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ENVIRONMENTAL HAZARD ASSESSMENT REPORT

CHLORINATED NAPHTHALENES

DECEMBER 1975

ENVIRONMENTAL PROTECTION AGENCY
OFFICE OF TOXIC SUBSTANCES
WASHINGTON, D.C. 20460

PREFACE

Our society uses thousands of chemical substances, with many of them released into the environment in varying quantities as production or handling losses, as waste materials, or as a direct consequence of their intended or unintended uses. Concern over possible effects of these chemicals has prompted the establishment by the Early Warning Branch of the Office of Toxic Substances of a program to review data on the release, exposure, and effects of chemical substances in order to assist in setting priorities for further study or possible regulatory action.

Detailed analyses on every commercial chemical are not practical. Selected materials are initially screened with a simple literature search; a limited number of these chemicals are selected for more detailed study. Criteria for this selection include volume of production, manner of use, market growth potential, exposure patterns, detection in the environment, known toxic effects, and functional or chemical relationships to known environmental pollutants. Chlorinated naphthalenes were selected for detailed study because of the serious occupational health problems suffered by workers exposed to the compounds, cattle poisoning incidents in the late 1940's and early to mid 1950's, 1972 production levels of some five million pounds, and chemical similarities to polychlorinated biphenyls. The early warning screening system uses diverse sources, including opinions of experts, referrals from other units of government, reports in the scientific and trade literature, predictive modelling, and public inquiries.

These hazard assessments are prepared from reviews of the subject substances supplemented by additional searches and inquiries to obtain the most complete and recent information available. Only data considered pertinent to an assessment of environmental hazard are reported in this series.

Although the assessments use as complete an information base as possible, additional information may be available or may become available. Therefore, these assessments are subject to revisions. The Office of Toxic Substances welcomes any additional pertinent data.

Recommendations in this document are those of the Office of Toxic Substances and may not represent an Agency consensus. Nor do they represent commitment to further action by the Environmental Protection Agency or any other organization. Tradenames and manufacturers are mentioned in this document for purposes of clarity and specificity only and do not constitute an endorsement of any product.

This report was written by Frank D. Kover. The Environmental Hazard Assessment Series is being prepared under the guidance of Dr. Farley Fisher, Chief of the Early Warning Branch, Office of Toxic Substances.

The literature review which preceded this assessment was conducted by Dr. Philip Howard and Mr. Patrick Durkin of the Syracuse University Research Corporation, Syracuse, New York. That review was supplemented by consultations with selected knowledgeable individuals both within and outside the Federal Government and is part of a report entitled Preliminary Environmental Hazard Assessment of Chlorinated Naphthalenes, Silicones, Fluorocarbons, Benzenepolycarboxylates, and Chlorophenols, available through the National Technical Information Service, Springfield, Virginia 22151 (NTIS accession number - PB-238 074/AS).

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Appreciation is expressed to the many individuals who provided information and reviewed drafts of this report. Special appreciation is expressed to the Office of Toxic Substances Staff, and to Dr. J.G. Vos of the Institute of Veterinary Pathology, University of Utrecht, the Netherlands, for valuable comments and information concerning European work on chlorinated naphthalenes and related chlorinated hydrocarbon compounds. Comments and suggestions incorporated into this report were contributed by Guy Nelson, U.S. EPA/NERC-Corvallis; Dr. Donald I. Mount, Director, EPA-National Water Quality Laboratory, Duluth; Dr. Gilman Veith, EPA-National Water Quality Laboratory, Duluth; Dr. Gerald Bowes, California Water Resources Board, Sacramento; John P. Lehman, EPA Office of Solid Waste Management Programs, Hazardous Waste Management Division, Washington, D. C.; George B. Morgan, EPA/NERC-Las Vegas; Dr. Charles F. Jelinek, FDA, Bureau of Foods, Division of Chemical Technology, Washington, D.C.; and Daniel F. McCarthy, U.S. International Trade Commission (formerly U.S. Tariff Commission).

CONCLUSIONS AND RECOMMENDATIONS

The largest use of the chlorinated naphthalenes employs the lower chlorinated compounds in a temporarily "closed" system (automobile capacitor). The extent to which these compounds leach out or are otherwise released from the capacitor to the environment has not been determined; should release occur, they are likely to be readily decomposed.

The more toxic higher chlorinated members of this class are produced at the rate of a half million pounds per year. Whether or not these compounds persist in the environment is unknown, but their chemical similarities to polychlorinated biphenyls (PCBs) arouse some suspicion. In addition, traces of higher chlorinated naphthalenes have been detected in one species of water fowl in the Netherlands. Only two reports of chlorinated naphthalenes in U.S. environmental samples have been cited. Amounts of these compounds released to the environment as a result of their use seem low.

Overall, the available information on the chlorinated naphthalenes suggests that the potential environmental hazard associated with these compounds warrants a moderate level of concern. The available monitoring data from limited U.S efforts could represent just some isolated contamination or indicate a more widespread problem that is just beginning to be detected.

Recommendations

1. The environmental hazard posed by these chemicals should be reassessed if: (a) chlorinated naphthalenes are detected with greater frequency in environmental samples by FDA (food), EPA, or others; (b) production levels of penta- and hexachloronaphthalenes double; or (c) new use(s) of these compounds with higher exposure potential is proposed.

2. The environmental persistence of the higher chlorinated naphthalenes should be determined.

3. Monitoring should be undertaken in the vicinities of electroplating activities discharges, waste oil discharges, electronic parts manufacturing wastes, landfill disposal activities, as well as effluent from the production plant. Samples should be taken from water sediments since these compounds are water insoluble. Some of this monitoring might be carried out in conjunction with future monitoring programs for PCBs and chlorinated hydrocarbons, and particularily pesticides.

4. Further investigation to determine the environmental fate of the penta- and hexachloronaphthalenes will be necessary if monitoring data indicate their presence near electroplating activities.

5. The potential for chlorinated naphthalenes to undergo epoxidation similiar to dieldrin has led to some concern about carcinogenic implications. The metabolic fate of these compounds with regard to epoxide formation should be determined.

SUMMARY OF TECHNICAL DISCUSSION

The chlorinated naphthalenes enjoyed large scale use prior to and during World War II. Capacitor and cable manufacturers used them as dielectrics and water repellents superior to paraffin wax. Other industrial applications of that era included use as additives for high-pressure lubricants, as wood preservatives, and as synthetic waxes and impregnants. Today their major uses are confined to use as a paper impregnant in automobile capacitors (dielectric), and as an oil additive to clean sludge and petroleum deposits from engines. Two minor uses of potential environmental significance are in the electroplating industry (stopoff compounds) and in the fabric dyeing industry.

Available production and market information on these compounds indicates that total production has declined somewhat over the last decade and a half, probably at least in part due to severe occupational skin problems (chloracne) associated with the higher chlorinated compounds, especially the penta- and hexachloronaphthalenes. The lower chlorinated naphthalenes (mono-, di-, tri-, and tetrachloronaphthalenes) form the bulk of today's market and are not associated with severe toxic manifestations.

The levels of chlorinated naphthalenes which are important from a toxicity standpoint show rather wide variation in toxic response from species to species. In general it is somewhat useful to consider the toxicity of the chlorinated naphthalenes to increase with the degree of chlorination. The penta- and hexachloronaphthalenes elicit the most severe toxic responses. Mono- and dichloronaphthalenes have relatively low toxicity. One of the most susceptible animals is the cow.

The lack of homogeneity in human response has prevented the establishment of a no-effect level. Industrial hygiene standards of 0.5 mg/m^3 in air for pentachloronaphthalene and 0.2 mg/m^3 for hexachloronaphthalene are designed to minimize the incidence of chloracne and to prevent liver damage.

A foreign report of traces of chlorinated naphthalenes in one species of fish eating birds appears to demonstrate a potential for bioaccumulation. Only two reports of detection in environmental samples from the U.S. are known.

I. GENERAL INFORMATION

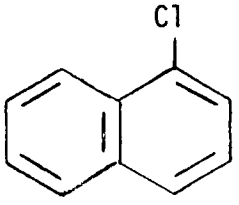
Physical and Chemical Characteristics

In general, the chlorinated naphthalenes are water insoluble waxy solids exhibiting a high degree of chemical and thermal stability. Their physical properties vary with the degree of chlorination. The mono- and dichloronaphthalenes are liquids at room temperature whereas the higher chlorinated compositions are solids. As the chlorine content increases, the specific gravity, boiling point, melting point, fire point and flash point all increase while vapor pressure and water solubility decrease.

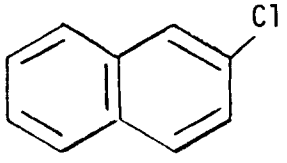
The structure of the chlorinated naphthalenes consists of the naphthalene double ring where any or all of the eight hydrogen atoms can be replaced with chlorine ($C_{10}H_{(8-x)}Cl_x$) (Figure 1.). The commercial products are generally mixtures with different degrees of chlorination and the dominant species is indicated by the percent chlorine content attributed to the product (Table I). No data from manufacturers are available on the ratios among the structural isomers in the commercial products. The commercial products are sold as refined chloronaphthalenes (Table I) and as chloronaphthalene crudes. The product bulletin (Koppers, a) describes the crudes as having essentially the same physical properties as the refined products, but "not held within close limits". It suggests they are suitable for many applications where dark colors are acceptable. Amounts of the crude forms produced are a minor portion of the total production (Hoy, 1975). Possible impurities of these products are chlorinated derivatives, corresponding to the impurities in coal tar, or petroleum-derived naphthalene feedstock which may include biphenyls, fluorenes, pyrenes, anthracenes, and dibenzofurans (Hunt and O'Neal, 1967). Koppers' Research Department analyzed the company's naphthalene feedstock (refined coal tar base) and did not detect any trace of biphenyls or dibenzofurans (Hoy, 1975).

FIGURE 1

MONOCHLORONAPHTHALENES



1-chloronaphthalene



2-chloronaphthalene

TABLE I

COMPARATIVE PROPERTIES OF HALOWAX CHLORONAPHTHALENES (KOPPERS, a)

PRODUCT NUMBER		1031	1000	1001	1099	1099B
1 COMPOSITION		Mono-Chlor	Mono-+Di-Chlor	Tri-+Tetra-Chlor	Tri-+Tetra-Chlor	Tri-+Tetra-Chlor
2 PHYSICAL FORM		LIQUID	LIQUID	FLAKES	FLAKES	FLAKES
3 CHLORINE CONTENT, % (Approximate)		22	26	50	52	52
4 SPECIFIC GRAVITY	@ 25°C	1.20	1.22	1.58	1.59	1.65
	@ 60°C	—	—	—	—	—
5 INITIAL BOILING POINTS	@ 30 MM	144°C	144°C	200°C	205°C	212°C
	@ 100 MM	180°C	180°C	234°C	241°C	248°C
	@ 760 MM	250°C	250°C	308°C	315°C	322°C
6 DISTILLATION RANGE		6% Max. 255°C	—	—	—	—
		95% Min. 265°C	80% Min. 282°C	—	—	—
		98% Min. 275°C	90% Min. 300°C	—	—	—
7 SOFTENING POINT(Melting Point), °C(Approx.)		–25	–33	93	102	115
C.O.C.		135	130	200	210	210
9 FIRE POINT, °C, C.O.C.				None to Boiling	None to Boiling	None to Boiling
10 SPECIFIC HEAT, Gm. Cal./Gm./°C		—	0.40 @ 50° 0.42 @ 100°	0.22 @ 15° 0.66 @ 100°	—	—
11 LATENT HEAT OF VAPORIZATION, Cal./Gm.		—		—	—	—
12 COLOR		White to Pale Straw	White to Pale Straw	White to Pale Straw	White to Pale Straw	Light Yellow
13 ACIDITY, MAXIMUM (Mg. of KOH/Gm.)		0.05	0.05	0.05	0.05	0.05
14 VISCOSITY, SAYBOLT UNIV. SEC. (APPROX.)		35 @ 25°C	34 @ 25°C	30 @ 130°C	31 @ 130°C	31 @ 130°C
15 VOLATILITY	200 Gms. with Surface 9.5 Sq. In for 10 Days @ Room Temp.	1.0%	1.5%	—	—	—
	Gms./Sq. In/Hr. @ 105°C	—	—	0.010	—	—
16 PENETRATION, 200 Gm., 5 Secs. @ 25°C(Approx.)		—	—	—	—	—
17 DIELECTRIC CONSTANT	8 FLASH POINT, °C	—	—	25°C 100°C	25°C 100°C	25°C 115°C
	@ 60 CYCLES/SEC.	—	—	— 4.1	— 4.1	— 4.0
	@ 1000 CYCLES/SEC.	—	—	5.3 4.1	5.3 4.1	5.3 4.0
18 POWER FACTOR	@ 60 CYCLES/SEC.	—	—	— 0.37	— 0.37	— —
	@ 1000 CYCLES/SEC.	—	—	0.002 0.005	0.002 0.005	0.002 .01
19 RESISTIVITY, MEGOHM CENTIMETERS				Over $1x10^{A}$ $1x10^{5}$	Over $1x10^{9}$ $1x10^{5}$	Over $1x10^{8}$ $1x10^{5}$

TABLE I (CONT.)
COMPARATIVE PROPERTIES OF HALOWAX CHLORONAPHTHALENES
(KOPPERS, a)

PRODUCT NUMBER		1013	1014	1051	2141
1. COMPOSITION		Tetra-+Penta-Chlor	Penta-+Hexa-Chlor	Octa-Chlor	Blend
2. PHYSICAL FORM		FLAKES	FLAKES	POWDER	CAKES
3. CHLORINE CONTENT, %(Approximate)		56	62	70	54
4. SPECIFIC GRAVITY	@ 25°C	1.67	1.78	2.00	1.63
	@ 60°C				
5. INITIAL BOILING POINTS	@ 30MM	222°C	242°C	310°C	
	@ 100 MM	258°C	278°C		
	@ 760 MM	328°C	344°C		
6. DISTILLATION RANGE					
7. SOFTENING POINT(Melting Point), °C(Approx.)		120	137	185	135
8. FLASH POINT, °C, C.O.C.		230	250	None to 430	
9. FIRE POINT, °C, C.O.C.		None to Boiling	None to Boiling	None to Boiling	
10. SPECIFIC HEAT, Gm. Cal./Gm./°C			0.19 @ 15° 0.48 @ 100°		
11. LATENT HEAT OF VAPORIZATION, Cal./Gm.					
12. COLOR		Light Yellow	Light Yellow	Light Yellow	Gray White
13. ACIDITY, MAXIMUM(Mg. of KOH/Gm.)		0.05	0.05	0.1	0.05
14. VISCOSITY, SAYBOLT UNIV. SEC.(Approx.)		33 @ 130°C	35 @ 150°C		183 @ 160°C
15. VOLATILITY	200 Gms. with Surface 9.6 Sq. In for 10 Days @ Room Temp.				
	Gms./Sq. In/Hr. @ 105°C	0.005	0.001		0.06 @ 140°C
16. PENETRATION,200 Gm,5 Sec. @ 25°C(Approx.)					24
17. DIELECTRIC CONSTANT		25°C 130°C	25°C 150°C		25°
	@ 60 CYCLES/SEC.	4.8 3.8	4.4 3.7		3.8
	@ 1000 CYCLES/SEC.	4.8 3.8	4.4 3.7		3.8
18. POWER FACTOR	@ 60 CYCLES/SEC.	0.002 0.45	0.0009 0.99		0.0006
	@ 1000 CYCLES/SEC.	0.0003 0.04	0.0002 0.44		0.0002
19. RESISTIVITY, MEGOHM CENTIMETERS		Over 1×10^8 1×10^5	Over 1×10^8 1×10^5		Over 1×10^8

Reactivity with environmental chemical species and potential complex formulations have not been studied. However, as discussed below some insights might be drawn from the chemical similarity of these compounds to polychlorinated biphenyls (PCBs).

Production Levels and Trends

The production process generally involves the chlorination of naphthalene in the presence of a ferric and antimony chloride catalyst. Foreign manufacturers of chlorinated naphthalenes are Bayer in Germany (Nibren waxes) and the Imperial Chemical Industries Ltd. in the United Kingdom (Seekay waxes). Crow (1970) has stated that in the United Kingdom only chlorinated naphthalenes with four chlorines or less are produced and sold. Personal communications with the International Trade Commission (formerly the U.S. Tariff Commission) revealed that the last reported imports of any chlorinated naphthalenes were 53 pounds (24 kg) of 1-chloronaphthalene in 1963 and 12,231 pounds (5,560 kg) of the same in 1964. Since that time no imports of chlorinated naphthalenes (by trade name or chemical name) have been reported.

The only U.S. manufacturer of chlorinated naphthalenes is the Koppers Company which produces them under the trade name of Halowaxes at a plant in Bridgeville, Pennsylvania, a few miles from Pittsburgh. In 1956, the total output was about 7 million pounds (about 3.24 million kilograms) (Hardie, 1964). Hardie (1964) suggested that the decline in use evident at the time was due to their serious disadvantages, such as their toxic nature in handling. In 1972 the market for chlorinated naphthalenes was less than 5 million pounds (2.27 million kilograms) (Koppers, 1973). Recent indications are that the market has continued to decline slightly over the last two years (Hoy, 1975).

Past and Present Use Patterns

Historically, the chlorinated naphthalenes were used in the 1930's and 40's as electrical cable insulating materials where they serve water repellant and flame resistant functions. This use led to recognition of the chlorinated naphthalenes as a serious occupational health problem during cable manufacture. Use of the penta- and hexachloronaphthalenes in cable manufacture was discontinued due to occupational health problems and the introduction of plastics as substitute materials after World War II (Hardie, 1964). Use as electrical insulating material in certain applications remains today mostly for capacitors where lower chlorinated members of the group, which exhibit a low order of toxicity, are employed. Uses as lubricant additive associated with feed pelletizing machinery and wood preservatives, both popular in the 40's and 50's, have been discontinued largely due to serious cattle poisoning incidents associated with those uses in the early 1950's.

Table II lists the various commercial mixtures presently marketed as Halowaxes and indicates the number of chlorines, the approximate percentage of the market and the current principal commercial uses. The tri- and tetrachloronaphthalenes (Halowax 1001 and 1099) are solids and make up more than half of the United States market. They are used almost exclusively as the paper impregnant in automobile capacitors. The second largest part of the market is the mono- and dichloronaphthalenes (Halowax 1000 and 1031 liquids), most of which are used as an oil additive to clean sludge and petroleum deposits in engines. These products find some use in the fabric dyeing industry, specifics about which are considered trade secrets by producers and users. The manufacturer's product bulletin indicates that monochloronaphthalene (Halowax 1031) is about 96% pure, containing predominantly 1-chloronaphthalene, and is used as a raw material for production of dyes.

The highly chlorinated naphthalenes (Halowax 1013 and 1014) are used mainly as electroplating stopoff compounds in relatively small quantities. Some specialized minor uses as an additive in automobile and industrial gear oils and cutting oils are also mentioned in the manufacturers product bulletin (Koppers, a). The product bulletin also mentions other possible minor applications of Halowax 1000 as solution polymerization solvents, gauge fluids, inert liquid seals for instruments, and photoelastic immersion fluids. Halowax 1001 is said to have applications in the paper coating and precision casting industries.

TABLE II
USES OF CHLORINATED NAPHTHALENES
(KOPPERS, 1973)

HALOWAX	% OF CHLORINATED ISOMERS	% MARKET* (1972)	USES
1000 1031	60% MONO 40% DI 95% MONO 5% DI	15–18%	ENGINE OIL ADDITIVE TO DISSOLVE SLUDGE AND DEPOSITS
1000 1031	60% MONO 40% DI 95% MONO 5% DI	10%	PROPRIETARY USES IN FABRIC
1001 1099	10% DI 40% TRI 40% TETRA 10% PENTA	65–66%	IMPREGNANT FOR AUTOMOBILE CAPACITORS
1013 1014	10% TRI 50% TETRA 40% PENTA 20% TETRA 40% PENTA 40% HEXA	8%	MOSTLY AS ELECTROPLATING STOPOFF COMPOUNDS, ALSO IMPREGNANT FOR CARBON ELECTRODES USED FOR CHLORINE PRODUCTION
1051	10% HEPTA 90% OCTA	.5%	UNKNOWN

*BASED ON MARKET OF LESS THAN 2.27×10^8_g (5 MILLION LBS.)

II. ENVIRONMENTAL EXPOSURE FACTORS

The potential for environmental exposure may be significant when these compounds are used as oil additives, electroplating stopoff compounds, and in the fabric dyeing industry. With the latter two uses, effluent discharges from point sources may release these compounds to the environment, while with the former, more widespread non-point sources would be involved. The largest use (about two-thirds of the market) of these compounds is as an impregnant for automobile capacitors. Automobile capacitors can be considered disposable items since they are often changed during engine tune-ups. This use is in a temporarily "closed" system, and the extent to which chlorinated naphthalenes will leach out or be released has not been determined. Previous use in products as insulation, e.g., old cables, could allow entry to the environment as a result of general waste disposal and physical breakdown of the products.

Chlorinated naphthalenes, like PCBs, exhibit a high degree of chemical and thermal stability as indicated by their resistance to most acids and alkalies and resistance to dehydrochlorination (Koppers, a). Although a number of researchers have recognized the similarity between the physical and chemical properties and uses of PCBs and chlorinated naphthalenes (Armour and Burke, 1971; Goerlitz and Law, 1972) and have developed analytical procedures for low level detection in environmental samples, only two reports of chlorinated naphthalenes contamination of the environment in the U.S have been reported. In addition, Koeman et al. (1973) detected traces of chlorinated naphthalenes during PCB and DDE residue analysis in cormorants (fish-eating birds) that were found dead in various parts of the Netherlands. In most cases the analytical procedures were developed to assure that chlorinated naphthalenes were not interfering with analysis for PCBs or organochlorine pesticides such as DDT. Some of the analytical techniques developed, especially gas chromatography-mass spectrometry (GC-MS), would allow detection and quantification of chlorinated naphthalenes in environmental samples.

The Food and Drug Administration (FDA), in their monitoring program for pesticides and other industrial chemicals, such as PCBs, in agricultural products, is able to determine the presence of chlorinated naphthalenes in food (grain, fruits, vegetables, milk, eggs, cheese, fish, etc.). This capability has been available since 1970. To date, no findings have been reported by FDA District Laboratories. This surveillance program is carried out on a continuing basis and would be in a good position to determine whether or not chlorinated naphthalenes become a significant contaminant of agricultural products (FDA, 1975).

Similarly, a personal communication with the EPA National Water Quality Laboratory in Duluth, Minnesota revealed that they have been monitoring samples of fish from the Great Lakes as well as many major rivers in the U.S. and have found no chlorinated naphthalenes as of February, 1975. The analytical chemist did indicate that some tentative findings of chloronaphthalenes had been made which, upon confirmatory analysis, proved to be other chlorinated hydrocarbons. For example, a tentative tetrachloronaphthalene identification was later found to be a compound with the same molecular weight, pentachlorophenol. Another tentative identification of octachloronaphthalene was later determined to be a pentachloroterphenyl with nearly the same molecular weight. One other tentative finding in Great Lakes herring gull extract awaits confirmation at the California Water Resources Control Board. The type of analytical procedures involved in identifying chlorinated naphthalenes seems to make their detection by those doing routine analyses for chlorinated hydrocarbons unlikely unless chlorinated naphthalenes are specifically sought.

A study of the distribution of polychlorinated biphenyls in the aquatic environment by Crump-Wiesner et al. (1973) led to the first report of chlorinated naphthalenes in an environmental sample in the United States. In analyzing sediment samples from a south Florida

drainage ditch, mixtures of chlorinated naphthalenes ranging from 1.25 to 5 mg/kg were found. Water samples overlying the sediments averaged 5.7 µg/l. Identification was confirmed by both microcoulometry and GC-MS. Discussions with the authors revealed that the drainage ditch was in the vicinity of an airport overhaul hangar.

Law and Goerlitz (1974), in a GC-MS study of chlorinated hydrocarbons in bottom material from streams tributary to San Francisco Bay, found 55 µg/kg of chlorinated naphthalenes present in a Guadalupe River sample. The authors point out that the sample came from an area of no apparent industrial activity.

Several instances of a disease called bovine hyperkeratosis (Olson, 1969) in the early 1950's were traced to chlorinated naphthalenes as a contaminant in pelletized cattle feed. This contamination was due to the use of a lubricant containing chlorinated naphthalenes in machines for pelletizing cattle feed. (See Biological Effects - Toxicity below).

Chlorinated naphthalenes have also been detected as a contaminant in foreign commercial PCB formulations (Phenoclor, Clophen and Kanechlor) along with chlorinated dibenzofurans. Early investigators did not detect chlorinated naphthalenes in domestic PCB formulations (Aroclors) (Vos et al., 1970; Roach and Pomerantz, 1974). Chlorinated naphthalenes are present in domestic PCBs but at lower levels than in foreign formulations. Bowes et al. (1975), using a more sensitive analytical technique, identified by MS three peaks of a chromatogram of Aroclor 1254 as chlorinated naphthalenes.

Environmental decomposition of chlorinated naphthalenes has received limited study. Only the monochlorinated naphthalenes have been studied under biological conditions similar to those found in the environment. Walker and Wittshire (1955) examined the decomposition of both 1-chloro- and 1-bromonaphthalene by soil bacteria and found that two species of bacteria, obtained from soil, would grow in a mineral salts medium with 1-chloronaphthalene as the sole carbon source. The isolation of

8-chloro-1,2-dihydro-1,2-dihydroxynaphthalene and 3-chlorosalicylic acid suggests the metabolic route shown in Figure 2. Similar results were reported for 2-chloronaphthalene by Canonica and coworkers (1957).

Okey and Bogan (1965) examined the rate of metabolism of 1-chloro- and 2-chloronaphthalene by sewage sludge bacteria that were first grown on unsubstituted naphthalene. The initial concentration of chlorinated substrates was 1 mg/l and the substrate was the only source of carbon. The following relative rates of metabolism were observed:

naphthalene >>> 2-chloronaphthalene>> 1-chloronaphthalene.

The microbial degradation of the highly chlorinated naphthalenes has not been studied. Gibson (1972) has suggested that the initial reactions in mammalian and microbial systems are quite different as is depicted in Figure 3. There is little certainty about the environmental fate of chlorinated naphthalenes. No literature references are available on the environmental stability and transport of chlorinated naphthalenes within the biosphere, including bioaccumulation and behavior in ecological food chains, although at least a potential for bioaccumulation appears to have been demonstrated since traces have been detected in one species of fish-eating birds (Koeman et al., 1973) and in stream sediments (Crump-Wiesner et al., 1973; Law and Goerlitz, 1974). Further, an evaluation of the physical and chemical data on these compounds together with the available data on mammalian and microbial metabolism and an intuitive correlation based on the similarities in chemical structure and physical properties (low water solubility, low volatility) between PCBs and chlorinated naphthalenes, indicate that the higher chloronaphthalenes are relatively stable and are likely to persist when released to the environment (Howard and Durkin, 1973).

Recent photolysis studies have shown a potential for photodegradation of polychlorinated naphthalenes in the environment. Experiments

FIGURE 2

Suggested Route of Decomposition of
1-Chloronaphthalene by Soil Bacteria.

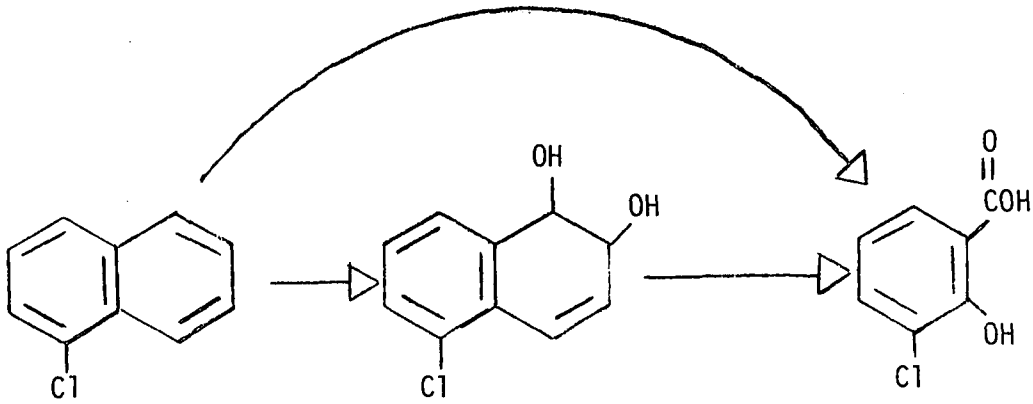
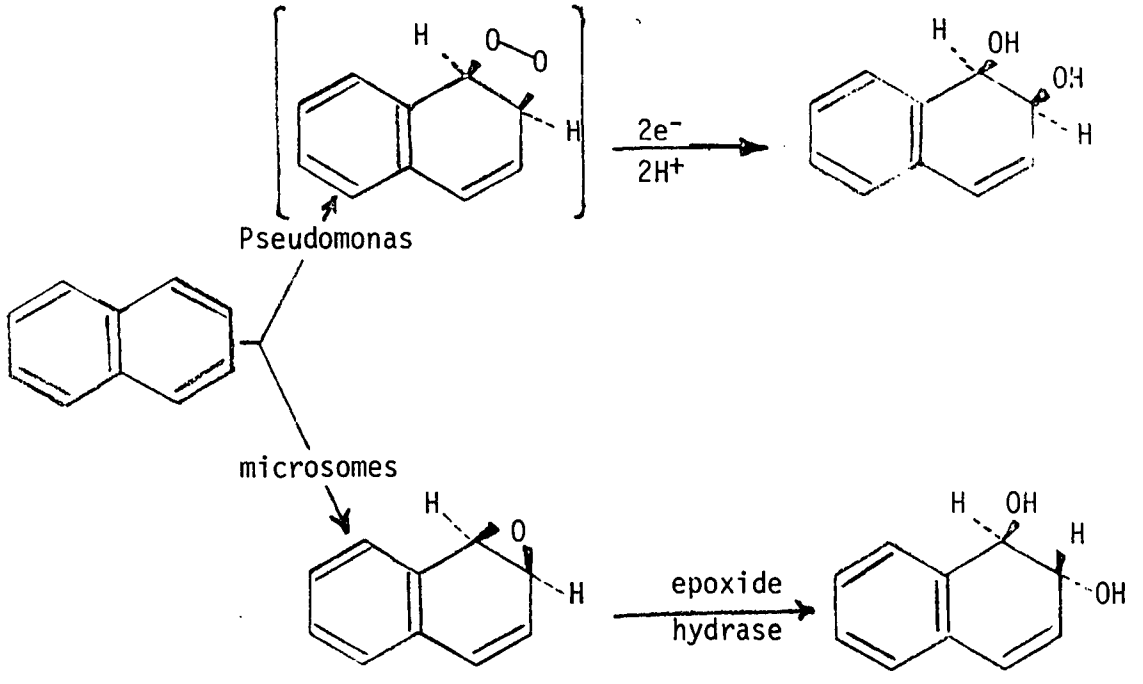


FIGURE 3

Proposed mechanism of naphthalene dihydrodiol formation in mammalian and microbial systems. (From Gibson, 1972)



various polychlorinated naphthalenes in methanol solution irradiated at a peak energy output of 300 nm resulted in dechlorination and dimerization. Sunlight irradiations were carried out on solid films in quartz vessels and resulted in insoluble polymeric material (Ruzo et al., 1975).

Another aspect about which there is little certainty but considerable concern is the potential epoxidation of the chlorinated naphthalenes to produce a relatively small stable agent capable of covalent linkage implicited in carcinogenicity of epoxides like dieldrin (Figure 3).

III. BIOLOGICAL EFFECTS

Metabolic Effects

The primary observed metabolic effect of the chlorinated naphthalenes is to interfere with the metabolism of carotene and its transformation to Vitamin A and is reflected in decreased plasma Vitamin A (Olson, 1969). The Vitamin A effect is highly variable and subject to species-specific variation (Hansel and McEntee, 1955). Goats, sheep, swine, mice, chickens, and rats are much less susceptible than cattle (Olson, 1969).

In the surveyed literature, male rabbits were the only subjects used to study the metabolism of chlorinated naphthalenes (Cornish and Block, 1958). The compounds studied were 1-chloronaphthalene, di-, tetra-, penta-, hepta-, and octachloronaphthalene. Naphthalene metabolites and the presence of unchanged compound were tested for in urine after administration by stomach tube of 1 gram of each test compound. 1-Chloronaphthalene, dichloronaphthalene, and tetrachloronaphthalene showed patterns of excretion similar to naphthalene. The excretion products of naphthalene are largely glucuronides with small amounts converted to mercapturic acid derivatives, sulfates, and phenolic compounds. The higher chlorinated naphthalenes did not yield an increase in these urinary metabolites. Less than 20% of the administered dose of penta- and heptachloronaphthalenes were found to be excreted in urine and feces.

This study suggested that the toxic symptoms produced in the rabbit by highly chlorinated naphthalenes can be related to the inability of the animal to metabolize and excrete these compounds. However, these compounds may be metabolized by pathways which yield excretory products not included in this study, or they may be deposited in the tissue, particularly fat depots, and metabolized or excreted unchanged over long periods of time (Cornish and Block, 1958).

Toxicity

Evaluation of the available toxicity information on the chlorinated naphthalenes indicates that the degree of toxicity, in general, increases with the degree of chlorination. Under acute conditions of human dermal exposure to mono, di, tri, and tetra compounds, slight or no observable reactions were reported. The higher chlorinated members of this class, on the other hand, especially the penta and hexa compounds, have been associated with dermal toxicity (chloracne) and liver damage of some severity under occupational exposure conditions prior to and during World War II years. A few fatalities from chloronaphthalene-induced liver necrosis from occupational exposure have been reported, the last in 1944. Recent study of occupational chloracne problems associated with chlorinated naphthalene exposure has shown limited systemic toxicity and no evidence of liver involvement (Kleinfeld, 1972).

Three natural routes are available for the human intake of chlorinated naphthalenes: ingestion, inhalation, and cutaneous absorption. Of these, Crow (1970) concluded, after a critical review of substances associated with chloracne pathology, that the more important route in occupational exposure is inhalation. The absence of chloracne in workers handling cold chloronapthalene solids (Collier, 1943; Crow, 1970) led to the recognition that the vapors from molten chlorinated naphthalenes are a critical factor in the toxic responses observed. However, the dermal absorption route should not be disregarded. Past occupational studies often failed to characterize adequately the exposure conditions so that the mode of entry in most situations is best considered as a probable combination of vapor inhalation and cutaneous absorption. In domestic animals, ingestion is by far the most common route of exposure and results in the most severe pathology (Huber and Link, 1962; Olson, 1969).

Because chlorinated naphthalenes have never enjoyed widespread household use, occupational rather than accidental or environmental exposure predominates in the relevant literature on human toxic effects. Two clinically distinct but often concurrent and possibly physiologically related syndromes have been described: liver necrosis and chloracne. (Chloracne is a general term and describes the skin irritation that can be produced not only by chlorinated naphthalenes but also by other chlorinated compounds including commercial grade biphenyls, a few specific benzenes, phenols, and dibenzofurans. Chloracne accompanied by itching, however, may be specific to the chlorinated naphthalenes.)

Any attempt to label these syndromes as acute or chronic is potentially misleading. Exposures of three to four months are often noted in the clinical literature (e.g., Schwartz and Peck, 1943; Collier, 1943; Greenburg et al., 1939). Human susceptibility is by no means homogeneous. Standard parameters such as age, sex, weight, general physical condition, and previous medical history show no clear correlation to chloronaphthalene pathogenesis (Greenburg et al., 1939). The situation is further complicated because precise dosage values are often not available. But, if some label is necessary, chronic is perhaps the best compromise, since the disease appears after an appreciable period of exposure and reversals are relatively gradual after exposure is discontinued. A more appropriate approach would probably be in terms of degree of damage as adopted by Collier (1943), i.e., slight, moderate, and severe.

Chloronaphthalene-induced liver necrosis has always been rare, with the last fatal case reported by Strauss in 1944. The worker was employed in proximity to a "Halowax Machine" in a wire cable manufacturing plant. No estimate of exposure was given. Occcasional deaths due to systemic poisoning (acute atrophy of the liver) as well as a number of nonfatal cases of liver disease were reported during the mid-30's to early 40's in workers repeatedly exposed to vapors of chlorinated naphthalenes. Every case but one was attributed to penta- and/or hexachloronaphthalenes (AIHA, 1966).

The symptomatic course of the disease is not unlike that of other forms of liver damage resulting in hepatitis with consequent jaundice, and may be accompanied by nausea, vomiting, loss of appetite, fatigue, fever, and/or acute abdominal pain (Kleinfeld et al., 1972; Collier, 1943). Autopsies of fatally exposed workers revealed severe yellow atrophy of the liver. Most researchers seem to agree that the liver is the primary internal organ directly damaged by chlorinated naphthalenes (Collier, 1943; Straus, 1944; Kleinfeld et al., 1972). Detailed descriptions of the pathology are available in the literature (See especially Greenburg, 1939). Understandably, very few detailed descriptions of liver damage are available for non-fatal exposures (Strauss, 1944).

Three fatal cases investigated by Greenburg (1939) revealed that all were exposed to chlorinated naphthalenes directly or indirectly in the workroom. A 17-year old girl worked at a plant that manufactured electrical condensers for use in radios. The condensers were impregnated with tri- and tetrachloronaphthalene. At the same time she was exposed to vapors of the higher chlorinated naphthalenes from the sealing operations conducted near the soldering tables where she was stationed. After seven months of soldering and labelling condensers which involved her in direct exposure, she became intensely jaundiced and was admitted to a hospital. She died two days later. Death was attributed to acute yellow atrophy of the liver.

Greenburg (1939) also described the fatal cases of two young men who, after working in a factory at coating wire with "wax", became jaundiced after 4 to 5 months of exposure. The wax used in the coating process contained higher chlorinated naphthalenes. The process involved a molten bath of the "wax" through which the wire to be coated was passed. The process was only partially enclosed with exhaust ventilation in use. Both died from acute yellow atrophy of the liver. All three cases reviewed by Greenburg (1939) revealed no predisposing causes for the conditions except exposure to chloronaphathalenes. No medical history suggesting a hepatic disorder prior to exposure was given.

Strauss (1944) reviewed 6 fatal cases related to exposure to Halowax. All deaths were attributed to acute yellow atrophy of the liver resulting from exposure to Halowax fumes in a "war plant". One case revealed a minute amount of arsenic (no quantitation) in the liver which, it was concluded, could have increased the susceptibility of the liver to Halowax exposure. Information given by Strauss on conditions of exposure was incomplete. Also, no information was given on which specific Halowax or chloronaphthalenes were involved.

Kleinfeld et al. (1972) could find no evidence of liver damage in a recent outbreak of chloracne associated with occupational exposure to wax containing a mixture of tetra-/pentachloronaphthalene. The melted wax used to insulate electrical components was applied by immersion. No exposure estimates were given, but Kleinfeld attributed the toxic effects observed to exposures to chlorinated naphthalenes through direct dermal contact and inhalation of vapors as a result of poor industrial hygiene practices, including an inadequate and poorly maintained exhaust ventilation system.

In contrast to the low incidence of liver damage, chloracne resulting from exposure to chlorinated naphthalenes is a common and persistent problem in manufacturing and use. Chlorinated naphthalene dermatitis was reported as early as 1918 (Jones, 1941) and remains a problem in spite of advances in industrial hygiene (Kleinfeld, 1972). The chloracne skin lesion is morphologically similar in all cases and has been referred to as the chloracne cyst--a sore 1 mm to 1 cm in diameter with an ill-defined central opening. These cysts are formed from necrotic material retained in the hair follicle or sebaceous gland and are covered by a horny layer of skin causing a dark crusty appearance (Crow, 1970). Hair follicles swell into acne-type sores and sebaceous glands degenerate. In severe cases, lesions may cover extensive areas of the body.

The lower chlorinated naphthalenes seem to be innocuous with respect to man. Mixtures of mono-/dichloronaphthalene (Halowax 1000) and tri-/tetrachloronaphthalene (Halowax 1001) at 500 mg/g in a mineral oil suspension applied to the human ear caused no response over a 30-day period. A mixture of penta-/hexachloronaphthalene (Halowax 1014) under the same conditions did cause acne, but heptachloronaphthalene (Halowax 1052) and octachloronaphthalene (Halowax 1051) did not (Shelly and Kligman, 1957). Even at concentrations as small as 30 mg/g acetone, typical chloracne developed in six weeks as penta-/hexachloronaphthalene was applied to the backs of human volunteers (Hambrick, 1957).

Chlorinated naphthalene toxicity in birds and non-human mammals has been studied in attempts to understand not only occupational hazards to man but also highly chlorinated naphthalene poisoning of cattle. In order to supplement available human clinical data, investigations have been conducted primarily with controlled exposures of known concentrations to rats. Investigations of cattle toxicity have concentrated primarily on a complete description of the syndrome and on attempts to induce a toxic response in other farm animals under closely monitored conditions. Cattle poisoning as described below usually involves a relatively high dose with rapid physical deterioration. Thus, it may be characterized as acute. By contrast, studies relating to occupational exposure usually involve attempts to elicit a gradual response to a minimum dosage and effects may thus be characterized as chronic.

Highly chlorinated naphthalene poisoning, also referred to as bovine hyperkeratosis or X-disease, was of major economic concern in the United States during the 1940's and 1950's. The disease was caused in most cases by accidental ingestion of chlorinated naphthalenes from lubricants in machines used to make pelletized feed (Crow, 1970). X-disease was also associated with the chemical's use in wood preservatives (Crow, 1970) and its use in wax for binding twine (Bentz and Herdmann, 1955). The relation of chlorination to toxicity in accidental cattle

poisoning seems to agree well with human toxicity experiences in that the penta-/hexachloronaphthalene are usually the toxic agents. However, octachloronaphthalene has been reported as having greater oral toxicity than hexachloronaphthalene in cattle (AIHA, 1966). As with reports on human exposure, detailed dosage data are often lacking in animal studies due not only to use of uncertain concentrations but also to ad libitum exposure.

The pathological course of bovine hyperkeratosis has been described in considerable detail and needs only a cursory examination in this report (Olson, 1969). A primary effect of chloronaphthalene poisoning is to interfere with the biotransformation of carotene to vitamin A. Chronologically, this is one of the first effects of exposure and many of the subsequent symptoms - especially of the skin and horns - may be due to vitamin A deficiency in the blood plasma. Vitamin A depression is quickly followed by inflammation of the oral mucosa, lacrimation, excessive salivation, and irregular food consumption. As the disease progresses, grossphysical effects may include a general thickening of the skin caused by over-development of the skin's horny layer with loss of hair (hyperkeratosis). The horns may show signs of degeneration or irregular growth. With continued exposure, the disease progresses through anemia, dehydration, loss of weight, fever, and death. Liver damage may be severe. (The resemblance of this syndrome to severe chloronaphthalene intoxication in man should be noted but no unequivocal comparisons can be made.) A combination of penta/hexachloronaphthalene at a total dosage of 5.5 mg/kg body weight given orally over a five day period will cause a sharp drop in plasma vitamin A by the end of the third day and depressed plasma vitamin A for over thirty days. A single oral dose of hexachloronaphthalene at 11 mg/kg body weight has caused mortality within two weeks (Olson, 1969).

A recent incident of hyperkeratosis in dairy cows was reported by Vos et al. (1971). The cause was determined to be contamination by PCBs and chlorinated naphthalenes of rubber mats used on the floor of the dairy barn. Skin lesions were localized in those areas where the cows

came in contact with the floor. GC-MS analysis of extracts of the mats revealed the presence of PCBs (60 % Cl) and predominantly hexachloronaphthalenes in a ratio of about 3:1.

Other domestic animals prove much less susceptible to chloronaphthalene poisoning than do cattle. Swine show no toxic effect to hexachloronaphthalene at ten times the above mentioned lethal dosage for cattle. Marked vitamin A depression is noted in swine only at dosages of 154 mg/kg body weight and death does not occur until 198 mg/kg body weight doses are given. Pentachloronaphthalene applied to the skin at 60 mg/liter, 3 liters per day, six times a week for six weeks - 180 mg/day for a total dose of 6.3 g - causes only mild hyperkeratosis (Link et al., 1958). Similar doses administered orally (176-200 mg/kg body weight over a 8-9 day period) cause only slight systemic effects and ataxia (Huber and Link, 1962). Although hyperkeratosis did not result from oral administration, lethal oral doses did result in moderate to severe liver damage ranging from yellow discoloration to swelling and hemorrhage. Following non-fatal oral doses, depression of plasma vitamin A was reversible upon oral administration of vitamin A (Link et al., 1958).

Experimental studies to produce toxic effects in sheep suggested that a ten-fold increase in dose over that required to produce toxicity in cattle is necessary. Sheep apparently have a greater tolerance for these compounds and do not show cutaneous hyperkeratosis or as excessive a drop in the plasma vitamin A level as that observed in cattle. Observed effects included nasal discharge, weakness, loss of weight, loss of appetite, ascites, necrosis and cirrhosis of the liver, and cardiovascular injury (Brock et al., 1957).

Ingestion studies with chloronaphthalenes have been conducted using broad breasted bronze poults (turkeys) and New Hampshire chickens. Feeding in both studies was ad libitum and, given the erratic effect of chloronaphthalene on the appetite, exact dosages cannot be meaningfully

approximated. With the broad breasted bronze poults, a mixture of penta-/hexachloronaphthalene (Halowax 1014), at concentrations of 5, 10, 50, and 100 ppm (mg/kg feed) for 40 days, gives an LC_{50} of 20 ppm with an average decrease in weight of the turkeys of 51 percent. Even at 5 ppm, weight gain was reduced by 33 percent with a 6.5 percent mortality and the prognosis was that prolonged feeding would result in death by marketing age. At 100 ppm, all of the broad breasted bronze poults died within 33 days. Gross histologic examination revealed enlarged and darkened livers as the only histopathologic manifestation, reinforcing the specificity of action found in human exposures. Similar to human topical application, octachloronaphthalenes at 125 ppm in feed caused no significant effect. The investigators speculated, but without elaboration, that this might reflect the high melting point and low solubility of octachloronaphthalene (Pudelkiewicz et al., 1958).

The New Hampshire chicken was studied in a subsequent experiment and found to be appreciably more resistant to penta-/hexachloronaphthalene (Halowax 1014) poisoning. A dose of 100 ppm only prevented egg production in the New Hampshire. With levels of 4, 20, 100, 500 and 2500 ppm in feed over 35 days, 100 percent fatality was achieved only with the highest level after a two week exposure period. A fourfold increase in dietary vitamin A markedly decreased effects. Again, enlarged fibrous livers were the most common pathological finding. Other pathological findings included lack of feather pigmentation and pericardial and peritoneal edema (Pudelkiewicz et al., 1959).

The clinical history of occupational poisoning due to chloronaphthalenes has stimulated much of the work done on "subacute" and "chronic" exposure of non-human mammals. A selective but representative sample of the available data is included in the following discussion. Because the toxic properties of the chlorinated naphthalenes vary considerably with the degree of chlorine substitution, the discussion is presented in ascending levels of chlorination.

Mono and Mono/Di Combinations

These compounds are commonly considered to have low toxicity. No effects to the skin were observed with daily topical application of mono-/dichlorinated naphthalenes to the human ear at 500 mg/g in mineral oil suspension for 30 days (Shelly and Kligman, 1957). However, when applied to the much more sensitive rabbit ear for 5-7 days, 1-chloronaphthalene produces mild reddening at 90 mg/g acetone and severe reddening - but without decrease of sebaceous glands - at 590 mg/g acetone (Hambrick, 1957). Inhalation and ingestion experiments were not encountered in the literature surveyed.

Dichloronaphthalenes

When applied topically to the rabbit ear at concentrations of 45 mg/g acetone and 290 mg/g acetone, dichloronaphthalene produced effects similar to those observed with 1-chloronaphthalene (Hambrick, 1957). When ingested in ad libitum feeding by the rat at 5 g/kg of feed for 15 days, liver weight was increased, growth impaired, and coat texture roughened (Wagstaff, 1971). No inhalation experiments were encountered.

Tri- and Tri/Tetra- Combinations

Topical application of trichloronaphthalenes to mice and rats at an unspecified concentration for 2 hr/day for 40-60 days produced no effects (Shakovskaya, 1953). This is in agreement with a study showing no effects from a mixture of tri-/tetrachloronaphthalenes applied to the human ear at 500 mg/g solvent for 30 days (Shelly and Kligman, 1957).

Experiments feeding trichloronaphthalene to mice at 2.5 mg/mouse/day for 20 days produced no effect (Shakhnovskaya, 1953). However, at 300 mg/rat/day for 9-136 days (total dose of 2.7 g - 41 g), a slight but

progressive increase in fatty accumulation in liver cells was evident (Bennett el al., 1938). Tri/tetrachloronaphthalene at 15 mg/kg body weight/day for 60 days (total dose of .9 g/kg body weight) had no observed effect in rabbits (Greenburg et al., 1939).

Inhalation experiments yield similar results with rats. At 0.05-0.2 mg/l for 2 hrs/day for 20 days and 1.31 mg/m^3 for 16 hrs/day for 134 days, no toxic signs developed (Shakhnovskaya, 1953, Bennett et al., 1938). But at 10.98 mg/m^3 for 16 hrs/day for 102 days, a slight liver discoloration appeared, and 5 percent of the rats showed increased fatty degeneration (Bennett, 1938).

Tetra/Penta- Combinations

With the introduction of the five chlorine atom compound, the first cases of severe poisoning develop. Rats fed 50 mg/rat/day for 63 days (total dose of 2.12 g/rat) became fatally intoxicated, showing jaundice and fatty degeneration of the liver (Bennett et al., 1938). Rabbits seem even more sensitive, with fatal intoxication at 15 mg/kg body weight/day subcutaneously injected for 12-26 days for a total dose of 180-390 mg/kg body weight (Greenburg et al., 1939). No inhalation or topical experiments were encountered.

Penta and Penta/Hexa Combinations

Pentachloronaphthalene alone has received relatively little attention. Applied to swine's skin at 60 mg/liter x 3 liters for 6 day/wk for 4 weeks (180 mg/day, total exposure 43.2 gm)., slight hyperkeratosis was produced (Link et al., 1958).

Combinations of penta-/hexachloronaphthalenes are among the most often cited in human toxicity studies and have also been studied in some detail in non-human mammals. Orally administered penta-/hexachloro-

naphthalenes mixtures have been found to be highly toxic to rabbits and rats. In rats, oral doses of 300 mg/rat/day (maximum dose of .99 g/rat) were fatal in 33 days or less. The livers were markedly yellow and showed signs of extreme fatty degeneration. A dosage of 100 mg/rat/day (.55 g/rat total dose) had the same effect over a 55 day period. Slower and less severe liver damage was noted with a dose of 62.5 mg/rat/day, but further details are not given (Bennett et al., 1938). In rabbits, the lethal dose is 15 mg/kg body weight/day for 12-26 days (total dose of 180-390 mg/kg body weight) with similar organ damage. (Greenburg et al., 1939)

Inhalation studies with rats show a similar dosage/effect relationship. Exposure to 1.16 mg/m^3 for 16 hrs/day for 52 days yields jaundice, enlarged yellow liver, and 69 percent fatality (Bennett et al., 1938).

Applied to the skin of the rabbit ear, 30 mg/g acetone/day for 5 days caused only mild dermatitis with follicular attenuation (Hambrick, 1957). In guinea pigs, a 2.5 mg/kg daily oral dose of technical grade pentachloronaphthalene in peanut oil was fatal after 48 days. Severe weight loss and fatty degeneration of the liver were noted at necropsy (Bentz and Herdmann, 1955).

Hexachloronaphthalene

Like pentachloronaphthalenes, hexachloronaphthalenes have received little attention. In ad libitum feeding to rats, 20 mg/kg and 63 mg/kg in the diet caused weight loss over an 84-day period and 200 mg/kg in the diet caused an unspecified number of fatalities (Weil and Goldberg, 1962). Exposure to the rabbit ear at 30 mg/g acetone for five days caused a decrease in sebaceous gland tissue (Hambrick, 1957).

Heptachloronaphthalene

No "chronic" studies on heptachloronaphthalene were encountered.

Octachloronaphthalene

The toxicity of octachloronaphthalene is somewhat problematical. Most current investigators consider it innocuous (Crow, 1970; Olson, 1969). No significant toxic effects have been observed after testing in man or chicken (Shelly and Kligman, 1957; Pudelkiewicz et al., 1958). However, ad libitum feeding of rats at dietary concentrations of .5 g, 2 g, or 5 g/kg for 22 days has shown a decrease in liver but not plasma vitamin A (Deadrick et al., 1955). Further, a single oral dose of 1 g/rabbit caused death in seven days (Cornish and Block, 1958).

IV. HANDLING PRACTICES, STANDARDS, AND REGULATIONS

The high thermal stability and resistance to chemical attack of the chlorinated naphthalenes reduces any instability problems which might otherwise be encountered during packing and transport. Liquid chlorinated naphthalenes (Halowax 1031 and 1000) are usually shipped and stored in 55-gallon steel drums and are occasionally transported in tank cars. The higher chlorinated solids are usually shipped in small quantities (50 lbs.) in fiber pack containers.

The manufacturer recommends that equipment using the Halowaxes be enclosed, and fumes and vapors be exhausted; individuals having a history of skin disease, liver disorders, or alcoholism should not be employed; work clothing should be completely supplied (including close-weave coveralls, socks, caps, underwear, gloves, and aprons), and the clothing should be changed twice a week; and face and hands should be washed before eating and a shower taken upon quitting work (Koppers, b).

The primary hepatotoxic agents for man seem to be penta- and hexachloronaphthalene (AIHA, 1966). Current industrial hygiene standards (TLV's) are 5 mg/m^3 for trichloronaphthalene, 2 mg/m^2 for tetrachloronaphthalene, 0.5 mg/m^3 for pentachloronaphthalene, and 0.2 mg/m^3 for hexachloronaphthalene (ACGIH, 1975). These levels are recommended to prevent liver damage and to minimize the incidence of chloracne. Where mixtures are used, the limit recommended for the most toxic compound must be taken into consideration when evaluating the exposure (ACGIH, 1971). These ACGIH standards are identical to those adopted by the Occupational Safety and Health Administration in 1971 as occupational exposure limits.

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15. Supplementary Notes

This is the first in a series of reports to be released by OTS/EPA which evaluate chemical compounds for environmental hazard potential.

16. Abstracts

The report is an analysis of available information on chlorinated naphthalenes pertinent to an assessment of the potential environmental hazard posed by these compounds. Aspects discussed are environmental exposure factors, biological effects, general information on uses, production and chemical properties as well as associated handling practices, and applicable standards and regulations. Conclusions as to current hazard potential are presented and recommendations for further study made.

17. Key Words and Document Analysis. 17a. Descriptors

naphthalene compounds
production capacity
marketing
utilization
toxicity
chemical properties
toxicology
occupational diseases
pollution

17b. Identifiers/Open-Ended Terms

chlorinated naphthalenes
environmental exposure
environmental effects

17c. COSATI Field/Group 06/A,C,F,J,M,O,T 07/C,D

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