EPA-540/1-86-014

Environmental Protection Agency

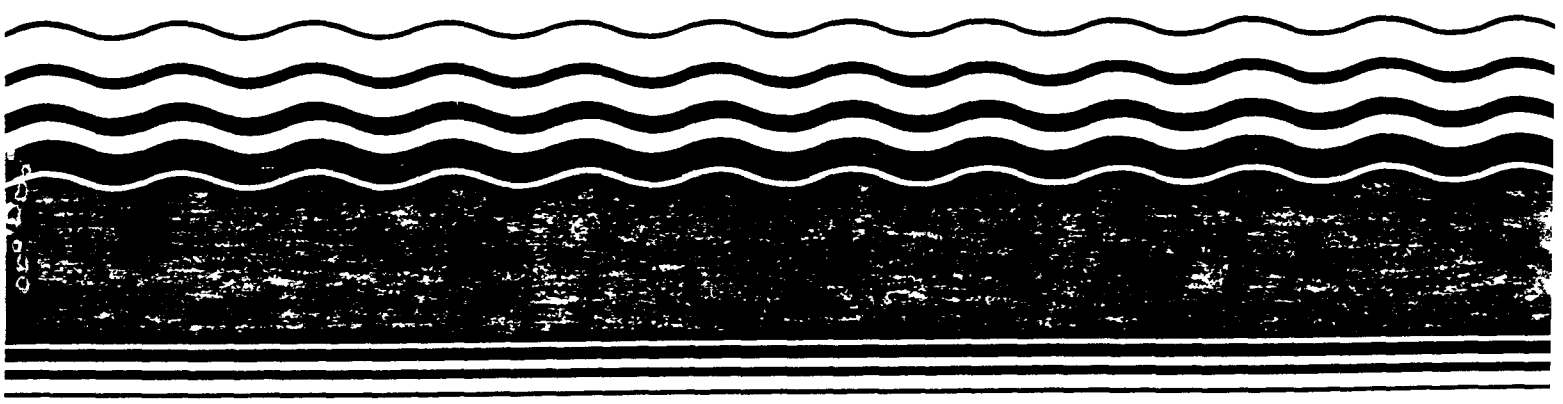
Office of Emergency and Remedial Response
Washington DC 20460

Office of Research and Development
Office of Health and Environmental Assessment
Environmental Criteria and Assessment Office
Cincinnati OH 45268

Superfund

EPA

HEALTH EFFECTS ASSESSMENT FOR NAPHTHALENE



EPA/540/1-86-014
September 1984

HEALTH EFFECTS ASSESSMENT FOR NAPHTHALENE

U.S. Environmental Protection Agency
Office of Research and Development
Office of Health and Environmental Assessment
Environmental Criteria and Assessment Office
Cincinnati, OH 45268

U.S. Environmental Protection Agency
Office of Emergency and Remedial Response
Office of Solid Waste and Emergency Response
Washington, DC 20460

DISCLAIMER

This report has been funded wholly or in part by the United States Environmental Protection Agency under Contract No. 68-03-3112 to Syracuse Research Corporation. It has been subject to the Agency's peer and administrative review, and it has been approved for publication as an EPA document. Mention of trade names or commercial products does not constitute endorsement or recommendation for use.

PREFACE

This report summarizes and evaluates information relevant to a preliminary interim assessment of adverse health effects associated with naphthalene. All estimates of acceptable intakes and carcinogenic potency presented in this document should be considered as preliminary and reflect limited resources allocated to this project. Pertinent toxicologic and environmental data were located through on-line literature searches of the Chemical Abstracts, TOXLINE, CANCERLINE and the CHEMFATE/DATALOG data bases. The basic literature searched supporting this document is current up to September, 1984. Secondary sources of information have also been relied upon in the preparation of this report and represent large-scale health assessment efforts that entail extensive peer and Agency review. The following Office of Health and Environmental Assessment (OHEA) source have been extensively utilized:

U.S. EPA. 1980a. Ambient Water Quality Criteria for Naphthalene. Environmental Criteria and Assessment Office, Cincinnati, OH. EPA-440/5-80-059. NTIS PB 81-117707.

U.S. EPA. 1982. Revision and Update of Hazard Profile on Naphthalene. Prepared by the Environmental Criteria and Assessment Office, Cincinnati, OH, OHEA for the Office of Solid Waste and Emergency Response, Washington, DC.

The intent in these assessments is to suggest acceptable exposure levels whenever sufficient data were available. Values were not derived or larger uncertainty factors were employed when the variable data were limited in scope tending to generate conservative (i.e., protective) estimates. Nevertheless, the interim values presented reflect the relative degree of hazard associated with exposure or risk to the chemical(s) addressed.

Whenever possible, two categories of values have been estimated for systemic toxicants (toxicants for which cancer is not the endpoint of concern). The first, the AIS or acceptable intake subchronic, is an estimate of an exposure level that would not be expected to cause adverse effects when exposure occurs during a limited time interval (i.e., for an interval that does not constitute a significant portion of the lifespan). This type of exposure estimate has not been extensively used or rigorously defined, as previous risk assessment efforts have been primarily directed towards exposures from toxicants in ambient air or water where lifetime exposure is assumed. Animal data used for AIS estimates generally include exposures with durations of 30-90 days. Subchronic human data are rarely available. Reported exposures are usually from chronic occupational exposure situations or from reports of acute accidental exposure.

The AIC, acceptable intake chronic, is similar in concept to the ADI (acceptable daily intake). It is an estimate of an exposure level that would not be expected to cause adverse effects when exposure occurs for a significant portion of the lifespan [see U.S. EPA (1980b) for a discussion of this concept]. The AIC is route specific and estimates acceptable exposure for a given route with the implicit assumption that exposure by other routes is insignificant.

Composite scores (CSs) for noncarcinogens have also been calculated where data permitted. These values are used for ranking reportable quantities; the methodology for their development is explained in U.S. EPA (1983b).

For compounds for which there is sufficient evidence of carcinogenicity, AIS and AIC values are not derived. For a discussion of risk assessment methodology for carcinogens refer to U.S. EPA (1980b). Since cancer is a process that is not characterized by a threshold, any exposure contributes an increment of risk. Consequently, derivation of AIS and AIC values would be inappropriate. For carcinogens, q_1^*s have been computed based on oral and inhalation data if available.

ABSTRACT

In order to place the risk assessment in proper context, the reader is referred to the preface of this document. The preface outlines limitations applicable to all documents of this series, as well as the appropriate interpretation and use of the quantitative estimates.

The absence of adequate toxicological data on naphthalene precludes estimation of acceptable intakes for any route.

ACKNOWLEDGEMENTS

The initial draft of this report was prepared by Syracuse Research Corporation under Contract No. 68-03-3112 for EPA's Environmental Criteria and Assessment Office, Cincinnati, OH. Dr. Christopher DeRosa and Karen Blackburn were the Technical Project Monitors and Helen Ball was the Project Officer. The final documents in this series were prepared for the Office of Emergency and Remedial Response, Washington, DC.

Scientists from the following U.S. EPA offices provided review comments for this document series:

Environmental Criteria and Assessment Office, Cincinnati, OH
Carcinogen Assessment Group
Office of Air Quality Planning and Standards
Office of Solid Waste
Office of Toxic Substances
Office of Drinking Water

Editorial review for the document series was provided by:

Judith Olsen and Erma Durden
Environmental Criteria and Assessment Office
Cincinnati, OH

Technical support services for the document series was provided by:

Bette Zwayer, Pat Daunt, Karen Mann and Jacky Bohanon
Environmental Criteria and Assessment Office
Cincinnati, OH

TABLE OF CONTENTS

TABLE OF CONTENTS (cont.)

LIST OF ABBREVIATIONS

ADI	Acceptable daily intake
AIC	Acceptable intake chronic
AIS	Acceptable intake subchronic
CAS	Chemical Abstract Service
CS	Composite score
ppm	Parts per million
RQ	Reportable quantity
STEL	Short-term exposure limit
TLV	Threshold limit value
TWA	Time-weighted average

1. ENVIRONMENTAL CHEMISTRY AND FATE

The relevant physical and chemical properties and environmental fate of naphthalene (CAS No. 91-20-3) are as follows:

Chemical class:	polycyclic aromatic hydrocarbon
Molecular weight:	128.19 (Callahan et al., 1979)
Vapor pressure at 25°C:	0.082 mm Hg (MacKay et al., 1982)
Water solubility at 25°C:	31.7 mg/ℓ (MacKay et al., 1980)
Log octanol/water partition coefficient:	3.37 (MacKay et al., 1980)
Bioconcentration factor:	146 (estimated from the equation of Veith et al., 1979)
Half-life in air:	0.7 (Atkinson et al., 1984)
Half-life in water:	~1 day (Callahan et al., 1979) 2.3 days (Zoeteman et al., 1980)
Half-life in soil:	<1 day (estimated)

The half-life for naphthalene in aquatic media caused by biodegradation can be estimated to be ~14 hours from the estimated biotransformation rate constant ($1x10^{-7}$ mℓ $cell^{-1}$ hr^{-1}) reported by Mabey et al. (1981) and the concentration of microorganisms at $5x10^{5}$ cell $mℓ^{-1}$ (Burns et al., 1982).

Pertinent data regarding the leachability of this compound in soils could not be located in the available literature. Considering the soil sorption coefficient (Kenaga and Goring, 1980) and the water solubility, this compound is expected to have higher mobility in soils compared with most other polycyclic aromatic hydrocarbons. The biodegradability of this compound in soils, however, is such that significant leaching into groundwater is not likely.

2. ABSORPTION

2.1. ORAL

Information regarding the absorption of naphthalene in humans and animals is limited. When ingested as a solid, naphthalene is sufficiently absorbed to cause toxicity in man (Chusid and Fried, 1955; Zuelzer and Apt, 1949; Nash, 1903; Gross et al., 1958; Haggerty, 1956). Furthermore, naphthalene seems to be more toxic when dissolved in oil before ingestion (Talakin, 1966). Sanborn and Malins (1977) suggested that naphthalene may be absorbed to a greater extent when ingested in water than in food.

2.2. INHALATION

Data regarding the absorption of inhaled naphthalene are limited; however, Valaes et al. (1963) reported toxicity and death in newborn infants exposed to naphthalene vapors from clothes or blankets that had been stored in or near the infants' rooms.

3. TOXICITY IN HUMANS AND EXPERIMENTAL ANIMALS

3.1. SUBCHRONIC

3.1.1. Oral. Data regarding subchronic oral exposure to naphthalene are limited. Several investigators (Anziulewicz et al., 1959; Zinkham and Childs, 1958) reported the occurrence of hemolytic anemia in infants born to mothers who had "sniffed" and ingested unspecified quantities of naphthalene (in the form of mothballs) during pregnancy. The mothers themselves were also anemic, but to a lesser extent than were the infants.

Several animal studies focused upon ocular effects of naphthalene, but failed to mention whether any other signs of toxicity were measured or observed. Fitzhugh and Buschke (1949) observed slight cataracts in weanling rats exposed to 2% naphthalene in the diet for 60 days. Assuming a rat consumes food equivalent to 5% of its body weight/day, this dietary level is equivalent to a dose of 1 g/kg bw/day.

In two separate studies, rabbits exposed to 1 g naphthalene/kg bw/day by gavage (either in light paraffin or an unspecified vehicle) for ~46 days developed cataracts (Ghetti and Mariani, 1956; Van Heyningen and Pirie, 1976) and degeneration of the retina (Van Heyningen and Pirie, 1976) within the first few days of treatment.

3.1.2. Inhalation. Subchronic data regarding the inhalation of naphthalene are limited to two studies of occupational exposure. Van der Hoeve (1906) reported that one man who worked with powdered naphthalene developed cataracts and retinal hemorrhage, while another man exposed similarly had chorioretinitis. Ghetti and Mariani (1956) reported that 8/21 workers exposed to unspecified "high" concentrations of naphthalene in a dye-manufacturing process developed cataracts. Since these men were <50 years of age, it is unlikely that they would have developed cataracts spontaneously.

3.2. CHRONIC

3.2.1. Oral. Pertinent data regarding the chronic toxicity of orally administered naphthalene could not be located in the available literature.

3.2.2. Inhalation. Pertinent data regarding chronic toxicity of inhaled naphthalene could not be located in the available literature.

3.3. TERATOGENICITY AND OTHER REPRODUCTIVE EFFECTS

3.3.1. Oral. Pertinent data regarding the teratogenicity of ingested naphthalene could not be located in the available literature; however, naphthalene or its metabolites are known to cross the placenta in sufficient quantities to cause fetotoxic effects (Anziulewicz et al., 1959; Van der Hoeve, 1913). These effects included hemolytic anemia in humans and cataracts and retinal damage in rabbits. Doses either were not estimated or were not reported in the secondary sources.

3.3.2. Inhalation. Pertinent data regarding the teratogenicity of inhaled naphthalene could not be located in the available literature.

3.4. TOXICANT INTERACTIONS

A woman exposed for 3 weeks to a combination of naphthalene and paradichlorobenzene (while mothproofing clothing) developed aplastic anemia 1 month after exposure (Harden and Baetjer, 1978). No other cases of aplastic anemia associated with exposure to either napthalene or paradichlorobenzene alone have been reported in the literature.

4. CARCINOGENICITY

4.1. HUMAN DATA

4.1.1. Oral. Pertinent data regarding the carcinogenicity of ingested naphthalene could not be located in the available literature.

4.1.2. Inhalation. Pertinent data regarding the carcinogenicity of inhaled naphthalene could not be located in the available literature; however, Wolf (1976) reported that 6/15 workers exposed to vapors of both naphthalene and coal tar developed laryngeal carcinomas (4) or neoplasms of the pylorus and cecum (2). There was no control group. Schmeltz et al. (1978), however, showed that the di-, tri- and tetramethyl naphthalene contaminants of coal tar were carcinogenic when applied to mouse skin but that naphthalene alone was not.

4.2. BIOASSAYS

4.2.1. Oral. In a study designed to investigate the carcinogenic effects of anthracene delivered by a naphthalene vehicle, the carcinogenicity of naphthalene alone was tested (Druckrey and Schmahl, 1955). A group of 28 BD I and BD III strain rats were exposed orally to 10 g naphthalene/rat for an unspecified amount of time and monitored for >1000 days. A second group of 10 rats were exposed subcutaneously to 0.82 g naphthalene/rat for a similar period of time. No tumors were observed in either group. No other studies pertaining to the carcinogenicity of orally administered naphthalene were located in the available literature.

4.2.2. Inhalation. Pertinent data regarding the carcinogenicity of inhaled naphthalene could not be located in the available literature.

4.3. OTHER RELEVANT DATA

The genotoxic activity of naphthalene has been tested in two *in vitro* systems. Cell transformation was not seen in rodent embryo cells pretreated with leukemia virus and exposed to concentrations of naphthalene up to 100 mg/ℓ of culture medium (Freeman et al., 1973; Rhim et al., 1974). Similarly, cell transformations were not seen in a murine mammary gland organ culture system at naphthalene concentrations up to 1000 mg/ℓ of culture medium (Tonelli et al., 1979).

Naphthalene was not found to be mutagenic in a number of bacterial/microsomal assay systems (McCann et al., 1975; Kraemer et al., 1974).

4.4. WEIGHT OF EVIDENCE

IARC has not evaluated the risk to humans associated with oral or inhalation exposure to naphthalene. Applying the criteria for evaluating the overall weight of evidence for carcinogenicity proposed by the Carcinogen Assessment Group of the U.S. EPA (Federal Register, 1984), the evidence for carcinogenicity of naphthalene in humans and animals is inadequate and the chemical is most appropriately designated a Group D-Not Classified chemical.

5. REGULATORY STANDARDS AND CRITERIA

The ACGIH (1983) has recommended a TLV-TWA of 50 mg/m^3 (10 ppm) and an STEL of 75 mg/m^3 for occupational exposure to naphthalene. Since eye irritation was seen at 15 ppm, this criterion was chosen "to prevent ocular effects but possibly not blood changes in hypersusceptibles." The OSHA standard for exposure to naphthalene in the workplace is a TWA of 50 mg/m^3 (Code of Federal Regulations, 1981).

6. RISK ASSESSMENT

6.1. ACCEPTABLE INTAKE SUBCHRONIC (AIS)

6.1.1. Oral. The only studies that defined exposure levels (Ghetti and Mariani, 1956; Van Heyningen and Pirie, 1976) failed to mention whether endpoints other than ocular effects were monitored. Since other effects such as hemolytic anemia have been associated with exposure to naphthalene, it would not be prudent to calculate an AIS on the basis of these studies.

6.1.2. Inhalation. The lack of pertinent, quantitative data precludes assessment of risk caused by inhalation of naphthalene.

6.2. ACCEPTABLE INTAKE CHRONIC (AIC)

6.2.1. Oral. Data regarding chronic oral exposure to naphthalene were not located in the available literature. For reasons given in Section 6.1.1., an AIC cannot be derived from the subchronic data. data from which a CS for oral exposure to naphthalene can be calculated have not been located in the available literature (U.S. EPA, 1983a).

6.2.2. Inhalation. Data regarding chronic exposure to naphthalene by inhalation could not be located in the available literature. Furthermore, it would be imprudent to use the TLV established by the ACGIH (1983) to calculate an AIC, since this value was established essentially on the basis of eye irritation and with the hypothesis that chronic exposure might cause more severe effects. Data from which a CS for inhalation exposure to naphthalene can be calculated have not been located in the available literature (U.S. EPA, 1983a).

6.3. CARCINOGENIC POTENCY (q_1*)

6.3.1. Oral. Naphthalene was not carcinogenic in strain BD I and BD III rats (Druckrey and Schmahl, 1955). Furthermore, mutagenicity and *in vitro* bioassays that tested only naphthalene yielded negative results. It is therefore not possible to derive a q_1* for oral exposure to naphthalene.

6.3.2. Inhalation. The lack of pertinent data regarding the carcinogenicity of inhaled naphthalene precludes assessment of carcinogenic potency.

8. REFERENCES

ACGIH (American Conference of Governmental Industrial Hygienists). 1983. Threshold Limit Values for Chemical Substances and Physical Agents in the Workroom Environment with Intended Changes for 1983-84. Cincinnati, OH. p. 26.

Anziulewicz, J.A., H.J. Dick and E.E. Chiarulli. 1959. Transplacental naphthalene poisoning. Am. J. Obstet. Gynecol. 78: 519-521. (Cited in U.S. EPA, 1983a)

Atkinson, R., S.M. Aschmann and J.N. Pitts, Jr. 1984. Kinetics of the reactions of naphthalene and biphenyl with OH radicals and with O_3 at 294±1K. Environ. Sci. Technol. 18: 110-113.

Burns, L.A., D.A. Cline and R.R. Lassiter. 1982. Exposure Analysis Modeling System (EXAMS). User Manual and System Documentation. Environmental Research Laboratory, ORD, U.S. EPA, Athens, GA. EPA 600/3-82-023.

Callahan, M.A., M.W. Slimak, N.W. Gabel, et al. 1979. Water-Related Environmental Fate of 129 Priority Pollutants. Vol. II. Office of Water Planning and Standards, Office of Water and Waste Management. U.S. EPA, Washington, DC. EPA 440/4-79-029b.

Chusid, E. and C.T. Fried. 1955. Acute hemolytic anemia due to naphthalene ingestion. Am. J. Dis. Child. 89: 612-614. (Cited in U.S. EPA, 1980a)

Code of Federal Regulations. 1981. OSHA Safety and Health Standards 29 CFR 1910.1000.

Druckrey, H. and D. Schmahl. 1955. Cancerogene wirkung von anthracen. Die Naturwissenschaften. 42: 159. (Ger.) (Cited in U.S. EPA, 1982)

Federal Register. 1984. Environmental Protection Agency. Proposed guidelines for carcinogenic risk assessment. 49 FR 46294-46299.

Fitzhugh, O.G. and W.H. Buschke. 1949. Production of cataract in rats by betatetralol and other derivatives of naphthalene. Arch. Opthal. 41: 572-582. (Cited in U.S. EPA, 1983a)

Freeman, A.E., et al. 1973. Transformation of cell cultures as an indication of the carcinogenic potential of chemicals. J. Natl. Cancer Inst. 51: 799-807. (Cited in U.S. EPA, 1980a; 1982)

Ghetti, G. and L. Mariani. 1956. Eye changes due to naphthalene. Med. Lav. 47: 533-538. (Cited in U.S. EPA, 1980a; 1983a)

Gross, R.T., et al. 1958. An hereditary enzymatic defect in erythrocyte metabolism: glucose-6-phosphate dehydrogenase deficiency. J. Clin. Invest. 37: 1176-1184. (Cited in U.S. EPA, 1980a)

Haggerty, R.J. 1956. Toxic hazards: naphthalene poisoning. New England J. Med. 255: 919-920. (Cited in U.S. EPA, 1980a)

Harden, R.A. and A.M. Baetjer. 1978. Aplastic anemia following exposure to paradichlorobenzene and naphthalene. J. Occup. Med. 20: 820-822. (Cited in U.S. EPA, 1980a)

Kenaga, E.E. and C.A. I. Goring. 1980. Relationships between water solubility, soil sorption, octanol-water partitioning and concentration of chemicals in biota. In: Aquatic Toxicology, ASTM STP 707, J.G. Eaton, P.R. Parrish and A.C. Hendricks, Ed., ASTM, Philadelphia, PA.

Kraemer, M., et al. 1974. S. typhimurium and E. coli to detect chemical mutagens. Arch. Pharmacol. 284: R46. (Cited in U.S. EPA, 1980a; 1982)

Mabey, W.R., J.H. Smith, R.T. Podoll, et al. 1981. Aquatic Fate Process Data for Organic Priority Pollutants. Monitoring and Data Support Div., Office of Water Regulations and Standards, Washington, DC. EPA 440/4-81-014.

MacKay, D., A. Bobra, W.Y. Shin and S.H. Yalkowsky. 1980. Relationships between aqueous solubility and octanol-water partition coefficients. Chemosphere. 9: 701-711.

MacKay, D., A. Bobra, D.W. Chan and W.Y. Shin. 1982. Vapor pressure correlations of low-volatility environmental chemicals. Environ. Sci. Technol. 16: 645-649.

McCann, J., et al. 1975. Detection of carcinogens as mutagens in the Salmonella/microsome test. Assay of 300 chemicals. Proc. Natl. Acad. Sci. 72: 5135-5139. (Cited in U.S. EPA, 1980a; 1982a)

Nash, L.F. 1903. Naphthalene poisoning. Br. Med. J. 1: 251. (Cited in U.S. EPA, 1980a)

Rhim, J.S., et al.* 1974. Evaluation of an in vitro assay system for carcinogens based on prior infection of rodent cells with nontransforming RNA tumor virus. J. Natl. Cancer Inst. 52: 1167-1173. (Cited in U.S. EPA, 1982)

Sanborn, H.R. and D.C. Malins. 1977. Toxicity and metabolism of naphthalene: a study with marine larval invertebrates. Proc. Soc. Exp. Biol. Med. 154: 151-155. (Cited in U.S. EPA, 1980a)

Schmeltz, I., et al.* 1978. Bioassays of Naphthalene and Alkyl Naphthalenes for Co-Carcinogenic Activity. Relation to Tobacco Carcinogenesis. In: Carcinogenesis, Vol. 3: Polynuclear Aromatic Hydrocarbons, P. Jones and I. Freudenthal, Ed. Raven Press, NY. p. 47-60. (Cited in U.S. EPA, 1980a)

Talakin, Yu. N. 1966. Sanitary-toxicological characteristics of α-naphthoquinone. Vop. Kommunal. Gig. 6: 37-43. (Cited in U.S. EPA, 1980a)

Tonelli, Q., et al. 1979. Transformation of cultured mouse mammary glands by aromatic amines and amides and their derivatives. Cancer Res. 39: 1784-1792. (Cited in U.S. EPA, 1980a; 1982)

U.S. EPA. 1980a. Ambient Water Quality Criteria for Naphthalene. U.S. EPA, Environmental Criteria and Assessment Office, Cincinnati, OH. EPA-440/5-80-059. NTIS PB 81-117707.

U.S. EPA. 1980b. Guidelines and Methodology Used in the Preparation of Health Effects Assessment Chapters of the Consent Decree Water Quality Criteria. Federal Register. 45:79347-79357.

U.S. EPA. 1982. Revision and Update of Hazard Profile on Naphthalene. Prepared by the Environmental Criteria and Assessment Office, Cincinnati, OH, OHEA for the Office of Solid Waste and Emergency Response, Washington, DC.

U.S. EPA. 1983a. Reportable Quantity for Naphthalene. Prepared by the Environmental Criteria and Assessment Office, Cincinnati, OH, OHEA for the Office of Solid Waste and Emergency Response, Washington, DC.

U.S. EPA. 1983b. Methodology and Guidelines for Reportable Quantity Determinations Based on Chronic Toxicity Data. Prepared by the Environmental Criteria and Assessment Office, Cincinnati, OH, OHEA for the Office of Solid Waste and Emergency Response, Washington, DC.

Valaes, T., et al. 1963. Acute hemolysis due to naphthalene inhalation. J. Ped. 63: 904-915. (Cited in U.S. EPA, 1980a)

Van der Hoeve, J. 1906. Choreoretinitis beim menschen durch die einwirking von naphthalin. Arch. Augenheilk. 56: 259. (Ger.) (Cited in U.S. EPA, 1980a; 1983a)

Van der Hoeve, J. 1913. Wirkung von naphthol auf die augen von menschen, tieren, und auf fatale augen. Graele Arch. Ophthal. 85: 305. (Cited in U.S. EPA, 1980a)

Van Heyningen, R. and A. Pirie. 1976. Naphthalene cataract in pigmented and albino rabbits. Exp. Eye Res. 22: 393-394. (Cited in U.S. EPA, 1983a)

Veith, G.D., D.L. Defoe and B.V. Bergstedt. 1979. Measuring and estimating the bioconcentration factor of chemicals in fish. J. Fish Res. Board Can. 36: 1040-1048.

Wolf, O. 1976. Cancer diseases in chemical workers in a former naphthalene cleaning plant. Deutche Gesundheitwesen. 31: 996-999. (Cited in U.S. EPA, 1980a)

Zinkham, W.J. and B. Childs. 1958. A defect of glutathione metabolism in erythrocytes from patients with a naphthalene-induced hemolytic anemia. Pediatrics. 22: 461-471. (Cited in U.S. EPA, 1983a)

Zoeteman, B.C.J., K. Harmsen, J.B.H.J. Linders, C.F. H. Morra and W. Slooff. 1980. Persistent organic pollutants in river water and groundwater of the Netherlands. Chemosphere. 9: 231-249.

Zuelzer, W.W. and L. Apt. 1949. Acute hemolytic anemia due to naphthalene poisoning. J. Am. Med. Assoc. 141: 185-190. (Cited in U.S. EPA, 1980a)

APPENDIX

Summary Table for Naphthalene

	Species	Experimental Dose/Exposure	Effect	Acceptable Intake (AIS or AIC)	Reference
Inhalation					
AIS				ND	
AIC				ND	
Carcinogenic potency				ND	
Oral					
AIS				ND	
AIC				ND	
Carcinogenic potency				ND	

ND = Not derived