Environmental Protection Agency
Office of Enforcement

TOXICITY OF ORGANIC COMPOUNDS FOUND IN PETROCHEMICAL EFFLUENTS

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National Enforcement Investigations Center
Denver, Colorado

CONTENTS

INTRODUCTION 1

TOXICITY INFORMATION 4

Acenaphthene 4
Butylphenol 4
2-*sec*-Butylcyclohexanol . . . 4
Cyclohexanone 5
2-Cyclohexylcyclohexanone . . 6
Cyclohexyl chloride 7
Dichlorobenzene 7
1,4-Dimethylcyclohexane . . . 8
3,6-Dimethyl-6-isopropyl-2-cyclohexanone 8
2,5-Dimethyltetradecane . . 8
Diphenyl 9
n-Dotriacontane 9
Ethoxyethyl acetate 9
Ethyl phthalate 9
Bis-(2-ethylhexyl) Fumarate .10
n-Hentriacontane 10
n-Heptacosane 10
n-Hexacosane 10
1-Methylfluorene 10
Methylindole 11
1-Methylnaphthalene . . . 12
Naphthalene 12
n-Nonacosane 12
2-Nonanone 12
n-Octacosane 13
n-Pentacosane 13
Phenanthrene 13
Phenyl Ether 13
RDX 13
Stilbene 15
Styrene 16
TNT 17
n-Tetracosane 19
n-Tetratriacontane . . . 19
n-Triacontane 19
Trichloroaniline 20
Trichlorobenzene 20
n-Tricosane 21
n-Tritriacontane 22
Xylene 22

INTRODUCTION

The National Enforcement Investigations Center has assessed the toxicity of organic compounds found in effluent discharges from the petrochemical industry. Among the on-line, bibliographic computerized data bases used to locate information on the various compounds were ENVIRONS, TOXICON, AMIC, MEDLINE, WIRSIC, EIS, Biological Abstracts and Chemical Abstracts. A profile was created in which the compound names were weighted against these terms:*

Aquatic
Bay
Bays
Biochem Oxygen Demand
Biochemical Oxygen Demand
Biol Oxygen Demand
Biological Oxygen Demand
B.O.D.
BOD
Brook*
Canal
Canals
Chem Oxygen Demand
Chemical Oxygen Demand
Coast
C.O.D.
COD
Contaminated Discharge*
Creek*
Delta
Deltas
Degener*
Degrad*
Disposal
Effluent*
Environmental Hazard*
Estuar*
Fate
Fresh Water*
Freshwater*
Gulf
Houston Ship Channel
Industrial Discharge*
Intertidal
Lagoon*
Lake*
Legal Tolerance*
Littoral
Marine*
Municipal Discharge*
Natural Water*
Ocean*
Outfall*
Persistence
Pond
Ponds*
Residue*
River*
Rivulet*
Salt Water*
Saltwater*
Sea
Seas
Seawater*
Sewage
Sewerage
Shallow water*
Stream*
Surface Water*
Surfacewater
Tidal
Tide
Tides
TOD
T.O.D.
Total Oxygen Demand
Tributar*
Water Way*
Waterway*
Waste*
Water Pollut*
Waters*

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* *Words followed by an asterisk received a "stem search," meaning that all endings such as singular, plural, participial and adverbial were searched.*

Of the 108 references found for 70 compound names, 13 names were cross-referenced and no specific information was found for 30 compounds.

This report contains information available as of July 1974 on the adverse effects of these 40 compounds:

Acenaphthene
Butylphenol
2-*sec*-Butylcyclohexanol
Cyclohexanone
2-Cyclohexylcyclohexanone†
Cyclohexyl chloride
Dichlorobenzene
1,4-Dimethylcyclohexane
3,6-Dimethyl-6-isopropyl-2-cyclohexanone
2,5-Dimethyltetradecane††
Diphenyl
n-Dotriacontane††
Ethoxyethyl acetate
Ethyl phthalate
Bis-(2-ethylhexyl) Fumarate
n-Hentriacontane††
n-Heptacosane††
n-Hexacosane††
1-Methylfluorene
Methylindole
1-Methylnaphthalene
Naphthalene
n-Nonacosane††
2-Nonanone
n-Octacosane††
n-Pentacosane††
Phenanthrene
Phenyl Ether
RDX
Stilbene
Styrene
TNT
n-Tetracosane††
n-Tetratriacontane††
n-Triacontane††
Trichloroaniline
Trichlorobenzene
n-Tricosane
n-Tritriacontane††
Xylene

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† *Cross-referenced under Cyclohexanona*
†† *Cross-referenced under n-Tricosane*

There was no specific information for these 30 compounds, even though they were found in discharges from the petrochemical industry:

Acenaphthalene
1-Bromo-2-chlorobenzene
Cadalene
m-Chloroaniline
Chloroheptadecane
Chlorohexadecane
Diisobutyl phthalate
1,4-Dimethylnaphthalene
1,6-Dimethylnaphthalene
2,2-Dimethyloctanol
4,4-Dimethyl-1-pentene
Di-*n*-butylketone
Di-*n*-octyl-phthalate
Di-(-2-ethylhexyl)adipate
1,11-Dodecadiene
2-Ethyl-1-Hexanol
Fluorocyclohexane
Hexamethylbenzene
Indene
2-Isopropyl-1,3-dioxolane
Isopropylnaphthalene
1-Methoxy-1-octooxyethane
3-Methylindene
2-Methylindene
Methylisopropylnaphthalene
2-Methylnaphthalene
p-(1,1,3,3-tetramethylnaphthalene)-Phenol
Trimethylnaphthalene
2,3,4-Trithiopenthane

TOXICITY INFORMATION

ACENAPHTHENE Used as an insecticide and fungicide.

Merck Index, 1970

N. Marimuthammal. Mutagenesis of Sugar Cane. 1. Effects of Chemical Mutagens. Proceedings of Indian Academy of Science Section B 68(3):131-142, 1968

J. F. Mesquita. Alterations of Cell Division in Allium cepa Root Meristen Cells Treated with Acenaphthene, C. R. Academy of Sci. Ser. D 265(4): 322-325, 1967

BUTYLPHENOL May be irritating to eyes and mucous membrane.

Merck Index, 1970

2-*sec*-BUTYLCYCLOHEXANOL This compound is a metabolite of butylcyclohexanone.[1] Cyclohexanol, like cyclohexanone, is a moderately toxic compound that possesses a high degree of cytogenetic activity. Savelova, Bruk, Klinkinan and Russkikh, based on toxicological considerations, recommended 0.5 mg/l of cyclohexanol in water be adopted as the limit of allowable cyclohexanol concentration in natural basin water.[2] However, more recent experiments have shown that cyclohexanol is a cytogenetic metabolite of cyclamate which has been banned for human consumption. *In vitro* experiments on human leukocyte cultures with cyclohexanol showed the cytogenetic effects of chromosome breaks, deformities, size abnormalities and achromatism.[3] Cyclohexanol had a thermodynamic activity in the range of 0.001 to 0.1 which gave a threshhold narcosis 50 toxicity to barnacle larvae with active appendages, but no forward movement in Elminius modistus larvae after 15 minutes.[4]

[1] *K. L. Cheo, T. H. Elliott and R. C. Tao. The Metabolism of Isomeric Tertbutylcyclohexanones. J. of Biochemistry, 104:198-204, 1967*

[2] *V. A. Savelova, E. S. Bruk, M. V. Klinkinan and V. V. Russkikh. Experimental Determination of the Limit of Allowable Cyclohexanol Concentration in Natural Basin Water. USSR Literature on Water Supply and Pollution Control, 5:40-58, 1966, US Dept. of Commerce*

[3] *J. P. Collin, E. P. Gondry, J. Lederer and A. M. Pottler-Arnould. Cytogenetic and Teratogenic Action of Cyclamates and its Metabolites. J. of Therapeutique, 47:357-363, 1971*

J. P. Collin. Cytogenetic Effect of Sodium Cyclamate, Cyclohexanone, and Cyclohexanol, in French. Diabete, 19:215-221, 1971

[4] *D. J. Crisp, A. O. Christie, and A. F. Ghobashy. Narcotic and Toxic Action of Organic Compounds on Barnacle Larvae. Compendium of Biochemistry and Physiology, 22:629-649, 1967*

CYCLOHEXANONE

Cyclohexanone has rather moderate toxicity but it is extremely potent cytogenetically. For example, its median lethal dose to rabbits is 1,000 ppm, to mice 1,950 ppm, and to rats 3,460 ppm.[1] Vertebnaya and Mozhaev likewise reported cyclohexanone to be a ketone of low sanitary-toxicological characteristics and suggested a limit of 1 mg/l in water basins.[2] However, more recent experiments have shown that cyclohexanone is a cytogenic metabolite of cyclamate which has been banned for human consumption. *In vitro* experiments on human leukocyte cultures with cyclohexanone showed the cytogenic effects of chromosome breaks, deformities, size abnormalities and archromatism.[3] In animal experiments, cyclohexanone has been found to be a potent inducer of cataracts.[4] With a thermodynamic activity in the range of 0.001 to 0.1, cyclohexanone gave a threshold narcosis 50 toxicity to barnacle larvae with active appendages but with no forward movement in Elminius modestus larvae after 15 minutes.[5] The biochemical purification of caprolactam wastes in the presence of domestic sewage reduced the concentration of cyclohexanone from 180 mg/l to nil.[6]

In die-away tests at about 18°C in a dilution water seeded with acclimatized activated sludge, 60 or 200 mg/l of cyclohexanone as sole carbon source was readily degraded, and COD removal was 96% in 2 days.[7]

[1] *Technical Assistance Data System: 72T16661, ENVIRONS, EPA*

[2] *P. I. Vertebnaya and E. A. Mozhaev. Limits of Allowable Concentrations of Methylethylketone and of Cyclohexanone in Water Basins. USSR Literature on Water Supply and Pollution Control, 3:30-34, 1962, US Department of Commerce*

[3] *J. P. Collin, E. Condry, J. Lederer and A. M. Pottler-Arnould. Cytogenetic and Teratogenic Action of Cyclamate and its Metabolites. Therapeutique, 47:357-363, 1971*

J. P. Collin, Cytogenetic Effect of Sodium Cyclamate, Cyclohexanone, and Cyclohexanol, in French. Diabete, 19:215-21, 1971

[4] *R. H. Rengstorff, J. P. Petral and V. M. Sim. Cataracts Induced in Guinea Pigs by Acetone, Cyclohexanone and Dimethyl Sulfoxide. Am. J. Optom., 49:308-29, 1972*

[5] *D. J. Crisp, A. O. Christie and A. F. Ghobashy. Narcotic and Toxic Action of Organic Compounds on Barnacle Larvae. J. Compendium of Biochemistry and Physiology, 22:629-649, 1967*

[6] *E. M. Arnoldov. Purification of Waste Waters and the Construction of Purification Installations at Enterprises of the Chemical Industry of the Donets Council of National Economy. Ochistka Ispolz. Stochn. Vod. prom. Vybrosov, Klev, in Russian, 1964, 40-45; Chem. Abstr. 63:11158, 1965*

[7] *P. Pitter and M. Kozderkova. Relation Between the Molecular Structure and Biological Degradability of Organic Compounds. 1. Biodegradability of Hydroaromatic and Cycloaliphatic Compounds by Activated Sludge. Sb. vys. 3K. Chem.-Technol. Praze 16:53-72, 1971*

2-CYCLOHEXYL-CYCLOHEXANONE — See CYCLOHEXANONE

CYCLOHEXYL CHLORIDE

Has suffocating odor.[1] The mosquito fish, Gambusia affinis had a TLM_{96} of 15 ppt.[2] Experimental animals exposed to 0.25 to 25 mg/l for six months exhibited disturbed conditioned reflexes.[3]

[1] *Merck Index, 1970*

[2] *J. E. Wallen, W. C. Greer and R. Lasater. Stream Pollution Toxicity to Gambusia affinis of Certain Pure Chemicals in Turbid Waters. Sewage and Industrial Wastes 29(6):695-711, 1957*

[3] *V. N. Orlavskii. Effect of Chlorocyclohexane on the Organoleptic Properties of Water and the Sanitation Conditions of Water Basins (in Russian). VOPR, Gig. NASELEN. NEST. 4:199-203, 1963*

DICHLOROBENZENE

Can cause injury to livers and kidneys. High concentrations cause CNS depression.[1] In pure cultures of the following marine plankton, no growth occurred but organisms were viable at 13 ppm: Protococcus sp., Chlorella sp., Dunaliella euchlora, Phaeodactylum tricornutum, and Monochrysis lutheri.[2] The toxicity of chlorobenzene and dichlorobenzene is on the same level; increasing the number of chlorine atoms in a benzene molecule does not affect the toxic action but affects only the degree of expressivity. In larger doses, the toxicity of dichlorobenzene depends more on the spatial distribution of chlorine atoms rather than their number; e.g., the ortho-isomer is more toxic than the para-isomer. In determining the maximum permissible concentrations of these compounds in bodies of water, chronic experiments were conducted with white rats to study acute intoxication, their effects on higher nervous activity, erythropoiesis, urinary 17-ketosteroids, and carcenogenic action. The action of the compounds was practically the same. Conditioned reflex activity was depressed showing a cerebral cortical effect; erythropoiesis was significantly decreased, with chlorobenzene producing eosinophila and ortho-dichlorobenzene, neutropenia. Ortho-dichlorobenzene, more than chlorobenzene, led to a sharp rise in urinary steroids. Although both benzenes increased, tissue acid phosphatase and sharply decreased tissue alkali phosphatase, no sign of carcenogenic action was found macroscopically, histologically, or histochemically. The maximum permissible concentration according to organoleptic

effect for chlorobenzene was determined to be 0.01 mg/l; for ortho-dichlorobenzene, 0.002 mg/l; and for para-dichlorobenzene, also 0.002 mg/l.[3]

[1] *Merck Index, 1970*

[2] *R. Ukeles. Growth of Pure Cultures of Marine Phytoplankton in the Presence of Toxicants, Applied Microbio. 10(6):532-537, 1962*

[3] *S. P. Varshavskia. The Comparative Sanitary and Toxicological Characteristics of Chlorobenzene and Dichlorobenzene (Ortho- and Para-Isomers) from the Point of View of Sanitation of Water Reservoirs. In Russian, Gigiena i Sanit. 33(10):15-21, 1968*

1,4-DIMETHYL-CYCLOHEXANE

No information has been received on this compound. However, its toxicity will be similar to that of cyclohexane. Fathead minnows had a TLM_{96} of 30 ppm to cyclohexane; bluegills had a TLM_{96} of 31 ppm; goldfish had the TLM_{96} of 33 ppm; and guppies had a TLM_{96} of 48 ppm.

Technical Assistance Data System: 72T16659, ENVIRONS, EPA

3,6-DIMETHYL-6-ISOPROPYL-2-CYCLOHEXANONE

2-cyclohexanone is a fungal metabolite of cyclohexene which can be further reduced enzymatically.

P. K. Bhattacharyya and K. Ganapathy. Microbiological Trends, Formations of Terpenes. VI. Studies on the Mechanism of Some Fungal Hydroxylation Reactions with the Aid of Model Systems. The Indian J. of Biochemistry, 2:137-145, 1965

E. Boyland and L. F. Chasseaud. Enzymes Catalyzing Conjugations of Glutathione with Alpha, Beta-Unsaturated Carbonyl Compounds. Biochemistry J., 109:651-661, 1968

2,5-DIMETHYL-TETRADECANE

See *n*-TRICOSANE

DIPHENYL — CNS depression, paralysis, and convulsions have been observed in experimental animals.

Merck Index, 1970

Additional information:

R. Viviani, G. Crisetig, V. Petruzzi, and P. Cortesi, Residues of Polychlorinated Biphenyls in Muscle Tissue of Clupeoid Fish in the Adriatic Sea. In Italian, Atti Soc. Ital. Sci. Vet. (ASISAI) 25:429-433, 1971

J. L. Mosser, N. S. Fisher and C. F. Wurster. Polychlorinated Biphenyls and DDT Alter Species Composition in Mixed Cultures of Algae, Science 176(4034):533-536, 1972

D. J. Wilpish. Polychlorinated Biphenyls (PCBs) in Seawater and Their Effects on the Reproduction of Gammarus oceanicus, Bulletin of Environmental Contamination and Toxicology 7(2):182-187, 1972

n-DOTRIACONTANE — See *n*-TRICOSANE

ETHOXYETHYL ACETATE — This compound is commonly known as Cellosolve acetate. It is produced by the Dow Chemical Company, Midland, Michigan; Eastman Kodak Company, Kingsport, Tennessee; Union Carbide Corporation, South Charleston, South Carolina; and the Olin Corporation, Brandenburg, Kentucky.[1] In spite of its intensive production and common availability, no references to its aquatic toxicity have been located.

[1] *Technical Assistance Data System: 72T16721, ENVIRONS, EPA*

ETHYL PHTHALATE — The lethal dose orally in rabbits is 1.0 g/kg.[1] Chronic toxicity tests with phthalic acid, to which ethyl phthalate will hydrolize in water, on laboratory animals at an exposure rate of 0.56 mg/kg daily for 6 months reduced thrombocyte concentrations, increased bilirubin excretion and caused morphological changes in internal organs. Based on this test, a maximum permissible level of phthalic acid in reservoir water was set at 0.5 mg/l.[2]

[1] *Merck Index, 1970*

[2] *K. F. Meleshchenko, N. V. Mironets, and R. K. Rozhkoveiskaya. Experimental Data to Substantiate the Maximum Permissible Concentration of Phthalic Acid (Phthalic Anhydride) in Water Reservoirs. In Russian, Gigiena i Sanit. 32(8):12-15, 1967*

Additional Information:

K. F. Meleshchenko. Maximum Permissible Concentration of Phthalic Acid (Phthalic Anhydride) in Water Bodies. Gigiena i Sanit. 32:167-171, 1967

bis-(2-ETHYLHEXYL) FUMARATE — This compound is an ester which is an extremely effective mosquito repellent, having a space residual time of at least 100 days.

H. Gouck, T. P. McGovern and M. Beroza. Chemicals Tested as Space Repellents Against Yellow Fever Mosquitoes. I. Esters. J. Econ. Entomol., 60:1587-1590, 1967

n-HENTRIACONTANE — See *n*-TRICOSANE

n-HEPTACOSANE — See *n*-TRICOSANE

n-HEXACOSANE — See *n*-TRICOSANE

1-METHYLFLUORENE — This compound, also known as ortho-biphenylenemethane, biphenylenemethane or 2, 2-*p*-methylenediphenyl (CAS Registry No. 1730376), has intense antitumor activity in many of its derivatives.

K. Agrawal. Fluorene Derivatives for Antitumor Activity, J. Med. Chem. 10(1):99-101, 1967

H. L. Pan and T. L. Fletcher. Derivatives of Fluorene XXI. New halogenofluorenes. II. Further Potential Antitumor Agents, J. Med. Chem., 8(4):491-497, 1965

H. L. Pan and T. L. Fletcher. Derivatives of Fluorene XXX. Rearrangement and Antitumor Activities on Some 9-Oxo-fluorene Oximes. 1. 6 5H-Phenanthridinones, J. Med. Chem., 12(5):822-825, 1969

METHYLINDOLE

There are several methylindoles, the most common being 3-methylindole, also known as skatole, which is infamous because it is the aromatic fraction which gives feces their distinctive aroma.[1] In addition to the distinctive aroma, indoles have been implicated in arthritis. A single injection of 0.26 µmoles of skatole once a week for 6 weeks elicited chronic arthritis. The arthritogenic effect of indolic substances is related to their lipophilic activity, which facilitates the diffusion of such substances into the synovial membranes.[2] The indoles have a negative effect upon tissue respiration by inhibiting cerebral oxygen consumption at concentrations below the pathological range.[3] In carefully controlled studies, compounds similar to the indoles which might be suspected of producing the noted effects were found not to have arthritogenic properties.[4] The indoles have also been implicated in pulmonary edema and emphysema.[5]

[1] *Technical Assistance Data System: 72T16884, ENVIRONS, EPA*

[2] *I. Nakoneczna, J. C. Forbes, and K. Rogers. Arthritogenic Effect of Indole, Skatole, and Other Tryptophan Metabolites in Rabbit. Amer. J. Pathol., 57:523-538, 1969*

[3] *P. T. Lascelles and W. H. Taylor. The Effect Upon Tissue Respiration in vitro of Metabolites Which May Accumulate in Hepatic Coma. J. Olin. Sci., 35:63-71, 1968*

[4] *K. S. Rogers, J. C. Forbes and Nakoneczna. Arthritogenic Properties of Lipophilic, Aryl Molecules. Proc. Soc. Exp. Biol. Med., 131:670-672, 1969*

[5] *J. R. Carlson, M. T. Yokoyama and E. Dickinson. Induction of Pulmonary Edema and Emphysema in Cattle and Goats with 3-Methylindole. Science, 176:298-299, 1972*

1-METHYL-NAPHTHALENE

Probably toxic.

Merck Index, 1970

NAPHTHALENE

A large dose by ingestion, inhalation, or skin absorption causes nausea, vomiting, headache, diaphoresis hematuria, hemolytic anemia, hepatic necrosis, convulsions and coma.[1] The fish, Gambusia affinis, in static acute bioassay had a TLM_{48} of 165 ppm.[2]

[1] *Merck Index, 1970*

[2] *I. E. Wallen, W. C. Grier and R. Lasater. Toxicity to Gambusia affinis of Certain Pure Chemicals in Turbid Waters, Sewage and Industrial Waste 29(6):695-711, 1957*

Additional Information:

B. De Jong. Contamination of Ground Water by Organic Substances in the Intake Area of Two Water Works. In German, Vom Wasser (VJWWAU) 38:141-156, 1971

n-NONACOSANE

See *n*-TRICOSANE

2-NONANONE

This compound has very potent biological activity as it is closely related to an alarm pheromone produced by the ant, Iridomyrmex pruinosus. 2-heptanone is produced by this species as an alarm pheromone. Laboratory and field studies with 2-nonanone showed it to be an alarm behavior-producing agent of similar activity to 2-heptanone.[1] In studies on the growth of the fungus Dipodascus aggregatus in culture media containing 2-nonanone there was an insignificant increase in growth.[2]

[1] *M. S. Blum Thomas, S. L. Warter and J. G. Traynham. Chemical Releasers of Social Behavior. VI. The Relaxation of Structure to Activity of Ketone as Releasers of Alarm for Iridomyrmex pruinosus. J. Insect Physiology, 12:419-427, 1966*

[2] *A. Hyman. Affect of Various Allphatic Aldehydes and Related Compounds on the Growth of Dipodascus aggregatus. J. Physiology of Plants, 22:1322-1328, 1969*

n-OCTACOSANE See *n*-TRICOSANE

n-PENTACOSANE See *n*-TRICOSANE

PHENANTHRENE Compound can cause photosensitization of skin and is considered a carcinogen.

Merck Index, 1970

Additional information:

B. H. Grossmann. Therapeutic Effects of Fluorene, Phenanthrene, and Xanthrene Derivatives on Fungal Diseases of Tomato, Nature 227(5264):1267-1268, 1970

PHENYL ETHER Chronic toxicity to warm blooded animals of 2.0 mg/kg daily was an ineffective dose during sanitary toxicological tests.

G. F. Amirkhanova and Z. V. Latypova. Experimental Basis for the Maximum Permissible Concentration of Diethyl Ether in Reservoir Waters. In Russian, Prom. Zagryazneniya Vodoemov No. 9:148-157, 1969

RDX RDX is also known as Hexahydro-1,3,5-trinitro-*s*-triazine, cyclotrimethylenetrinitramine, cyclonite, and hexogen. In a case of accidental industrial poisoning with RDX, human subjects lapse into unconsciousness with no advance warning. Unconsciousness lasted from several minutes to 24 hours, and upon recovery there were headaches, periods of stupor, nausea, disorientation, vomiting and weakness. No other abnormal physical findings were found, and there were no changes in the blood or urine. Treatment was supported and recovery was apparently complete with no sequelae.[1] Data concerning toxicological effects of RDX to humans are extremely limited. Seventeen cases of toxic reactions which occurred between 1939 and 1942 in

Italian workers handling powdered RDX in the drying, cooling, sealing and packing process of its manufacture have been described.[2] Ten had generalized convulsions of clonic-tonic type followed by postictal coma; four had loss of consciousness without convulsions; two had vertigo; and one had vomiting and confusion. Similar cases in German workers handling finely pulverized RDX powder have been described.[3] The author experienced no fatal cases; however, he alluded to German newspaper reports in the post World War II period that paper bags used for wrapping food after having been used for packaging RDX caused four deaths. Oral ingestion of RDX by rats or dogs resulted in hyperirritability, viciousness, generalized convulsion of a clonic-tonic type and death.[4] Similar symptoms have been noted.[5] In acute toxicity studies of rats the LD_{50} was found to be approximately 200 mg/kg in non-fasting rats, and 50 to 100 mg/kg in fasting rats. In chronic toxicity studies, the LD_{50} was approximately 50 mg/kg ingested daily, with a wide variation in the total dose that was fatal. In cases of acute poisoning, the vascular supply of the central nervous system appeared affected through changes in the fibrous material of the vessel wall; degeneration of the nerve cells was also observed. The most affected area was the spinal cord; less so the brain stem; and least the cortex. In chronic exposures, not only the central nervous system but also the liver, lungs, and heart were involved. Principal changes occurred again in the fibrous material blood vessels, leading to impaired blood circulation and metabolism. The fatty acid metabolism especially was affected, and secondary degeneration occurred throughout the organ systems. It was found that RDX injected intraperitoneally caused convulsions and death in rats in 9 to 121 minutes; subcutaneous and intravenous injection of RDX also caused a rapid onset of convulsions. Doses as low as 10 mg/kg intraperitoneally and 18 mg/kg intravenously caused death. Thus, relatively small quantities of RDX, if absorbed, are capable of causing toxic symptoms and death in laboratory animals.

[1] *A. S. Kaplan, O. F. Berghout and A. Peczenik. Human Intoxication from RDX. Arch. Environ. Health, 10:ISS 6, 877-883, 1965*

[2] *M. Barsotti and G. Crotti. Epileptic Attacks as Manifestations of Industrial Intoxication Caused by Trimethylenetrinitroamine, Lavoro 40:107-112, 1949*

[3] *W. Vogel. Hexogen Poisoning in Human Beings, in German. Zbl Arbeitsmed 1:51-54, 1951*

[4] *W. F. Von Oettingen, et al. Toxicity and Potential Dangers of Cyclotriethylenetrinitramine. J. of Industrial Hygiene, 31:21-31, 1949*

[5] *F. W. Sunderman, J. K. Clark and E. S. Bills. Compilation of Informal Monthly Reports on Hazards to Health of Individuals Working with RDX, May 1943-June 1944, National Defense Research Committee of the Office of Scientific Research and Development, NORC Contract No. OEM sr-962, unclassified, 1944*

STILBENE

This compound, also known as bibenzal, or bibenzylidene (CAS Registry Number 588590), is isosteric with azobenzene. In view of the apparent importance of molecular shape in compounds having carcenogenic and tumor inhibitory activity, stilbene derivatives should have such activity. In fact, stilbene was first produced because it was suspected that it would have an acaricidal activity.[1] Because stilbene is estrogenic it was thought that it might prevent heart failure in middle-aged males without producing secondary female sex characteristics. This was found to be the case. Stilbene itself is considered to be non-carcenogenic.[2] In addition to the intense estrogenic activity,[3,4,5] stilbene derivatives have biocidal properties such as anti-fungal[6] or cancerostatic.[7]

[1] *W. A. Sexton. Chemical Constitution and Biological Activity, pp 409-410, D. Van Nostrand Company, 1963*

[2] *G. E. Mikhailovskii and Yup Kozlov. Inclusion of Polycyclic Hydrocarbon Molecules into the Respiratory Chain as one of the Basic Mechanisms of Chemical Carcinogenesis, Biofizika 12(5):938-941, 1967*

[3] *R. L. Preston, J. E. Martin, J. E. Blakely, and W. H. Pfander. Structural Requirements for the Growth Response of Certain Estrogens in Ruminants, J. Anim. Sci., 24(2):338-340, 1964*

[4] *N. P. Buu-Hoi, G. Saint-Ruf, and G. Beauvillain. Differences in Estrogenic Activity Between Trans-Stilbene and Trans-Ar-Decadeuteriostilbene, C. R. Acad. Sci., Ser. D., 268(8):1221-1222, 1969*

[5] *T. S. Danowski, N. R. Limaye, R. E. Cohn, B. J. Grimes, J. V. Narduzzi, and C. Moses. Species Differences in Lipid and Endocrine Gland Response to a Stilbene Derivative, J. Pharm. Sci., 55(6):635-636, 1966*

[6] *L. Drobnica, M. Zemanova, P. Hemec, K. Antos, P. Kristian, and A. Martvon and Zavokska. Antifungal Activity of Isothiocyanates and Related Compounds. III. Derivatives of Biphenyl, Stilbene, Azobenzene, and Several Polycondensed Aromatic Hydrocarbons, Appl. Microbiol., 16(4):582-587, 1968*

[7] *K. Horakova, L. Drobnica, P. Nemnc, P. Kristian, K. Antos, A. Martvon. Cytotoxic and Cancerostatic Activity of Isothiocyanates and Related Compounds. III. Effect of Stilbene, Azobenzene, and Polycondensed Aromatic Hydrocarbon Isothiocyanate Derivatives on Hela Colis, Neoplasma 16(3):231-237, 1969*

STYRENE

Styrene may be irritating to eyes and mucous membrane, and in high concentrations it is narcotic.[1] In acute static bioassay the following fish had TLM_{96} of: Pimephales pronelas, 51 ppm; Lempomis macrochirus, 22 ppm; Carassius auratus, 68 ppm; and Lebistes reticulatus, 68 ppm.[2]

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TNT This compound is also known as 2,4,6-Trinitrotoluene or *s*-Trinitrotoluene. TNT has been found to chemically induce many degenerative diseases through long periods of moderate exposure. 2,4,6-Trinitrotoluene has been found to cause hepatitis, cataracts, fatty liver, jaundice, dyspancreatism, and increased glycolysis.[1] In a study by Manoilova and Zakharovi a total of 360 persons occupationally exposed to this toxic substance for at least 5 years were examined. In 45.3% an eye lesion taking the form of a singular specific cataract was discovered, which may appear as the first and only clinical manifestation of poisoning. No severe internal changes were demonstrable. Most frequently occurring were astheno-vegetative syndrome, chronic gastritis with subnormal acidity and mild forms of hepatitis.[2] One hundred parts per million of TNT were found to produce a complete kill of the algae, Microcystis

aeruginosa, in five days.[3] The anaerobic digestion of sludge from sewage containing 60 mg/l of TNT was practically unaffected by the TNT.[4] TNT can be decomposed in sewage aerobically but only at concentrations of a few mg/l. It was found to be utilized by infusoria and flagellates as nitrogen or carbon sources; concentrations of 0.5-1.0 mg/l only slightly retarded the self-purification of water. The limit suggested for sewage treatment was 10 mg/l.[5] A concentration of 2 mg/l of TNT reduced the 5-day BOD of sewage at 18.3°C by 6%, 5 mg/l by 15% and 20 mg/l by 35%.[6] In laboratory experiments using percolating filters impregnated with Nocardia, 100 mg/l of TNT was not decomposed, but slow decomposition occurred anaerobically with the microflora of domestic sewage.[7] TNT at concentrations of 5 to 50 mg/l was found to be destroyed during the anaerobic digestion of sewage sludges at 36°C.[8] 100 mg/l of TNT was slightly oxidized by phenol-adapted bacteria in Warburg respirometer experiments at 30°C.[9]

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n-TETRACOSANE See *n*-TRICOSANE

n-TETRA-TRIACONTANE See *n*-TRICOSANE

n-TRIACONTANE See *n*-TRICOSANE

TRICHLOROANILINE

Difluoroaniline, 4-fluoroaniline, and 3-bromoaniline caused methaemoglobin formation in various animal species lowering the level of intact haemoglobin.

S. McLean, G. A. Starmer and J. Thomas. Methaemoglobin Formation by Aromatic Amines. J. Pharm. Pharmac., 21:441-450, 1969

TRICHLOROBENZENE

The toxicity of chlorobenzene, dichlorobenzene and trichlorobenzene is on the same level; increasing the number of chlorine atoms in a benzene molecule does not affect the toxic action, but affects only the degree of expressivity. In determining the maximum permissible concentrations of these compounds in bodies of water, chronic experiments were conducted with white rats. Conditioned reflex activity was depressed showing a cerebral cortical effect; erythropoiesis was significatnly decreased, with chlorobenzene producing eosinophilia and ortho-dichlorobenzene, neutropenia. Ortho-dichlorobenzene, more than chlorobenzene, led to a sharp rise in urinary steroids.[1] Results of an investigation by Gurfein and Pavlova indicated that 0.03 mg/l of either di- or trichlorobenzene could be recommended as the limit of allowable concentration in water basins. Such conclusions were arrived at on the basis of the organoleptic index.[2] For chlorobenzene, Pickering and Henderson reported a TLM_{24} of 29 ppm for fathead, 24 ppm for bluegills, 73 ppm for goldfish, and 45 ppm for guppies.[3] Exposure of fresh water micro life to 100 ppm of trichlorobenzene results in a 98% kill; chronic feeding has caused loss of hair in experimental animals.[4]

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n-TRICOSANE

The long chain paraffin hydrocarbons generally should have little if any effect on water quality as they are all produced naturally and metabolized. Excessive concentrations would of course have a deleterious effect on BOD. For example, tobacco produces the following straight chain hydrocarbons: heptacosane, octacosane, nonacosane, triacontane, hentriacontane, dotriacontane, tritriacontane, tetratriacontane and pentatriacontane.[1] From the standpoint of water purification the behavior of these long-chain hydrocarbons will be very similar to their fatty acids which has been studied by Grin. His experiments established that 0.1 mg/l of the fatty acids (range C_5-C_{20}) was the minimal concentration which effected the BOD. Therefore, this concentration was regarded as the threshold BOD effect in water. Tests had also shown that the mineralization rate of domestic sewage type of organic matter in the presence of 3 to 4 mg/l of fatty acids was the same as in the control tests. Fatty acids lowered the rate of water auto-purification processes beginning with 5 mg/l. Ammonia accumulation was of a slower rate in the presence of fatty acids in the test samples than in the controls during the first six days; thereafter it gradually exceeded the control rates. Nitrification rate varied with the concentration and with the fraction type of the fatty acid. The second nitrification phase, formation of nitrites, manifested a higher sensitivity to the arresting effects of fatty acids; the arrest intensity was directly proportional to the fatty acid concentration.[2]

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n-TRITRIACONTANE See *n*-TRICOSANE

XYLENE TLM values for various fish are: Pimephales promelas - TLM_{96} of 21 ppm; Lepomis machrochirus - TLM_{96} of 22 ppm; Carassius auratus - TLM_{96} of 24 ppm; Lebistes reticulatus - TLM_{96} of 39 ppm. Daphnia magna in static acute bioassay had LD_{50} of 1 ppt.[2] *p*-Xylene had a thermodynamic activity in the range of 0.001 to 0.1 and gave a threshold narcosis 50 toxicity to barnacle larvae with active appendages but with no forward movement in Elminius modestus larvae after 15 minutes.[3] Based on the combined results of chronic sanitary-toxicological and organoleptic experiments Rubleva, in 1962, concluded that 0.08 mg/l should be adopted as the limit of allowable concentration for xylene in water basins.[4] This value was lowered to 0.05 by Cherkinskii in 1966 on the basis of additional test results.[5] More recently (1968) Kashin, Kulinskaya, and Mikhailovskaya found that the prolonged effect of small concentrations of *m*-xylene resulted in inhibition of agglutinin formation and functional activity of the adrenal cortex, disorders of acetycholin mediation and protein-forming function of the liver and loss of weight. They concluded that xylene possesses high toxicity and the permissible concentration should be further decreased.[6]

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