ENVIRONMENTAL CONTAMINANTS ENCYCLOPEDIA

PAHS 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.

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> **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).

PAHs as a group (Polycyclic Aromatic Hydrocarbons, also known as Polynuclear Aromatic Hydrocarbons, discussion includes alkyl PAHs)

Brief Introduction:

Br.Class: General Introduction and Classification Information:

Polycyclic aromatic hydrocarbons (PAHs) are sometimes referred to as polynuclear aromatic hydrocarbons or as polycyclic aromatic compounds. Aromatics (including PAHs) are considered to be the most acutely toxic component of petroleum products, and are also associated with chronic and carcinogenic effects [770]. Aromatics are often distinguished by the number of rings they possess, which may range from one to five [770]. (For a list of these, see the Associated Chemicals or Topics section below). PAHs do not include the lighter, mono-aromatic (one ring) BTEX (benzene, toluene, ethylbenzene, and xylenes) compounds, the other group of hazardous aromatics commonly found in many petroleum fuels [771]. Aromatics with two or more rings are referred to as PAHs [770]. There are more than 100 different PAH compounds [788].

PAHs are among organic compounds which EPA classifies as Base/Neutrals (Extractable) [1010] and semi-volatile organics [1013].

In the strictest definition, PAHs are composed of "two or more fused aromatic (benzene) rings" [177]. However, most people consider the most important criteria in classifying PAHs to be whether or not two benzene rings are present in the chemical structure (Charlie Henry, Louisiana State University, personal communication). Therefore, a compound such as biphenyl is considered by most environmental chemists to be a PAH even though the two ring structure is joined directly rather than "fused."

In general, PAHs may be divided into two groups, depending upon their physical and chemical properties: low-molecular-weight PAHs, containing three or fewer aromatic rings, and high-molecular-weight PAHs, containing more than three aromatic rings (most information on the forms and fate of PAHs in the aquatic environment, however, is available for only a few compounds, specifically naphthalene, anthracene, benz(a)anthracene and benzo(a)pyrene) [754]. See Associated Chemicals or Topics section below for a list of the low- and high-molecular weight PAHs of concern.

PAHs may or may not have substituent groups attached to

one or more rings [794]. Alkyl and chlorine groups attached to PAHs change the fate and effects characteristics, often for the worse (see more detail in Br.Haz section below).

PAHs are a subset of a category of chemicals referred to as aromatic hydrocarbons (AHs) in some publications.

Sixteen of the common PAHs typically analyzed in standard contract laboratory scans have been listed by the Environmental Protection Agency among 129 priority pollutants [58]. Five of them are also listed among the 25 hazardous substances thought to pose the most significant potential threat to human health at priority superfund sites [93].

PAHs can be grouped into different categories, depending on their molecular weight and number of rings present. Often a lab analysis lists a total polycyclic aromatic hydrocarbon (tPAH) concentration (see Laboratory and/or Field Analyses section below). It is important to keep in mind that the individual PAH compounds included in "total" PAH measurements vary. For example, some studies include only the 16 EPA Priority Pollutant PAHs [634,853] or less (one study only included 11 PAHs [653]) in their TPAH value, while other studies include as many as 18 [697] or 24 [680] PAHs. See Associated Chemicals or Topics section below for more information on various ways "total PAHs" have been grouped.

PAHs are among the most hazardous compounds in oil spills [177]. PAHs are found in crude oil, used motor oil, soot, smoke from incomplete combustion, and in various complex mixtures of hazardous chemicals (such as creosote). The main sources of human exposure are food intake and inhalation (Rita Schoeny, Environmental Protection Agency, Cincinnati, personal communication). Food intake can be an important route for fish and wildlife too, along with direct contact in water, sediment, and soil environments.

ATSDR summary: PAHs are a group of chemicals that are formed during the incomplete burning of coal, oil, gas, wood, garbage, or other organic substances, such as tobacco and charbroiled meat [881]. There are more than 100 different PAHs [881]. PAHs generally occur as complex mixtures (for example, as part of combustion products such as soot), not as single compounds [881]. PAHs usually occur naturally, but they can be manufactured as individual compounds for research purposes; however, not as the mixtures found in combustion products [881]. As pure chemicals, PAHs generally exist as colorless, white, or pale yellow- green solids [881]. They can have a faint, pleasant odor [881].

PAHs have been found in at least 585 of the EPA's 1350 National Priorities List hazardous waste sites (as of Oct, 1993) [788].

PAHs most often occur in environmental samples as a minute fraction of a complex mixture of hydrocarbons, and it is the exception rather than the rule to find only one or two of these compounds occurring together. It is therefore of major importance that the methodology employed in such analyses include separation techniques for the determination and identification of individual PAHs [794].

PAHs are valuable for identifying spilled oil, distinguishing between sources of hydrocarbons in the environment, and providing information on the extent of oil weathering and degradation [468].

Petrogenic (related to crude oil and its products) PAHs characteristically have a greater percentage of alkyl PAHs compared to parent compounds, while pyrogenic (generated by high temperatures) PAHs tend to have a predominance of parent compound PAHs compared to alkyl PAHs [942]. In 1996, NASA scientists presented a somewhat underwhelming argument that lack of alkylation was evidence for a martian rather than an earthly origin for PAHs on a meteorite (see Uses/Sources section below).

Several PAHs are on many of the hazardous substances lists. If the following amounts of individual PAHs are released to the environment within a 24-hour period, EPA must be notified: 1 pound of benzo[b]fluoranthene, benzo[a]pyrene, or dibenz[a,h]anthracene; 10 pounds of benz[a]anthracene; 100 pounds of acenaphthene, chrysene, fluoranthene, or indeno[1,2,3-c,d]pyrene; or 5,000 pounds of acenaphthylene, anthracene, benzo[k]fluoranthene, benzo[g,h,i]perylene, fluorene, phenanthrene, or pyrene [881].

Br.Haz: General Hazard/Toxicity Summary:

Typically, both humans and fish and wildlife will not be exposed to an individual PAH, but to a mixture of PAHs [881]. Studies in animals have also shown that PAHs can cause harmful effects on skin, body fluids, and the body's system for fighting disease after both short-and long-term exposure [881]. PAHs are changed by all tissues in the body into many different substances [881]. Some of these substances are more harmful and some are less harmful than the original PAHs [881].

Some studies have concluded that the acute toxicity of

PAHs in oil appears to be a function of its di-aromatic hydrocarbon (that is, two-ring hydrocarbons such as naphthalene) content [770,854]. See W.Misc. section below for more details.

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).

Hazards to Fish, Wildlife and other Non-Human Biota:

Probably the most important target analytes in natural resource damage assessments for oil spills are PAHs and the homologous series (alkylated) PAHs [468]. Alkylated PAHs are often more abundant (at least for petrogenic PAHs), persist for a longer time, and are sometimes more toxic than the parent PAHs [468]. For example, methyl phenanthrene is more toxic than the parent compound phenanthrene. Metabolism of PAHs does not necessarily mean a reduction in the biological potency of the compound to harm the organism, since the metabolites are often more hazardous [469,889].

In a Park Service groundwater investigation at Colonial National Historical Park performed in response to contamination by Fuel Oil 5, 92.4% of the concentrations of various PAHs detected in groundwater were alkyl PAHs. All 39 PAHs and alkyl PAHs analyzed with the NOAA protocol expanded scan [828], were present in groundwater. Of course, all 39 PAHs were also present in fresh fuel oil 5, in much higher concentrations. Fresh fuel oil 5 also contained mostly alkyl compounds rather than parent compounds (see Chem.Detail section below for more details). Similar results were documented in a Park Service sediment investigation at Petersburg National Battlefield performed in response to contamination by Diesel Fuels (1D and 2D). In the Petersburg Study, 97.6% of the PAHs concentrations detected in sediments were alkyl PAHs. All 39 PAHs and alkyl PAHs analyzed were present in the contaminated sediments (Personal Communication, Roy Irwin, National Park Service, 1996). Since alkyl PAHs are such a high percentage of total PAHs in several petroleum products, they should not be

ignored in hazard or risk assessments.

Hazards from PAHs in the aquatic environment are often difficult to assess due to the great number of PAHs and alkyl PAHs of potential concern and the number of variables which can either increase or decrease the risk. The traditional concern of environmental toxicologists related to PAHs has focused on the metabolic breakdown of Benzo(a)pyrene and several other PAHs into metabolites far more carcinogenic and otherwise hazardous than the original parent compounds [889]. Total carcinogenic PAHs as low as 1.0 mg/kg have been shown to induce tumors in brown bullhead catfish [40]. However, the significance of cancer in fish and other aquatic populations has not been adequately determined [889].

PAHs tend to partition into sediments and soils, with relatively small concentrations of many PAHs showing up in water due to low solubilities [889]. Except for the more soluble and particularly toxic two-ring compounds such as the naphthalenes [770,854], water toxicity has historically not been thought to be a big problem for most PAHs. It had been thought that when the PAH molecular weight reaches that of three-ring compounds, an aqueous concentration equal to the solubility in water was required to elicit aquatic toxicity, and that heavier compounds would not exhibit acute toxicity even at solubility concentrations, the maximum concentrations water would hold at ordinary temperatures and pressures [779].

However, recent studies have shown that some of the heavier PAHs can exhibit acute water toxicity at levels below solubilities due to photo-enhanced toxicity in the presence of ultraviolet (UV) or other types of solar radiation [779,911,887].

The common PAH naphthalene is selectively phytotoxic, with alkyl compounds being most toxic [366], with effects on benthic aquatic invertebrates and fish. Phototoxicity of PAHs has been seen in a wide variety of aquatic organisms, including aquatic plants [911].

Phototoxicity of 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. Tests performed in the presence of UV or other solar radiation showed greatly increased toxicity to those same organisms

at PAH concentrations below maximum solubility [888,889,911,887].

Some experts now believe that much of the real hazard to aquatic systems from PAHs may result from photo-induced toxicity caused by PAH excited states in the presence of oxygen and UV radiation in sunlight [889,911]. It has been discovered that UV and other solar radiation penetrates deeper into naturally waters than had been previously thought [887]. It has also been confirmed that the toxicity of certain PAHs in the presence of sunlight or UV light can be hundreds of times greater than would be the case in the absence of photo sources [779,889,911].

Phototoxicity to Daphnia magna (zooplankton) has been documented for [887]:

Acridine
Anthracene
Benzo(a)anthracene
Benzo(b)anthracene
Benzanthrone
Benzo(k)fluoranthene
Benzo(a)fluorene
Benzo(b)fluorene
Benzo(ghi)perylene
Benzo(a)pyrene
Benzo(e)pyrene
Chrysene
Dibenzo(ah)anthracene
Fluoranthene
Perylene
Pyrene

Note: the above table 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].

Phototoxicity has been predicted using quantitative structure-activity relationship (QSAR) estimates for the following PAHs [891]:

Coronene
Dibenzo(a,j)anthracene
Benzo(a)chrysene
Benzo(b)chrysene
Benzo(b)triphenylene

Benzene and naphthalene are predicted not to be phototoxic using QSAR estimates [891].

Any benchmarks or criteria which have been developed for phototoxic PAH compounds or for any PAHs which have not been tested in the presence of UV or sunlight, may be under-protective [889]. Phototoxicity is potentially so important in aquatic toxicity of PAHs that the following additional summary statements are provided:

> Alkyl- and hydroxy- substituted PAHs tend to have similar phototoxicity potentials compared to the (unsubstituted) parent PAH compounds [888]. Alkene, nitro, and chloro substituents, however, have a greater effect on phototoxicity than alkyl or hydroxy substitutions [888].
>
> Photodegradation of PAHs into more toxic or harmful compounds, including free-radical PAH intermediates, is perhaps part of the mechanism of action for photo-induced increases in toxicity [779,889,911].

Potential effects of PAHs can relate to the fact that they can serve as both oxidizing and reducing agents:

> Oxidation: The most important processes contributing to the degradation of PAHs in water are chemical oxidation, photo-oxidation, and biodegradation by aquatic microorganisms [207,788]. PAHs (and many of their metabolites) are capable of causing oxidative damage within organisms [844]. Melancon provided a 1995 summary of biological effects of oxidative stress and protective mechanisms [844], and Arfsten et al. reviewed phototoxicity in 1996 [911]. PAHs produce an increase in intracellular oxidation and chromosomal breaks or gene rearrangements [571]. Some of the breakdown products of photodegradation of PAHs are of a concern related to mutagenicity, carcinogenicity, and general toxicity [779,911] (see Fate.detail section for discussion of photodegradation, photolysis, and photo-oxidation of PAHs).
>
> Reduction: Reduction potential can relate to mutagenicity and therefore potential carcinogenicity. Nitro-PAHs such as 3-

nitrofluoranthene (3NF) and 1 phenyl-4-nitro-naphthalene (1P4NN) form when free radical NOx compounds combine with PAHs in urban air have been investigated concerning potential mutagenicity related to reduction potential; only 3NF was strongly mutagenic (Science News, Volume 138).

For perylene and benzo(b)anthracene, phototoxicity may be mitigated by degradation [888]. However, alkyl PAHs typically bioconcentrate to a greater degree and degrade more slowly than their parent compounds. This is relevant to phototoxicity because many typical site investigations have only analyzed for parent compound PAHs and have therefore missed the alkyl PAHs which biodegrade more slowly in soils. These alkyl PAHs may still be inducing phototoxicity in exposed organisms after the parent compounds have been broken down.

Thousands of PAHs and alkyl PAHs have not yet been tested for phototoxicity, but quantitative structure-activity relationship (QSAR) investigations should help narrow which PAHs may exhibit this tendency [888].

PAHs which do not exhibit phototoxicity include phenanthrene and fluorene [888,887].

Organisms at risk from photo-enhanced effects of PAHs could potentially include those living or feeding in PAH contaminated sediments, fish or amphibian eggs or larvae in shallow water or the surface microlayer, and any other biota exposed to PAHs and solar radiation (see Interactions section below for more details).

Note: For a recent review of phototoxicity, see the following reference: Author: DP Arfsten, DJ Schaeffer, DC Mulveny, Title: The effects of near ultraviolet radiation on the toxic effects of polycyclic aromatic hydrocarbons in animals and plants: A review. Source: Ecotoxicology and Environmental Safety 33: 1-24 [911].

One of the reasons that PAHs are a potential risk to aquatic biota is that fuels or crude oils containing PAHs are so often spilled into aquatic environments. For example, heavier and more

persistent PAHs are found in gasolines (among other oil products) [796]. Although they make up small percentages of gasolines, they are more persistent than most other constituents of gasoline and tend to have greater carcinogenic and other chronic impact potential. Thus, although PAHs typically represent a small percentage of the total mass of volume of gasoline spilled, a few months later the PAHs represent a relatively large proportion of the hazardous components which still remain in contaminated soils or sediments (by then the also hazardous but more mobile and volatile BTEX compounds have often migrated into the air or groundwater) [796].

In general, although uptake of both low- and high-molecular-weight PAHs is relatively rapid in vertebrate aquatic species (like fish), metabolism and depuration are also rapid [754].

Our understanding of the effects of most PAHs and their hazardous metabolic breakdown products is very incomplete and changing rapidly as research is completed [40]. The environmental effects of the non-carcinogenic PAHs are poorly understood [40]. Given these factors, the best policy for preventing PAH impacts to fish and wildlife is to reduce or eliminate them wherever possible [40].

In the aquatic environment, naphthalenes are particularly hazardous PAHs due to their particular combination of mobility, toxicity, and general environmental hazard (personal communication, Roy Irwin, National Park Service, 1996, summary of information from references 770,854, and numerous other papers and personal communications). Alkyl naphthalenes pose similar hazards and are usually found in the same petroleum products as naphthalenes, often in higher concentration than the parent compound (naphthalene) [796].

Benthic organisms living in sediments are especially at risk for PAH-contaminated sediments [469].

Potential Effects on Humans:

Many of the same complications for predicting effects of PAH mixtures on humans are true for predictions related to fish and wildlife (see section above). The traditional concern of human toxicologists related to PAHs has focused on the metabolic breakdown of Benzo(a)pyrene and several other PAHs to metabolites far more carcinogenic and

otherwise hazardous than the original parent compounds [889].

Although PAHs usually occur in the presence of other PAHs, the combined effects of mixtures of PAHs upon humans is not well known, the main carcinogens tend to be metabolites rather than parent compounds, and there is very little information on some of the suspect compounds; the bottom line is that precise risk assessments of mixtures of PAHs is a very difficult task (Rita Schoeny, Environmental Protection Agency, Cincinnati, personal communication). 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 (BAP, the most studied PAH) as the standard (Rita Schoeny, Environmental Protection Agency, Cincinnati, personal communication, 1995).

In the environment, humans are most likely to be exposed to PAH vapors or PAHs that are attached to dust and other particles in the air [881]. Sources include cigarette smoke, vehicle exhausts, asphalt roads, coal, coal tar, wildfires, agricultural burning, residential wood burning, municipal and industrial waste incineration, and hazardous waste sites [881]. Background levels of some representative PAHs in the air are reported to be 0.02-1.2 nanograms per cubic meter (ng/m3; a nanogram is one-millionth of a milligram) in rural areas and 0.15-19.3 ng/m3 in urban areas [881]. You may be exposed to PAHs in soil near areas where coal, wood, gasoline, or other products have been burned [881].

Humans may be exposed to PAHs in the soil at or near hazardous waste sites, such as former manufactured-gas factory sites and wood-preserving facilities [881]. PAHs have been found in some drinking water supplies in the United States [881]. In the home, PAHs are present in tobacco smoke, from wood fires, creosote-treated wood products, cereals, grains, flour, bread, vegetables, fruits, meat, processed or pickled foods, and contaminated cow's milk or human breast milk [881]. Food grown in contaminated soil or air may also contain PAHs [881]. Cooking meat or other food at high temperatures, which happens during grilling or charring, increases the amount of PAHs in the food [881].

PAHs can enter humans through lungs humans you

breathe air that contains them (usually stuck to particles or dust) [881]. Cigarette smoke, wood smoke, coal smoke, and smoke from many industrial sites may contain PAHs [881]. People living near hazardous waste sites can also be exposed by breathing air containing PAHs [881].

Emphasis on BAP has increased tremendously due to its carcinogenicity, relative ease of analysis, and the belief by investigators that BAP serves as an indicator for the presence of other PAHs which contaminate the environment [794]. In evaluating the environmental effects of chemical mixtures, the "indicator chemical approach" is often used. This involves selecting a subset of chemicals from the whole mixture that represents the "worst-case" in terms of mobility and toxicity [745]. This approach can be used with crude oil with the "subsets" of chemicals being volatile organics such as BTEX (if present) and polycyclic aromatic hydrocarbons (PAHs) such as BAP. Although not PAHs, BTEX compounds are of concern in spills in most of the lighter petroleum fuels because the BTEX compounds are relatively soluble in water, highly mobile in the environment, and represent the more volatile and soluble components of crude oil. PAHs are not highly mobile but are of concern because they are prevalent in crude oil, represent the heavier or less volatile crude oil components, and several are known animal carcinogens [745]. A major deficiency of using the indicator chemical approach (or the similar surrogate chemical or toxic equivalency approaches) is that often the hazard of many alkyl PAHs, which are more persistent and often more common than the parent compounds used as indicators, are not thoroughly considered.

Immunological Effects: Immunosuppressive effects of PAHs have been documented in mammals [40,41]. PAHs have been shown to alter the immune system of mammals [835]. Commonly reported effects of petroleum and individual PAHs on living organisms are impaired immune systems for mammals [835]. The greater the carcinogenicity, the greater the effect on the immune system [40]. Additional detail on immune and immune vs. cancer issues from ATSDR [881]:

> 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].

Chlorine substituted naphthalenes (and probably other chlorinated PAHs) are very toxic to aquatic organisms and are also very persistent [207]. This is one reason that chlorine should probably not be used to disinfect waste or drinking water having high concentrations of PAHs [207]. Ozone could be used to break down PAHs without producing chlorinated PAHs [207].

The ATSDR has published a human toxicological profile for PAHs in general [881]. It covers:

acenaphthene
acenaphthylene

anthracene
benz[a]anthracene
benzo[a]pyrene
benzo[e]pyrene
benzo[b]fluoranthene
benzo[g,h,i]perylene
benzo[j]fluoranthene
benzo[k]fluoranthene
chrysene
dibenz[a,h]anthracene
fluoranthene
fluorene
indeno[1,2,3-c,d]pyrene
phenanthrene
pyrene

The 17 PAHs listed above were the PAHs identified at the highest concentrations at NPL hazardous waste sites [881]. This is probably at least partly because these are ones looked for most commonly, rather than these being the only common PAHs at petroleum impacted hazardous waste sites (Roy Irwin, National Park Service, Personal Communication, 1997). Some, but not all, of highlights of ATSDR information on effects of PAHs [881] has been incorporated into this document.

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

Higher weight PAHs include some of the most carcinogenic chemicals known to man. Many PAHs, and several breakdown products of PAHs have been documented to be tumorigenic [40]. In general, the heavier (4-, 5-, and 6-ring) PAHs have greater carcinogenic potential than the lighter (2- and 3-ring) PAHs [796]. The 4- to 7-ring PAHs have been especially implicated in the carcinogenic effect of used oil [519].

There are many references in the literature to complex mixtures of PAHs seeming to be associated with cancer in both fish and humans. Correlation between PAH metabolites in the gallbladder or the degree of PAH pollution in sediments has been positively correlated with DNA adducts or hepatic neoplasms in fish [793; Reprinted with permission from Environmental Toxicology and Chemistry, Volume 13, Van Der Weiden, M.E.J., F.H.M. Hanegraaf, M.L. Eggens, M. Celander, W. Seinen, and M. Van Den Berg. Temporal induction of cytochrome P450 1A in the Mirror Carp (Cyprinus Carpio) after administration of several polycyclic aromatic hydrocarbons. Copyright 1994 SETAC]. EPA has historically given polycyclic (polynuclear) aromatic hydrocarbons (PAHs) as a group the regulatory classification of "carcinogenic" [302,446].

However, literature sources vary in classifying various individual PAHs as carcinogens or non-carcinogens.

Some glucuronides (metabolic conjugates) are less toxic than the parent compound, but one PAH glucuronide (N-hydroxyacetylaminofluorene glucuronide) is actually a stronger carcinogen than the parent compound N-hydroxyacetylaminofluorene [483].

PAHs are common in the aquatic environment and sometimes may present the greatest carcinogenic insult [794]. Some PAH compounds were among the first to be definitely categorized as carcinogens, and perhaps 50 of them (mostly the heavier compounds) show carcinogenic properties (Rita Schoeny, Environmental Protection Agency, Cincinnati, personal communication, 1995).

Those performing either ecological or human risk assessments often report total carcinogenic PAHs and total non-carcinogenic PAHs and are therefore faced with the sometimes considerable challenge of deciding which PAHs at a given site to classify in each category. The cellular mechanisms of action are most likely similar for different species. Some carcinogenic/mutagenic aromatic hydrocarbons are actively taken up by fish and metabolized to derivatives that may lead to tumorous lesions [781]. Although some xenobiotics are thus detoxified, others, such as certain PAHs, are transformed into metabolites which are highly toxic, mutagenic, or carcinogenic to the host [207]. Several researchers have suggested that metabolic activity of the MFO system is a prerequisite for PAH induced carcinogenesis and mutagenesis [207].

However, different species vary in how quickly they breakdown PAH parent compounds and carcinogenic intermediates. Different species and individuals also vary in their susceptibility to carcinogens. The modern trend is towards incorporating the route of exposure and species into the statement, for example, stating that the compound is carcinogenic to mice by inhalation but not by ingestion.

Although more specificity is now recommended, the carcinogenicity classification schemes are presently still very general and sometimes in disagreement with each other. However, it is of interest how many of the 1980s "non-carcinogenic" and "weak carcinogen" ratings [40,302] have been changed to stronger warnings (either "probable carcinogens", "possible carcinogens", or "not-classifiable") as more information has become available [881]. It is especially notable that so few PAHs, and none of the PAHs commonly analyzed at hazmat sites using EPA semi-volatile scan 8270, still have a totally and

clearly clean bill of health as relates to carcinogenicity (see list provided below). The least worrisome thing that can be said of any of them is that they are "not classifiable" or that not all of the tests indicated carcinogenicity on a weight of evidence basis, or that the testing done has been insufficient to say one way or another. The record of all those common parent compound PAHs formerly classified by EPA as "noncarcinogenic" to humans [302] has been at least somewhat clouded as relates to ecorisk (for details, see list provided below).

Does it make sense to continue to classify certain PAHs as "non-carcinogenic" in site-specific risk assessments when they officially classified as "not classifiable" or when their records are somehow clouded or incomplete? Possibly not, especially since so many different complex mixtures of PAHs are carcinogenic and since the list of PAHs and alkyl PAHs used in typical risk assessments is usually very incomplete compared to the total list of carcinogenic alkyl PAHs likely to be present at the sites being assessed. Alkyl substitution often confers or enhances carcinogenic potential of PAHs.

Very few alkyl PAHs have been broadly tested for carcinogenicity, but it is known that both dimethylbenzo(a)anthracene and its parent compound benzo(a)anthracene are carcinogenic [40,793,788,881]. Methylbenzo(a)anthracene is actually more carcinogenic than its parent compound benzo(a)anthracene, and dimethylbenzo(a)anthracene is still more carcinogenic [40].

Both cholanthrene and its 3 methyl alkyl cholanthrene counterpart are carcinogenic [40,793]. It is also known that alkylation does not significantly change phototoxicity [888] and that there are some relationships between phototoxicity and potential carcinogenicity (see discussion above). Thus it would not be surprising to discover that a notable number of alkyl PAHs are carcinogenic although they are not now typically added to the list of "carcinogenic PAHs" considered in risk assessments.

Consider the case of naphthalene. Naphthalene is an example of a common PAH formerly classified as generally non-carcinogenic by EPA [302,446] and previously often given a non-carcinogenic rating in risk assessments. It is now classified as non-classifiable [766,IRIS] with a note in IRIS [893] that it will probably be upgraded to "possibly carcinogenic" soon (confirmed by Bob McGaughy, EPA, personal communication, 1996). There is currently some (mixed) evidence of naphthalene carcinogenicity to mice exposed by inhalation [867] as well as some

indications that naphthalene may act as a promoter for lung tumors started by other carcinogens [766]. Since naphthalenes often occur in petroleum hydrocarbon mixtures which contain strong carcinogens, a carcinogenic promoter role may prove environmentally significant.

> EPA IRIS Note ADDED IN JULY 1995 [893]: Subsequent to the verification of this cancer assessment in 1990, the National Toxicology Program completed a two-year cancer bioassay (1991); its results suggest that naphthalene may be more appropriately classified as a possible human carcinogen (Group C under current EPA guidelines) [893]. The NTP concluded, "Under the conditions of these 2-year studies, there was no evidence of carcinogenic activity of naphthalene in male B6C3F1 mice exposed by inhalation to concentrations of 10 or 30 ppm for 6 hours daily, 5 days per week, for 103 weeks [893]. There was some evidence of carcinogenic activity of naphthalene in female B6C3F1 mice, as indicated by the increased incidences of pulmonary alveolar/bronchiolar adenomas [893]."

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).

Many PAHs seem to have at least some indications of carcinogenicity somewhere in their testing literature. Since the cellular or subcellar modes of carcinogenicity tend to be similar no matter how the carcinogenic PAH metabolite arrived on the scene, is there any logical reason to classify a PAH as "non-carcinogenic" when one is not to sure one way or another?

When considering the logic of various approaches, it is appropriate to consider photoinduction and metabolic breakdown properties of various PAHs. Some of the photodynamic properties that can be used to model and predict phototoxicity can also be used as predictors of carcinogenicity [887].

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1987 SETAC].

Numerous authors have found a correlation between those PAHs which are phototoxic versus those which are carcinogenic [911]. Phototoxicity thus represents one potential clue related to carcinogenicity, since the structure of certain PAHs that makes them effective photodynamic toxicants may also make them effective carcinogens [887]. PAHs (and many of their breakdown products) are capable of causing oxidative damage within organisms [844]. Oxidative stress from free radical compounds produced by photodegradation of certain PAHs is involved in the presumed mode of action of phototoxicity [889], and some of the photodegradation breakdown products of PAHs are also potentially involved in mutagenicity and carcinogenicity [207,779,889].

The higher molecular weight PAHs benzo(a)pyrene, benzo(g,h,i)perylene, indeno(1,2,3-cd)pyrene, and compounds of molecular weight 302 were found to be the principal mutagenic compounds in a coal-tar-contaminated sediment [816]. Benzo(g,h,i)perylene and Benzo(a) pyrene are phototoxic. Indeno(1,2,3-cd)pyrene is known to be carcinogenic [881] but phototoxicity status is unknown. Benzo(a)pyrene is strongly phototoxic (see information below). Like phototoxicity, mutagenicity represents one clue suggesting possible carcinogenicity.

The carcinogenic action of PAH was correlated with phototoxicity [779]. Phenanthrene and fluorene, two PAHs which are not considered to be phototoxic [887] have carcinogenicity considered "not classifiable" by EPA and IARC [788,881]. Strong correlations between carcinogenic potential and phototoxic potential of PAHs have been documented many times since the correlation was first documented in invertebrates in 1939 [911]. It is also recognized that PAHs can enhance UV carcinogenicity [911]. Now that carcinogenicity ratings have changed, the strongest carcinogens also tend to be phototoxic, and it is still of interest to compare the lists of PAHs known or suspected to be carcinogens (below) to the lists of those known to induce phototoxicity (See Br.Hazard and Interactions sections).

Since fewer and fewer PAHs are classified as (definitely) non-carcinogenic as time goes along, and since the mechanism for phototoxicity includes the production of free radicals that could be involved in carcinogenicity, those PAHs known or suspected to be phototoxicants, but not already confirmed as carcinogens should perhaps be subjected to more testing for carcinogenicity (Roy Irwin, personal communication, National Park Service, 1996).

The debates on exactly how to perform both ecological and

human risk assessments on the complex mixtures of PAHs, alkyl PAHs, and other aromatics typically found at contaminated sites and oil spills, are likely to continue. There are some clearly wrong ways to go about it, but defining clearly right ways is more difficult. PAHs usually occur in complex mixtures rather than alone. Perhaps the most unambiguous thing that can be said about complex PAH mixtures is that such mixtures are often hazardous in many ways, including carcinogenicity and phototoxicity. (James Huckins, National Biological Survey/USGS, and Roy Irwin, National Park Service, personal communication, 1996, see also Arfsten et al) [911].

One way to approach site specific risk assessments is to collect the complex mixture of PAHs and other lipophilic organic contaminants in a semipermeable membrane device (SPMD, also known as a fat bag) [894,895,896], retrieve the organic contaminant mixture from the SPMD, then test the mixture for carcinogenicity, toxicity, and phototoxicity (James Huckins, National Biological Service, and Roy Irwin, National Park Service, personal communication, 1996).

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]. Studies of people show that individuals exposed by breathing or skin contact for long periods to mixtures that contain PAHs and other compounds can also develop cancer [881].

The Department of Health and Human Services (DHHS) has determined that benz[a]anthracene, benzo[b]fluoranthene, benzo[j]fluoranthene, benzo[k]fluoranthene, benzo[a]pyrene, dibenz[a,h]anthracene, and indeno[1,2,3-c,d]pyrene are known animal carcinogens [881]. The International Agency for Research on Cancer (IARC) has determined the following: benz[a]anthracene and benzo[a]pyrene are probably carcinogenic to humans; benzo[b]fluoranthene, benzo[j]fluoranthene, benzo[k]fluoranthene, and indeno[1,2,3-c,d]pyrene are possibly carcinogenic to humans; and anthracene, benzo[g,h,i]perylene, benzo[e]pyrene, chrysene, fluoranthene, fluorene, phenanthrene, and pyrene are not classifiable as to their carcinogenicity to humans [881]. EPA has determined that benz[a]anthracene, benzo[a]pyrene, benzo[b]fluoranthene, benzo[k]fluoranthene, chrysene, dibenz[a,h]anthracene, and indeno[1,2,3-c,d]pyrene are probable human

carcinogens and that acenaphthylene, anthracene, benzo[g,h,i]perylene, fluoranthene, fluorene, phenanthrene, and pyrene are not classifiable as to human carcinogenicity [881]. Acenaphthene has not been classified for carcinogenic effects by the DHHS, IARC, or EPA [881].

The National Institute for Occupational Safety and Health [881]. (NIOSH) concluded that occupational exposure to coal products can increase the risk of lung and skin cancer in workers [881]. NIOSH established a recommended occupational exposure limit, time-weighted average (REL-TWA) for coal tar products of 0.1 milligram of PAHs per cubic meter of air (0.1 mg/m3 for a 10-hour workday, within a 40-hour workweek [881]. The American Conference of Governmental Industrial Hygienists (ACGIH) recommends an occupational exposure limit for coal tar products of 0.2 mg/m3 for an 8-hour workday, within a 40-hour workweek [881]. The Occupational Safety and Health Administration (OSHA) has established a legally enforceable limit of 0.2 mg/m3 averaged over an 8-hour exposure period [881]. Mineral oil mists have been given an IARC classification of 1 (sufficient evidence of carcinogenicity) [881]. The OSHA Permissible Exposure Limit (PEL) for mineral oil mist is 5 mg/m3 averaged over an 8-hour exposure period [881]. NIOSH has concurred with this limit, and has established a recommended occupational exposure limit (REL-TWA) for mineral oil mists of 5 mg/m3 for a 10-hour work day, 40-hour work week, with a 10 mg/m3 Short Term Exposure Limit (STEL) [881].

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].

However, several of the tumorigenic PAHs (i.e., the nonalternant PAHs) discussed in this profile do not have a bay region, or have been shown not to be similarly activated via a simple bay region epoxide [881]. This observation has important implications regarding the

expression of carcinogenicity for the nonalternant PAHs [881]. If these chemicals are activated to carcinogens via a mechanism that differs from alternant PAHs, then they may also differ with respect to tumor site and species specificity [881]. A prerequisite for conversion of PAHs into these active bay region diol epoxides is the presence of cytochrome P-450 and associated enzymes responsible for this conversion [881]. These enzymes can be found primarily in the liver, but they are also present in the lung, intestinal mucosa, and other tissues [881]. Thus, factors such as distribution to the target tissue(s), solubility, and intracellular localization proximate to these enzymes figure prominently in the expression of a PAH's carcinogenicity [881].

The induction of one enzyme particularly important to the metabolism of PAHs, AHH, is also known to be under genetic control [881]. Given the heterogeneity of human genotypes, it is likely that certain human subpopulations exist that are more susceptible to AHH induction and thus more susceptible to the induction of cancer [881]. Once the reactive bay region epoxide is formed, it may covalently bind to DNA and other cellular macromolecules and presumably initiate mutagenesis and carcinogenesis [881]. Indeed, the level of DNA- adduct formation has been found to correlate with tumor induction activity for a number of PAHs in newborn rat liver and lung and in mouse skin [881].

More recent summary on individual PAHs: The following PAHs have either been termed carcinogens in general, have been found to be carcinogenic in at least certain settings, have not been adequately tested, have mixed records concerning carcinogenicity, have not yet been classified, or are considered "not classifiable." Without additional site- specific justification, it is questionable to use any of the following PAHs in risk assessment lists of "non-carcinogenic" PAHs (see indented information for a few details, and the entry for the compound for more details):

Acenaphthene

Acenaphthene had not been classified for carcinogenic effects by DHHS, IARC, or EPA, as of 1995 [881].

This compound has not been treated as a carcinogen for calculation purposes in some EPA risk-based (RBC and PRG) models [868,903], but this tentative distinction was made for the purpose of choosing a modeling scenario based on current (often inadequate) knowledge rather than for the purpose of strongly

stating that this compound is definitely not a carcinogen; the non-carcinogenic benchmarks are sometimes nearly as low as the carcinogenic benchmarks (Stan Smucker, personal communication, EPA, 1996).

IRIS EPA Carcinogenicity Assessment: Under Review as of 1996 [893].

Acenaphthylene

IRIS Database 1996 EPA Information [893]:

Evidence for classification as to human carcinogenicity; weight-of-evidence classification:

Classification: D; not classifiable as to human carcinogenicity

EPA: "not classifiable as to human carcinogenicity" [881].

EPA Historical (modeling purposes only) Classification: Carcinogen [302,446].

Acridine

Phototoxic [494,887]. Although not definitive, as discussed above, phototoxicity represents one clue suggesting possible carcinogenicity.

Anthracene

IRIS Database 1996 EPA Information [893]:

Evidence for classification as to human carcinogenicity; weight-of-evidence classification:

Classification: D; not classifiable as to human carcinogenicity

International Agency for Research on Cancer (IARC) rating of "not classifiable as to carcinogenicity to humans" (Class D) [788,881]:

EPA: "not classifiable as to human carcinogenicity" [881].

EPA Historical (modeling purposes only) Classification: Carcinogen [302,446].

This compound has not been treated as a carcinogen for model calculation purposes in some EPA risk-based (RBC and PRG) models [868,903], but this tentative distinction was made for the purpose of choosing a modeling scenario based on current (often inadequate) knowledge rather than for the purpose of strongly stating that this compound is definitely not a carcinogen; the non-carcinogenic benchmarks are sometimes nearly as low as the carcinogenic benchmarks (Stan Smucker, personal communication, EPA, 1996).

This is a very phototoxic PAH [887]. Phototoxic [494,911]. Although not definitive, as discussed above, phototoxicity represents one clue suggesting possible carcinogenicity. UV light greatly increases the toxicity of anthracene to bluegill sunfish [841].

Benzo(a)anthracene

IRIS 1996 EPA information [893]:

Evidence for classification as to human carcinogenicity; weight-of-evidence classification:

Classification:

B2 (probable human carcinogen)

Both EPA and the Department of Health and Human Services (DHHS) have determined that this compound may reasonably be anticipated to be a carcinogen (Class B2, probable human carcinogen [788,881].

International Agency for Research on Cancer (IARC) rating of "Probably Carcinogenic to Humans" (B2) [788].

EPA Historical (modeling purposes only) Classification: Carcinogen [302,446].

This is a phototoxic PAH [891,911,887]. Although not definitive, as discussed above, 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): The

factor for Benzo(a)anthracene compared to BAP is 0.1 [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, this 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).

Formerly considered only weakly carcinogenic [40].

This compound has been treated as a carcinogen for model calculation purposes in some EPA risk-based (RBC or risk-based concentration and PRG or Preliminary remediation goals) models [868,903].

Benzo(a)anthracene, methyl

More carcinogenic than benzo(a)anthracene parent compound, with potency depending on substitution position [40].

Benzo(a)anthracene, dimethyl

Strongly carcinogenic [40].
More carcinogenic than benzo(a)anthracene parent compound, and methyl benzo(a)anthracene [40].

In fish, the greatest hazards of PAHs are their carcinogenic properties, especially with metabolites of PAHs such as 7,12-dimethylbenz(a)anthracene [793].

Both EPA and the Department of Health and Human Services (DHHS) have determined that this compound may reasonably be anticipated to be a carcinogen (Class B2, probable human carcinogen) [788,881].

Benzo(b)anthracene

This is a phototoxic PAH [887]. Although not definitive, as discussed above, phototoxicity represents one clue suggesting possible carcinogenicity.

Benzo(a)chrysene

This is a (predicted) phototoxic PAH [891]. Although not definitive, as discussed above, phototoxicity represents one clue suggesting possible carcinogenicity.

Benzo(b)chrysene

This is a (predicted) phototoxic PAH [891]. Although not definitive, as discussed above, phototoxicity represents one clue suggesting possible carcinogenicity.

Benzo(a)fluoranthene

Related compounds benzo(k)fluoranthene, benzo(b)fluoranthene, and benzo(j)fluoranthene are all carcinogenic. Phototoxicity to Daphnia magna (zooplankton) has been documented for a related compound benzo(k)fluoranthene [887]. However, no specific information could be found on carcinogenicity or phototoxicity of benzo(a)floranthene.

Benzo(b)fluoranthene

EPA 1996 IRIS classification information [893]:

> Evidence for classification as to human carcinogenicity, weight-of-evidence classification:
>
> > Classification:
> >
> > > B2 (probable human carcinogen) [893].

Both EPA and the Department of Health and Human Services (DHHS) have determined that this compound may reasonably be anticipated to be a carcinogen (Class B2, probable human carcinogen) [788,881].

International Agency for Research on Cancer (IARC) rating of "Possibly Carcinogenic to Humans" [788].

Relative (equivalency factor) oral carcinogenic potency value compared to Benzo(a)pyrene (BAP, which is ranked 1.0): The factor for Benzo(b)fluoranthene compared to

BAP is 0.1 [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, this 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).

Carcinogenic [40].

Synonym of Benzofluoranthene, 3,4-: EPA Historical (modeling purposes only) Classification: Carcinogen [302,446].

Phototoxicity to Daphnia magna (zooplankton) has been documented for a related compound benzo(k)fluoranthene [887]. However, no specific information could be found on carcinogenicity or phototoxicity of benzo(b)floranthene.

Benzo(j)fluoranthene

Carcinogenic [40].

Both EPA and the Department of Health and Human Services (DHHS) have determined that this compound may reasonably be anticipated to be a carcinogen (Class B2, probable human carcinogen) [788,881].

International Agency for Research on Cancer (IARC) rating of "Possibly Carcinogenic to Humans" [788].

Benzo(k)fluoranthene

IRIS 1996 EPA information [893]:

> Evidence for classification as to human carcinogenicity; weight-of-evidence classification:
>
> > Classification B2 (probable human carcinogen).

Both EPA and the Department of Health and Human Services (DHHS) have determined that this compound may reasonably be anticipated to

be a carcinogen (Class B2, probable human carcinogen) [788,881].

International Agency for Research on Cancer (IARC) rating of "Possibly Carcinogenic to Humans" [788].

Historical EPA Classification: Carcinogen [302,446].

This is a phototoxic PAH [891,887]. Although not definitive, as discussed above, 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): The factor for benzo(k)fluoranthene compared to BAP is 0.01 [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, this 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).

Benzo(a)fluorene

Termed "Non-carcinogenic" in 1987 [40]. However, some other PAHs given this same classification in 1980s have now been classified otherwise, and since this PAH has shown some phototoxicity potential, perhaps additional carcinogenicity testing is in order.

This is a phototoxic PAH [887]. Although not definitive, as discussed above, phototoxicity represents one clue suggesting possible carcinogenicity.

Benzo(b)fluorene

Termed "Non-carcinogenic" in 1987 [40]. However, some others given this same classification in 1987 have now been classified otherwise, and since this PAH has shown some phototoxicity potential, perhaps

additional carcinogenicity testing is in order.

This is a phototoxic PAH [887]. Although not definitive, as discussed above, phototoxicity represents one clue suggesting possible carcinogenicity.

Benzo(g,h,i)perylene

EPA 1996 IRIS Database [893]:

> Evidence for classification as to human carcinogenicity, weight-of-evidence classification:
>
> > Classification: D; not classifiable as to human carcinogenicity

International Agency for Research on Cancer (IARC) rating of "not classifiable as to carcinogenicity to humans" (Class D) [788].

EPA: "not classifiable as to human carcinogenicity" [881].

EPA Historical (modeling purposes only) Classification: Carcinogen [302,446].

This is a phototoxic PAH [891,887]. Although not definitive, as discussed above, phototoxicity represents one clue suggesting possible carcinogenicity.

Found to be one of the three principal mutagenic compounds in a coal-tar-contaminated sediment [816]. The other two [benzo(a)pyrene and indeno(1,2,3-cd)pyrene] are known to be carcinogenic [881]. Mutagenicity represents one clue suggesting possible carcinogenicity.

Benzo(c)phenanthrene

Strongly carcinogenic [40,911]. Very phototoxic [911].

Benzo(a)pyrene

EPA (IRIS) 1996 [893]:

> Evidence for classification as to human carcinogenicity; weight-of-evidence classification

Classification B2 (probable human carcinogen) [893].

Strongly carcinogenic [40].

In fish, the greatest hazards of PAHs are their carcinogenic properties, especially with metabolites of PAHs such as benzo(a)pyrene (BaP) [793].

Both EPA and the Department of Health and Human Services (DHHS) have determined that this compound may reasonably be anticipated to be a carcinogen (Class B2, probable human carcinogen) [788,881].

International Agency for Research on Cancer (IARC) rating of "Probably Carcinogenic to Humans" (B2) [788].

EPA Historical (modeling purposes only) Classification: Carcinogen [302,446].

This is a very phototoxic PAH [891,887]. Although not definitive, as discussed above, phototoxicity represents one clue suggesting possible carcinogenicity.

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

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).

Benzo(e)pyrene

International Agency for Research on Cancer (IARC) rating of "not classifiable as to carcinogenicity to humans" (Class D) [788].

This is a phototoxic PAH [891,887]. Although not definitive, as discussed above, phototoxicity represents one clue suggesting possible carcinogenicity.

Some co-carcinogenic activity was noted when combined with mixtures of other PAHs in dermal treatments of mice [40].

EPA IRIS 1996: classification under review [893].

The 4- to 7-ring PAHs have been especially implicated in the carcinogenic effect of used oil [519].

B(E)P Inhibited 7,12-dimethylbenz(a)anthracene skin tumor-initiation in mice by 84%. [366, Slaga et al; Cancer Lett 7(1) 51-59 (1979)].

Benzo(b)triphenylene

This is a (predicted) phototoxic PAH [891]. Although not definitive, as discussed above, phototoxicity represents one clue suggesting possible carcinogenicity.

Biphenyl

EPA 1996 IRIS Database [893]:

> Classification as to human carcinogenicity: weight-of-evidence classification:
>
> Classification: D; not classifiable as to human carcinogenicity

Phototoxicity: no information found. Possible reference of interest: Hirayama, T., M. Nohara, H. Shindo and S. Fukui. 1981. Mutagenicity assays of photochemical reaction products of biphenyl (BP) and o-phenylphenol (OPP) with NOx. Chemosphere. 10(2): 223-228 [893].

Carbazole

This compound has been treated as a carcinogen for model calculation purposes in some EPA risk-based (RBC) models [903].

Cholanthrene

Carcinogenic [40].

Cholanthrene, 3 methyl

In fish, the greatest hazards of PAHs are their carcinogenic properties, especially with metabolites of PAHs such as 3-methylcholanthrene (3-MC) [793].

Strongly carcinogenic [40].

Chrysene

EPA 1996 IRIS Human carcinogenicity weight-of-evidence classification:

Classification:

B2 (probable human carcinogen) [893].

This compound has been treated as a carcinogen for model calculation purposes in some EPA risk-based (RBC and PRG) models [868,903].

Earlier International Agency for Research on Cancer (IARC) rating of "not classifiable as to carcinogenicity to humans" (Class D) [788].

Relative (equivalency factor) oral carcinogenic potency value compared to benzo(a)pyrene (BAP, which is ranked 1.0): The factor for chrysene compared to BAP is 0.001 [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, this 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).

Weakly carcinogenic [40].

This is a phototoxic PAH [887]. Although not definitive, as discussed above, phototoxicity represents one clue suggesting possible carcinogenicity.

Chrysene, C1-

This is a grouping of alkyl PAHs rather than a single compound. There is inadequate evidence that 1-methylchrysene is carcinogenic to experimental animals. There is limited evidence that 2-, 3-, 4- and 6-methylchrysenes are carcinogenic to experimental animals. There is sufficient evidence that 5-methylchrysene is carcinogenic to experimental animals [847].

However, alkyl substitution often confers or enhances carcinogenic potential of PAHs.

Chrysene, C2-

No information found on this specific grouping of alkyl PAHs.

However, alkyl substitution often confers or enhances carcinogenic potential of PAHs.

Chrysene, C3-

No information found on this specific grouping of alkyl PAHs.

However, alkyl substitution often confers or enhances carcinogenic potential of PAHs.

Chrysene, C4-

No information found on this specific grouping of alkyl PAHs.

However, alkyl substitution often confers or enhances carcinogenic potential of PAHs.

Dibenz(a,c)anthracene

Weakly carcinogenic [40].

Dibenz(a,h)anthracene

Synonym of Dibenzo(a,h)anthracene.

EPA 1996 IRIS database information [893]:

Human carcinogenicity weight-of-evidence classification:

Classification B2 (probable human carcinogen).

Animal carcinogenicity data:

Sufficient. Dibenz(a,h)anthracene has been shown to be carcinogenic when administered to mice by the oral route (Snell and Stewart, 1962, 1963).

Both EPA and the Department of Health and Human Services (DHHS) have determined that this compound may reasonably be anticipated to be a carcinogen (Class B2, probable human carcinogen) [788,881].

Strongly carcinogenic [40].

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 is BAP [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).

EPA Historical (modeling purposes only) Classification: Carcinogen [302,446].

This is a very phototoxic PAH [891,887]. Although not definitive, as discussed above, phototoxicity represents one clue suggesting possible carcinogenicity.

For Dibenz(ah)anthracene--there is experimental evidence of mammary carcinogenesis [571].

Dibenz(a,j)anthracene

Weakly carcinogenic [40].

This is a (predicted) phototoxic PAH [891]. Although not definitive, as discussed above, phototoxicity represents one clue suggesting possible carcinogenicity.

Dibenzo(a,c)fluorene

Weakly carcinogenic [40].

Dibenzo(a,g)fluorene

Weakly carcinogenic [40].

Dibenzo(a,h)fluorene

Weakly carcinogenic [40].

Dibenzo(a,1)pyrene

Weakly carcinogenic [40].

Dibenzo(a,e)pyrene

Carcinogenic [40].

Dibenzo(a,h)pyrene

Strongly carcinogenic [40].

Dibenzo(a,i)pyrene

Strongly carcinogenic [40].

Dibenzothiophene

No information found.

Dibenzothiophene, C1-

Cannot be given a rating because this is a grouping of alkyl PAHs rather than a single compound.

Dibenzothiophene, C2-

Cannot be given a rating because this is a grouping of alkyl PAHs rather than a single compound.

Dibenzothiophene, C3-

Cannot be given a rating because this is a grouping of alkyl PAHs rather than a single compound.

Fluoranthene

Information from EPA 1996 IRIS database [893]:

Human carcinogenicity weight-of-evidence classification:

Classification: D; not classifiable as to human carcinogenicity, based on no human data and inadequate data from animal bioassays.

Human carcinogenicity data: None.

Animal carcinogenicity data: Inadequate.

This compound has not been treated as a carcinogen for model calculation purposes in some previous EPA risk-based (RBC and PRG) models [302,406,868,903], but this tentative distinction was made for the purpose of choosing a modeling scenario based on current (often inadequate) knowledge rather than for the purpose of strongly stating that this compound is definitely not a carcinogen; the non-carcinogenic benchmarks are sometimes nearly as low as the carcinogenic benchmarks (Stan Smucker, personal communication, EPA, 1996).

However, these classifications may have been premature. Fluoranthene is a potent co-carcinogen [870].

NOTE: A co-carcinogen is a noncarcinogenic chemical that, when present with another carcinogenic chemical, enhances that chemical's carcinogenicity [494].

Some co-carcinogenic activity was noted for fluoranthene when combined with mixtures of other PAHs in dermal treatments of mice [40]. PAH compounds usually occur in the presence of other PAH compounds, and one of the few things that is relatively clear is that PAH mixtures in water, sediments, and organism internal tissues often tend to be both carcinogenic and phototoxic [911].

International Agency for Research on Cancer (IARC) rating of "not classifiable as to carcinogenicity to humans" (Class D) [788].

EPA 1995: "not classifiable as to human carcinogenicity" [881].

This is a phototoxic PAH [494,887]. Although not definitive, as discussed above,

phototoxicity represents one clue suggesting possible carcinogenicity.

Fluoranthenes + Pyrenes, C1-

No information was found on this particular grouping of alkyl PAHs.

However, alkyl substitution often confers or enhances carcinogenic potential of PAHs.

The following information relates to fluoranthene and pyrene compounds:

Both fluoranthene and pyrene are considered "not classifiable as to carcinogenicity to humans" (Class D) [788,893].

Both fluoranthene and pyrene are phototoxic PAHs [891,911,887]. The phototoxic effects of pyrene to mosquito larvae were similar to (the strong) phototoxic effects of BAP [911]. Although not definitive, as discussed above, phototoxicity represents one clue suggesting possible carcinogenicity.

These compounds have not been treated as carcinogens for model calculation purposes in some EPA risk-based (RBC and PRG) models [868,903], but this tentative distinction was made for the purpose of choosing a modeling scenario based on current (often inadequate) knowledge rather than for the purpose of strongly stating that this compound is definitely not a carcinogen; the non-carcinogenic benchmarks are sometimes nearly as low as the carcinogenic benchmarks (Stan Smucker, personal communication, EPA, 1996). EPA Historical (modeling purposes only) for both fluoranthene and pyrene: Not a Carcinogen [302,446].

However, these classifications may have been premature. Fluoranthene is a potent co-carcinogen [870].

NOTE: A co-carcinogen is a noncarcinogenic chemical that, when present with another carcinogenic chemical, enhances that chemical's carcinogenicity [494].

Some co-carcinogenic activity was noted for both fluoranthene and for pyrene when combined with mixtures of other PAHs in dermal treatments of mice [40]. PAH compounds usually occur in the presence of other PAH compounds, and one of the few things that is relatively clear is that PAH mixtures in water, sediments, and organism internal tissues often tend to be both carcinogenic and phototoxic [911].

Fluorene

EPA 1996 IRIS database information [893]:

Human carcinogenicity weight-of-evidence classification:

Classification: D; not classifiable as to human carcinogenicity

BASIS: Based on no human data and inadequate data from animal bioassays.

Human carcinogenicity data: None.

Animal carcinogenicity data: Inadequate.

International Agency for Research on Cancer (IARC) rating of "not classifiable as to carcinogenicity to humans" (Class D) [788].

EPA: "not classifiable as to human carcinogenicity" [881].

This compound has not been treated as a carcinogen for model calculation purposes in some EPA risk-based (RBC and PRG) models [868,903], but this tentative distinction was made for the purpose of choosing a modeling scenario based on current (often inadequate) knowledge rather than for the purpose of strongly stating that this compound is definitely not a carcinogen; the non-carcinogenic benchmarks are sometimes nearly as low as the carcinogenic benchmarks (Stan Smucker, personal communication, EPA, 1996).

EPA Historical (modeling purposes only) Classification: Carcinogen [302,446].

This is not a phototoxic PAH [888,891,887]. Although not definitive, as discussed above, phototoxicity represents one clue suggesting possible carcinogenicity.

Fluorene, C1-

No information found on this group of alkyl PAHs.

Fluorene, C2-

No information found on this group of alkyl PAHs.

Fluorene, C3-

No information found on this group of alkyl PAHs.

Indeno(1,2,3-cd)pyrene

EPA 1996 IRIS database information [893]:

Classification as to human carcinogenicity weight-of-evidence:

Classification B2 (probable human carcinogen)

BASIS: Based on no human data and sufficient data from animal bioassays. Indeno(1,2,3-cd)pyrene produced tumors in mice following lung implants, subcutaneous injection and dermal exposure. Indeno(1,2,3-cd)pyrene tested positive in bacterial gene mutation assays.

HUMAN CARCINOGENICITY DATA: None

ANIMAL CARCINOGENICITY DATA: Sufficient.

This compound has been treated as a carcinogen for model calculation purposes in some EPA risk-based (RBC and PRG) models [868,903].

EPA has determined that this compound is a probable human carcinogen (Class B2, probable human carcinogen) [788,881].

The Department of Health and Human Services (DHHS) states that it is a known animal carcinogen [881].

International Agency for Research on Cancer (IARC) rating of "Possibly Carcinogenic to Humans" [788].

Relative (equivalency factor) oral carcinogenic potency value compared to Benzo(a)pyrene (BAP, which is ranked 1.0): The factor for indeno(1,2,3-cd)pyrene compared to BAP is 0.1 [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, this 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).

Formerly considered weakly carcinogenic [40].

EPA Historical (modeling purposes only) Classification: Carcinogen [302,446].

Naphthalene

EPA 1996 IRIS Database [893]:

Evidence for classification as to human carcinogenicity; weight-of-evidence classification:

Classification: D; not classifiable as to human carcinogenicity

BASIS: Based on no human data and inadequate data from animal bioassays.

HUMAN CARCINOGENICITY DATA: None.

ANIMAL CARCINOGENICITY DATA: Inadequate.

However, according to information in IRIS [893], naphthalene may soon be upgraded to "possible carcinogen." status (confirmed by Robert McGaughy, EPA,

personal communication, 1996). Further details:

> EPA IRIS Note ADDED IN JULY 1995 [893]: Subsequent to the verification of this cancer assessment in 1990, the National Toxicology Program completed a two-year cancer bioassay (1991); its results suggest that naphthalene may be more appropriately classified as a possible human carcinogen (Group C under current EPA guidelines) [893]. The NTP concluded, "Under the conditions of these 2-year studies, there was no evidence of carcinogenic activity of naphthalene in male B6C3F1 mice exposed by inhalation to concentrations of 10 or 30 ppm for 6 hours daily, 5 days per week, for 103 weeks [893]. There was some evidence of carcinogenic activity of naphthalene in female B6C3F1 mice, as indicated by the increased incidences of pulmonary alveolar/bronchiolar adenomas [893]."

Mixed evidence has partly implicated naphthalene in lung cancer of female mice but not in male mice, or rats of either sex [867].

EPA Historical (modeling purposes only) Classification: Not a Carcinogen [302,446].

Naphthalene was predicted not to be phototoxic using QSAR estimates [891].

There is some (mixed) evidence of naphthalene carcinogenicity to mice exposed by inhalation [867,893] as well as some indications that naphthalene may act as a promoter for lung tumors started by other carcinogens [766]. Since naphthalenes often occur in petroleum hydrocarbon mixtures which contain strong carcinogens, a carcinogenic promoter role may prove environmentally significant.

Naphthalene, C1-C4

Cannot be given a rating because this is a grouping of alkyl PAHs rather than a single compound.

Perylene

International Agency for Research on Cancer (IARC): not classifiable as to carcinogenicity to humans or animals due to no adequate data or inadequate evidence [365]. Several other dibenzanthracene-related compounds are considered carcinogens (see dibenzanthracene ratings above).

This is a phototoxic PAH [891,887]. Although not definitive, as discussed above, phototoxicity represents one clue suggesting possible carcinogenicity.

Several other dibenzanthracene-related compounds are considered carcinogens (see dibenzanthracene ratings above).

No information in EPA IRIS, PRG, RBC sources [868,893,903].

Phenanthrene

EPA 1996 IRIS database information [893]:

Evidence for classification as to human carcinogenicity; weight-of-evidence classification

Classification: D; not classifiable as to human carcinogenicity

BASIS: Based on no human data and inadequate data from a single gavage study in rats and skin painting and injection studies in mice.

HUMAN CARCINOGENICITY DATA: None.

ANIMAL CARCINOGENICITY DATA: Inadequate.

International Agency for Research on Cancer (IARC) rating of "not classifiable as to carcinogenicity to humans" (Class D) [788].

EPA: "not classifiable as to human carcinogenicity" [881].

EPA Historical (modeling purposes only) Classification: Carcinogen [302,446].

Several references [887,888,891] do not

include phenanthrene among PAHs found to be phototoxic, but one reference does list it as phototoxic [494]. Although not definitive, as discussed above, phototoxicity represents one clue suggesting possible carcinogenicity.

Pyrene

EPA 1996 IRIS database information [893]:

> Classification as to human carcinogenicity; weight-of-evidence classification:
>
> > Classification: D; not classifiable as to human carcinogenicity
> >
> > BASIS: Based on no human data and inadequate data from animal bioassays.
>
> HUMAN CARCINOGENICITY DATA: None.
>
> ANIMAL CARCINOGENICITY DATA: Inadequate.

This compound has not been treated as a carcinogen for model calculation purposes in some EPA risk-based (RBC and PRG) models [868,903], but this tentative distinction was made for the purpose of choosing a modeling scenario based on current (often inadequate) knowledge rather than for the purpose of strongly stating that this compound is definitely not a carcinogen; the non-carcinogenic benchmarks are sometimes nearly as low as the carcinogenic benchmarks (Stan Smucker, personal communication, EPA, 1996).

However, some co-carcinogenic activity was noted when combined with mixtures of other PAHs in dermal treatments of mice [40].

This is a phototoxic PAH [891,911,887]. The phototoxic effects to mosquito larvae were similar to phototoxic effects of BAP [911]. Although not definitive, as discussed above, phototoxicity represents one clue suggesting possible carcinogenicity.

International Agency for Research on Cancer (IARC) rating of "not classifiable as to carcinogenicity to humans" (Class D) [788].

EPA: "not classifiable as to human

carcinogenicity" [881].

Some co-carcinogenic activity was noted when combined with mixtures of other PAHs in dermal treatments of mice [40].

The following PAHs have been termed non-carcinogens and we have not found any information to the contrary:

Aceanthrylene

Non-carcinogenic [40].

Anthanthrene

Non-carcinogenic [40].

Benzo(g,h,i)fluoranthene

Non-carcinogenic [40].

Benzo(c)fluorene

Non-carcinogenic [40].

Naphthacene

Non-carcinogenic [40].

Cancer vs. Total PAHs or Mixtures of PAHs:

As detailed above, perhaps the most unambiguous thing that can be said about PAH mixtures is that complex mixtures of PAHs tend to be carcinogenic and possibly phototoxic. Often the complex mixture of PAHs and other lipophilic organic contaminants should be collected in a semipermeable membrane device [894,895,896], retrieved from the SPMD, then tested for carcinogenicity, toxicity, and phototoxicity (James Huckins, National Biological Service, and Roy Irwin, National Park Service, personal communication, 1996).

Humans exposed to mixtures of PAHs and other compounds by inhalation or skin contact routes of exposure can develop cancer [881]. As mentioned above, EPA has historically given polynuclear (polycyclic) aromatic hydrocarbons (PAHs) as a group the regulatory classification of "carcinogenic" [302,446].

When considering total PAHs, one must first ask: which one? In other words, which and how many PAHs were totaled? For NOAA, the previous "Total

polycyclic aromatic hydrocarbons" (tPAH) was the sum concentration of 18 PAH compounds: biphenyl, naphthalene, 1-methylnaphthalene, 2-methylnaphthalene, 2,6-dimethylnaphthalene, acenaphthalene, fluorene, phenanthrene, 1-methylphenanthrene, anthracene, fluoranthene, pyrene, benz(a)anthracene, chrysene, benzo(a)pyrene, benzo(e)pyrene, perylene, and dibenz(a,h)anthracene [698]. However, many other risk assessors have totaled more or fewer PAHs to arrive at totals.

Downstream of a creosote superfund site (Eagle Harbor site in Puget Sound), sediment concentrations of total PAHs above 1.0 ppm dry weight had positive correlations with incidence of liver cancer in fish [124,125, confirmed by Don Malins, Pacific Northwest Research Foundation, personal communication]. In this assessment, only priority pollutant, parent compound PAHs were considered. The Eagle Harbor total PAH dry weight concentration of 1.0 ppm might typically correspond to a wet weight concentration of about 0.5 ppm, a total carcinogenic PAH level of 0.083 to 0.166 ppm and an individual Benzo(a)pyrene concentration of perhaps 0.0267 to 0.0369 ppm [124,125].

Other researchers have also documented carcinogenic impacts from low levels of PAHs in sediments. For example, sediments from the Buffalo River, New York with concentrations of total carcinogenic PAHs as low as 1.0 mg/kg induced tumors in brown bullhead catfish [40]. The otherwise hardy brown bullhead (Ictalurus nebulosus) is notably susceptible to PAHs, though evidence of cancer may not appear for 2 years after the initial exposure [81,82].

The concentration of total PAHs (16 individual PAH compounds) analyzed in the Hamilton Harbor sediment sample (137 ug/g) is similar to the concentrations of total PAHs reported for other areas where high occurrence of fish tumors have been observed, such as Black Rock Harbor, Connecticut (131 ug/g), the Black River entering Lake Erie (129 ug/g), and Eagle Harbor in Puget Sound (120 ug/g). Hepatic neoplasms have also been observed in mummichog fish (Fundulus heteroclitus) from areas where the total PAH concentration in sediments were as high as 2,200 ug/g. On the other hand, hepatic neoplasms were observed in black croaker fish (Cheilotrema saturnum) from San Diego Bay, where the total PAH concentration in sediment was reported to be only 7 ug/g. Therefore, it appears that total PAH concentrations in sediment are not always a

reliable indicator of the potential for the development of tumors in bottom-dwelling fish. Others have observed that PAH concentrations explained only 35% of the observed variation in neoplasm prevalence in English sole from various areas in Puget Sound [780; Reprinted with permission from Environmental Toxicology and Chemistry, Volume 14(1):79-91, Balch, G.C., C.D. Metcalfe and S.Y. Huestis, "Identification of Potential Fish Carcinogens in Sediment from Hamilton Harbor, Ontario, Canada", Copyright 1994 SETAC].

PAHs can function as estrogenic contaminants [571] (see details in Br.Dev section below).

Carcinogenicity vs. Plants

Although the data base is small, phytotoxicity from PAHs were rare; some plants can evidently catabolize the carcinogen benzo(a)pyrene, but metabolic pathways have not been clearly defined [40]. Some plants contain chemicals known to protect against PAH effects [40]. Some green plants contain ellagic acid, a substance that can destroy the diol epoxide form of benzo(a)pyrene, inactivating its carcinogenic and mutagenic potential [40]. This is an important factor since when PAHs do degrade through metabolism, they often break down into even more toxic, carcinogenic, and mutagenic compounds [40].

Some PAHs may promote plant growth, and the degree of the promoting effect corresponded to the oncogenic activity of the hydrocarbon. The six polycyclic aromatic hydrocarbons found in plants were tested one at a time or in combination. Considerable growth-promotion was noted (near to 100% in some cases) with the effectiveness of hydrocarbons ranked as follows: (1) Benzo(a)pyrene (2) Benzo(a)anthracene (3) Indeno (1,2,3-cd)pyrene, Benzo(b)fluoranthene (4) Fluoranthene (5) Benzo(ghi)perylene. [Graf W, Nowak W; Arch Hyg Bakt 150: 513-28 (1968) as cited in Health & Welfare Canada; Polycyclic Aromatic Hydrocarbons p.67 (1979) Report No. 80-EHD-50] [366].

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

Endocrine Disruption:

Commonly reported effects of petroleum and

individual PAHs on living organisms are altered endocrine functions for fish and birds [835].

PAHs can function as xenoestrogens [924]. Experimental evidence reveals that polycyclic aromatic hydrocarbons (PAHs) affect estrogen production and metabolism and thus function as xenoestrogens. For two of these--benzo(a)pyrene and dibenz(ah)anthracene--there is experimental evidence of mammary carcinogenesis [571].

The PAH naphthalene is an endocrine disrupter; when elevated in water to 10 ppm, naphthalene has caused crawfish ovaries to shrink, resulting in fewer eggs and smaller offspring [883,884] (Naphthalene confirmed as an endocrine disrupter by Milton Fingerman, Tulane University, personal communication, 1996). Noncarcinogenic effects of naphthalene exposure range from subtle changes in detoxifying enzymes to liver damage and interference with reproductive behavior [773].

Breast cancer risk factors can be linked to total lifetime exposure to bioavailable estrogens. Experimental evidence reveals that several classes of compounds, including polycyclic aromatic hydrocarbons (PAHs), affect estrogen production and metabolism and thus function as xenoestrogens [571]. For two of these--benzo(a)pyrene and dibenz(ah)anthracene--there is experimental evidence of mammary carcinogenesis [571].

Some limited gavage experiments with anthracene, fluoranthene, and fluorene exposures of mice produced no obvious endocrine effects [881],

PAHs are listed on many different general endocrine disruption lists [514,569,571,575]. These lists are sometimes based on little or circumstantial evidence. Ah receptor binding activity makes chemcals suspect related to male endocrine reproductive system effects [1025]. A group of halogenated aromatics that cause male reproductive risk can also activate AH receptors [1025].

Some experts believe that all AHH or Ah (receptor) active contaminants are endocrine disrupters, some disagree. Most experts would agree that more work needs to done before we thoroughly understand potential effects of PAHs on endocrine disruption in various species (Roy Irwin, National Park Service, Personal Communication, 1997).

In one study of human breast cells, several PAHs

that bind to the Ah receptor and are found in cigarette smoke, decrease estrogen-induced cell proliferation [1025].

One study showed an association of coke oven emissions with excess prostate cancer mortality, but it is unclear the role which PAHs and endocrine disruption may have played, 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].

General Reproduction:

Commonly reported effects of petroleum and individual PAHs also include impaired reproduction and reduced growth and development for plants, invertebrates, fish, reptiles, amphibians, and birds [835].

The testes and ovaries contain rapidly proliferating cells and therefore should be considered susceptible to damage by PAHs. The reproductive toxicity data in animals for the PAHs are limited. The available animal studies exclusively discuss the reproductive effects of benzo(a)pyrene [788].

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].

Teratogenicity and Mutagenicity:

Many PAHs, and several breakdown products of PAHs have been documented to be teratogenic and mutagenic to a variety of fish and wildlife, including fish, birds, amphibians, and mammals [40]. The higher molecular weight PAHs benzo(a)pyrene, benzo(ghi)perylene, indeno(1,2,3-cd)pyrene, and compounds of molecular weight 302 were found to be the principal mutagenic compounds in a coal-tar-contaminated sediment. The organism affected was the bacteria Salmonella typhimurium [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].

The higher molecular weight PAHs benzo(a)pyrene, benzo(ghi)perylene, indeno(1,2,3-cd)pyrene, and compounds of molecular weight 302 were found to be mutagenic [816].

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

Because of their low water-solubility and hydrophobic nature, PAHs tend to be associated primarily with inorganic and organic material in suspended and bed sediments [754]. Concentrations of PAHs in aquatic ecosystems are generally highest in sediments, intermediate in aquatic biota and lowest in the water column [754].

It is not known how rapidly or completely lungs absorb PAHs [881]. Drinking water and swallowing food, soil, or dust particles that contain PAHs are other routes for these chemicals to enter a human body, but absorption is generally slow when PAHs are swallowed [881]. Under normal conditions of environmental exposure, PAHs could enter a body from skin contact with soil that contains high levels of PAHs (this could occur near a hazardous waste site) or with used crankcase oil or other products (such as creosote) that contain PAHs [881]. The rate at which PAHs enter a human body by eating, drinking, or through the skin can be influenced by the presence of other compounds [881]. PAHs can enter all the tissues of a body that contain fat [881]. They tend to be stored mostly in kidneys, liver, and fat [881]. Smaller amounts are stored in the spleen, adrenal glands, and ovaries [881].

The MFO system acts to degrade aromatic and a number of other organic compounds (including PAHs) by (phase I) hydroxylation and/or (by phase II) conjugation with a glucuronic acid [574]. The reaction of organic contaminants with uridine diphosphate glucuronic acid (UDPGA) is called glucuronidation or glucuronide conjugation, a phase II metabolic process [483,494,855,982]. The transfer is mediated by glucuronyl transferase enzymes in the endoplasmic reticulum where hydroxylated phase I metabolites of lipophilic xenobiotic compounds are produced [483,855]. Several PAHs are changed to more water soluble forms by glucuronidation [366,483,494,609,982]. Several other breakdown pathways are also imporant for BaP and many

other PAHs [366,982].

BCFs of PAHs in fish and crustaceans have frequently been reported to be in the range of 100-2000. One study reported that, in general, bioconcentration was greater for the higher molecular weight compounds than for the lower molecular weight compounds [788].

Alkyl PAHs are more persistent than the parent PAHs [468]. They also tend to bioaccumulate to a greater degree [347,885]. Alkyl substitution usually decreases water solubility [754]. Increased alkylation in phenanthrene appears to increase bioconcentration tendencies [347]. Whereas phenanthrene (parent compound, no methyl groups attached) had higher concentrations in soil than in earth worms, the concentrations for 3,6,-dimethylphenanthrene were higher in earthworms than in soil [347]. This is not surprising since alkylation increases Kow and drastically changes other physical/chemical parameters. For more detailed discussions, see Chem.detail section of this entry and individual PAH entries. Fate summary information reflecting the Kow and other physical differences between alkyl and parent compound PAHs:

> Coho salmon accumulated the more highly alkylated naphthalenes in muscle tissue faster than the less-substituted aromatics [851]. PAH Alkyl naphthalenes were more persistent in sediments [521,885] and amphipod tissues [885] than naphthalene. Persistence increases with increasing alkyl substitution, with methyl naphthalene being more persistent than naphthalene and dimethyl naphthalene being still more persistent in both sediments and amphipod tissues [885].

In order to determine the presence and toxicity of PAHs in a water body, it is necessary to gather data both from the water column and sediment [653]. PAHs have a short residence time in aqueous solution and, when present in the water column, they are usually a result of recent or chronic pollution [653].

PAHs are not uniformly distributed throughout the aquatic environment; instead, higher concentrations are generally encountered near point sources. PAHs are expected to decrease logarithmically with distance from the source. Concentrations of PAHs are extremely variable, in part reflecting the degree of urban and industrial development and the specific use of the water [754].

PAHs have been detected in groundwater either as a result of migration directly from contaminated surface waters or through the soil (for example, fluorene). PAHs have also

been shown to be transported laterally within contaminated aquifers [788].

Metabolic transformations of PAHs into even more hazardous chemicals could also occur in sediments, soils, and various species of fish and wildlife [40,70]. Metabolic degradation of carcinogenic PAHs proceeds very slowly in subsurface soil or sediment environments. This is because these environments are low in oxygen and sunlight [40].

Photoinduced breakdown of PAHs into hazardous components can be dramatic (see Br.Hazard section above and Interactions section below).

Breakdown in soil and water generally takes weeks to months and is due mostly to the actions of microorganisms [788].

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

The movement of PAHs in the environment depends on properties such as how easily they dissolve in water, and how easily they evaporate into the air [881]. PAHs in general do not easily dissolve in water [881]. They are present in air as vapors or stuck to the surfaces of small solid particles [881]. They can travel long distances before they return to earth in rainfall or particle settling [881].

Some PAHs evaporate into the atmosphere from surface waters, but most stick to solid particles and settle to the bottoms of rivers or lakes [881]. In soils, PAHs are most likely to stick tightly to particles [881]. Some PAHs evaporate from surface soils to air [881]. Certain PAHs in soils also contaminate underground water [881].

The PAH content of plants and animals living on the land or in water can be many times higher than the content of PAHs in soil or water [881]. PAHs can break down to longer-lasting products by reacting with sunlight and other chemicals in the air, generally over a period of days to weeks [881]. Breakdown in soil and water generally takes weeks to months and is caused primarily by the actions of microorganisms [881].

The route by which PAHs and other xenobiotics enter the body may determine their fate and organ specificity [881]. For example, an inhaled compound may bypass the first-pass effect of the liver and reach peripheral tissues in concentrations higher than one would see after oral exposures [881]. Enzyme activities among tissues are

variable [881].

Most PAHs that enter the human body leave within a few days, primarily in the feces and urine [881].

Synonyms/Substance Identification:

For an extensive list of names of individual PAHs, see the Associated Chemicals and Topics section below.

Associated Chemicals or Topics (Includes Transformation Products):

NOTE: PAHs is a broad topic and readers can search for information under the name of individual PAHs. Since PAHs are one of the most hazardous groups of hydrocarbons in petroleum products, the reader should also refer to various oil topics:

Oil Spills
Petroleum, General
Crude Oil

Although some studies only analyze for the 16 Priority Pollutant PAHs, analyzing environmental samples for only the PAHs considered as EPA "priority pollutants" is no longer considered adequate for many purposes [564]. Therefore, we are emphasizing more complete lists than just the 16 priority PAHs, primarily those of at least 39 PAHs recommended by NOAA [828] and various laboratories that have been involved in oil contamination studies.

A long-term study by NOAA used the sum concentration of the following 18 PAH compounds for its measurement of tPAH [697] (NOTE: EPA Priority Pollutant PAHs are noted by the reference [634]):

EPA Priority Pollutant PAHs [634]:

Low molecular weight:

2-ring compounds:
- Acenaphthylene [634]
- Biphenyl
- Naphthalene [634]
- 1-Methylnaphthalene
- 2-Methylnaphthalene
- 2,6-Dimethylnaphthalene

3-ring compounds:
- Anthracene [634]
- Fluorene [634]
- Phenanthrene [634]
- 1-Methylphenanthrene

High molecular weight:

4-ring compounds:

Benzo(a)anthracene [634]
Fluoranthene [634]
Pyrene [634]

5-ring compounds:
Chrysene [634]
Benzo(a)pyrene [634]
Benzo(e)pyrene
Dibenz(a,h)anthracene [634]
Perylene [697]

The NOAA National Status and Trends Program's tPAH list includes 24 individual compounds; the list includes all 18 listed above plus the following 6 compounds [680] (NOTE: EPA Priority Pollutant PAHs are noted by the reference [634]):

Low molecular weight:
Acenaphthene [634]
1,6,7-Trimethylnaphthalene

High molecular weight:

Benzo(b)fluoranthene [634]
Benzo(k)fluoranthene [634]
Ideno(1,2,3-c,d)pyrene [634]
Dibenzo(a,h)anthracene [634]

Other PAHs, not in the above list, yet included in some PAH scans and discussed separately in this document, include:

2,3,5-Trimethylnaphthalene
Benzo(e)fluoranthene

NOTE: Some of the toxicity values, environmental concentrations, and other criteria in the rest of this PAH entry are for individual PAH compounds rather than just for PAHs as a group or for total PAHs. These individual values give the reader a brief idea of various PAH toxicities; however, more complete information on individual PAH compounds can be found separately in entries for each of these compounds.

The PAHs listed on various laboratory scans for PAHs vary by protocol, method, and lab. Historically, polycyclic aromatic hydrocarbons (PAHs) analyzed in typical laboratory scans included primarily only parent compounds and priority pollutant PAHs:

Anthracene (CAS number 120-12-7).

Benzo(a)anthracene (CAS number 56-55-3, synonym of 1,2-benzanthracene).

Benzo(b)fluoranthene (CAS number 205-99-2, synonym of Benzofluoranthene, 3,4-or 2,3-Benzofluoranthrene or 2,3-

Benzofluoranthene).

Benzo(k)fluoranthene (CAS number 207-08-9, sometimes misspelled Benzo(k)fluoranthrene).

Benzo(a)pyrene (CAS number 50-32-8).

Benzo(e)pyrene (CAS number 192-97-2).

Note: Benzo(e)pyrene has been on FWS scans but is not on all other standard scans for PAHs. For example, it has been absent from the scan LSU has historically done (Lynette Stevens, National Park Service, personal communication, 1994).

Benzo(g,h,i)perylene (CAS number 191-24-2).

Dibenz(a,h)anthracene (CAS number 53-70-3, synonym of 1,2,5,6-dibenzanthracene and dibenzo(a,h) anthracene).

Chrysene (CAS number 218-01-9).

Fluoranthene (C16-H10, CAS number 206-44-0, sometimes misspelled Fluoranthrene).

Fluorene (CAS number 86-73-7).

Naphthalene (CAS number 91-20-3)

Phenanthrene (CAS number 85-01-8).

Pyrene (CAS number 129-00-0).

Other PAHs included in some historical PAH scans included:

Acenaphthene (CAS number 83-32-9)
Acenaphthylene (CAS number 208-96-8)
Benzofluoranthenes (as a group)
Dimethylnaphthalene
Indeno(1,2,3-c,d)pyrene (CAS number 193-39-5)
1-Methylnaphthalene
2-Methylnaphthalene
Perylene
2,3,5-Trimethylnaphthalene

The historical scans proved inadequate [468] for use in risk or damage assessments, and a more current and rigorous "expanded scan of PAHs" included parent compounds and various alkyl homologs [828]:

Acenaphthene
Acenaphthylene
Anthracene
Anthracenes, C1-*

Anthracenes, C2-*
Anthracenes, C3-*
Anthracenes, C4-*
Benzo(a)anthracene
Benzo(b)fluoranthene
Benzo(k)fluoranthene
Benzo(g,h,i)perylene
Benzo(e)pyrene
Benzo(a)pyrene
Biphenyl
Chrysene
Chrysenes, C1-
Chrysenes, C2-
Chrysenes, C3-
Chrysenes, C4-
Dibenzo(a,h)anthracene
Dibenzothiophene

NOTE: Although opinions differ as to whether dibenzothiophene is a PAH, it is listed as such in several sources [795,468,495].

Dibenzothiophenes, C1-
Dibenzothiophenes, C2-
Dibenzothiophenes, C3-
Fluoranthene
Fluoranthenes, C1-*
Fluoranthenes/Pyrenes, C1-*
Fluorene
Fluorenes, C1-
Fluorenes, C2-
Fluorenes, C3-
Ideno(1,2,3,c,d)pyrene
Naphthalene
Naphthalenes, C1-
Naphthalenes, C2-
Naphthalenes, C3-
Naphthalenes, C4-
Perylene
Phenanthrene
Phenanthrenes/Anthracenes, C1-*
Phenanthrenes/Anthracenes, C2-*
Phenanthrenes/Anthracenes, C3-*
Phenanthrenes/Anthracenes, C4-*
Pyrene
Pyrenes, C1*

Specific Isomers included in expanded scan [828]:

Naphthalene, 1-Methyl
Naphthalene, 2,6-Dimethyl
Naphthalene, 1,6,7-Trimethyl
Phenanthrene, 1-Methyl

*NOTE: Due to difficulty in separating similar alkyl PAHs, some labs combine alkyl phenanthrenes and

anthracenes (both 3-ring PAHs), and some combine C1 (alkyl) fluoranthenes and pyrenes (both 4-ring compounds) (Guy Denoux, Geochemical and Environmental Research Group, Texas A&M University, personal communication, 1996).

Other "expanded scans of PAHs" delete biphenyl while adding Benzo(e)fluoranthene (Lisa Lefkovitz, Battelle NW, personal communication, 1995).

Some labs and programs also analyze for the following specific isomers. Many of these have been included in the NOAA National Status and Trends Program [680,697,828]:

Naphthalene, 2,6-Dimethyl
Naphthalene, 1-Methyl
Naphthalene, 2-Methyl
Naphthalene, 1,6,7-Trimethyl
Naphthalene, 2,3,5-Trimethyl
Phenanthrene, 1-Methyl

Chlorine vs. PAHs and chlorinated PAHs:

Sodium hypochlorite in water treatment plants effectively oxidizes most PAHs [207]. Chlorine substituted naphthalenes (and probably other chlorinated PAHs) are very toxic to aquatic organisms and are also very persistent [207]. This is one reason that chlorine should probably not be used to disinfect waste or drinking water having high concentrations of PAHs [207]. Ozone could be used to break down PAHs without producing chlorinated PAHs [207].

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

W.Low (Water Concentrations Considered Low):

No information found. See individual PAHs entries.

W.High (Water Concentrations Considered High):

Groundwater levels of PAHs near U.S. wood treatment facilities and a coal gasification plant have been found to be elevated. Groundwater from the site of a Seattle coal and oil gasification plant, which ceased operation in 1956, contained acenaphthylene in the range of 0.01-0.25 mg/L with an average concentration of 0.098 mg/L. Additional PAHs detected at this site include acenaphthene, fluorene, phenanthrene, fluoranthene, pyrene, and chrysene. Individual PAHs in the groundwater from five 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 [788].

Water concentrations of heavier (3 ring and above) is quite limited due to low water solubilities. However, phototoxicity can result in water toxicity even at very low concentrations (see Br.Hazard section above and W.Misc. section below).

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

> PAHs have been detected in urban runoff generally at concentrations much higher than those reported for surface water. Data collected as part of the Nationwide Urban Runoff Program indicate concentrations of individual PAHs in the range of 300-10,000 ng/L, with the concentrations of most PAHs above 1,000 ng/L (Cole et al. 1984). In a recent study by Pitt et al. (1993) which involved the collection and analysis of approximately 140 urban runoff samples from a number of different source areas in Birmingham, Alabama, and under various rain conditions, fluoranthene was one of two organic compounds detected most frequently (23% of samples). The highest frequencies of detection occurred in roof runoff, urban creeks, and combined sewer overflow samples. The maximum reported concentration of fluoranthene in these samples was 130 ug/L [881].
>
> Industrial effluents also have elevated PAH levels. Morselli and Zappoli (1988) reported elevated PAH levels in refinery waste waters, with concentrations for most PAHs in the range of 400 ng/L (benzo[b]fluoranthene) to 16,000 ng/L (phenanthrene). In an analysis of STORET data covering the period 1980-88, Staples et al. (1985) reported median concentrations in industrial effluents of less than 10 ug/L) for 15 PAHs. The number of samples ranged from 1,182 (benzo[b]fluoranthene) to 1,288 (phenanthrene); the percentage of samples in which PAHs were detected ranged from 1.5 (benzo[g,h,i]perylene) to 7.0 (fluoranthene) [881].
>
> Few data are available on the concentrations of PAHs in U.S. groundwater. Basu and Saxena (1978b) reported total PAH concentrations in groundwater from three sites in Illinois, Indiana, and Ohio to be in the range of 3-20 ng/L. Groundwater levels of PAHs near a coal and oil gasification plant and U.S. wood treatment facilities have been found to be elevated. Groundwater samples from the site of a Seattle coal and oil gasification plant which ceased operation in 1956 were found to contain acenaphthylene, acenaphthene, fluorene,

phenanthrene, fluoranthene, pyrene, and chrysene at concentrations ranging from not detected (detection limit 0.005 mg/L) to 0.25, 0.18, 0.14, 0.13, 0.05, 0.08, and 0.01 mg/L, respectively (Turney and Goerlitz 1990). 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 (Rosenfeld and Plumb 1991) [881].

An evaluation of the analytical data from 358 hazardous waste sites with over 5,000 wells indicated that anthracene, fluoranthene, and naphthalene were detected (practical quantitation limit, 10- 200 ug/L) in groundwater from at least 0.1% of the sites in three of the ten EPA Regions into which the United States is divided (Garman et al. 1987). 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].

W.Typical (Water Concentrations Considered Typical):

See W.Misc heading below for PAHs in a reservoir due to motor boat activity.

River water may contain significant concentrations of PAHs (0.02-3.79 ug/L total PAHs) [754].

Total concentrations of PAHs in surface waters ranged from 4.7 ng/L in Buffalo, NY to 600 ng/L in Pittsburgh, PA [788].

Polynuclear aromatic hydrocarbons were found in the Delaware River in Pennsylvania, and in rainwater in Switzerland. In both cases, all PAHs were present at sub-ug/L concentrations and probably originated with man-made sources [500].

Trace amounts of polynuclear aromatic hydrocarbons were found in 22 samples of rain and snow from Norway. PAHs found were phenanthrene, anthracene, fluoranthene, and others. They probably originated from combustion of fossil fuels [500].

PAHs have been detected in urban runoff generally at concentrations much higher than those reported for surface water. Data collected as part of the nationwide Urban Runoff Program indicate concentrations of individual PAHs in the range of 300-10,000 ng/L with concentrations of most PAHs above 1000 ng/L [788].

Groundwater usually contains PAHs at concentrations approximately one order of magnitude lower than those of surface waters (0.0009-4 ug/L total PAHs) [754]. One study of three sites (Illinois, Indiana, Ohio) found total PAH concentrations in groundwater to be in the range of 3 to 20 ng/L [788].

Reported maximum concentrations for total PAHs (based on 15 PAHs) in finished drinking water of 10 U.S. cities ranged from 4 to 24 nanograms per liter (ng/L; a liter is slightly more than a quart) [881]. Concentrations in untreated drinking water ranged from 6 to 125 ng/L [788].

Liquid domestic sewage: <1.9 ug/L [207].
Industrial sewage: 5-15 ug/L [207].

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

> PAHs have been detected in surface waters of the United States. In an assessment of STORET data covering the period 1980-82, Staples et al. (1985) reported 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 number of samples ranged from 630 (naphthalene) to 926 (fluoranthene); 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].
>
> Basu and Saxena (1978a) reported concentrations of selected PAHs in surface waters used as drinking water sources in four U.S. cities (Huntington, West Virginia; Buffalo, New York; and Pittsburgh and Philadelphia, Pennsylvania). Total concentrations of PAHs ranged from 4.7 ng/L in Buffalo to 600 ng/L in Pittsburgh. 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].

During April and May 1990, Hall et al. (1993) analyzed 48-hour composite samples from three locations in the Potomac River and three locations in the upper Chesapeake Bay for eight PAHs: perylene, fluorene, phenanthrene, anthracene, fluoranthene, pyrene, benz[a]anthracene, and chrysene. Pyrene was the only PAH found (0.42 ug/L) in these samples; it was detected in only one of nine Chesapeake Bay samples and not detected in any of the Potomac River samples (detection limit, 0.04 ug/L) [881].

In a more recent study by Pham et al. (1993), raw water samples from 5 areas in the St. Lawrence River and its tributaries were analyzed for 12 PAHs. The highest mean total PAH concentrations were observed in samples collected in the spring (27.3 ng/L) and autumn (21.03 ng/L), which was attributed to snow melt and increased runoff during these respective seasons. The lowest mean total PAH concentration was observed in summer (14.63 ng/L). High molecular weight PAHs were detected more frequently in the spring and autumn samples. Phenanthrene, benzo[b]fluoranthene, fluoranthene, and pyrene were predominant, comprising on average 33.8%, 17.4%, 17.1% and 12.8% of the total PAHs, respectively. With the exception of anthracene and benzo[b]fluoranthene, a general decrease in concentration with increasing molecular weight was observed [881].

Data summarized by Sorrel et al. (1980) indicate low levels of PAHs in finished drinking waters of the United States. Reported maximum concentrations for total PAHs (based on measurement of 15 PAHs) in the drinking water of 10 cities ranged from 4 to 24 ng/L; concentrations in untreated water ranged from 6 to 125 ng/L. The low concentrations of PAHs in finished drinking water were attributed to efficient water treatment processes. Shiraishi et al. (1985) found PAHs in tap water at concentrations of 0.1-1.0 ng/L, primarily as chlorinated derivatives of naphthalene, phenanthrene, fluorene, and fluoranthene. The significance to human health of these compounds is

not known (Eisler 1987) [881].

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):

Note: The acute toxicity of PAHs in water appears to be a function of its di-aromatic hydrocarbon (that is, two-ring hydrocarbons such as naphthalene) content [770,854] (see W.Misc. section below for details).

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].

The freshwater and saltwater Final Chronic Values (the concentration protecting aquatic life from chronic toxicity) for phenanthrene, a non-carcinogenic PAH, are 6.3 ug/L and 4.6 ug/L, respectively [127].

Water Quality Criteria for Polynuclear Aromatics (PAHs) in ug/L [446]:

Freshwater Acute Criteria: None Published

Freshwater Chronic Criteria: None Published

Marine Acute Criteria: Insufficient data to develop criteria. Lowest Observed Effect Level: 300

Marine Chronic Criteria: None Published

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 1992.

Many of the water quality criteria, standards, and screening benchmarks are in the lower ppb range and relate to individual PAHs rather than total PAHs. The following are a few examples of freshwater

aquatic benchmarks for individual PAHs:

Acenaphthene (ug/L) [649]:

23 = National ambient water quality final chronic value

Anthracene (ug/L) [649]:

0.024 = Secondary Acute Value
0.0013 = Secondary Chronic Value
0.09 = Estimated Lowest Chronic Value - Fish
< 2.1 = Lowest Chronic Value - Daphnids
0.35 = Estimated Lowest Test EC20 - Fish
> 8.2 = LOWEST TEST EC20 - DAPHNIDS

Benzo(a)anthracene (ug/l) [649]:

0.49 = Secondary acute value
0.027 = Secondary chronic value
0.65 = Estimated lowest chronic value - daphnid

Benzo(a)pyrene (ug/L) [649]:

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

Note from Roy Irwin: There are some human health MCL's for PAHs. For example: Drinking Water MCL for Benzo(a)pyrene = 2 ug/L [653].

Fluoranthene (ug/L) [649]:

33.6 = National Ambient Water Quality Final Acute Value

6.16 = National Ambient Water Quality Final Chronic Value

Methyl naphthalene, 1- (ug/L) [649]:

2.08 = Secondary chronic value

Naphthalene (ug/L) [649]:

23.4 = Secondary chronic value

Phenanthrene (ug/L) [649]:

6.3 = National ambient water quality criterion - acute (proposed)

W.Plants (Water Concentrations vs. Plants):

No information found for total PAHs. The following information is for one representative individual PAH: For fluoranthene, the 96-hr EC50 for the alga Selenastrum capricornutum, was 54.4 mg/L [754].

W.Invertebrates (Water Concentrations vs. Invertebrates):

No information found for total PAHs. The following information is for one representative individual PAH: For fluoranthene, the 48-hr EC50 for Daphnia magna was 325 mg/L [754].

Misc. References:

Author EAJ Bleeker, MC Buckertdejong, HG Vandergeest, MHS Kraak Title Toxicity of nitrogen-containing polycyclic aromatic hydrocarbons (NPAH) to the midge Chironomus riparius (Diptera) Source Proceedings of the Section Experimental and Applied Entomology of the Netherlands Entomological Society (N.E.V.), Vol 7, 1996 (1996) Page(s) 197-202

Author C Vanderkraan, WKRE Vanwingerden Title Use of the eggs of the large marsh grasshopper (Stethophyma grossum L) for testing the effects of polycyclic aromatic hydrocarbons (PAH) Source Proceedings of the Section Experimental and Applied Entomology of the Netherlands Entomological Society (N.E.V.), Vol 7, 1996 (1996) Page(s) 217-222

W.Fish (Water Concentrations vs. Fish):

No information found for total PAHs. The following information is for representative individual PAHs:

For fluoranthene, the 96-hr LC50 for bluegill was 3.98 mg/L [754].

In continuous-flow toxicity tests with embryo-larval stages of rainbow trout and largemouth bass, the LC50 values for phenanthrene were 0.04 and 0.18 mg/L, respectively [754].

In the presence of sunlight, anthracene was acutely toxic to fish at concentrations in the

order of 12 ug/L [754].

Histological and skeletal abnormalities were observed in rainbow trout alevins reared in aqueous solutions containing benzo(a)pyrene at concentrations as low as 0.08 ug/L [754].

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

No information found. See individual PAHs entries.

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

Water Quality Criteria for Polynuclear Aromatics (PAHs) in ug/L [446]:

NOTE: In 1980, criteria for PAHs were calculated for total PAHs rather than for each individual PAH compound. This approach was taken both because there were insufficient data to calculate individual criteria and because the environmental exposure pathway for these chemicals would likely involve contact with complex PAH mixtures. Current (1993) criteria for individual PAH compounds can be found under the entry for that particular compound [689].

Human Health (10E(-6) Risk Level for Carcinogens) ug/L [446]:

Published Criteria for Water and Organisms: 0.0028

Published Criteria for Organisms Only: .0311

IRIS Recalculated (9/90) Criteria for Water and Organisms: None Published

IRIS Recalculated (9/90) Criteria for Organisms Only: None Published

Drinking Water MCL: None Published

Criteria Federal Register Notice Number: 45 FR 79339

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

1992.

Human Health (10E(-5) Risk Level for All Carcinogenic PAHs [40]: 0.028 ug/L.

Human Health (10E(-6) Risk Level for All Carcinogenic PAHs [40]: 0.0028 ug/L.

Human Health (10E(-7) Risk Level for All Carcinogenic PAHs [40]: 0.00028 ug/L.

The World Health Organization (WHO) in Europe recommends that the total concentration of six specific PAHs not exceed 200 ng/L for domestic drinking waters. The six specified PAHs are fluoranthene, benzo(b)fluoranthene, benzo(k)fluoranthene, benzo(a)pyrene, benzo(ghi)perylene, and indeno(1,2,3-cd)pyrene [795].

The total concentration of six specific PAHs should not exceed 0.0135 to 0.2 ug/L for drinking waters. The six specified PAHs are fluoranthene, benzo(b)fluoranthene, benzo(k)fluoranthene, benzo(a)pyrene, benzo(ghi)perylene, and indeno(1,2,3-cd)pyrene [40].

Other Historical Human Water Standards [366]:

The attempt to develop a drinking water criterion for Polyaromatic nuclear 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 benz(a)pyrene (BaP) 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 p.C-118 (1980)].

European standard for polycyclic aromatic hydrocarbons is: ground waters, up to 0.05 ug/l; drinking waters, up to 0.1 ug/l. /Polycyclic aromatic hydrocarbons/ [Borneff J, Kunte H; Arch Hyg Bacteriol 153 (3): 220-229 (1969) as cited in Health and Welfare Canada; Polycyclic Aromatic

Hydrocarbons p.106 (1979) Report No. 80-EHD-50].

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

Some studies have concluded that the acute toxicity of PAHs in oil appears to be a function of its di-aromatic hydrocarbon (that is, two-ring hydrocarbons such as naphthalene) content [770,854]. Toxicity resulting from fresh Prudhoe Bay Crude oil water soluble fractions and dispersed crude oil was likely the result of the combination of monoaromatic and di-aromatic compounds found in fresh oil [854]. Data from shrimp (Pandalus danae) exposure showed a decrease in toxicity with decreasing amounts of total aromatics in the mono- and di-aromatic range (benzene, alkylbenzenes and naphthalenes) as the fresh product was distilled in order to approximate field conditions [854]. The naphthalenes still present in the Stage I oil (that is, the stage where the benzenes and alkylbenzenes had been removed) produced significant mortality [854]. Stage II (which lacked benzenes, alkylbenzenes, and naphthalenes) did not produce significant mortality for shrimp in either the water soluble fraction or dispersed phase [854]. In amphipod sediment toxicity tests conducted in an Exxon Valdez spill investigation, concentrations of 2-ring PAHs correlated better with toxicity than did concentrations of total PAHs or 4- and 5-ring PAHs. This was partly due to differences in water solubility and bioavailability (Bill Stubblefield, ENSR, Ft. Collins, CO, personal communication, 1995).

It had previously been thought that when the PAH molecular weight reaches that of three-ring compounds, an aqueous concentration equal to the solubility in water was required to elicit aquatic toxicity, and that heavier compounds would not exhibit acute toxicity even at maximum solubility [779]. However, recent studies have shown that some of the heavier PAHs can exhibit acute toxicity at levels below solubilities due to photoinduced enhanced toxicity in the presence of UV or other types of solar radiation [779,887]. For additional details on photoinduced toxicity, see Interactions section.

The potential impact of motorboat activity on the Occoquan (drinking-water) reservoir east of Washington, D.C. was evaluated at both marina and non-marina sites. The presence of PAHs (especially the lower molecular weight compounds like acenaphthene and naphthalene) in June during peak boating activity, and the absence of PAHs in October, a period of low boating activity, indicated boating to be a source of PAH to the water [653].

More detail [653]: There appears to be a

relationship between the amount of boating activity and the concentration of PAHs. Since PAHs are rapidly lost from the water column due to volatilization and sedimentation, the presence of PAHs, especially the lower molecular wight compounds (eg. acenaphthene and naphthalene), in the water column during June probably resulted from recent PAH inputs. The presence of PAHs in June during peak boating activity, and the absence of PAHs in October, a period of low boating activity, further indicated boating to be at least one of the sources of PAH to the water. The PAH concentration found in the Occoquan reservoir water column ranged from not detectable to 4.12 ug/L for total PAH concentrations and from not detectable to 2.14 ug/L for individual PAH concentration for June. Removal mechanisms such as photolysis, volatilization, sedimentation, or hydrological processes may explain losses of PAHs from the water column throughout the summer. This is in agreement with another study which found the highest hydrocarbon concentrations in Lake Metigoshe, North Dakota, in July, at a time of peak boating activity and the lowest concentrations in October, at a time of low boating activity [653].

A two-way analysis of variance test showed that at the 95% confidence level, there were no significant differences in the total aqueous PAH concentration found at marina and nonmarina sites. The data suggests that PAH concentration in the water column was not localized and that mixing, dilution, or other PAH sources may have influenced PAH distribution. For June, the Fountainhead Marina and Bull Run Marina at the Occoquan reservoir appeared to have lower aqueous total PAH concentrations than LRCP Marina and Hooes Run. Both Hooes Run and LRCP Marina are located in inlets; Hooes Run has substantial residential development. The marina sites at Fountainhead and Bull Run which reside on the main channel of the reservoir were probably impacted by recent boat activity and then longitudinal mixing, dilution, and transport resulted in lower PAH concentrations being detected throughout the water column. This is supported by the observed data which illustrates that the median total PAH concentration for inlet sites was much greater than for non-inlet sites, independent of a marina at either type of site [653].

While aqueous total PAH levels differed between the marina and nonmarina sites they were not significant. However, the PAH levels differed

significantly in the sediments (see corresponding study information in sediment section). It can be concluded from this that in order to determine the presence and toxicity of PAHs it is necessary to gather data both from the water column and sediment.[653].

Permitting only use of motorboats with less than 10 horse power engines in the Occoquan drinking reservoir, appeared to result in minor adverse impacts on water quality from boating activity [653].

Removal mechanisms, such as photolysis, volatilization, sedimentation, or hydrological processes may explain losses of PAHs from the water column throughout the summer. However decreases in boating in October resulted in the absence of PAHs in the water column [653].

PAH's were detected in a study of motorboat activity on a major drinking water reservoir (Occoquan reservoir). Total PAH concentration of < or equal to 4 ug/L were present in the water during June, a period of peak boating activity, but aqueous PAHs were not detected during October, a period of low boating activity. The PAH concentration found in the Occoquan reservoir water column ranged from not detectable to 4.12 ug/L for total PAH concentrations and from not detectable to 2.14 ug/L for individual PAH concentration for June. PAHs were detected in the sediments during both sampling periods; sediment concentrations for total PAHs were generally <700 ug/kg. The PAH profile of June water samples was representative of combustion and petrogenic sources, while the PAH profiles found in the sediments were typical of combustion. Boating was shown to be a PAH source to the water column during periods of high boating activity [653].

The attempt to develop a drinking water criterion for polynuclear aromatic hydrocarbons as a class is hindered by several gaps in the scientific data base: (1): The polynuclear aromatic hydrocarbon class is composed of numerous compounds having diverse biological effects and varying carcinogenic potential. A "representative" polynuclear aromatic hydrocarbon 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 polynuclear aromatic hydrocarbon may not be scientifically sound. (3): No chronic animal toxicity studies involving oral exposure to polynuclear aromatic hydrocarbon 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 p.C-118 (1980)] [366].

In fish the greatest hazards of PAHs are their carcinogenic properties, especially with metabolites of PAHs such as benzo(a)pyrene (BaP), 7,12-dimethylbenz(a)anthracene, and 3-methylcholanthrene (3-MC). These metabolites are produced after metabolic bioactivation by the cytochrome P450 1A isoform (the cytochrome P450 1A isoform is a form of cytochrome P450) [793; Reprinted with permission from Environmental Toxicology and Chemistry, Volume 13, Van Der Weiden, M.E.J., F.H.M. Hanegraaf, M.L. Eggens, M. Celander, W. Seinen, and M. Van Den Berg. Temporal induction of cytochrome P450 1A in the Mirror Carp (Cyprinus Carpio) after administration of several polycyclic aromatic hydrocarbons. Copyright 1994 SETAC].

Survival of striped bass was significantly reduced in laboratory tests by exposing them to a mixture of organic (including PAH) and inorganic contaminants at concentrations similar to those found on east coast spawning grounds [89]. Doubling the concentrations of each contaminant in the mixture further increased the lethality of the mix and the bioaccumulation of PAHs [89].

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

Sed.Low (Sediment Concentrations Considered Low):

No information found. See individual PAHs entries.

Sed.High (Sediment Concentrations Considered High):

PAH concentrations in sediments downstream from an aluminum smelter in British Columbia were found to be 1000 to 10,000 times higher (up to 2829 ug/g dry wt for 12 PAHs) than those found in the water column in Sydney Harbor, Nova Scotia [754].

Total (priority pollutant only) PAHs in sediments of the Mississippi River downstream of St Paul exceeded 16 mg/Kg [912].

Drainage stream sediments of a wood-preserving facility near Pensacola, Florida, contained various PAHs at individual levels up to 140,000 ug/kg (anthracene) [788].

NOAA National Status and Trends Program (1984-1990) [698]: High concentration for tPAH in fine-grained sediment = 4000 ng/g dry wt at 4.6% total organic carbon (TOC) dry wt.

NOTES:
* "High" concentration is the geometric mean plus one standard deviation on the log normal distribution.
* Total polycyclic aromatic hydrocarbons (tPAH) is the sum concentration of 18 PAH compounds: biphenyl, naphthalene, 1-methylnaphthalene, 2-methylnaphthalene, 2,6-dimethylnaphthalene, acenaphthalene, fluorene, phenanthrene, 1-methylphenanthrene, anthracene, fluoranthene, pyrene, benz(a)anthracene, chrysene, benzo(a)pyrene, benzo(e)pyrene, perylene, and dibenz(a,h)anthracene.
* Samples taken from over 200 sites on U.S. east and west coasts, and Gulf of Mexico; half the sites in rural areas, and half near urban areas (within 10 mi of cities in excess of 100,000 people, but away from obvious "hot spots").
* "Fine-grained" sediment is <or= 64 um [698].

Sed.Typical (Sediment Concentrations Considered Typical):

See also information [780] in Sed.Fish section below.

PAH concentrations in sediments are generally higher than those in surface water (that is, in the range of ppb rather than ppt) [788].

Sediment concentrations of total PAHs in SW Lake Erie near a large coal-fired power plant were generally in the range of 530-700 ug/kg, although concentrations in river and near-shore sediments reached nearly 4000 ug/kg (4 ppm). Another study found total concentrations of 3-7 ring PAHs from two lakes in the Adirondack acid lake region of 2,660 ug/kg and 770 ug/kg. Concentrations of PAHs in sediments from Cape Cod and Buzzards Bay in Massachusetts and the Gulf of Maine have been reported to be in the range of 540-1,300 ppb. Average concentrations of total PAHs in sediments from three coastal South Carolina marinas were reported to range from 35.6 to 352.3 ug/kg. Total PAH concentrations in bottom sediments from the main stem of the Chesapeake Bay were reported at 45-8,920. Benzo(a)pyrene levels in bottom sediments of the Great Lakes have been reported to range from 34-490 ppb [788].

Typical Total PAH Concentrations in Sewage sludge: 1-30 mg/kg [207].

NOAA National Status and Trends Program (1984-1990) [698]: Geometric mean ("typical") tPAH concentration in fine-grained sediment = 810 ng/g dry wt at 1.4% total organic carbon (TOC) dry wt.

NOTES:
* Total polycyclic aromatic hydrocarbons (tPAH) is the sum concentration of 18 PAH compounds: biphenyl, naphthalene, 1-methylnaphthalene, 2-methylnaphthalene, 2,6-dimethylnaphthalene, acenaphthalene, fluorene, phenanthrene, 1-methylphenanthrene, anthracene, fluoranthene, pyrene, benz(a)anthracene, chrysene, benzo(a)pyrene, benzo(e)pyrene, perylene, and dibenz(a,h)anthracene.
* Samples taken from over 200 sites on U.S. east and west coasts, and Gulf of Mexico; half the sites in rural areas, and half near urban areas (within 10 mi of cities in excess of 100,000 people, but away from obvious "hot spots").
* "Fine-grained" sediment is <or= 64 um [698].

PAHs were detected in a study of motorboat activity on a major drinking water reservoir. Total PAH concentration of < or equal to 4 ug/L were present in the water during peak boating activity, but aqueous PAHs were not detected during low boating activity. PAHs were detected in the sediments during both sampling periods; sediment concentrations for total PAHs were generally <700 ug/kg. The PAH profile of June water samples was representative of combustion and petrogenic sources, while the PAH profiles found in the sediments were typical of combustion. Boating was shown to be a PAH source to the water column during periods of high boating activity [653]. Additional detail:

> The ANOVA test for the June sediment data showed that there was a significant difference in the total PAH concentration (p=0.05) and the phenanthrene concentration (p=0.049) for the marina and nonmarina sites. In addition, there was a significant difference for the individual PAH concentrations of phenanthrene, fluoranthene, and pyrene for October; this demonstrated localized impacts of boating at marina sites [653]. That marina sediments would contain higher levels of PAHs than nonmarina sediments during a period of high boating activity is consistent with marina sediments absorbing PAHs from motorboat fuel and combustion. Lower median total PAH concentrations were observed for inlet sites compared to noninlet sites [653].
>
> Only a few PAHs (eg. phenanthrene, pyrene, and

fluoranthene) were commonly found throughout the [Occoquan] reservoir sediments, unlike the water samples, in which all 11 of the PAHs monitored were frequently found. In both June and October, phenanthrene, pyrene, and fluoranthene were found at between 20 and 65% of sediment sites and also had high combined concentrations [653].

Concentration ratios for fluoranthene/pyrene (F/PY) and phenanthrene/anthracene (P/A) have been widely used to determine sources of PAHs. Sediments from urban areas tend to have lower F/PY ratios than those from remote areas. Sediments from lakes in urban areas are prone to contaminations from urban runoff which contains both raw fuel and combustion sources, whereas sediments in remote sites are primarily contaminated by atmospheric deposition of combustion materials. For urban sites, F/PY ratios were found in the range of 0.95-1.4; the ratio shifted to higher values in the range of 1.2-1.7 for rural areas. Values of P/A in the range of 3-11 are indicative of an urbanized area, specifically urban runoff. Higher P/A values, eg >11, are indicative of remote sites that are primarily influenced by atmospheric deposition [653].

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

Sediments are major sinks for PAHs, primarily because of the low solubility of these compounds and their strong affinity for organic carbon in particulate matter. PAH concentrations in sediment are generally much higher than those detected in surface water, i.e., in the range of ug/kg (ppb) rather than ng/kg (ppt) [881].

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].

Sediment concentrations for total PAHs were generally in the range of 530-700 ug/kg, although concentrations in river and near-shore sediments reached nearly 4,000 ug/kg (4 ppm). Heit et al. (1981) reported total concentrations of PAHs (3-7 ring PAHs) from two lakes in the Adirondack acid lake region of 2,660 ug/kg and 770 ug/kg (calculated from data presented). Average concentrations of total PAHs in sediments from three coastal South Carolina marinas were reported to range from 35.6 to 352.3 ug/kg (Marcus et al. 1988). 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].

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).

Ontario Ministry of Environment Freshwater Sediment Guidelines for benthic organisms, 1993 [761]:

Lowest Effect Level = 4000 ug/kg dry weight

Severe Effect Level = 10,000 ug/kg organic carbon (to a maximum of 10% organic carbon)

New York Dept of Environmental Conservation, Freshwater Navigational Dredging; Interim Guidance for benthic organisms, 1994 [761]:

No Appreciable Contamination: <1000 ug/kg dry weight

Moderate Contamination: 1000-35,000 ug/kg dry weight

High Contamination: >35,000 ug/kg dry weight

St. Lawrence River Interim Freshwater Sediment Criteria for benthic organisms, 1993 [761]:

No Effect for LPAH (low PAH, molecular weight <200) = 100 ug/kg dry weight

No Effect for HPAH (high PAH, molecular weight >200) = 1,000 ug/kg dry weight

The calculation of concern levels for PAHs in sediments is complicated by the fact that so many

investigators have used different combinations of PAHs in their analyses [233]. 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 (1995) suggested that the potential for biological effects of Total PAH sorbed to sediments was highest in sediments where its concentration exceeded the 44,792 ppb dry weight Effects Range-Median (ERM) concentration and was lowest in sediments where its concentration was less than the 4,022 ppb dry weight Effects Range-Low (ERL) concentration [664]. For low-molecular weight PAHs (listed near beginning of this entry) NOAA reports an ERM of 3,160 ppb and an ERL of 552 ppb. For high-molecular weight PAHs (see list above), NOAA reports an ERM of 9,600 ppb and an ERL of 1,700 ppb [664].

To improve the original 1990 guidelines [233], the 1995 report included percent incidence of effects for ranges below, above, and between the ERL and ERM values. These numbers represent the percent of the total number of data entries within each concentration range in which biological effects were observed [664]:

For Total PAHs [664]:

<ERL	14.3
ERL-ERM	36.1
>ERM	85.0

For Low-molecular weight PAHs [664]:

<ERL	13.0
ERL-ERM	48.1
>ERM	100

For High-molecular weight PAHs [664]:

<ERL	10.5
ERL-ERM	40.0
>ERM	81.2

Sediment standards are not available for most individual PAHs, but a scientist working on the interagency task force developing sediment standards has stated that interim standards for many other PAHs will be in the 1-20 ppm range, and indications are that final criteria values may be much lower (Chris Ingersoll, U.S. Fish and Wildlife Service, personal communication, 1994).

Sed.Plants (Sediment Concentrations vs. Plants):

See Sed.Misc section below for general information

on PAH-sediment-plant interactions.

Sed.Invertebrates (Sediment Concentrations vs. Invertebrates):

No information found. See individual PAHs entries.

Sed.Fish (Sediment Concentrations vs. Fish):

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].

Downstream of a creosote superfund site (Eagle Harbor site in Puget Sound), concentrations of total PAHs above 1.0 ppm dry weight had positive correlations with incidence of liver cancer in fish [124,125, confirmed by Don Malins, Pacific Northwest Research Foundation, personal communication]. A total PAH dry weight concentration of 1.0 ppm might typically correspond to a wet weight concentration of about 0.5 ppm, a total carcinogenic PAH level of 0.083 to 0.166 ppm and an individual Benzo(a)pyrene concentration of perhaps 0.0267 to 0.0369 ppm.

Other researchers have also documented carcinogenic impacts from low levels of PAHs in sediments. For

example, sediments from the Buffalo River, New York with concentrations of total carcinogenic PAHs as low as 1.0 mg/kg induced tumors in brown bullhead catfish [40].

The concentration of total PAHs (16 individual PAH compounds) analyzed in the Hamilton Harbor sediment sample (137 ug/g dry wt) is similar to the concentrations of total PAHs reported for other areas where high prevalences of fish tumors have been observed, such as Black Rock Harbor, Connecticut (131 ug/g dry wt), the Black River entering Lake Erie (129 ug/g dry wt), and Eagle Harbor in Puget Sound (120 ug/g dry wt). However, hepatic neoplasms have also been observed in mummichog (Fundulus heteroclitus) from areas where PAH concentration in sediment are as high as 2,200 ug/g dry wt. On the other hand, hepatic neoplasms were observed in black croaker (Cheilotrema saturnum) from San Diego Bay, where the total PAH concentration in sediment was reported to be only 7 ug/g dry wt. Therefore, it appears that total PAH concentrations in sediment are not always a reliable indicator of the potential for the development of tumors in bottom-dwelling fish. Others have observed that PAH concentrations explained only 35% of the observed variation in neoplasm prevalence in English sole from various areas in Puget Sound [780; Reprinted with permission from Environmental Toxicology and Chemistry, Volume 14(1):79-91, Balch, G.C., C.D. Metcalfe and S.Y. Huestis, "Identification of Potential Fish Carcinogens in Sediment from Hamilton Harbor, Ontario, Canada", Copyright 1994 SETAC.].

The following information is for one representative individual PAH: 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].

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

No information found. See individual PAHs entries.

Sed.Human (Sediment Concentrations vs. Human):

No information found. See individual PAHs entries.

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

Over 190 PAH compounds were found in sediment samples from Galveston Bay (Brian Cain, FWS, personal communication, 1993).

In amphipod sediment toxicity tests conducted in an Exxon Valdez spill investigation, concentrations of 2-ring PAHs correlated better with toxicity than did concentrations of total PAHs or 4- and 5-ring PAHs. This was partly due to differences in water solubility and bioavailability (Personal communication, Bill Stubblefield, 1995).

In a study of the effects of motor boat activity on the water and sediment quality of a drinking water reservoir near Washington, D.C., only a few PAHs (like phenanthrene, pyrene, and fluoranthene) were commonly found in the sediments, unlike the water samples, in which all 11 of the PAHs monitored were frequently found [653].

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]. (Total organic carbon (TOC) in sediments influences the dose at which many compounds are toxic, PAHs (Dr. Denny Buckler, FWS Columbia, personal communication)).

There are many instances where well-documented bioconcentration factors (BCFs) or bioaccumulation factors (BAFs) are not available for chemicals and yet the literature gives us reason to believe that certain sediment concentrations of various chemicals are harmful to fish and wildlife. A good example is provided by the various chemicals classified as PAHs. Concentrations of total carcinogenic PAHs above 1.0 ppm have been associated with tumor formation in catfish exposed to sediments from the severely polluted Buffalo River in New York [40].

It is apparent that very low levels of PAHs in sediments can impact bottom fish, and so in some situations quite low detection limits are appropriate for analyses of PAHs in sediments. If researchers at the Eagle Harbor superfund site had used minimum detection limits for individual PAHs above 0.002 ppm, they would not have had the low level resolution to determine the 1.0 ppm total PAH level above which PAH concentrations were positively correlated with liver cancer in fish [124]. This provides a convincing argument for using the lowest possible minimum detection limits (1 ppb dry weight or lower is recommended for sediments and tissues) at sites where one suspects low levels of PAHs as important

contaminants.

PAHs can be absorbed from soils through plant roots and translocated to other parts of the plant [40]. PAHs accumulated in lab plants grown in contaminated soil [40]. Presumably this also occurs in sediments and aquatic plants [40].

Metabolic transformations of PAHs into even more hazardous chemicals can happen through microbial degradation of PAHs in soils or sediments. This provides an example of a situation where human health based standards may not be protective of biota living in soil or sediment.

In fish the greatest hazards of PAHs are their carcinogenic properties, especially with metabolites of PAHs such as benzo(a)pyrene (BaP), 7,12-dimethylbenz(a)anthracene, and 3-methylcholanthrene (3-MC). These metabolites are produced after metabolic bioactivation by the cytochrome P450 1A isoform (the cytochrome P450 1A isoform is a form of cytochrome P450) [793].

Survival of striped bass was significantly reduced in laboratory tests by exposing them to a mixture of organic (including PAH) and inorganic contaminants at concentrations similar to those found on east coast spawning grounds [89]. Doubling the concentrations of each contaminant in the mixture further increased the lethality of the mix and the bioaccumulation of PAHs [89].

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

Soil.Low (Soil Concentrations Considered Low):

No information found. See individual PAHs entries.

Soil.High (Soil Concentration Considered High):

The following information is for representative individual PAHs: Soil samples collected from the site of a Seattle coal and oil gasification site, which ceased operation in 1956, contained pyrene levels up to 4,300 ug/kg. Soil samples collected from the Fountain Avenue Landfill in New York contained total PAH concentrations of 400-10,000 ug/kg [788]. 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]:

Acenaphthene	1,368
Acenaphthylene	187
Anthracene	3,037
Benz(a)anthracene	397
Benzo(a)pyrene	159
Benzo(b)-fluoranthene	552
Benzo(k)-fluoranthene	446
Benzo(j)-fluoranthene	1.2
Benzo(g,h,i)perylene	16
Chrysene	1,586
Dibenz(a,h)anthracene	3,836
Fluoranthene	3,664
Fluorene	1,792
Indeno-(1,2,3-c,d)pyrene	316
Naphthalene	5,769
Phenanthrene	4,434
Pyrene	1,303

Soil.Typical (Soil Concentrations Considered Typical):

The following information is for representative individual PAHs: PAHs are ubiquitous in soil and are distributed globally. One study found benzo(g,h,i)perylene and fluoranthene at concentrations above 150 ug/kg in arctic soils. Soil samples collected from remote wooded areas of Wyoming contained total PAH concentrations of up to 210 ug/kg [788]. Total PAH concentrations of 4,000-8,000 ug/kg were found in the soil near a complex road interchange in Switzerland, while a level of 2,300 ug/kg was measured in an area removed from the road [788].

Five to fifteen ppm TPH (total petroleum hydrocarbons) in soil, although not a good measure of PAHs, is a realistic background level on a Texas intercoastal waterways spoil island (Brian Cain, Fish and Wildlife Service Contaminants Specialist, Houston, personal communication, 1995).

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

> PAHs are ubiquitous in soil. Because anthropogenic combustion processes are a major source of PAHs in soils, soil concentrations have tended to increase over the last 100-150 years, especially in urban areas (Jones et al. 1989a, 1989b). Background concentrations for rural, agricultural, and urban soils (from the United States and other countries) are below. In general, concentrations ranked as

follows: urban greater than agricultural greater than rural. Evidence of the global distribution of PAHs was given by Thomas (1986) who detected benzo[g,h,i]perylene and fluoranthene at concentrations above 150 ug/kg in arctic soils. Soil samples collected from remote wooded areas of Wyoming contained total PAH concentrations of up to 210 ug/kg [881].

Recent data on PAH concentrations in soil at contaminated sites are summarized in the table below. Because of the different sampling methods and locations at each site, this tabulation does not provide a reliable inter-site comparison. Additional studies indicate significantly elevated concentrations of PAHs at contaminated sites. Soil samples collected from the Fountain Avenue Landfill in New York City contained PAH concentrations ranging from 400 to 10,000 ug/kg (Black et al. 1989). 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 (Loehr et al. 1993). 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) in rural soil [881]:

Acenaphthene 1.7
Benzo(a)anthracene 5-20
Benzo(a)pyrene 2-1,300
Benzo(b)fluoranthene 0-30
Benzo(g,h,i)perylene 10-70
Benzo(k)fluoranthene 0-110
Chrysene 38.3
Fluoranthene 0.3-40
Ideno(1,2,3-c,d)pyrene 10-15
Phenanthrene 30.0
Pyrene 1-19.7

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

Acenaphthene 6
Acenaphthylene 5
Anthracene 11-13
Benzo(a)anthracene 5-20
Benzo(a)pyrene 4.6-900
Benzo(b)fluoranthene 58-200
Benzo(e)pyrene 53-130
Benzo(g,h,i)perylene 66
Benzo(k)fluoranthene 58-250
Chrysene 78-120
Fluoranthene 120-210
Fluorene 9.7
Ideno(1,2,3-c,d)pyrene 63-100
Phenanthrene 48-140
Pyrene 99-150

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

Benzo(a)anthracene 169-59,000
Benzo(a)pyrene 165-220
Benzo(b)fluoranthene 5,000-62,000
Benzo(e)pyrene 60-14,000
Benzo(g,h,i)perylene 900-47,000
Benzo(k)fluoranthene 300-26,000
Chrysene 251-640
Fluoranthene 200-166,000
Ideno(1,2,3-c,d)pyrene 8,000-61,000
Phenanthrene 48-140
Pyrene 145-147,000

Above values derived from (see ATSDR [881] for details): IARC 1973; White and Vanderslice 1980; Windsor and Hites 1979; Edwards 1983; Butler et al. 1984; Vogt et al. 1987; and Jones et al. 1987 [881].

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):

No information found. See individual PAHs entries.

Soil.Plants (Soil Concentrations vs. Plants):

See Soil.Misc section below for general information on PAH-soil-plant interactions.

Soil.Invertebrates (Soil Concentrations vs. Invertebrates):

No information found. See individual PAHs entries.

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

No information found. See individual PAHs entries.

Soil.Human (Soil Concentrations vs. Human):

Metabolic transformations of PAHs into even more hazardous chemicals could also happen through microbial degradation of PAHs in soils or sediments. See also Br.Car. section above for problems in adding carcinogenic vs. non-carcinogenic PAHs.

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

PAHs are ubiquitous in soil. In general, PAH concentrations in soils rank as follows: urban > agricultural > rural [788].

PAHs may also be translocated in plants and may accumulate in plants grown in contaminated soil [40]. Presumably this also occurs in sediments and aquatic plants and therefore might impact herbivorous species of fish and wildlife. Although some research seems to indicate that interior portions of above-ground vegetables do not accumulate high concentrations of PAHs, plants do translocate PAHs from roots to other plant parts, such as developing shoots [40].

In a series of soil and hydrocultures of the higher plants, tobacco, rye, and radish, as well as algae cultures of lower plants (Chlorella vulgaris, Scenedesmus obligurus, and Ankistrodesmus) /results indicate/ that certain polycyclic aromatic hydrocarbons have growth-promoting effects on plants. Further, the degree of the promoting effect corresponded to the oncogenic activity of the hydrocarbon. The six polycyclic aromatic hydrocarbons found in plants were tested one at a time or in combination. Considerable growth-promotion was noted (near to 100% in some cases) with the effectiveness of hydrocarbons ranked as follows: (1) Benzo(a)pyrene (2) Benzo(a)anthracene (3) Indeno (1,2,3-cd)pyrene, Benzo(b)fluoranthene (4) Fluoranthene (5)

Benzo(ghi)perylene. [Graf W, Nowak W; Arch Hyg Bakt 150: 513-28 (1968) as cited in Health & Welfare Canada; Polycyclic Aromatic Hydrocarbons p.67 (1979) Report No. 80-EHD-50] [366].

Guddal (1969) first reported the isolation of anthracene, pyrene, and fluoranthene from chrysanthemum roots grown in contaminated soils near a gasworks. These findings stimulated interest in the PAH content of soils and plants as well as the possible endogenous formation in plants and microorganisms. In a 1983 publication, investigators concluded that on the basis of available data, three routes of PAH passage into plants have been considered: air deposition, adsorption from soil and water, and biochemical synthesis. There appears to be general agreement that available data support the first two routes; however, there is considerable controversy over the synthesis route [794].

Misc. References:

Author GF Fries Title Ingestion of sludge applied organic chemicals by animals Source Science of the Total Environment 185: 1-3 (JUN 21 1996) Page(s) 93-108

Author AJ Beck, DL Johnson, KC Jones Title The form and bioavailability of non-ionic organic chemicals in sewage sludge-amended agricultural soils Source Science of the Total Environment 185: 1-3 (JUN 21 1996) Page(s) 125-149

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. See individual PAHs entries.

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

No information found. See individual PAHs entries.

Tis.Invertebrates:

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

No information found. See individual PAHs entries.

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

No information found. See individual PAHs entries.

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

NOAA National Status and Trends (NS&T) Program (1988-1993): Geometric mean ("typical") and two "high" (**) concentrations of total PAHs in mollusks collected from about 190 sites in coastal and estuarine areas of the U.S. (units = ng/g dry wt) [787]:

YEAR	CONCENTRATION
1988	350
1989	310
1990	270
1990	260 [697]
1990	1020**
1990	890** [697]
1991	250
1992	280
1993	300

** = "high" is defined as the mean plus one standard deviation of the logarithms of the individual site means [787].
* Total PAH concentration is sum of 24 individual PAH compounds: biphenyl, naphthalene, 1-methylnaphthalene, 2-methylnaphthalene, 2,6-dimethylnaphthalene, acenaphthene, fluorene, phenanthrene, 1-methylphenanthrene, anthracene, fluoranthene, pyrene, benz(a)anthracene, chrysene, benzo(a)pyrene, benzo(e)pyrene, perylene, dibenz(a,h)anthracene, 1,6,7-trimethylnaphthalene, acenaphthylene, benzo(b)fluoranthene, benzo(ghi)perylene, benzo(k)fluoranthene, and indeno(1,2,3-cd)pyrene. [697] values do not include the last six compounds.
* Samples were whole soft parts of oysters and mussels
* includes sites on east and west coasts, Gulf of Mexico, Alaska and Hawaii; half the sites in rural areas, and half near urban areas (within 20km of cities in excess of 100,000 people, but away from obvious "hot spots").

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 as yet.

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

No information found for total PAHs. The following information is for one representative individual PAH: 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.

TYPICAL TISSUE CONCENTRATIONS IN TEXAS:
The following text is quoted from the Trinity River Report [201] for reference comparison with values from other areas. PAHs were analyzed in only 33 Trinity River samples due to cost:

MAXIMUM LEVELS: The highest PAH concentrations in our study were from a composite whole-body sample of 100 mosquitofish from site 9 at south Loop 12 just downstream of Dallas Central Sewage Treatment plant. The concentrations of total PAHs in mosquitofish from site 9 was 60.79 mg/kg. This is an extremely elevated level and was confirmed by duplicate GC/mass spectrometry analysis.

The individual PAHs detected in this mosquitofish sample (and their respective concentrations in mg/kg listed in parenthesis) included naphthalene (0.19); fluorene (0.50); phenanthrene (7.2); anthracene (1.1); fluoranthene (10); pyrene (8.7); 1, 2-benzanthracene (5.3); chrysene (4.9); benzo(b)fluoranthene (3.5); benzo(k)fluoranthene (3.2); benzo(e)pyrene (1.2); 1,2,5,6-dibenzanthracene (1.6); benzo(g,h,i) perylene (8.1); and benzo(a)pyrene (5.3). These concentrations, including those of the heavier PAHs like benzo(a)pyrene, are much higher than those implicated in a high incidence of liver cancer

in bullhead catfish from a severely polluted river in Ohio [40,87]. The next highest concentrations of total PAHs were 0.75 mg/kg, in a composite whole-body sample of smallmouth buffalo fish from site 11 (one mile downstream), and 0.51 mg/kg from a composite whole-body sample of redfin shiners from site 18, a storm drain site in downtown Fort Worth. At the time of our collections, there was a slick of used motor oil on the surface of the water at site 18. Soon after we notified them of the oil slick at site 18, the City of Fort Worth Health Department discovered that a new car dealer in downtown Fort Worth was illegally dumping used motor oil into a storm drain which flowed directly to the river at that site.

All other elevated levels of total PAHs (>0.1 mg/kg) were also either from sites downstream of Dallas (3 mosquitofish samples, one carp sample, and one longnose gar sample) or from site 18 (one fatty tissue sample dissected from Texas cooter turtles). Previous studies by other agencies have reported concentrations of PAHs in sediments from site 9 in the 0.5 to 0.7 mg/kg range.

All 13 Trinity River samples from relatively clean waters upstream of Dallas or Fort Worth had total PAH levels below 0.05 mg/kg. Concentrations in this range may be the result of atmospheric fallout from nearby urban sources.

GRADIENT MONITORING LEVELS:
Regarding mosquitofish PAH residues, sites 9, 10, and 15 had mosquitofish levels of PAHs higher than those reported [65] from a Pecos River site which experiences recurrent oil pollution. However, only site 9 had levels above the surprisingly high levels reported from mosquitofish from the Rio Grande River at Big Bend National Park [65].

The high quantity of benzo(a)pyrene in our mosquitofish sample from site 9 is of concern because quantities this high are very unusual in fish; the carcinogenic PAHs usually are broken down quickly in the liver [40]. Analyses of PAHs in fish tissues often show only traces of PAHs even when the sediments contain high concentrations of these compounds [70].

Greatly elevated concentrations of PAHs in fish or sediments can be indicative of localized contaminant hot spots, with much lower concentrations a half mile downstream (Brian Cain, personal communication). Environmental degradation of PAHs such as fluorene can be reduced by low dissolved oxygen and low algal productivity [92]. Both conditions are common at our site 9, where the highest concentrations of PAHs were found.

Our report on Big Bend National Park [65] showed generally lower (but surprisingly present) PAH concentrations from a rural stretch of the Rio Grande.

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. See individual PAHs entries.

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):

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].

Male and female mice were exposed to 0, 175, 350, or 700 mg/kg/day acenaphthene by gavage for 13 weeks [881]. No signs of cardiovascular distress were seen during life for any dose group, and no gross or microscopic damage was seen upon necropsy [881]. Similar findings were reported after 13-week administration of 1,000 mg/kg/day anthracene, and 500 mg/kg/day fluoranthene, or 500 mg/kg/day fluorene [881].

No other information found. See individual PAHs entries.

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

Marine vertebrates commonly have trace levels of PAHs in their tissues [713]. Few data on PAHs in marine mammals are available [713].

No information found for total PAHs or PAHs in general. The following information is for representative individual PAHs:

In one study, four species of seals and six species of whales were collected. Skeletal muscle tissue from the mid dorsal region was analyzed (using fluorimetry). Values ranged from 0.10 to 1.21 ppm dry wt in terms of chrysene equivalents and 0.26 to 5.51 ppm dry wt in terms of petroleum equivalents. Although most PAH concentration values were low, a few high values from fishing areas demonstrate the need for more comprehensive information on PAHs in marine mammals. Baseline levels of hydrocarbons in marine mammal species worldwide are needed [713].

After the Exxon Valdez spill, one yearling brown bear (Ursus arctos) found dead had elevated bile naphthalene and phenanthrene concentrations (160,000 ppb and 18,000 ppb respectively) [713].

Tis.Human:

A) Typical Concentrations in Human Food Survey Items:

The level of PAHs in the typical U.S [881]. diet is less than 2 parts of total PAHs per billion parts of food (ppb), or less than 2 micrograms per kilogram of food (ug/kg; a microgram is one-thousandth of a milligram) [881].

PAHs have been reported in smoked fish and meats, grilled and roasted foods, root and leaf vegetables, vegetable oils, grains, plants, fruits, seafoods, whiskeys, etc. Sources of such contamination include curing smokes, contaminated oils, polluted air and water, modes of cooking, processing, or food preparation, food additives, and endogenic or biosynthesis by plants and microorganisms [794]. Differences in hydrocarbon content may be ascribed to the many variables involved in the smoking process, including type of generator, combustion temperature, and degree of smoking. Two studies found that protective coverings, including loose cotton fabrics and especially cellophane, reduced the PAH content of the smoked meats significantly [794].

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):

Daily intake of total PAHs from food should not exceed 1.6 to 16 ug [40]. Daily intake of total PAHs from all sources should not exceed 1.7 to 16.6 ug [40].

EPA has suggested that taking into your body each day the following amounts of individual PAHs is not likely to cause any harmful health effects: 0.3 milligrams (mg) of anthracene, 0.06 mg of acenaphthene, 0.04 mg of fluoranthene, 0.04 mg of fluorene, and 0.03 mg of pyrene per kilogram (kg) of your body weight (one kilogram is equal to 2.2 pounds) [881]. Actual exposure for most of the United States population occurs from active or passive inhalation of the compounds in tobacco smoke, wood smoke, and contaminated air, and from eating the compounds in foods [881]. Skin contact with contaminated water, soot, tar, and soil may also occur [881]. Estimates for total exposure in the United States population have been listed as 3 mg/day [881].

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

EPA has suggested that taking into your body each day the following amounts of individual PAHs is not

likely to cause any significant (noncancer) harmful health effects: 0.3 mg anthracene, 0.06 mg acenaphthene, 0.04 mg fluoranthene, 0.04 mg fluorene, and 0.03 mg pyrene per kg body weight [788].

Misc. References:

Author DH Phillips Title DNA adducts in human tissues: Biomarkers of exposure to carcinogens in tobacco smoke Source Environmental Health Perspectives 104: Suppl. 3 (MAY 1996) Page(s) 453-458

Author RJ Albertini, JA Nicklas, JP Oneill Title Future research directions for evaluating human genetic and cancer risk from environmental exposures Source Environmental Health Perspectives 104: Suppl. 3 (MAY 1996) Page(s) 503-510

Author T Kuljukka, R Vaaranrinta, T Veidebaum, M Sorsa, K Peltonen Title Exposure to PAH compounds among cokery workers in the oil shale industry Source Environmental Health Perspectives 104: Suppl. 3 (MAY 1996) Page(s) 539-541

Author H Autrup, AB Vestergaard Title Transplacental transfer of environmental genotoxins - Polycyclic aromatic hydrocarbon-albumin in nonsmoking women Source Environmental Health Perspectives 104: Suppl. 3 (MAY 1996) Page(s) 625-627

Author A Verdina, R Zito, G Cortese, A Zijno, R Crebelli Title Modulation of DNA binding in vivo by specific humoral immunological response: A novel host factor in environmental carcinogenesis? Source Environmental Health Perspectives 104: Suppl. 3 (MAY 1996) Page(s) 679-682

Author JH Vanwijnen, R Slob, G Jongmansliedekerken, RHJ Vandeweerdt, F Woudenberg Title Exposure to polycyclic aromatic hydrocarbons among Dutch children Source Environmental Health Perspectives 104: 5 (MAY 1996) Page(s) 530-534

Tis.Misc. (Other Tissue Information):

From the available data, there is little apparent correlation between PAH concentrations in aquatic

organisms and their phylatic (taxonomic) position, trophic status and preferred habitat [754].

One researcher found that hydrocarbon concentrations in ringed seals declined less rapidly in muscle than in liver or blubber tissues. Although fat and liver may have higher concentrations of hydrocarbons, muscle tissue may be a better indication of hydrocarbon concentrations over time [713].

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].

Guddal (1969) first reported the isolation of anthracene, pyrene, and fluoranthene from chrysanthemum roots grown in contaminated soils near a gasworks. These findings stimulated interest in the PAH content of soils and plants as well as the possible endogenous formation in plants and microorganisms. In a 1983 publication, investigators concluded that on the basis of available data, three routes of PAH passage into plants have been considered: air deposition, adsorption from soil and water, and biochemical synthesis. There appears to be general agreement that available data support the first two routes; however, there is considerable controversy over the synthesis route [794].

Studies conducted at atmosphere-polluted areas in the United States and Germany noted a strong relationship between air pollution and the occurrence of benzo(a)pyrene in grains and vegetables [794].

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

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 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).

Bioaccumulation relates to Kow, the higher the number, the more tendency to bioccumulate.

Log Kow values for PAHs [971]:

Naphthalene:	3.37
C1-Naphthalene:	3.87
C2-Naphthalene:	4.37

C3-Naphthalene:	5.0
C4-Naphthalene:	5.55
Acenaphthylene:	4.07
Acenaphthene:	3.92
Fluorene:	4.18
C1-Fluorene:	4.97
C2-Fluorene:	5.2
C3-Fluorene:	5.5
Anthracene:	4.54
Phenanthrene:	4.57
C1-Phenanthrene:	5.14
C2-Phenanthrene:	5.51
C3-Phenanthrene:	6
C4-Phenanthrene:	6.51
Dibenzothiophene:	4.49
C1-Dibenzothiophene:	4.86
C2-Dibenzothiophene:	5.5
C3-Dibenzothiophene:	5.73
Fluoranthene:	5.22
Pyrene:	5.18
C1-Fluoranthene/pyrene:	5.72
Benzo(a)anthracene:	5.91
Chrysene:	5.86
C1-Chrysene:	6.42
C2-Chrysene:	6.88
C3-Chrysene:	7.44
C4-Chrysene:	8
Benzo(b)fluoranthene	5.80
Benzo(k)fluoranthene:	6.0
Benzo(a)pyrene:	6.04
Indeno(1,2,3-c,d)pyrene:	7.0
Dibenz(a,h)anthracene:	6.75
Benzo(g,h,i)perylene:	6.50

Aquatic organisms can bioaccumulate some PAH compounds [70]. Species lower down on the food chain, such as certain zooplankton, phytoplankton, and invertebrates (like mussels and molluscs) can bioaccumulate PAHs. They will lose much of the accumulated hydrocarbon products if clean water is again available. However, if oil exposure is chronic, the hydrocarbons may enter more stable tissue (like depot lipids) and as long as the animal is in positive nutritional balance, it will only very slowly release the hydrocarbons [713].

Biomagnification of petroleum hydrocarbons through the food chain has not been demonstrated in marine mammals, probably due to their cytochrome P450 system [713]. The bioaccumulation and persistence of PAHs in the food chain is opposite that seen for other chemicals such as some PCBs and certain other organochlorines which tend to concentrate in the top predators. Because it is the species lower in the food chain that concentrate PAHs, those species (like bowhead whales and walrus) that feed at that lower level are at higher risk of bioaccumulation than species (like killer whales) that feed higher in the food chain on fish. Fish also have an enzyme system for clearing hydrocarbons, thus they are

not likely to bioaccumulate hydrocarbons. However, colder waters can slow down the metabolism and elimination of hydrocarbons, thus animals feeding in arctic waters have a greater chance of bioaccumulating some hydrocarbons [713].

In model ecosystems, high-molecular-weight PAHs were accumulated and magnified in lower organisms; however, fish were usually able to metabolize and depurate the compounds relatively rapidly, precluding biomagnification as a significant process. In general, although uptake of both low- and high-molecular-weight PAHs is relatively rapid in vertebrate species, metabolism and depuration are also rapid. Estimates of whole-body half-lives for a variety of PAHs were usually less than 7 d in a number of different fish species. Hence, bioaccumulation of PAHs, at least for vertebrate species, is considered to be relatively short-term in the aquatic environment [754].

Half-lives for elimination of PAHs in fish ranged from less than 2 days to 9 days [788].

Although some PAHs have relatively high calculated octanol/water partition coefficients, measured bioaccumulation factors are lower than would be predicted. This may be the result of the difficulty in differentiation truly dissolved PAHs from "total" PAHs in the water. Bioconcentration factors of 100 to 1000 were reported for Daphnia pulex, with factors increasing with molecular weight. Bioconcentration factors of 100 to 10,000 were found for sediment and biota relative to water for benzo(a)pyrene. Oysters had bioconcentration factors of 1000 for PAHs with three rings [754].

BCFs of PAHs in fish and crustaceans have frequently been reported to be in the range of 100-2000. One study reported that, in general, bioconcentration was greater for the higher molecular weight compounds than for the lower molecular weight compounds [788].

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

Bioconcentration factors for benzo(a)pyrene range from 930 in the mosquitofish (Gambusia affinis) to 134,240 in Daphnia pulex. Bioconcentration factors for anthracene range up to 3500 in the mayfly (Hexagenia sp.) [754].

There is ample evidence that fish exposed to petroleum in sediments, water, or through the diet, accumulate hydrocarbons in tissues and body fluids. Some of the aromatic hydrocarbons are converted metabolically to metabolites that remain in tissues for prolonged periods. PAH ingestion by fish is more apt to reach critical organs if it comes across gill membranes than if it is ingested into the gut (Denny Buckler, FWS Columbia, personal communication).

Interactions:

Phototoxicity: PAHs exhibiting greatly enhanced toxicity to both fish and zooplankton species in the presence of solar radiation include [779,887]:

Anthracene
Benzo(a)pyrene.

PAHs showing substantially increased toxicity to Daphnia magna zooplankton in the presence of solar radiation include [887]:

Acridine
Benzanthrone
Benzo(a)anthracene
Benzo(b)anthracene,
Benzo(k)fluoranthene
Benzo(a)fluorene
Benzo(b)fluorene
Benzo(ghi)perylene
Benzo(e)pyrene
Chrysene
Dibenzo(ah)anthracene
Fluoranthene
Pyrene

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PAHs showing some (minor) increased toxicity to Daphnia magna zooplankton in the presence of solar radiation include [887]:

Benzo(a)fluorene
Perylene

Many other PAHs would be expected to be associated with phototoxicity based on physicochemical properties [887]. However, PAHs and alkyl PAHs have not been tested for phototoxicity, and water concentrations of PAHs have not been considered a big problem partly because no toxicity had been observed even at solubility concentrations in lab tests of some PAHs in the absence of sunlight or UV radiation [887].

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Those PAHs exhibiting no phytotoxicity to Daphnia magna zooplankton include [887]:

Carbazole
Fluorene
Phenanthrene
Triphenylene

Short wave length, visible, and ultraviolet solar radiation of sufficient energy to cause photo-enhanced toxicity can reach to significant depths in the hydrosphere [887]. Organisms at risk from photo-enhanced toxicity of PAHs could potentially include those living or feeding in PAH contaminated sediments, fish eating PAH contaminated invertebrates, fish or amphibian eggs and larvae in shallow water or the surface microlayer, and any other biota exposed to PAHs and solar radiation. Photo-enhanced toxicity may involve some of the same mechanisms as carcinogenic potential [887].

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Studies in illuminated stream microcosms have shown both juvenile bluegill sunfish and the invertebrate (zooplankton) Daphnia pulex to be hundreds of times more sensitive to the toxic effects of anthracene than would be the case in the absence of UV or other types of solar radiation [779]. Anthracene was acutely toxic (100% mortality) to the bluegill at concentrations of 12 ug/L in less than 9 hrs [779]. Twelve ug/L is an environmental relevant concentration, since the water solubility of anthracene at 25 degrees C is 43-75 ug/L [848].

Although high-molecular-weight PAHs are associated primarily with particulate matter, a wide variety of soluble organic substances, including humic substances, may solubilize them and, hence, increase their aqueous mobility [754].

Many PAHs are not carcinogenic; some of them may act as synergists and some as competitive inhibitors [794].

Creosote, which was made in the U.S. by distillation of wood tar, is a complex mixture of PAHs, phenolic compounds, guaiacol, cresol, methylcresol, dioxins, dibenzofurans, and other chemicals [366]. Recent research suggests that at least one of the dioxins previously thought to be a very potent carcinogen may actually be a "promoter" of carcinogenesis [403] when cancer is initiated by another carcinogen (such as the many carcinogenic PAHs present in creosote). Thus it is inappropriate to single out only certain high level PAHs in a carcinogenic risk analysis when there are other carcinogens or cancer promoters such as dioxins, dibenzofurans, and nitrogen-containing aromatic compounds (NCACs) present in creosote. Mixtures of these compounds may be much more hazardous than any single component, especially if understanding of the hazards posed by the components is limited by the use of higher than optimal detection limits in initial site surveys.

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

Uses/Sources:

PAHs are generally not produced commercially in the United

States except as research chemicals [881]. However, PAHs are found in coal, coal tar, and in the creosote oils, oil mists, and pitches formed from the distillation of coal tars [881].

A few PAHs are used in medicines and to make dyes, plastics, and pesticides [881]. Others are contained in asphalt used in road construction [881]. They can also be found in substances such as crude oil, coal, tar pitch, creosote, and roofing tar [881]. They are found throughout the environment in the air, water, and soil [881]. They can occur in the air, either attached to dust particles or as solids in soil or sediment [881].

PAHs enter the environment mostly as releases to air from volcanoes, forest fires, residential wood burning, and exhaust from automobiles and trucks [881]. They can also enter surface water through discharges from industrial plants and waste water treatment plants, and they can be released to soils at hazardous waste sites if they escape from storage containers [881].

The primary sources of exposure to PAHs for most of the U.S [881]. population are inhalation of the compounds in tobacco smoke, wood smoke, and ambient air, and consumption of PAHs in foods [881]. For some people, the primary exposure to PAHs occurs in the workplace [881]. PAHs have been found in coal tar production plants, coking plants, bitumen and asphalt production plants, coal-gasification sites, smoke houses, aluminum production plants, coal tarring facilities, and municipal trash incinerators [881]. Workers may be exposed to PAHs by inhaling engine exhaust and by using products that contain PAHs in a variety of industries such as mining, oil refining, metalworking, chemical production, transportation, and the electrical industry [881]. PAHs have also been found in other facilities where petroleum, petroleum products, or coal are used or where wood, cellulose, corn, or oil are burned [881]. People living near waste sites containing PAHs may be exposed through contact with contaminated air, water, and soil [881].

Some PAHs and PACs (polyaromatic compounds) are often from petroleum sources while others are typically the result of incomplete combustion. Many can originate from many different sources. The most important petroleum-source PAHs/PACs include (all are on the expanded scan) [468]:

Chrysene (CAS Number 218-01-9): Present in some heavy crudes in significant amounts; also important are the homologous series compounds (alkylated compounds C1 through C4).

Fluorene (CAS Number 86-73-7): Present in significant amounts, especially the homologous series (alkylated compounds C1 through C3).

Naphthalene (CAS Number 91-20-3): Often present in significant amounts; also important are the homologous series compounds (alkylated compounds C1 through C4).

Phenanthrene (CAS Number 85-01-8): Often present in significant amounts; also important are the homologous series compounds (alkylated compounds C1 through C4).

Anthracene (CAS Number 120-12-7): Present in petroleum but usually not very high quantities.

Dibenzothiophene: Can be present in significant amounts, especially the homologous series (alkylated compounds C1 through C3). Note: Dibenzothiophene and related compounds are not universally considered to be PAHS.

Many of the entries for individual PAHs are quite long and additional information on uses and sources of PAHs is found in these compound-specific entries. PAHs originate from many sources, including pyrogenic (often from incomplete combustion of petroleum or woody products), motor oil (both new and used), various petroleum based fuels including crude oils (see Chemical/Physical section above), oil drips off of motor vehicles, and weathering or eroding asphalt or tires.

Urban runoff is a major source of PAHs, the biggest source in Chesapeake Bay, for example, and includes components from spilled oils and breakdown components from tires and asphalt (Greg Foster, George Mason University, personal communication, 1996). Man-induced and/or man-controlled combustion processes which contribute PAHs include the burning of coal, production of coke in the iron and steel industry, catalytic cracking of petroleum, heating and power generation, emissions from transportation vehicles, asphalt paving, and coal tar pitch [794].

Petrogenic (related to crude oil and its products) PAHs characteristically have a greater percentage of alkyl PAHs compared to parent compounds, while pyrogenic (generated by high temperatures) PAHs tend to have a predominance of parent compound PAHs compared to alkyl PAHs [942]. There are a few polynuclear aromatic hydrocarbons found in nature, such as retene, perylene, and 3,4-benzopyrene [500]. However, it is not always easy to determine if a particular PAH is from natural or anthropogenic sources. Consider the case of perylene:

Perylene is not universally considered a "pyrogenic" (resulting from high temperatures) PAH or a "petrogenic" (coming from crude oil or oil products) PAH [942]. In sediments perylene can be formed from early diagenesis of plant pigments so its presence in sediments is not necessarily indicative of anthropogenic contamination (Gregory Foster, George Mason University, personal communication 1995. Perylene has been said to be one of the few polynuclear aromatic hydrocarbons found in nature [500].

However, the statement that perylene is not petrogenic may be somewhat misleading. Perylene does occur in petroleum products, including:

South LA crude [177],

Fuel Oil 5 (Chuck Rafkind, National Park Service, Personal Communication, 1996),

Sediments contaminated by Diesel Fuel (1D and 2D) (Ray Ahlbrandt, National Park Service, Personal Communication, 1996),

Used Engine Oil [519].

Other parent compound PAHs often considered pyrogenic, PAHs which are not found predominantly in alkyl versions, also occur in crude and many of the same products listed above, so finding them does not necessarily rule out petrogenic sources.

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].

Concentration ratios for fluoranthene/pyrene (F/PY) and phenanthrene/anthracene (P/A) have been widely used to determine sources of PAHs. Sediments from urban areas tend to have lower F/PY ratios than those from remote areas [653].

Fingerprinting of combinations of PAHs and degree of alkylation was used to distinguish between Exxon Valdez crude oils and natural seep oils in Alaska [942].

During the summer of 1996, NASA announced that PAHs had been found on a rock alleged to be 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. Petrogenic (related to crude oil and its products) PAHs characteristically have a greater percentage of alkyl PAHs compared to parent compounds, while pyrogenic (generated by

high temperatures) PAHs tend to have a predominance of parent compound PAHs compared to alkyl PAHs [942]. This is also true of signatures of PAHs from earthly origins.

Some of the PAHs in isolated surface waters are believed to result from atmospheric deposition. However, for any given body of water, the major source of PAHs could vary [788].

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]. Used motor oil typically has a much higher concentration of PAHs than new motor oil [40,519]. Used crankcase oil was identified as the main source of PAHs and related sulfur heterocyclics in stormwater in Philadelphia, PA [207].

Creosote, which was made in the U.S. by distillation of wood tar, is a complex mixture of PAHs, phenolic compounds, guaiacol, cresol, methylcresol, dioxins, dibenzofurans, and other chemicals [366].

In rural areas a considerable portion of PAHs in streams comes from highways [43]. Citizens or businesses who illegally pour used motor oil into storm drains are polluting urban rivers with PAHs. Aquatic environments also receive PAHs from sewage treatment plants or atmospheric deposition [40].

Although PAHs occur in smoked or barbecued fish and can occur naturally in the environment in small concentrations, raw fish from unpolluted areas usually do not contain detectable concentrations of PAHs [40]. Highly elevated concentrations of PAHs in the environment are usually the result of contamination from petroleum products or various industrial or combustion activities [40].

The most important hazardous components of most gasolines are PAHs, alkyl PAHs, the BTEX compounds, and additives [560]. Some gasolines have more content of PAH and alkyl PAH compounds than other gasolines [560].

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

> There is a relatively large body of data characterizing PAH air levels at a variety of U.S. sites. Caution must be used in interpreting and comparing results of different studies, however, because of the different sampling methods used. PAHs occur in the atmosphere in both the particle phase and the vapor phase [881].
>
> Three-ring PAH compounds are found in the atmosphere primarily in the gaseous phase, whereas, five- and six-ring PAHs are found mainly in the particle phase; four-ring PAH compounds are found in both phases. To fully characterize atmospheric PAH levels, both particle- and vapor-phase samples must be collected. Many of the earlier monitoring studies used filter sampling methods, which provided information on particle-phase PAH concentrations only, and which did not account for

losses of some of the lower molecular weight PAHs by volatilization. As a result, the early use of particulate samples may have resulted in an underestimation of total PAH concentrations. More recent monitoring studies often use sampling methods that collect both particle- and vapor-phase PAHs and that prevent or minimize volatilization losses, thus providing more reliable characterization of total atmospheric PAH concentrations (Baek et al. 1991) [881].

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].

More recently, Greenberg et al. (1985) evaluated atmospheric concentrations of particulate phase PAHs at four New Jersey sites (three urban and one rural) over two summer and winter seasons during 1981-82. Urban PAH concentrations were approximately 3-5 times higher than those at the rural site; in addition, winter concentrations were approximately 5-10 times higher than summer concentrations. Geometric mean concentrations of ten PAHs (benzo[a]pyrene, benzo[k]fluoranthene, benzo[b]fluoranthene, benzo[k]fluoranthene, bins[a]enthrones, indeno[1,2,3-c,d]pyrene, benzo[g,h,i]perylene, pyrene, and chrysene) ranged from 0.03 to 0.62 ng/m3 in urban areas and from 0.01 to 0.12 ng/m3 in the rural area during the summer seasons. During the winter seasons, geometric mean concentrations of these PAHs ranged from 0.40 to 11.15 ng/m3 in urban areas and from 0.08 to 1.32 ng/m3 in the rural area. Geometric mean concentrations of benzo[a]pyrene ranged from 0.11 to 0.23 ng/m3 (urban) and 0.04 to 0.06 ng/m3 (rural) during the summer seasons, and from 0.69 to 1.63 ng/m3 (urban) and 0.17 to 0.32 (rural) during the winter seasons. A more extensive study by Harkov and Greenberg (1985) of atmospheric benzo[a]pyrene concentrations at 27 New Jersey sites indicated similar differences in mean urban (0.6 ng/m3) and rural (0.3 ng/m3) concentrations. Significant seasonal trends were also observed, with mean benzo[a]pyrene concentrations during the winter more than an order of magnitude greater than during the summer [881].

Several other studies provide evidence that atmospheric concentrations of particle-phase PAHs are higher in winter than in summer. In a 1981-82 study conducted in the Los Angeles area, atmospheric concentrations of 10 PAHs (anthracene, fluoranthene, pyrene, chrysene,

benz[a]anthracene, combined benzo[a]pyrene and perylene, benzo[b]fluoranthene, benzo[k]fluoranthene, benzo[a]pyrene, and combined benzo[g,h,i]perylene, and indeno[1,2,3-c,d]pyrene) ranged from 0.14 to 1.45 ng/m3 (with an average of 0.43 ng/m3) during the summer (August-September), and from 0.40 to 4.46 ng/m3 (with an average of 1.28 ng/m3 during the winter (February-March) (Grosjean 1983). A similar seasonal variation in particle-phase PAH concentrations in the Los Angeles atmosphere was seen in an earlier 1974-75 study (Gordon 1976). Quarterly geometric mean concentrations of 11 PAHs (pyrene, fluoranthene, benz[a]anthracene, chrysene, benzo[a]pyrene, benzo[e]pyrene, benzo[b]fluoranthene, benzo[j]fluoranthene, benzo[k]fluoranthene, benzo[g,h,i]perylene, and indeno[1,2,3-c,d]pyrene) ranged from 0.06 to 2.71 ng/m3 (with an average of 0.45 ng/m3) during the May-October period, and from 0.26 to 8.25 ng/m3 (with an average of 1.46 ng/m3) during the November-April period. The highest and lowest concentrations were observed during the fourth (November-January) and second (May-July) quarters, respectively. Ratios of fourth quarterly and second quarterly geometric mean concentrations ranged from 3.9 for indeno[1,2,3-c,d]pyrene to 7.5 for benzo[a]pyrene and 9.8 for benz[a]anthracene. Possible factors contributing to these seasonal variations in PAH levels include the following: changes in emission patterns; changes in meteorological conditions (i.e., daylight hours and temperature); and changes in space heating emissions, volatilization, and photochemical activity [881].

Certain monitoring data suggest that ambient levels of some PAHs may be decreasing. Faoro and Manning (1981) analyzed a limited sample of U.S. National Air Surveillance Network data updated through 1977, which indicated that benzo[a]pyrene concentrations have shown consistent, sizable declines during the period from 1967 to 1977 at 26 urban sites and 3 background sites studied (data not provided) [881].

Over the past two decades, the ambient air levels of PAHs in a number of major cities have been characterized. Although data from studies in different areas cannot be used to indicate definitive temporal trends in PAH air levels, a comparison of the results of these studies yields no strong suggestion that the ambient air levels of PAHs may be decreasing, except in traffic tunnels [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. Similar results were obtained in an obtained in an earlier (1974-1975) study of atmospheric particle-phase PAHs in the Los Angeles area, where ambient annual geometric mean concentrations ranged from 0.17 ng/m3 for benzo[j]fluoranthene to 3.27 ng/m3 for benzo[g,h,i]perylene (Gordon 1976). The annual geometric mean concentration of benzo[a]pyrene was 0.46 ng/m3; most individual PAHs had annual geometric mean concentrations of less than 0.6 ng/m3. The relatively high levels of benzo[g,h,i]perylene found in these studies have been attributed to high levels of automobile emissions, which are known to contain high levels of benzo[g,h,i]perylene relative to other PAHs (Santodonato et al. 1981). During the same time period, Fox and Staley (1976) reported somewhat higher ambient average concentrations of particle-phase PAHs in College Park, Maryland, ranging from 3.2 ng/m3 for benzo[a]pyrene to 5.2 ng/m3 for pyrene [881].

In a 1985-86 study, reported average ambient concentrations (combined particle- and vapor-phase) of eight PAHs in Denver ranged between 0.83 ng/m3 for benzo[k]fluoranthene and 39 ng/m3 for phenanthrene (Foreman and Bidleman 1990). In a study conducted in Hamilton, Ontario, between May 1990 and June 1991, the concentrations of PAHs in respirable air particulate samples were found to range from 0.6 ng/m3 for phenanthrene into 4.3 ng/m3 for benzo[g,h,i]perylene, and 5.1 ng/m3 for combined benzo[b,j,k]fluoranthenes (Legzdins et al. 1994). In a recent limited study, mean concentrations of particle-phase PAHs in New York City air were reported to range from 0.11 ng/m3 for anthracene to 4.05 ng/m3 for benzo[g,h,i]perylene (Tan and Ku 1994) [881].

Atmospheric PAH concentrations have been found to be significantly elevated in areas of enclosed traffic tunnels. In a 1985-86 study in the Baltimore Harbor Tunnel the average concentrations of particle-phase PAHs ranged from 2.9 ng/m3 for anthracene to 27 ng/m3 for pyrene (Benner and Gordon, 1989). These values are up to an order of magnitude lower than those obtained in 1975 by Fox and Staley (1976), which ranged from 66 ng/m3 for benzo[a]pyrene to 120 ng/m3 for pyrene. Benner and Gordon (1989) postulated that the observed decrease in PAH concentrations over the 1975-85 decade resulted from the increasing use of catalytic converters in U.S. automobiles over that period. These authors also reported concentrations of PAHs in a typical vapor-phase sample

from the Boston Harbor Tunnel for four PAHs included in this profile: anthracene (32.3 ng/m3), fluoranthene (25.6 ng/m3), phenanthrene (184 ng/m3), and pyrene (28.3 ng/m3. They emphasized that the vapor-phase samples included PAHs inherently present in the vapor phase as well as the more volatile 3- and 4-ring PAHs that may be desorbed from particles during sampling. These results underscore the need to evaluate both particle- and vapor-phase samples to obtain more reliable estimates of total atmospheric PAH concentrations [881].

Forms/Preparations/Formulations:

See individual PAHs entries.

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

Low-molecular-weight PAHs are more soluble and volatile and have less affinity for surfaces than do high-molecular-weight PAHs (see Associated Chemicals or Topics section above for list of low- and high-molecular-weight PAHs). In general, vapor pressures decrease, as do water solubilities, with increasing molecular weight. Also, alkyl substitution usually decreases water solubility [754].

The low molecular weight PAHs have Henry's law constants in the range of 10(-3) to 10(-5) atm-m(3)/mol; medium molecular weight PAHs have constants in the 10(-6) range; and high molecular weight PAHs have values in the range of 10(-5) to 10(-8). Compounds with values ranging from 10(-3) to 10(-5) are associated with significant volatilization, while compounds with values less than 10(-5) volatilize from water only to a limited extent [788].

The low molecular weight PAHs have Koc values in the range of 10(3) to 10(4), which indicates a moderate potential to be adsorbed to organic carbon in the soil and sediments. The medium molecular weight compounds have Koc values in the 10(4) range. High molecular weight PAHs have Koc values in the range of 10(5) to 10(6), which indicates stronger tendencies to adsorb to organic carbon [788].

Petroleum-derived PAH assemblages contain higher amounts of the smaller molecular wight PAHs such as naphthalene, acenaphthene, and fluorene, and also alkylated PAHs such as methylnaphthalenes. A higher ratio of three to four ring PAHs and a higher ratio of three to five ring PAHs exists in petroleum and petroleum-polluted sediments compared to recently formed or uncontaminated sediments [653].

The combustion-derived or pyrolytic PAH assemblages tend to contain more of the higher molecular weight and higher membered-ring PAH compounds such as phenanthrene, fluoranthene, pyrene, and benzo(a)pyrene, and also contain fewer of the smaller molecular wight PAHs such as naphthalene. Thus the PAH assemblage can be indicative of whether the PAH source is of petrogenic or pyrolytic/combustion origin [653].

PAHs generally have low solubilities (eg. < or equal to 34.4 mg/L, the value for naphthalene) and high octanol-water partition coefficients (eg. > or equal to 2344, the value for naphthalene) and are more likely to be found associated with the sediments and suspended solids than in aqueous solution. The lower molecular weight PAHs (acenaphthene, naphthalene, fluorene) may be rapidly lost from the water column due to volatilization and microbial degradation, while the large molecular weight PAHs [B(a)A, B(a)P] are more susceptible to losses due to photo-oxidation and may be removed as a result of sedimentation. Thus PAHs have a short residence time in aqueous solution and, when present in the water column, they are usually a result of recent or chronic pollution [653].

Typical PAH concentrations in various fuels and petroleum products:

Concentrations of PAHs in Two Crude and Two Refined Fuel Oils [177]:

Note: The composition of chemicals making up petroleum hydrocarbon batches is quite variable, so in spill scenarios, it is often first necessary to determine the exact composition of the oil in the particular spill in question. The following concentrations in mg/kg (ppm) are from API reference oils:

Compound	South LA crude	Kuwait crude	No. 2 fuel oil	Bunker C residual
Naphthalene	400	400	4,000	1,000
1-Methylnaphthalene	800	500	8,200	2,800
2-Methylnaphthalene	900	700	18,900	4,700
Dimethylnaphthalenes	3,600	2,000	31,100	12,300
Trimethylnaphthalenes	2,400	1,900	18,400	8,800
Fluorenes	200	<100	3,600	2,400
Phenanthrene	70	26	429	482
1-Methylphenanthrene	111	-	173	43
2-Methylphenanthrene	144	89	7,677	828
Fluoranthene	5.0	2.9	37	240
Pyrene	3.5	4.5	41	23
Benz(a)anthracene	1.7	2.3	1.2	90
Chrysene	17.56	6.9	2.2	196
Triphenylene	10	2.8	1.4	31
Benzo(ghi)fluoranthene	1	<1		
Benzo(b)fluoranthene	<0.5	<1		
Benzo(j)fluoranthene	<0.9	<1		
Benzo(k)fluoranthene	<1.3	<1		
Benzo(a)pyrene	0.75	2.8	0.6	44
Benzo(e)pyrene	2.5	0.5	0.1	10
Perylene	34.8	<O.l	-	22
Benzo(ghi)perylene	1.6	<1		

Details of PAH content (ng/mg or ppm) in one fresh sample of NSFO (Fuel Oil 5, Chuck Rafkind, National Park Service, Personal Communication, 1996):

Naphthalene:	34.3
C1-Naphthalene:	4086.9
C2-Naphthalene:	4865.4
C3-Naphthalene:	4793.7
C4-Naphthalene:	2688.5
Biphenyl:	3.5
Acenaphthylene:	4.1
Acenaphthene:	111.2
Fluorene:	216.0
C1-Fluorene:	658.8
C2-Fluorene:	1277.0
C3-Fluorene:	1243.8
Anthracene:	96.4
Phenanthrene:	778.2
C1-Phenanthrene/anthracene:	2116.3 (includes both)
C2-Phenanthrene/anthracene:	2716.7 "
C3-Phenanthrene/anthracene:	1923.3 "
C4-Phenanthrene/anthracene:	820.5 "
Dibenzothiophene:	25.7
C1-Dibenzothiophene:	1396.1
C2-Dibenzothiophene:	2155.9
C3-Dibenzothiophene:	1975.5
Fluoranthene:	31.6
Pyrene:	177.9
C1-Fluoranthene/pyrene:	566.1
Benzo(a)anthracene:	41.1
Chrysene:	74.3
C1-Chrysene:	312.1
C2-Chrysene:	370.8
C3-Chrysene:	29.9
C4-Chrysene:	19.7
Benzo(b)fluoranthene	11.0
Benzo(k)fluoranthene:	0.6
Benzo(e)pyrene:	29.8
Benzo(a)pyrene:	19.3
Perylene	10.6
Indeno(1,2,3-c,d)pyrene:	2.3
Dibenz(a,h)anthracene:	4.0
Benzo(g,h,i)perylene:	11.4

Note: the above PAHs and alkyl PAHs were analyzed by a GC/MS/SIM (Selective Ion Mode) 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).

Details of PAH content (ng/L or ppt, compare to the above listed ppm concentrations by dividing the below-listed ppt concentrations below by 1000000) 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):

Naphthalene:	530.8
C1-Naphthalene:	2463.7
C2-Naphthalene:	12044.7
C3-Naphthalene:	45345.1
C4-Naphthalene:	48336.8
Biphenyl:	129.7
Acenaphthylene:	81.2
Acenaphthene:	1517.6
Fluorene:	1229.3
C1-Fluorene:	11424.5
C2-Fluorene:	28680.7
C3-Fluorene:	32509.9
Anthracene:	1972.5
Phenanthrene:	7136.3
C1-Phenanthrene/anthracene:	31377.0 (includes both)
C2-Phenanthrene/anthracene:	49447.3 "
C3-Phenanthrene/anthracene:	41754.1 "
C4-Phenanthrene/anthracene:	22250.2 "
Dibenzothiophene:	8377.8
C1-Dibenzothiophene:	24742.0
C2-Dibenzothiophene:	44033.0
C3-Dibenzothiophene:	43900.3
Fluoranthene:	818.8
Pyrene:	5900.6
C1-Fluoranthenes/pyrenes:	16248.3 (includes both)
Benzo(a)anthracene:	1053.5
Chrysene:	1817.1
C1-Chrysene:	7398.8
C2-Chrysene:	9910.6
C3-Chrysene:	1048.5
C4-Chrysene:	625.9
Benzo(b)fluoranthene	399.2
Benzo(k)fluoranthene:	39.7
Benzo(e)pyrene:	1062.3
Benzo(a)pyrene:	602.7
Perylene	428.6
Indeno(1,2,3-c,d)pyrene:	106.8
Dibenz(a,h)anthracene:	117.1
Benzo(g,h,i)perylene:	421.4

Note: the above PAHs and alkyl PAHs 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).

Details of PAH content (mg/kg or ppm) in one fresh sample of Exxon Valdez Crude Oil [971]:

Note: these values are wet weight (Jerry Neff, Battelle Ocean Sciences, Duxbury, MA, personal communication 1996):

Naphthalene:	622 mg/kg = ppm
C1-Naphthalene:	1400 mg/kg = ppm
C2-Naphthalene:	1780 mg/kg = ppm
C3-Naphthalene:	1410 mg/kg = ppm
C4-Naphthalene:	696 mg/kg = ppm
Acenaphthylene:	0 mg/kg = ppm
Acenaphthene:	2 mg/kg = ppm
Fluorene:	93 mg/kg = ppm
C1-Fluorene:	224 mg/kg = ppm
C2-Fluorene:	366 mg/kg = ppm
C3-Fluorene:	394 mg/kg = ppm
Anthracene:	0 mg/kg = ppm
Phenanthrene:	262 mg/kg = ppm
C1-Phenanthrene:	572 mg/kg = ppm
C2-Phenanthrene:	722 mg/kg = ppm
C3-Phenanthrene:	576 mg/kg = ppm
C4-Phenanthrene:	446 mg/kg = ppm
Dibenzothiophene:	217 mg/kg = ppm
C1-Dibenzothiophene:	449 mg/kg = ppm
C2-Dibenzothiophene:	635 mg/kg = ppm
C3-Dibenzothiophene:	579 mg/kg = ppm
Fluoranthene:	2 mg/kg = ppm
Pyrene:	10 mg/kg = ppm
C1-Fluoranthene/pyrene:	82 mg/kg = ppm
Benzo(a)anthracene:	2 mg/kg = ppm
Chrysene:	46 mg/kg = ppm
C1-Chrysene:	89 mg/kg = ppm
C2-Chrysene:	138 mg/kg = ppm
C3-Chrysene:	115 mg/kg = ppm
C4-Chrysene:	0 mg/kg = ppm
Benzo(b)fluoranthene	6 mg/kg = ppm
Benzo(k)fluoranthene:	0 mg/kg = ppm
Benzo(a)pyrene:	0 mg/kg = ppm
Indeno(1,2,3-c,d)pyrene:	1 mg/kg = ppm
Dibenz(a,h)anthracene:	1 mg/kg = ppm
Benzo(g,h,i)perylene:	2 mg/kg = ppm
Total PAHs	11,317 mg/kg = ppm

Details of PAH content (mg/kg or ppm) in salmon carcass (fatty viscera removed, so the concentrations may have been higher from whole body samples) 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 fish tissues [971]. These values are wet weight (Jerry

Neff, Battelle Ocean Sciences, Duxbury, MA, personal communication 1996):

Naphthalene:	7.15 ug/kg = ppb
C1-Naphthalene:	65.11 ug/kg = ppb
C2-Naphthalene:	29.75 ug/kg = ppb
C3-Naphthalene:	93.95 ug/kg = ppb
C4-Naphthalene:	36.63 ug/kg = ppb
Acenaphthylene:	0 ug/kg = ppb
Acenaphthene:	0 ug/kg = ppb
Fluorene:	6.86 ug/kg = ppb
C1-Fluorene:	12.63 ug/kg = ppb
C2-Fluorene:	22.87 ug/kg = ppb
C3-Fluorene:	13.64 ug/kg = ppb
Anthracene:	0 ug/kg = ppb
Phenanthrene:	22.97 ug/kg = ppb
C1-Phenanthrene:	28.48 ug/kg = ppb
C2-Phenanthrene:	20.45 ug/kg = ppb
C3-Phenanthrene:	12.43 ug/kg = ppb
C4-Phenanthrene:	1.71 ug/kg = ppb
Dibenzothiophene:	19.65 ug/kg = ppb
C1-Dibenzothiophene:	19.68 ug/kg = ppb
C2-Dibenzothiophene:	15.96 ug/kg = ppb
C3-Dibenzothiophene:	7.44 ug/kg = ppb
Fluoranthene:	0 ug/kg = ppb
Pyrene:	0 ug/kg = ppb
C1-Fluoranthene/pyrene:	0.62 ug/kg = ppb
Benzo(a)anthracene:	0.72 ug/kg = ppb
Chrysene:	2.5 ug/kg = ppb
C1-Chrysene:	0.71 ug/kg = ppb
C2-Chrysene:	0.48 ug/kg = ppb
C3-Chrysene:	0.16 ug/kg = ppb
C4-Chrysene:	0.56 ug/kg = ppb
Benzo(b)fluoranthene	0 ug/kg = ppb
Benzo(k)fluoranthene:	0 ug/kg = ppb
Benzo(a)pyrene:	0 ug/kg = ppb
Indeno(1,2,3-c,d)pyrene:	0 ug/kg = ppb
Dibenz(a,h)anthracene:	0 ug/kg = ppb
Benzo(g,h,i)perylene:	0 ug/kg = ppb
Total PAHs	436 ug/kg = ppb

Details of PAH 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):

Naphthalene:	12.9 ug/kg = ppb
C1-Naphthalene:	17.3 ug/kg = ppb
C2-Naphthalene:	247 ug/kg = ppb
C3-Naphthalene:	905 ug/kg = ppb
C4-Naphthalene:	850 ug/kg = ppb
Acenaphthylene:	0 ug/kg = ppb
Acenaphthene:	0 ug/kg = ppb
Fluorene:	38.3 ug/kg = ppb
C1-Fluorene:	383 ug/kg = ppb
C2-Fluorene:	1317 ug/kg = ppb
C3-Fluorene:	1535 ug/kg = ppb
Anthracene:	0 ug/kg = ppb
Phenanthrene:	356 ug/kg = ppb
C1-Phenanthrene:	1924 ug/kg = ppb
C2-Phenanthrene:	3834 ug/kg = ppb
C3-Phenanthrene:	2438 ug/kg = ppb
C4-Phenanthrene:	796 ug/kg = ppb
Dibenzothiophene:	260 ug/kg = ppb
C1-Dibenzothiophene:	1344 ug/kg = ppb
C2-Dibenzothiophene:	2743 ug/kg = ppb
C3-Dibenzothiophene:	2743 ug/kg = ppb
Fluoranthene:	10.7 ug/kg = ppb
Pyrene:	32.9 ug/kg = ppb
C1-Fluoranthene/pyrene:	302 ug/kg = ppb
Benzo(a)anthracene:	0 ug/kg = ppb
Chrysene:	411 ug/kg = ppb
C1-Chrysene:	658 ug/kg = ppb
C2-Chrysene:	521 ug/kg = ppb
C3-Chrysene:	239 ug/kg = ppb
C4-Chrysene:	43.9 ug/kg = ppb
Benzo(b)fluoranthene	27.4 ug/kg = ppb
Benzo(k)fluoranthene:	0 ug/kg = ppb
Benzo(a)pyrene:	65.8 ug/kg = ppb
Indeno(1,2,3-c,d)pyrene:	0 ug/kg = ppb
Dibenz(a,h)anthracene:	2.63 ug/kg = ppb
Benzo(g,h,i)perylene:	7.41 ug/kg = ppb
Total PAHs	24,051 ug/kg = ppb

Table: Concentrations of individual polynuclear aromatic hydrocarbons (PAHs) in two crude oils 10(-6)g/g oil in ppm [747]:

COMPOUND	So. Louisiana crude	Kuwait crude
pyrene	4.3	4.5
fluoranthene	6.2	2.9
benz(a)anthracene	3.1	2.3
chrysene	23	6.9
triphenylene	13	2.8
benzo(a)pyrene	1.2	2.8
benzo(e)pyrene	3.3	0.5

If the above-summarized 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 no lower than 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, 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, as detailed above, 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.

Additional example from a separate study:

Details of contaminants (ng/g or ppb dry weight) in samples of sediments contaminated by Diesel Fuel (1D and 2D, historically possibly mixed with a few other petroleum products), two sites in Branch Creek, Petersburg National Battlefield (Ray Ahlbrandt, National Park Service, Personal Communication, 1996):

Site O = outfall area S1B, Sediments here smelled like Diesel.

Site DS = One mile downstream of S1B, still in Branch Creek, 50 feet upstream of confluence with Harrison Creek

Parameter	Site O	Site DS
Naphthalene:	15.1	10.3
C1-Naphthalene:	51.0	13.8
C2-Naphthalene:	910.5	25.1
C3-Naphthalene:	3698.1	52.1
C4-Naphthalene:	4915.9	26.1
Total Naphthalenes	9590.6	127.40

Note: there is some evidence of carcinogenicity for naphthalenes: see entries for naphthalenes and PAHs.

Parameter	Site O	Site DS
Biphenyl:	11.8	1.8
Acenaphthylene:	4.3	4.4
Acenaphthene:	21.7	29.7
Fluorene:	43.0	48.4

C1-Fluorene:	483.3	27.2
C2-Fluorene:	1371.2	22.5
C3-Fluorene:	1880.8	39.7
Total Fluorenes	3,778.3	137.8
Anthracene:	17.4	88.4
Phenanthrene:	107.3	423.9
C1-Phenanthrene/anthracene:	1190.5	181.1
C2-Phenanthrene/anthracene:	1936.8	79.9
C3-Phenanthrene/anthracene:	1019.2	44.3
C4-Phenanthrene/anthracene:	318.3	17.7
Total P/A	4,455.8	323.0
Dibenzothiophene:	30.2	30.2
C1-Dibenzothiophene:	296.0	26.0
C2-Dibenzothiophene:	769.1	19.8
C3-Dibenzothiophene:	626.6	10.7
Total DBZT	1,721.9	86.7
Fluoranthene:	42.5	500.5
Pyrene:	78.6	421.0
C1-Fluoranthene/pyrene:	153.6	241.3
Heavier and often more carcinogenic PAHs:		
Benzo(a)anthracene *:	12.3	183.7
Chrysene *:	21.5	173.9
C1-Chrysene *:	26.3	82.3
C2-Chrysene *:	39.1	31.9
C3-Chrysene *:	3.3	2.8
C4-Chrysene *:	5.5	22.1
Total Chrysenes *:	95.7	313.0
Benzo(b)fluoranthene *:	23.5	194.7
Benzo(k)fluoranthene *:	5.3	79.3
Benzo(e)pyrene **:	9.9	84.7
Benzo(a)pyrene *:	8.3	84.7
Perylene **:	2.5	29.0
Indeno(1,2,3-c,d)pyrene *:	8.2	87.5
Dibenz(a,h)anthracene *:	2.4	25.9
Benzo(g,h,i)perylene **:	10.7	70.4

* Clearly Carcinogenic

** Not Able to Classify one way or the other (not sure about carcinogenicity, not classifiable, often due to lack of data. This does not mean clearly never

carcinogenic; sometimes it means there is not enough information to tell.

Note: The above PAHs and alkyl PAHs were analyzed by a GC/MS/SIM NOAA protocol [828].

If the above-summarized Park Service sediment investigation at Petersburg National Historical Battlefield (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.

Additional brief analysis of the above data from Petersburg National Battlefield (Roy Irwin, National Park Service, Personal Communication, 1996):

Overview: This was a one-time sampling, so not too much can be read into the results. However, the initial data seems generally consistent with a mixed petroleum product input with strong diesel component (as suspected) and it is of note that all 39 PAHs and alkyl PAHs were found in samples taken one mile downstream as well as even higher concentrations of many of the lighter (and less persistent) PAHs at the outfall. Magnesium, a known contaminant in diesel, is higher near the outfall and non-detected downstream. Other petroleum product suspect metals such as vanadium, nickel, and lead (from historic spills of leaded gas?) show the same trend. On the other hand, most of the heavier and more persistent PAHs had higher concentrations downstream. These heavier PAHs may be accumulating there after gradual transport downstream with sediment loads, while the lighter (and more prone to biodegradation) PAHs such as naphthalenes may be breaking down before they get so far downstream.

PAHs: Total naphthalenes of 9591 ppb at the outfall are far above concern effects benchmarks from Oakridge (407 ppb at 1% organic carbon). Only rough rather than precise comparisons can be made with Oakridge benchmarks since TOC was not analyzed along with the other

contaminants listed in the above table. However, NOAA suggested that the potential for biological effects for naphthalene of this contaminant sorbed to sediments was highest in sediments where its concentration exceeded the 2100 ppb dry weight Effects Range-Median (ERM) concentration and was lowest in sediments where its concentration was less than the 160 ppb dry weight Effects Range-Low (ERL) concentration [664]. The ERL and ERM levels were developed for marine and estuarine sediments, so they are only a rough benchmark for freshwater sediments, though commonly used for that purpose due to the lack of many other benchmarks. Nevertheless, using both NOAA and Oakridge values, impacts would be suspected from the naphthalene levels in sediments near the outfall.

Naphthalene is selectively phytotoxic, with alkyl compounds being most toxic [366], with effects on benthic aquatic invertebrates and fish. Phototoxicity of PAHs has been seen in a wide variety of aquatic organisms, including aquatic plants [911]. Anthracene concentrations of 17.4 ppb at the outfall and 88.4 one mile downstream are above the concern effects benchmark from Oakridge (0.3 ppb). Extractable organic matter was low (0.025) at the downstream site, so the 88.4 ppb level found there may be a concern. If the C1 to C4 Anthracenes were added, this exceedance would be more dramatic. Total chrysenes downstream are approaching but not exceeding the lowest published concern level: 386 ppb ERL NOAA level [664]. Benzo(a)anthracene levels in sediments downstream (183 ppb) somewhat exceeded the Oakridge Estimated equivalent sediment quality criterion of 108 ppb [652]. Benzo(a)pyrene levels in sediments downstream (157.6) exceed the OakRidge Estimated equivalent sediment quality criterion of 140 ppb [652]. Total fluorene sediment concentration of 3.778 ppm found upstream exceed 0.54 mg/kg dry weight (microtox) to 3.6 mg/kg dry weight (amphipod) AET benchmarks [416]. Fluoranthene levels downstream (500.5 ppb) were higher downstream, where this biodegradation resistant PAH may be accumulating; although 500 ppb is not extremely elevated, effects in sediments have been observed with mean fluoranthene concentrations as low as 382 ppb [444].

Total carcinogenic PAHs: in sediments downstream at site S6a, total carcinogenic PAHs just barely exceeded the 1 ppm level which had been shown to induce tumors in bullhead catfish [40]. Total carcinogenic PAHs were those in above tables from Benzo(a)anthracene, except for (excluding) the non-classifiable compounds Benzo(e)Pyrene, Perylene, and Benzo(g,h,i)perylene. Actually, none of the PAHs commonly analyzed by EPA method 8270 for semi-volatiles or even the NOAA expanded scan are clearly classified as non-carcinogens; the least

damaging thing one can say about any of them is that they are not classifiable due to lack of data.

In a different investigation, PAH concentrations (ug/g oil sampled) 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, respectively [790]:

Note: the below table 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]:

Naphthalene: 562, 14, 4
C1-Naphthalene: 1307, 150, 52
C2-Naphthalene: 1739, 740, 283
C3-Naphthalene: 1377, 970, 473
C4-Naphthalene: 767, 760, 423
Acenaphthylene: ND, ND, ND
Acenaphthene: ND, ND, ND
Fluorene: 80, 44, 27
C1-Fluorene: 208, 180, 98
C2-Fluorene: 306, 400, 198
C3-Fluorene: 310, 370, 245
Anthracene: ND, ND, ND
Phenanthrene: 222, 200, 124
C1-Phenanthrene/anthracene: 488, 660, 410
C2-Phenanthrene/anthracene: 629, 870, 564
C3-Phenanthrene/anthracene: 456, 640, 507
C4-Phenanthrene/anthracene: 256, 370, 263
Dibenzothiophene: 189, 150, 73
C1-Dibenzothiophene: 389, 460, 258
C2-Dibenzothiophene: 567, 860, 529
C3-Dibenzothiophene: 508, 880, 593
Fluoranthene: ND, ND, ND
Pyrene: 9, 7, 7
C1-Fluoranthene/pyrene: 63, 68, 70
Benzo(a)fluoranthene: ND, ND, 1
Chrysene: 41, ND, 54
C1-Chrysene: 73, 120, 120
C2-Chrysene: 93, 150, 144
C3-Chrysene: 79, 120, 101
C4-Chrysene: 64, 69, 58
Benzo(a)fluoranthene [sic]: 6, ND, 1
Benzo(k)fluoranthene: ND, ND, 2
Benzopyrene: 12, ND, 1
Indeno(1,2,3-c,d)pyrene: ND, ND, ND
Dibenz(a,h)anthracene: ND, ND, ND
Benzo(g,h,i)perylene: ND, ND, 1

ND = not detected.

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. Estimates of total carcinogenic PNAs in crude oil ranges from 12 to < 100 ppm [734].

PAHs are present at 1-2 weight % in crude oils [788].

In one study, the sum of 26 individual PAHs represented 0.17% of a waste crankcase oil, or 1.2% of the aromatic fraction [519].

Note: the table below was 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]:

Table: Concentrations of Polycyclic Aromatic Compounds in Used Engine Oil [519]:

COMPOUND	CONCENTRATION (ppm)
Low-molecular-weight PAHs	
Naphthalene	52.0
Acenaphthylene	1.5
Acenaphthene	3.7
Fluorene	67.0
Phenanthrene	200.0
Anthracene	22.0
High-molecular-weight PAHs	
Fluoranthene	55.0
Pyrene	120.0
Benz(a)anthracene	38.0
Chrysene	45.0
Benzofluoranthenes	46.0
Benzo(e)pyrene	32.0
Benzo(a)pyrene	15.0
Perylene	1.1
Indeno(1,2,3-cd)pyrene	14.0
Dibenz(ah)anthracene	1.5
Benzo(ghi)perylene	72.0
High-molecular-weight PACs	
Dibenzothiophene	1.9
Alkylated PAHs	
Naphthalenes	
C-1 naphthalene	31.0
C-2 naphthalene	60.0

C-3 naphthalene	80.0
C-4 naphthalene	52.0
Phenanthrenes	
C-1 phenanthrene	300.0
C-2 phenanthrene	300.0
C-3 phenanthrene	140.0
C-4 phenanthrene	35.0

Gasolines contain a small but significant amount of PAHs including naphthalene and alkyl naphthalenes [797]. Naphthalenes make up from 0.09 to 0.49 weight percent of gasoline and from 0.08 to 0.5 volume percent of various gasolines [796].

The following PAHs are found in unleaded, premium unleaded, and leaded gasolines (ranges in % volume of gasoline given in parentheses) [796]:

Anthracene (1.55 to 1.84 % volume of gasoline)
Benzo(b)fluoranthene (3.9 % volume of gasoline)
Fluoranthene (1.84 % volume of gasoline)

Benzo(a)pyrene, a particularly carcinogenic and persistent heavy PAH, is found in gasoline in concentrations of 0.19 to 2.8 mg/kg (ppm), while benzo(e)pyrene, another heavy and persistent PAH is found in unleaded, premium unleaded, and leaded gasolines at a (presumably typical) concentration of 0.3 mg/kg (ppm) [796].

Some PAHs and PACs (polyaromatic compounds) are often from petroleum sources while others are typically the result of incomplete combustion. Many can originate from many different sources. The most important petroleum-source PAHs/PACs include (all are on the expanded scan) [468]:

Chrysene (CAS Number 218-01-9): Present in some heavy crudes in significant amounts; also important are the homologous series compounds (alkylated compounds C1 through C4).

Fluorene (CAS Number 86-73-7): Present in significant amounts, especially the homologous series (alkylated compounds C1 through C3).

Naphthalene (CAS Number 91-20-3): Often present in significant amounts; also important are the homologous series compounds (alkylated compounds C1 through C4).

Phenanthrene (CAS Number 85-01-8): Often present in significant amounts; also important are the homologous series compounds (alkylated compounds C1 through C4).

Anthracene (CAS Number 120-12-7): Present in petroleum but usually not very high quantities.

Dibenzothiophene: Can be present in significant amounts, especially the homologous series (alkylated compounds C1 through C3). Note: Dibenzothiophene and related compounds are not universally considered to be PAHS.

Over 190 PAH compounds were found in sediment samples from Galveston Bay (Brian Cain, FWS, personal communication, 1994).

For Kow figures for PAHs, see Bio.Detail section above.

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

The movement of PAHs in the environment depends on properties like their water solubility, vapor pressure, and molecular weight [788]. Concentrations of PAHs in aquatic ecosystems are generally highest in sediments, intermediate in aquatic biota and lowest in the water column [754]. Because of their low water-solubility and hydrophobic nature, PAHs tend to be associated primarily with inorganic and organic material in suspended and bed sediments [754].

PAH glucuronides include [366,609]:

> Naphthalene in sediments was metabolized to 1,2-dihydro-1,2-dihydroxynaphthalene glucuronide. [Varanasi et al; Aquat Toxicol 1 (1): 49 (1981)].
>
> Phenanthrene metabolites 48 hr after intragastric admin of 75 mg/kg to coalfish were all 5 of possible monohydroxy deriv & trans-phenanthrene-1,2-dihydrodiol & trans-phenanthrene-9,10-dihydrodiol. Trans-phenanthrene-1,2-dihydrodiol was excreted as glucuronide &/or sulfate conjugate. [Solbakken JE et al; Acta Pharmacol Toxicol 46 (2): 127-32 (1980)].
>
> The 1,2-, 3,4-, and 9,10-dihydrodiols are excreted either free or conjugated with glucuronic acid in the urine of rats and rabbits following IP admin of phenanthrene. These metabolites have also been detected in vitro following incubation of phenanthrene with liver preparations from guinea pigs, rats and mice. Further oxidative metabolism of the 1,2-dihydrodiol by rat-liver preparations to the 1,2-diol-3,4-epoxide has also been reported. [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 423 (1983)]

Water Fate:

> It had previously been thought that when the PAH molecular weight reaches that of three-ring compounds, an aqueous concentration equal to the solubility in water was required to elicit aquatic toxicity, and that heavier compounds would not exhibit acute toxicity even at maximum solubility [779]. However, recent studies have shown that some of the heavier PAHs can exhibit acute toxicity at levels below solubilities due to photoinduced

enhanced toxicity in the presence of UV or other types of solar radiation [779,887]. For additional details on photoinduced toxicity, see Interactions section.

PAHs (higher concentrations at least) have a short residence time in aqueous solution and, when present in the water column, they are usually a result of recent or chronic pollution [653].

The most important processes contributing to the degradation of PAHs in water are chemical oxidation, photo-oxidation, and biodegradation by aquatic microorganisms [207,788]. The rate and extent of photodegradation vary widely among the PAHs. Factors such as water depth, turbidity, and temperature affect the rate of photodegradation [788]. Photolysis of many PAHs is expected to occur near the water surface, with the higher molecular weight PAHs such as BaP being more sensitive to photolysis [207].

The photo-oxidation of dissolved PAHs is relatively rapid under both laboratory and natural sunlight illumination; half-lives range from 30 min to 23 d. In natural waters, photolysis of dissolved PAHs proceeds at a slower rate; half-lives are 20-60% longer. Little or no photolysis occurs in the presence of particulate matter, because of either direct absorption or light scattering in the water column [754].

The low molecular weight PAHs have Henry's law constants in the range of 10(-3) to 10(-5) atm-m(3)/mol; medium molecular weight PAHs have constants in the 10(-6) range; and high molecular weight PAHs have values in the range of 10(-5) to 10(-8). Compounds with values ranging from 10(-3) to 10(-5) are associated with significant volatilization, while compounds with values less than 10(-5) volatilize from water only to a limited extent. Half-lives for volatilization of benz(a)anthracene and benzo(a)pyrene (high molecular weight PAHs) from water have been estimated to be greater than 100 hrs. One researcher also stated that lower molecular weight PAHs could be substantially removed by volatilization if suitable conditions (high temperature, low depth, high wind) were present. Half-lives for volatilization of anthracene (a low molecular weight PAH) were estimated to be 18 hrs in a stream with moderate current and wind, versus about 300 hrs in a body of water with a depth of 1 meter and no current [788].

In general, volatilization half-lives from 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].

Fate vs. Fish:

There is ample evidence that fish exposed to petroleum in sediments, water, or through the diet, accumulate hydrocarbons in tissues and body fluids. Some of the aromatic hydrocarbons are converted metabolically to metabolites that remain in tissues for prolonged periods. PAH ingestion by fish is more apt to reach critical organs if it comes across gill membranes than if it is ingested into the gut (Denny Buckler, FWS Columbia, personal communication).

Fish can be exposed to contaminants through their diet or skin, but the major route of exposure to water-soluble compounds is believed to be through the gills. Ingestion of contaminants adsorbed to particulates can be substantial (especially in marine species drinking water), but it is of interest to estimate exposure via the gills. According to Kiceniuk and Jones, the ventilation rate of trout measuring between 10 and 70 g can be estimated at 2 to 15 ml/min. The oil used in our investigation had a density of 0.828 g/ml and contained 14% aromatics and 65.4% aliphatics (by weight). Therefore we can transform the volume of used crankcase oil that was orally injected into the fish to a time of exposure at concentrations reported in environmental cases [519].

> Note: the text above 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].

Fate vs. Plants:

Investigators (as of 1983) concluded that on the basis of available data, three routes of PAH passage into plants have been considered: air deposition, adsorption from soil and water, and biochemical synthesis. There appears to be general agreement that available data support the first two routes; however, there is considerable controversy over the synthesis route [794].

Although some research seems to indicate that interior portions of above-ground vegetables do not accumulate high concentrations of PAHs, plants do translocate PAHs from roots to other plant parts, such as developing shoots [40]. This factor may have significance for herbivorous species of fish and wildlife. Some plants

can evidently catabolize benzo(a)pyrene, a PAH which some authors have referred to as the ultimate carcinogen, but metabolic pathways have not been clearly defined. When PAHs do degrade in plants through metabolism, they often break down into even more toxic, carcinogenic, and mutagenic compounds [40]. The PAH biomagnification potential of vegetation in terrestrial and aquatic food chains needs to be measured for a variety of PAHs in both field and laboratory experiments before we will have a complete understanding of these transformations [40].

Fate vs. Mammals:

In mammals, the mixed function oxygenase (MFO) enzyme system is responsible for initiating the breakdown of lipophilic organic compounds like PAHs [207]. Although some xenobiotics are thus detoxified, others, such as certain PAHs, are transformed into metabolites which are highly toxic, mutagenic, or carcinogenic to the host [207]. Several researchers have suggested that metabolic activity of the MFO system is a prerequisite for PAH induced carcinogenesis and mutagenesis [207].

Fate vs. Invertebrates:

Clams evidently do break down PAHs, although with a different (enzyme) system and at apparently different rates than fish [469]. The C2 and C3 alkylated phenanthrene isomers (dimethylyphenanthrene and trimethylphenanthrene) show relative concentration decreases over time that may indicate isomer-specific metabolism in clams [469]. Bivalves taken from chronically polluted harbors and transferred to clean waters release PAHs very slowly since some of the PAHs are in storage in tissue lipids [469].

Fate of PAHs in Soil or Sediments:

Degradation of PAHs in aquatic sediments in the absence of oxygen and penetrating radiation is very slow [40].

The "Koc" indicates the chemical's potential to bind to organic carbon in soil and sediment. The low molecular weight PAHs have Koc values in the range of 10(3) to 10(4), which indicates a moderate potential to be adsorbed to organic carbon in the soil and sediments. The medium molecular weight compounds have Koc values in the 10(4) range. High molecular weight PAHs have Koc values in the range of 10(5) to 10(6), which indicates stronger tendencies to adsorb to organic carbon [788].

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].

Volatilization of acenaphthene, anthracene, fluorene, and phenanthrene (low molecular weight PAHs) from soil may be substantial. Of 14 PAHs studied in two soils, volatilization was found to account for about 20% of the loss of 1-methylnaphthalene and 30% of the loss of naphthalene; volatilization was not an important loss mechanism for the other compounds. Lower molecular weight compounds may also volatilize from sediments; this process is not significant for the higher molecular weight compounds [788].

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].

The half-life of B(b)F has been estimated to be 610 days in terrestrial environments. One source reported that 90% of an application of B(a)A remained after 11 months of incubation with bacteria. This would correspond to a half-life of approximately 2200 days for B(a)A based on first-order degradation. Another source estimated that the half-life of B(a)A in terrestrial environments is 430 days [734].

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

Another study showed how biodegradation of PAHs was related to molecular weight. The 2- and 3-ring PAHs (naphthalene, fluorene, and phenanthrene) degraded rapidly. The 4-ring PAHs (fluoranthene, pyrene, benzo(a)anthracene, and chrysene) generally were biodegraded 50% in a few months. The 5-ring PAHs (benzo(b)fluoranthene and benzo(a)pyrene) decreased slowly over a period of years [815].

Regarding naphthalene and alkyl naphthalenes, the parent compound naphthalene is the first to degrade; so as

petroleum products age, the percentage of alkyl naphthalenes vs. naphthalene increases, but most standard EPA standard scans (even 8270) do not pick up alkyl naphthalenes [796].

Sinking vs. Floating oil:

Heavy fuel oils or crude oils which are high in density should be subjected to a rigorous determination of submergence characteristics so that on-scene response groups can be warned about the possibility of the oil sinking. Sinking oil could lead to a significant environmental impact because the sensitive and often economically valuable benthic environment could become contaminated with oil and resultant PAHs. More research in this area should lead to suggested protocols for a density increase assessment of spilled oils [554].

In general, even though density of oil increases through weathering, the density will rarely increase to that of freshwater (specific gravity approximately 1.00, density 1000 kg/m3) or marine water (specific gravity approximately 1.024, density 1024 kg/m3). However, heavy fuels such as no. 6 may weather to densities heavier that water since the unweathered density already exceeds 900 kg/m3 (that is, a typical No. 6 fuel oil has an API gravity around 12.3, which corresponds to a density of 971 kg/m at 22 +or- 2 degrees Celsius) [554].

When does one have to be concerned about heavy oil fractions sinking through the water column and ending up in bottom sediments?

Many of the heavier PAHs have specific gravities much higher than 1.0. So why don't they sink out of oil slicks, and head for the bottom sediments, more often than they do? According to Jackie Michel, Research Planning Inc., S. Carolina, a member of a team that provides support to NOAA at spills, the reasons and considerations include the following (Personal Communication to Roy Irwin, 1993):

> Oil fractions seldom sink out of slicks right away. Little separation occurs in most marine spills. The oil mass usually stays together. Only a very small amount of the oil typically dissolves in water.
>
> Oil is viscous and it stays together. The heavier PAHs are dissolved in the lighter fractions (toluene, benzene, xylene) and are more attracted to these compounds than to water. These forces can overcome the forces of gravity, at least for a time.

It is true that as the light fractions are removed over time by evaporation, sinking can begin to occur as the oil gets heavier and begins to pick up particulates. As emulsification sets in, water surrounds oil droplets.

It should be kept in mind that sinking oil fractions are less likely in marine waters (specific gravity approx. 1.034) than in freshwaters (specific gravity of 1.00) and that the heavy compounds of most concern, PAHs, only make up 2-10% of many spilled petroleum products.

In addition to PAHs, there are other heavy compounds in typical oil slicks, such as heavy asphaltines (compounds with specific structures, not "asphalt", waxes, resins, tars, etc.).

Environmental Fate [609]:

In order to clarify synthetically the situation concerning air pollution by suspended particulate matter in the vicinity of the Meishin Expressway, polycyclic aromatic hydrocarbons, which are typical substances adsorbed on suspended particulate matter, were determined by the HPLC method, and the distribution of the polycyclic aromatic hydrocarbons concn was examined. The concn of benzo(a)pyrene at a point 60 m from the road edge was 2.93 ng/cu m (9.11 ug/g dust). The concn of benzo(ghi)perylene was 6.62 ng/cu m (20.6 ug/g dust) at this point, being the highest of all the polycyclic aromatic hydrocarbons that were analyzed in this study. The concn of polycyclic aromatic hydrocarbons that were contained in 1 g of dust were the highest in the suspended particulate matter fraction from 0.7 to 1.6 micron in diameter. The concn of polycyclic aromatic hydrocarbons that were contained in particles under 5.4 microns in diameter, and which are absorbed extremely easily into the lung, occupied more than 90% of the concn of polycyclic aromatic hydrocarbons that were contained in the total suspended particulate matter. When there was a high frequency of perpendicular wind, the concn of polycyclic aromatic hydrocarbons in suspended particulate matter fractions under 1.6 micron in diameter showed a marked tendency to have a clear concn-distance profile, but when there was a high frequency of parallel wind, it was observed that these concn at a point 120 m from the road edge were much higher than at the road edge. The correlation coefficient between the concn of benzo(a)pyrene and the traffic volume of large vehicles was elevated with the increase of distance from the road edge and was statistically significant at a point 290 m from the road edge. [Kita Y; Nippon Eiseigaku Zasshi 44 (2): 673-84 (1989)].

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 1991; IARC 1983) [881].

In addition to being conjugated, the 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 1978; Cohen et al 1976; Kiefer et al 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 1978; Cohen et al 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. 1976) [881]. Activation of benzo[a]pyrene has also been detected in human fetal esophageal cell culture (Chakradeo et al. 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 1988) [881].

Approximately 50% of the benzo[a]pyrene that was intratracheally instilled in hamsters was metabolized in the nose (Dahl et al 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 1989b) [881]. The prevalence of quinone production was not seen in hamsters as it was in rats (Dahl et al 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. 1988) [881]. In vitro metabolism of benzo[a]pyrene in the ethmoid turbinates of dogs resulted in a prevalence of phenols (Bond et al. 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. 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. 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. 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. 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. 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. 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. 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. 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. 1976, 1980) [881]. This metabolite is highly tumorigenic (Levin et al. 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. 1985) and human tissue preparations (Harris et al. 1979) [881].

Studies using rat liver microsomes have shown that hydroxy metabolites of benzo[a]pyrene undergo glucuronidation (Mackenzie et al. 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. 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. 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. 1993), and in rat liver microsomes (Kawamoto et al. 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. 1983b) [881]. Indeno[1,2,3-c,d]pyrene stimulated the bay region oxidation (3, 4-oxidation) of benz[a]anthracene (Jacob et al. 1985) [881]. Similar xenobiotic effects were observed with chrysene as a substrate (Jacob et al. 1987) [881]. While some enzyme activities are being enhanced, alternate enzymatic pathways may be suppressed (Jacob et al. 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. 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. 1987) [881].

Rat mammary epithelial cells (RMEC) have been shown to activate PAHs (Christou et al. 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. 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. 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. (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. (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. (1985, 1987) [881]. Furthermore, Fiume et al. (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. 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. 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. 1993b; Weyand et al. 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].

Detailed information about the biocatlysis/biodegradation fate of naphthalene, a reprensentative light PAH compound, is included on the University of Minnesota Biocatlysis/Biodegradation Database (Available on the interet in July, 1997, www.nmsr.labmesd.umn.edu).

Other detailed Fate information on PAHs 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. 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 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 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. 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. 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. 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. 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. 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. 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. 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 [881].

Misc. References on Fate: See also:

Author	RC Loehr, MT Webster
Title	Behavior of fresh vs aged chemicals in soil
Source	Journal of Soil Contamination 5: 4 (1996)
Page(s)	361-383

Author	N Alawadhi, R Aldaher, A Elnawawy, MT Balba
Title	Bioremediation of oil-contaminated soil in

Kuwait .1. Landfarming to remediate oil-contaminated soil Source Journal of Soil Contamination 5: 3 (1996) Page(s) 243-260

Author AL Juhasz, ML Britz, GA Stanley Title Degradation of high molecular weight polycyclic aromatic hydrocarbons by Pseudomonas cepacia. Source Biotechnology Letters 18: 5 (MAY 1996) Page(s) 577-582

Author IT Yeom, MM Ghosh, CD Cox, KG Robinson Title Micellar solubilization of polynuclear aromatic hydrocarbons in coal tar-contaminated soils (vol 29, pg 3015, 1995) Source Environmental Science & Technology 30: 6 (JUN 1996) Page(s) 2104

Author H Melcer, P Steel, WK Bedford Title Removal of polycyclic aromatic hydrocarbons and heterocyclic nitrogen compounds in a municipal treatment plant Source Water Environment Research 67: 6 (SEP-OCT 1995) Page(s) 926-934

Author MM Grundy, MN Moore, SM Howell, NA Ratcliffe Title Phagocytic reduction and effects on lysosomal membranes by polycyclic aromatic hydrocarbons, in haemocytes of Mytilus edulis Source Aquatic Toxicology 34: 4 (APR 1996) Page(s) 273-290

Author E Deziel, G Paquette, R Villemur, F Lepine, JG Bisaillon Title Biosurfactant production by a soil Pseudomonas strain growing on polycyclic aromatic hydrocarbons Source Applied and Environmental Microbiology 62: 6 (JUN 1996) Page(s) 1908-1912

Author E Kiehlmann, L Pinto, M Moore Title The biotransformation of chrysene to trans-1,2- dihydroxy-1,2-dihydrochrysene by filamentous fungi Source Canadian Journal of Microbiology 42: 6 (JUN 1996) Page(s) 604-608

Author M Bouchez, D Blanchet, JP Vandecasteele Title The microbiological fate of polycyclic aromatic hydrocarbons: Carbon and oxygen balances for bacterial degradation of model compounds Source Applied Microbiology and Biotechnology 45: 4 (MAY 1996) Page(s) 556-561

Laboratory and/or Field Analyses:

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Recommended detection limits:

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

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 above for more details).

In a similar vein, if the Park Service sediment investigation at Petersburg National Historical Battlefield (see Chem.Detail section above, 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).

Analyses of sediments:

The past use of EPA method 8270 [861] for analyses of PAHs in sediments was often deficient because the detection limits used were too high. For example, the detection limit on phenanthrene in

sediments analyzed from a Park Service site at Fort Darling was listed as 1600 ppb, whereas many now recommend using a detection limit no higher than 10 ppb (1 ppb is best). In this case, harmful levels of phenanthrene and other PAHs could have been present but the test would not have detected them, because the detection limit used was too high (Roy Irwin, National Park Service, personal commuication, 1997]. It is usually better to perform an expanded scan for PAHs and alkylated homologues [828], with detection limits no lower than 1 ppb dry weight in solids. 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, utilizing sonic extraction.

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]. (see HPLC sections below for more detail).

Some labs (such as Coastal Environments Lab in Encinitas, California) have recommend P450 Reporter Gene System (RGS) screening for sediments to determine which are most severely contaminated with PAHs before proceeding to GC/MS testing. However, the system is also activated by certain PCBs, dioxins, and other compounds (see biomarker section below for details).

Compounds in Expanded Scans:

An "expanded scan of PAHs" done by the Geochemical and Environmental Research Group Laboratory includes parent compounds and various alkyl homologs [828]: The expanded list includes most of the PAHs recommended by the NOAA's National Status and Trends program [680,828]:

Acenaphthene
Acenaphthylene
Anthracene
Benzo(a)anthracene
Benzo(a)fluoranthene
Benzo(k)fluoranthene
Benzo(g,h,i)perylene
Benzo(e)pyrene
Benzo(a)pyrene
Biphenyl
Chrysene
Chrysene, C1-
Chrysene, C2-
Chrysene, C3-
Chrysene, C4-
Dibenzo(a,h)anthracene
Dibenzothiophene

NOTE: Although opinions differ as to whether dibenzothiophene is a PAH, it is listed as such in several sources [795,468,495].

Dibenzothiophene, C1-
Dibenzothiophene, C2-
Dibenzothiophene, C3-
Fluoranthene
Fluoranthenes/Pyrenes, C1-
Fluorene
Fluorene, C1-
Fluorene, C2-
Fluorene, C3-
Ideno(1,2,3,c,d)pyrene
Naphthalene
Naphthalene, C1-
Naphthalene, C2-
Naphthalene, C3-
Naphthalene, C4-
Perylene
Phenanthrene
Phenanthrenes/Anthracenes, C1-
Phenanthrenes/Anthracenes, C2-
Phenanthrenes/Anthracenes, C3-
Phenanthrenes/Anthracenes, C4-

Additional Details on NOAA expanded scan: PAHs Lab Analyses, NOAA Protocol Expanded Scan for PAHs and Alkyl Homologues of PAHs Using Gas Chromatography-Mass Spectrometry with Selective Ion Mode Enhanced Detection Limits (GC/MS/SIM) [828]:

Recommended by: National Park Service Staff, Fish and Wildlife Service Contaminants Program, NOAA Status and Trends Program, EPA EMAP Program, Many Consultants working on Exxon Valdez Spill, Many Laboratories.

Pros: A more complete list of analytes including alkyl homologues, suitably low detection limits, good utility for both risk/hazard assessment and for obtaining basic clues about possible sources (provides a crude fingerprint).

Cons: A bit more expensive than EPA standard method 8270 and substantially more expensive than rough screening scans. Not all labs are set up to do it.

Detection limits:

Recommended water sample detection limits are 1-10 ng/L (ppt) while recommended tissue, soil, and sediment sample detection limits for the expanded scan for PAHs are 1 ug/kg (ppb) dry wt.

Typical Costs per Sample (based on a survey of several laboratories in 1995):

Lab 1 (BSEQ): $425 per sample including extraction.

Lab 2 (GERG): $250 to $400 per sample depending on details. For low numbers of samples with no previous extractions, $400 for water, $425 for sediment, and $450 for tissues. As low as $250 if extraction not included (less than standard 8270 scan).

Lab 3 (BNW): $425 including extraction, or as low as $225 each for 40 samples if extraction already done.

Lab 4 (CAS): $300 for one class of chemicals (PAHs), up to $600 for all 8270 method analytes and lower detection limits (Method 8270/SIM, detection limits 1 ppb water, 20 pb tissues, and 10 ppb sediments).

Lab 5 (ADL): $425 to $600 including extraction. If a lot of extra chemical classes or advance fingerprinting is specified: up to $1000 per sample.

Summary: An alternative which works for many purposes (hazard assessment, source determination, surveys of hazardous compounds in weathered as well as fresh oils.

Another current and rigorous "expanded scan of PAHs" [679] done by the Battelle Marine Sciences Lab in Sequim, Washington, and the Battelle Ocean Science, Environmental Systems and Technology Division in Duxbury, Massachusetts, is very similar to the Texas A.& M. scan listed above except that the Battelle GC/MS/SIM expanded scan (detection limits 1-5 ppb dry wt. in sediments and tissue, 1-10 ppt in water) deletes biphenyl while adding Benzo(e)fluoranthene (Lisa Lefkovitz, Battelle NW, personal communication, 1995):

Some labs and programs add the following specific isomers (some of these have been included in the NOAA National Status and Trends Program, as noted in the NOAA list in Associated Chemicals or Topics section above [680,697]):

Naphthalene, 2,6-Dimethyl
Naphthalene, 1-Methyl
Naphthalene, 2-Methyl
Naphthalene, 1,6,7-Trimethyl
Naphthalene, 2,3,5-Trimethyl
Phenanthrene, 1-Methyl

Examples of standard method protocols for PAHs published by various parts of EPA as well as some other agencies are outlined below:

Holding Times:

Water Samples:

Both NPDES effluent discharge permit applications [1010] and RCRA (SW-846) solid and hazardous waste applications [1013] call for the following maximum holding times: 7 days until extraction and 40 days after extraction.

Solids Samples:

EPA RCRA methods for semi-volatiles in solids in SW-846 call for holding times of 14 days until extraction and 40 days after extraction [1013].

The need to get rid of headspace to prevent loss of certain PAHs (such as naphthalenes) tends to discourage the freezing of soil and other samples. However, the Fish and Wildlife Service and some other groups nevertheless freeze some soil samples. If this can be accomplished without compromising the sample (for example, breaking a glass container), the freezing tends to stop biodegradation. Once frozen, holding times for samples of semi-volatiles such as PAHs in solids is on the order of decades (John Moore, Fish and Wildlife Service, Personal Communication, 1997).

Containers:

Both EPA and APHA (Standards Methods Book) recommend glass containers for the collection of organic compounds [141,1010,1013]. EPA also recommends teflon lined caps for solids samples of semi-volatiles [1010,1013].

Guidance from other federal agencies (USGS, FWS, NOAA) also recommends glass containers for organics, and discourages the use of plastic containers for a variety of reasons (Roy Irwin, National Park Service, Personal Communication, 1997, based on a glance through recent internal guidance of several agencies).

Some federal agency quality control procedures call for voiding or red-flagging the results of organic analyses if the lab receives the sample in plastic containers (Roy Irwin, National Park Service, Personal Communication, 1997). The APHA pointed out some the potential hazards of the use of certain plastic containers for storing organic samples [141]:

A) Potential contamination of the sample via leaching of compounds from the plastic, and/or

B) The plastic container walls can sometimes be attacked by certain organics and fail, and/or

C) The possibility that some of organic compound

will dissolve into the walls of the plastic container, reducing the concentration of the compound in the container [141].

For the relatively volatile PAHs such as naphthalenes, not even vials are not the best choice for avoiding false negatives in soil samples through volatilization losses, since the use of brass liners for collection resulted in 19 fold higher VOCs than when 40 mL vials were used [798] The third update of EPA's SW-846 RCRA guidance authorizes the storage of soil samples of volatiles in EnCore TM (or equivalent, no government endorsement implied) samplers as long the sample is analyzed within 48 hours after collection [1013]. Several states also authorize the use of EnCore TM or equivalent containers for temporary (48 hour) storage containers (Donalea Dinsmore, State of Wisconsin DNR, personal communication, 1997).

Certain plastic polymers present less of a problem related to potential losses of volatiles than others. Some plastic is found in the latest approved EnCore TM samplers. Some states also give the reader the option of using plastic in collecting devices. For example, related to methods for gasoline range petroleum hydrocarbons, Wisconsin states that organics can be collected using a 30 ml plastic syringe with the end sliced off, a brass tube, an EnCore TM sampler or other appropriate devices (Donalea Dinsmore, State of Wisconsin DNR, personal communication, 1997). A plastic syringe is also mentioned as an option in the third update of RCRA methods in SW-846 [1013]. The thinking appears to be that plastic is less of a threat in a collecting device, with momentary contact, than in a storage container where contact times are longer.

Typical "standard method" protocols recommend proper cleaning of glass containers before use. Some collectors simply use pre-cleaned jars from I-Chem, Eagle Pitcher, or other private suppliers (no government endorsement implied). EPA [1010], USGS, and most other federal agencies recommend cleaning procedures for the glass containers, usually involving detergent rinsing, baking, and sometimes HCL rinses (Roy Irwin, National Park Service, Personal Communication, 1997).

Field Protocols:

Standard field collection method protocols are published or internally distributed by the Fish and Wildlife Service, the USGS, DOE, NOAA, and EPA. These recommendations change over time, with the newest recommendations sometimes being quite different than the old, thereby producing different results. The USGS NAWQA protocols call of sieving of sediment samples composites,

a practice that might result in the loss of relatively volatile PAHs such as the naphthalenes.

The Fish and Wildlife Service methods are similar in many ways to NOAA field protocols [676]. Many recommended EPA field methods for organics are not very detailed, although the 3rd update of SW-846 for RCRA solid waste methods is becoming more detailed [1013].

The various EPA methods for organics are different from each other, with the selection of the appropriate method depending upon the specific application (RCRA vs. CERCLA vs. NPDES permits, vs. Drinking Water, etc.) [861,1010,1013]. The EPA-recommended field methods are scattered through various EPA and ASTM publications.

EPA methods typically include recommendations that grab samples rather than composites be utilized for organics, and require the proper cleaning of collection bottles and collecting gear for both volatile and semi-volatile organics [1010,1013]. In other publications, EPA recommends caution in the use of composite soil samples whether organic or inorganic, citing statistical complications and stating that the compositing of samples cannot, in general, be justified unless for a stated specific purpose and unless a justification is provided [1017].

For PAHs (lab method 610) and other semi-volatiles, EPA recommended in 1994: that "conventional sampling practices" be followed as specified by ASTM D-3370 (3370-95a is a recent number), "Standard Practices for Sampling Water from Closed Conduits" [1010,1012]. No field methods are specified when not sampling from pipes [1010,1012].

Regardless of what lab methods are used, the investigator should take special precautions to prevent the escape of relatively light PAHs during sample shipment, storage, extraction, and cleanup [798]. This is especially true for soil and sediment sampling. The results of analyses of the lighter semi-volatiles (such as naphthalenes) can be dramatically effected by small details such as how the samples are collected, stored, held, and analyzed in the lab, since volatile compounds can readily volatilize from samples in both field and lab procedures. If the investigator knows that the sample will contain significant quantities of the lighter semi-volatiles such as naphthalenes, field and lab precautions should be taken just as if the investigator were handling volatiles (see Benzene entry for details). For example, for the lighter semi-volatiles, it may be prudent to use EPA method 5021 in SW 846, a generic "headspace" method for the collection of volatiles in soils and sediments

[1013].

Standard field methods for sampling contaminated soils for various types of contaminants were summarized by EPA in 1991 [1020]. These methods seem generally consistent with SCS recommendations, but are not necessarily 100% consistent with other protocols suggested by other parts of EPA [1013], and are not consistent with methods suggested by other agencies, such as the Fish and Wildlife Service.

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. As mentioned in the disclaimer at the top of this entry, contaminants data from different labs, different states, and different agencies, collected by different people, are often not very comparable.

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]. The basics of these quality assurance plans for chemical analyses should include the following quality control steps:

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. Typical lab quality control techniques should have included the following considerations (Roy Irwin, National Park Service, Personal Communication, 1997, summary based on various EPA and FWS documents):

Procedural Blanks should be analyzed to assure that no contaminants are added during the field and lab processing of

the samples. The standards for adequacy depend on the method and the media being measured.

Different federal agencies publish different acceptable limits. For one program, NOAA stated that at least 8% of samples should be blanks, reference or control materials [676].

The basic idea is that neither samples nor blanks should be contaminated. Because the only way to measure the performance of the modified procedures is through the collection and analysis of uncontaminated blank samples in accordance with this guidance and the referenced methods, it is highly recommended that any modifications be thoroughly evaluated and demonstrated to be effective before field samples are collected [1003].

Duplicate samples are analyzed to provide a measure of precision of the methods. The standards for adequacy depend on the method and the media being measured.

Different federal agencies publish different acceptable limits. There appears to be an inverse relationship between precision and sensitivity [676].

Some EPA methods state that a field duplicate must be collected at each sampling site, or one field duplicate per every ten samples, whichever is more frequent [1003]. Some protocols call for the preparation of one Ongoing precision and recovery (OPR) standard for every ten or fewer field samples. Great care should be taken in preparing ongoing precision and recovery standards [1003].

Spiked samples are analyzed to provide a measure of the accuracy of the analysis methods. The standards for adequacy depend on the method and the media being measured.

Different federal agencies publish different acceptable limits.

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. Many of the analyses which have been done for oil have used inappropriate methods

This is one reason for using the NOAA expanded scan for PAHs [828]; or method 8270 [1013] modified for 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 parent compound PAH scans (Roy Irwin, National Park Service, Personal Communication, 1997).

PAHs are often analyzed when crude oil or oil products are spilled. This is as it should be, since PAHs are among the more hazardous of the constituents in crude oil and many oil products (see Chem.Detail section of PAHs entry). However, it is not always easy to determine which combinations of lab methods to use for crude oil and oil products. The following is a proposed decision Tree (dichotomous key) for selection of lab methods for measuring contamination from mid-range petroleum products (Roy Irwin, National Park Service, Personal Communication, 1997):

> In choosing a lab method, it should be kept in mind that many mid range products (such as Diesel, No. 2 Fuel Oils, and Light Crudes) can be expected to exhibit the following characteristics [741]:
>
> -Moderately volatile; will leave residue (up to 1/3 of spilled amount)
> -Moderate concentrations of toxic (soluble) compounds
> -Will "oil" intertidal resources with long-term contamination potential
> -Has potential for subtidal impacts (dissolution, mixing, sorption onto suspended sediments)
> -No dispersion necessary
> -Cleanup can be very effective

Decision Tree (dichotomous key) for selection of lab methods for measuring contamination from light crude oils and middle distillate petroleum products (all diesels, jet fuels, kerosene, Fuel oil 2, Heating Oil 2):

1a. Your main concern is biological effects of petroleum products.....2

1b. Your main concern is cleanup or remediation but no ecological or human resources are at risk.....3

2a. The resource at risk is primarily humans via a drinking water pathway, either the contamination of groundwater used for drinking water, or the fresh* or continuing contamination of surface waters used as drinking water, or the risk is primarily to aquatic species in confined** surface waters from a fresh* spill, or the risk is to surface waters re-emerging from contaminated groundwater resources whether the spill is fresh* or not; the medium and/or pathway of concern is water rather than sediments, soil, or tissues 4

2b. The resource at risk is something else.....5

3a. The spilled substance is a fresh* oil product of known

composition: If required to do so by a regulatory authority, perform whichever Total Petroleum Hydrocarbon (TPH) analysis specified by the regulator. However, keep in mind that due to its numerous limitations, the use of the common EPA method 418.1 for Total Petroleum Hydrocarbons is not recommended as a stand-alone method unless the results can first be consistently correlated (over time, as the oil ages) with the better NOAA protocol expanded scan*** for polycyclic aromatic hydrocarbons (PAHs) and alkyl PAHs. If not required to perform an EPA method 418.1-based analysis for TPH, instead perform a Gas Chromatography/Flame Ionization Detection (GC/FID) analysis for TPH using the spilled substance as a calibration standard. GC/FID methods can be sufficient for screening purposes when the oil contamination is fresh*, unweathered oil and when one is fairly sure of the source [657]. If diesel 1D was spilled, perform TPH-D (1D) using California LUFT manual methods (typically a modified EPA method 8015) [465] or a locally available GC/FID method of equal utility for the product spilled. However, no matter which TPH method is used, whether based on various GC/FID or EPA method 418.1 protocols, the investigator should keep in mind that the effectiveness of the method typically changes as oil ages, that false positives or false negatives are possible, and that the better Gas Chromatography-Mass Spectrometry-Selected Ion Mode (GC/MS/SIM) scans (such as the NOAA expanded scan***) should probably be performed at the end of remediation to be sure that the contamination has truly been cleaned up.

3b. The spilled product is not fresh* or the contamination is of unknown or mixed composition.....6

4. Analyze for Benzene, Toluene, Ethyl Benzene, and Toluene (BTEX) compounds in water as part of a broader scan of volatiles using EPA GC/MS method 8260. The older standard EPA GC/MS method 8240 protocol was sufficient for some applications, but the standard EPA method 8240 (and especially the less rigorous EPA BTEX methods such as method 8020 for soil and method 602 for water) are all inadequate for generating scientifically defensible information for Natural Resource Damage Assessments [468]. The standard EPA methods are also inadequate for risk assessment purposes. Thus, when collecting information for possible use in a Natural Resource Damage Assessment or risk assessment, it is best to ask the lab to analyze for BTEX compounds and other volatile oil compounds using a modified EPA GC/MS method 8260 method using the lowest possible Selected Ion Mode detection limits and increasing the analyte list to include as many alkyl BTEX compounds as possible. Also analyze surface or (if applicable) ground water samples for polycyclic aromatic hydrocarbons (PAHs) and alkyl PAHs using the NOAA protocol expanded scan*** modified for water samples using methylene chloride extraction. If the contaminated water is groundwater, before the groundwater is determined to be

remediated, also analyze some contaminated sub-surface soils in contact with the groundwater for BTEX compounds (EPA GC/MS method 8260) [1013], and PAHs (NOAA protocol expanded scan***). The magnitude of any residual soil contamination will provide insight about the likelihood of recontamination of groundwater resources through equilibria partitioning mechanisms moving contamination from soil to water.

5a. The medium of concern is sediments or soils.....6

5b. The medium of concern is biological tissues.....7

6. Perform the NOAA protocol expanded scan*** for polycyclic aromatic hydrocarbons (PAHs) and alkyl PAHs. If there is any reason to suspect fresh* or continuing contamination of soils or sediments with lighter volatile compounds, also perform EPA GC/MS method 8260 [1013] using the lowest possible Selected Ion Mode (SIM) detection limits and increasing the analyte list to include as many alkyl Benzene, Toluene, Ethyl Benzene, and Xylene (BTEX) compounds as possible.

7a. The problem is direct coating (oiling) of wildlife or plants with spilled oil product.....8

7b. The problem is something else.....9

8. Perform NOAA protocol expanded scan*** for polycyclic aromatic hydrocarbons (PAHs) and alkyl PAHs and/or GC/FID fingerprinting of the coating oil only if necessary to identify the source or exact oil. If the source is known and no confirmation lab studies are necessary: dispense with additional chemical laboratory analyses and instead document direct effects of coating: lethality, blinding, decreased reproduction from eggshell coating, etc., and begin cleaning activities if deemed potentially productive after consolations with the Fish and Wildlife Agencies.

9a. The concern is for impacts on water column organisms such as fish or plankton).....10

9b. The concern is for something else (including benthic organisms).....11

10. If exposure to fish is suspected, an HPLC/Fluorescence scan for polycyclic aromatic hydrocarbon (PAH) metabolites in bile may be performed to confirm exposure [844]. For bottom-dwelling fish such as flounders or catfish, also analyze the bottom sediments (see Step 6 above). Fish which spend most of their time free-swimming above the bottom in the water column can often avoid toxicity from toxic petroleum compounds in the water column, but if fish are expiring in a confined** habitat (small pond, etc.), EPA GC/MS method 8260 and the NOAA protocol expanded scan*** for PAHs could be performed to see if Benzene, Toluene, Ethyl Benzene, and Xylene (BTEX),

naphthalene, and other potentially toxic compounds are above known acute toxicity benchmark concentrations. Zooplankton populations impacted by oil usually recover fairly quickly unless they are impacted in very confined** or shallow environments [835] and the above BTEX and PAH water methods are often recommended rather than direct analyses of zooplankton tissues.

11a. The concern is for benthic invertebrates: analyze invertebrate whole-body tissue samples and surrounding sediment samples for polycyclic aromatic hydrocarbons (PAHs) and alkyl PAHs using the NOAA protocol expanded scan***. If the spill is fresh* or the source continuous, risk assessment needs may also require that the sediments which form the habitat for benthic invertebrates be analyzed for Benzene, Toluene, Ethyl Benzene, and Xylene (BTEX) and other volatile compounds using EPA GC/MS method 8260 or modified EPA method 8260 in the Selected Ion Mode (SIM). Bivalve invertebrates such as clams and mussels do not break down PAHs as well or as quickly as do fish or many wildlife species. They are also less mobile. Thus, bivalve tissues are more often directly analyzed for PAH residues than are the tissues of fish or wildlife.

11b. The concern is for plants or for vertebrate wildlife including birds, mammals, reptiles, and amphibians: polycyclic aromatic hydrocarbons (PAHs) and other petroleum hydrocarbons break down fairly rapidly in many wildlife groups and tissues are not usually analyzed directly. Instead direct effects are investigated and water, soil, sediment, and food items encountered by wildlife are usually analyzed for PAHs and alkyl PAHs using the NOAA protocol expanded scan***. If the spill is fresh* or the source continuous, risk assessment needs may also require that these habitat media also be analyzed for Benzene, Toluene, Ethyl Benzene, and Xylene (BTEX) and other volatile compounds using EPA GC/MS method 8260 or modified EPA method 8260 in the Selected Ion Mode (SIM). Less is known about plant effects. However, the same methods recommended above for the analyses of water (Step 4 above) and for sediments or soils (Step 6 above) are usually also recommended for these same media in plant or wildlife habitats. If wildlife or plants are covered with oil, see also Step 8 (above) regarding oiling issues.

* Discussion of the significance of the word "fresh": The word "fresh" cannot be universally defined because oil breaks down faster in some environments than in others. In a hot, windy, sunny, oil-microbe-rich, environment in the tropics, some of the lighter and more volatile compounds (such as the Benzene, Toluene, Ethyl Benzene, and Xylene compounds) would be expected to disappear faster by evaporation into the environment and by biodegradation than in a cold, no-wind, cloudy, oil-microbe-poor environment in the arctic. In certain habitats, BTEX and other relatively water soluble compounds will tend to move to groundwater and/or

subsurface soils (where degradation rates are typically slower than in a sunny well aerated surface environment). Thus, the judgement about whether or not oil contamination would be considered "fresh" is a professional judgement based on a continuum of possible scenarios. The closer in time to the original spill of non-degraded petroleum product, the greater degree the source is continuous rather than the result of a one-time event, and the more factors are present which would retard oil evaporation or breakdown (cold, no-wind, cloudy, oil-microbe-poor conditions, etc.) the more likely it would be that in the professional judgement experts the oil would be considered "fresh." In other words, the degree of freshness is a continuum which depends on the specific product spilled and the specific habitat impacted. Except for groundwater resources (where the breakdown can be much slower), the fresher the middle distillate oil contamination is, the more one has to be concerned about potential impacts of BTEX compounds, and other lighter and more volatile petroleum compounds.

To assist the reader in making decisions based on the continuum of possible degrees of freshness, the following generalizations are provided: Some of the lightest middle distillates (such as Jet Fuels, Diesel, No. 2 Fuel Oil) are moderately volatile and soluble and up to two-thirds of the spill amount could disappear from surface waters after a few days [771,835]. Even heavier petroleum substances, such as medium oils and most crude oils will evaporate about one third of the product spilled within 24 hours [771]. Typically the volatile fractions disappear mostly by evaporating into the atmosphere. However, in some cases, certain water soluble fractions of oil including Benzene, Toluene, Ethyl Benzene, and Xylene (BTEX) compounds move down into groundwater. BTEX compounds are included in the more volatile and water soluble fractions, and BTEX compounds as well as the lighter alkanes are broken down more quickly by microbes than heavier semi-volatiles such as alkyl PAHs and some of the heavier and more complex aliphatic compounds. Thus after a week, or in some cases, after a few days, there is less reason to analyze surface waters for BTEX or other volatile compounds, and such analyses should be reserved more for potentially contaminated groundwaters. In the same manner, as the product ages, there is typically less reason to analyze for alkanes using GC/FID techniques or TPH using EPA 418.1 methods, and more reason to analyze for the more persistent alkyl PAHs using the NOAA protocol expanded scan***.

** Discussion of the significance of the word "confined": Like the word "fresh" the word "confined" is difficult to define precisely as there is a continuum of various degrees to which a habitat would be considered "confined" versus "open." However, if one is concerned about the well-being of ecological resources such as fish which spend most of their time swimming freely above the bottom, it makes more sense to spend a smaller proportion of analytical funding for water column and surface water analyses of Benzene, Toluene, Ethyl Benzene, and Xylene (BTEX) and other volatile or acutely toxic compounds if the spill is in open and/or deep waters rather than shallow or "confined" waters. This is because much of

the oil tends to stay with a surface slick or becomes tied up in subsurface tar balls. The petroleum compounds which do pass through the water column often tend to do so in small concentrations and/or for short periods of time, and fish and other pelagic or generally mobile species can often swim away to avoid impacts from spilled oil in "open waters." Thus in many large oil spills in open or deep waters, it has often been difficult or impossible to attribute significant impacts to fish or other pelagic or strong swimming mobile species in open waters. Lethality has most often been associated with heavy exposure of juvenile fish to large amounts of oil products moving rapidly into shallow or confined waters [835]. Different fish species vary in their sensitivity to oil [835]. However, the bottom line is that in past ecological assessments of spills, often too much money has been spent on water column analyses in open water settings, when the majority of significant impacts tended to be concentrated in other habitats, such as benthic, shoreline, and surface microlayer habitats.

*** The lab protocols for the expanded scan of polycyclic aromatic hydrocarbons (PAHs) and alkyl PAHs have been published by NOAA [828].

End of decision tree key.

In response to oil spills, it is important to analyze samples for petroleum PAHs, important alkyl PAHs, and the standard PAHs using the expanded scan of PAHs [828]. This degree of specificity is necessary because oil spill effects are related not so much to the gross amount of oil present as to the levels of key toxic components [468]. Expanded scans typically search for a long list of PAHs and alkylated PAHs at very low detection limits [828]. PAHs in such scans are typically identified by Gas Chromatography with Mass Spectrometry (GC/MS) in the selective ion mode (SIM). In the SIM mode, the GC/MS records intensify for ions that are diagnostic for specific PAHS. Modifications of older lab practices are necessary to get appropriately low detection limits.

Expanded scans for PAHs are often used in conjunction with analyses for aliphatic biomarkers such as triterpanes, steranes, and hopane to help "fingerprint" potential sources. Dibenzothiophenes and related dibenzothiophenol compounds, though not universally considered PAHs, are often included to help complete fingerprinting aspects.

Abstracts on Fingerprinting:

Wade, T.L., T.J. Jackson, T.J. McDonald, J.L. Sericano, and J.M. Brooks. 1993. Oyster Polynuclear Aromatic Hydrocarbon Fingerprinting Applied to the Apex Barge Oil Spill. Society of Environmental Toxicology and Chemistry (SETAC) 14th annual meeting. Westin Galleria and Oaks Houston, TX., (Nov. 14-18 1993), p. 17.

An estimated 692,000 gallons of catalytic feed

stock oil was spilled into Galveston Bay on July 28, 1990, when a tanker collided with three Apex barges in the Houston Ship Channel. Oysters were collected and analyzed from Galveston Bay Todd's Dump (GBTD) before the spill (235 days) and after the spill (6, 37, 132, 495, and 851 days). Oysters were also collected from Galveston Bay Redfish Island (GBRI), a site known to be impacted by the spill, 37 and 110 days after the spill. The spilled oil was also analyzed. The concentration of 18 polynuclear aromatic hydrocarbons (PAHs), measured as part of the National Oceanic and Atmospheric Administration's National Status & Trends (NS&T) showed a sharp increase from 100 ng/g (235 days before the spill) to over 600 ng/g (one week after the spill). Concentrations of these 19 PAHs were also found at GBRI. Fingerprinting techniques applied to data from oyster analyses demonstrated the presence of bioavailable Apex Barge oil 37, 110, 132 days after the spill at GBTD and GBRI. Fingerprinting becomes less diagnostic with time due to possible environmental weathering of the oil.

A.G. Requejo, T. McDonald, G. Denoux, M.C. Kennicutt, R. Sassen, and J.M. Brooks. 1993. Multivariate Analysis of Environmental Data: A tool for interpreting results of "fingerprinting" analyses. Society of Environmental Toxicology and Chemistry (SETAC) 14th annual meeting. Westin Galleria and Oaks, Houston, TX., (Nov. 14-18 1993), p. 17.

Chemical Analyses of environmental samples using "fingerprinting" techniques often result in large quantities of data for each sample. For example, a typical soil or sediment analysis might include concentrations of targeted saturated hydrocarbons, polynuclear aromatic hydrocarbons, chlorinated hydrocarbons, and trace metals, in addition to bulk parameters such as organic carbon and nitrogen content and grain size distributions. The sheer volume and diversity of this type of data can make its interpretation difficult. Multivariate analytical techniques such as Principal Components Analysis (PCA) are ideally suited for the reduction and synthesis of such data sets. PCA employs eigenvector analysis to evaluate the degree of similarity between samples and establish the interrelationship between measured analytes. The major advantages of PCA in comparison to traditional data interpretation approaches are that it is fast, objective, and employs all the data measured. The utility of this approach will be demonstrated using several different sets of

environmental "fingerprinting " data. Included among these are fluorescence and polynuclear aromatic hydrocarbon data from bioremediated soil samples containing petroleum and trace organic and inorganic data from estuarine sediments (Casco Bay, Maine).

Notes on fish bile analysis:

Since many fish species metabolize PAHs faster than other organisms (such as mussels or clams), some contaminants specialists analyze fish bile for PAH metabolites. The presence of the metabolites is a sign that the fish have been exposed to PAHs. A large number (perhaps 30 per site) of samples is typically needed to account for natural variability, as bile can be depleted in an individual fish which has just eaten a big meal (Rick Roy, Fish and Wildlife Service, personal communication). Due to such complications, as well as the difficulty of interpreting the significance of the data once it is obtained, some contaminants specialists prefer dispense with bile analysis and instead to measure PAHs and their alkylated homologues directly in other media (such as sediments, shell fish, or mosquitofish).

A measure of the exposure to aquatic contamination and of the capacity to metabolize PAHs by biota is the induction of the P450 1A enzyme in fish liver [793; Reprinted with permission from Environmental Toxicology and Chemistry, Volume 13, Van Der Weiden, M.E.J., F.H.M. Hanegraaf, M.L. Eggens, M. Celander, W. Seinen, and M. Van Den Berg. Temporal induction of cytochrome P450 1A in the Mirror Carp (Cyprinus Carpio) after administration of several polycyclic aromatic hydrocarbons. Copyright 1994 SETAC].

A promising new method to assess exposure in fish from oil spills is to measure polynuclear aromatic hydrocarbon (PAH) metabolites in fish bile. PAH exposure in fish may not be accurately assessed by tissue burden because fish rapidly convert PAHs to a variety of metabolites. Biliary PAH metabolites are a short-term environmental indicator of the bio-available level of PAH to fish from oil spills [545].

Currently PAH metabolite concentrations in fish bile cannot be directly related to fish mortalities because comparative dose-response experiments have not been conducted. Minimally, however, one may be able to assume that the PAH metabolite levels in the bile represent recent levels of environmental exposure [545].

Biomarkers: MFO enzymes play a role in the metabolism of liposoluble foreign compounds including PAHs. The most sensitive catalytic probes for determining induction in fish appear to be EROD and AHH. In most fish species, these cytochrome P450 dependent enzymes are highly inducible upon exposure to PAH-type compounds. The activity of MFO enzymes is a useful early biochemical indicator of pollution in the environment [519].

Some common biochemical indicators include the following:

1) PAH exposure causes an increase in MFO activity [625].
2) PAHs can function as xenoestrogens [924].

3) PAHs produce and increase in intracellular oxidation and chromosomal breaks or gene rearrangements [924].
4) PAH exposure causes and increase in glutathione levels.
5) EROD induction was observed in the liver of flounder after exposure to petroleum sources of PAHs [519].

Various EPA Methods (Note: as mentioned above, many of the standard EPA methods are inadequate for risk or damage assessment purposes):

EPA (RCRA Group) publishes requirements for solid waste methods in 40 CFR Part 261, Appendix III, with details in the following periodically updated publication [1013]:

Environmental Protection Agency. 1997. Test methods for evaluating solid waste, physical/chemical methods, SW-846, EPA Office of Solid Waste and Emergency Response, EPA, Washington, D.C. Update 3 finalized in 1997. Available from NTIS or GPO. Previous 1995 update 2 was available on CD-ROM [1013].

RCRA (SW-846) methods tend to include provisions for using the specified method or something better. RCRA SW-846 methods typically require instrument calibration before analyses, but some labs don't do it, and many labs actually use some kind of hybrid between RCRA, CERCLA, or various other "standard protocols" (Roy Irwin, Park Service, Personal Communication, 1997, based on conversations with various EPA and private lab staff members). The guidance in SW-846 must be used in some states, but is considered "guidance of acceptable but not required methods" in most federal applications. In the past, EPA has also published separate (not SW-846) guidance documents with suggestions on field sampling and data quality assurance related to sampling of sediments [1016] and soils [1017,1018,1019].

EPA (CERCLA) publishes various Contract Laboratory Program (CLP) methods documents periodically, available from EPA and NTIS. CLP methods were designed for use in contaminated areas and often have detection limits that are not low enough for use in relatively clean areas or where low detection levels are needed in comparison with low concentration criteria or benchmarks. CERCLA CLP methods tend to require things done exactly per contract specifications. A few examples of CLP publications (this list is not complete) [861]:

User's Guide CLP CERCLA User's Guide to the Contract Laboratory Program. USEPA - Office of Emergency and Remedial Response. Dec 1988

9240_0-0XFS Multi-Media/Conc Superfund OSWER CERCLA Multi-Media, Multi-Concentration Organic/Inorganic Analytical Service for Superfund, Quick Reference Fact Sheets, 9240.0-08FS (organic) and 9240-0-09FS (inorganic), August 1991. The

organic/inorganic analytical service provides a technical and contractual framework for laboratories to apply EPA/Contract Laboratory Program (CLP) analytical methods for the isolation, detection and quantitative measurement of 33 volatile, 64 semi-volatile, 28 pesticide/Aroclor, and 24 inorganic target analytes in water and soil/ sediment environmental samples.

AOC/Contract Laboratory Program (CLP), Routine Analytical Services, Summary on EPA Home Page under Superfund Subdirectory, EPA Office of Remedial and Emergency Response, 1997, Internet.

Other less rigorous scanning methods for various PAHs in drinking water have included High pressure liquid chromatography (EPA 550, 550.1); gas chromatographic/mass spectrometry (EPA 525): PQL= 0.0002 mg/L [893].

Alternatives to Expanded Scans:

Semi-Volatiles, Lab Analyses using EPA Method 8270 Recommended in the past by: Consultants, Many Laboratories, EPA.

Pros: A more complete list of analytes than the screening methods (although not as complete as the expanded scans). Includes semi-volatiles and priority pollutants other than oil compounds, which could possibly be an advantage if one just happened to find a contaminant not expected in oil.

Cons: More expensive than screening scans. List of oil analytes incomplete compared to expanded scan, standard detection limits used often to high, making the scan prone to false negatives. The typically high detection limits often used with the standard EPA method 8270 makes the scan less than desirable than the modified (SIM) version for hazard/risk assessment or contamination mapping purposes.

Typical Costs per Sample (based on a survey of several laboratories in 1995):

Lab 1 (GCC):

EPA Method Analysis: Price per sample ($)
GC/MS Volatile Organics (Method 625, 8270):

Water	$400
Soil	$425

Lab 2 (CAS): $300-380 for standard EPA 8270 (detection limits 10 ppb water, ~600 ppb tissue, ~300 ppb sediments; up to $600 for all modified method 8270/SIM with lower detection limits (1 ppb

water, 20 pb tissues, and 10 ppb sediments).

Lab 3 (GERG) As low as $350 if extraction not included.

Lab 4 (ADL): $350-400.

Summary: An alternative which includes some, but not nearly all of the oil compounds (mostly just the priority pollutant, parent compound PAHs). It is better than screening methods but not nearly as appropriate for many oil applications as the NOAA expanded scan (see discussion above). An alternative which works for some general CERCLA applications, but is not particularly well suited to oil contamination.

EPA Description [861]:

OSW 8270A S Semivolatiles - Soil 228 SW-846 CGCMS ug/kg EQL Method 8270A "Semivolatile Organic compounds by Gas Chromatography/Mass Spectrometry (GC/MS): Capillary Column Technique" A table of analyte specific sample preparation procedures that may be used is given in this method [861]. The two procedures that cover most of the analytes are 3510 and 3580 [861]. Prior to using this method, the samples should be prepared for chromatography using the appropriate sample preparation and cleanup methods [861]. This method describes chromatographic conditions that will allow for the separation of the compounds in the extract [861].

OSW 8270A W Semivolatiles - Water 228 SW-846 CGCMS ug/L EQL Method 8270A "Semivolatile Organic compounds by Gas Chromatography/Mass Spectrometry (GC/MS): Capillary Column Technique" A table of analyte specific sample preparation procedures that may be used is given in this method [861]. The two procedures that cover most of the analytes are 3510 and 3580 [861]. Prior to using this method, the samples should be prepared for chromatography using the appropriate sample preparation and cleanup methods [861]. This method describes chromatographic conditions that will allow for the separation of the compounds in the extract [861].

EPA Method 8100.

This a GC/FID method which is typically less rigorous and often has higher detection limits and less specifivity that the expanded scan. There are various modifications of the standard 8100 analysis. For example, a Diesel Range Organics

analysis is sometimes referred to as a DRO Modified EPA method 8100. The GC/FID method is a modification of EPA methods 8100 and 8015 and ASTM method 3328-90 [657].

> Method 8100 is used for the analysis of various PAHs. Samples are extracted, concentrated and analyzed using direct injection of both neat and diluted organic liquids. The method provides two optional GC columns that are better than Column 1 and that may help resolve analytes from interferences. Solvents, reagents and glassware may introduce artifacts. Other interferences may come from coextracted compounds from samples [731].

Some labs have used EPA 8100 to analyze for a few PAHs not on other standard scans, such as (Gary Rosenlieb, National Park Service, Personal Communication, 1997):

Dibenz(a,h)acridine

Dibenz(a,j)acridine

7H-Dibenzo(c,g)carbazole

Dibenzo(a,i)pyrene

Dibenzo(a,e)pyrene

Dibenzo(a,h)pyrene

3-Methylchloanthrene

EPA gave the following description of EPA Method 8100 in the EMMI database [861]:

> OSW 8100 PAH's - CGCFID 24 SW-846 (CGCFID) ug/L Method 8100 "Polynuclear Aromatic Hydrocarbons" Method 8100 provides gas chromatographic conditions for the detection of certain polynuclear aromatic hydrocarbons [861]. Prior to the use of this method, appropriate sample extraction techniques must be used [861]. Both neat and diluted organic liquids (Method 3580, Waste Dilution) may be analyzed by direct injection [861]. A 2-5 uL aliquot of the extract is injected into a gas chromatograph using the solvent flush technique, and compounds in the GC effluent are detected by a flame ionization detector [861]. If interferences prevent proper detection of the analytes of interest, the method may also be performed on extracts that have undergone cleanup using silica gel column cleanup [861].
>
> Typical 8100 prices (based on 1992 prices supplied by Global Geochemistry Corporation) -- no government endorsement implied). The following prices may have

changed by the time you read this and are supplied only to show the relative costs vs typical prices for other analyses mentioned in this entry; actual lab prices will vary. The listed cost is per sample, assuming only one to four samples. With most labs, as the number of samples increases, and/or the number of different analysis performed on each sample increases, the price per sample decreases:

For Polynuclear Aromatic Hydrocarbons (Methods 610, 8100):

EPA Method Analysis:	Price per sample ($)
Water	125
Soil	140

HPLC Screening:

In cases where a less expensive screening scan is desired, consider using an HPLC/Fluorescence scan method for sediment or bile metabolite samples. Such scans are available from laboratories at Texas A. and M., Arthur D. Little, the NOAA lab in Seattle.

Some labs use screening HPLC fluorescence methods to screen for alkylated naphthalenes and dibenzothiophenes that fluoresce at naphthalene wavelengths and the alkylated phenanthrenes that fluoresce at phenanthrene wavelengths [521]. Other HPLC/fluorescence scans are used to examine fish bile directly for the presence of metabolites of PAHs such as naphthalene [523]. In these methods, ratios of phenanthrene or naphthalene equivalents to benzo(a)pyrene (BaP) equivalents from bile screening can be used to differentiate the source of the aromatic hydrocarbons contamination [523]. The naphthalene/BaP ratios should be higher in fish exposed to crude oil than those exposed in pyrogenic urban PAHs in sediments [523].

Typical minimum sample sizes for aliphatic and/or aromatic scans include the following (John Moore, NBS, personal communication, 1990):

Tissue	15 G
Sediment	100 G
Water	500 ML

Other information on Petroleum Hydrocarbons, Fluorescence Screening Methods [521,522]:

Recommended By: National Park Service Staff, NOAA staff, and many laboratories for certain screening applications (such as crude oil).

Pros: Not much more expensive, and less prone to false negatives and various other problems than some of the more common screening methods (TPH-EPA 418.1 and Oil and

Grease). Less expensive than some of the more rigorous scans. Screening measures the total fluorescence of oil components while GC/MS measures individual aromatic compounds [521]. Thus, HPLC/fluorescence screening allowed detecting lower concentrations of petroleum-related aromatic compounds in samples contaminated by Prudhoe Bay Crude Oil than did analysis by GC/MS [521]. The HPLC/fluorescence scan can be used for analyses of fish bile: the scan looks at bile directly for the presence of metabolites of PAHs: naphthalene, phenanthrene, and benzo[a]pyrene. The technique does not identify or quantify actual PAH compounds, but subsequent gas chromatography analyses can done to confirm the initial findings. Even the semi-quantitative Total Scanning Fluorescence (TSF) done inexpensively by labs such as GERG are a better measure of PAH contamination than GC/FID, which measures less persistent and less hazardous aliphatics.

> Additional Pros: HPLC Fluorescence screening methods have been performed extensively by NOAA to locate hotspots for crude oil contamination. NOAA's experience with the Exxon Valdez spill indicated that concentrations of aromatic hydrocarbons measured by HPLC/Fluorescence screening were highly correlated with the sums of Aromatic hydrocarbons determined by GC/MS, thus validating the screening method as an effective tool for estimating concentrations of petroleum-related aromatic hydrocarbons in sediments. Moreover, differences in HPLC chromatographic patterns among sediments suggested different sources of contamination, e.g., crude oil or diesel fuel. Allows crude determinations related to sources: HPLC/Fluorescence analyses allowed at least rough differentiation between aromatic hydrocarbons which may have originated from diesel fuel versus those from boat traffic [521] and The procedure was successfully applied to fingerprinting' gasolines, kerosines, diesel oils, heavy fuel oils, lubricating oils, and ship bilge oils [AUTHOR: Saner WA; Fitzgerald GE, II PUBLICATION YEAR: 1976 TITLE: Thin-Layer Chromatographic Technique for Identification of Waterborne Petroleum Oils JOURNAL: Environmental Science and Technology SOURCE: Vol. 10, No. 9, p 893-897, September 1976. 6 fig, 4 tab, 7 ref.].

Cons: The fluorescence methods do not identify or quantify actual PAH compounds, but subsequent gas chromatography analyses can done to confirm the initial findings (Tom MacDonald, Texas A&M, personal communication to Roy Irwin). Not as good for biological effects and potential source/origin interpretation as the

more rigorous methods listed below in section B): Risk/Hazard Assessment and Source Identification Methods.

Other HPLC EPA methods [861]:

EMSLC 550 PAH's - LLE/HPLCUV & FL 16 DRINKING_WATER_1 HPLCFL ug/L MDL "Determination of Polycyclic Aromatic Hydrocarbons in HPLCUV Drinking Water by Liquid-Liquid Extraction and HPLC with Coupled Ultraviolet and Fluorescence Detection" A measured volume of sample, approximately 1 L, is serially extracted with methylene chloride using a separatory funnel [861]. The methylene chloride extract is dried and concentrated to a volume of 1 mL [861]. A 3.0 mL portion of acetonitrile is added to the extract and concentrated to a final volume of 0.5 mL [861]. The extract analytes are then separated by HPLC [861]. Ultraviolet adsorption (UV) and fluorescence detectors are used with HPLC to quantitatively measure the PAHs [861].

EMSLC 550.1 PAH's - LSE/HPLCUV & FL 16 DRINKING_WATER_1 HPLCFL ug/L MDL "Determination of Polycyclic Aromatic Hydrocarbons in HPLCUV Drinking Water by Liquid-Solid Extraction and HPLC with Coupled Ultraviolet and Fluorescence Detection" This method is for determination of polycyclic aromatic hydrocarbons (PAH) in drinking water sources and finished drinking water [861]. Polycyclic aromatic hydrocarbons and internal standards are extracted from a water sample by passing 1 liter of sample through a cartridge containing about 1 gram of a solid inorganic matrix coated with a chemically bonded C-18 organic phase (liquid-solid extraction, LSE) [861]. The compounds are eluted from the cartridge or disk with a small quantity of methylene chloride, dried, and concentrated further to 1 mL [861]. A 3.0 mL portion of acetonitrile is added to the extract and concentrated to a final volume of 0.5 mL [861]. The extract is then separated by HPLC and detected by UV and fluorescence detectors [861]. Matrix interferences have been found for benzo(a)-anthracene, benzo(a)pyrene and benzo(g,h,i)perylene [861]. Poor recovery has been experienced for these analytes in dechlorinated tap water [861]. The nature of the interferences has not been fully assessed [861].

Note: methods 550 and 550.1 are recommended in IRIS for drinking water analyses [893].

Typical Costs per Sample (based on a survey of several

laboratories in 1995):

Example 1 (Lab GERG): For TSF (Total Scanning Fluorescence):

Tissue: $20 (or $60 with extraction).
Sediment: $20 (or $60 with extraction).

Summary: An acceptable screening technique for certain types of oil products if the results at the particular site are first found to be consistently correlated with the results of the more rigorous methods such as the NOAA expanded scan.

EPA Method 8310:

EPA Method 8310 (for Polynuclear Aromatic Hydrocarbons):

One EPA method used for PAHs. Method 8310 is used for the analysis of 16 polynuclear aromatic hydrocarbons (PAHs). Samples are extracted, concentrated and analyzed using HPLC with detection by UV and fluorescence detectors. Solvents, reagents and glassware may introduce artifacts. Other interferences may come from coextracted compounds from samples [731].

EPA description [861]:

OSW 8310 PAH's - HPLCFL, HPLCUV 16 SW-846 HPLCFL ug/L MDL Method 8310 "Polynuclear Aromatic Hydrocarbons" HPLCUV Method 8310 provides high performance liquid chromatographic (HPLC) conditions for the detection of certain polynuclear aromatic hydrocarbons [861]. Prior to use of this method, appropriate sample extraction techniques must be used [861]. A 5 - 25 uL aliquot of the extract is injected into an HPLC, and compounds in the effluent are detected by ultraviolet and fluorescence detectors [861]. If interferents prevents proper detection of the analytes of interest, the method may also be performed on extracts that have undergone cleanup using silica gel column cleanup (Method 3630) [861].

EPA Method 610:

EPA Description [861]:

EMSLC 610 PAH's - GCFID, HPLCUV, HPLCFL 16 WASTEWATER GCFID ug/L MDL "Polynuclear Aromatic Hydrocarbons" HPLCFL HPLCUV This is a chromatographic method

applicable to the determination of polynuclear aromatic hydrocarbons in municipal and industrial discharges as provided under 40 CFR 136.1 [861]. When this method is used to analyze unfamiliar samples, compound identifications should be supported by at least one additional qualitative technique [861]. This method provides for both high performance liquid chromatographic (HPLC) and gas chromatographic (GC) approaches for the determination of PAHs [861]. The gas chromatographic procedure does not adequately resolve the following four pairs of compounds: anthracene and phenanthrene; chrysene and benzo(a)anthracene; benzo(b)fluoranthene and benzo(k)fluoranthene; and dibenzo(a,h)anthracene and indeno(1,2,3-cd)pyrene [861]. Unless the purpose for the analysis can be served by reporting the sum of an unresolved pair, the liquid chromatographic approach must be used for these compounds [861]. The liquid chromatographic method does resolve all of the PAHs listed [861]. A measured volume of sample, approximately 1-L, is extracted with methylene chloride using a separatory funnel [861]. The methylene chloride extract is dried and concentrated to a volume of 10 mL or less [861]. The extract is then separated by HPLC or GC [861]. Ultraviolet (UV) and fluorescence detectors are used with HPLC to identify and measure the PAHs [861]. A flame ionization detector is used with GC [861]. The method provides a silica gel column cleanup procedure to aid in the elimination of interferences that may be encountered [861].

The water-soluble fractions of unleaded gasoline, kerosene and diesel fuel were evaluated by EPA Methods 602, 610, and 625 [AUTHOR: Thomas DH; Delfino JJ PUBLICATION YEAR: 1991 TITLE: Gas Chromatographic/Chemical Indicator Approach to Assessing Ground Water Contamination by Petroleum Products JOURNAL: Ground Water Monitoring Review GWMRDU SOURCE: Vol. 11, No. 4, p 90-100, Fall 1991. 5 fig, 6 tab, 24 ref. Florida Dept. of Environmental Regulation Contract WM246, part of an abstract contributed by Carol Schuler, Fish and Wildlife Service, Portland Oregon].

Typical prices (based on 1992 prices supplied by Global Geochemistry Corporation) -- no government endorsement implied). The following prices may have changed by the time you read this and are

supplied only to show the relative costs vs typical prices for other analyses mentioned in this entry; actual lab prices will vary. The listed cost is per sample, assuming only one to four samples. With most labs, as the number of samples increases, and/or the number of different analysis performed on each sample increases, the price per sample decreases:

For Polynuclear Aromatic Hydrocarbons (Methods 610, 8100):

EPA Method Analysis: Price per sample ($)

Water	125
Soil	140

GC/FID Screens in General:

Gas Chromatography with Flame Ionization Detection (GC/FID,:

Recommended For Analyses of Fresh Aliphatics By: National Park Service staff, many consultants (used extensively at Exxon Valdez spill for aliphatics), and many laboratories for certain screening applications.

Pros: Less prone to false negatives and various other problems than some of the more common screening methods (TPH-EPA 418.1 and Oil and Grease). More inexpensive than rigorous scans.

Cons: Not as good for biological effects and potential source/origin interpretation as the more rigorous methods listed below in section B): Risk/Hazard Assessment and Source Identification Methods. At some labs, not too much cheaper than the (more complete) NOAA expanded scan for PAHs. Concentrates on aliphatics, which are less persistent and less hazardous than PAHs.

Typical Costs:

Lab 1: The FID analysis cost about $225 to 300 per sample in 1994 (it partly depends on the number of samples).

Lab 2 (BNW): The GC/FID fuel fingerprint was available for about $220 per sample in early 1995.

Lab 3 (GERG): TPH by GC/FID: $100 for water or sediment. Similar GC-C15 method at GERG lab as low as $70.

Lab 4 (BDUX): The GC/FID analysis was available for

about $100 per sample for 150 samples in early 1995.

Lab 5 (ADL): The GC/FID analysis was available for about $300 per sample for in early 1995.

Summary: An acceptable screening technique for certain types of oil products: It can be sufficient for screening purposes when the oil contamination is fresh, unweathered oil and when one is fairly sure of the source [657]. The GC/FID scan is used for screening higher levels of fresh or moderately degraded hydrocarbons of mostly known composition, and is not as good as the expanded GC/MS SIM scan for identifying the full range of PAHs down to lowest known benchmark levels. It could be used if the results at the particular site are first found to be consistently correlated with the results of the more rigorous methods listed below in section B): Risk/Hazard Assessment and Source Identification Methods.

Detection Limits: The detection limits used for most modified GC/FID methods ranges from 1 to 10 mg/kg dry weight for soils and from 10 to 50 ug/L for waters [657].

Total Petroleum Hydrocarbons:

Various TPH methods don't measure PAHs very well. Among the other problems with total petroleum hydrocarbon analyses: Different methods used to generate total petroleum hydrocarbon concentrations, or other similar measures of petroleum contamination, all produce very different numbers [831]. For example, gasoline saturated soil produced the following concentrations (mg/kg = ppm) [831]:

Total Volatile Solids by EPA 160.4: 3,200
TPH by EPA 418.1: 140,110
TPH-G (GRO by GC/FID or GC/MS): 1,500
Naphthalene (a PAH) by EPA 8270: 13
Benzene by EPA 8260: 3.4
Ethyl Benzene by EPA 8260: 77
Toluene by EPA 8260: 150
Xylene by EPA 8260: 420
Original Gasoline by Column Mass Differences: 15,300

As the product spills or moves to or through different media, the above given proportions change. For example, aqueous leaching of the gasoline saturated soils documented above reduced TPH 418.1 more than it reduced TPH-G [831]. Following aqueous leaching, the concentrations were the following [831]:

Total Volatile Solids by EPA 160.4: 3,600
TPH by EPA 418.1: <25
TPH-G (GRO by GC/FID or GC/MS): 390-400
Naphthalene by EPA 8270: 2.7

Benzene by EPA 8260: <0.025
Ethyl Benzene by EPA 8260: 3.7
Toluene by EPA 8260: 0.13
Xylene by EPA 8260: 25
Original Gasoline by Column Mass Differences: 15,200

Additional Information on EPA method 418.1: Petroleum Hydrocarbons expressed as Total Petroleum Hydrocarbons (TPH).

Recommended By: Many State agencies, some consulting firms, and some laboratories for certain regulatory and screening applications (often leaking underground storage tanks). Total Petroleum Hydrocarbons (TPH) is an indicator that is commonly used in cleanup standards for motor fuel contaminated soils. Its chief popularity is a function of its relatively simple and inexpensive analytical procedure, plus its title, which intuitively (but incorrectly) provides the impression that it measures all petroleum hydrocarbons.

Pros: Inexpensive. Necessary in certain states due to regulatory requirements, so if one has to do it anyway it is not an extra expense. Said to readily detect heavy petroleum products such as motor oil, tar, and asphalt [497] (but misses many hazardous heavy PAHs (see below).

Cons: Not well suited to many types of oil contamination or to the more persistent hazardous constituents in oil. Done differently by different labs and prone to numerous problems too lengthy to list here (for details, see entry entitled: Petroleum Hydrocarbons expressed as Total Petroleum Hydrocarbons (TPH). Usually useless for interpretation related to biological effects and potential sources/origins. Low values tend to give the mistaken impression that a site is clean when it really isn't (prone to false negatives). A field test of bioremediation of soils contaminated with Bunker C at a refinery in Beaumont, Texas, utilized oil and grease data, which (although the data was quite variable) seemed to indicate bioremediation was taking place [728]. A comparison of the oil and grease data at this site with TPH data at this site suggested the same thing, that the data was quite variable but if anything, the oil was being slowly being cleaned up by bioremediation (Bruce Herbert, Texas A. and M., Department of Geology, personal communication, 1995). However, a later study of the same site utilizing the expanded scan for PAHs (a modified EPA 8270 including alkyl homologues and lower detection limits), indicated that very little bioremediation of hazardous alkyl PAHs and multi-ring PAHs was actually taking place [727]. Thus, utilizing either oil and grease or TPH analyses would tend to lead one to the faulty conclusion that the harmful compounds were being naturally cleaned up at an acceptable rate. This is

partly because the TPH and oil and grease methods tend to favor the lighter and less alkylated PAHs, whereas many of the carcinogenic and longer lasting PAHs are the heavier multi-ringed and alkylated compounds.

Additional cons: Prone to interference from water. Not only does it not cover heavier PAHs, the method specifically states that it does not accurately measure the lighter fractions of gasoline (which would include BTEX) [497]. When the regulatory objective is protection of groundwater quality, it would seem most appropriate to focus on specific and more mobile compounds like BTEX as the best indices of potential groundwater risks [497].

Original EPA description:

EMSLC 418.1 Total Petroleum Hydrocarbons 1 MCAWW SPECTR mg/L DL "Petroleum Hydrocarbons, Total Recoverable" The sample is acidified to pH <2 and serially extracted with fluorocarbon-113 in a separatory funnel [861]. Interferences are removed with silica gel adsorbent [861]. Infrared analysis of the extract is performed by direct comparison with standards [861].

Typical Costs per Sample (based on a survey of several laboratories in 1995):

Total Petroleum Hydrocarbons (Method 418.1)

Water, LAB GGC: $60
Soil, LAB GGC: $65

Lab BNW: $60-70

Soil, Lab Gerg $50

Lab ADL: $100

Summary: An often unacceptable oil screening method which is nevertheless required by many states. Numerous technical procedural shortcomings limit its utility as an accurate and reliable analytical method [497]. Its current widespread use as a soil cleanup criterion is a function of a lack of understanding of its proper application and limitations [497]. This method is sometimes marginally acceptable for screening certain types of oil contamination if the results at the particular site are first found to be consistently correlated with the results of the more rigorous methods, such as the NOAA expanded scan.

Detailed information on Oil and Grease Methods:

Has been recommended in the past by: Some consulting firms and laboratories for certain screening applications.

Pros: Inexpensive.

Cons: Responds to vegetable oil and other non-petroleum substances. Not well suited to many of the more hazardous and more persistent constituents in oil. When applied to petroleum products, prone to numerous problems too lengthy to list here (for details, see entry entitled: Oil and Grease. Usually useless for interpretation related to biological effects and potential sources/origins. Oil and grease is difficult (if not impossible) to interpret related to petroleum hydrocarbon levels; scatter plots of oil and grease levels versus the levels of petroleum hydrocarbons often appear random (Brian Cain, U.S.Fish and Wildlife Service, personal communication). A field test of bioremediation of soils contaminated with Bunker C at a refinery in Beaumont, Texas, utilized oil and grease data, which (although the data was quite variable) seemed to indicate bioremediation was taking place [728]. A comparison of the oil and grease data at this site with TPH data indicated that TPH was suggesting the same thing, that the data was quite variable but if anything, the oil was being slowly being cleaned up by bioremediation (Bruce Herbert, Texas A. and M., Department of Geology, personal communication, 1995). However, a later study of the same site utilizing the expanded scan for PAHs [828] (a modified EPA 8270 including alkyl homologues and lower detection limits), indicated that very little bioremediation of hazardous alkyl PAHs and multi-ring PAHs was actually taking place [727]. Thus, utilizing either oil and grease or TPH analyses would tend to lead one to the faulty conclusion that the harmful compounds were being naturally cleaned up at an acceptable rate. This is partly because the TPH and oil and grease methods tend to favor the lighter and less alkylated PAHs, whereas many of the carcinogenic and longer lasting PAHs are the heavier multi-ringed and alkylated compounds.

Typical Costs per Sample (based on a survey of several laboratories in 1995):

Lab BNW: $60

Summary: Designed for other purposes, oil and grease is usually unacceptable as screening method for petroleum based oil contamination. It has nevertheless sometimes been used for this purpose by certain industrial and consulting firm groups.

This method would only be marginally acceptable for

screening certain types of oil contamination if the results at the particular site are first found to be consistently correlated with the results of the more rigorous methods such as the NOAA expanded scan.

There are numerous oil and grease methods published by APHA, ASTM, USGS, and EPA [861]. EPA's methods are described as follows [861]:

EMSLC 413.1 Oil and Grease - Gravimetric 1 MCAWW GRAV mg/L RNGE "Oil and Grease, Total Recoverable (Gravimetric, Separatory Funnel Extraction)" The sample is acidified to a low pH (less than 2) and serially extracted with fluorocarbon-113 in a separatory funnel [861]. The solvent is evaporated from the extract and the residue weighed [861].

EMSLC 413.2 Oil and Grease - IR 1 MCAWW SPECTR mg/L RNGE "Oil and Grease, Total Recoverable (Spectrophotometric, Infrared)" The sample is acidified to a low pH (less than 2) and serially extracted with fluorocarbon-113 [861]. The oil and grease is determined by comparison of the infrared absorbance of the sample extract with standards [861].

OSW 9070 Oil & Grease 1 SW-846 GRAV mg/L RNGE Method 9070 "Total Recoverable Oil and Grease (Gravimetric, Separatory Funnel Extraction)" The one-liter sample is acidified to a low pH (2) and serially extracted with fluorocarbon-113 in a separatory funnel [861]. The solvent is evaporated from the extract and the residue is weighed [861].

OSW 9071A Oil & Grease (Sludge) 1 SW-846 GRAV mg/L RNGE Method 9071A "Oil and Grease Extraction Method for Sludge Samples" A 20 g sample of wet sludge with a known dry-solids content is acidified to pH 2.0 with 0.3 mL concentrated HCl [861]. Magnesium sulfate monohydrate will combine with 75% of its own weight in water in forming MgSO4_7H2O and is used to dry the acidified sludge sample [861]. Anhydrous sodium sulfate is used to dry samples of soil and sediment [861]. After drying, the oil and grease are extracted with Fluorocarbon-113 using the Soxhlet apparatus [861].

USGS O3108 Extractable Oil and Grease 1 USGS_METHODS GRAV mg/L RNGE "Oil and grease, extractable, extraction-gravimetric" This method is suitable for the determination of oil and grease in water-suspended-sediment mixtures containing at

least 1 mg/L of the analyte [861]. A sample is extracted twice with trichloro- trifluoroethane and the extract is evaporated at 20C to leave a nonvolatile residue whose weight represents an estimate of the extractable organic matter in the sample [861].

EAD 1652 Oil and Grease 1 EAD_METHODS GRAV mg/L DL "Oil and Grease by Solid Phase Extraction" This method is used to determine total oil and grease and oil and grease amenable to solid phase extraction [861]. This method measures the materials that may be extracted on a bonded silica solid phase sorbent material from surface water, saline water, industrial, and domestic wastewater [861]. It is applicable to the determination of relatively non-volatile hydrocarbons, vegetable oils, animal fats, waxes, soaps, greases, and related matter [861]. The method is not applicable to measurement of light hydrocarbons that volatilize at temperatures below 70C [861]. Petroleum fuels in the range from gasoline through No [861]. 2 fuel oils are completely or partially lost in the solvent removal operation [861]. The sample is acidified to a pH <2 and drawn through a bonded silica sorbent material [861]. The oil and grease remain on the solid phase sorbent while the aqueous phase passes through [861]. The oil and grease are then eluted with an organic solvent into an evaporating vessel [861]. The solvent is evaporated from the extract, and the remaining residue is weighed [861].

Biomarker Screens:

Biomarker screens (such as biomarker P450 RGS) screens have also been used by some individuals.

Recommended in the past by: Some individuals, consulting firms, and laboratories for certain screening applications.

Pros: Inexpensive.

Cons: Not widely used, triggered by organochlorines and various hazardous compounds not found in oil. Impossible to use for risk assessment or general interpretation related to biological effects and potential sources/origins (used only to identify hotspots).

Typical Costs per Sample (based on a survey of several laboratories in 1995): $75-$150 (Lab CAS). Water samples wouldn't require extraction, so just $75; but CAS labs suggest extraction to concentrate the compounds (due

to low water solubility); this will increase detection power. The breakdown of the quoted $75-150 charge for the P450 RGS scans is as follows: $75 for the actual test; includes 3 reps, $20-75 for the extraction technique: $20 for extraction of high level contamination; sonicate in solvent, called "short extraction" 50 for EPA 3540 extraction, $75 for tissue extraction, so $95-125 is a typical estimate for P450 RGS scans.

Summary: An alternative which is not yet widely used or accepted. This method would only be acceptable for screening certain types of fresh light product contamination if the results at the particular site are first found to be consistently correlated with the results of the more rigorous methods such as the NOAA expanded scans for PAHs.

EPA Method 525: Determination of Organic Compounds in Drinking Water by Liquid-Solid Extraction and Capillary Column Gas Chromatography/Mass Spectrometry. EPA method 525 allows low detection limits [893]. Detection limits (PQL) = 0.0002 mg/L [893]. Thus the method is used for drinking water and risk assessment and is described by EPA as follows [861]:

EMSLC 525 Organics in Water DRINKING_WATER CGCMS "Determination of Organic Compounds in Drinking Water by Liquid-Solid Extraction and Capillary Column Gas Chromatography/Mass Spectrometry" Organic compound analytes, internal standards, and surrogates are extracted from a water sample by passing 1 liter of sample water through a cartridge containing about 1 gram of a solid inorganic matrix coated with a chemically bonded C18 organic phase (liquid-solid extraction, LSE) [861]. The organic compounds are eluted from the LSE cartridge with a small quantity of methylene chloride, and concentrated further by evaporation of some of the solvent [861]. The sample components are separated, identified, and measured by injecting an aliquot of the concentrated methylene chloride extract into a high resolution fused silica capillary column of a gas chromatography/ mass spectrometry (GC/MS) system [861]. Compounds eluting from the GC column are identified by comparing their measured mass spectra and retention times to reference spectra and retention times in a data base [861]. Reference spectra and retention times for analytes are obtained by the measurement of calibration standards under the same conditions used for samples [861]. The concentration of each identified component is measured by relating the MS response of the quantitation ion produced by that compound to the MS response of the quantitation ion produced by a compound that is used as an internal standard [861]. Surrogate analytes, whose concentrations are known in every sample, are measured with the same internal standard calibration procedure

[861].

Misc. Oil or PAH Related Scans:

EPA Method 625:

Semivolatile priority pollutant organics method 625 is inadequate for generating scientifically defensible information for Natural Resource Damage Assessments [468]. Method 625 is a GC/MS method [861]. 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].

The water-soluble fractions of unleaded gasoline, kerosene and diesel fuel were evaluated by EPA Methods 602, 610, and 625 [AUTHOR: Thomas DH; Delfino JJ PUBLICATION YEAR: 1991 TITLE: Gas Chromatographic/Chemical Indicator Approach to Assessing Ground Water Contamination by Petroleum Products JOURNAL: Ground Water Monitoring Review GWMRDU SOURCE: Vol. 11, No. 4, p 90-100, Fall 1991. 5 fig, 6 tab, 24 ref. Florida Dept. of Environmental Regulation Contract WM246, part of an abstract contributed by Carol Schuler, Fish and Wildlife Service, Portland Oregon].

EPA descibes method 625 as follows [861]:

EMSLC 625 Base/Neutrals and Acids 84 WASTEWATER GCMS ug/L MDL "Base/neutrals and Acids" A measured volume of sample, approx 1-L, is serially extracted with methylene chloride at a pH greater than 11 and again at a pH less than 2 using s separatory funnel or a continuous extractor [861]. The methylene chloride extract is dried, concentrated to a volume of 1 mL, and analyzed by GC/MS [861]. Qualitative identification of the analytes in the extract is performed using the retention time and the relative abundance of three characteristic masses (m/z) [861]. Quantitative analysis is performed using internal standard techniques with a single characteristic m/z [861].

EMSLC 625-S Organics in Sludge - ABN 80 WASTEWATER_1 CGCMS ug/L MDL "Protocol for the Analysis of Extractable Organic GCMS Priority Pollutants in Industrial and Municipal Wastewater Treatment Sludge" This method uses repetative solvent extraction aided by a high-speed homogenizer [861]. The

extract is separated by centrifugation and removed with a pipette or syringe [861]. Sludges are extracted at pH >= 11 to isolate base/neutral compounds and at pH <= 2 to isolate acidic compounds [861]. Extracts containing base/neutral compounds are cleaned by silica gel or florisil chromatography or by gel permeation chromatography (GCP) [861]. Extracts containing the acidic compounds are cleaned by GPC [861]. The organic priority pollutants are determined in the cleaned extracts by capillary column or packed column GC/MS [861]. Extract cleanup by slilica gel or Florisil chromatography and analysis by capillary GC/MS (HRGC/MS) is preferred since HRGC/MS allows easier data interpretation [861].

EPA Method 3450 (Extraction):

EPA method 3450 is just the extraction; not the actual analysis for oil compounds. Typical costs: (Lab CAS): $50 for EPA 3540 extraction.

Outdated Scans: Historically, only parent-compound PAHs were analyzed in typical laboratory scans (such as the previous standard Fish and Wildlife Service contract laboratory scan). There is less literature on the biological effects of alkylated PAHs than on their parent compounds. However, alkylated PAH information is necessary for tracing sources, and toxicological information does exist for alkylated naphthalenes and a few other alkylated PAHs (Paul Boehm, Arthur D. Little, Inc., Cambridge, Mass, personal communication to Roy Irwin).

Information on biological methods from the ATSDR (see ATSDR for information on embedded references) [881]:

BIOLOGICAL SAMPLES Several analytical techniques have been used to determine trace levels of PAHs in biological tissues and fluids including adipose tissue, lungs, liver, skin, hair, blood, urine, and feces. These include gas chromatography coupled with flame ionization detection (GC/FID), gas chromatography coupled with a mass spectrometry (GC/MS), high-performance liquid chromatography (HPLC) coupled with an ultraviolet (UV) or fluorescence detector, and thin-layer chromatography (TLC) with fluorescence detection.

Recently, Liao et al. (1988) developed a relatively simple and rapid procedure for purifying human and bovine adipose tissue extracts so that trace levels of complex mixture of target analytes (including PAHs) could be detected and quantified by capillary GC/MS. By employing an activated Florisil column, Liao and co-workers showed that lipid contaminants bind effectively (more than 99.75%) with Florisil, thereby producing a relatively clean sample extract. A detection limit

at a low ng/g level and an average sample recovery of 85% were achieved (Gay et al. 1980; Liao et al. 1988; Modica et al. 1982) [881]. For a table listing biological methods, see ATSDR [881].

Obana et al. (1981) reported the identification and quantification of six PAHs on EPA's priority pollutant list: anthracene, pyrene, benzo[b]fluoranthene, benzo[k]fluoranthene, benzo[a]pyrene, and benzo[g,h,i]perylene using the HPLC/fluorescence detector technique. Levels measured in human, tissue ranged from 0.006 to 0.460 ng/g. Following extraction of the PAHs from the sample matrices by saponification with KOH, the extract was cleaned on alumina and silica gel columns, prior to quantitation. The known carcinogens, benz[a]anthracene and dibenz[a,h]anthracene, were not detected (detection limit 0.005 ng/g). The HPLC/UV detection technique has also been used to simultaneously determine fluoranthene, benz[a]anthracene, and pyrene in blood and lung tissues (Brandys et al. 1989). A detection limit of ppb (ng/g or ng/mL), satisfactory recoveries (65-109%), and adequate precision (less than or equal to 19% relative standard deviation [RSD]) were achieved (Brandys et al. 1989).

In addition to direct measurement of PAHs in biological tissues, it is also possible to determine the concentration of metabolites in biological fluids. Pyrene is predominantly excreted as a 1-hydroxy-pyrene conjugate (glucoronate and sulfate), although 1,2-dihydroxy-1,2-dihydropyrene conjugates are also excreted in urine (Grimmer et al. 1993). Phenanthrene, on the other hand, is mainly excreted as dihydrodiol conjugates. The metabolites of phenanthrene that have been detected in human urine are 1-hydroxyphenanthrene, 2-hydroxyphenanthrene, 3-hydroxyphenanthrene, 4-hydroxyphenanthrene, 9-hydroxyphenanthrene, 1,2-dihydroxy-1,2-dihydrophenanthrene, 3,4-dihydroxy-3,4-dihydrophenanthrene, and 9, 10-dihydroxy-9,10-dihydrophenanthrene (Grimmer et al. 1993). There are apparently individual variations in the phenanthrol (hydroxyphenanthrene) and phenanthrene dihydrodiol conjugates excreted in the 24-hour urine samples (Grimmer et al. 1993). The major metabolite of benzo[a]pyrene in human tissue and body fluid is 7,8,9,10-tetrahydroxy-7,8,9,10- tetrahydrobenzo[a]pyrene (Weston et al. 1993a, 1993b).

Becher and Bjorseth (1983, 1985) and Becher (1986) developed an HPLC method for biological monitoring of PAHs and PAH metabolites in the urine of humans following occupational exposure to PAHs. Using the HPLC/fluorescence detector technique, recoveries of the individual PAH compounds varied between 10 and 85% with the more volatile 3-ring PAHs having the lowest recoveries. A detection limit of less than 1 ug of PAHs per mmol of creatinine was obtained. HPLC equipped with a fluorescence detector has also been used to measure 1-

pyrenol (1- hydroxypyrene, a pyrene metabolite) in urine of workers exposed to PAHs in coal tar pitch with a detection limit of 0.45 nmol/L (Tolos et al. 1990). Recovery and precision data were not reported. A strong correlation was observed between the concentrations of urinary 1-hydroxypyrene in workers and environmental PAHs, indicating that pyrene and may be used as a biomarker of exposure for assessing worker exposure to coal tar pitch containing pyrene (Tolos et al. 1990). Since 1-hydroxypyrene glucuronide is approximately 5 times more fluorescent than 1-hydroxypyrene, the former may be a more sensitive biomarker for PAH exposure (Strickland et al. 1994). A sensitive HPLC/synchronous fluorescence spectroscopic method is a available for the determination of 1- hydroxypyrene glucuronide (Strickland et al. 1994). Hecht et al. (1979) employed an HPLC analytical technique for determining the concentrations of benzo[a]pyrene and its metabolites in the feces of humans and rats following consumption of charcoal-broiled beef. A detection limit of 0.05 ug of benzo[a]pyrene metabolites per gram of sample was noted with HPLC/UV detection [881].

There is considerable evidence that PAHs are enzymatically converted to highly reactive metabolites that bind covalently to macromolecules such as DNA, thereby causing mutagenesis and carcinogenesis in experimental animals. Thus, benzo[a]pyrene, a prototype of the carcinogenic PAHs and the most thoroughly studied PAH, is activated by microsomal enzymes to 7b,8a-dihydroxy-(9a , 10a)-epoxy-7,8,9,10-tetrahydrobenzo[a]pyrene (BPDE) and binds covalently to DNA, resulting in formation of BPDE-DNA adducts (Harris et al. 1985; Haugen et al. 1986; Uziel et al. 1987). Sensitive methods are available to detect PAH-DNA adducts in the blood and tissues of humans and animals. These include immunoassays, i.e., enzyme-linked immunosorbent assays (ELISA), radioimmunoassay (RIA), dissociation-enhanced lanthanide fluoroimmunoassay (DELFIA), and ultrasensitive enzyme radioimmunoassay (USERIA); 32P- and 35S-postlabelling with radioactivity counting; surface-enhanced Raman spectroscopy; and synchronous luminescence spectroscopy (SLS) (Gorelick and Wogan 1989; Gorelick and Reeder 1993; Harris et al. 1985; Haugen et al. 1986; Helmenstine et al. 1993; Herikstad et al. 1993; Lau and Baird 1991; Perera et al. 1988; Phillips et al. 1987; Schoket et al. 1993) [881].

The ELISA technique is used for detection of antibodies in serum bound to BPDE-DNA adducts. The USERIA method involves measuring the immunological response of BPDE-DNA in the presence of rabbit anti-serum [881].

Abbreviations: FID = gas chromatography/flame ionization detector; GC/MS = gas chromatography/mass spectrometry; Gua = Guanosine; H 2 O = water; HCL = hydrogen chloride; HPLC = high performance liquid [881].

Tests related to human exposures: In the human body, PAHs are changed into chemicals that can attach to substances within the body [881]. The presence of PAHs attached to these substances can then be measured in body tissues or blood after exposure to PAHs [881]. PAHs or their metabolites can also be measured in urine, blood, or body tissues [881]. Although these tests can show that you have been exposed to PAHs, these tests cannot be used to predict whether any health effects will occur or to determine the extent or source of your exposure to the PAHs [881]. It is not known how effective or informative the tests are after exposure is discontinued [881]. These tests to identify PAHs or their products are not routinely available at a doctor's office because special equipment is required to detect these chemicals [881].

Information on environmental samples from ATSDR (see ATSDR for additional detail) [881]:

One of the difficulties associated with determination of PAHs in environmental samples is the complexity of PAH mixture in these samples [881]. Even after extensive and rigorous clean-up, the PAH fraction may contain hundreds of compounds [881]. Analytical methods that offer combinations of good chromatographic resolving power and detector selectivity are usually required to quantify selected compounds in such mixtures [881]. There is essentially a three-step procedure for the analysis and determination of PAHs in environmental samples: (1) extraction and isolation of PAHs from the sample matrix; (2) clean-up of the PAH mixtures from impurities and fractionation of PAH into subgroups; and (3) identification and quantitative determination of the individual components in each of these subgroups [881]. The collection of PAHs from air for quantification requires special considerations [881]. Some of the PAHs, especially those with lower molecular weights, exist primarily in the vapor phase while PAHs with higher molecular weights exist primarily in the particulate phase [881]. Therefore, a combination of a particulate filter (usually glass-fiber filter) and an adsorbent cartridge (usually XAD-2 or polyurethane foam) is used for the collection of PAHs [881]. Therefore, collection methods that use either a filtration system or an adsorbent alone may be incapable of collecting both particulate and vapor phase PAHs [881]. In addition, a few PAHs are known to be susceptible to oxidation by ozone and other oxidants present in the air during the collection process [881]. The commonly used methods for the extraction of PAHs from sample matrices are Soxhlet extraction, sonication, or partitioning with a suitable solvent or a solvent mixture [881]. Dichloromethane, cyclohexane, benzene, and methanol have been widely used as solvents [881]. Supercritical fluid extraction (SFE) of heterogeneous environmental samples with carbon dioxide in the presence of a modifier, such as 5-10% methanol or dichloromethane is preferable to the conventional extraction method because SFE is much less time consuming and has

comparable or better PAH extraction recovery than the conventional methods [881].

Abstracts and Misc. Publications: Other Information on PAH methods: See also:

MC Poirier, A Weston. 1966. Human DNA adduct measurements: State of the art. Environmental Health Perspectives 104: Suppl. 5: Page(s) 883-893.

ELC Lin, SM Cormier, JA Torsella. 1996. Fish biliary polycyclic aromatic hydrocarbon metabolites estimated by fixed-wavelength fluorescence: Comparison with HPLC-fluorescent detection
Source. Ecotoxicology and Environmental Safety 35: 1, (OCT 1996), Page(s) 16-23

G Talaska, P Underwood, A Maier, J Lewtas, N Rothman, M Jaeger. 1996. Polycyclic aromatic hydrocarbons (PAHs), nitro-PAHs and related environmental compounds: Biological markers of exposure and effects. Environmental Health Perspectives 104: Suppl. 5 (OCT 1996), Page(s): 901-906.

P Strickland, D Kang, P Sithisarankul. 1996. Polycyclic aromatic hydrocarbon metabolites in urine as biomarkers of exposure and effect. Environmental Health Perspectives 104: Suppl. 5 (OCT 1996), Page(s) 927-932.

JF Uthe, RK Misra, TL King, CJ Musial. 1996. Estimating analytical variances in measurement of polycyclic aromatic hydrocarbons and application to monitoring contaminants in American lobster (Homarus americanus). Journal of AOAC International 79: 3 (MAY-JUN 1996), Page(s) 797-802/

RM Burgess, RA Mckinney, WA Brown, JG Quinn. 1996. Isolation of marine sediment colloids and associated polychlorinated biphenyls: An evaluation of ultrafiltration and reverse-phase chromatography Source Environmental Science & Technology 30: 6 (JUN 1996), Page(s) 1923-1932.

MC Meckes, TJ Wagner, J Tillman. 1996. Solvent extraction of polynuclear aromatic hydrocarbons and polychlorinated biphenyl from river sediments Environmental Technology 17: 5 (MAY 1996), Page(s) 525-531.