Technical Fact Sheet

npic.orst.edu/factsheets/archive/naphtech.html

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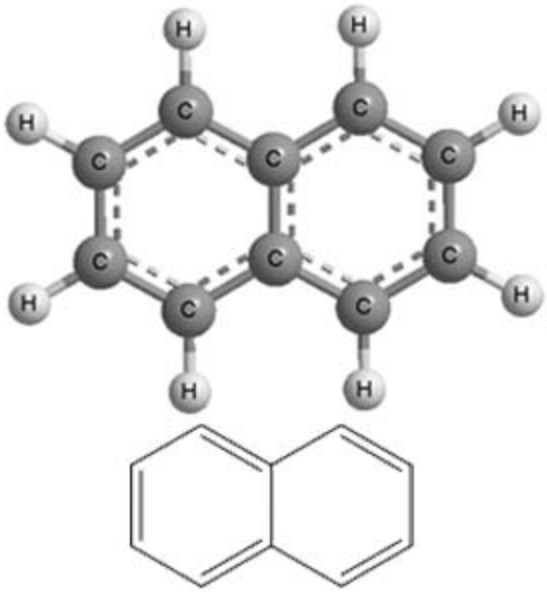
Naphthalene

As of 2011, NPIC stopped creating technical pesticide fact sheets. The old collection of technical fact sheets will remain available in this archive, but they may contain out-of-date material. NPIC no longer has the capacity to consistently update them. To visit our general fact sheets, click here. For up-to-date technical fact sheets, please visit the Environmental Protection Agency's webpage.

Molecular Structure -
Naphthalene

Laboratory Testing: Before pesticides are registered by the U.S. EPA, they must undergo laboratory testing for short-term (acute) and long-term (chronic) health effects. Laboratory animals are purposely given high enough doses to cause toxic effects. These tests help scientists judge how these chemicals might affect humans, domestic animals, and wildlife in cases of overexposure.



Chemical Class and Type:

Naphthalene and other polycyclic aromatic hydrocarbons (PAHs) are released from incomplete combustion processes originating in industry, domestic sources including cigarette smoke and motor vehicle exhaust, as well as natural events such as forest fires.[5]

Physical / Chemical Properties:

- Naphthalene is a white crystalline or colorless to brown solid. Naphthalene has a distinct aromatic odor.[3,6]
- Vapor pressure[1]: 0.087 mmHg
- Octanol-Water Partition Coefficient (log K_{ow})[1]: 3.29
- Henry's constant[1]: 4.6 x 10^{-4} atm·m^3/mol
- Molecular weight[1,3]: 128.18 g/mol
- Solubility (water)[7]: 3 mg/100 mL (30 mg/L) at room temperature
- Soil Sorption Coefficient (K_{oc})[3]: Values from 200-1470 have been reported worldwide in a variety of soil types.

Uses:

- Naphthalene is primarily used in the United States in the production of plastics, dyes, resins, lubricants, and fuels.[4] As a pesticide, most naphthalene is used to control moths in indoor storage areas. Products are also registered to repel squirrels, bats and other animals in attics, around structures and gardens.[3] Uses for individual products containing naphthalene vary widely. Always read and follow the label when applying pesticide products.
- Signal words for products containing naphthalene may range from Caution to Danger. The signal word reflects the combined toxicity of the active ingredient and other ingredients in the product. See the pesticide label on the product and refer to the NPIC fact sheets on Signal Words and Inert or "Other" Ingredients.
- To find a list of products containing naphthalene which are registered in your state, visit the website http://npic.orst.edu/reg/state_agencies.html select your state then click on the link for "State Products."

Mode of Action:

Target Organisms

- Naphthalene is used for the control of clothes moths. In this application, the naphthalene vapors fill the airtight container and kill the insects. No information was found on the mode of action of naphthalene in insects.
- Naphthalene is registered for use as a wildlife repellent in some products. In this application, it is meant to be effective via inhalation and subsequent avoidance of the odor in the treated area.[3]

Non-target Organisms

- In humans, the metabolite alpha-naphthol has been linked to the development of hemolytic anemia in some cases following ingestion or extensive dermal or inhalation exposure. Susceptibility appears to be exacerbated by a deficiency in the glucose 6-phosphate dehydrogenase enzyme, or G-6-PD. The enzyme is inherited through a sex-linked gene so that males are more likely to express the deficiency. The deficiency is also more prevalent in people of African or Mediterranean descent.[10]
- Newborns appear to be more susceptible to developing hemolysis following exposure to naphthalene. This susceptibility may be due to their less-developed ability to conjugate and excrete naphthalene metabolites.[1] In two studies, several young children developed hemolytic anemia following exposure to clothing, bedding and diapers that had been stored with naphthalene mothballs. Many but not all of these children appeared to have the G-6-PD enzyme deficiency.[11,12] Fetuses have developed hemolytic anemia *in utero* following their mothers' inhalation exposure.[13]

Acute Toxicity:

Oral

Acute oral LD_{50} values in rats range from 2200 mg/kg to 2600 mg/kg.[1,17,18] Based on these values, the U.S. EPA considered naphthalene to be low in toxicity via oral exposure.[3] See the text boxes on **Toxicity Classification** and **LD_{50}/LC_{50}**.

LD_{50}/LC_{50}: A common measure of acute toxicity is the lethal dose (LD_{50}) or lethal concentration (LC_{50}) that causes death (resulting from a single or limited exposure) in 50 percent of the treated animals. LD_{50} is generally expressed as the dose in milligrams (mg) of chemical per kilogram (kg) of body weight. LC_{50} is often expressed as mg of chemical per volume (e.g., liter (L)) of medium (i.e., air or water) the organism is exposed to. Chemicals are considered highly toxic when the LD_{50}/LC_{50} is small and practically non-toxic when the value is large. However, the LD_{50}/LC_{50} does not reflect any effects from long-term exposure (i.e., cancer, birth defects or reproductive toxicity) that may occur at levels below those that cause death.

Dermal

- The U.S. EPA considered primary eye irritation to be slight to moderate and skin irritation to be moderate based on studies with rabbits. Naphthalene is considered to be low in toxicity for both exposure routes.[3]
- Naphthalene was not found to be a skin sensitizer in guinea pigs.[3,17]

TOXICITY CLASSIFICATION - NAPHTHALENE

High Toxicity	Moderate Toxicity	Low Toxicity	Very Low Toxicity

Acute Oral LD_{50}	Up to and including 50 mg/kg (≤ 50 mg/kg)	Greater than 50 through 500 mg/kg (>50-500 mg/kg)	Greater than 500 through 5000 mg/kg (>500-5000 mg/kg)	Greater than 5000 mg/kg (>5000 mg/kg)
Inhalation LC_{50}	Up to and including 0.05 mg/L (≤0.05 mg/L)	Greater than 0.05 through 0.5 mg/L (>0.05-0.5 mg/L)	Greater than 0.5 through 2.0 mg/L (>0.5-2.0 mg/L)	Greater than 2.0 mg/L (>2.0 mg/L)
Dermal LD_{50}	Up to and including 200 mg/kg (≤200 mg/kg)	Greater than 200 through 2000 mg/kg (>200-2000 mg/kg)	Greater than 2000 through 5000 mg/kg (>2000-5000 mg/kg)	Greater than 5000 mg/kg (>5000 mg/kg)
Primary Eye Irritation	Corrosive (irreversible destruction of ocular tissue) or corneal involvement or irritation persisting for more than 21 days	Corneal involvement or other eye irritation clearing in 8 - 21 days	Corneal involvement or other eye irritation clearing in 7 days or less	Minimal effects clearing in less than 24 hours
Primary Skin Irritation	Corrosive (tissue destruction into the dermis and/or scarring)	Severe irritation at 72 hours (severe erythema or edema)	Moderate irritation at 72 hours (moderate erythema)	Mild or slight irritation at 72 hours (no irritation or erythema)

The highlighted boxes reflect the values in the "Acute Toxicity" section of this fact sheet. Modeled after the U.S. Environmental Protection Agency, Office of Pesticide Programs, Label Review Manual, Chapter 7: Precautionary Labeling. https://www.epa.gov/sites/default/files/2018-04/documents/chap-07-mar-2018.pdf

Inhalation

The inhalation LC_{50} for naphthalene was determined to be 0.4 mg/L or 77.7 ppm in an unspecified animal species, and it is considered moderately toxic via inhalation by the U.S. EPA.[3]

Signs of Toxicity - Animals

- Three dogs fed varying amounts of naphthalene either as a single dose or over the course of five days exhibited lethargy, diarrhea, anorexia, and in one case ataxia.[2,3] Vomiting, tremors, depression, and mydriasis, or dilation of the pupils have also been reported in dogs.[2,4]

- Other clinical signs noted in dogs include hemolytic anemia, the presence of Heinz bodies indicating damaged hemoglobin, reduced hemoglobin content, fragmented blood cells, reticulocytosis and leukocytosis in the blood.[23,24]
- Rats and mice were found to be less sensitive to the hemolytic effects of naphthalene than dogs.[1]

Signs of Toxicity - Humans

- Inhalation of naphthalene vapor has been associated with headaches, nausea, vomiting and dizziness. Hemolysis, the abnormal breakdown of red blood cells, may occur following ingestion or sufficient dermal exposure to either naphthalene or to naphthalene-treated fabric. People with a deficiency of the enzyme glucose-6-phosphate dehydrogenase, or G-6-PD, are at greater risk of developing hemolytic anemia. G-6-PD deficiency is an inherited trait.[10] Infants may develop hemolysis even if not deficient in G-6-PD.[11]
- In humans, cataracts and other ocular injury have been reported following acute and chronic occupational exposure to naphthalene.[1] However, there are many documented cases of severe acute exposures that did not lead to ocular damage in people. The potential for naphthalene to cause damage to human eyes is uncertain.[26]
- Children who ingested mothballs containing naphthalene exhibited diarrhea, vomiting, lethargy, lack of appetite, fever, abdominal pain, painful urination (dysuria), and dark wine-colored or discolored urine.[12,23] Upon admission to the hospital, the children were diagnosed with acute hemolytic anemia and hemoglobinuria.[23]
- Additional signs of toxicity in children include convulsions and coma. Infants may develop encephalopathy and kernicterus, a form of brain damage, due to the presence of increased levels of methemoglobin, hemoglobin, and bilirubin in their plasma.[10,11]
- Liver and kidney damage has been noted following ingestion of naphthalene in humans, but these effects have not been consistently seen in animals.[1]
- Always follow label instructions and take steps to minimize exposure. If any exposure occurs, be sure to follow the First Aid instructions on the product label carefully. For additional treatment advice, contact the Poison Control Center at 1-800- 222-1222. If you wish to discuss an incident with the National Pesticide Information Center, please call 1-800-858-7378.

Chronic Toxicity:

Animals

- Researchers dosed mice with naphthalene by gavage at 27, 53, or 267 mg/kg daily for 14 days. Mice treated with 267 mg/kg weighed significantly less at the end of the experiment than the other groups. At this dose, males experienced an average decrease of 30% in thymus weight, and females lost significant spleen mass but gained lung mass.[19]

- The LD_{50} for female CD-1 mice was determined to be 353.6 mg/kg based on an eight-day oral exposure.[20]
- Mice were dosed with naphthalene by gavage at doses of 5.3, 53.0, and 133.0 mg/kg daily for 90 days. The females given the highest dose experienced decreases in brain, liver, and spleen weights but these effects were not found in the males.[19]
- The chronic dietary NOAEL was identified as 100 mg/kg/day and the LOAEL identified as 200 mg/kg/day based on studies in rats.[3] See the text box on **NOAEL, NOEL, LOAEL, and LOEL**.
  NOAEL: No Observable Adverse Effect Level

  NOEL: No Observed Effect Level

  LOAEL: Lowest Observable Adverse Effect Level

  LOEL: Lowest Observed Effect Level

- Researchers conducted a 4-week inhalation study of naphthalene exposure in rats and identified a NOAEL of 3 ppm or 16 mg/m^3 and an inhalation LOAEL of 10 ppm or 52 mg/m^3. Signs of toxicity at the LOAEL were nasal lesions.[27]
- A 13-week inhalation study in which rats were exposed to naphthalene only through the nose resulted in a NOAEL of 1.0 ppm or 5.2 mg/m^3 and a LOAEL of 2.0 ppm or 10.0 mg/m^3 because of increased nasal lesions in exposed animals.[3]
- Researchers applied naphthalene to the skin of rats for 90 days. They observed atrophy of the seminiferous tubules in males and changes in the liver, kidney, thyroid, and urinary bladder in addition to non-neoplastic lesions in the cervical lymph node in the females. The NOAEL was set at 300 mg/kg/day and the LOAEL at 1000 mg/kg/day. Researchers also noted hyperkeratosis and acanthosis in the skin of exposed female rats.[28]

Humans

- Children aged 1.5-36 months have developed hemolytic anemia following dermal exposure to diapers or other clothing stored with naphthalene mothballs or playing in a room used to store naphthalene mothballs.[29] Infants developed hemolytic anemia and other complications following inhalation exposure to clothing, blankets, linens, or diapers stored in or around naphthalene.[11] See the text box on **Exposure**.
  Exposure: Effects of naphthalene on human health and the environment depend on how much naphthalene is present and the length and frequency of exposure. Effects also depend on the health of a person and/or certain environmental factors.

- A 68-year-old woman developed aplastic anemia following inhalation and possibly dermal exposure to naphthalene and paradichlorobenze, which she placed into containers with stored clothing several hours a day for three weeks while working for a clothing resale business. The woman had worked in a room with poor ventilation and no air conditioning during hot weather, which contributed to high vapor concentrations of naphthalene and paradichlorobenzene.[30]

Endocrine Disruption:

Atlantic croaker fish (*Micropogonias undulatus*) exposed to naphthalene at either 0.5 or 1.0 ppm daily during sexual maturation demonstrated reduced rates of sexual maturity and arrested or reduced egg development. Reduced egg growth was associated with decreased gonadal steroid levels in plasma.[31]

Carcinogenicity:

Animals

- Researchers exposed male and female F344 rats to naphthalene vapors at 10, 30, or 60 ppm for 6 hours a day, five days a week for two years. Respiratory epithelial adenomas and olfactory epithelial neuroblastomas occurred in the noses of both males and females. In addition, non-cancerous lesions were also noted in the lungs and noses of the animals.[32]
- The National Toxicology Program (NTP) concluded that there was some evidence of carcinogenic activity of naphthalene in female B6C3F1 mice. When these mice were exposed to naphthalene vapors for two years at 10 or 30 ppm, they developed increased rates of bronchiolar and alveolar adenomas. No evidence of carcinogenic activity was found in male mice exposed to the same doses.[33]
- Embryonic roundworms (*Caenorhabditis elegans*) were used in mechanistic studies of the carcinogenicity of naphthalene. The authors hypothesized that the 1,4-naphthoquinone metabolite of naphthalene suppressed normal cell apoptosis.[34]

Humans

- The International Agency for Research on Cancer (IARC) of the World Health Organization (WHO) concluded that there was inadequate evidence to evaluate the carcinogenicity of naphthalene to humans, but found sufficient evidence in animals to conclude that naphthalene is carcinogenic. IARC concluded their evaluation by placing naphthalene in Group 2B, possibly carcinogenic to humans.[4] See the text box on **Cancer**.
  Cancer: Government agencies in the United States and abroad have developed programs to evaluate the potential for a chemical to cause cancer. Testing guidelines and classification systems vary. To learn more about the meaning of various cancer classification descriptors listed in this fact sheet, please visit the appropriate reference, or call NPIC.

- Naphthalene is classified as Group C, possible human carcinogen, by the U.S. EPA. This is based on limited evidence of carcinogenicity following inhalation exposure in animals, and lack of data for humans.[35]

Reproductive or Teratogenic Effects:

Animals

- The Agency for Toxic Substances and Disease Registry (ATSDR) reviewed 45 studies that examined the genotoxic potential of naphthalene. Of these studies, 10 found evidence of chromosomal aberrations, gene mutations, recombination abnormalities, or DNA fragmentation.[1]
- Pregnant rabbits were fed 400 mg/kg naphthalene during days 6-18 of their gestation. No adverse effects were noted.[36]
- Researchers dosed pregnant mice by gavage with 300 mg/kg/day during gestation days 7-14. The mice had reduced weight gain, reduced survival rates, and gave birth to fewer young than the controls, although the young appeared normal.[20]
- The difference in results between the rabbit and rat studies above may have been due to the use of different carriers during administration, which could have altered the absorption potential of the compound.[1]
- Female rats were dosed with 50, 150, or 450 mg/kg/day of naphthalene during days 6-15 of gestation. Researchers measured decreases in maternal weight gain of 31% and 53% in the mid-dose and high-dose groups, respectively, compared to the controls. No teratogenic effects were noted.[1,37]

Humans

- Hemolytic anemia has been reported in infants born to mothers who ingested mothballs or inhaled high concentrations of naphthalene vapors during pregnancy.[13,38]

- Naphthalene was detected in human breast milk from women in four urban areas near industrial facilities or chemical manufacturing plants.[39] Naphthalene in the nursing infants was not reported and no further information was found.
- The umbilical cord blood of pregnant women exposed to naphthalene and other volatile organic compounds including methylcyclopentane and tetrachloroethylene contained elevated levels of cytokine-producing T-cells. These data suggest that naphthalene may cross the placenta and affect the immune system of the newborn.[40]

Fate in the Body:

Absorption

- Researchers applied 3.3 μg/cm^2 naphthalene to the skin of rats either alone or in combination with sandy or clay soil. Half of the dermally applied pure naphthalene reached the bloodstream in 2.1 hours. The absorption half-life of naphthalene and clay soil was 2.8 hours, and for naphthalene and sandy soil, 4.6 hours.[41] Although adsorption to soil slowed the absorption of naphthalene, the total amount eventually absorbed by the skin was unchanged.[41]
- Daily use of baby oil may have increased the absorption of naphthalene through the skin of an infant who wore diapers stored in naphthalene and subsequently developed acute hemolytic anemia.[42]
- Once inhaled, naphthalene is thought to enter the body by passive diffusion across the alveolar membranes. Researchers have proposed that naphthalene enters the body through intestinal membrane following oral exposure.[1] Evidence suggests that absorption of naphthalene flakes or balls through the intestine can continue for several days after ingestion due to the slow dissolution of the solid form.[1]
- No data were found quantifying rates of absorption of naphthalene in humans following either oral or dermal exposure.

Distribution

- Tissue distribution of naphthalene was examined following dermal exposure in rats. After 48 hours, 0.56 ± 0.14% (mean ± SE) of the dose was found in the treated skin, 0.02 ± 0.01% in the ileum, 0.01 ± 0.00% in the duodenum, and 0.01 ± 0.00% in the kidney. Most of the dose was excreted.[41]
- Naphthalene metabolites formed in the liver may reach the lungs via the bloodstream.[43]

- Researchers dosed chickens, swine, and dairy cows via oral intubation for 1 day in an acute study and for 31 days in a chronic exposure study to evaluate the distribution of naphthalene in tissues.[44] The kidneys of the chronically exposed chickens contained the greatest residual naphthalene and metabolites (2.4 ± 0.22% [mean ± SE] of the original dose), followed by lungs, spleen, liver, and heart.[44] Naphthalene residues in the acutely exposed chickens were greatest in the kidneys, fat, lungs, and the liver.[44] The kidneys contained 42.9 ± 1.05% of the dose 24 hours after exposure. Kidney concentrations dropped to 8 ± 1.07% in 72 hours.[44]
- Chickens orally dosed with naphthalene laid eggs with detectable levels of the naphthalene in both the albumin and yolk. Eggs contained the chemical following acute and chronic exposures.[44]
- Swine fat contained the greatest residues in the acute exposure case, but the heart, spleen and liver contained the greatest residues following chronic exposure. The fat of acutely exposed swine contained 3.48 ± 2.16% (mean ± SE) of the total dose after 24 hours and 2.18 ± 1.16% after 72 hours. In the chronically exposed swine, maximum tissue concentrations contained less than 0.2% of the dose.[44]
- Researchers examined the liver, kidneys, lungs, heart, spleen, and fat of dairy cows given oral doses of naphthalene. Tissue concentrations were all less than 1% following either chronic or acute exposure.[44] Residues of naphthalene were found in the cows' milk.[44]
- Naphthalene was detected in six of eight samples of human breast milk taken from women residing in urban areas. Concentrations were not reported.[39]

Metabolism

- Naphthalene is metabolically activated by a number of cytochrome P450 enzymes.[5] These metabolites are subsequently detoxified by glutathione.[9] They may also be metabolized by P450 enzymes and epoxide hydrolase prior to excretion.[1]
- The metabolite naphthalene oxide appears to be largely responsible for the toxicity of naphthalene in lung tissue, although other metabolites may also play a role.[9]
- Reactive metabolites of naphthalene include 1,2-naphthoquinone, 1,4-naphthoquinone, 1,2-dihydroxy-3,4-epoxy-1,2,3,4- tetrahydronaphthalene, and 1,2-naphthalene oxide. These metabolites may be involved in the toxicity of naphthalene.[1]
- Researchers repeatedly exposed mice to naphthalene via inhalation and noted reduced susceptibility of lung tissue to necrosis compared with damage from single exposures.[9,45] Researchers concluded that this resistance to injury may be due to increased levels of glutathione synthesis in the lung tissue.[9,46]
- Researchers have identified 30 metabolites of naphthalene in the urine of mammals. In rodents, the most common urinary metabolites were 1-naphthol, 2-naphthol, 1-naphthyl glucuronide, 1-naphthyl sulfate, 1,2-dihydro-1,2-dihydroxynaphthalene, 1,2-dihydroxynaphthalene, and 1,2-dihydro-2-hydroxy-1-napahthylglucuronide.[5]

- The metabolite 1,2-naphthoquinone reacted with the ocular tissues in rabbits dosed with naphthalene via gavage. The aqueous humor and ciliary body of the eye were affected, and the lenses were damaged from the reaction of 1,2-naphthoquinone with enzyme and structural proteins, as well as coenzymes.[47]

Excretion

- Workers were exposed to low levels of naphthalene, naphthols, and methylated naphthalenes during naphthalene distillation processes at a coke plant. Their urine contained the metabolites 1-naphthol, 2-naphthol, and 1,4-naphthoquinone.[48]
- Excretion rates of 1-napthol ranged from 0.19 to 0.31 mg/h in factory workers, with maximum excretion occurring 2-3 hours following cessation of the workday exposure. The biological half-life of the metabolite 1-naphthol was estimated at 4 hours. All of the workers in this study were smokers.[49]
- Researchers applied radio-labeled naphthalene to the skin of rats as pure compound, adsorbed to sandy soil, and adsorbed to clay soil. Excretion was measured in the feces, urine, and expired air for 48 hours after dosing. Rats treated with pure naphthalene excreted 70.3 ± 4.5% (mean and SE) in their urine, 3.7 ± 0.7% in their feces, and 13.6 ± 3.4% from their lungs. Rats exposed to treated sandy soil excreted 84.7 ± 3.8% of the naphthalene in their urine, 2.4 ± 0.1% in their feces, and 0.9 ± 0.1% from their lungs. Rats exposed to treated clay soil excreted 86.7 ± 0.4% of the radio-labeled dose in their urine, 2.2 ± 0.2% in their feces, and 5.9 ± 2.5% in expired air.[41]
- Rats exposed dermally to naphthalene excreted less than 0.5% of the parent compound in their urine. They also excreted the urinary metabolites 2,7-diydroxynaphthalene and 1,2-dihydroxynaphthalene. The metabolites 1,2-naphthoquinone, 1-naphthol, and 2-naphthol were also present in lower concentrations.[41]
- Naphthalene was excreted in the milk of a dairy cow within 8 hours of a single oral exposure of 30.69 mg. Other cows were orally exposed to 5.12 mg naphthalene per day for 31 days.[44] Upon cessation of exposure, the concentration of naphthalene in the milk dropped rapidly until it was no longer detected on day 33, 2 days after dosing ceased.[44]
- Chickens exposed to naphthalene via gavage in a single acute exposure or 31 consecutive daily exposures eliminated 75- 80% of the dose within 48 hours after exposure ceased.[44]

Medical Tests and Monitoring:

- Biomarkers of exposure to naphthalene have been reported in the scientific literature.[5] Scientists have used high-pressure liquid chromatography and gas chromatography to detect and quantify naphthalene metabolites in urine of laboratory animals and humans.[5] This method of testing for exposure to naphthalene has not been well-studied in humans, and its clinical significance is currently unknown.

- Smoking has been considered a major source of urinary metabolites of naphthalene.[5,50] In addition, levels of urinary naphthols may be affected by genetic polymorphisms in enzyme expression.[50] Exposure to the insecticide carbaryl may also lead to the formation of 1-naphthol, the metabolite used in workplace exposure monitoring.[9]
- Methods also exist for detecting naphthalene and its metabolites in blood, urine, feces, breast milk, and body fat but these tests are not routinely done in a doctor's office.[1]

Environmental Fate:

Soil

- Naphthalene may be lost from soil via evaporation, volatilization, and biodegradation. The relative importance of each pathway will vary depending on soil depth and the presence and composition of soil biota, including bacteria, fungi, cyanobacteria, and algae.[51,52]
- Soil and sediment bind naphthalene to a moderate extent depending on soil type. Naphthalene will move rapidly through sandy soil. However, increasing organic carbon content will increase naphthalene's sorption to soil.[51]
- Naphthalene will evaporate from the soil surface, but this process will decrease with increasing soil depth.[51]
  The "half-life" is the time required for half of the compound to break down in the environment.

  1 half-life = 50% remaining
  2 half-lives = 25% remaining
  3 half-lives = 12% remaining
  4 half-lives = 6% remaining
  5 half-lives = 3% remaining

  Half-lives can vary widely based on environmental factors. The amount of chemical remaining after a half-life will always depend on the amount of the chemical originally applied. It should be noted that some chemicals may degrade into compounds of toxicological significance.

- The effective half-lives for volatilization from soil at 1 cm and 10 cm depths were 1.1 days and 14.0 days, respectively. These values were determined through modeling a system that assumed no evaporation.[53] See the text box on **Half-life**.
- Biodegradation will remove naphthalene from soil, with an estimated half-life of more than 80 days. If the soil is contaminated with other polycyclic aromatic hydrocarbons (PAHs), biodegradation may be much more rapid, with a half-life of a few hours.[51]

- Naphthalene is broken down by soil bacteria to naphthalene diol, salicylic acid, and catechol. Some bacteria may utilize naphthalene as their sole carbon source.[54] Bacterial oxidation pathways include five metabolites of naphthalene along the degradation pathway: *cis*-1,2-dihydroxy-1,2-dihydronaphthalene, 1,2-dihydroxynaphthalene, *cis*-o-hydroxybenzalpyruvic acid, salicylic acid, and catechol, which was subsequently subject to ring cleavage.[55]
- Fungal degradation of naphthalene produces naphthalene-1,2-oxide via cytochrome P-450 oxidation. The oxide can be subsequently hydrolyzed to *trans*-1,2-dihydroxy-1,2-dihydronaphthalene, or alternatively conjugate with glucuronide or sulfate to break down first to 1-naphthol and 2-naphthol and subsequently to 4-hydroxy-1-tetralone.[55] Other reported metabolites include naphthalene *trans*-1,2-dihydrodiol, 1,2-naphthoquinone, and 1,4-naphthoquinone.[52]

Water

- Naphthalene will be lost from water by volatilization, sorption, photolysis, and biodegradation.[51] The relative contributions of these processes will depend in part on the water's characteristics, including depth, flow rate, and contamination level.[51]
- Wastewater from secondary treatment plants was discharged into spreading basins for groundwater recharge. Naphthalene concentrations in the wastewater declined 68-94% during movement across the settling basin.[56]
- Researchers modeling volatilization of naphthalene from water concluded that evaporation is affected more by water movement than by air movement above the water's surface.[57] Maximum volatilization would be expected to occur under conditions of warm temperatures, shallow waters, and wind. Surface films, aerosol formation, waves, water depth, and current speed will affect volatilization rates.[57]
- The presence of algae in the water at concentrations of 1-10 mg chlorophyll a per liter of water increased the photodegradation of naphthalene.[58]
- Cyanobacteria and microalgae metabolize naphthalene.[52] The primary metabolite was found to be 1-naphthol, with 4-hydroxy- 1-tetralone and *cis*-naphthalene dihydrodiol as lesser metabolites.[59,60]

Air

- Hydroxyl radicals react with naphthalene in the atmosphere at a rate of 2.17 x 10-11 cm^3/molecules sec.[61] Based on this rate, the half-life of naphthalene in air is less than one day.[1] Nitrate may also react with naphthalene in the atmosphere.[51]
- Napthalene degraded to form 1-naphthol, 2-naphthol, 1-nitronaphthalene, and 2-nitronaphthalene in the presence of hydroxyl radicals and nitric oxides. Researchers noted that other unidentified metabolites also were present.[62]

Plants

- Naphthalene may be deposited on foliage as a result of volatilization from the soil. In addition, naphthalene may be transferred from the plant's roots to the shoots during transpiration.[63]
- Naphthalene adsorbed onto the roots of fescue and alfalfa placed in an aqueous solution containing 18.4 mg/L dissolved naphthalene. The amount adsorbed was affected by the lipid contents of the roots and plant age.[64]
- The marsh grass Spartina alterniflora contained more residues of naphthalene in its roots than in the new shoot growth when grown in pots containing contaminated soil.[65] Concentrations in soil were four orders of magnitude greater than those found in shoots.[65] Roots contained up to 43 µg/g and leaves contained up to 0.2 µg/g naphthalene.[65]
- PAH residues in cabbage, carrots, leeks, lettuce and endive grown in an industrial area in northern Greece were most similar to the mixture in air, suggesting that gaseous deposition is a major accumulation pathway.[66]

Indoor

- Naphthalene has been detected in indoor air. It is a product of combustion when organic materials are burned. Tobacco smoking, cooking, and moth repellents may all be sources of naphthalene indoors.[1]
- Clothing stored with naphthalene mothballs may adsorb the naphthalene. The clothing itself then may act as a source of naphthalene in indoor air.[67]
- Indoor air concentrations measured in 24 low-income houses in Durham, NC ranged from 334 to 9700 ng/m^3 naphthalene, with a mean concentration of 2190 ng/m^3 (SD=1870 ng/m^3).[68] House dust contained a mean concentration of 0.33 µg/g naphthalene (SD=0.85 µg/g).[68] Samples were collected from rural and urban areas, and included both smokers' and nonsmokers' households.[68]

Food Residue

- No tolerances were found for naphthalene in food.
- Cabbage, carrots, leeks, lettuce and endives were grown in an industrial area in northern Greece, then washed and peeled. The vegetables contained naphthalene at concentrations ranging from 0.37 µg/kg (cabbage) to 63 µg/kg (endive) dry weight.[66]
- Researchers measured naphthalene residues in samples of the food consumed by 24 low-income households in North Carolina. They found 0.01-18.7 µg/kg naphthalene in the adults' food (mean= 3.75 µg/kg) and 0.10-54.9 µg/kg naphthalene in the food of the children (mean= 4.08 µg/kg).[68]

Ecotoxicity Studies:

Birds

The acute oral LD_{50} in bobwhite quail (*Colinus virginianus*) was 2690 mg/kg, and the LC_{50} was estimated as greater than 5620 mg/kg. In both cases, naphthalene was considered by the U.S. EPA as practically nontoxic by ingestion.[3]

Fish and Aquatic Life

- The 96-hour LC_{50} for rainbow trout (*Onchorynchus mykiss*) exposed to naphthalene was 2.0 mg/L. The NOAEC was 0.86 mg/L. The 96-hour LC_{50} in bluegill sunfish (*Lepomis macrochirus*) was 3.2 mg/L and the NOAEC was 1.4 mg/L. Researchers estimated the 96-hour LC_{50} in fathead minnows (*Pimephales promelas*) to be 6.6 mg/L. Naphthalene is moderately toxic to these three species of fish.[3] Embryo-larval toxicity in fathead minnows was noted at concentrations of 0.85 mg/L, and the NOAEC was 0.62 mg/L.[69]
- The 40-day NOAEC for coho salmon (*Onchoryncus kisutch*) exposed to naphthalene was 0.37 mg/L and the 40-day LOAEC was 0.67 mg/L. No ecotoxicity category was assigned.[3] Chronic exposure resulted in reductions in feeding, growth, and survival rates.[69]
- Rainbow trout (*Salmo gairdneri*) exposed to 9.2 ppb naphthalene for 72 hours followed by 72 hours of exposure to clean water contained increasing concentrations of naphthalene through the exposure period. Fish skin contained 2103 ppb naphthalene and 440 ppb of metabolites at 72 hours of exposure. Depuration for 72 hours reduced naphthalene levels to 223 ppb and metabolites to 275 ppb in the skin.[70] Epidermal mucus was suggested to be an excretion pathway for some contaminants.[70]
- Atlantic croaker fish (*Micropogonias undulatus*) were exposed to naphthalene at concentrations of 0.5 or 1.0 ppm for up to eight weeks, beginning two weeks before sexual maturity. Fewer exposed fish reached sexual maturity, and many fish that matured exhibited reduced ovarian growth and delayed or arrested oocyte development.[31]

  EC_{50}: The median effective concentration (EC_{50}) may be reported for sublethal or ambiguously lethal effects. This measure is used in tests involving species such as aquatic invertebrates where death may be difficult to determine. This term is also used if sublethal events are being monitored.

  Newman, M.C.; Unger, M.A. *Fundamentals of Ecotoxicology*; CRC Press, LLC.: Boca Raton, FL, 2003; p 178.

- Reports regarding the toxicity of naphthalene to water fleas (*Daphnia magna*) are variable. The 48-hour EC_{50} was 1.6 mg/L, the NOAEC for mortality as an endpoint was 0.48 mg/L, and the NOAEC for sublethal effects was greater than 8.8 mg/L. Naphthalene is moderately toxic to water fleas according to the U.S. EPA.[3] See the text box on **EC_{50}**.

- Pacific oysters (*Crassostrea gigas*) exposed to naphthalene had a 96-h EC_{50} of 199 mg/L, which placed naphthalene in the U.S. EPA's practically nontoxic category for this species. In contrast, the 96-h LC_{50} of grass shrimp (*Palaemonetes pugio*) was 2.35 mg/L, which is considered moderately toxic.[3]
- Naphthalene is considered slightly toxic to the green algae (*Chlorella vulgaris*) by the U.S. EPA. The 48-hour EC_{50} was 33 mg/L.[3]

Terrestrial Invertebrates

- No data were found on the acute toxicity of naphthalene to the honey bee (*Apis mellifera*).
- Soil invertebrates (*Folsomia candida*, or springtail) were exposed to naphthalene in chronic tests. The NOAEC was 88 µmol/kg soil and the LOAEC was 409 µmol/kg soil based on effects on reproduction and survival.[3]
- Earthworms (*Enchytaeus crypticus*) were also studied following chronic exposure to naphthalene in soil. The NOAEC was 220 µmol/kg soil and the LOAEC was 2045 µmol/kg soil.[3]
- *Pseudomonas putida* bacteria were added to soil that had been mixed with naphthalene 68 days earlier, and soil that had just been mixed with naphthalene. The bacteria metabolized the freshly treated soil much more rapidly, suggesting that aging of naphthalene in the soil matrix made it unavailable for biodegradation.[71] In both cases, most of the degradation occurred in the first 3 days.[71]
- In the same study, earthworms (*Eisenia foetida*) incorporated 2.3% of radioactive naphthalene from freshly treated soil into their bodies, but only 1.4% of radioactive naphthalene that had been added to soil 68 days earlier.[71]

Regulatory Guidelines:

- The U.S. EPA set the chronic reference dose or cRfD for naphthalene at 0.1 mg/kg/day and acute reference dose or aRfD at 0.4 mg/kg/day for naphthalene based on studies of exposure in rats.[3] See the text box on **Reference Dose (RfD)**.
  Reference Dose (RfD): The RfD is an estimate of the quantity of chemical that a person could be exposed to every day for the rest of their life with no appreciable risk of adverse health effects. The reference dose is typically measured in milligrams (mg) of chemical per kilogram (kg) of body weight per day.

  U.S. Environmental Protection Agency, Integrated Risk Information System, IRIS Glossary, 2009. https://www.epa.gov/iris/iris-glossary#r

- The U.S. EPA classifies naphthalene as Group C, possible human carcinogen.[35] The IARC classification is Group 2B, possibly carcinogenic to humans.[4] See the text box on **Cancer**.

- The Recommended Exposure Limit (REL) for a time-weighted average (TWA) is 10 ppm or 50 mg/m^3, and the Short Term Exposure Limit (STEL) is 15 ppm or 75 mg/m^3.[7,2]
- The Permissible Exposure Limit (PEL) is 10 ppm or 50 mg/m^3 for naphthalene.[7,2]

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References:

1. *Toxicological Profile for Naphthalene, 1-methylnaphthalene, and 2-methylnaphthalene*; U.S Department of Health and Human Services, Public Health Service, Agency for Toxic Substances and Disease Registry. https://wwwn.cdc.gov/TSP/ToxProfiles/ToxProfiles.aspx?id=240&tid=43 (accessed March 2010), updated Sept 2010.
2. Bischoff, K. Naphthalene. *Clinical Veterinary Toxicology*; Plumlee, K. H., Ed.; Mosby: St. Louis, MO, 2004, pp. 163-164.
3. *Reregistration Eligibility Decision (RED): Naphthalene*; EPA 738-R-07-010; U.S. Environmental Protection Agency, Office of Prevention, Pesticides and Toxic Substances, Office of Pesticide Programs, U.S. Government Printing Office: Washington, DC, 2008.
4. WHO. *IARC Monographs on the Evaluation of Carcinogenic Risks to Humans*; International Agency for Research on Cancer, World Health Organization: Lyon, France, 2002; pp 367-435.
5. Preuss, R.; Angerer, J.; Drexler, H. Naphthalene - An environmental and occupational toxicant. *Int. Arch. Occup. Environ. Health* 2003, 76, 556-576.
6. *Hazardous Substances Data Bank (HSDB): Naphthalene*; U.S. Department of Health and Human Services, National Institutes of Health, National Library of Medicine. https://pubchem.ncbi.nlm.nih.gov/source/hsdb/184 (accessed March 2010), updated June 2005.
7. WHO. *Naphthalene (PIM 363)*; International Programme on Chemical Safety, World Health Organization. https://inchem.org/documents/pims/chemical/pim363.htm (accessed March 2010), updated Sept 2000.
8. Bogen, K. T.; Benson, J. M.; Yost, G. S.; Morris, J. B.; Dahl, A. R.; Clewell, H. J., III; Krishnan, K.; Omiecinski, C. J. Naphthalene metabolism in relation to target tissue anatomy, physiology, cytotoxicity and tumorigenic mechanism of action. *Reg. Toxicol. Pharmacol.* 2008, 51, S27-S36.

9. Buckpitt, A. R.; Boland, B.; Isbell, M.; Morin, D.; Shultz, M.; Baldwin, R.; Chan, K.; Karlsson, A.; Lin, C.; Taff, A.; West, J.; Fanucchi, M.; Van Winkle, L.; Plopper, C. G. Naphthalene-induced respiratory tract toxicity: metabolic mechanisms of toxicity. *Drug Metab. Rev.* 2002, 34 (4), 791-820.
10. Reigart, J. R.; Roberts, J. R. Fumigants. *Recognition and Management of Pesticide Poisonings*, 5th ed.; U.S. Environmental Protection Agency, Office of Prevention, Pesticides and Toxic Substances, Office of Pesticide Programs, U.S. Government Printing Office: Washington, DC, 1999; pp 156-162.
11. Valaes, T.; Doxiadis, S. A.; Fessas, P. Acute hemolysis due to naphthalene inhalation. *J. Pediatr.* 1963, 63, 904-915.
12. Santucci, K.; Shah, B. R. Association of naphthalene with acute hemoyltic anemia. *Acad. Emerg. Med.* 2000, 7, 42-47.
13. Molloy, E. J.; Doctor, B. A.; Reed, M. D.; Walsh, M. C. Perinatal toxicity of domestic naphthalene exposure. *J. Perinatol.* 2004, 24, 792-793.
14. Phimister, A. J.; Lee, M. G.; Morin, D.; Buckpitt, A. R.; Plopper, C. G. Glutathione depletion is a major determinant of inhaled naphthalene respiratory toxicity and naphthalene metabolism in mice. *Toxicol. Sci.* 2004, 82, 268-278.
15. Phimister, A. J.; Nagasawa, H. T.; Buckpitt, A. R.; Plopper, C. G. Prevention of naphthalene-induced pulmonary toxicity by glutathione prodrugs: roles for glutathione depletion in adduct formation and cell injury. *J. Biochem. Mol. Toxicol.* 2005, 19 (1), 42-51.
16. Wilson, A. S.; Davis, C. D.; Williams, D. P.; Buckpitt, A. R.; Pirmohamed, M.; Park, B. K. Characterization of the toxic metabolite(s) of naphthalene. *Toxicol.* 1996, 114, 233-242.
17. Papciak, R. J.; Malloy, V. T. Acute toxicological evaluation of naphthalene. *J. Am. Coll. Toxicol.* 1990, 1 (1), 17-19.
18. Gaines, T. B. Acute toxicity of pesticides. *Toxicol. Appl. Pharmacol.* 1969, 14, 515-534.
19. Shopp, G. M.; White, K. L., Jr.; Holsapple, M. P.; Barnes, D. W.; Duke, S. S.; Anderson, A. C.; Condie, L. W.; Hayes, J. R.; Borzelleca, J. F. Naphthalene toxicity in CD-1 mice: general toxicology and immunotoxicology. *Fundam. Appl. Toxicol.* 1984, 4, 406-419.
20. Plasterer, M. R.; Bradshaw, W. S.; Booth, G. M.; Carter, M. W. Developmental toxicity of nine selected compounds following prenatal exposure in the mouse: naphthalene, p-nitrophenol, sodium selenate, dimethyl phthalate, ethylenethiourea, and four glycol ether derivatives. *J. Toxicol. Environ. Health* 1985, 15, 25-38.
21. West, J. A. A.; Pakehham, G.; Morin, D.; Fleschner, C. A.; Buckpitt, A. R.; Plopper, C. G., Inhaled naphthalene causes dose dependent clara cell cytotoxicity in mice but not in rats. *Toxicol. Appl. Pharmacol.* 2001, 173, 14-119.
22. Witschi, H. R.; Pinkerton, K. E.; Van Winkle, L. S.; Last, J. A. Toxic responses of the respiratory system. *Casarette and Doull's Toxicology: The basic science of poisons*, 7th ed.; Klaassen, C. D., Ed.; McGraw Hill Medical: New York, 2008; p 613.
23. Zuelzer, W. W.; Apt, L. Acute hemolytic anemia due to naphthalene poisoning. *J. Am. Med. Assoc.* 1949, 141 (3), 185-190.

24. Desnoyers, M.; Hebert, P. Heinz body anemia in a dog following possible naphthalene ingestion. *Vet. Clin. Pathol.* 1995, 24 (4), 124-125.
25. *Health Effects Support Document for Naphthalene*; EPA 822-R-03-005; U.S. Environmental Protection Agency, Office of Water, Health and Ecological Criteria Division, U.S. Governement Printing Office: Washington, DC, 2003.
26. Grant, W. M. *Toxicology of the Eye*, 3rd ed.; Charles C. Thomas: Springfield, IL, 1986, pp 650-645.
27. Coombs, D. 27. Naphthalene 4-week inhalation study in rats. Unpublished lab project no. LDA 1/921559, 1993, submitted to U.S. Environmental Protection Agency by Huntingdon Research Center, Ltd. EPA MRID 42934901 *Reregistration Elegibility Decision (RED) Naphthalene*; U.S. Environmental Protection Agency, Office of Prevention, Pesticides and Toxic Substances, Office of Pesticide Programs, U.S. Government Printing Office: Washington, DC, 2008, p 14.
28. Frantz, S.; Van Miller, J.; Papciak, R. Ninety-day (sub-chronic) dermal toxicity study with naphthalene in albino rats: final report. Unpublished project no. 49- 539, 1986, submitted to U.S. Environmental Protection Agency by Union Carbide Bushy Run Research Center. EPA MRID 40021801. *Reregistration Elegibility Decision (RED) Naphthalene*; U.S. Environmental Protection Agency, Office of Prevention, Pesticides and Toxic Substances, Office of Pesticide Programs, U.S. Government Printing Office: Washington, DC, 2008, p 14.
29. Shah, B. R.; Santucci, K. Naphthalene induced acute hemolytic-anemia in children with glucose-6-phosphate-dehydrogenase (G-6-PD) deficiency - naphthalene has no legitimate place on the market as a moth repellent. *Pediatr. Res.* 1995, 37 (4), A144.
30. Harden, R. A.; Baetjer, A. M. Aplastic anemia following exposure to paradichlorobenzene and naphthalene. *J. Occup. Med.* 1978, 20 (12), 820-822.
31. Thomas, P.; Budiantara, L. Reproductive life history stages sensitive to oil and naphthalene in Atlantic croaker. *Mar. Environ. Res.* 1995, 39, 147-150.
32. Abdo, K. M.; Grumbein, S.; Chou, B. J.; Herbert, R. Toxicity and carcinogenicity study in F344 rats following 2 years of whole-body exposure to naphthalene vapors. *Inhal. Toxicol.* 2001, 13, 931-950.
33. *NTP Technical Report on the toxicology and carcinogenesis studies of naphthalene (CAS No. 91-20-3) in B6C3F1 mice*; U.S. Department of Health and Human Services, Public Health Service, National Institutes of Health, National Toxicology Program: Research Triangle Park, NC, 1992; pp 35-36.
34. Kokel, D.; Li, Y.; Qin, J.; Xue, D., The nongenotoxic carcinongens naphthalene and para-dichlorobenzene suppress apoptosis in Caenorhabditis elegans. *Nat. Chem. Biol.* 2006, 2 (6), 338-345.
35. *Integrated Risk Information System (IRIS): Naphthalene (CASRN 91-20-3)*; U.S. Environmental Protection Agency. https://cfpub.epa.gov/ncea/iris2/chemicalLanding.cfm?substance_nmbr=436 (accessed March 2010), updated Jan 2008.

36. Developmental toxicity study in rabbits: naphthalene. Unpublished report number PH 329-TX-001 85, 1986, submitted to U.S. Department of Health and Human Services by Pharmakon Research International, Inc. *Toxicologial Profile for naphthalene, 1-methylnaphthalene, and 2-methylnaphthalene*; U.S Department of Health and Human Services, Public Health Service, Agency for Toxic Subtances and Disease Registry: Atlanta, 2005; p 75.
37. *Developmental toxicity of naphthalene (CAS No. 91-20-3) administered by gavage to Sprague-Dawley (CD) rats on gestational days 6 through 15: Final study report and appendix*; U.S. Department of Health and Human Services, Public Health Service, National Institutes of Health, National Toxicology Program, TER91006: Research Triangle Park, NC, 1991, p 76.
38. Anziulewicz, J. A.; Dick, H. J.; Chiarulli, E. E. Transplacental naphthalene poisoning. *Am. J. Obstet. Gynecol.* 1959, 78 (3), 519-521.
39. Pellizzari, E. D.; Hartwell, T. D.; Harris, B. S. H., III; Waddell, R. D.; Whitaker, D. A.; Erickson, M. D. Pureable organic compounds in mother's milk. *Bull. Environ. Contam. Toxicol.* 1982, 28, 322-328.
40. Lehmann, I.; Thoelke, A.; Rehwagen, M.; Rolle-Kampczyk; Schlink, U.; Schulz, R.; Borte, M.; Diez, U.; Herbarth, O. The influence of maternal exposure to volatile organic compounds on the cytokine secretion profile of neonatal T cells. *Environ. Toxicol.* 2002, 17 (3), 203-210.
41. Turkall, R. M.; Skowronsi, G. A.; Abdel-Rahman, M. S. A comparative study of the kinetics and bioavailability of pure and soil-adsorbed naphthalene in dermally exposed male rats. *Arch. Environ. Contam. Toxicol.* 1994, 26, 504-509.
42. Schafer, W. B. Acute hemolytic anemia related to naphthalene. *Pediatrics* 1951, 7, 172-174.
43. Buckpitt, A. R., Evidence for hepatic formation, export and covalent binding of reactive naphthalene metabolites in extrahepatic tissues *in vivo*. *J. Pharmacol. Exp. Ther*. 1983, 225 (1), 8-16. 44. Eisele, G. R. Naphthalene distribution in tissues of laying pullets, swine, and dairy cattle. *Bull. Environ. Contam. Toxicol.* 1985, 34, 549-556.
44. O'Brien, K. A. F.; Suverkropp, C.; Kanekal, S.; Plopper, C. G.; Buckpitt, A. R. Tolerance to multiple doses of the pulmonary toxicant, naphthalene. *Toxicol. App. Pharmacol.* 1989, 99, 487-500.
45. West, J. A. A.; Buckpitt, A. R.; Plopper, C. G. Elevated air 46. way GSH reysnthesis confers protection to Clara cells from naphthalene injury in mice made tolerant by repeated exposures. *J. Pharmacol. Exp. Ther*. 2000, 294 (2), 516-523.
46. Van Heyningen, R.; Pirie, A. The metabolism of naphthalene and its toxic effect on the eye. *Biochem. J*. 1967, 102, 842-852.
47. Bieniek, G. Urinary naphthols as an indicator of exposure to naphthalene. *Scand. J. Work Environ. Health* 1997, 23, 414-420.
48. Bieniek, G., The presence of 1-naphthol in the urine of industrial workers exposed to naphthalene. *Occup. Environ. Med.* 1994, 51, 357-359.

49. Yang, M.; Koga, M.; Tawamoto, T. A study for the proper application of urinary naphthols, new biomarkers for airborne polycyclic aromatic hydrocarbons. *Arch. Environ. Contam. Toxicol.* 1999, 36, 99-108.
50. Howard, P. H. Naphthalene. *Handbook of Environmental Fate and Exposure Data for Organic Chemcials*; Jarvis, W. F.; Sage, G. W.; Basu, D. K.; Gray, D. A.; Meylan, W.; Crosbie, E. K., Eds; Lewis Publishers: Chelsea, MI, 1989; Vol. 1, pp 408-422.
51. Cerniglia, C. E. Biodegradation of polycyclic aromatic hydrocarbons. *Biodegradation* 1992, 3, 351-368.
52. Jury, W. A.; Spencer, W. F.; Farmer, W. J. Behavior assessment model for trace organics in soil: III. Application of screening model. *J. Environ. Qual.* 1984, 13 (4), 573-479.
53. Treccani, V.; Walker, N.; Wiltshire, G. H., The metabolism of naphthalene by soil bacteria. *J. Gen. Microbiol.* 1954, 11, 341-348.
54. Cerniglia, C. E., Microbial metabolism of polycyclic aromatic hydrocarbons. *Adv. Appl. Microbiol.* 1984, 30, 31-71.
55. Bouwer, E. J.; McCarty, P. L.; Bouwer, H.; Rice, R. C. Organic contaminant behavior during rapid infiltration of secondary wastewater at the Phoenix 23rd Avenue Project. *Water Res.* 1984, 18 (4), 463-472.
56. Southworth, G. R. The role of volatilization in removing polycyclic aromatic hydrocarbons from aquatic environments. *Bull. Environ. Contam. Toxicol.* 1979, 21, 507-514.
57. Zepp, R. G.; Schlotzhauer, P. F. Influence of algae on photolysis rates of chemicals in water. *Environ. Sci. Technol.* 1983, 17, 462-466.
58. Cerniglia, C. E.; van Baalen, C.; Gibson, D. T., Metabolism of naphthalene by the cyanobacterium Oscillatoria sp., strain JCM. *J. Gen. Microbiol.* 1980, 116, 485-494.
59. Cerniglia, C. E.; Gibson, D. T.; van Baalen, C. Oxidation of naphthalene by cyanobacteria and microalgae. *J. Gen. Microbiol.* 1980, 116, 495-500.
60. Atkinson, R. Kinetics and mechanisms of the gas-phase reactions of the hydroxyl radical with organic compounds under atmospheric conditions. *Chem. Rev.* 1986, 86, 69-201.
61. Atkinson, R.; Arey, J.; Zielinska, B.; Aschmann, S. M., Kinetics and products of the gas-phase reactions of OH radicals and N2O5 with naphthalene and biphenyl. Environ. Sci. Technol. 1987, 21, 1014-1022.
62. Gao, Y.; Collins, C. D. Uptake pathways of polycyclic aromatic hydrocarbons in white clover. *Environ. Sci. Technol.* 2009, 43 (16), 6190-6195.
63. Schwab, A. P.; Al-Assi, A. A.; Banks, M. K. Adsorption of naphthalene onto plant roots. *J. Environ. Qual.* 1998, 27, 220-224.
64. Watts, A. W.; Ballestero, T. P.; Gardner, K. H. Uptake of polycyclic aromatic hydrocarbons (PAHs) in salt marsh plants Spartina alterniflora grown in contaminated sediments. *Chemosphere* 2006, 62, 1253-1260.

65. Kipopoulou, A. M.; Manoli, E.; Samara, C. Bioconcentration of polycyclic aromatic hydrocarbons in vegetables grown in an industrial area. *Environ. Pollut.* 1999, 106, 369-380.
66. De Coensel, N.; Desmet, K.; Sandra, P.; Gorecki, T. Domestic sampling: exposure assessment to moth repellent products using ultrasonic extraction and capillary GC-MS. *Chemosphere* 2008, 71, 711-716.
67. Chuang, J. C.; Callahan, P. J.; Lyu, C. W.; Wilson, N. K. Polycyclic aromatic hydrocarbon exposures of children in low-income families. *J. Expo. Anal. Environ. Epidemiol.* 1999, 2, 85-98.
68. *Revised Ecological Risk Assessment for the Reregistration Elegibility Decision (RED) for Naphthalene*; U.S. Environmental Protection Agency, Offices of Prevention, Pesticides, and Toxic Substances, Environmental Fate and Effects Division, U.S. Government Printing Office: Washington, DC, 2008.
69. Varanasi, U.; Uhler, M.; Stranahan, S. I. Uptake and release of naphthalene and its metabolites in skin and epidermal mucus of salmonids. *Toxicol. Appl. Pharmacol.* 1978, 44, 277-289.
70. Kelsey, J. W.; Alexander, M., Declining bioavailability and inappropriate estimation of risk of persistent compounds. *Environ. Toxicol. Chem.* 1997, 16 (3), 582-585.
71. *NIOSH Pocket Guide to Chemical Hazards: Naphthalene*; U.S. Department of Health and Human Services, Centers for Disease Control and Prevention, National Institure for Occupational Safety and Health. http://www.cdc.gov/NIOSH/npg/npgd0439.html (accessed March 2010), updated Sept 2005.