ENVIRONMENTAL CONTAMINANTS ENCYCLOPEDIA

BENZO[A]PYRENE (BaP) ENTRY

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Like a library or most large databases (such as EPA's national STORET water quality database), this document contains information of variable quality from very diverse sources. In compiling this document, mistakes were found in peer reviewed journal articles, as well as in databases with relatively elaborate quality control mechanisms [366,649,940]. A few of these were caught and marked with a "[sic]" notation, but undoubtedly others slipped through. The [sic] notation was inserted by the editors to indicate information or spelling that seemed wrong or misleading, but which was nevertheless cited verbatim rather than arbitrarily changing what the author said.

Most likely additional transcription errors and typos have been added in some of our efforts. Furthermore, with such complex subject matter, it is not always easy to determine what is correct and what is incorrect, especially with the "experts" often disagreeing. It is not uncommon in scientific research for two different researchers to come up with different results which lead them to different conclusions. In compiling the Encyclopedia, the editors did not try to resolve such conflicts, but rather simply reported it all.

It should be kept in mind that data comparability is a major problem in environmental toxicology since laboratory and field methods are constantly changing and since there are so many different "standard methods" published by EPA, other federal agencies, state agencies, and various private groups. What some laboratory and field investigators actually do for standard operating practice is often a unique combination of various standard protocols and impromptu "improvements." In fact, the interagency task force on water methods concluded that [1014]:

> It is the exception rather than the rule that water-quality monitoring data from different programs or time periods can be compared on a scientifically sound basis, and that...
>
> No nationally accepted standard definitions exist for water quality parameters. The different organizations may collect data using identical or standard methods, but identify them by different names, or use the same names for data collected by different methods [1014].

Differences in field and laboratory methods are also major issues related to (the lack of) data comparability from media other than water: soil, sediments, tissues, and air.

In spite of numerous problems and complexities, knowledge is often power in decisions related to chemical contamination. It is therefore often helpful to be aware of a broad universe of conflicting results or conflicting expert opinions rather than having a portion of this information arbitrarily censored by someone else. Frequently one wants to know of the existence of information, even if one later decides not to use it for a particular application. Many would like to see a high percentage of the information available and decide for themselves what to throw out, partly because they don't want to seem uniformed or be caught by surprise by potentially important information. They are in a better position if they can say: "I knew about that data, assessed it based on the following quality assurance criteria, and decided not to use it for this application." This is especially true for users near the end of long decision processes, such as hazardous site cleanups, lengthy ecological risk assessments, or complex natural resource damage assessments.

For some categories, the editors found no information and inserted the phrase "no information found." This does not necessarily mean that no information exists; it

simply means that during our efforts, the editors found none. For many topics, there is probably information "out there" that is not in the Encyclopedia. The more time that passes without encyclopedia updates (none are planned at the moment), the more true this statement will become. Still, the Encyclopedia is unique in that it contains broad ecotoxicology information from more sources than many other reference documents. No updates of this document are currently planned. However, it is hoped that most of the information in the encyclopedia will be useful for some time to come even with out updates, just as one can still find information in the 1972 EPA Blue Book [12] that does not seem well summarized anywhere else.

Although the editors of this document have done their best in the limited time available to insure accuracy of quotes as being "what the original author said," the proposed interagency funding of a bigger project with more elaborate peer review and quality control steps never materialized.

The bottom line: The editors hope users find this document useful, but don't expect or depend on perfection herein. Neither the U.S. Government nor the National Park Service make any claims that this document is free of mistakes.

The following is one topic entry (one file among 118). See the file entitled RERENCE for the indentity of numbered references in brackets. See the README file for an introduction, an explanation of how to search and otherwise use this document, the oganization of each entry, information quality, copyright issues, and other entries (other topics) covered.

HOW TO CITE THIS DOCUMENT: As mentioned above, for critical applications it is better to obtain and cite the original publication after first verifying various data quality assurance concerns. For more routine applications, this document may be cited as:

> **Irwin, R.J., M. VanMouwerik, L. Stevens, M.D. Seese**, and **W. Basham.** 1997. Environmental Contaminants Encyclopedia. National Park Service, Water Resources Division, Fort Collins, Colorado. Distributed within the Federal Government as an Electronic Document (Projected public availability on the internet or NTIS: 1998).

Benzo(a)pyrene (BaP, B(a)P, BP, CAS number 50-32-8)

Brief Introduction:

Br.Class: General Introduction and Classification Information:

Benzo(a)pyrene is a high molecular weight, 5-ring PAH, and an EPA Priority Pollutant and EPA-classified carcinogen [446,697,634].

Benzo(a)pyrene's (BaP) release to the environment is quite wide-spread since it is an ubiquitous product of incomplete combustion [366].

Benzo(a)pyrene is included on the expanded scan list used by the Geochemical and Environmental Research Group (GERG) Laboratory at Texas A&M [828]. This list includes most of the PAHs recommended by the NOAA's National Status and Trends program [680].

One of the PAHs found by NASA in 1996 on a rock alleged to be a meteorite from mars (see Uses/Sources section below for details).

On many hazardous substances lists. If only one pound of BAP is released to the environment within a 24-hour period, EPA must be notified [881].

Br.Haz: General Hazard/Toxicity Summary:

The heavier (4-, 5-, and 6-ring) PAHs are more persistent than the lighter (2- and 3-ring) PAHs and tend to have greater carcinogenic and other chronic impact potential [796].

In some ways, BAP is a sentinel or worst case PAH, particularly from a carcinogenic standpoint. From what is currently known about benzo[a]pyrene, the federal government has developed regulatory standards and guidelines to protect people from the potential health effects of PAHs in drinking water [881].

Acute toxicity is rarely reported in humans, fish, or wildlife, as a result of exposure to low levels of a single PAH compound such as this one. PAHs in general are more frequently associated with chronic risks. These risks include cancer and often are the result of exposures to complex mixtures of chronic-risk aromatics (such as PAHs, alkyl PAHs, benzenes, and alkyl benzenes), rather than exposures to low levels of a single compound (Roy Irwin, National Park Service, Personal Communication, 1996, based on an overview of literature on hand). See also: PAHs as a group entry.

Potential effects of PAHs on humans were summarized by the Agency for Toxic Substances and Disease Registry in a 1995 toxicological profile for polycyclic aromatic hydrocarbons [881], so no lengthy summary will be attempted here.

In an effort somewhat similar to the TCDD equivalents method for dioxins, risk assessment researchers are considering assigning relative potency to various PAHs using Benzo(a)pyrene as the standard because it is the most studied PAH (Rita Schoeny, Environmental Protection Agency, Cincinnati, personal communication, 1995).

> NOTE: Emphasis on B(a)P has increased tremendously due to its carcinogenicity, relative ease of analysis, and the belief by investigators that this compound serves as an indicator for the presence of other PAHs which contaminate the environment [794].

Immune Effects:

> Most human health concerns for B(a)P relate to cancer; other concerns include immunosuppression, genetic damage, and various reproductive problems [366].
>
> All the steps necessary for cellular transformation and cancer induction were demonstrated in cultured human skin fibroblasts: inducible AHH activity, altered cellular proliferation kinetics, and DNA damage [881]. Thus, humans are likely to be susceptible to tumor induction by PAHs by these mechanisms [881]. Carcinogenic PAHs have been suggested to have an effect on immune function, thereby allowing the induction of carcinogenesis, while noncarcinogenic PAHs do not affect immune function [881].
>
> Humoral immunity was monitored in male iron foundry workers in Poland [881]. Coke oven workers (199) were compared to cold-rolling mill workers (76) [881]. The groups were similar with respect to age, length of employment, and smoking habits [881]. The results showed that coke oven workers, exposed to high concentrations of atmospheric PAHs, including fluoranthene, perylene, pyrene, benzo[a]pyrene, chrysene, benz[a]anthracene, dibenz[a,h]anthracene, and benzo[g,h,i]perylene, had reduced levels of serum immunoglobins [881]. The workers most exposed to PAHs worked at the topside area of the coke ovens [881]. Benzo[a]pyrene exposure was used as a reference point [881]. Coke oven workers, exposed to 0.0002-0.50 mg/m3 benzo[a]pyrene, were compared to cold-

rolling mill workers, whose exposure to benzo[a]pyrene was 3-5 orders of magnitude less [881]. Average length of employment was 15 years [881]. IgG, IgA, IgM, and IgE concentrations were measured [881]. Coke oven workers exhibited a marked depression of mean serum IgG and IgA, compared to mill workers [881]. IgM tended to decrease, whereas IgE tended to increase in the coke oven workers [881]. The biological significance of this finding is unclear and is not addressed by the authors [881].

Benzo(a)pyrene, a lipophilic promutagen, reached maximal concentrations in the thoracic duct lymphatic circulation within 2 hr after gastric instillation. Benzo(a)pyrene in lymph obtained by throacic duct cannulation decreased to approximately control levels within 4 hr after treatment. When lymph was not allowed to enter the blood vascular circulation, serum levels of benzo(a)pyrene increased very slowly, suggesting minimal mesenteric blood vascular absorption of the lipophilic hydrocarbon. Benzo(a)pyrene partitions into lymph lipoproteins as a function of the lipoprotein concentration. Data suggest that low-density lipoproteins may take up benzo(a)pyrene more efficiently than do very low-density or high-density lipoproteins, and that lymph components other than lipoproteins do not take up and transport benzo(a)pyrene. The authors propose that lipophilic xenobiotic compounds interact with cells of the immune system via lymphatic lipoprotein transport of potentially mutagenic, carcinogenic, or immunosuppressive agents. [Busbee DL et al; J Toxicol Environ Health 13 (1): 43-51 (1984)].

For additional details on immunological effects of PAHs in general, see ATSDR [881].

Br.Car: Brief Summary of Carcinogenicity/Cancer Information:

Several of the PAHs, including benz[a]anthracene, benzo[a]pyrene, benzo[b]fluoranthene, benzo[j]fluoranthene, benzo[k]fluoranthene, chrysene, dibenz[a,h]anthracene, and indeno[1,2,3-c,d]pyrene, have caused tumors in laboratory animals when they breathed these substances in the air, when they ate them, or when they had long periods of skin contact with them [881].

EPA (IRIS) 1996 [893]:

Evidence for classification as to human

carcinogenicity; weight-of-evidence classification

Classification: B2; probable human carcinogen [893].

HUMAN CARCINOGENICITY DATA: Inadequate. Lung cancer has been shown to be induced in humans by various mixtures of polycyclic aromatic hydrocarbons known to contain BAP including cigarette smoke, roofing tar and coke oven emissions. It is not possible, however, to conclude from this information that BAP is the responsible agent.

Human data specifically linking benzo(a)pyrene (BAP) to a carcinogenic effect are lacking. There are, however, multiple animal studies in many species demonstrating BAP to be carcinogenic following administration by numerous routes. BAP has produced positive results in numerous genotoxicity assays.

ANIMAL CARCINOGENICITY DATA: Sufficient [893].

The International Agency for Research on Cancer (IARC) and the EPA have determined that benzo(a)pyrene is probably carcinogenic to humans [788]. The Department of Health and Human Services (DHHS) has determined that benzo(a)pyrene may reasonably be anticipated to be a carcinogen [788].

IARC Summary and Evaluation: No data are available in humans. Sufficient evidence of carcinogenicity in animals. OVERALL EVALUATION: Group 2A: The agent is probably carcinogenic to humans [366].

The heavier PAHs (such as benzo(a)pyrene) are such potent carcinogens that they have been known to produce tumors in test animals from single exposures to very small quantities [40].

PAHs express their carcinogenic activity through biotransformation to chemically reactive intermediates that then covalently bind to cellular macromolecules (i.e., DNA) leading to mutation and tumor initiation [881]. The products of PAH metabolism include epoxide intermediates, dihyhdrodiols, phenols, quinones, and their various combinations [881]. The bay region (e.g., the sterically hindered, cup-shaped area between carbons 10 and 11 of benzo[a]pyrene or 1 and 12 of benz[a]anthracene) diol epoxide intermediates of PAHs are considered to be the ultimate carcinogen for alternant PAHs [881]. These diol epoxides are easily converted into carbonium ions (carbocations) which are alkylating agents

and thus mutagens and initiators of carcinogenesis [881]. Therefore, the carcinogenic and toxic potential of PAHs relies on their metabolites [881].

For benzo(a)pyrene there is experimental evidence of mammary carcinogenesis [571].

Drinking water concentrations even lower than a concentration (which was formerly) commonly used as a detection limit (10 ug/L) may result in an unacceptable human cancer risk [209].

B(a)P is used extensively as a positive control in a variety of /laboratory mutagenicity & carcinogenicity/ short-term tests. [366, IARC. Monographs on the Evaluation of the Carcinogenic Risk of Chemicals to Man. Geneva: World Health Organization, International Agency for Research on Cancer, 1972-1985. (Multivolume work).,p. V32 215 (1983)].

This is a very phototoxic PAH [891,887; Reprinted with permission from Environmental Toxicology and Chemistry, Volume 6, Newstead, J.L. and J.P. Geisy. Predictive models for photoinduced acute toxicity of polycyclic aromatic hydrocarbons to Daphnia magna. Copyright 1987. SETAC]. Although not definitive, phototoxicity represents one clue suggesting possible carcinogenicity.

Relative (equivalency factor) oral carcinogenic potency value compared to benzo(a)pyrene (BAP, which is ranked 1.0): 1.0; the only other PAH ranked 1.0 besides BAP is Dibenz(a,h)anthracene [EPA, 1993, Provisional Guidance for Quantitative Risk Assessment of Polycyclic Aromatic Hydrocarbons]. Although the information is based on mouse skin painting studies, until better guidance is available, the relative oral carcinogenic potency value may be used in superfund site human risk assessments in conjunction with the oral carcinogenic slope factor for benzo(a)pyrene found in EPA's IRIS database [893] (Stan Smucker, EPA Region 9, personal communication, 1996).

Amphibians are reported to be quite resistant to PAH carcinogenicity due to their inability to produce mutagenic metabolites of benzo(a)pyrene and perylene [957]. However, PAHs usually occur in the company of other PAHs and the surface eggs and larvae of amphibians, especially those at high altitude, may be prone to acute impacts from phototoxic properties of mixtures of PAHs (Roy Irwin, National Park Service, personal communication, 1996).

Br.Dev: Brief Summary of Developmental, Reproductive, Endocrine, and Genotoxicity Information:

Mice fed high levels of benzo[a]pyrene during pregnancy had difficulty reproducing and so did their offspring [881]. The offspring of pregnant mice fed benzo[a]pyrene also showed other harmful effects, such as birth defects and decreased body weight [881]. Similar effects could occur in people, but we have no information to show that these effects do occur [881].

B(a)P is embryotoxic & teratogenic in mice; the inducibility of aryl hydrocarbon hydroxylase activity in dams & fetuses is an important factor in determining these effects. A reduction in fertility in ... male & female offspring was observed in mice following exposure ... in utero. B(a)P undergoes metabolism to reactive electrophiles capable of binding covalently to DNA. It was active in assays for bacterial DNA repair, bacteriophage induction & bacterial mutation; mutation in Drosophila melanogaster; DNA binding, DNA repair, sister chromatid exchange, chromosomal aberrations, point mutation & transformation in mammalian cells in culture; & in tests in mammals in vivo, including DNA binding, sister chromatid exchange, chromosomal aberration, sperm abnormality & the somatic specific locus (spot) test. There is sufficient evidence that benzo(a)pyrene is active in short-term tests. [IARC. Monographs on the Evaluation of the Carcinogenic Risk of Chemicals to Man. Geneva: World Health Organization, International Agency for Research on Cancer, 1972-1985. (Multivolume work).,p. V32 216 (1983)] [366].

Adverse effects such as decreased fertility and total sterility in mice and decreased incidence of pregnant female rats at parturition were reported following oral exposure to benzo(a)pyrene [788].

Benzo(a)pyrene may be a xenoestrogen since there is experimental evidence of its association with mammary carcinogenesis (and breast cancer risk factors can be linked to total lifetime exposure to bioavailable estrogens) [571].

Some combustion pollutants (certain PAHs) have made it onto lists of potential endocrine disrupters [514,569,575]. One study showed an association of coke oven emissions with excess prostrate cancer mortality, but it is unclear the role which PAHs and endocrine disruption may be playing, if any [1025]. A 1997 EPA issue paper on endocrine disruption made relatively little mention of PAHs, but did mention that much more study is needed to clarify endocrine disruption issues in general [1025].

Benzo(a)pyrene was found to be a principal mutagenic compound when exposed to a bacteria [816; Reprinted with

permission from Environmental Toxicology and Chemistry, Volume 14, Marvin, C.H., J.A. Lundrigan, B.E. McCarry and D.W. Bryant. "Determination and genotoxicity of high molecular mass polycyclic aromatic hydrocarbons isolated from coal-tar-contaminated sediment." Copyright 1995 SETAC].

Of six polycyclic aromatic hydrocarbons found to have growth-promoting effects on plants, B(a)P was most potent. It was further found that the degree of the promoting effect corresponded to the oncogenic activity of the hydrocarbon [366].

Benzo(a)pyrene also has growth-promoting effects on some planktonic algae [366].

Br.Fate: Brief Summary of Key Bioconcentration, Fate, Transport, Persistence, Pathway, and Chemical/Physical Information:

The breakdown and biotransformation of BAP in living things is quite complex, involving glucuronidation and the cytochrome P450 biotransformation enzymes, and various other pathways [982].

The heavier (4-, 5-, and 6-ring) PAHs are more persistent than the lighter (2- and 3-ring) PAHs [796].

B(a)P is highly lipophilic and can bioconcentrate to high levels in some aquatic organisms. It bioaccumulated in lower organisms such as snails, but it can be degraded by higher organisms [754].

Environmental Fate/Exposure Summary [366]:

> Benzo(a)pyrene's (BaP) release to the environment is quite wide spread since it is an ubiquitous product of incomplete combustion. It is largely associated with particulate matter, soils, and sediments. Although environmental concentrations are highest near sources, its presence in places distant from primary sources indicates that it is reasonably stable in the atmosphere and capable of long distance transport. When released to air it may be subject to direct photolysis, although adsorption to particulates apparently can retard this process. It may also be removed by reaction with O3 (half-life 37 min) and NO2 (half-life 7 days), and an estimated half-life for reaction with photochemically produced hydroxyl radicals is 21.49 hr. If released to water, it will adsorb very strongly to sediments and particulate matter, bioconcentrate in aquatic organisms which can not

metabolize it, but will not hydrolyze. It may be subject to significant biodegradation, and direct photolysis may be important near the surface of waters; adsorption, however, may significantly retard these two processes. Evaporation may be important with a half-life of 43 days predicted for evaporation from a river 1 m deep, flowing at 1 m/sec with a wind velocity of 3 m/sec; adsorption to sediments and particulates will limit evaporation. If released to soil it will be expected to adsorb very strongly to the soil and will not be expected to appreciably leach to the groundwater, although its presence in some samples of groundwater illustrates that it can be transported there. It will not be expected to hydrolyze or significantly evaporate from soils and surfaces. It may be subject to appreciable biodegradation in soils. Human exposure will be from inhalation of contaminated air and consumption of contaminated food and water. Especially high exposure will occur through the smoking of cigarettes and the ingestion of certain foods (eg smoked and charcoal broiled meats and fish). (SRC)

Clams and mussels have been used to measure body burdens of B(a)P and other PAHs in aquatic organisms, since fish break down the compound faster.

Synonyms/Substance Identification:

Benz(a)pyrene [366]
3,4-Benz(a)pyrene [366]
3,4-Benzopyrene [870]
3,4-Benzypyrene [870]
3,4-BP [870]
BP [366,870]
BaP [366]
B(a)P [870]
Benzo(d,e,f)chrysene [870]
1,2-Benzpyrene [870]
1,2-Benzopyrene [870]
6,7-Benzopyrene [870]
RCRA Waste Number U022 [870]

Molecular Formula [366]:
C20-H12

Associated Chemicals or Topics (Includes Transformation Products):

See also entries for:

PAHs (Polycyclic Aromatic Hydrocarbons) as a group

Related But Different Compounds [366]: Benzo(e)pyrene

Benzo(a)pyrene is metabolized to approximately 20 primary and secondary oxidized metabolites and to a variety of conjugates (see HSDB [366] information immediately below). Several metabolites can induce mutations, transform cells and/or bind to cellular macromolecules; however only a 7,8-diol-9, 10-epoxide is presently considered to be an ultimate carcinogenic metabolite [847].

Metabolites/Metabolism/Breakdown Products [366]:

The predominant metabolites of benzo(a)pyrene in mammals are 3- & 9-hydroxybenzo(a)pyrene; benzo(a)pyrene-1,6-quinone & benzo(a)pyrene- 3,6-quinone; & benzo(a)pyrene-4,5-dihydrodiol, benzo(a)pyrene-7,8-dihydrodiol & benzo(a)pyrene-9,10-dihydrodiol.

Several metabolites /of B(a)P/ can induce mutations, transform cells &/or bind to cellular macromolecules; however, only a 7,8-diol-9,10-epoxide is presently considered to be an ultimate carcinogenic metabolite. [IARC. Monographs on the Evaluation of the Carcinogenic Risk of Chemicals to Man. Geneva: World Health Organization, International Agency for Research on Cancer, 1972-1985. (Multivolume work).,p. V32 215 (1983)].

Fish exposed to benzo(a)pyrene (b(a)p) & naphthalene in sediment containing prudhoe bay crude oil. B(a)P metab to greater extent. Naphthalene in sediments was metabolized to 1,2-dihydro-1,2-dihydroxynaphthalene glucuronide. [Varanasi et al; Aquat Toxicol 1 (1): 49 (1981)].

Water Data Interpretation, Concentrations and Toxicity (All Water Data Subsections Start with "W."):

W.Low (Water Concentrations Considered Low):

No information found.

W.High (Water Concentrations Considered High):

Average benzo(a)pyrene concentration in the groundwater near five U.S. wood treatment facilities was 57 ppb (0.057 mg/L) [788].

Individual PAHs in the groundwater from 5 U.S. wood treatment facilities were reported at average concentrations of 57 ppb (0.057 mg/L) for benzo[a]pyrene to 1,825 ppb (1.8 mg/L) for phenanthrene [881].

W.Typical (Water Concentrations Considered Typical):

B(a)P concentrations found in the environment are: tap water, 0.2-1000 ng/L; rain water, 2.2-7.3 ng/L; subterranean water, 0.4-7 ng/L [847].

The B(a)P concentration in surface waters of the Atlantic region of Canada is 0.003 ug/L (based on 61 samples taken 1980-81) [754].

Information from ATSDR on PAHs in water (for information on embedded references, see ATSDR) [881]:

> Mean concentrations of benzo[a]pyrene in the Great Lakes have been detected at levels between 0.03 and 0.7 ppt (ng/L) (Environment Canada 1991) [881].
>
> DeLeon et al. (1986) analyzed surface water from 11 locations in the Mississippi River. Seventeen PAHs were identified in the samples at levels ranging from 1 ng/L for 6 compounds to a high of 34 ng/L for phenanthrene. The highest concentration of phenanthrene was detected in a sample collected near New Orleans, Louisiana, near an industrial area, implicating industrial effluent or surface runoff from this area as a possible source [881].

Water Concentrations [366]:

> DRINKING WATER: 15 USA cities, 87% pos, 0.1-2.1 parts per trillion, avg 0.55 parts per trillion(1). USA, 0.20- parts per trillion(3); USA: 6 cities, finished and distribution water, <1 parts per trillion; 11 cities, 0.2-1.6 parts per trillion; 8 cities, <1-1 parts per trillion(4). Treated surface waters used as drinking waters: River Rhine, 0.5 parts per trillion, Lake Constance, 1.7, English River, 9 parts per trillion (1). West Germany, 1968, 6 samples, 100% pos, 0.5-4.0 parts per trillion(2). The Netherland, avg 2 parts per trillion, max 15 parts per trillion(3). UK, 1974-75 7 distributed treated surface water systems, Trace-<3 parts per trillion; 8 groundwater distribution systems, 1974-77, trace-<10 ppt, max during repair work on parts of 1 system, 101 parts per trillion(5). Norway, 4 samples, <0.05-0.29 parts per trillion(6). [(1) Santodonato J et al; Health and Economic Assessment of Polynuclear Aromati Hydrocarbons. Lee SD, Grant L, eds Park Forest South IL: Pathotox Pub Inc. p 119-22 (1981) (2) Verschueren K; Handbook of environmental data on organic chemicals. 2nd ed Von Nostrand Reinhold NY p.272 (1983) (3) Kraybill HF; NY Aca Sci Ann 298: 80-9 (1977) (4) Sorrell RK et al; Environ Inter 4: 245-54 (1980) (5) Crane RI et al; A Survey of

Polycyclic Aromatic Hydrocarbon Levels in British Waters TR-158 Medmenham, UK: Water Research Centre p 47 (1981) (6) Kveseth K et al; Chemosphere 11: 623-39 (1982)].

GROUNDWATER: Elkhart, IN, 4 parts per trillion, Fairborn, OH, 0.3 parts per trillion, Champaign, IL, not detected, unspecified sites in West Germany, 0.4 parts per trillion(1). West Germany, 1968, 10 samples, 0.4-3.8 parts per trillion(2). The Netherlands, 232 groundwaer pumping stations, max concn 1 ppb(3). Germany, 1963-64, 3 sites, 0.1-23.4 parts per trillion(4). [(1) Santodonato J et al; Health and Economic Assessment of Polynuclear Aromatic Hydrocarbons. Lee SD, Grant L, eds Park Forest South, IL Pathotox Pub Inc. p.119 (1981) (2) Verschueren K; Handbook of Environmental Data on Organic Chemicals. 2nd ed Von Nostrand Reinhold NY p 272 (1983) (3) Zoeteman BCJ et al; Sci Total Environ 21: 187-202 (1981) (4) Sorrell RK et al; Environ Internat 4: 245-54 (1980)].

SURFACE WATER: US STORET Database, 914 water stations, pos, 0.6-80 parts per trillion, avg 23 parts per trillion(1); USSR, 2 rivers, 17 and 10 parts per trillion(1); Thames River, UK, 3 stations, 0.13-0.35 ug/l, avg 0.21 ug/l(1). Germany, 1963-64, 4 rivers, 6 sites, 0.6-114.0 parts per trillion; USSR, 5 sites < 0.1-13,000 parts per trillion (max Moscow Reservoirs); UK: Severn River, 5 sites, 1.5-13.5 parts per trillion, Thames River, 2 sites, 130-210 parts per trillion; River Trent and tributaries, 11 sites, in solution, 0.1-1.8 parts per trillion, in suspended solids, 0.8-504.0 parts per trillion (max River Trent at Keadby)(3. UK, 1973-76, 9 rivers, 25 sites, 51 samples, 3.8-531 parts per trillion(4). [(1) Santodonato J et al; Health and Economic Assessment of Polynuclear Aromatic Hydrocarbons. Lee SD, Grant L, eds Park Forest South, IL: Pathotox Pub Inc. p 119-20 (1981) (2) Staples CA et al; Environ Toxicol Chem 4: 131-42 (1985) (3) Sorrell RK et al; Environ Inter 4: 245-54 (1980) (4) Crane RI et al; A Survey of Polycyclic Aromatic Hydrocarbon Levels in British Waters TR-158 Medmenham, UK: Water Research Centre 47 pp (1981)].

RAINWATER: 2.2-7.3 ppt(1). The Netherland, 3 rainfall events, Dec 10, 1982: 3 samples taken 5, 12, and 23 min after onset of rain: 390, 9, 6 parts per trillion; same date, 4 samples taken 8, 18, 27, and 38 min after onset of second rain event: 75, 25, 18, 0 (not detected) parts per trillion; Sept, 1983, 8 samples taken 12-25 min after onset of

rain: 10-37 parts per trillion(2). [(1) IARC; Polynuclear aromatic compounds, Part 1. Chemical, environmental and experimental data. 32: 432-45 (1983) (2) VanNoort PCM, Wondergem; Environ Sci Technol 19: 1044-8 (1985)].

Effluent Concentrations [366]:

US STORET Database, 1253 water stations, 2.3% pos, median <10 ppb(1). Sludge from 12 UK sewage treatment plants, 16-400 ppb (dry wt), 0.35-11.43 ppm (dry wt)(2). Bekkelaget Sewage Treatment Plant, Oslo, Norway, <3-5 parts per trillion(3). Those industries with mean raw or treated wastewater concn exceeding 100 ppb includes (max raw wastewater concn, ppb): coal mining (140), iron and steel manufacturing (14,000), nonferrous metals manufacturing (570), timber products processing (2,700)(4). Estimates of total emissions >100 metric tons/yr: coal-fired residential furnaces, coke production, forest fires, burning coal refuse banks; estimates >10 metric tons/yr: coal-fired industrial boilers, residential fireplaces, iron and steel sintering, commercial incinerators, open burning of auto components and leaves, trucks and automobiles, and tire wear(5). Identified, not quantified in emissions from biomass gasifier(6). Estimated emissions from mobile sources, 1979, 43 metric tons; estimated total annual emission, 1975, 346-1,676 (intermediate 588) metric tons, 1985, 67-885 (intermediate 358) metric tons(7). Industrial effluent wastewaters: by-products, 12-16 ppb, oil refineries, 0.05-3.6 ppb; domestic effluent, 0.038-0.074, final effluent of sewage work, 0.03 ppb(8). National Urban Runoff Program, 15 cities, 13% pos, 86 samples, 4% pos, 1-10 ppb(9). [(1) Staples CA et al; Environ Toxicol Chem 4: 131-42 (1985) (2) McIntyre AE et al; Anal Letters 14: 291-309 (1981) (3) Kveseth K et al; Chemosphere 11: 623-39 (1982) (4) USEPA; Treatability Manual; pp.1.10.5-1 to 1.10.5-4 USEPA-600/2-82-001A (1981) (5) Santodonato J et al; Health and Ecological Assessment of Polynuclear Aromatic Hydrocarbons. Lee SD, Grant L, eds Park Forest South, IL: Pathotox Pub Inc. p 131-36 (1981) (6) Desilets DJ et al; Environ Sci Technol 18: 386-91 (1984) (7) National Research Council; Polycyclic Aromatic Hydrocarbons: Evaluation of Sources and Effects. National Acad Press Washington, DC (1983) (8) Verschueren K; Handbook of Environmental Data on Organic Chemicals. 2nd ed Von Nostrand Reinhold NY p 272 (1983) (9) Cole RH et al; J Water Pollut Control Fed 56: 898-908 (1984)].

W.Concern Levels, Water Quality Criteria, LC50 Values, Water Quality Standards, Screening Levels, Dose/Response Data, and Other Water Benchmarks:

W.General (General Water Quality Standards, Criteria, and Benchmarks Related to Protection of Aquatic Biota in General; Includes Water Concentrations Versus Mixed or General Aquatic Biota):

Cautionary note: This is a very phototoxic compound (see more detailed discussion the entry on PAHs as a group). Therefore, any of the water criteria which have been developed for it using bioassays performed in the absence of UV light may be under-protective. Phototoxicity of certain PAHs was discovered when organisms which had survived lab exposures to PAHs died quickly after being moved into sunlight. An increase in toxicity due to photo-induced changes is called phototoxicity. For certain PAHs, tests performed in the presence of UV or other solar radiation show greatly increased toxicity to those same organisms at PAH concentrations below maximum solubility [888,889,911,887; Reprinted with permission from Environmental Toxicology and Chemistry, Volume 6, Newstead, J.L. and J.P. Geisy. Predictive models for photoinduced acute toxicity of polycyclic aromatic hydrocarbons to Daphnia magna. Copyright 1987. SETAC]. The reader should be aware that the authors of this document have not yet been able to determine which of the following criteria and benchmarks were developed in the presence or absence of UV light:

National EPA Water Quality Criteria in ug/L :

Freshwater Acute Criteria: None Published [446,893].

Freshwater Chronic Criteria: None Published [446,893].

Marine Acute Criteria: 3.0E+2 ug/L is the lowest effect concentration found in the literature (LEC) [893].

Marine Chronic Criteria: None Published [446].

NOTE: Before citing a concentration as EPA's water quality criteria, it is prudent to make sure you have the latest one. Work on the replacement for the Gold Book [302] was

underway in March of 1996, and IRIS is updated monthly [893].

Oak Ridge National Lab, 1994: Ecological Risk Assessment Freshwater Screening Benchmarks for concentrations of contaminants in water [649]. For a definition of meaning of each benchmark, see entry entitled: Benchmarks, Ecological Risk Assessment Screening Benchmarks. To be considered unlikely to represent an ecological risk, field concentrations should be below all of the following benchmarks [649]:

NOTE: Although most of the lab tests done to develop water quality criteria and other benchmarks were originally based on "total" values rather than "dissolved" values, the lab settings were typically fairly clean and the numbers generated by the lab tests are therefore often even more comparable to field "dissolved" values than to field "total" values (Glen Suter, Oak Ridge National Lab, Personal Communication, 1995). For more information on dissolved vs. total concentrations, and EPA suggested conversion fractions for metals, see entry entitled "Dissolved."

Benzo(a)pyrene (ug/L):

0.24 = Secondary Acute Value
0.014 = Secondary Chronic Value
0.30 = Estimated Lowest Chronic Value - Daphnids
> 2.99 = Lowest test EC20 - Fish

Canada's Interim Assessment Criterion for benzo(a)pyrene in water is 0.01 ug/L [656].

NOTE: a) For most of the organic chemical parameters in [656], criteria are based on analytical detection limits; b) criterion is considered "Interim" since complete supporting rationale do not exist.

The IJC (1983) recommended that the concentration of benzo(a)pyrene in water should be less than 0.01 ug/L (based on the WHO limit for drinking water). They also noted that other 3- to 5-ring PAHs are carcinogenic and may be of equal or greater concern [754].

As of July 1992, the proposed Ambient Water Quality Standard for New York state is 0.200 ug/L [859].

W.Plants (Water Concentrations vs. Plants):

No information found.

W.Invertebrates (Water Concentrations vs. Invertebrates):

See also [649] information in W.General section above.

LC50 for Daphnia pulex (water flea) was 5 ug/L (ppb) (0.005 mg/L, ppm) for a 96-hr exposure [998].

LC50 for Neanthes arenaceodentata (polychaete) was <1000 ug/L (1.0 mg/L) for a 96-hr exposure [998].

W.Fish (Water Concentrations vs. Fish):

See also [649] information in W.General section above.

Histological and skeletal examinations were performed on rainbow trout alevins reared in 0.00, 0.08, 0.21, 0.39, 1.48, 2.40, or 2.99 ng/ml aqueous benzo(a)pyrene (BaP). Nuclear pycnosis and karyorrhexis were most common in neuroectodermal and mesodermal derivatives and in liver of B(a)P-treated alevins. Microphthalmia was noted in 17% of the test fish and was frequently associated with a patent optic fissure. Depressed mitotic rates in the retina and brain, but not liver, were seen in alevins reared in 0.21 to 1.48 ng/ml aqueous B(a)P. Test alevins had a significantly higher incidence of skeletal malformations in the skull and vertebral column and abnormalities of vertebral arcualia often corresponded to areas of kyphoscoliotic flexures. The ecological significance of such morphological abnormalities would be decreased feeding and growth and inability to escape predation, leading to reduced survival. Persistent mixed function oxygenase induction in less affected larvae would lead to continuing production of cytotoxic, mutagenic, and carcinogenic B(a)P metabolites resulting in anemia, impaired ability to respond to environmental stress and disease, and possibly latent tumorigenesis. [Hose JE et al; Arch Environ Contam Toxicol 13 (6): 675-84 (1984)] [366].

W.Wildlife (Water Concentrations vs. Wildlife or Domestic Animals):

Oak Ridge National Lab, 1994: Risk Assessment Screening Benchmarks for Wildlife derived for No-

Observed-Adverse-Effect (NOAEL) levels. To be considered unlikely to represent an ecological risk, water concentrations should be below the following benchmarks for each species present at the site [650]:

SPECIES	WATER CONC (ppm)
Mouse (test species)	0.00000
Short-tailed Shrew	5.71400
Little Brown Bat	9.87600
White-footed Mouse	3.69300
Meadow Vole	6.46300
Cottontail Rabbit	3.06200
Mink	3.17600
Red Fox	2.26600
Whitetail Deer	1.26800

W.Human (Drinking Water and Other Human Concern Levels):

EPA 1996 IRIS database information [893]:

IRIS 1996 Ambient Water Quality Criteria for Human Health Considering Water & Fish routes of exposure: 2.8E-3 ug/liter [893].

Older Reference to same value: Human Health (10-6 = E-06) Risk Level for Carcinogens: Published Criteria for Water and Organisms: 0.0028 [689,928].

IRIS 1996 Ambient Water Quality Criteria for Human Health Considering fish route only: 3.11E-2 ug/liter [893].

Older reference to same value: Published Criteria for Organisms: 0.0311 [689,928]. Older Discussion of Same Values as Water Standards for Humans [366]:

For the maximum protection of human health from the potential carcinogenic effects due to exposure of polynuclear aromatic hydrocarbons through ingestion of contaminated water and contaminated aquatic organisms, ... therefore, the levels which may result in incremental increase of cancer risk over the lifetime are estimated at 1x10-5, 1x10-6, and 1x10-7. The

corresponding criteria /for ambient water/ are 28.0 ng/l, 2.8 ng/l, and 0.28 ng/l, respectively. If the above estimates are made for consumption of aquatic organisms only, excluding consumption of water, the levels are 344.0 ng/l, a 31.1 ng/l, and 3.11 ng/l respectively. /Polynuclear aromatic hydrocarbons based on benzo(a)pyrene as the model PAH/ [USEPA; Ambient Water Quality Criteria Doc: Polynuclear Aromatic Hydrocarbons p.C-121 (1980)].

Reference: 45 FR 79318 (11/28/80).

Discussion: For the maximum protection from the potential carcinogenic properties of this chemical, the ambient water concentration should be zero. However, zero may not be obtainable at this time, so the recommended criteria represents a E-6 estimated incremental increase of cancer over a lifetime. The values given represent polynuclear aromatic hydrocarbons as a class [893].

Unit Risk: 2.1E-4 per ug/liter [893]. The unit risk is the quantitative estimate in terms of either risk per ug/L drinking water. Drinking Water Concentrations at Specified Risk Levels: Risk Level Concentration E-4 (1 in 10,000) 5E-1 ug/liter E-5 (1 in 100,000) 5E-2 ug/liter E-6 (1 in 1,000,000) 5E-3 ug/liter [893].

Drinking Water Health Advisories: empty [893].

MCLG: As of July 1992, the U.S. EPA Maximum Contaminant Level Health Goal (MCLG) for B(a)P in drinking water is 0 mg/L [893].

MCL: The Maximum Contaminant Level (MCL) is 0.0002 mg/L [859,893,952]. Older reference: Drinking Water MCL [653]: 2 ug/L.

NOTE: The attempt to develop a drinking water criterion for polynuclear aromatic hydrocarbons (PAH) as a class is hindered by several gaps in the scientific data base: (1) The PAH class is composed of numerous compounds having diverse biological effects and varying

carcinogenic potential. A "representative" PAH mixture, has not been defined. (2) The common practice of using data derived from studies with benzo(a)pyrene to make generalizations concerning the effects of environmental PAH may not be scientifically sound. (3) No chronic animal toxicity studies involving oral exposure to PAH mixtures exist. (4) No direct human data concerning the effects of exposure to defined PAH mixtures exist. /Polynuclear aromatic hydrocarbons/ [USEPA; Ambient Water Quality Criteria Doc: Polynuclear Aromatic Hydrocarbons] [366].

Reference: 55 FR 30370 (07/25/90) [893].
Discussion: The proposed MCL is equal to the PQL and is associated with a maximum lifetime individual risk of 1E-4 [893].

EPA Region 9 Preliminary remediation goals (PRGs) for tap water [868]: 9.2E-03 (0.0092) ug/L.

As of July 1992, the Canadian Health and Welfare value and the Ontario Ministry of the Environment value for Maximum Acceptable Concentration (MAC) of B(a)P in drinking water are both 0.010 ug/L [859].

The warm water- and cold water sport fish community human cancer criteria for B(a)P in Wisconsin public water supplies are each 0.023 mg/L [881].

The warm water- and cold water sport fish community human cancer criteria for B(a)P in Wisconsin non-public water supplies are each 0.1 mg/L [881].

Numeric Water Quality Criteria in Arizona [881]:

Domestic water supply: 0.003 ug/L
Fish consumption: 0.002 ug/L
Full body contact: 0.12 ug/L

Criteria for human health protection in Missouri [881]:

Fish consumption: 0.03 ug/L
Drinking water supply: 0.003 ug/L
Groundwater: 0.003 ug/L

The groundwater standard for B(a)P in New Mexico is 0.0007 mg/L [881].

Public Health Groundwater Quality Standards in

Wisconsin [881]:

Enforcement standard: 0.003 ug/L
Preventative action: 0.0003 ug/L

As of July 1992, the World Health Organization (WHO) Guideline Value for B(a)P in drinking water is 0.010 ug/L [859].

NOTE: Value is based upon a maximum acceptable risk of 1 additional case of cancer per 100,000 population (or 1x10(-5)) [859].

W.Misc. (Other Non-concentration Water Information):

This is a very phototoxic PAH [891,887; Reprinted with permission from Environmental Toxicology and Chemistry, Volume 6, Newstead, J.L. and J.P. Geisy. Predictive models for photoinduced acute toxicity of polycyclic aromatic hydrocarbons to Daphnia magna. Copyright 1987. SETAC]. See cautionary note under W.General section above.

A review of groundwater monitoring data from 479 waste disposal sites (178 CERCLA or Superfund sites, 173 RCRA sites, and 128 sanitary/municipal landfill sites) located throughout the United States indicated that 14 of the PAHs included in this profile were detected at frequencies ranging from 2 detections at one site in one EPA Region for indeno[1,2,3-c,d]pyrene, to 85 detections at 16 sites in 4 EPA Regions for fluorene (Plumb 1991). Benzo[a]pyrene was detected 13 times at 6 sites in 6 EPA Regions. Concentrations were not reported [881].

PAHs have been detected in surface waters of the United States. In an assessment of STORET data covering the period 1980-82, it was reported that median concentrations in ambient water of less than 10 ug/L for 15 PAHs (acenaphthene, acenaphthylene, anthracene, benz[a]anthracene, benzo[b]fluoranthene, benzo[k]fluoranthene, benzo[g,h,i]perylene, benzo[a]pyrene, chrysene, fluoranthene, fluorene, indeno[1,2,3-c,d]pyrene, naphthalene, phenanthrene, and pyrene). The percentage of samples in which these PAHs were detected ranged from 1.0 (benzo[g,h,i]perylene) to 5.0 (phenanthrene) and 7.0 (naphthalene) [881].

Sediment Data Interpretation, Concentrations and Toxicity (All Sediment Data Subsections Start with "Sed."):

Sed.Low (Sediment Concentrations Considered Low):

No information found.

Sed.High (Sediment Concentrations Considered High):

Analyses of sewage sludges from 50 publicly owned treatment works by the U.S. Environmental Protection Agency (1985): The mean concentration of BaP was 256.6 ppm [347].

Sed.Typical (Sediment Concentrations Considered Typical):

Benzo(a)pyrene was detected in 94.5 % of urban-bay samples from the Puget Sound area. The mean concentration was 1417 ug/kg (ppb) dry weight, while the median concentration was 280 ug/kg (ppb) [852].

Benzo(a)pyrene was detected in 67% of non-urban-bay samples from the Puget Sound area. The mean concentration was 2223 ug/kg (ppb) dry weight, while the median concentration was 77 ug/kg (ppb) [852].

NOTE: The above values are not normalized for total organic carbon (TOC) content. Urban bay concentrations may be lower than non-urban bay concentrations due to more frequent dredging practices in urban bays, and also to the fact that most of the urban bays are at the mouths of rivers which are continually depositing "clean" sediment into these bays.

The control site in one Great Lakes study had a sediment concentration of <0.01 mg/kg [145].

Benzo(a)pyrene levels in bottom sediments of the Great Lakes have been reported to range from 34-490 ppb [788].

B(a)P concentration range in dried sediments from lakes: 1-2380 ug/kg [847].

Concentrations of B(a)P in freshwater sediments range from 0.001 to 17 ug/g dry weight [754].

Benzo[a]pyrene levels in bottom sediments of the Great Lakes have been reported to range from 34 to 490 ppb (ug/kg) (Environment Canada 1991) [881].

Sediment Concentrations [366]:

SEDIMENTS: Marine and estuarine, 8 sites, 0-3030 ppb (dry wt)(max black mud from Bay of Naples)(8). Marine sediments: Bay of Naples, 6 samples, 3 sites, conc-dry weight (depth), 1.4-530 ppb (2-120 m), 2 sites highly polluted, 100-3000 ppb (15-45 m), 1 site in vicinity of volcanic pollution, 260-960 ppb (55 m)(1); French Mediterranean coast: 0-2

m depth, 16-5000 ppb; 0-0.03 m, 1800 ppb; 0.08-0.13 m, 5000 ppb; 2 m, 16 ppb; sand, 0-5 m, 15-34 ppb, mud, 102 m, not detected (nd)(1). French Channel and Atlantic coast, 11 sites, mud and sand, nd-1700 ppb(1). Greenland, west coast, sand 0.2 m, 5 ppb(1). Wilderness Lake, Ontario, 1976, 13 ppb (dry weight); West Germany, water solids, four rivers, 0.1-2.0 ppm (2). Niagara River at Niagara-on-the-Lake, 1975-82, suspended sediments, 190-20,000 ppb(3). Columbia River, WA, 1979, suspended particulates, 10 samples, 5-97 ppb (min June, max Jan)(4). Duwamish River delta, Seattle, WA/Puget Sound, WA, 1.16 pm (wet wt)(5). 6 Western US lakes, <2-305 ppb (dry wt)(max Los Angeles reservoir, 27-29 cm depth)(6). [(1) Santodonato J et al; Health and Economic Assessment of Polynuclear Aromatic Hydrocarbons. Lee SD, Grant L, eds Park Forest South, IL Pathotox Pub Inc. p 131-6 (1981) (2) Verschueren K; Handbook of Environmental p 273 (1983) (3) Kuntz KW; Toxic Contaminants in the Niagara River, 1975-1982. Technical Bulletin No. 134 Burlington, Ontario (1984) (4) Prahl FG et al; Environ Sci Technol 18: 687-93 (1984) (5) Varanasi U et al; Environ Sci Technol 19: 836-41 (1985) (6) Heit M; pp.89-103 in Environmental Measurement Laboratory-Environmental Quarterly, Hardy EP Jr, ed (1978) (7) Edwards NT; J Total Environ Qual 12: 427-41 (1983) (8) Sims RC, Overcash MR; Res Rev 88: 1-68 (1983)].

Sed.Concern Levels, Sediment Quality Criteria, LC50 Values, Sediment Quality Standards, Screening Levels, Dose/Response Data and Other Sediment Benchmarks:

Sed.General (General Sediment Quality Standards, Criteria, and Benchmarks Related to Protection of Aquatic Biota in General; Includes Sediment Concentrations Versus Mixed or General Aquatic Biota):

Oak Ridge National Lab, 1994: Risk Assessment Screening Benchmarks for Sediment Concentrations. To be considered unlikely to represent an ecological risk, field concentrations should be below all of the following benchmarks in mg/kg (ppm) dry weight [652]:

Benzo(A)Pyrene (Values in mg/kg dry wt.): 0.14 is the Estimated equivalent sediment quality criterion at 1% Organic Carbon.

Various Other Concern Levels for Sediment Concentrations (Dry Weight):

EPA, 1988: The interim sediment criteria value proposed by EPA was 11.0 mg/kg [145].

AET 1988: The apparent effects threshold concentrations for toxic effects for Benzo(a)pyrene in sediments proposed for Puget Sound ranged from 1.6 mg/kg dry weight (microtox) to 3.6 mg/kg dry weight (benthic) [416]. Although the authors of the Puget Sound AETs have cautioned that Puget Sound AETs may not be appropriate for comparison with data from other geographic areas, so few (toxic) concern levels for this chemical have been published that the proposed Puget Sound concern level is included in this text as a reference item.

NOTE: Much lower concentrations of this PAH are of concern related to their contribution to "total and especially total carcinogenic PAH" sums (see "PAHs as a group" entry).

NOAA 1995: After studying its own data from the National Status and Trends Program as well as many literature references concerning different approaches to determining sediment criteria, NOAA suggested that the potential for biological effects of this contaminant sorbed to sediments was highest in sediments where its concentration exceeded the 1600 ppb dry weight Effects Range-Median (ERM) concentration and was lowest in sediments where its concentration was less than the 430 ppb dry weight Effects Range-Low (ERL) concentration [664]. To improve the original 1990 guidelines [233], the 1995 report included percent (ratios) incidence of effects for ranges below, above, and between the ERL and ERM values. These numbers represent the number of data entries within each concentration range in which biological effects were observed divided by the total number of entries within each range [664]. (For more information see entries entitled "ERM" and "ERL"):

<ERL	10.3
ERL-ERM	45.0
>ERM	88.5

The Canadian AET concentration (adapted from NOAA 1990) for B(a)P sorbed to marine sediments is 0.700 mg/kg dry weight [864]. An AET is defined as the

lowest concentration of a compound in sediment at which biological effects (usually changes in composition of benthic invertebrate communities) are observed to occur [864].

Ontario Ministry of the Environment Freshwater Sediment Guidelines, 1993. Lowest effect level: 370 ug/kg dry weight. Severe effect level: 1,440 mg/kg organic carbon [761].

St. Lawrence River Interim Freshwater Sediment Criteria, 1992. No effect: 10-100 ug/kg dry weight. Minimal effect: 500 ug/kg dry weight. Toxic effect: 70 mg/kg organic carbon [761].

Environment Canada Interim Sediment Quality Assessment Values. Toxic effect level: 31.9 ug/kg dry weight. Probable effect level: 782.0 ug/kg dry weight [761].

Sed.Plants (Sediment Concentrations vs. Plants):

No information found.

Sed.Invertebrates (Sediment Concentrations vs. Invertebrates):

No information found.

Sed.Fish (Sediment Concentrations vs. Fish):

The IJC (1983) recommended that the concentration of benzo(a)pyrene in sediments or in organisms serving as a food source for fish should not exceed 1.0 ug/g. They also noted that other 3- to 5-ring PAHs are carcinogenic and may be of equal or greater concern [754].

Collier and Varanasi exposed English sole to sediment extracts containing 0.0104 mg of PAHs (sum of 22 PAHs) per gram sediment and to B(a)P (fish weighing 69 g at 13 degrees C). A fourfold induction of AHH enzymes was observed in liver, with a B(a)P dose of 0.1 mg/kg and with 0.01 mg/kg of PAHs in sediments. In our study, a threefold induction of EROD enzymes was observed in liver, when fish were exposed to a concentration of total unsaturated compounds between 0.2 to 1.7 g/kg (150 ul of used oil). When PACs are expressed as a sum of 26 components, this concentration translates into 3.0 to 21 mg/kg, or into 25 to 176 ug/kg if expressed in terms of B(a)P. Comparison of these two mixtures of chemicals, crankcase and

contaminants in sediments, points to 300 to 2,100 times stronger effects from the contaminants in sediments than from crankcase (0.01 compared to 3.0-21 mg/kg). However, if the level of B(a)P is included in the comparison, there is hardly any difference in concentrations (0.1 compared to 0.025-0.176 mg/kg). Again, this comparison neglects the effect of other variables [519; Reprinted with permission from Environmental Toxicology and Chemistry, Volume 12, Upshall, C., J.F. Payne and J. Hellou. "Induction of MFO enzymes and production of bile metabolites in rainbow trout (Oncorhynchus mykiss) exposed to waste crankcase oil." Copyright 1992 SETAC].

Sed.Wildlife (Sediment Concentrations vs. Wildlife or Domestic Animals):

No information found.

Sed.Human (Sediment Concentrations vs. Human):

No information found.

Sed.Misc. (Other Non-concentration Sediment Information):

In an assessment of STORET data covering the period 1980-1982, Staples et al. (1985) reported median concentrations in sediment of less than or equal to 500 ug/kg dry weight for 15 PAHs (acenaphthene, acenaphthylene, anthracene, benz[a]anthracene, benzo[b]fluoranthene, benzo[k]fluoranthene, benzo[g,h,i]perylene, benzo[a]pyrene, chrysene, fluoranthene, fluorene, indenopyrene, naphthalene, phenanthrene, and pyrene). The number of sample ranged from 236 (anthracene) to 360 (benzo[a]pyrene, fluoranthene); the percentage of samples in which these PAHs were detected ranged from 6.0 (acenaphthene, benzo[b]fluoranthene, benzo[k]fluoranthene, indeno[1,2,3-c,d]pyrene) to 22.0 (fluoranthene, pyrene) [881].

Soil Data Interpretation, Concentrations and Toxicity (All Soil Data Subsections Start with "Soil."):

Soil.Low (Soil Concentrations Considered Low):

No information found.

Soil.High (Soil Concentrations Considered High):

A benzo(a)pyrene concentration of 650,000 ug/kg was measured in soil 10 meters from an industrial plant in Germany [788].

Soil Concentrations (mg/kg dry weight) Polycyclic Aromatic Hydrocarbons (PAHs) at Contaminated Sites. Highest values found at wood preserving, gas works, and coking site plants (mg/kg dry weight) [881]:

Benzo(a)pyrene 159

Soil.Typical (Soil Concentrations Considered Typical):

Misc. Soil Concentrations (mg/kg dry weight) [881]:

In a 1988 study at a hazardous waste land treatment site for refinery process wastes, which had been operative since 1958, average PAH concentrations in surface soils (0-30 cm) ranged from not detected (detection limits 0.1-2.0 mg/kg dry weight) for acenaphthylene, acenaphthene, anthracene, benz[a]anthracene, and benzo[k]fluoranthene to 340 mg/kg dry weight for dibenz[a,h]anthracene [881]. In addition to dibenz[a,h]anthracene, the three most prevalent compounds at this depth were benzo[a]pyrene (204 mg/kg), benzo[b]fluoranthene (130 mg/kg), and chrysene (100 mg/kg). PAH concentrations decreased with increasing depth and the majority of PAHs were not detected at depths below 60 cm. At 90-135 cm, only phenanthrene (1.4 mg/kg), pyrene (4.0 mg/kg), chrysene (0.9 mg/kg), and dibenz[a,h]anthracene (0.8 mg/kg) were found [881].

Background Soil Concentrations of Polycyclic Aromatic Hydrocarbons (PAH concentration in ug/kg) [881]:

(The below table is not indented to allow it to fit the margins):

Compound	Rural soil	Agricultural Soil	Urban Soil
Benzo(a)pyrene	2-1,300	4.6-900	165-220

Information from HSDB [366]:

Agricultural soil: Czechoslovakia, 8.3-42.1 ppb, Italy, 8-800 ppb(1,2). Forest soil: Massachusetts and eastern Connecticut, 40-1300 ppb, near Lake Constance, 1.5-2.5 ppb (dry weight), W. Germany, south of Darmstadt, 1.5-4.0 ppb (dry weight)(1,2). Iceland, near airfield, 785 ppb; near highway traffic, up to 2,000 ppb(1). 6 countries, remote from industrial- vehicular sources, 1.5-1300 ppb (dry wt)(max, mixed forest in Massachusetts)(7). [(1) Santodonato J et al; Health and Economic

Assessment of Polynuclear Aromatic Hydrocarbons. Lee SD, Grant L, eds Park Forest South, IL Pathotox Pub Inc. p 131-6 (1981) (2) Verschueren K; Handbook of Environmental p 273 (1983) (3) Kuntz KW; Toxic Contaminants in the Niagara River, 1975-1982. Technical Bulletin No. 134 Burlington, Ontario (1984) (4) Prahl FG et al; Environ Sci Technol 18: 687-93 (1984) (5) Varanasi U et al; Environ Sci Technol 19: 836-41 (1985) (6) Heit M; pp.89-103 in Environmental Measurement Laboratory-Environmental Quarterly, Hardy EP Jr, ed (1978) (7) Edwards NT; J Total Environ Qual 12: 427-41 (1983) (8) Sims RC, Overcash MR; Res Rev 88: 1-68 (1983)].

SOILS: 11 soils, 1.5-8010 ppb (max petroleum refinery soil)(8). USSR: Moscow and vicinity, 4 sites, 7-346 ppb, 3 industrial sites, 350-11,000 ppb(1,2). [(1) Santodonato J et al; Health and Economic Assessment of Polynuclear Aromatic Hydrocarbons. Lee SD, Grant L, eds Park Forest South, IL Pathotox Pub Inc. p 131-6 (1981) (2) Verschueren K; Handbook of Environmental p 273 (1983) (8) Sims RC, Overcash MR; Res Rev 88: 1-68 (1983)].

Soil.Concern Levels, Soil Quality Criteria, LC50 Values, Soil Quality Standards, Screening Levels, Dose/Response Data and Other Soil Benchmarks:

Soil.General (General Soil Quality Standards, Criteria, and Benchmarks Related to Protection of Soil-dwelling Biota in General; Includes Soil Concentrations Versus Mixed or General Soil-dwelling Biota):

Soviet Union Maximum Allowable Concentration in Soils, 1984: 0.02 mg/kg dry weight [347].

Canada's Interim Assessment Criterion for benzo(a)pyrene in soil is 0.1 ug/g dry weight [656].

NOTE: a) "Interim" means complete supporting rationale do not exist; b) for most of the organic parameters in [656], criteria are based on analytical detection limits and are intended to provide general guidance only for the protection of both human and environmental health [656].

Canada's Interim Remediation Criteria for benzo(a)pyrene in soil for three different land-uses (ug/g dry weight) [656]:

Agricultural = 0.1
Residential/Parkland = 1
Commercial/Industrial = 10

NOTE: a) "Interim" means complete supporting rationale do not exist; b) if contaminant concentrations exceed the criterion for a current or anticipated land use at a site, then the need for further investigation and/or remediation exists; c) criteria are relevant to protection of both human and environmental health [656].

Soil criteria for evaluating the severity of contamination under the Dutch Soil Cleanup (Interim) Act (1982): 0.1 ppm indicates a background concentration of BaP. 1 ppm indicates a moderate soil contamination of BaP. 10 ppm indicates a threshold value for BaP contamination which requires immediate cleanup [347].

Acceptable on-site soil concentrations approved by the Ontario Ministry of the Environment for the Texaco and Shell refinery sites (1987): The acceptable soil concentration for BaP is 0.004-0.005 ppm [347].

Soil.Plants (Soil Concentrations vs. Plants):

No information found.

Soil.Invertebrates (Soil Concentrations vs. Invertebrates):

Concentrations of PAH's in bioassay earthworms and bioassay soil from 15 sites at the Times Beach Confined Disposal Facility in Buffalo, N.Y. (1987): The mean concentration of BaP in the soil was 3.8 ppm (dry weight) the range was 0.36-8.6 ppm. The mean concentration of BaP in earthworms was 1.3 ppm (ash-free dry weight), the range was 0.14-7.4 ppm [347].

Soil.Wildlife (Soil Concentrations vs. Wildlife or Domestic Animals):

No information found.

Soil.Human (Soil Concentrations vs. Human):

EPA 1996 National Generic Soil Screening Level (SSL) designed to be conservative and protective at the majority of sites in the U.S. but not

necessarily protective of all known human exposure pathways, land uses, or ecological threats [952]:

SSL = 0.09 mg/kg for ingestion pathway [952].

SSL = 0.4 to 8 mg/kg for protection from migration to groundwater at 1 to 20 Dilution-Attenuation Factor (DAF) [952].

See also Canada's Interim Criteria [656] in Soil.General section above.

EPA 1995 Region 9 Preliminary remediation goals (PRGs) [868]:

Residential Soil: 0.061 mg/kg wet weight
Industrial Soil: 0.26 mg/kg wet weight

NOTE:
1) Values are based on a one-in-one million cancer risk.
2) PRGs focus on the human exposure pathways of ingestion, inhalation of particulates and volatiles, and dermal absorption. Values do not consider impact to groundwater or ecological receptors.
3) PRGs are slightly lower concentrations than EPA Region III RBCs, which consider fewer aspects [903].

EPA Region III Risk-Based Concentration (RBC) for soil screening levels related to potential transfers from soil to groundwater: 4 mg/kg dry weight [903].

Soil.Misc. (Other Non-concentration Soil Information):

Some plants can evidently catabolize benzo(a)pyrene, but metabolic pathways have not been clearly defined [40].

Tissue and Food Concentrations (All Tissue Data Interpretation Subsections Start with "Tis."):

Tis.Plants:

A) As Food: Concentrations or Doses of Concern to Living Things Which Eat Plants:

No information found.

B) Body Burden Residues in Plants: Typical, Elevated, or of Concern Related to the Well-being of the Organism Itself:

Plant Concentrations [366]:

Natural background levels: chrysanthemum, 1.20 ppb (dry wt); post oak (leaves), 30 ppb (dry wt), little bluestem (leaves), 30 ppb (dry wt)(1). [(1) Sims RC, Overcash MR; Res Rev 88: 1-68 (1983)].

Benzo(a)pyrene is a component of the condensate isolated from Citrullus colocynthis (coloquint) seeds. [Habs M et al; J Cancer Res Clin Oncol 108 (1): 154-6 (1984)].

Tis.Invertebrates:

A) As Food: Concentrations or Doses of Concern to Living Things Which Eat Invertebrates:

No information found.

B) Concentrations or Doses of Concern in Food Items Eaten by Invertebrates:

No information found.

C) Body Burden Residues in Invertebrates: Typical, Elevated, or of Concern Related to the Well-being of the Organism Itself:

See information under Tis.Fish, C), below.

Details of benzo(a)pyrene content (ug/kg or ppb) in whole body samples of mussels) from Snug Harbor, Alaska, an area heavily oiled by the Exxon Valdez Crude Oil, 4/15/89 [971]:

Note: Concurrent measurements of water quality, as well as equilibrium partitioning estimates of water quality based on concentrations in fish and mussels, both confirm that PAH concentrations did not exceed water quality criteria at the time these concentrations were measured in mussel tissues [971]. These values are wet weight (Jerry Neff, Battelle Ocean Sciences, Duxbury, MA, personal communication 1996):

benzo(a)pyrene: 65.8 ug/kg = ppb

Tis.Fish:

A) As Food: Concentrations or Doses of Concern to Living Things Which Eat Fish (Includes FDA Action Levels for

Fish and Similar Benchmark Levels From Other Countries):

No information found.

B) Concentrations or Doses of Concern in Food Items Eaten by Fish:

The IJC (1983) recommended that the concentration of benzo(a)pyrene in sediments or in organisms serving as a food source for fish should not exceed 1.0 ug/g. They also noted that other 3- to 5-ring PAHs are carcinogenic and may be of equal or greater concern [754].

C) Body Burden Residues in Fish: Typical, Elevated, or of Concern Related to the Well-being of the Organism Itself:

The B(a)P content in fish is quite variable; demersal species do not appear to contain significantly higher amounts of B(a)P than do pelagic (out in the open sea) species [754].

The benzo(a)pyrene concentration in mosquitofish with extremely elevated total PAH concentrations (60.79 mg/kg) was 5.3 mg/kg [201]. This B(a)P concentration is of concern because quantities this high are very unusual in fish [201].

No detections of this compound were made in certain samples of Exxon Valdez fish [971].

Rainbow trout withstood the injection of up to 30 mg/kg B(a)P [754].

Fish/Seafood Concentrations [366]:

Raw fish from unpolluted waters usually do not contain detectable amounts of polyaromatic hydrocarbons (PAH), but smoked or cooked fish contain varying levels of PAH(1). The following conc were noted for benzo(a)pyrene. Smoked fish, 12 species, not detected (nd)-4.4 ppb(1,2). Smoked oysters, 9 ppb; cooked scallops, 9.9 ppb(1). Uncooked marine seafood: mussel, not detected (nd)-540 ppb (max, sample from Italian coast)(2,3); oysters, 4 locations, 0.1-7.0 ppb; codfish, nd-1.5 ppb; clams, 4 locations, nd-3 ppb; crab, nd-3 ppb; mollusk, 2.4-60 ppb (max, Greenland); codfish, Greenland, 15 ppb; sardine, Italy, 65 ppb; shrimp, Palacios, TX, nd(2,3). Lake trout, Lake Maskinonge,

Ontario, Canada, <1 ppb(2). Mussels, Saudafjord, Norway, 1976, 0.517-20.8 ppm (dry wt)(4). 0.78 ppb, corb-shell, 0.21 ppb, shortnecked crab, 0.41-1.79 ppb(5). [(1) Santodonato J et al; Health and Economic Assessment of Polynuclear Aromatic Hydrocarbons. Lee SD, Grant L, eds Park Forest South, IL Pathotox Pub Inc. p 124-26 (1981) (2) Verschueren K; Handbook of Environmental Data on Organic Chemicals. 2nd ed Von Nostrand Reinhold NY p 275-6 (1983) (3) Fazio T, Howard JW; pp.461-506 in Handbook of Aromatic Hydrocarbons; Bjorseth A, ed (1983) (4) Bjorseth A et al; Sci Total Environ 13: 71-86 (1979) (5) Takatsuki K et al; JAOAC 68: 945-49 (1985)].

Tis.Wildlife: Terrestrial and Aquatic Wildlife, Domestic Animals and all Birds Whether Aquatic or not:

A) As Food: Concentrations or Doses of Concern to Living Things Which Eat Wildlife, Domestic Animals, or Birds:

No information found.

B) Concentrations or Doses of Concern in Food Items Eaten by Wildlife, Birds, or Domestic Animals (Includes LD50 Values Which do not Fit Well into Other Categories, Includes Oral Doses Administered in Laboratory Experiments):

LD50 Mouse intraperitoneal about 250 mg/kg [IARC. Monographs on the Evaluation of the Carcinogenic Risk of Chemicals to Man. Geneva: World Health Organization, International Agency for Research on Cancer, 1972-1985. (Multivolume work).,p. V32 213 (1983)] [366].

Oak Ridge National Lab, 1994: Risk Assessment Screening Benchmarks for Wildlife derived from No-Observed-Adverse-Effect (NOAEL) levels. To be considered unlikely to represent an ecological risk, wet-weight field concentrations should be below the following benchmarks for each species (listed below) present at the site [650]:

SPECIES	NOAEL (mg/kg/day)	FOOD CONC (ppm)
Mouse (test species)	1.00000	0.00000
Short-tailed Shrew	1.25700	2.09500
Little Brown Bat	1.58000	4.74000

White-footed Mouse	1.10800	7.16800
Meadow Vole	0.88100	7.75500
Cottontail Rabbit	0.29600	1.49900
Mink	0.31400	2.29500
Red Fox	0.19100	1.91400
Whitetail Deer	0.08300	2.69600

Oral exposure to 120 mg/kg/day benzo[a]pyrene has resulted in decreased survival time in two strains of mice (DBA/2N and AKR/N) whose hepatic aryl hydrocarbon hydroxylase (AHH) activity is not induced by PAHs ("nonresponsive" mice) [881]. AHH is a microsomal enzyme believed to be responsible for the metabolism of benzo[a]pyrene [881]. All of the mice in the treatment group died, with at least half the deaths occurring within 15 days of dosing [881]. Only three mice in the control group died [881]. Death appeared to be caused by bone marrow depression (aplastic anemia, pancytopenia), leading to hemorrhage or infection [881]. In contrast, only 6 of 90 (7%) mice with inducible AHH activity ("responsive" mice) similarly exposed to benzo[a]pyrene died over the same period of time [881]. The authors concluded that the decreased survival in the nonresponsive mice was associated with a single gene difference encoding aromatic hydrocarbon responsiveness and was dependent on route of exposure [881]. Benzo[a]pyrene was not as rapidly metabolized by the liver and excreted following oral administration in nonresponsive mice as in responsive mice [881]. Therefore, more benzo[a]pyrene was available to reach the target tissue (i.e., bone marrow) in the nonresponsive mice, resulting in bone marrow depression and death [881].

C) Body Burden Residues in Wildlife, Birds, or Domestic Animals: Typical, Elevated, or of Concern Related to the Well-being of the Organism Itself:

No information found.

Tis.Human:

A) Typical Concentrations in Human Food Survey Items:

See also Tis.Fish, C), above for B(a)P concentrations in Fish and Seafood.

Food Survey Results [366]:

PPB of benzo(a)pyrene in foodstuff: fresh vegetables 2.85-24.5; Vegetable oils 0.4-1.4;

Coconut oil 43.7; Margarine 0.4-0.5; Mayonnaise 0.4; Coffee 0.3-1.3; Tea 3.9; Grain 0.19-4.13; Oysters & mussels 1.5-9.0; Smoked sausage 12.5-18.8; Singed meat 35-99. Broiled meat 0.17-0.63; Charcoal-broiled steak 8.0; Broiled mackerel 0.9; Barbecued beef 3.3; Barbecued ribs 10.5. /From table/ [Searle, C. E. (ed.). Chemical Carcinogens. ACS Monograph 173. Washington, DC: American Chemical Society, 1976. 706].

In fruit ... & cereals B(a)P content depends on their source (closeness to industrial areas or traffic highways). ... Following amt of B(a)P were found: salad, 2.8-5.3 ug/kg; spinach, 7.4 ug/kg; tomatoes, 0.2 ug/kg; kale, 12.6-48.1 ug/kg; soya beans, 3.1 ug/kg; apples, 0.1-0.5 ug/kg; other fruits, 2-8 ug/kg. [IARC. Monographs on the Evaluation of the Carcinogenic Risk of Chemicals to Man. Geneva: World Health Organization, International Agency for Research on Cancer, 1972-1985. (Multivolume work).,p. V3 101 (1973)].

In meat or fish amt of B(a)P present depends on method of cooking: time of exposure, distance from heat source & whether or not melted fat is allowed to drop into heat source. [IARC. Monographs on the Evaluation of the Carcinogenic Risk of Chemicals to Man. Geneva: World Health Organization, International Agency for Research on Cancer, 1972-1985. (Multivolume work).,p. V3 100 (1973)].

United Kingdom, 1979, total diets, 0.0036 ug/kg/day; range (avg) ppb in food classes: cereals, 0.12-0.79 (0.32), meat, 0.02-0.08 (0.05), oils and fats, 0.019-3.74 (0.13), fruit and sugar, 0.03-0.10 (0.07), root vegetables, 0.02-0.13 (0.06), beverages, not detected (nd)-0.02 (0.008)(3). Raw meat does not normally contain polyaromtic hydrocarbons (PAH), but smoked or cooked meat may contain varying amounts(1). Levels of B(a)P found in cooked meats were affected by several factors including cooking time, distance from heat source, type of heat source (eg charcoal, gas, electric), fat content, and whether the heat source is above or below the meat(1). Smoked meat products, trace-55.0 ppb (max, heavily-smoked ham); barbecued (bbq) beef, 3.5 ppb; bbq ribs, 10.5 ppb; charcoal broiled steaks,

5.8 ppb; broiled t-bone steaks: cooked with charcoal-hot, 50.4 ppb, cooked with flame-hot, 4 ppb(1). Hamburger: raw, <0.1 ppb, cooked, 2.5 ppb; smoked meat, avg of 47 products, 0.225 ppb(2). Vegetables: source not noted, (nd)-7.4 ppb, polluted environments, 0.1-24.3 ppb (max, parsley)(1). Grains and cereal products, nd-60.0 ppb (max, wheat sprouts); fruits, source not noted, nd-1.5 ppb, polluted environments, 0.2-29.7 ppb (max, plums); baker's yeast, 0.5-12.2 ppb; roasted coffee: moderately dark, nd, darkest, nd-4.0 ppb, instant, 0.02-0.06 ppb; tea, nd-16 ppb; dark rum, 1.0 ppb; whiskey, 0.04 ppb; vegetable oils, 7 types, nd-10.6 ppb (max, sunflower oil); butter, nd; margarine, 0.2-6.8 ppb(1,2). [(1) Santodonato J et al; Health and Economic Assessment of Polynuclear Aromatic Hydrocarbons. Lee SD, Grant L, eds Park Forest South, IL Pathotox Pub Environmental Data on Organic Chemicals. 2nd ed Von Nostrand Reinhold NY p 274-75 (1983) (3) Dennis MJ et al; Fd Chem Toxicol 21: 569-74 (1983)].

T-bone steak (long cooking time): 50 ug/kg; Charcoal-broiled or barbecued meat: 2.6-11.2 ug/kg; Icelandic home smoked meat: up to 23 ug/kg; Meat hung close to stove: 107 ug/kg; Smoked meat, ham and sausages: 0.02 - 14.6 ug/kg; Broiled meat or sausages: 0.17 - 0.63 ug/kg; Gas-broiled fish: 0.9 ug/kg; Smoked fish: trace to 37 ug/kg; Salad: 2.8 - 5.3 ug/kg; Spinach: 7.4 ug/kg; Tomatoes: 0.2 ug/kg; Kale: 12.6 - 18.1 ug/kg; Soybeans: 3.1 ug/kg; Apples: 0.1 - 0.5 ug/kg; Cereals: 0.25 - 0.84 ug/kg; Margarine or mayonnaise: 0.2 - 6.8 ug;kg; Crude vegetable oils: 0.4 - 36 ug/kg; coconut oil, olive oil, plant cooking fat, plant oil: up to 43.7 ug/kg; Coconut fat: up to 62 ug/kg; Roasted or black coffee: 0.1 - 4 ug/kg; Malted coffee: 15ug/kg; Tea: 3.9 - 21.3 ug/kg. /From table/ [Health & Welfare Canada; Polycyclic Aromatic Hydrocarbons p.29-30 (1979) Report No. 80-EHD-50].

... In baker's dry yeast ... 1.8-40.4 ug/kg ... Dietetic yeast or feed yeasts grown on mineral oil show lower content. ... 0.04 ug/kg in one of 15 brands of whiskey; in prunes dried by different methods, concn ranged from 0.2-1.5 ug/kg ... Detected in algae ... In leaves from different kinds of trees. [IARC. Monographs on the Evaluation of the Carcinogenic Risk of Chemicals to Man. Geneva:

World Health Organization, International Agency for Research on Cancer, 1972-1985. (Multivolume work).,p. V3 102 (1973)].

B) Concentrations or Doses of Concern in Food Items Eaten by Humans (Includes Allowable Tolerances in Human Food, FDA, State and Standards of Other Countries):

Slope Factor: 7.3E+0 per mg/(kg/day) [868,893,952]. The slope factor is the result of application of a low-dose extrapolation procedure and is presented as the risk per mg/kg/day [868,893]. Oral Reference Dose (RfD): none given [893].

For risk to human adults eating fish, separate carcinogenic and non-carcinogenic risk-based fish tissue concentrations were calculated [903]. The following EPA Region III fish tissue risk-based concentration (RBC) benchmark utilizes the lower of the two concentrations (carcinogenic), rounded to two significant figures [903]:

RBC = 0.00043 mg/Kg wet weight.

C) Body Burden Residues in Humans: Typical, Elevated, or of Concern Related to the Well-being of Humans:

Milk Concentrations [366]:

Dutch milk, 51 samples, 0.03-0.66 ppb, avg 0.03 ppb(1). Dried milk, Canada: skim, 0.1 ppb, infant formula, 1.2 ppb(2). United Kingdom, 1979, 0.005-0.02 ppb, avg 0.01 ppb(3). [(1) Betlem JP; Food Inspection Service Amsterdam, Report No. 15, Amsterdam, The Netherlands (1981) (2) Lawrence JF, Weber DF; J Agric Food Chem 32: 794-7 (1984) (3) Dennis MJ et al; Fd Chem Toxicol 21: 569-74 (1983)].

Milk of nursing mothers was analyzed for the presence of benzo(a)pyrene. Levels of 7.6 to 387 ng/ml (with the mean value of 129.5 ng/ml) were found in sample of ten nursing mothers. [Health & Welfare Canada; Polycyclic Aromatic Hydrocarbons p.38 (1979) Report No. 80-EHD-50].

Human Body Burdens [366]:

Human liver, 6 samples, 100% pos 10-22 ppt (wet wt), avg 784 parts per trillion; fat, 9 samples, 89% pos, 16-59 ppt (wet wt), avg 21.4 parts per trillion(1). [(1) National Research

Council; Polycyclic Aromatic Hydrocarbons: Evaluation of Sources and Effects, National Acad Press Washington, DC p 6-36 to 6-37 (1983)].

Milk of nursing mothers was analyzed for the presence of benzo(a)pyrene. Levels of 7.6 to 387 ng/ml (with the mean value of 129.5 ng/ml) were found in sample of ten nursing mothers. [Health & Welfare Canada; Polycyclic Aromatic Hydrocarbons p.38 (1979) Report No. 80-EHD-50].

Tis.Misc. (Other Tissue Information):

This is a very phototoxic PAH [887,891]. See cautionary note under W.General section above.

Benzo(a)pyrene concentrations in marine organisms range from nondetectable (usually <0.0001 ug/g dry wt) to as high as 5 ug/g dry wt [754].

Bio.Detail: Detailed Information on Bioconcentration, Biomagnification, or Bioavailability:

If released to water, it will adsorb very strongly to sediments and particulate matter, bioconcentrate in aquatic organisms which can not metabolize it, but will not hydrolyze. (SRC).

During the Exxon Valdez spill, bioconcentration explained the buildup of PAHs in tissues better than biomagnification; most accumulation was of an equilibrium partitioning nature across the gills rather than from the food chain [971]. Immature fish seem to have higher bioconcentration of PAHs than adults, perhaps because their PAH breakdown systems are not fully developed and at times perhaps because of a higher percentage of lipid tissues (yolk tissues, etc) [971] (confirmed by Jerry Neff, Battelle Ocean Sciences, Duxbury, MA, personal communication 1996).

B(a)P is rapidly metabolized by mosquito fish and slowly metabolized by snails [Chapter 18 in 177].

34 different log bioconcentration factor values (log BCF) from the literature range from -0.155 for capitella capitata to 6.95 for p. hoyi [848]. Other values are [848]: 3.45, 3.90, and 4.11 for Daphnia magna; 2.69, 3.69, and 4.45 for bluegill sunfish; 2.68 for fish; 3.52 for algae; 3.51 for worms; 1.140 for polychaete sp; 1.10 for macrochirus; and 5.65 for mixed microbial population.

Bioconcentration factors (BCF) of 100 to 10,000 were found for sediment and biota relative to water for benzo(a)pyrene [754]. Bioconcentration factors for benzo(a)pyrene range from 930 in the mosquitofish (Gambusia affinis) to 134,240 in Daphnia pulex [754].

The amount of benzo(a)pyrene metabolism by aquatic organisms has been ranked as follows: fish > shrimp > amphipod crustaceans > clams [788].

Half-life depuration by oysters is 18 days, by bluegill sunfish is 67 hours, and by s. heringianus is 52 hours [848].

Calculated half-lives in different tissues of sea bass: 12.4 days for fat, 5.1 and 6.5 days for kidney, 5.1 days for intestine, 4.8 days for gallbladder, 4.5 days for spleen, 2.9 days for muscle, 2.4 days for whole body, 2.3 days for gonads, 2.3 days for gills, and 2.2 days for liver [848].

Concentrations of PAH's in bioassay earthworms and bioassay soil from 15 sites at the Times Beach Confined Disposal Facility in Buffalo, N.Y. (1987): The mean concentration of BaP in the soil was 3.8 ppm (dry weight) the range was 0.36-8.6 ppm. The mean concentration of BaP in earthworms was 1.3 ppm (ash-free dry weight), the range was 0.14-7.4 ppm [347].

Information from HSDB [366]:

Reported BCF: Oysters (Crassostrea virginica), 3000(1); Rainbow trout, 920(2); Bluegills, 2,657(3); Daphnia magna, 1000(5); Daphnia pulex, 13,000(6). The presence of humic acid in solution have been shown to decr bioconcentration: eg, Daphnia magna, BCF 1716 (humic material (hm) 0.3 ppm dissolved organic carbon (DOC), BCF 979 (hm 1.5 ppm DOC), BCF 838 (hm 5.7 ppm DOC)(4). [(1) Verschueren K; Handbook of Environmental Data on Organic Chemicals. 2nd ed Von Nostrand Reinhold NY p.27 (1983) (2) Spehar RL et al; J Water Pollut Control Fed 52: 1703-74 (1980) (3) McCarthy JF, Jimenez BD; Environ Sci Technol 19: 1072-6 (1985) (4) Leversee GJ et al; Can J Fish Aquat Sci 40: 63-9 (1983) (5) McCarthy JF; Arch Environ Contam Toxicol 12: 559-68 (1983) (6) Biddinger GR, Gloss SP; Res Rev 91: 103-45 (1984) (7) Santodonato J et al; Health and Economic Assessment of Polynuclear Aromatic Hydrocarbons. Lee SD, Grant L, eds Park Forest South, IL: Pathotox Pub Inc. p.160-7 (1981)].

Gillichthys mirabilis (mudsucker) exposed to bioconcentration factor of 0.048; Oligocottus maculosus (tidepool sculpin) exposed to benzo(a)pyrene /concn not specified/ for 1 hr exhibited a bioconcentration factor of 0.13; Citharichthys stigmacus (sand dab) exposed to benzo(a)pyrene /concn not specified/ for 1 hr exhibited a bioconcentration factor of 0.02. /Edible tissue/ [Lee RG et al; Mar Biol 17: 201 (1972) as cited in USEPA; Ambient Water Quality Criteria Doc: Chloroalkyl Ethers p.B-4 (1980) EPA 440/5-80-030].

... Some marine organisms have no detectable aryl hydrocarbons hydroxylase enzyme systems, namely: phytoplankton, certain zooplankton, mussels (Mytilus edulis), scallops (Placopecten sp), and snails (Litternia littorea). ... Those organisms which lack a metabolic detoxification enzyme system, tend to accumulate polycyclic aromatic hydrocarbons. /Polycyclic aromatic hydrocarbons/ [Malins DC; Ann NY Acad Sci 298: 482-496 (1977) as cited in: Health and Welfare Canada; Polycyclic Aromatic Hydrocarbons p.37 (1979) Report No. 80-EHD-50].

Polycyclic Aromatic Hydrocarbons (PAH) were analyzed in surfacial sediments & benthic organisms in southeastern lake Erie, near a large coal-fired power plant. Sediment concn (530-770 ppb PAH) were relatively homogenous throughout most of the 150 square km area, although river & nearshore concentrations reached 4 ppm. Oligochaete worms did not bioconcentrate (on wet wt basis) any of the PAH. Chironomide midges collected 1 km offshore exhibited bioconcentration of 5 PAH one of which was pyrene. Further offshore, these apparent bioconcentrations disappeared, with midges at near equilibrium with sediments. /PAH/ [Eadie BJ et al; Chemosphere 11 (2): 185-92 (1982)].

Interactions:

Photoinduced toxicity may occur for benzo(a)pyrene [779].

Oral absorption of benzo[a]pyrene is enhanced by some oils (such as corn oil) in the gastrointestinal tract [881].

Information from HSDB [366]:

> ... Experimental evidence supports the antineoplastic effect of selenium with regard to benzo(a)pyrene-induced ... skin tumors in mice. [Doull, J., C.D.Klassen, and M.D. Amdur (eds.). Casarett and Doull's Toxicology. 3rd ed., New York: Macmillan Co., Inc., 1986. 617].
>
> Rabbits treated with benzo(a)pyrene developed cardiac arrhythmias when exposed by inhalation to 8100 ppm trichloroethylene or 15000 ppm halothane to a greater extent & at lower doses of epinephrine challenge than did controls. Benzo(a)pyrene increased the metabolism of trichloroethylene. The basis of the arrhythmogenic action of benzo(a)pyrene was unrelated to its ability to induce xenobiotic metabolism. [Carlson GP, White JF; Toxicol Lett (AMST) 15 (1): 43-8 (1983)].
>
> In mouse hepatic microsomes, Dietary Butylated Hydroxyanisole (BHA) enhanced the total metabolism of benzo(a)pyrene (BP) but decreased the microsomal metabolism of (BP)-7,8-diol, especially the formation of (BP)-trans-7,8-diol-anti-9,10-oxide. The altered metabolism of benzo(a)pyrene is believed to be due to the induction of new cytochrome p-450 species by dietary BHA. Thus, bha can affect benzo(a)pyrene metabolism by exerting its inhibitory effect directly & by altering the composition of microsomal monooxygenase enzymes after a few days of exposure. [Sydor W JR et al; Carcinogenesis 4 (2): 131-6 (1983)].
>
> The pharmacokinetics of benzo(a)pyrene (BaP) in the isolated perfused rabbit lung (IPL) following

pretreatment of the whole animal or simultaneous administration to the isolated perfused rabbit lung with n-dodecane, ferric oxide, crude airborne particulate (CAP), fly ash or sulfur dioxide have been investigated using a one compartment model. The rate constant for the appearance (ka) of B(a)P in the blood, the clearance of B(a)P from the blood, and the rate of appearance of B(a)P metabolites (RAM) were the kinetic parameters determined. B(a)P entered the blood rapidly with an average half-life of 11 min in experiments in which the isolated perfused rabbit lungs received only B(a)P on perfusion. The logarithms of the clearances from these experiments were linearly correlated with the B(a)P metabolites. In these experiments, pretreatment of the whole animal with B(a)P produced a 48-55-fold incr in B(a)P clearance while pretreatment with n-dodecane increased the clearance 4-fold in comparison with no pretreatment. Pretreatment with ferric oxide, or ferric oxide and B(a)P increased the clearance by factors of 5.5 and 1.5, respectively, over those of unpretreated and B(a)P pretreated experiments. [Morgan DD et al; Toxicology 33 (3-4): 275-89 (1984)].

Ip injection of channel catfish (Ictalurus punctatus) with 100 ug benzo(a)pyrene, Aroclor 1254, or naphthalene, singly and in combinations, affected the levels of the brain neurotransmitters norepinephrine, dopamine, and 5-hydroxytryptamine, but the effect showed no discernible pattern. The effects of combinations of the chemicals did not appear to be predictable from the effects of individual chemicals. In several instances, the change in the level of neurotransmitter in fish receiving a combination of chemicals was greater than in fish receiving either chemical alone. [Fingerman SW, Short EC; Bull Environ Contam Toxicol 30 (2): 147-51 (1983)].

The modifying effect of solvents on the carcinogenicity of B(a)P is well demonstrated by comparison between the effects of 3 weekly paintings with different concn of B(a)P either in n-dodecane/decalin (50:50 mixture) or in decalin on C3H/He mice. When n-dodecane/decalin was the solvent, 5 malignant tumors appeared among l24 mice painted with the lowest concn ... 0.00002%, & the tumor incidence increased at higher doses. With decalin, no skin tumors developed in any ... mice below 0.02% concn. [IARC. Monographs on the Evaluation of the Carcinogenic Risk of Chemicals to Man. Geneva: World Health Organization, International Agency for Research on Cancer, 1972-1985. (Multivolume work).,p. V3 105 (1973)].

In inhalation study, of group of 21 rats exposed to mixture of 10 mg/cu m b(a)p & 3.5 Ppm so2 /sulfur dioxide/ for 1 hr/day for more than a yr, 2 developed squamous cell carcinomas of lung. In another group of 21

rats which received additional treatment with so2 of 10 ppm for 6 hr/day, squamous cell carcinomas appeared in five rats. No tumors were found among 3 rats receiving so2 only. No group was exposed to b(a)p alone. [IARC. Monographs on the Evaluation of the Carcinogenic Risk of Chemicals to Man. Geneva: World Health Organization, International Agency for Research on Cancer, 1972-1985. (Multivolume work).,p. V3 107 (1973)].

Carcinogenicity of hydrocarbon mixt predominantly found in automobile exhaust gas condensate was attributed to the syncarcinogenic action of benzo(a)pyrene, dibenz(a,h)anthracene, benz(a)anthracene, & benzo(b)fluoranthene. The 4 carcinogenic hydrocarbons were tested in mice with single dosage levels of 4-12 mug. [Schmaehl D et al; IARC SCI Publ 16 (Air pollut cancer man, proc hanover int carcinog meet, 2ND): 53-9 (1977)].

The frequencies of base-line and benzo(a)pyrene induced sister chromatid exchanges (SCE) were measured in peripheral blood lymphocytes from 22 male asbestos-exposed workers and 10 nonexposed workers of comparable age. A clear association between cigarette smoking and asbestos exposure in the sensitivity of lymphocytes to BP was observed. Among asbestos-exposed workers, lymphocytes from those who smoked cigarettes were significantly more susceptible to the induction of sister chromatid exchanges by in vitro exposure to BP (p= 0.01) than were lymphocytes from nonsmokers. Active smoking elevated the base-line sister chromatid exchange frequency in both asbestos-exposed and noneposed workers (p= 0.001), and an interaction between smoking and asbestos in the production of base-line sister chromatid exchange was suggested (p= 0.07). Asbestos exposure alone was not associated with an enhanced susceptibility to the induction of sister chromatid exchanges by BP or with an elevation of base-line sister chromatid exchange. Increased age was associated with an increase in sister chromatid exchanges inducibility by BP (p= 0.01), and a history of smoking was marginally associated with sister chromatid exchanges inducibility by BP (p= 0.07). These findings support the hypothesis that an increased susceptibility of asbestos-exposed individuals to polyaromatic hydrocarbon-induced cancer results from an enhanced sensitivity to the induction of genetic damage rather than to an asbestos-induced differential cellular metabolic capacity. [Kelsey KT et al; JNCI 77 (2): 321-7 (1986)].

Uses/Sources:

See also Chem.detail section below for B(a)P concentrations in

various petroleum products.

Is benzo(a)pyrene (BAP) natural, pyrogenic, or petrogenic? It can be all three:

> BAP is a product of incomplete combustion and there are natural pyrogenic sources including volcanoes and forest fires [366]. BAP is also formed from pyrolysis of anthracene [366]. So BAP has pyrogenic sources. However, there is also some evidence for biosynthesis of BAP by plants and bacteria (natural sources), yet BAP is also found in crude oils and many other oil products (petrogenic sources) [366]. BAP has been found in gasoline; fresh motor oil; used motor oil; used motor oil; various crude oils; diesel oil (gasoil); asphalt; and coal tar pitch [366].

During the summer of 1996, NASA announced that PAHs had been found on a martian meteorite. Three to 6 ring PAHs found included phenanthrene, pyrene, chrysene, perylene, and benzo(a)pyrene, with less than 10% of the mass being alkyl PAHs. It was said that the meteorite PAHs were typified by little alkylation and a lack a dibenzothiophene, making the PAHs different than found in the typical earth atmosphere. However, another unidentified mass of alkyl PAH compounds were also found and NASA acknowledged that PAHs have been found in a wide range of extraterrestrial materials [McKay et.al. 1996, manuscript entitled "Search for Life on Mars: Possible Biogenic Activity in Martian Meteorite ALH84001," a NASA paper available at the time of the NASA press release].

> Note from Roy Irwin: This represents and interesting and somewhat speculative attempt to link fingerprinting of PAH combinations to possible life on mars. NASA admits that the PAHs alone do not prove there was life on mars, and I may personally remain a bit skeptical until more comprehensive and convincing evidence is presented.

Several monitoring studies indicate that there are higher concentrations of PAHs in urban air than in rural air. Pucknat (1981) summarized 1970 data from the U.S. National Air Surveillance Network and reported that benzo[a]pyrene concentrations in 120 U.S. cities were between 0.2 and 19.3 ng/m3. Ambient benzo[a]pyrene concentrations in nonurban areas ranged between 0.1 and 1.2 ng/m3 [881].

In a 1981-82 study that characterized air levels of 13 PAHs in Los Angeles, Grosjean (1983) reported mean ambient particle-phase PAH concentrations ranging from 0.32 ng/m3 for benzo[k]fluoranthene to 3.04 ng/m3 for combined benzo[g,h,i]perylene and indeno[1,2,3-c,d]pyrene. Mean concentrations of anthracene, fluoranthene, pyrene, chrysene, benz[a]anthracene, combined perylene and benzo[e]pyrene, benzo[b]fluoranthene, and benzo[a]pyrene were 0.54; 0.94, 1.62, 0.97, 0.48, 0.43. 0.94, and 0.64 ng/m3, respectively [881].

Major Uses [366]:

... used extensively as a positive control in a variety of /laboratory mutagenicity & carcinogenicity/ short-term tests. [IARC. Monographs on the Evaluation of the Carcinogenic Risk of Chemicals to Man. Geneva: World Health Organization, International Agency for Research on Cancer, 1972-1985. (Multivolume work).,p. V32 215 (1983)].

Research chemical [SRI].

Not used commercially in usa [SRI].

Natural Occurring Sources [366]:

... In crude oils, shale oils, & coal tars, & is emitted with gases & fly ash from active volcanoes. [Clayton, G. D. and F. E. Clayton (eds.). Patty's Industrial Hygiene and Toxicology: Volume 2A, 2B, 2C: Toxicology. 3rd ed. New York: John Wiley Sons, 1981-1982. 3362].

Since benzo(a)pyrene is a product of incomplete combustion, there will be natural sources including volcanoes and forest fires(SRC). There is some evidence for biosynthesis by plants(2) and bacteria(1,2). Crude oils, 0.40-1.66 ppm(1), 0.1-3.6 ppm(3). [(1) Verschueren K; Handbook of Environmental Data on Organic Chemicals. 2nd ed Von Nostrand Reinhold NY pp. 263-4 (1983) (2) Sims RC, Overcash MR; Res Rev 88: 1-68 (1983) (3) IARC; Polynuclear Aromatic Compounds, Part 1. Chemical, Environmental and Experimental Data. 32: 211-24 (1983)].

Occurs in fossil fuels. [SRI].

Artificial Sources [366]:

/Found/ in furnace blacks, automobile tires & rubber stoppers ... /The concentrations measured/ did not diminish on processing, aging or wear. [IARC. Monographs on the Evaluation of the Carcinogenic Risk of Chemicals to Man. Geneva: World Health Organization, International Agency for Research on Cancer, 1972-1985. (Multivolume work).,p. V3 102 (1973)].

B(a)P has been produced by pyrolysis of anthracene at 950 deg c ... Of dicetyl at 800 deg c ... of carbohydrates, amino acids & fatty acids at 700 deg c ... At 500 deg c ... tobacco constituents at 650 deg c ... Aliphatic hydrocarbons, 30 mg/kg at 800 deg c ... Found ... /In/ agar-agar, natural dyes, humectants, glues, starches logwood. [IARC. Monographs on the Evaluation of the Carcinogenic Risk of Chemicals to Man. Geneva: World Health Organization, International Agency for Research on Cancer, 1972-1985. (Multivolume work).,p. V3 96 (1973)].

Ubiquitous product of incomplete combustion, occurring in exhaust from motor vehicles and other gasoline and diesel engines, emmission from coal-, oil-, and wood-burning stoves and furnaces, cigarette smoke; general soot and smoke of industrial, municipal, and domestic origin, and cooked foods, especially charcoal-broiled(1). Incinerators, coke ovens, and asphalt processing and use(2). [(1) IARC; Polynuclear Aromatic Compounds, Part 1. Chemical, Environmental and Experimental Data. 32: 211-24 (1983) (2) Verschueren K; Handbook of Environmental Data on Organic Chemicals. 2nd ed Von Nostrand Reinhold NY p.265 (1983)].

Air pollution from motor transport exhaust gases was studied in Kazan, Russian SFSR, USSR, in 1974-1977. In the central part of town, where motor transport movement was most intense, the concn of CO, NOx and benz(a)pyrene in the air was higher than in new industrial regions. There was a direct relationship between concentrations of these substances and the intensity of motor transport flow. ... [Dauton FF; Kazan Med Zh 61 (3): 61-3 (1980)].

In gasoline: 0.135 mg/l - 8.28 mg/kg; In fresh motor oil: 0.02 - 0.10 mg/kg; In used motor oil: 5.8 mg/l; In used motor oil after 5,000 km: 83.2 - 162.0 mg/kg; In used motor-oil after 10,000 km: 110.0 - 242.4 mg/kg, In Kuwait crude oil: 2.8 ppm; In South Louisiana crude oil: 2.8 ppm; In diesel oil (gasoil): 0.026 mg/l; In asphalt up to 0.027 wt%; In coal tar pitch: up to 1.25 wt %. [Verschueren, K. Handbook of Environmental Data of Organic Chemicals. 2nd ed. New York, NY: Van Nostrand Reinhold Co., 1983. 264].

Coal tar processing, petroleum refining, shale refining, ... kerosene processing, heat and power generation [Verschueren, K. Handbook of Environmental Data of Organic Chemicals. 2nd ed. New York, NY: Van Nostrand Reinhold Co., 1983. 263].

Mainstream cigarette smoke per cigarette: 20-40 ug, 4.7-7.8 ug, 0.0129-0.0354 ug, 135 ug; per 100 cigarettes: 4.6 ug, 3.1 ug, 2.5 ug, 1.7-2.3 ug, 0.0018-0.008 ug; cigar smoke, 3.4-5.1 ug/100 g, 2.2-4 ug/cigar; mainstream smoke of marijuana cigarettes, 2.9 ug/100 cigarettes, 3.1 ug/cigarette(1). [(1) IARC; Polynuclear aromatic compounds, Part 1. Chemical, Environmental and Experimental Data. 32: 211-24 (1983)].

Emissions from typical European gasoline engine (1608 cu cm) ... using leaded and unleaded commercial gasolines: 1.9 - 26.0 ug/l fuel burnt. In exhaust condensate of gasoline engine: 0.05 - 0.08 mg/l gasoline consumed. [Verschueren, K. Handbook of Environmental Data of Organic Chemicals. 2nd ed. New York, NY: Van Nostrand

Reinhold Co., 1983. 263].

Grimmer et al. reported concentrations of benzo(a)pyrene to be 7,226 times higher in "used" compared to "fresh" oil [519; Reprinted with permission from Environmental Toxicology and Chemistry, Volume 12, Upshall, C., J.F. Payne and J. Hellou. "Induction of MFO enzymes and production of bile metabolites in rainbow trout (Oncorhynchus mykiss) exposed to waste crankcase oil." Copyright 1992 SETAC].

Forms/Preparations/Formulations:

No information found.

Chem.Detail: Detailed Information on Chemical/Physical Properties:

Solubilities:

0.000172 to 0.0080 mg/L at 25 degrees C (most values near 0.0038 mg/L) [848].

Almost insoluble in water [870].

Sol in benzene, toluene, xylene; sparingly sol in alcohol, methanol [The Merck Index. 10th ed. Rahway, New Jersey: Merck Co., Inc., 1983. 157] [366].

Sol in ether [Clayton, G. D. and F. E. Clayton (eds.). Patty's Industrial Hygiene and Toxicology: Volume 2A, 2B, 2C: Toxicology. 3rd ed. New York: John Wiley Sons, 1981-1982. 3346] [366].

Solubility in aqueous caffeine is higher than in water; also, native DNA has a solubilizing effect [IARC. Monographs on the Evaluation of the Carcinogenic Risk of Chemicals to Man. Geneva: World Health Organization, International Agency for Research on Cancer, 1972-1985. (Multivolume work).,p. V3 92 (1973)] [366].

Vapor Pressure [848]:

8.53x10(-10) to 2.53x10(-5) Pa at 25 degrees C (most values near 7x10(-7) Pa).

Boiling Point:

Boiling point: > 360 deg C at 760 mm Hg [Mackison, F. W., R. S. Stricoff, and L. J. Partridge, Jr. (eds.). NIOSH/OSHA - Occupational Health Guidelines for Chemical Hazards. DHHS(NIOSH) PublicationNo. 81-123 (3 VOLS). Washington, DC: U.S. Government Printing Office, Jan. 1981. 3] [366].

495 degrees C [848].

Melting Point [366]:

1. 179-179.3 DEG C [The Merck Index. 10th ed. Rahway, New Jersey: Merck Co., Inc., 1983. 157].

Molecular Weight [366]:

252.30 [The Merck Index. 10th ed. Rahway, New Jersey: Merck Co., Inc., 1983. 157].

Density/Specific Gravity [366]:

1.351 [IARC. Monographs on the Evaluation of the Carcinogenic Risk of Chemicals to Man. Geneva: World Health Organization, International Agency for Research on Cancer, 1972-1985. (Multivolume work).,p. V3 91 (1973)].

Octanol/Water Partition Coefficient, log Kow [848]:

4.05 to 8.50 (most near 6.1) [848].

Sorption Partition Coefficient, log Koc:

4.0 to 8.3 (most near 6.26) [848].

Reported Kocs: 3,950,00-5,830,000 [sic] experimental. Koc for binding to dissolved organic carbon in 3 natural waters, 18,000-52,000; Koc for binding to Aldrich humates, 890,000 [366].

Color/Form [366]:

Pale yellow monoclinic needles from benzene & methanol [Weast, R.C. (ed.). Handbook of Chemistry and Physics. 60th ed. Boca Raton, Florida: CRC Press Inc., 1979.,p. C-203].

Crystals may be monoclinic or orthorhombic [The Merck Index. 10th ed. Rahway, New Jersey: Merck Co., Inc., 1983. 157].

Yellowish plates (from benzene and ligroin) [Weast, R.C. (ed.) Handbook of Chemistry and Physics. 67th ed. Boca Raton, FL: CRC Press, Inc., 1986-87.,p. C-141].

Odor [366]:

Faint aromatic odor [Mackison, F. W., R. S. Stricoff, and L. J. Partridge, Jr. (eds.). NIOSH/OSHA - Occupational Health Guidelines for Chemical Hazards. DHHS(NIOSH) PublicationNo. 81-123 (3 VOLS). Washington, DC: U.S. Government Printing Office, Jan. 1981. 1].

Reference [734] reports that crude oil contains on the average

approximately 1% PNAs (a.k.a. PAHs) by weight and approximately 1 ppm B(a)P. The B(a)P concentration in crude oils from the Persian Gulf, Libya, and Venezuela were measured at 0.04, 1.3, and 1.6 ppm, respectively [734].

Concentrations of benzo(a)pyrene in South Louisiana crude, Kuwait crude, No. 2 fuel oil, and Bunker C residual were 0.75, 2.8, 0.6, and 44 mg/kg (ppm), respectively [177]. Another study showed concentrations of benzo(a)pyrene in South Louisiana crude and Kuwait crude were 1.2 and 2.8 10(-6)g/g oil (ppm), respectively [747].

Benzopyrene concentrations were determined for three different crude oil sample types taken from the Exxon Valdez oil spill. Concentrations in 1) unweathered oil from the tanker itself (March 1989), 2) oil skimmed from the water immediately after the spill and held in the skimmer barge for about 90 days (July 1989), and 3) weathered oil from Prince William Sound shorelines (May 1989) were: 12, ND (not detected), and 1 ug/g oil sampled, respectively [790; Reprinted with permission from Environmental Toxicology and Chemistry, Vol.14(11), W.A. Stubblefield, G.A. Hancock, W.H. Ford, and R.K. Ringer, "Acute and Subchronic Toxicity of Naturally Weathered Exxon Valdez Crude Oil in Mallards and Ferrets." Copyright 1995 SETAC].

Benzo(a)pyrene content in one fresh sample of NSFO (Fuel Oil 5, Chuck Rafkind, National Park Service, Personal Communication, 1996): 19.3 ng/mg (ppm)

Benzo(a)pyrene content in one sample of groundwater subjected to long term contamination of NSFO (Fuel Oil 5), possibly mixed with some JP-4, motorgas, and JP-8, Colonial National Historical Park Groundwater Site MW-10 (Chuck Rafkind, National Park Service, Personal Communication, 1996): 602.7 ng/L (ppt).

> NOTE: the above two PAH concentrations were analyzed by a GC/MS/SIM NOAA protocol [828] modified with methylene chloride extraction for use with water samples (Guy Denoux, Geochemical and Environmental Research Group, Texas A&M University, personal communication 1996).

Waste crankcase oil contains several toxic components including up to 30% aromatic hydrocarbons, with as much as 22 ppm benzo(a)pyrene (a PAH) [75]. Naphthalene, benzo(a)pyrene, fluorene, and phenanthrene are common PAH components of used motor oil [75].

Benzo(a)pyrene concentrations in fresh motor oil: 0.008-0.266 mg/kg [847].

Benzo(a)pyrene concentrations in Used Engine Oil: 15.0 ppm [519]; and 5.2-35.1 mg/kg [847].

Benzo(a)pyrene is found in gasoline in concentrations of 0.19 to 2.8 mg/kg (ppm) [796].

Benzo(a)pyrene content (mg/kg or ppm) in one fresh sample of Exxon Valdez Crude Oil [971]: 0 mg/kg = ppm

Fate.Detail: Detailed Information on Fate, Transport, Persistence, and/or Pathways:

Surface water half-lives of B(a)P were 0.37-1.1 hours (based on photolysis half-life in water) and 0.045 day (under mid-December sunlight) [848].

In general, volatilization half-lives from water surfaces are longer than 100 h for high-molecular-weight PAHs, such as benz(a)anthracene and benzo(a)pyrene, and shorter than 100 h for low-molecular-weight PAHs, such as naphthalene and anthracene. However, these numbers may vary depending upon surface wind velocity and turbulence [754,788].

Half-life in groundwater is 2,736 to 25,440 hours, based on estimated unacclimated aqueous aerobic biodegradation half-life [848].

Sorption of PAHs to soil and sediments increases with increasing organic carbon content and is also directly dependent on particle size. One researcher found from 3 to 4 times more anthracene and about 2 times more fluoranthene, benz(a)anthracene, and benzo(a)pyrene were retained by marsh sediment than by sand [788].

Values from the literature for half-lives in soil [848]: >2 days; 347 days for 5 mg/kg treatment, and 218 days for 50 mg/kg treatment; 309 days for Kidman sandy loam soils, and 229 days for Mclaurin sandy loam soils; 1,368 to 12,720 hours (based on aerobic soil dieaway test data at 10-30 degrees C); >50 days; 0.3 to >300 weeks; and 8.2 yrs.

One study of a field bioremediation site showed that all 2- and 3-ring PAHs were degraded to nondetect by day 280. The higher ring PAHs such as pyrene and benzo(a)pyrene continued to degrade over a three-year period [814].

Benzo(a)pyrene (B(a)P), benz(a)anthracene (B(a)A), and benzo(b)fluoranthene (B(b)F) are all carcinogenic polyaromatic hydrocarbons (CaPNAs) for which half-life estimates have been published. The primary fate mechanisms of these constituents is likely to be biodegradation. According to one source, 72% of B(a)P applied to soil remained after 16 months of incubation with bacteria. Based on first-order degradation, it is estimated that this would correspond to a half-life of approximately 1000 days for B(a)P. Another source estimated that the terrestrial half-life of B(a)P is approximately 290 days [734].

One study showed how biodegradation of PAHs was related to molecular weight. The 2- and 3-ring PAHs degraded rapidly. The 5-ring PAHs such as benzo(a)pyrene decreased slowly over a period of years [815].

Detailed Fate information from ATSDR (See ATSDR for embedded references) [881]:

> PAHs are absorbed through the lungs by transport across the mucus layer lining the bronchi (Bevan and Ulman 1991) [881]. In general, PAHs are lipophilic compounds that can cross the lungs through passive diffusion and partitioning into lipids and water of cells (Gerde et al [881]. 1991, 1993a, 1993b) [881]. The rapid, blood-bound redistribution of hydrocarbons at low blood concentrations from lungs to other organs indicates that diffusion is the rate-determining step (Gerde et al [881]. 1991) [881]. The absorption rates vary among the

PAHs, probably depending on the octanol/water partition coefficient [881]. Essentially all of gastrically instilled benzo[a]pyrene is absorbed via uptake of fat-soluble compounds (Busbee et al [881]. 1990) [881].

Oral absorption of benzo[a]pyrene is enhanced by some oils (such as corn oil) in the gastrointestinal tract [881]. The mechanism of dermal absorption of PAHs is most likely passive diffusion through the stratum corneum (Yang et al [881]. 1986) [881]. PAHs and their metabolites are distributed to tissues by transport through the blood [881]. Therefore, PAHs reach more-perfused tissues rapidly following exposure and are eliminated more slowly from lessperfused tissues (Bartosek et al [881]. 1984) [881]. A large fraction of orally absorbed benzo[a]pyrene is believed to be transported by lipoproteins from the gastrointestinal tract to the blood via the thoracic duct lymph flow (Busbee et al [881]. 1990) [881]. The carcinogenic mechanism of action of alternant PAHs is fairly well elucidated, but it is not as well described for nonalternant PAHs [881]. Furthermore, it is not known exactly how PAHs affect rapidly proliferating tissues [881]....

In order to assess whether there was any correlation between carcinogenic potency and the ability to induce P-450 isonezymes, several indices of P-450 isoenzyme activity (O -demethylation of ethoxyresorufin, metabolic activation of 2-amino-6-methyldipyrido [1,2-:3',2'd]imadazol [Glu-P-I] to mutagens, and immunological detection of polyclonal antibodies against purified rat P-450 I) were measured in microsomal preparations incubated with benzo[a]pyrene and benzo[e]pyrene (Ayrton et al [881]. 1990) [881]. While both PAHs increased several parameters of P-450-I activity, benzo[a]pyrene was markedly more potent than benzo[e]pyrene [881]. Based on these results, the authors concluded that the carcinogenic potency of the PAHs tested could be predicted by the degree to which they induced these enzymes [881]. Changes in the cytochrome P-450 system can affect the carcinogenicity of the PAHs [881]. This system is susceptible to induction by the PAHs themselves as well as other chemicals commonly found in the environment [881]. The degree and specificity (i.e., which enzymes are affected) of induction depend on the tissue and species and strain [881].... Alexandrov and Rojas-Moreno 1990) [881]. Furthermore, no benzo[a]pyrene-DNA-adducts were found in rat skin, which is known to be resistant to PAH-induced skin tumor formation (Alexandrov and Rojas-Moreno 1990) [881]. The types of adducts formed in various tissues may dictate target organ susceptibility to PAH-induced carcinogenicity [881]. Various metabolites of benzo[a]pyrene were administered to rats intraperitoneally and DNA adducts from lung, liver, and lymphocytes were measured (Ross et al [881]. 1991) [881]. The only metabolites that led to DNA binding were 2-, 9-, and 12-hydroxybenzo[a]pyrene and the trans -7,8-dihydrodiol of benzo[a]pyrene [881]. The authors suggested that different DNA adducts resulting from the in vivo metabolism of

benzo[a]pyrene in different tissues may be related to tissue specificity of benzo[a]pyrene-induced carcinogenicity [881]. Although the bulk of this work on PAH-induced carcinogenicity has been done in animal models and animal in vitro systems indicates that these same mechanisms of activation may be involved in humans [881]. For example, induction of AHH and formation of the reactive intermediate, benzo[a]pyrene 7,8-dihydrodiol, has been observed in the epithelial tissue from human hair follicles (Merk et al [881]. 1987) [881]. .... The effects of dermally applied benzo[a]pyrene alone or following dermal pretreatment with the prostaglandin synthetase inhibitor, indomethacin, on contact hypersensitivity (cell-mediated immunity), production of antibodies to DNP (humoral immunity), and the induction of skin tumors was studied in male BALBc mice treated for 6 weeks to 6 months (Andrews et al [881]. 1991b) [881]. A group of mice treated with acetone served as controls [881]. Skin tumors were observed in the mice treated with benzo[a]pyrene beginning at week 18 of treatment [881]. Pretreatment with indomethacin significantly increased (by 21%) the latency of tumor induction by benzo[a]pyrene and significantly reduced (by 46%) the weight of benzo[a]pyrene-induced skin tumors [881]. Based on these findings, the authors suggested that benzo[a]pyrene-induced skin carcinogenesis may be mediated by a mechanism that involves prostaglandin suppression of cellular immunity [881]. Undoubtedly, several other factors yet to be determined are involved in the ultimate expression of PAH-induced toxicity and carcinogenicity.

Metabolism summary from ATSDR (See ATSDR for embedded references) [881]:

The lipophilicity of PAHs enables them to readily penetrate cellular membranes and remain in the body indefinitely [881]. However, the metabolism of PAHs renders them more water-soluble and more excretable [881]. Metabolism of PAHs occurs in all tissues [881]. The metabolic process involves several possible pathways with varying degrees of enzyme activities [881]. The activities and affinities of the enzymes in a given tissue determine which metabolic route will prevail [881].

The metabolism of PAHs has been studied extensively in vitro and in vivo [881]. The most commonly used system is the rat liver microsomal fraction, although other species are also used [881]. Cells and cultured tissues from human and other animals have also significantly contributed to the elucidation of the PAH metabolic scheme [881].

The structural similarity of PAHs contributes to the similarities that exist in their biotransformation [881]. Benzo[a]pyrene metabolism has been extensively reviewed and will be used as a model for PAH metabolism [881]. In the many microsomal, cell, and cultured tissue preparations that have

been examined, the metabolic profiles are qualitatively similar [881]. However, there are differences in the relative levels and rates of formation of specific metabolites among tissues and cell preparations used from various animal species and strains [881]. These differences are susceptible to change as a result of pretreatment of the animals with either inducers or inhibitors of particular enzymes [881]. Furthermore, it is known that the metabolism of alternant PAHs (such as benzo[a]pyrene, benz[a]anthracene, chrysene, and dibenz[a,h]anthracene) differs from nonalternant PAHs (such as benzo[b]fluoranthene, benzo[k]fluoranthene, benzo[j]fluoranthene, and indeno[1,2,3-c,d]pyrene) [881]. Therefore, the metabolism of benzo[b]fluoranthene will also be discussed as a model for nonalternant PAH metabolism...

Benzo[a]pyrene is metabolized initially by the microsomal cytochrome P-450 systems to several arene oxides [881]. Once formed, these arene oxides may rearrange spontaneously to phenols, undergo hydration to the corresponding trans-dihydrodiols in a reaction catalyzed by microsomal epoxide hydrolase, or react covalently with glutathione, either spontaneously or in a reaction catalyzed by cytosolic glutathione-S-transferases (IARC 1983) [881]. Phenols may also be formed by the P-450 system by direct oxygen insertion, although unequivocal proof for this mechanism is lacking [881]. 6-Hydroxybenzo[a]pyrene is further oxidized either spontaneously or metabolically to the 1,6-, 3,6-, or 6,12-quinones [881]. This phenol is also a presumed intermediate in the oxidation of benzo[a]pyrene to the three quinones catalyzed by prostaglandin endoperoxide synthetase (Panthanickal and Marnett 1981) [881]. Evidence exists for the further oxidative metabolism to two additional phenols [881]. 3-Hydroxybenzopyrene is metabolized to the 3,6-quinone and 9-hydroxy-benzo[a]pyrene is further oxidized to the K-region 4,5-oxide, which is hydrated to the corresponding 4,5-dihydrodiol (4,5,9-triol) [881]. The phenols, quinones, and dihydrodiols can all be conjugated to glucuronides and sulfate esters; the quinones also form glutathione conjugates (Agarwal et al [881]. 1991; IARC 1983) [881].

In addition to being conjugated, dihydrodiols undergo further oxidative metabolism [881]. The cytochrome P-450 system metabolizes benzo[a]pyrene-4,5-dihydrodiol to a number of uncharacterized metabolites, while the 9,10-dihydrodiol is metabolized predominantly to its 1- and/or 3-phenol derivative with only minor quantities of a 9, 10-diol-7,8-epoxide being formed [881]. In contrast to the 9,10-diol, benzopyrene-7,8-diol is metabolized to a 7,8-dihydrodiol-9,10-epoxide, and phenol-diol formation is a relatively minor pathway [881]. The diol epoxides can be conjugated with glutathione either spontaneously or by a glutathione-S-transferase catalyzed reaction [881]. They may also hydrolyze spontaneously to tetrols (Hall and Grover 1988) [881].

Benzo[a]pyrene was metabolized in vitro by human bronchial epithelial and lung tissue to the 9,10-dihydrodiol, 7,8-dihydrodiol, and small quantities of the 4,5-dihydrodiol and 3- hydroxybenzo[a]pyrene, all of which are extractable into ethyl acetate (Autrup et al [881]. 1978; Cohen et al [881]. 1976; Kiefer et al [881]. 1988) [881]. These metabolites also conjugated with glutathione and sulfates, but none conjugated with glucuronide [881]. The rate of formation of the dihydrodiols was greater in the bronchial epithelium than in the lung (Autrup et al [881]. 1978; Cohen et al [881]. 1976) [881]. This may render some areas of the respiratory tract more sensitive to the effects of carcinogens [881]. One principal difference seen in human lung was the generation of a major ethyl acetate-soluble metabolite that was identified as the sulfate conjugate of 3-hydroxybenzo[a]pyrene, benzo[a]pyrene-3-yl-hydrogen sulfate [881]. This sulfate is very lipid soluble and, thus, would not be readily excreted in the urine (Cohen et al [881]. 1976) [881]. Activation of benzo[a]pyrene has also been detected in human fetal esophageal cell culture (Chakradeo et al [881]. 1993) [881].

Intratracheal instillation of benzo[a]pyrene to rats resulted in quinones constituting the highest concentration of metabolites in the lung and the liver within 5 minutes after instillation (Weyand and Bevan 1986, 1988) [881]. An in vitro study with rat lung demonstrated that the lung tissue has a high capacity to form quinones originating from oxidation at the six position of benzo[a]pyrene to form quinones and subsequently to water-soluble products [881]. Ozone exposure resulted in an increase in the metabolism of benzo[a]pyrene metabolites with the greatest increase observed in the formation of metabolites generated by oxidation at the six position [881]. The proposed retention of quinones following ozone exposure might lead to cytotoxicity associated with superoxide-anion generation by quinone-quinol redox-cycling [881]. However, the high levels of benzo[a]pyrene used in this in vitro study may not relate to what occurs in vivo [881]. Metabolism of benzo[a]pyrene at carbon six was higher at a lower dose than at the higher dose [881]. Therefore, quinone production and detoxification may represent a major pathway of lung PAH detoxification in vivo (Basett et al [881]. 1988) [881].

Approximately 50% of the benzo[a]pyrene that was intratracheally instilled in hamsters was metabolized in the nose (Dahl et al [881]. 1985) [881]. The metabolite produced in the hamster nose included tetrols, the 4,5-, 7,8-, and 9,10-dihydrodiol, quinones, and 3-and 9-hydroxybenzo[a]pyrene [881]. Similar metabolites were detected in nasal and lung tissues of rats inhaling benzo[a]pyrene (Wolff et al [881]. 1989b) [881]. The prevalence of quinone production was not seen in hamsters as it was in rats (Dahl et al [881]. 1985; Weyand and Bevan 1987a, 1988) [881]. In monkeys and dogs, dihydrodiols, phenols, quinones, and tetrols were identified

in the nasal mucus following nasal instillation of benzo[a]pyrene (Petridou-Fischer et al [881]. 1988) [881]. In vitro metabolism of benzo[a]pyrene in the ethmoid turbinates of dogs resulted in a prevalence of phenols (Bond et al [881]. 1988) [881]. However, small quantities of quinones and dihydrodiols were also identified [881]. Rat lung microsomes facilitated the dissociation of small amounts of benzo[a]pyrene from diesel particles, but only a small fraction of the amount dissociated was metabolized (Leung et al [881]. 1988) [881]. The ability to dissociate benzo[a]pyrene was related to the lipid content of the microsomal fraction [881]. Microsomes are able to enhance the slow dissociation of a small amount of benzo[a]pyrene from diesel particles in a form that can be metabolized [881]. Free benzo[a]pyrene was principally and extensively metabolized to the 9,10-dihydrodiol [881].

A human hepatoma cell line (HepG2) has high benzo[a]pyrene-metabolizing activity and converts benzo[a]pyrene to metabolites (Diamond et al [881]. 1980) [881]. When [3H]-benzo[a]pyrene was added to the incubate, a large fraction of the radioactivity was not extractable into chloroform [881]. The extractable fraction contained 9,10-dihydrodiols, 7,8-dihydrodiols, quinones, 3-hydroxybenzo[a]pyrene, and the unchanged parent compound [881]. The cell lysate also consisted of the same metabolites, but the proportions of 3-hydroxybenzo[a]pyrene and the parent compound were much higher than in the medium [881]. Conversely, the proportion of water-soluble metabolites in the cell lysate was lower than in the medium [881]. Treatment of the medium and cell lysate with -glucuronidase converted only 5-7% of the water-soluble metabolites to chloroform-extractable material [881]. Aryl sulfatase had no effect on radioactivity [881]. These results suggested that this human liver tumor cell line does not extensively utilize the phenol detoxification pathway (Diamond et al [881]. 1980) [881].

Metabolism of benzo[a]pyrene in the primary culture of human hepatocytes primarily resulted in the formation of 3-hydroxybenzo[a]pyrene, 4,5-dihydrodiol, 9,10-dihydrodiol, and 7,8-dihydrodiol (Monteith et al [881]. 1987) [881]. As the dose of benzo[a]pyrene increased, the amount of metabolites increased linearly [881]. Binding to DNA was associated with the amount of unconjugated 7,8-dihydrodiol [881]. DNA binding was linear up to a benzo[a]pyrene concentration of 100 mol [881]. At this concentration, binding increased 64-844 times over the extent of binding at 10 mol [881]. As the concentration of benzo[a]pyrene increased, the ratio of dihydrodiol/phenolic metabolites also increased [881]. Although the capacity to form dihydrodiols was not saturated at 100 mol benzo[a]pyrene, there was a change in the relative proportion of the dihydrodiol metabolites formed as the dose of benzo[a]pyrene increased [881]. As benzo[a]pyrene concentration increased, the 9,10-dihydrodiol was the more

prevalent metabolite, but levels of 7,8-dihydrodiol also increased (Monteith et al [881]. 1987) [881].

Epoxide hydrolase is a microsomal enzyme that converts alkene and arene oxides to dihydrodiols [881]. Appreciable enzyme activity was observed in human livers [881]. Comparison of epoxide hydrolase activities with various substrates revealed that the human liver has a single epoxide hydrolase with broad substrate specificity (Kapitulnik et al [881]. 1977) [881]. Epoxide hydrolase activity is also present in other tissues and increases the likelihood for carcinogenic effects in these organs [881]. Ethyl acetate extracts of human and rat bladder cultures contained 9,10-dihydrodiol, 7,8-dihydrodiol, and 3-hydroxybenzo[a]pyrene [881]. Covalent binding of [3H]-benzo[a]pyrene with DNA occurring in both human and rat bladder cultures suggested that benzo[a]pyrene-7,8-diol-9,10-epoxide is generated [881]. The urothelium of the bladder clearly has the ability to generate the ultimate carcinogen (Moore et al [881]. 1982) [881].

Hepatic microsomes from rats induced with 3-methylcholanthrene convert benzo[a]pyrene to benzo[a]pyrene-7,8-diol-9,10-epoxide (BPDE) 10 times faster than untreated microsomes [881]. The rate-limiting step in BPDE formation is the competition for P-450 between benzo[a]pyrene and the 7,8-dihydrodiol [881]. Formation of BPDE is directly correlated with the 3-methylcholanthrene inducible form(s) of P-450 (Keller et al [881]. 1987) [881]. Formation of the proximate carcinogen, 7,8-dihydrodiol, is stereoselective [881]. Rabbit hepatic microsomes generated more of the 7R,8R enantiomer with an optical purity of 90% (Hall and Grover 1987) [881]. The major stereoisomer formed by rat liver microsomes is (+)-diol-epoxide-2 (R,S,S,R absolute conformation) (Jerina et al [881]. 1976, 1980) [881]. This metabolite is highly tumorigenic (Levin et al [881]. 1982) and gives rise to the major adduct formed upon reaction with DNA [881]. The adduct is a diol epoxide-deoxyguanosine formed by alkylation at the exocyclic nitrogen (N-2) of deoxyguanosine [881]. This diol epoxide-deoxyguanosine has been isolated from several animal species (Autrup and Seremet 1986; Horton et al [881]. 1985) and human tissue preparations (Harris et al [881]. 1979) [881].

Studies using rat liver microsomes have shown that hydroxy metabolites of benzo[a]pyrene undergo glucuronidation (Mackenzie et al [881]. 1993) [881]. Assays with three different DNA-expressed glucuronidases from human liver indicate preferential glucuronidation for the 2- and 5-hydroxy, 4- and 11-hydroxy, or 1-, 2-, and 8-hydroxy derivatives of benzo[a]pyrene [881]. There are differences in preferential activities for the glucuronidation of various benzo[a]pyrene metabolites among the various DNA-expressed glucuronidases from human liver, with some glucuronidases being relatively or totally inactive toward this class of compounds (Jin et al [881]. 1993) [881]. The results of this

study suggest that the relative content of particular types of glucuronidases in a cell or tissue may be important for determining the extent to which a particular carcinogen is deactivated [881]. Several xenobiotics can induce enzymes to influence the rat liver microsomal metabolite profiles of various PAHs [881]. For example, AHH, the cytochrome P-450 isoenzyme believed to be primarily responsible for the metabolism of benzo[a]pyrene and other PAHs, is subject to induction by PAHs [881]. Treatment of pregnant and lactating rats with a single intraperitoneal dose of Aroclor 1245 increased the metabolism of benzo[a]pyrene by liver microsomes from pregnant and fetal rats 9-fold and 2-fold, respectively, and 2-fold in lactating rats (Borlakoglu et al [881]. 1993) [881]. The pretreatment enhanced the formation of all metabolites, but the ratio of the 7,8-diol (the proximate carcinogen) was increased 3-fold in lactating rats and 5-fold in pregnant rats [881]. Similar results were observed in rabbit lung microsomes (Ueng and Alvares 1993) [881]. Cigarette smoke exposure has been shown to increase PAH metabolism in human placental tissue (Sanyal et al [881]. 1993), and in rat liver microsomes (Kawamoto et al [881]. 1993) [881]. In studying benz[a]anthracene metabolism, some xenobiotics were found to be weak or moderate inducers, but even less efficient ones altered the benz[a]anthracene profile significantly [881]. Thiophenes equally enhanced oxidation at the 5,6- and the 8,9-positions [881]. Benzacridines favored K-region oxidation (5,6-oxidation) (Jacob et al [881]. 1983b) [881]. Indeno[1,2,3-c,d]pyrene stimulated the bay region oxidation (3, 4-oxidation) of benz[a]anthracene (Jacob et al [881]. 1985) [881]. Similar xenobiotic effects were observed with chrysene as a substrate (Jacob et al [881]. 1987) [881]. While some enzyme activities are being enhanced, alternate enzymatic pathways may be suppressed (Jacob et al [881]. 1983a) [881].

Rat liver microsomes also catalyzed benzo[a]pyrene metabolism in cumene hydroperoxide (CHP)-dependent reactions which ultimately produced 3-hydroxybenzo[a]pyrene and benzo[a]pyrene-quinones (Cavalieri et al [881]. 1987) [881]. At low CHP concentrations, 3-hydroxybenzo[a]pyrene was the major metabolite [881]. As CHP concentrations increased, levels of quinones increased and levels of 3-hydroxybenzo[a]pyrene decreased [881]. This effect of varying CHP levels was reversed by preincubating with pyrene [881]. Pyrene inhibited quinone production and increased 3-hydroxybenzo[a]pyrene production [881]. Pretreatment with other PAHs like naphthalene, phenanthrene, and benz[a]anthracene nonspecifically inhibited the overall metabolism [881]. The binding of benzo[a]pyrene to microsomal proteins correlated with quinone formation [881]. This suggested that a reactive intermediate was a common precursor [881]. The effects of pyrene on benzo[a]pyrene metabolism indicated that two distinct microsomal binding sites were responsible for the formation of 3-hydroxybenzo[a]pyrene and

benzo[a]pyrene-quinone (Cavalieri et al [881]. 1987) [881].

Rat mammary epithelial cells (RMEC) have been shown to activate PAHs (Christou et al [881]. 1987) [881]. Cytochrome-P-450 in RMEC is responsible for the monooxygenation of DMBA [881]. Prior exposure of cultured cells to benz[a]anthracene induced DMBA metabolism [881]. The metabolite profile following benz[a]anthracene-induction was significantly different from the profile obtained with purified P-450c, the predominant PAH-inducible enzyme in rat river [881]. The bay region 3,4-dihydrodiol, which was not formed with P-450c, was clearly detectable in RMEC [881]. Low epoxide hydrolase activity in the benz[a]anthracene-induced RMEC limited the formation of all other DMBA dihydrodiols [881]. The DMBA monooxygenase activity of benz[a]anthracene-induced RMEC limited the formation by -naphthaflavone [881]. The study concluded that DMBA metabolism by RMEC depended on the induction of P-450c and at least one additional form of P-450 that is sensitive to -naphthaflavone (Christou et al [881]. 1987) [881].

As expected from results of other studies, the perfused rat lung can release high quantities of benzo[a]pyrene metabolites and conjugates into the perfusate (Molliere et al [881]. 1987) [881]. Addition of a liver to this perfusion system up gradient from the lungs reduces the concentration of parent compound and free metabolites to less than 20% of that seen in the liver's absence [881]. The liver provides a protective effect on the lung to inhibit covalent binding of benzo[a]pyrene metabolites to pulmonary macromolecules [881].

The effects of various factors that can modify the hepatic clearance of PAHs, specifically benz[a]anthracene and chrysene, were studied by Fiume et al [881]. (1983) [881]. The hepatic clearance and rate constants of these PAHs were significantly reduced in the perfused livers of fasted rats relative to those of fed rats [881]. This reduction was attributed to a decrease in aryl hydrocarbon hydroxylase activity [881]. Fasting also accelerated the depletion of cytochrome P-450 and other microsomal enzymes [881]. In contrast, pretreatment of the rats with these PAHs resulted in increased clearance of both hydrocarbons from the perfusion medium when compared to control rats [881].

It was also noted by Fiume et al [881]. (1983) that the livers of male rats demonstrated a significantly higher hepatic clearance of benz[a]anthracene than female rats, perhaps suggesting a sexual difference with aryl hydrocarbon hydroxylase activity [881]. Similar findings regarding sexual differences in the metabolism of chrysene by rat livers were also reported by Jacob et al [881]. (1985, 1987) [881]. Furthermore, Fiume et al [881]. (1983) demonstrated that age can play a role in PAH metabolism [881]. The hepatic clearance of PAHs in older rats (2 years) was significantly less than

the hepatic clearance in younger rats (8 weeks) [881]. However, activation of benzo[a]pyrene to mutagenic derivatives, as measured by the Salmonella typhimurium test, with hepatic microsomes from male rats from 3 weeks to 18 months of age showed no age-dependent changes (Hilali et al [881]. 1993) [881]. Nonalternant PAHs, in contrast to several alternant PAHs, do not appear to exert their genotoxic effect primarily through the metabolic formation of simple dihydrodiol epoxides [881]. In the case of benzo[b]fluoranthene, there is evidence to suggest that metabolism to the dihydrodiol precursor to its bay-region dihydrodiol does occur [881]. Rather than this metabolite being converted to its dihydrodiol epoxide; however, it appears to be extensively converted to its 5- hydroxy derivative [881]. It is the further metabolism of this phenolic dihydrodiol to 5,9, 10-trihydro y-11,12-epoxy-9,10,11,12-tetrahydrobenzo[b]fluoranthene that has been linked to the genotoxic activity of benzo[b]fluoranthene in mouse skin (Weyand et al [881]. 1993b) [881]. In the case of benzo[j]fluoranthene, two potentially genotoxic metabolites have been identified [881]. These are the trans -4, 5- and 9,10-dihydrodiols of benzo[j]fluoranthene [881]. It is the conversion of trans - 4,5-dihydro-4,5-dihydroxybenzo[j]fluoranthene to anti -4,5-dihydroxy-5,6a-epoxy-4,5,6,6a-tetrahydrobenzo[j]fluoranthene that is principally associated with DNA adduct formation in mouse skin (La Voie et al [881]. 1993b; Weyand et al [881]. 1993a) [881]. Benzo[k]fluoranthene in rat microsomes was shown to result in the formation of 8,9-dihydrodiol [881]. This dihydrodiol can form a dihydrodiol epoxide that is not within a bay region [881]. This may represent an activation pathway of benzo[k]fluoranthene that may be associated, in part, with its genotoxic activity [881]. In the case of nonalternant PAHs, reactive metabolites, that deviate from classical bay region dihydrodiol epoxides, have been linked to their tumorigenic activity [881].

Misc. Information on Environmental Fate from the HSDB (see HSDB for more detail) [366]:

> TERRESTRIAL FATE: If benzo(a)pyrene is released to soil it will be expected to adsorb very strongly and will not be expected to leach to the groundwater; however, its presence in some groundwater samples indicates that it can be transported there by some mechanism. It will not hydrolyze, and evaporation from soils and surfaces is not expected to be significant. Biodegradation tests in soils have resulted in a wide range of reported half-lives: 2 days to 1.9 yr; based on these values and the apparent lack of a significant competing fate process, biodegradation may be an important process in soils. (SRC)
>
> AQUATIC FATE: If released to water, benzo(a)pyrene (BaP)

will be expected to adsorb very strongly to sediments and particulate matter. It will not hydrolyze but will be expected to bioconcentrate in aquatic organisms that can not metabolize it. It has been shown to be susceptible to significant metabolism by microorganisms in some natural waters without use as carbon or energy source, but in most waters and in sediments it has been shown to be stable towards biodegradation. B(a)P will be expected to undergo significant photodegradation near the surface of waters. Evaporation may be significant with a predicted half-life of 43 days for volatilization from a river 1 m deep, flowing at 1 m/sec with a wind velocity of 3 m/sec. Adsorption to sediments and particulates may significantly retard biodegradation, photodegradation, and evaporation. (SRC)

ATMOSPHERIC FATE: Benzo(a)pyrene (BaP) released to the atmosphere will likely be associated with particulate matter and may be subject to moderately long transport, depending mainly on the particle size distribution and climactic conditions which will determine the rates of wet and dry deposition. Its presence in areas remote from primary sources demonstrates the potential for this long range transport as well as B(a)P's considerable stability in the air. A half-life of 1.4 years has been reported for removal of B(a)P from the gas phase by rainout and has a lifetime of 7.9 days for removal by aerosol particles(1). It may be subject to direct photodegradation but evidence suggests that this process is retarded by the material being in the adsorbed state. Half-life for reaction of a thin film of B(a)P with 0.19 ppm O3 is 37 min and for reaction of adsorbed B(a)P with NO2 is 7 days. The estimated half-life for reaction with photochemically produced hydroxyl radicals is 21.49 hr. [(1) Cupitt LT; Fate of Toxic Hazards in the Air Environment USEPA-600/3-80-084 (1980].

Biodegredation [366]:

In natural waters 61.7% benzo(a)pyrene (BaP) cometabolized in 4 weeks when phenanthrene was used as a carbon source, 16.5% cometabolism with naphthalene as growth substrate(1). No degradation in 24 hr in water from estuarine Skidaway River in 24 hr in Jan and June; half-life of 3500 days for biodegradation was reported after 96 days in June(2). Degradation of 56-67% B(a)P was observed in nutrient/B(a)P solutions seeded with sewage and incubated for 4-7 day periods, with the last 3 seeds derived from the previous incubation period(3). Half-life for microbial transformation in water from a stream 0.5 km below coke effluent discharge, >1400 hr(4). No degradation to CO2 observed in seawater at 12 deg C in the dark after 48 hr; half-life 24 hr after addition of water extract of fuel oil, 970 days(5). No degradation

was observed in enrichment culture studies using natural waters for up to 6 weeks as well as with effluents from waste treatment plants and industrial effluents(6). Half-lives for degradation in sediments: oil contaminated stream, 2.4 yr, uncontaminated stream, 39.6 yr(7); stream 0.5 km below coke effluent discharge, 1300 hr, stream 0.2 km below petroleum storage depot, >20,000 hrs, uncontaminated stream, >20,000 hr(4). Soil biodegradation half-lives (days): <15 deg C, 5 values, 37-694, avg 251; 15-25 deg C, 7 values, 30-420, avg 198; >25 deg C, 8 values, 2-7, avg 4.3; also reported for > 25 deg C range, 39 days for an initial conc of 9,100 ppm(8). [(1) McKenna EJ; Biodegradation of Polynuclear Aromatic Hydrocarbon Pollutants by Soil and Water Microorganisms. Water Resources Center 113: 1-25 (1976) (2) Lee RF; pp.611-6 in Proc of the 1977 Oil Spill Conf Amer Petroleum Inst (1977) (3) Fochtman EG; Biodegradation and Carbon Adsorption of Carcinogenic and Hazardous Organic Compounds USEPA-600/S2-81-032 (1981) (4) Herbes SE et al; pp.113-28 in The Scientific Basis of Toxicity Assessment. Witshih H ed (1980) (5) Verschueren K; Handbook of Environmental Data on Organic Chemicals. 2nd ed Von Nostrand Reinhold NY pp.270-1 (1983) (6) Smith JH et al; Environmental Pathways of Selected Chemicals in Freshwater Systems: Part II. Laboratory Studies USEPA-600/7-78-074 (1978) (7) Herbes SE, Schwall LR; Appl Environ Microbiol 35: 306-16 (1978) (8) Sims RC, Overcash MR; Res Rev 88: 1-68 (1983)].

Abiotic Degredation [366]:

Data for the oxidation rates of benzo(a)pyrene coated on quartz surface & exposed to ozone or sunlight are presented. From about eight products detected in these experiments, three have been identified as quinones based on uv-absorption spectrometry & mass spectrometry. [Rajagopalan R et al; Sci total environ 27 (1): 33-42 (1983)].

Polyaromatic hydrocarbons do not contain hydrolyzable groups and would therefore not be expected to hydrolyze(1). Benzo(a)pyrene (BaP) absorbs light at >290 nm and may, therefore, undergo direct photolysis(1); calculated half-life for photolysis in surface waters in midsummer at 40 deg North is 0.54 hr(2); however, since B(a)P occurs mainly adsorbed to particulate matter in the environment, data concerning B(a)P in the pure state may not be relevant(3), but may be relevant to spill situations. It has been reported that adsorption of B(a)P on kaolinite clay inhibits photolysis(4) and B(a)P adsorbed on fly ash showed 17% degradation when exposed to sunlight for 3.8 hr(5). Half-lives for B(a)P in various waters with 1% acetonitrile added and irradiated at 366 nm: pure water, 0.44 hr, pure water with humic

acid, 2.8 hr, Lake Tahoe water, 1.1 hr, Searsville Pond water, 0.48 hr, Coyote Creek water, 1.1 hr(6). Half-life for thin film of B(a)P exposed to 0.19 ppm O3, 37 min(7); half-life for reaction of adsorbed B(a)P with NO2, 7 days(9). The estimated half-life for reaction of B(a)P with photochemically produced radicals hydroxyl radicals is 21.49 hr(10). air in light, 22% (48 hr), smog in light, 50% (1 hr); B(a)P adsorbed on soot, air in light, 10% (48 hr), smog, 18% (1 hr)(8). [(1) Callahan MA et al; p.98-1 to 98-27 in Water-Related Environmental Fate of 129 Priority Pollutants Vol 2 USEPA-440/4-79-029b (1979) (2) Herbes SE et al; pp.113-28 in The Scientific Basis of Toxicity Assessment; Witschi H, ed Elsevier/North-Holland Biomedical Press (1980) (3) Bjorseth A, Olufsen BS; pp.507-24 in Handbook of Aromatic Hydrocarbons; Bjorseth A, ed (1981) (4) Sims RC, Overcash MR; Res Rev 88: 1-68 (1983) (5) Korfmacher WA et al; Environ Sci Technol 14: 1094-9 (1980) (6) Smith JH et al; Environmental Pathways of Selected Chemicals in Freshwater Systems: Part II. Laboratory Studies USEPA-600/7-78-074 (1978) (7) Lane DA, Morris K; Adv Environ Sci Technol 8: 137-54 (1977) (8) Syracuse Research Corporation; Hazard Assessment on Polycyclic Organic Mater. Syracuse Research Corporation pp 249 TR69-115 (1980) (9) National Research Council; Polycyclic Aromatic Hydrocarbons: Evaluation of Sources and Effects. National Acad Press Washington, DC (1983) (10) GEMS; Graphical Exposure Modeling System. Fate of Atmospheric Pollutants (FAP) Data Base. Office of Toxic Substances. USEPA (1986)].

Exposure of benzo(a)pyrene adsorbed on calcite to white fluorescent light. T 1/2 is dependent on the production of oxygen. T 1/2: 11 hr in an atmosphere of oxygen. /From table/ [Castegnaro, M., G. Grimmer, O. Hutzinger, W. Karcher, H. Kunte, M. LaFontaine, E.B. Sansone, G. Telling, and S.P. Tucker (eds.). Laboratory Decontamination and Destruction of Carcinogens in Laboratory Wastes: Some Polycyclic Aromatic Hydrocarbons. IARC Publications No. 49. Lyon, France: International Agency for Research on Cancer, 1983. 47].

Soil Adsorption/Mobility [366]:

Reported Koc: 3,950,00-5,830,000 experimental(1,2). Koc for binding to dissolved organic carbon in 3 natural waters, 18,000-52,000; Koc for binding to Aldrich humates, 890,000(3). [(1) Sims RC, Overcash MR; Res Rev 88: 1-68 (1983) (2) Smith JH et al; Environmental Pathways of Selected Chemicals in Freshwater Systems: Part II. Laboratory Studies USEPA-600/7-78-074 (1978) (3) Landrum PF et al; Environ Sci Technol 18: 187-92 (1984)].

Volatilization from Water/Soil [366]:

The reported estimated theoretical maximum half-life for volatilization from a model river 1 m deep, flowing at 1 m/sec with a wind velocity of 4 m/sec is 18 days; physical factors, such as adsorption, which will slow volatilization, were not considered in this estimation and it was concluded that vaporization will be insignificant under all conditions(1). Predicted half-life for volatilization from a model river 1 m deep, flowing at 1 m/sec with a wind velocity of 3 m/sec is 43 days(3,SRC), qualified by adsorption. Volatilization half-life (hr) predicted by the one-compartment model: 140 (river), 350 lake); measured half-life in a rapidly stirred aqueous solution was 22 hr; benzo(a)pyrene/O2 reaeration rate ratio, 0.0036(2). [(1) Southworth GR et al; Bull Environ Contam Toxicol 21: 507-14 (1979) (2) Smith JH et al; Environmental Pathways of Selected Chemicals in Freshwater Systems: Part II. Laboratory Studies USEPA-600/7-78-074 (1978) (3) Lyman WJ et al; Handbook of Chemical Property Estimation Methods. Environmental Behavior of Organic Compounds. McGraw-Hill NY p 15-1 to 15-34 (1982)].

Biological Half-Life [366]:

... /In mice/ hydrocarbon/deoxyribonucleoside adduct showed approx parallel dose-response curves. The half-life of the b(a)p/deoxyribonucleoside adducts & the total radioactivity bound to the DNA were 4.5 & 5.5 Days. ... [Ashurst SW et al; Cancer res 43 (3): 1024-9 (1983)].

Absorption, Distribution and Excretion [366]:

... Readily absorbed from the intestinal tract & tend to localize primarily in body fat & fatty tissues such as breast. Disappearance of B(a)P from blood & liver of rats following single IV injection is very rapid, having a half-life in blood of less than 5 min & a half-life in liver of 10 min. In ... blood & liver ... initial rapid elimination phase is followed by slower disappearance phase, lasting 6 hr or more. ... A rapid equilibrium is established between B(a)P in blood & that in liver & ... the cmpd fast disappearance from blood is due to ... metabolism & distribution in tissues. [National Research Council. Drinking Water & Health, Volume 4. Washington, DC: National Academy Press, 1981. 257].

B(a)P crosses the placenta in mice & rats ... [IARC. Monographs on the Evaluation of the Carcinogenic Risk of Chemicals to Man. Geneva: World Health Organization, International Agency for Research on Cancer, 1972-1985. (Multivolume work).,p. V32 214 (1983)].

(14)C metabolites were secreted into bile of rats within 7 min of iv dose of (14)c-benzo(a)pyrene. Pretreatment

of animals with this carcinogen ... enhanced biliary secretion of (14)C. [The Chemical Society. Foreign Compound Metabolism in Mammals. Volume 2: A Review of the Literature Published Between 1970 and 1971. London: The Chemical Society, 1972. 153].

Male rats cannulated in the bile duct received iv injections of radiolabeled benzo(a)pyrene noncovalently bound to the very-low-density, low-density, or high-density lipoproteins in equimolar amounts. Cumulative biliary excretions of benzo(a)pyrene complexed with rat lipoproteins were 39.6, 24.6, & 21.2% For very-low density, low-density, & high-density lipoprotein, respectively. Values for excretion of benzo(a)pyrene complexed with rat or human lipoproteins were comparable. Excretion increased as the degree of b(a)p hydroxylation increased. The excretion of b(a)p bound to very-low-density, low-density, or high-density lipoproteins in aroclor-induced rats was not greater than the control. Hence, 60-80% of injected b(a)p & 50-60% of injected b(a)p metabolites were not excreted immediately in control or induced animals. This b(a)p may represent a carcinogen pool that is slowly excreted. [Shu HP, Bymun EN; Cancer res 43 (2): 485-90 (1983)].

Benzo(a)pyrene metabolism & macromolecular binding were studied in explants from 4 tissues (bladder, skin, bronchus, & esophagus) from 8 human donors sampled within 4 hr after death. Explants were incubated with b(a)p for 24 hr, then metabolites extracted & analyzed. Fibroblasts were grown from explants from 2 patients & also incubated with b(a)p. Metabolite profiles were qualitatively the same for explants & fibroblasts with similar product ratios, although fibroblasts were less active in B(a)P metabolism. Dna binding studies showed a broad variance among patients & tissues with the relative distribution being the widest in bladder, followed by skin, bronchus, & esophagus, respectively. [Selkirk JK et al; Cancer lett (Shannon, IREL) 18 (1): 11-9 (1983)].

...

The uptake and distribution of ... benzo(a)pyrene in northern pike (Esox lucius) were investigated by whole body autoradiography and scintillation counting. (3)H-Benzo(a)pyrene was administered in the diet or in the water. The levels of this cmpd employed corresponded to levels found in moderately polluted water. The uptake and distribution of this cmpd and its metabolites were followed from 10 hr to 21 days after the initial exposure. The autoradiography patterns observed with both routes of administration suggested that benzo(a)pyrene was taken up through the gastrointestinal

system and the gills, metabolized in the liver and excreted in the urine and bile. The gills may not have been a major route of excretion for benzo(a)pyrene and its metabolites in the northern pike. Benzo(a)pyrene may have been taken up from the water directly into the skin of this fish. Benzo(a)pyrene and its metabolites were heterogeneously distributed in the kidney of the northern pike. Little radioactivity accumulated in the adipose tissue. With scintillation counting, uptake of radioactivity from the water occurred rapidly in all organs, reaching a plateau in most cases after about 0.8 days. The concentrations of radioactivity in different organs ranged between 50 (many organs) and 80,000 (gallbladder + bile)-fold that found in the surrounding water. Since most of the radioactivity recovered in different organs of the pike after 8.5 days of exposure was in the form of metabolites, metabolism may have played an important role in the bioconcentration of xenobiotics in fish. [Balk L et al; Toxicol Appl Pharmacol 74 (3): 430-49 (1984)].

Polynuclear aromatic hydrocarbons (PAH), some of which are potent carcinogens, are common environmental pollutants. The transport processes for these hydrophobic compounds into cells and between intracellular membranes are diverse and are not well understood. A common mechanism of transport is by spontaneous desorption and transfer through the aqueous phase. From the partitioning parameters, we have inferred that the rate limiting step involves solvation of the transfer species in the interfacial water at the phospholipid surface. Transfer of 10 PAH ... out of phosphatidylcholine vesicles has been examined. ... Results show that the molecular volume of the PAH is a rate-determining factor. Moreover, high performance liquid chromatography (HPLC) data confirms the hypothesis that the rate of transfer is correlated with the size of the molecule and with the partitioning of the molecule between a polar and hydrocarbon phase. The kinetics and characteristics of the spontaneous transfer of carcinogens are likely to have a major impact on the competitive processes of PAH metabolism within cells. [Plant AL et al; Chem-Biol Interact 44 (3): 237-46 (1983)].

Poeciliopsis lucida and Poeciliopsis monacha are freshwater viviparous fishes susceptible to tumorigenesis by exposure to waterborne procarcinogens. The hepatic monooxygenase system and the uptake, toxicity, and distribution of benzo(a)pyrene (BP) were characterized as a first step in exploring relationships between xenobiotic metabolism and cancer in these fishes. Waterborne BP was lethal at a dose of 3.75 mg/l with a 24 hr exposure. During a 24 hr exposure to 1.0 mg/l (3.97

umol/l) BP, an average of 8.27 nmol of BP was taken up per fish. Of this total, 64-70% was in the gallbladder or gut, indicating rapid metabolism and excretion. Basal levels of aryl hydrocarbon hydroxylase (AHH) activity were fairly high, about 0.6 nmol/min/mg. Maximal induction by BP occurred at a dose of 1.0 mg/l, but with aryl hydorcarbon hydroxylase activities only about twice the levels in untreated fish. Sensitivity to inhibition by alpha-naphthoflavone increased slightly in treated fish. Induced aryl hydrocarbon hydroxylase and also 7-ethoxyresorufin O-deethylase activities declined slowly after a single treatment, approaching pre-exposure levels after 7 days. ... [Kathryn A et al; Am Soc Pharm Exp Ther 15 (4): 449 (1987)].

Comparison of disposition of benzo(a)pyrene (BaP) among Sprague-Dawley rats, Gunn rats, hamsters, and guinea pigs was performed. B(a)P was administered intratracheally to animals, and the rate of excretion of radioactivity into bile, types of metabolites of B(a)P in bile, and distribution of radioactivity were qualitatively similar among these species although quantitative differences were observed. In hamsters, the rate of excretion was essentially independent of dose at the concentrations examined (0.16 and 350 ug). The major difference between hamsters and the other species was that increased amounts of radioactivity were retained in lung of hamsters at the lower dose with a proportional decrease in the amount of radioactivity excreted into bile. The types and relative amounts of conjugated and nonconjugated metabolites of B(a)P were similar in bile of Sprague-Dawley rats and hamsters. Smaller amounts of glucuronides and larger amounts of sulfate conjugates were detected in bile of Gunn rats than in bile of Sprague-Dawley rats or hamsters. Metabolites in bile of guinea pigs were markedly different from those in the other species in that approximately 90% of the metabolites were thioether conjugates. ... [Weyland EH, Bevan DR; Am Soc Pharm Exp Ther 15 (4): 442 (1987)].

In an attempt to understand route of administration dependency, 3H-benzo(a)pyrene, (14)C-urethane and (14)C-acrylamide were administered as single doses orally or topically to male SENCAR mice. Distribution in skin, stomach, liver, and lung was determined for time periods up to 48 hr. The binding of these compounds to DNA, RNA, and protein in these tissues was determined 6 and 48 hr after administration. For all three compounds, high concentrations were found in the skin following topical application, but very little material reached this target organ following oral administration. The internal organs generally contained more material after oral administration compared to topical application, whereas the opposite was true for the skin. Differences in

distribution to the skin and binding to macromolecules following oral or topical administration cannot explain the greater tumorigenicity of urethane and acrylamide after oral administration in the SENCAR mouse. [Carlson GP et al; Environ Health Perspect 68: 53-60 (1986)].

Other HSDB Notes on fate-related pharmacokinetics [366]:

Serum was taken from fasting, male Sprague-Dawley rats and the uptake of (14)C-benzo(a)pyrene (B(a)P) and subsequent extraction of bound B(a)P was determined by radio-scintillation techniques. The initial uptake velocity for B(a)P was similar to human serum for all concentrations of B(a)P used. Maximum uptake of B(a)P was estimated at 120 ug/ml for rat serum. In rat serum, low-density lipoproteins contained 50% of the bound B(a)P, high- density lipoproteins contained 40%, and albumin contained 10% of the total B(a)P added. In rat serum, the HDL component contained 40-50% of B(a)P, and the LDL component contained 19-22% of the added B(a)P. After removal of the lipoproteins from the serum, bound B(a)P was associated entirely with albumin. [Aaarstad K et al; Toxicology 47 (3): 235-45 (1987)].

Lobster (Homarus americanus) in Maine and the spiny lobster (Panulirus argus) in Florida were selected as exptl invertebrate models for studies of the disposition and metabolism of xenobiotics. Hepatopancreas microsomes from both species contained relatively high amounts of cytochrome p450 (> = 1 nmol/mg protein), but NADPH-dependent monooxygenase activity was very low or undetectable with several substrates, including benzo(a)pyrene. NADPH-dependent cytochrome c reductase. Activity was also very low in these microsomes. (14)C-Benzo(a)pyrene (1 mg/kg) was metabolized and excreted very slowly after intracardial administration to lobsters. The half-life for disappearance of radiolabel was approximately 2 mo, and most of the radioactivity was stored in the hepatopancreas. Similar studies in the spiny lobster demonstrated that metab and excretion were considerably faster in this species (half-life approximately 1 wk in the summer and approximately 2 wk in the winter). In reconstituted monooxygenase systems containing spiny lobster hepatopancreas cytochrome p450, benzo(a)pyrene was metabolized primarily to phenolic products (approximately 50% of the total metabolites). Benzo(a)-metabolites, benzo(a)pyrene 4,5-, 7,8-, and 9,10-benzo(a)pyrene-dihydrodiol were formed in approximately equal amounts and accounted for approximately 20% of the total metabolites. Collectively, these results are consistent with the production of carcinogenic metabolites of benzo(a)pyrene in vivo by the marine crustacean species examined. [Bend JR et al; Proc Int Symp Princess Takamatsu Cancer Res

Fund 11: 179-94 (1981)].

Metabolism [366]:

The facial selectivity of purified rat liver cytochrome p-450c toward epoxidation of benzo(a)pyrene (bp) at its 4,5-position (k-region) was determined by formation, separation, & quantitation of diastereomeric trans-addition products of glutathione with enzymatically produced benzo(a)pyrene 4,5-oxide. Correlation of the glutathione conjugates obtained from bp 4,5-oxide derived from cytochrome p-450c catalyzed oxidation of bp with those obtained from the synthetic enantiomers indicated that 97% of the enzymatically formed arene oxide was the (+)-(4s,5r) enantiomer. [Armstrong RN et al; Biochem biophys res commun 100 (3): 1077-84 (1981)].

Human liver microsomal fractions from 13 different individuals were characterized with respect to SDS (sodium dodecylsulfate)- polyacrylamide gel electrophoretic profiles & regional specificity in the metabolism of the polyaromatic hydrocarbon benzo(a)pyrene. Pronounced interindividual differences in the composition of microsomal proteins in the molecular wt range of 49,000-60,000 were found. Most of the variations among profiles of microsomal proteins are interindividual differences in the composition of isoenzymes of cytochrome p-450. Large variations among the human liver microsomal samples were also seen in benzo(a)pyrene metabolism. The results indicate the presence of 7-8 different forms of cytochrome p-450 in human liver microsomes & interindividual variations seen in drug metabolism may at least in part be explained by variations in the distribution of these isoenzymes. [Ekstroem G et al; Acta pharmacol toxicol 50 (4): 251-60 (1982)].

The ability of three purified forms of rat liver cytochrome p-450 to metabolically activate benzo(a)pyrene to mutagenic products was examined using salmonella typhimurium strains TA98 & g46 in a reconstituted monooxygenase system. The isozymes examined were cytochrome P450-PB (the major phenobarbital inducible form), & the two major 3-mc inducible forms (cytochromes P-448(52,000 mol wt) & P-448(55,000 mol wt)). Only cytochrome P-448(55) metabolizes benzo(a)pyrene & its 7,8-dihydrodiol derivative to mutagenic products. [Roberstson IG et al; Carcinogenesis 4 (1): 93-6 (1983)].

Colonic biopsy specimens from patients with ulcerative colitis & normal subjects were studied for the ability to metabolize benzo(a)pyrene. Approx 73% of 30 colonic biopsy specimens from 7 ulcerative colitis patients could metabolize benzo(a)pyrene to oxidized products, with an

average production of 11.6 Nmol/mg biopsy protein. In contrast, 39% of 23 biopsy specimens from 5 normal persons showed an average metabolic activity, 2.79 Nmol. Benzo(a)pyrene oxidation activity in colonic tissue from colitis patients was, on the average, fourfold greater than that in normal subjects. This study suggest that the colonic mucosa of patients with ulcerative colitis has a greater ability than that of normal subjects to oxidize such chemicals possibly to electrophiles with higher mutagenic potential. [Mayhew JW et al; Gastroenterology 85 (2): 328-34 (1983)].

Laboratory and/or Field Analyses:

Recommended detection limits:

Most of the PAH methods which have been commonly used historically for routine monitoring, including PAH parent compound standard methods:

EPA 8270 (8270 includes several PAH parent compounds along with a long list of other organics) for solid waste/RCRA applications [1013], and

EPA NPDES method 610 as specified in 40 CFR Part 136 (method 610 includes 16 PAH parent compounds) [1010],

EPA method 625 for Base/Neutral Extractables (method 625 includes several PAH parent compounds along with a long list of other organics) as specified in 40 CFR Part 136 [1010],

are all inadequate for generating scientifically defensible information for Natural Resource Damage Assessments [468]. These standard EPA scans do not cover important alkyl PAHs and do not utilize low-enough detection limits. When biological effects, ecological risk assessment, damage assessment, or bio-remediation are being considered, detection limit should be no higher than 1-10 ng/L (ppt) for water and 1 ug/kg (ppb) dry weight for solids such as tissues, sediments, and soil.

Note: Utilizing up to date techniques, many of the better labs can use detection limits of 0.3 to 1 ppb for tissues, sediments, and soils. When no biological resources are at risk, detection limits for solids should nevertheless generally not be above 10 ppb. One reason that low detection limits are needed for PAHs is that so many of the criteria, standards, and screening benchmarks are in the lower ppb range (see various entries on individual PAHs).

In the past, many methods have been used to analyze for PAHs [861,1010,1013]. However, recent (1991) studies have indicated that EPA approved methods used for oil spill assessments (including total petroleum hydrocarbons method 418.1, semivolatile priority pollutant organics methods 625 and 8270, and volatile organic priority pollutant methods 602, 1624, and 8240) are all inadequate for generating scientifically defensible information for Natural Resource Damage Assessments [468]. These general organic chemical methods are deficient in chemical selectivity (types of constituents analyzed) and sensitivity (detection limits); the deficiencies in these two areas lead to an inability to interpret the environmental significance of the data in a scientifically defensible manner [468].

For risk, damage assessment, drinking water, or to determine if biodegradation has occurred, the NOAA expanded scan for PAHs and alkyl PAHs [828], or equivalent rigorous and comprehensive scans. (such as SW-846 method 8270 modified for Selective Ion Mode detection limits and an equivalent list of parent compound and alkyl PAH analytes), are recommended.

If a Park Service groundwater investigation at Colonial National Historical Park performed in response to contamination by Fuel Oil 5 had utilized EPA semi-volatile scan 8270 or any of the other typical EPA scans (625, etc.) all of which only include parent compounds and typically utilize detection limits in the 170-600 ppb range, the false conclusion reached would have been that no PAHs were present in significant (detection limit) amounts. This false negative conclusion would have been made because the parent compound PAHs present constituted only 7.6% of the PAHs detected in groundwater by the expanded scan [828], and the highest concentration found for any parent compound was 8.4 ppb, far below the detection limits used on the older standard EPA scans. Utilizing the NOAA protocol expanded scan [828], it was determined that 92.4% of the total concentration values of the PAHs detected in groundwater were alkyl PAHs, and that all 39 PAHs and alkyl PAHs were present. Of course, all 39 PAHs were also present in the fresh product, in much higher concentrations, and also having alkyl compounds with the highest percentage of higher values compared to parent compounds (see Chem.Detail section in separate PAHs entry for more details).

In a similar vein, if the Park Service sediment investigation at Petersburg National Historical Battlefield (see Chem.Detail section in separate PAHs entry, this study was performed in response to contamination by Diesel) had utilized EPA semi-volatile scan 8270 or any of the other typical EPA scans (625, etc.), all of which only include parent compounds and often utilize detection limits no lower than the 170-600 ppb range, the false conclusion reached would have been that only one PAH was present in significant (detection limit) amounts. This false negative conclusion would have been made because the parent compound PAHs present constituted only 2.4% of the PAHs detected in sediments, and the highest concentration found for any parent compound except pyrene was 85.5 ppb, far below the detection limits used on the older standard EPA scans. Pyrene was 185 ppb, which would have been non-detected on many of the EPA scans, but not all. However,

utilizing the NOAA protocol expanded scan [828], it was determined that 97.6% of total quantity of PAHs detected in sediments were alkyl PAHs, and that all 39 PAHs and alkyl PAHs were present in these sediments.

When taking sediment samples for toxic organics such as PCBs, PAHs, and organochlorines, one should also routinely ask for total organic carbon analyses so that sediment values may be normalized for carbon. This will allow comparison with the newer EPA interim criteria [86,127]. TOC in sediments influences the dose at which many compounds are toxic (Dr. Denny Buckler, FWS Columbia, personal communication).

In some cases (where the expanded scans are too expensive) an alternative recommendation is that one screen sediments with a size-exclusion high-performance liquid chromatography (HPLC)/fluorescence method. The utility and practicality of the HPLC bile and sediment screening analyses were demonstrated on board the NOAA R/V Mt. Mitchell during the Arabian Gulf Project. Estimates of petroleum contamination in sediment and fish were available rapidly, allowing modification of the sampling strategy based on these results [522].

Variation in concentrations of organic contaminants may sometimes be due to the typically great differences in how individual investigators treat samples in the field and in the lab rather than true differences in environmental concentrations. This is particularly true for volatiles and for the relatively lighter semi-volatiles such as the naphthalene PAHs, which are so easily lost at various steps along the way. Contaminants data from different labs, different states, and different agencies, collected by different people, are often not very comparable. See disclaimer at the beginning of this entry for additional detail.

As of 1997, the problem of lack of data comparability (not only for water methods but also for soil, sediment, and tissue methods) between different "standard methods" recommended by different agencies seemed to be getting worse, if anything, rather than better. The trend in quality assurance seemed to be for various agencies, including the EPA and others, to insist on quality assurance plans for each project. In addition to quality control steps (blanks, duplicates, spikes, etc.), these quality assurance plans call for a step of insuring data comparability [1015,1017]. However, the data comparability step is often not given sufficient consideration. The tendency of agency guidance (such as EPA SW-846 methods and some other new EPA methods for bio-concentratable substances) to allow more and more flexibility to select options at various points along the way, makes it harder in insure data comparability or method validity. Even volunteer monitoring programs are now strongly encouraged to develop and use quality assurance project plans [1015,1017].

At minimum, before using contaminants data from diverse sources, one should determine that field collection methods, detection limits, and lab quality control techniques were acceptable and comparable. The goal is that the analysis in the concentration range of the comparison benchmark concentration should be very precise and accurate.

It should be kept in mind that quality control field and lab

blanks and duplicates will not help in the data quality assurance goal as well as intended if one is using a method prone to false negatives. Methods may be prone to false negatives due to the use of detection limits that are too high, the loss of contaminants through inappropriate handling, or the use of an inappropriate methods such as many of the EPA standard scans. This is one reason for using the NOAA expanded scan for PAHs [828]; or method 8270 [1013] modified for Selective Ion Mode (SIM) detection limits (10 ppt for water, 0.3 to 1 ppb for solids) and additional alkyl PAH analytes; or alternative rigorous scans. These types of rigorous scans are less prone to false negatives than many of the standard EPA scans for PAH parent compounds (Roy Irwin, National Park Service, Personal Communication, 1997).

For a much more detailed discussion of the great many different lab and field methods for PAHs in general, see the entry entitled PAHs as a group (file name starting with letter string: PAHS). There the reader will find much more detailed discussions of lab methods, holding times, containers, comparability of data from different methods, field sampling methods, quality assurance procedures, the relationship of various methods to each other, the various EPA standard methods for various EPA programs, the pros and cons of various methods, and additional documentation concerning why many standard EPA methods are inadequate for certain applications. A decision tree key for selecting the most appropriate methods for oil or oil products spills is also provided in the lab section of the PAHs entry. Due to the length of these discussions, they are not repeated here (see PAHs entry).