single 6-hr exposure to n-hexane. Urinary metabolites were quantified following a single n-hexane exposure. n-Hexane concentrations achieved an apparent steady state within two hours in all tissues. Peak blood concentrations of n-hexane were 1, 2, 8 and 12 ug/ml and peak sciatic nerve concentrations were 12, 48, 130 and 430 ug/g at 500 (1,390 mg/m^3), 1,000 (2,780 mg/m^3), 3,000 (8,340 mg/m^3) and 10,000 (27,800 mg/m^3) ppm, respectively. The halflives of n-hexane and MBK were on the order of 1 to 2 hours in all tissues except the kidneys ($K^{1/2}$ = 5 to 6 hrs). The data showed a complex relationship between n-hexane exposure and peak concentrations of the remaining metabolites. Tissue concentrations of 2,5-HD were not proportional to dose. Highest 2,5-HD concentrations were found following exposure to 1,000 ppm n-hexane in the blood, kidneys and sciatic nerve (6.1, 55 and 25 ug/g, respectively). The data indicated that the metabolism and elimination of n-hexane were dependent upon exposure concentration. Consequently, n-hexane exposure concentration cannot be directly correlated with tissue 2,5-HD concentrations.

Excretion

° The metabolites of n-hexane in urine and their concentrations following n-hexane (commercial) exposure of shoe factory workers were: 2-hexanol (0.5 mg/liter), 2,5-hexanedione (10.1 mg/liter), O-valerolactone (2.4 mg/liter) and 2,5-dimethylfuran (5.2 mg/liter) (Perbillini et al., 1980).

IV. HEALTH EFFECTS

Humans

- Hershkowitz et al. (1971) reported the effects of inhalation of n-hexane vapor on three female employees who worked in a furniture factory. n-Hexane concentrations in the room averaged 650 (1,807 mg/m^3) ppm with peaks up to 1,300 (3,614 mg/m^3) ppm. The first symptoms appeared 2 to 4 months after the beginning of exposure and the three employees were hospitalized 6 to 10 months later when they complained of one or more of the following symptoms: headache, burning sensation of the face, abdominal cramps, numbness and weakness of the distal extremities. Physical examination revealed bilateral foot-drop gait, bilateral wrist drop and absence of Achilles tendon reflexes. Electromyographic examination of these patients indicated fibrillation potentials in the small muscles of the hands and feet. Biopsies of the anterior tibial muscle and sural nerves of two of the patients revealed that the muscles contained small angulated fibers and other fibers with clear central zones (denervation type injury). Small bundles of axons from the muscle sections were studied by electron microscopy and found to contain dense bodies and fibrous formations, increased numbers of neurofilaments and abnormal membranous structures with clumped and degenerated mitochondria. Motor-end plates also were damaged, with swollen terminal axoplasmic expansions, an increased number of degenerated mitochondria and an increased number of glycogen granules, dense bodies, large osmophilic membranes, synaptic folds and vesicles. The investigators reported that the health of these employees improved after leaving their employment.

Animals

Short-term Exposure

- Kimura et al. (1971) studied the oral toxicity of a single dose of n-hexane in different age groups of rats: newborn (1 to 2 days old, 5 to 8 g), 14 days old (16 to 50 g), young adult (80 to 160 g) and older adult (300 to 470 g). The undiluted solvents were administered orally to non-fasted rats. A precise LD_{50} value for n-hexane could not be determined for the newborn rats because of measurement limitations, but doses of less than 1 ml/kg body weight were lethal. The acute oral LD_{50} was 24.0 ml/kg (15.7 g/kg) for 14-day old rats, 49.0 ml/kg (32.1 g/kg) for young adults and 43.5 ml/kg (28.9 g/kg) for older adult rats.

- Hewett et al. (1980) carried out experiments in which groups of male adult Sprague-Dawley rats were given a single oral dose of 1,290 mg/kg of n-hexane solubilized in corn oil (control animals received corn oil alone). This segment of the experiment was to provide evidence of potentiation of chloroform toxicity in rats pretreated with n-hexane, methyl n-butyl ketone or 2,5-hexanedione. n-Hexane-induced hepatotoxicity was estimated 42 hours later by measuring enzyme activity of glutamic-pyruvic transferase (GPT) and ornithine carbamyl transferase (OCT) in the plasma of animals.

The extent of cell damage was assessed by observing histological changes in the liver and nephrotoxicity was evaluated by monitoring the ability of renal cortical slices to accumulate an organic anion (p-aminohippurate) and cation (tetraethylammonium) and by determining the blood urea nitrogen content. The investigators reported that the 1,290 mg/kg dose of n-hexane produced no measurable effects either on organ weight (liver, kidney) or on any parameters described earlier. However, a single oral dose of n-hexane in rats produced minimal changes in renal histology as indicated by the presence of degenerated tubules in sections from these animals.

° Howd et al. (1982) studied the relation between schedules of exposure to n-hexane and plasma levels of 2,5-hexanedione. Male Fischer rats were exposed repeatedly to high concentrations of n-hexane: 4,000 ppm for 8 hours/day for 5 days/week; 48,000 ppm for 10 minutes every half hour for 8 hours/day, 5 days/week; 40,000 ppm for 10 minutes every half hour, on a background of 4,000 ppm continuous, for 8 hours/day, 5 days/week. Concentrations of n-hexane in blood and brain were linearly related to the concentrations of n-hexane in the chamber after a 10-minute exposure, and declined thereafter, with half-lives of about 2-1/2 and 4 minutes in blood and brain, respectively. Despite the rapid elimination of n-hexane, neurotoxic levels of 2,5-hexanedione (2,5-HD) were formed from repeated 10-minute exposures to a high concentration of n-hexane when the inter-exposure interval was 20 minutes. Neurotoxic levels of 2,5-HD also resulted from continuous exposure to much lower concentrations of n-hexane. Both exposure schedules (4,000 ppm for 8 hours/day and 10 minutes every half hour exposure) caused an increase in 2,5-HD concentrations in blood after repeated daily treatments. The authors suggested that the minimal sustained plasma 2,5-HD concentration that will result in neurotoxicity appears to be less than 50 ug/ml in the rat.

° In vitro toxicity of n-hexane and 2,5-hexanedione using isolated perfused rabbit hearts is reported (Raye, 1983). The hearts were perfused using Langendorf's procedure and modified Anderson's coronary perfusion apparatus. The force of cardiac contraction was significantly reduced following one hour perfusion with 9.6 mg/L concentration of n-hexane and with 0.35% v/v concentration of 2,5-hexanedione.

Dermal/Ocular Effects

° Jakobson et al. (1982) reported results of uptake via the blood and elimination of n-hexane (one of 10 organic solvents) following epicutaneous exposure of anesthetized guinea pigs. The concentration of n-hexane in the blood was monitored over a 6-hour period of n-hexane exposure of anesthetized guinea pigs. A glass ring chamber (area: 3.1 cm^2) 4 mm in thickness and 10 mm in height was glued to a clipped area of skin on the back of the guinea pigs. This glass ring chamber contained 1.0 ml of n-hexane solvent for the study. With n-hexane, the concentrations in the blood at 0.5 and 6 hrs were 0.58 and 0.23 ug/ml of n-hexane, respectively.

- Nomiyama and Nomiyama (1975) investigated the absorption rates of n-hexane and toluene through the skin of humans. An unspecified number of subjects immersed their hands up to the wrists in a dish containing analytically pure n-hexane (95% n-hexane) for 1 minute. At intervals following skin exposure, breath, blood and urine samples were analyzed for n-hexane by gas chromatographs. The authors were unable to detect hexane in either the breath or the blood of any of the subjects following exposure to n-hexane. The detection limit for n-hexane was 1 ppm in the breath and 3.5 ppm in blood. The authors did not describe any physiological effects.

Long-term Exposure

- Krasavage et al. (1980) studied the relative neurotoxicities of n-hexane by the appearance of hind-limb weakness. Charles River male rats were given oral doses of n-hexane at 570 mg/kg (6.6 mmol/kg) 5 days/week for 90 days or 1,140 or 4,000 mg/kg doses for 120 days. As soon as hind-limb weakness clinical signs occurred, the animals were killed and the tissues were examined for histopathological changes. No clinical or histological signs of neuropathy were observed in the animals at dose levels of 570 or 1,140 mg/kg n-hexane (although body weights were depressed at all three dose levels compared to controls). At a dose level of 4,000 mg/kg n-hexane, the clinical and histological signs of neuropathy occurred at approximately 101 days. The histological changes included multi-focal axonal swellings, adaxonal myelin infolding and paranodal myelin retraction. In addition to neuropathy, histological examination of testicular tissue revealed varying stages of atrophy of the germinal epithelium following the administration of 4,000 mg/kg n-hexane.

- Takeuchi et al. (1980) studied the neurotoxicity of n-hexane in Wistar strain male rats following inhalation exposure to 3,000 ppm (8,340 mg/m^3) of n-hexane for 12 hours a day for 16 weeks. The nerve conduction velocity and the distal latency measured before the beginning of the exposure and after the experiment showed that (1) n-hexane disturbed the conduction velocity of the motor nerve and the mixed nerve and prolonged the distal latency in the rat's tail and (2) the neuromuscular junction and the muscle fiber of the rats exposed to n-hexane were impaired severely as seen by light and electron microscopy.

- Cavender et al. (1984) reported the results of a 13-week vapor inhalation study of n-hexane in rats with emphasis on neurotoxic effects. Male and female Fischer-344 rats were exposed to 0, 3,000 (8,340 mg/m^3), 6,500 (18,070 mg/m^3) or 10,000 (27,800 mg/m^3) ppm n-hexane vapors 6 hours per day, 5 days per week, for 13 weeks. The 13-week exposures had no adverse effect on the growth of female rats. However, the mean body weight gain of male rats in the 10,000 (27,800 mg/m^3) ppm was significantly lower than for controls at 4 weeks of exposure and thereafter. In addition to the depression in body weight gain, the males exposed to 10,000 (27,800 mg/m^3) ppm had slightly but significantly lower brain weights at necropsy. No adverse testicular effects were noted. Axonopathy was observed in the tibial nerve in

four of five male rats from the 10,000 ppm group and one of five male rats in the 6,500 (18,070 mg/m^3) ppm group and in the medulla from one male rat in the 10,000 (27,800 mg/m^3) ppm group. These axonal changes were detectable only in teased nerve fiber preparations or in Epon embedded specimens. Histopathologic studies on Formalin fixed tissues did not reveal any lesions that were attributed to n-hexane exposure.

Reproductive Effects

- Bus et al. (1979) studied the effects of maternal inhalation exposure to n-hexane on the size and survival of newborn Fischer 344 rats. Pregnant rats were exposed for 6 hours per day to 1,000 (2,780 mg/m^3) ppm (3.5 g/m^3) n-hexane on days 8 to 12, 12 to 16, or 8 to 16 of gestation. No significant changes in fetal resorption, body weights, visible anomalies or the incidence of soft tissue and skeletal anomalies were noted in any of the treatment groups. The post-natal growth of pups born to dams exposed to n-hexane at 1,000 (2,780 mg/m^3) ppm (3.5 g/m^3) 6 hours/day on days 8 through 16 of gestation was depressed significantly ($P < 0.05$) compared to controls for up to 3 weeks after birth. However, litter weights of treated pups had returned to control values by 7 weeks after birth. No anatomic defects or neuropathic symptoms were noted in treated pups.

Developmental Effects

- Marks et al. (1980) stated that n-hexane was not teratogenic in mice up to a dose level of 9.90 g/kg/day. In this experiment, pregnant outbred albino mice (CD-1) received n-hexane once daily by gavage at doses up to 2.20 g/kg/day on days 6-15 of gestation. Other pregnant mice received higher hexane doses (up to 9.90 g/kg/day), employing a three times a day injection schedule. At the lower, once-daily doses only one dam died and no teratogenic effects occurred. Higher hexane doses were toxic: 2 of 25 dams treated with 2.83 g/kg/day, 3 of 34 treated with 7.92 g/kg/day and 5 of 33 treated with 9.90 g/kg/day died. At the 7.92 and 9.90 g/kg/day doses, the average fetal weight was significantly ($P < 0.05$) reduced, but the incidence of malformations in treated and vehicle (cottonseed oil) control groups did not differ significantly. Thus, n-hexane was not teratogenic even at doses toxic to the dam.

Mutagenicity

- No information was found in the available literature on the mutagenic effects of n-hexane.

Carcinogenicity

- No information was found in the available literature on the carcinogenic effects of n-hexane.

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:

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

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 Health Advisory

The results of the Hewlett et al. (1980) study in which a group of Sprague-Dawley male rats were given a single oral dose of 1,290 mg/kg n-hexane can be used for the derivation of a One-day HA, even though these studies (in which other chemicals also were screened) were not designed specifically to examine the toxicity of n-hexane. This dose produced no measurable effects on the following parameters after 42 hours: relative liver weight, relative kidney weight, plasma glutamic-pyruvic transaminase, plasma ornithine carbamyl transferase, hepatic and renal histological changes, uptake of p-aminohippurate and tetraethylammonium ion by kidney slices, and blood urea nitrogen. These negative findings (except for body weight at the single data point are consistent with the results reported by Krasavage et al. (1980) in a 90-day study. However, a single oral dose of n-hexane in rats produced minimal changes in renal histology as indicated by the presence of degenerated tubules in sections from these animals.

The One-day HA for the 10-kg child is calculated as follows:

$$\text{One-day HA} = \frac{(1{,}290 \text{ mg/kg/day}) \ (10 \text{ kg})}{(1 \text{ L/day}) \ (1{,}000)} = 12.9 \text{ mg/L } (13{,}000 \text{ ug/L})$$

where:

1,290 mg/kg/day = LOAEL based on minimal adverse effect in male rats.

10 kg = assumed body weight of a child.

1,000 = uncertainty factor, chosen in accordance with NAS/ODW guidelines for use with a LOAEL from an animal study.

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

Ten-day Health Advisory

Appropriate studies for the derivation of a Ten-day HA are not available. Use of the Longer-term HA for the 10-kg child of 4 mg/L is recommended.

Longer-term Health Advisory

The inhalation study by Takeuchi et al. (1980) was not considered because the parameters were not examined in detail. However, a Longer-term HA can be derived from a study (Krasavage et al., 1980) in which Charles River rats were given oral doses of 570 mg/kg n-hexane 5 days/week for 90 days, 1,140 mg/kg or 4,000 mg/kg for 120 days. Clinical and histological signs of neuropathy were absent in the animals at dose levels of 570 and 1,140 mg/kg n-hexane (although body weights were depressed at all three dose levels compared to control). The lowest dose administered (570 mg/kg) can be considered a LOAEL. A safety factor of 1,000 will be used since only one species was considered in the study and the data obtained were part of a broader study dealing with relative neurotoxicity of n-hexane, methyl n-butyl ketone and their metabolites.

The Longer-term HA for a 10-kg child is calculated as follows:

$$\text{Longer-term HA} = \frac{(570\ \text{mg/kg/day})(10\ \text{kg})\ (5)}{(1{,}000)\ (1\ \text{L/day})\quad (7)} = 4.07\ \text{mg/L}\ (4{,}000\ \text{ug/L})$$

where:

570 mg/kg/day = LOAEL based on depressed body weight in animals.

10 kg = assumed body weight of a child.

5/7 = conversion of 5 day/week dosing schedule to 7 day/week.

1,000 = uncertainty factor, chosen in accordance with NAS/ODW for use with a LOAEL from an animal study.

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

The Longer-term HA for a 70 kg adult is:

$$\text{Longer-term HA} = \frac{(570\ \text{mg/kg/day})\ (70\ \text{kg})\ (5)}{(1{,}000)\ (2\ \text{L/day})\quad (7)} = 14.3\ \text{mg/L}\ (14{,}000\ \text{ug/L})$$

where:

570 mg/kg/day = LOAEL based on depressed body weight in rats.

70 kg = assumed body weight of an adult.

1,000 = uncertainty factor, chosen in accordance with NAS/ODW guidelines for use with a LOAEL 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.

Appropriate studies for the derivation of a Lifetime HA are not available at this time. The Krasavage et al. (1980) study was not considered because the data obtained were part of a broader study dealing with other chemicals.

Evaluation of Carcinogenic Potential

- No information was found in the available literature on the carcinogenic effects of n-hexane.
- According to the EPA classification scheme (U.S. EPA, 1986), n-hexane may be classified as Group D.

VI. OTHER CRITERIA, GUIDANCE AND STANDARDS

- An occupational threshold limit value (TLV) of 100 ppm was set by ACGIH (1976).

VII. ANALYTICAL METHODS

- There is no standardized method for the determination of hexane in drinking water samples. However, hexane may be determined by a

purge-and-trap gas chromatographic/mass spectrometric procedure used for the determination of volatile organic compounds in water (U.S. EPA, 1985). This method calls for the bubbling of an inert gas through the sample and trapping hexane on an adsorbant material. The adsorbant material is heated to drive off n-hexane onto a gas chromatographic column. The gas chromatograph is temperature programmed to separate the method analytes which are then detected by the mass spectrometer.

VIII. TREATMENT TECHNOLOGIES

- No data are available on the removal of n-hexane by conventional or other treatment technologies. However, the physical properties and structure of the compound, as well as its similarity to other straight chain aliphatic hydrocarbons, suggest that several treatment methods may be effective in removing n-hexane.

- ESE (1982) considered adsorption a potential treatment technique for hexane on the basis of its structure and low solubility. According to McGuire and Suffet (1980), non-polar saturated hydrocarbons such as hexane should be adsorbed on granulated activated carbon (GAC). However, only limited data demonstrating hexane removal by GAC is available. In a full-scale study, average hexane concentrations in water were reduced from 0.2 ppb to 0.1 ppb on passage through each of two 5 ft diameter (1.6 m), 11 ft (3.4 m) GAC contactors containing Westvaco 12 x 40 GAC. The hydraulic loading for each contactor was 7.4 gpm/ft^2 and the Empty Bed Contact Time was 15.2 min.

- Packed column aeration also may remove n-hexane from drinking water. McCarty et al. (1979) found that the Henry's Law Constant for a chemical is a good indicator of the relative amenability of that chemical to aeration. Accordingly, the Henry's Law Constant for n-hexane (1×10^{-1} atm-m^3/mole) suggests that this substance will be amenable to removal from solution by air stripping. For example, this value is significantly higher than that for chloroform (3.4×10^{-3} atm-m^3/mole), a substance known to be amenable to air stripping (Singley and Bilello, 1981). Air stripping is an effective, simple and relatively inexpensive process for removing many organics from water. However, this process transfers the contaminant directly to 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 potential health hazards associated with the chemical.

- The boiling point of n-hexane (69°C) and of its azeotropic mixture with water [94.4% n-hexane, 61.6°C CCRC, 1979)] suggest that boiling would be an effective means of removing n-hexane from aqueous systems. However, the potential health hazard from hexane inhalation would have to be considered.

- A study reported by Quentin et al. (1977) removed n-hexane in water from 7.3 mg/L to 0.3 mg/L after treatment with alum and a polymeric flocculant. This suggests that a conventional treatment process such as coagulation/sedimentation may be effective in reducing n-hexane.

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