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Ambient Water Quality Criteria for Fluoranthene

AMBIENT WATER QUALITY CRITERIA FOR

FLUORANTHENE

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FOREWORD

Section 304 (a)(1) of the Clean Water Act of 1977 (P.L. 95-217), requires the Administrator of the Environmental Protection Agency to publish criteria for water quality accurately reflecting the latest scientific knowledge on the kind and extent of all identifiable effects on health and welfare which may be expected from the presence of pollutants in any body of water, including ground water. Proposed water quality criteria for the 65 toxic pollutants listed under section 307 (a)(1) of the Clean Water Act were developed and a notice of their availability was published for public comment on March 15, 1979 (44 FR 15926), July 25, 1979 (44 FR 43660), and October 1, 1979 (44 FR 56628). This document is a revision of those proposed criteria based upon a consideration of comments received from other Federal Agencies, State agencies, special interest groups, and individual scientists. The criteria contained in this document replace any previously published EPA criteria for the 65 pollutants. This criterion document is also published in satisifaction of paragraph 11 of the Settlement Agreement in Natural Resources Defense Council, et. al. vs. Train, 8 ERC 2120 (D.D.C. 1976), modified, 12 ERC 1833 (D.D.C. 1979).

The term "water quality criteria" is used in two sections of the Clean Water Act, section 304 (a)(1) and section 303 (c)(2). The term has a different program impact in each section. In section 304, the term represents a non-regulatory, scientific assessment of ecological effects. The criteria presented in this publication are such scientific assessments. Such water quality criteria associated with specific stream uses when adopted as State water quality standards under section 303 become enforceable maximum acceptable levels of a pollutant in ambient waters. The water quality criteria adopted in the State water quality standards could have the same numerical limits as the criteria developed under section 304. However, in many situations States may want to adjust water quality criteria developed under section 304 to reflect local environmental conditions and human exposure patterns before incorporation into water quality standards. It is not until their adoption as part of the State water quality standards that the criteria become regulatory.

Guidelines to assist the States in the modification of criteria presented in this document, in the development of water quality standards, and in other water-related programs of this Agency, are being developed by EPA.

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FLUORANTHENE

CRITERIA

Aquatic Life

The available data for fluoranthene indicate that acute toxicity to freshwater aquatic life occurs at concentrations as low as 3,980 µg/l and would occur at lower concentrations among species that are more sensitive than those tested. No data are available concerning the chronic toxicity of fluoranthene to sensitive freshwater aquatic life.

The available data for fluoranthene indicate that acute and chronic toxicity to saltwater aquatic life occur at concentrations as low as 40 and 16 µg/l, respectively, and would occur at lower concentrations among species that are more sensitive than those tested.

Human Health

For the protection of human health from the toxic properties of fluoranthene ingested through water and contaminated aquatic organisms, the ambient water criterion is determined to be 42 µg/l.

For the protection of human health from the toxic properties of fluoranthene ingested through contaminated aquatic organisms alone, the ambient water criterion is determined to be 54 µg/l.

INTRODUCTION

Fluoranthene, a polynuclear aromatic hydrocarbon (PAH), is produced by the pyrolysis of organic raw materials such as coal and petroleum at high tempertures (Andelman and Snodgrass, 1974). It is also known to occur naturally as a product of plant biosynthesis (Borneff, et al. 1968).

Fluoranthene (1,2-benzacenaphthene or Idryl) has the molecular formula $C_{16}H_{10}$. It has a molecular weight of 202, a melting point of 111°C, a boiling point of approximately 375°C, and a vapor pressure of 0.01 mm Hg at 25°C. It is soluble in water to the extent of 265 µg/l (Davis, et al. 1942; Klevens, 1950).

Fluoranthene is ubiquitous in the environment and has been detected in air in the U.S. (Searle, 1976), in foreign and domestic drinking waters (Harrison, et al. 1975; Basu and Saxena, 1977, 1978; U.S. EPA, 1977), and in foodstuffs (Howard, et al. 1966a,b,c).

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Aquatic Life Toxicology*

INTRODUCTION

Bluegill, Daphnia magna, and the alga, Selenastrum capricornutum, have been studied using static test procedures and unmeasured concentrations. The range of LC_{50} and EC_{50} values is 3,980 to 325,000 µg/l and the bluegill is most sensitive.

In contrast to the relationship between freshwater fish and invertebrate species, the mysid shrimp and a polychaete are much more sensitive to fluoranthene than the sheepshead minnow. The numerical relationship between acute and chronic effect concentrations of fluoranthene on the mysid shrimp is small, with the acute-chronic ratio being 2.5.

EFFECTS

Acute Toxicity

Daphnia magna is more resistant than the bluegill (Table 1) with a 48-hour EC_{50} value of 325,000 µg/l; the 96-hour LC_{50} value for the bluegill is 3,980 µg/l (U.S. EPA, 1978).

The 96-hour LC_{50} values for the mysid shrimp and a polychaete are 40 and 500 µg/l, respectively (Table 1). The sheepshead minnow was exposed to concentrations of fluoranthene as high as 560,000 µg/l with no observed LC_{50} value (Table 4).

Chronic Toxicity

The chronic value for the mysid shrimp is 16 µg/l (Table 2) and when this concentration is divided by the acute value a ratio of 2.5 is obtained.

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*The reader is referred to the Guidelines for Deriving Water Quality Criteria for the Protection of Aquatic Life and Its Uses in order to better understand the following discussion and recommendation. The following tables contain the appropriate data that were found in the literature, and at the bottom of each table are calculations for deriving various measures of toxicity as described in the Guidelines.

Plant Effects

The freshwater alga, Selenastrum capricornutum, has been exposed to fluoranthene and the 96-hour EC_{50} values for cell numbers and chlorophyll a are 54,400 and 54,600 µg/l, respectively (Table 3).

The 96-hour EC_{50} values for chlorophyll a and cell numbers of the saltwater alga, Skeletonema costatum, are 45,000 and 45,600 µg/l, respectively.

Residues

No measured, steady-state bioconcentration factors are available for freshwater or saltwater organisms and fluoranthene.

Summary

The bluegill (96-hour LC_{50} = 3,980 µg/l) is much more sensitive to fluoranthene than the cladoceran, Daphnia magna (48-hour EC_{50} = 325,000 µg/l). No chronic data are available for freshwater organisms. The 96-hour EC_{50} values for the alga, Selenastrum capricornutum, were 54,400 and 54,600 µg/l.

The saltwater mysid shrimp and a polychaete were much more sensitive than the sheepshead minnow. The LC_{50} values for the invertebrate species were 40 and 500 µg/l; the 96-hour LC_{50} value for the sheepshead minnow was greater than 560,000 µg/l. The chronic value and acute-chronic ratio for the mysid shrimp were 16 µg/l and 2.5, respectively. The EC_{50} values for the saltwater alga were 45,000 and 45,600 µg/l.

CRITERIA

The available data for fluoranthene indicate that acute toxicity to freshwater aquatic life occurs at concentrations as low as 3,980 µg/l and would occur at lower concentrations among species that are more sensitive

than those tested. No data are available concerning the chronic toxicity of fluoranthene to sensitive freshwater aquatic life.

The available data for fluoranthene indicate that acute and chronic toxicity to saltwater aquatic life occur at concentrations as low as 40 and 16 µg/l, respectively, and would occur at lower concentrations among species that are more sensitive than those tested.

Table 1. Acute values for fluoranthene

Species	Method*	LC50/EC50 (µg/l)	Species Acute Value (µg/l)	Reference
		FRESHWATER SPECIES		
Cladoceran, *Daphnia magna*	S, U	325,000	325,000	U.S. EPA, 1978
Bluegill, *Lepomis macrochirus*	S, U	3,980	3,980	U.S. EPA, 1978
		SALTWATER SPECIES		
Polychaete (immature), *Neanthes arenaceodentata*	S, U	500	500	Rossi & Neff, 1978
Mysid shrimp (juvenile), *Mysidopsis bahia*	S, U	40	40	U.S. EPA, 1978

* S = static, U = unmeasured

No Final Acute Values are calculable since the minimum data base requirements are not met.

Table 2. Chronic values for fluoranthene (U.S. EPA, 1978)

Species	Method*	Limits (µg/l)	Chronic Value (µg/l)
	SALTWATER SPECIES		
Mysid shrimp, Mysidopsis bahia	LC	12-22	16

* LC = life cycle or partial life cycle

Acute-Chronic Ratio

Species	Chronic Value (µg/l)	Acute Value (µg/l)	Ratio
Mysid shrimp, Mysidopsis bahia	16	40	2.5

Table 3. Plant values for fluoranthene (U.S. EPA, 1978)

Species	Effect	Result (µg/l)
	FRESHWATER SPECIES	
Alga, *Selenastrum capricornutum*	EC50 96-hr cell numbers	54,400
Alga, *Selenastrum capricornutum*	EC50 96-hr chlorophyll *a*	54,600
	SALTWATER SPECIES	
Alga, *Skeletonema costatum*	EC50 96-hr chlorophyll *a*	45,000
Alga, *Skeletonema costatum*	EC50 96-hr cell numbers	45,600

Table 4. Other data for fluoranthene (U.S. EPA, 1978)

Species	Duration	Effect	Result (µg/l)
	SALTWATER SPECIES		
Sheepshead minnow (adult), Cyprinodon variegatus	96 hrs	LC50	>560,000

REFERENCES

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Mammalian Toxicology and Human Health Effects

EXPOSURE

Ingestion from Water

The sources of fluoranthene in aqueous environments are both natural and man-made. The occurrence of fluoranthene in water sediments, bacteria, algae, and plant materials in areas remote from industry and human habitation suggest natural origin. Suess (1970) has suggested that fluoranthene in surface waters arises from contamination of estuaries and coastal waters by shipping and harbor oil, industrial and municipal effluents, atmospheric fallout, precipitation, and road run-off.

The two most important properties influencing the concentrations of fluoranthene in water are its stability and solubility. Its relatively high molecular weight and relative nonpolarity make fluoranthene very insoluble in water. Although the solubility of fluoranthene in water at 25°C is only 265 µg/l (Klevens, 1950), its presence in environmental waters can be increased by detergents, solvents and by adsorption on the surface of solid matter, both biotic and abiotic.

Studies have shown that fluoranthene can be adsorbed and concentrated on a variety of particulate matter. Thus, the presence of particulate matter in suspended and settled form in natural waters can be sources of relatively high concentrations of fluoranthene. Analyzing sediment samples from Buzzards Bay, Mass., Giger and Blumer (1974) found the concentration of fluoranthene to be from 110 µg/kg to 790 µg/kg of dry sediment. Similarly, the analysis of recent sediments from a Swiss lake and river showed the

fluoranthene content as 0.42 µg/g and 0.39 µg/g of dry material, respectively (Giger and Schaffner, 1978). River particulate matter, on the other hand, was found to contain 5.7 µg/g of fluoranthene (Giger and Schaffner, 1978). The analysis of sediments from an English valley showed fluoranthene concentrations of 0.6 to 13.8 µg/g of dry sediments (John and Nickless, 1977). There is evidence of accumulation of fluoranthene in edible aquatic organisms. Thus, it is considered necessary to monitor fluoranthene levels not only in surface waters, but also in contaminated water since the use of these waters for irrigation also can spread fluoranthene into other foodstuffs (Shabad and Il'nitskii, 1970).

Industrial effluents from oil refineries, coke production, plastic and dyestuff industries, and industries using high temperature furnaces are some of the primary sources of man-made fluoranthene (Harrison, et al. 1975). The fluoranthene concentration in an industrial effluent was determined to be 2,198 ng/l (Borneff and Kunte, 1965). Except for pyrene, the amount of fluoranthene in this industrial effluent was much higher than that of all other individual polynuclear aromatic hydrocarbons (PAH) determined.

The fluoranthene concentration in municipal effluents was determined by Borneff and Kunte (1965). The value in domestic effluents was determined to be 273 to 352 ng/l. Although fluoranthene can be found in human urine and feces, the concentrations found in domestic sewage are unlikely to originate exclusively or primarily from this source (Harrison, et al. 1975). Other possible contributing sources include the washing of clothing, infiltration from soil, washout from the atmosphere and road run-off

(Harrison, et al. 1975). When the sewage contained a high percentage of industrial effluents, the fluoranthene level was found to be high and varied from 2,660 ng/l to 3,420 ng/l (Borneff and Kunte, 1965).

Road run-off can be an important factor in increasing the fluoranthene content of sewage. Fluoranthene in road run-off can arise in a number of ways. Bituminous road surfaces (Borneff and Kunte, 1965), car tire wear (Falk, et al. 1964), and vehicle exhausts (McKee and McMahon, 1967; Commins, 1969) contribute to the increased fluoranthene content in road run-off. Road run-off was primarily responsible for the increase of fluoranthene levels in sewage, from 352 ng/l on a dry day to 16,350 ng/l during a heavy rain (Borneff and Kunte, 1965). This finding of Borneff and Kunte has been confirmed by Acheson, et al. (1976) who found that highway run-off samples contained higher levels of fluoranthene (0.49 µg/l to 1.10 µg/l) than Thames River water (0.11 µg/l to 0.27 µg/l).

The removal of fluoranthene from water by conventional sewage treatment processes was investigated by Borneff and Kunte (1967). Removal of fluoranthene during primary sedimentation was found to be 62 to 66 percent (from an initial value of 3.23 - 43.5 µg/l to 1.22 - 14.6 µg/l) and the removal was 91 to 99 percent (final value of 0.28 - 0.26 µg/l) after biological purification with activated sludge processes.

The fluoranthene level in surface waters (lakes and rivers) was determined by a number of authors. Borneff and Kunte (1964, 1965) found the concentration of fluoranthene in German rivers to be in the range from 38.5 to 761 ng/l. Acheson, et al. (1976)

determined the value for Thames River water in England to be from 140 to 360 ng/l. Analyzing fourteen water samples from the Winnipesaukee, Oyster and Cocheco rivers in New Hampshire, Keegan (1971) detected fluoranthene in three samples, and the concentration ranged from 320 to 1,000 ng/l.

One surface water supply used for drinking water in England was analyzed for fluoranthene, and the concentration was found to be 150 ng/l (Harrison, et al. 1976). Surface waters in the U.S. were analyzed by Basu and Saxena (1977,1978) and Basu, et al. (1978). These investigators detected fluoranthene in four of the seven surface waters sampled. The average fluoranthene concentration in the positive samples was 325.7 ng/l with a range of 23.5 ng/l to 408.3 ng/l. These authors also analyzed three ground water samples and failed to detect any fluoranthene.

The fluoranthene levels in U.S. drinking waters were analyzed by Basu and Saxena (1977,1978) and Basu, et al. (1978). Of the 16 water supplies monitored, four showed positive fluoranthene levels. The concentrations of fluoranthene in the four positive samples were 2.4, 4.3, 8.9, and 94.5 ng/l, with an average of 27.5 ng/l. The analytical limit of detection for fluoranthene in these studies was 2.3 ng/l. The U.S. EPA also conducted the National Organic Monitoring Survey (U.S. EPA, 1977) to determine the frequency of occurrence of fluoranthene in drinking water supplies. Seventeen out of 110 samples analyzed showed the presence of fluoranthene (limit of detection = 10 ng/l). The mean fluoranthene concentration in positive samples in this study was 20.0 ng/l, with a range of concentrations varying from 10 ng/l to 80 ng/l. The values for

fluoranthene concentrations in various surface and drinking waters are shown in Table 1.

The removal of fluoranthene during drinking water treatment processes was studied by Harrison, et al. (1976). A fluoranthene concentration in river intake water of 150 ng/l was reduced to 140 ng/l when stored in a reservoir, reduced to 81 ng/l after filtration, and further reduced to 45 ng/l after chlorination. Thus, there was a 70 percent total reduction of fluoranthene concentration. The removal efficiency with a full water treatment process involving flocculation, activated carbon treatment, filtration, and chlorination was studied by Basu and Saxena (1977,1978) and Basu, et al. (1978). They found an 87.5 to 100 percent reduction in fluoranthene levels. The removal efficiency was 100 percent when two stages of activated carbon purification were used. These removal efficiencies are presented in Table 2.

There is no epidemiological evidence to prove that polynuclear aromatic hydrocarbons (PAH) in general, and fluoranthene, in particular, found in drinking water are related to the development of cancer (Andelman and Snodgrass, 1974). Also, Shabad and Il'nitskii (1970) stated that the amount of carcinogenic PAH consumed by man in water is typically only 0.1 percent of the amount consumed from food. Nevertheless, accumulation of PAH in edible aquatic organisms can greatly increase this amount (Andelman and Snodgrass, 1974). The use of contaminated water for irrigation also can spread PAH into other foodstuffs (Shabad and Il'nitskii, 1970). Therefore, in 1970, the World Health Organization (WHO, 1970) recommended that the concentration of six representative PAH (including

TABLE 1

Fluoranthene Concentrations in Various Water Samples

Water Source	Wastewater Containing: Domestic sewage (dry day)	Domestic sewage (heavy rain)	Domestic and industrial sewage	Surface water	Surface water used for drinking water	Ground Water	Drinking Water
Concentration (ng/l)	2 273-352	16350	3040 (2660-3420)[a]	320-1000 140-360 38.5-761	325.7 (23.5-408.3) 150	N.D.[b]	27.5 (2.4-94.5) 20.0 (10-80)
Reference	Borneff and Kunte, 1965	Borneff and Kunte, 1965	Borneff and Kunte, 1965	Keegan, 1971; Borneff and Kunte (1964, 1965); Acheson, et al. 1976	Basu and Saxena, 1977; Basu and Saxena, 1978; Basu, et al. 1978	Basu and Saxena, 1978	Basu and Saxena, 1977; Basu and Saxena, 1978; Basu, et al. 1978; U.S. EPA, 1977

[a] Values in parentheses are concentration ranges

[b] N.D.: not detected

TABLE 2

Fluoranthene Removal Efficiencies as a Result of Water Treatment[a]

Water Source	Initial conc., ng/l	Final conc., ng/l	% Reduction
Pittsburgh, Pa.	408.3	N.D.[b]	100
Huntington, Va.	23.5	2.4	89.7
Philadelphia, Pa.	114.3	8.9	92.2
Wheeling, W. Va.	756.5	94.5	87.5

[a]Sources: Basu and Saxena, 1977, 1978; Basu, et al. 1978.

[b]N.D.: not detected.

fluoranthene) in drinking water not exceed 0.2 μg/l. It further recommended that there should be at least one center in each country capable of carrying out investigations of PAH in drinking water. However, from the data given for fluoranthene (Table 1) and the other PAH data available in the references provided, the PAH level in U.S. drinking waters is well below the WHO recommended level.

Ingestion from Food

PAH formed through both natural and man-made sources can enter the food chain in a variety of ways. The absorption of PAH from the soil by various plant roots and translocation to the shoots is well documented (Lo and Sandi, 1978). Some plant waxes act as collectors of PAH present in polluted air (Hetteche, 1971). It has been shown that 10 percent of benzo(a)pyrene (BaP) in lettuce, kale, spinach, leeks, and tomatoes can be removed by cold water washing, an indication that it was originally deposited externally (Lo and Sandi, 1978). Oysters and clams collected from moderately polluted waters also concentrate PAH (Cahnmann and Kuratsune, 1957; Guerrero, et al. 1976). Food additives and packages as well as dairy waxes containing PAH increase PAH levels in processed foods. Hexane, a commercial solvent used to extract edible vegetable oils, is also a source of PAH contamination. PAH present in food-grade carbon blacks used for food processing can be transported to the food products. Curing smoke and other pyrolysis products used during food cooking add to the level of PAH in food.

It has been demonstrated by Zitko (1975) that PAH are not bioaccumulated along the food chain. However, Bjørseth (1978)

concluded that both common and horse mussels bioaccumulated PAH, although not to the same degree. Dunn and Stich (1976) have shown that mussels cannot metabolize BaP upon their removal from water. In water, mussels released 79 percent of naphthalene in three days, with a half-life of 1.3 days. The BaP release from both clams and mussels in water took place with a half-life of 2 to 5 weeks.

The fluoranthene levels in various foods are discussed individually below.

Various European workers have reported the presence of PAH in fruits and related products (International Agency for Research on Cancer (IARC), 1973). However, fluoranthene concentrations were not reported in this IARC study. No study from North America concerning PAH levels in fruits and related products was reported. Kuratsune and Hueper (1958, 1960) published PAH levels in coffee soot and roasted coffee. The coffee soot was found to contain 340 to 1,000 ppb fluoranthene. The moderately dark and fully roasted (darkest) coffees contained 1 to 7 ppb and 0 to 15 ppb fluoranthene, respectively. Grimmer and Hildebrandt (1967) determined the fluoranthene content in coconut and reported values of 0.3 ppb, 3.9 ppb and 92.7 ppb for fresh, sun-dried, and smoke-dried coconut, respectively.

Fluoranthene was also qualitatively detected from germinated rye, wheat, and lentil seedlings, although none was detected in the ungerminated products (Graf and Nowak, 1966). These authors also demonstrated the uptake of fluoranthene in radishes from polluted environments. According to Borneff (1977), the main human intake of PAH comes from fruits, vegetables, and bread. He estimated that

the total PAH intake from all of these sources amounted to 3-4 mg/person/year.

The fluoranthene levels found in these products are shown in Table 3. The relatively high levels of fluoranthene found indicate that the heating of such oils might have led to a slight increase of fluoranthene concentration (Lo and Sandi, 1978). In a total diet study, Howard, et al. (1968) found only trace amounts (less than 0.5 ppb) of seven PAH (fluoranthene not studied) in the composite sample containing the fats and oils. However, Borneff (1977) estimated that the yearly human intake of PAH from these sources was 0.1 mg.

Raw meat does not normally contain fluoranthene, but smoked or cooked meat may contain varying amounts of fluoranthene. The pyrolysis of fats, and incomplete combustion of the fuel contribute to the fluoranthene content in meats. Casing around the meat changes PAH levels in cooked meats. Cellulose casing is more effective as a barrier to the passage of PAH than is gut casing (Simon, et al. 1969). Further investigations have shown that the amount of PAH in broiled meats is directly proportional to the temperature of the treatment (Lijinsky and Ross, 1967). The dependency of fluoranthene content in meat and meat products on all the above factors is summarized in Tables 4 and 5.

Fish from unpolluted waters usually do not contain detectable amounts of PAH. Smoked and cooked food, however, contain varying levels of fluoranthene. The amount of fluoranthene depends on the method of cooking, that is, the nature of the heat source, the temperature of combustion, and the degree of smoking in the case of

TABLE 3

Fluoranthene Levels in Vegetable Fats, Oils and Shortenings

Product	Concentration, ppb	Reference
Linseed oil (unrefined)	15.1	Grimmer & Hildebrandt, 1967
Cocoa butter oil (unrefined)	20.5	Grimmer & Hildebrandt, 1967
Coconut oil (smoke-dried)	372.0	Grimmer & Hildebrandt, 1967
Coconut oil (hot-air dried)	255.0	Biernoth & Rost, 1967
Coconut oil (commercial)	445.0	Biernoth & Rost, 1967
Coconut oil (dried copra, treated with slaked lime)	18.0	Biernoth & Rost, 1967
Cotton seed oil (unrefined)	7.0	Grimmer & Hildebrandt, 1967
Ground nut oil (unrefined)	8.4	Grimmer & Hildebrandt, 1967
Palm oil (unrefined)	7.1	Grimmer & Hildebrandt, 1967
Palm-kernel oil (unrefined)	39.0	Grimmer & Hildebrandt, 1967
Pumpkin seed oil	25.0	Biernoth & Rost, 1967
Rapeseed oil (unrefined)	10.9	Grimmer & Hildebrandt, 1967
Soybean oil (unrefined)	6.4	Grimmer & Hildebrandt, 1967
Soybean oil	1.3[a] (0.6-2.6)	Howard, et al. 1966c
Sunflower oil	21.1	Grimmer & Hildebrandt, 1967
Wesson oil	N.D.[b]	Lijinsky & Ross, 1967
n-paraffin oil (acid-washed for yeast fermentation)	1.9 (1.0-3.0)	McGinnis, 1975
n-paraffin oil (silica-gel treated for yeast fermentation)	3.8 (0.5-7.3)	McGinnis, 1975
Olive oil	3.2 (2.2-4.4)	Howard, et al. 1966c
Peanut oil	3.3	Howard, et al. 1966c

[a]Values in parenthesis are ranges in concentrations

[b]N.D.: not detected.

TABLE 4

Fluoranthene Levels in Meat and Meat Products
Under a Variety of Conditions

Product	Concentration, ppb	Reference
Charcoal broiled steak (lab. preparation)	20.0	Lijinsky & Shubik, 1965b
Charcoal broiled ribs (commercial)	49.0	Lijinsky & Shubik, 1965b
Charcoal broiled steak (commercial)	43.0	Lijinsky & Shubik, 1965b
Liquid smoke	10.0-16.0	Lijinsky & Shubik, 1965a,b
Smoked ham	14.0	Howard, et al. 1966a;
	0.6-2.9	Malanoski, et al. 1968;
	4-156	Lo & Sandi, 1978
Smoked bacon	8.0	Lijinsky & Shubik, 1965b
	35.0	Lo & Sandi, 1978
Smoked chipped beef	0.6	Howard, et al. 1966a
Smoked frankfurters	6.4	Howard, et al. 1966a
Smoked mutton (lab.)	4.6	Bailey & Dungal, 1958
Smoked mutton (commercial)	18.0	Thorsteinsson, 1969
Smoked pork roll	3.1	Howard, et al. 1966a
Smoked barbecued beef	2.0	Malanoski, et al. 1968
Smoked mutton sausage (commercial)	6.0	Thorsteinsson, 1969
Home-smoked mutton (close to stove & with cover)	35.0	Thorsteinsson, 1969
Home-smoked mutton (close to stove & without cover)	303.0	Thorsteinsson, 1969
Home-smoked mutton (distant from stove & with cover)	47.0	Thorsteinsson, 1969
Home-smoked lamb	158.0	Thorsteinsson, 1969
Cold-smoked sausage (with casing)	40.0	Lo & Sandi, 1978
Cold-smoked sausage (without casing)	7.2	Lo & Sandi, 1978
Hot-smoked sausage (with casing)	35.2	Lo & Sandi, 1978
Hot-smoked sausage (without casing)	13.0	Lo & Sandi, 1978
Hot-smoked salami (without casing)	5.6	Lo & Sandi, 1978
Hot-smoked mortadella without casing)	22.0	Lo & Sandi, 1978

TABLE 5

Effect of Fat Content and Temperature of Cooking on Fluoranthene Levels in Cooked Meats*

Product and Cooking Method	Concentration, ppb
Charcoal broiled:	
Hamburger, fat (hot)[a]	13.3
Hamburger, fat (cold)[a]	6.4
Hamburger, lean (hot)	0.3
Hamburger, lean (cold)	1.3
Hamburger (no drip pan)	0.2
Hamburger frozen (hot)	4.9
Pork Chop (hot)	22.5
Chicken (hot)	1.1
Sirloin steak (hot)	12.6
T-Bone steak (hot)	19.8
Flame broiled:	
T-Bone steak (hot)	19.0

*Source: Lijinsky and Ross, 1967

[a]cold: 25 cm from heat source; hot: 7 cm from heat source; fat: 21% fat; lean: 7% fat

smoked fish. In a study sponsored by the U.S. Food and Drug Administration, PAH levels in unsmoked and smoked fish were compared (Howard, et al. 1966a,b). In addition to fish, various other marine foods were investigated and found to contain fluoranthene (Table 6). According to a recent estimate by Borneff (1977), the total human intake of PAH from smoked meat, smoked fish and drinking water sources amounts to 0.05 mg/person/year. The fluoranthene levels detected in a variety of dairy and bakery products are listed in Table 7.

A bioconcentration factor (BCF) relates the concentration of a chemical in aquatic animals to the concentration in the water in which they live. The steady-state BCFs for a lipid-soluble compound in the tissues of various aquatic animals seem to be proportional to the percent lipid in the tissue. Thus the per capita ingestion of a lipid-soluble chemical can be estimated from the per capita consumption of fish and shellfish, the weighted average percent lipids of consumed fish and shellfish, and a steady-state BCF for the chemical.

Data from a recent survey on fish and shellfish consumption in the United States were analyzed by SRI International (U.S. EPA, 1980). These data were used to estimate that the per capita consumption of freshwater and estuarine fish and shellfish in the United States is 6.5 g/day (Stephan, 1980). In addition, these data were used with data on the fat content of the edible portion of the same species to estimate that the weighted average percent

TABLE 6

Fluoranthene Levels in Fishes and Other Sea Foods

Product	Concentration, ppb	Reference
Unsmoked haddock	1.6	Howard, et al. 1966a
Unsmoked herring (salted)	0.8	Howard, et al. 1966a
Unsmoked salmon (canned)	1.8	Howard, et al. 1966a
Smoked Cod	0.5	Dungal, 1961
Smoked haddock	1.1	Lijinsky & Shubik, 1965a
Smoked herring	3.0	Howard, et al. 1966a
Smoked herring (dried)	1.8	Howard, et al. 1966a
Smoked Red fish	4.0	Dungal, 1961
Smoked salmon	6.0	Lijinsky & Shubik, 1965b
	3.2	Howard, et al. 1966a
Smoked sturgeon	2.4	Howard, et al. 1966a
Smoked trout	N.D.	Howard, et al. 1966a
	12.0	Thorsteinsson, 1969
Smoked white fish	4.6	Baily & Dungal, 1958
Smoked eel	4.0	Thorsteinsson, 1969
Smoked lump fish	2.0	Thorsteinsson, 1969
Horse mackerel (gas broiled)	3.6-7.0	Lo & Sandi, 1978
Horse mackerel (electric broiled)	0.2-5.2	Lo & Sandi; 1978
Kale	82-6760	Hetteche, 1971
Algae, Chlorella vulgaris	650	Borneff, et al. 1968
Algae, Scenedesmus acutus	44	Payer, et al. 1975

N.D.: not detected

TABLE 7

Fluoranthene Concentrations Determined in Yeast and Cheese

Product	Baking Yeast				Brewing Yeast	Smoked Gouda cheese	Cheddar cheese
	French	German	Scottish	Russian			
Concentration, ppb	18.5-21.2	17.2-66.8	93.5	32.1	8.6	2.8	0.8
Reference			Grimmer, 1974		Grimmer, 1974	Howard, et al. 1966a	Howard, et al. 1966a

lipids for consumed freshwater and estuarine fish and shellfish is 3.0 percent.

No measured steady-state bioconcentration factor (BCF) is available for fluoranthene, but the equation "Log BCF = (0.85 Log P) - 0.70" can be used (Veith, et al. 1979) to estimate the BCF for aquatic organisms that contain about 7.6 percent lipids (Veith, 1980) from the octanol/water partition coefficient (P). Since no measured log P value could be found, a log P value of 4.90 was calculated for fluoranthene using the method described in Hansch and Leo (1979). Thus, the steady-state bioconcentration factor is estimated to be 2,900. An adjustment factor of 3.0/7.6 = 0.395 can be used to adjust the estimated BCF from the 7.6 percent lipids on which the equation is based to the 3.0 percent lipids that is the weighted average for consumed fish and shellfish. Thus, the weighted average bioconcentration factor for fluoranthene and the edible portion of all freshwater and estuarine aquatic organisms consumed by Americans is calculated to be 2,900 x 0.395 = 1,150.

Inhalation

A variety of PAH, including fluoranthene, have been detected in ambient air. Because of its carcinogenic properties, BaP has been most extensively monitored and has frequently been used as an indicator of ambient PAH. However, the relative amount of individual PAH in ambient air is dependent on the location. This has been demonstrated by Stocks, et al. (1961) by studying ambient rural, suburban, and urban air in England. The exact amount of fluoranthene intake by inhalation is difficult to determine because of the different sources of exposure, such as, tobacco smoke inhalation,

occupational exposure, and exposure to ambient air. The fluoranthene exposure due only to inhalation of ambient air will be discussed in this section.

Concentrations of fluoranthene are different in various cities and at different times of the year. The concentrations are usually highest during the winter months, probably from heating sources (Sawicki, 1962). However, there may be some exceptions to this high winter - low summer concentration pattern. It has been suggested that in areas with significant industrial emissions of PAH the fluoranthene level may remain uniform throughout the year (U.S. EPA, 1974). In other areas such as Los Angeles, which do not require heating during winter, automobile and industrial emissions control the PAH pattern in the ambient air (Gordon, 1976). The fluoranthene concentration in Los Angeles ambient air during four quarterly periods of 1974-1975, May-July, Aug.-Oct., Nov.-Jan., and Feb.-Apr. were 0.38 ppb, 0.15 ppb, 0.24 ppb, and 0.68 ppb, respectively (Gordon, 1976).

The declining trend of fluoranthene concentration in U.S. ambient air from the 1960's to 1970's may be from the decreased use of coal for power generation. Also contributing to this decline is the improved disposal of solid wastes and restrictions on open burning (U.S. EPA, 1974).

The possibility of long distance transport of PAH which might result in PAH contamination in areas downwind from large emission sources has been studied by Lunde and Bjørseth (1977). They determined that samples with trajectories from Western Europe contained about 20 times more fluoranthene than samples with trajectories

from northern Norway. This proves that some PAH, including fluoranthene, are stable enough to be transported from distant industrial sources to the suburban and rural areas.

Fluoranthene levels determined in various locations and at different times are presented in Table 8.

Various factors, particularly smoking, can alter the concentration of fluoranthene in indoor environments. Under standard smoking conditions the smoke of a cigarette generated between puffs (sidestream-smoke) contains 1,255 ng of fluoranthene per cigarette compared to the smoke which is inhaled (mainstream-smoke) which contains 272 ng of fluoranthene per cigarette (Grimmer, et al. 1977). In a 36 m^3 room with ventilation equal to a single air change per hour, the smoke from 5 cigarettes per hour from 2 smokers produced an average level of 99 ng/m^3 of fluoranthene in air samples collected over a period of 8 hours from an average of two tests (Grimmer, et al. 1977).

Dermal

No direct information is available on the importance of dermal absorption in total human exposure to fluoranthene. Fluoranthene can be absorbed through the skin by animals (see Absorption). For those humans exposed to only background levels of fluoranthene, dermal absorption is not likely to be a significant route of entry.

PHARMACOKINETICS

There are no data available concerning the pharmacokinetics of fluoranthene in humans. Moreover, animal studies have not been conducted for the specific purpose of supplying pharmacokinetic data on fluoranthene. Nevertheless, it is possible to make limited

TABLE 8

Ambient Fluoranthene Levels at Different Locations

Location	Concentration, μg/1,000 m^3	Reference
Average U.S. Urban air, 1963[a]	4.0	Hoffman & Wynder, 1968
Birmingham, AL, 1964-65	5.5	U.S. EPA, 1975
Detroit, MI, 1965	0.19-15.0	Hoffman & Wynder, 1968
Los Angeles, CA, 1973	0.1-3.4	Hoffman & Wynder, 1977
College Park, MD, 1976	4.1	Fox & Staley, 1976
Baltimore Harbor Tunnel, MD, 1976	93.0	Fox & Staley, 1976
Los Angeles, CA,1976	0.31	Gordon, 1976
Providence, RI, 1977	0.16-1.5	Krstulovic, et al. 1977
England, Urban, 1961		
Summer	6.5	Stocks, et al. 1961
Winter	44.9	
England, Bus Depot, 1961		
Summer	5.0	Stocks, et al. 1961
Winter	40.0	
England, Bus Garage, 1961		
Summer	5.0	
Winter	83.0	Stocks, et al. 1961
England Tunnel, 1961		
Summer	24.0	Stocks, et al. 1961
Winter	54.5	
England Suburban, 1961		
Summer	4.6	Stocks, et al. 1961
Winter	26.6	
England, Rural, 1961		
Summer	4.5	Stocks, et al. 1961
Winter	10.5	
Rome, 1966	2.1-4.5	Hoffman & Wynder, 1977
Rome, 1972	1.0-18.0	Hoffman & Wynder, 1977
Budapest, 1975	10.4	Kertesz-Saringer & Morlin, 1975
Sidney, 1965	0.06-2.6	Hoffman & Wynder, 1977
Ontario, 1966	0.3-10.6	Hoffman & Wynder, 1977
Ontario, 1962 inversion period	0.6-41.0	Hoffman & Wynder, 1977
Norway, 1977	0.17-0.32	Lunde & Bjørseth, 1977
Switzerland, 1978	12.9	Giger & Schaffner, 1978
Ohmuta, Japan, 1978	5.75	Tokiwa, et al. 1977

[a]Values from a composite sample of downtown areas in approximately 100 cities.

assumptions based on the results of animal studies conducted with other PAH that are structurally similar to fluoranthene.

Absorption

The demonstrated toxicity of fluoranthene by oral and dermal administration indicates that it can pass across epithelial membranes (Smyth, et al. 1962). The high lipid solubility of fluoranthene supports this observation. Animal studies with structurally related PAH, such as benzo(a)pyrene, chrysene, 7,12-dimethylbenz-(a)anthracene, benz(a)anthracene, and 3-methylcholanthrene, confirmed that intestinal transport readily occurs, primarily by passive diffusion (Rees, et al. 1971). In addition, there is ample evidence to indicate that benzo(a)pyrene (and presumably other PAH) is easily absorbed through the lungs (Kotin, et al. 1959; Vainio, et al. 1976).

Distribution

The tissue distribution and accumulation of fluoranthene has not been studied. It is known, however, that other PAH (e.g., benzo(a)pyrene, 7,12-dimethylbenz(a)anthracene, 3-methylcholanthrene, phenanthrene) were found in a wide variety of body tissues following their absorption in experimental rodents (Kotin, et al. 1959; Bock and Dao, 1961; Flesher, 1967). Relative to other tissues, PAH such as fluoranthene can be expected to localize primarily in body fat and fatty tissues.

The potential for transplacental passage of fluoranthene cannot be predicted. With other PAH, passage into the fetus following intragastric or intravenous administration to pregnant rats has been variable (Shendrikova and Aleksandrov, 1974).

Metabolism

Experimental studies have not been conducted on the metabolism of fluoranthene. However, it is well established that the metabolism of PAH is accomplished by the microsomal enzyme complex of mixed-function oxidases, often termed aryl hydrocarbon hydroxylase. This enzyme system has been studied extensively and is the subject of numerous reviews (Conney, 1967; Gelboin, 1967; Marquardt, 1977). These microsomal oxidases, while most abundant in the liver, have been found in most mammalian tissues. This enzyme complex is responsible for the metabolic detoxification of PAH, but also activates PAH to toxic and carcinogenic metabolites.

As a group, PAH are metabolized to substances that have been arbitrarily divided into two groups on the basis of solubility. In one group are metabolites that can be extracted from an aqueous incubation mixture by an organic solvent. This group consists of ring-hydroxylated products such as phenols and dihydrodiols. Numerous studies indicate that epoxide intermediates are involved in the formation of phenolic metabolites for the expression of toxic and carcinogenic effects (Sims and Grover, 1974; Sims, 1976; Jerina and Daly, 1974; Jerina, et al. 1977).

In the second group of metabolites are water-soluble products that remain after extraction with an organic solvent. It is generally agreed that most of these PAH derivatives are formed by conjugation of the hydroxylated products with glutathione, glucuronic acid, or sulfate. This process would render the derivatives more hydrophilic and presumably less toxic.

It is reasonable to assume that fluoranthene is metabolized in a manner which is consistent with the general biochemical scheme for biotransformation of PAH. However, the exact chemical structure of fluoranthene metabolites or their chemical and biological reactivity is not presently known.

Excretion

There is no direct information available concerning the excretion of fluoranthene in experimental animals or man. Limited inferences can be drawn from animal studies with related PAH, however.

In 1936 it was recognized that various PAH were excreted primarily through the hepatobiliary system and the feces (Peacock, 1936; Chalmers and Kirby, 1940). However, the rate of disappearance of various PAH from the body and the principal routes of excretion are influenced both by structure of the parent compound and the route of administration (Heidelberger and Weiss, 1951; Aitio, 1974). Moreover, it has been shown that the rate of disappearance of benzo(a)pyrene from body tissues can be markedly stimulated by prior treatment with inducers of microsomal enzymes (e.g., benzo(a)pyrene, 7,12-dimethylbenz(a)anthracene, 3-methylcholanthrene, chrysene) (Schlede, et al. 1970a,b; Welch, et al. 1972). From the available evidence concerning excretion of PAH in animals, it is apparent that extensive bioaccumulation is not likely to occur.

EFFECTS

Acute, Subacute, and Chronic Toxicity

Smyth and coworkers (1962) determined the acute toxicity of fluoranthene following oral and inhalation exposures in rats and dermal administration to rabbits. The acute oral LD_{50} for fluoranthene, determined using groups of five male Carworth-Wistar rats, was 2.00 g/kg (range from 1.27 to 3.13 g/kg). The dermal LD_{50} in rabbits resulting from 24-hour contact with fluoranthene was 3.18 g/kg (range from 2.35 to 4.29 g/kg). Exposure of six male or female albino rats to concentrated vapors of fluoranthene for eight hours produced no mortality. Taken together, these results from animal studies indicate that fluoranthene has a relatively low acute toxicity. Where deaths occurred, no information was reported concerning target organs or specific cause of death.

In earlier studies, Haddow and coworkers (1937) examined the effect of various PAHs, including fluoranthene, on body growth in hooded rats of the Lister strain. A single intraperitoneal injection of 30 mg fluoranthene dissolved in sesame oil had no adverse effect on body weight gain over a 24-day observation period. By comparison, certain carcinogenic PAH (10 mg of benzo(a)pyrene or dibenz(a,h)anthracene) caused an initial weight reduction followed by resumption of growth at a reduced rate.

Only limited data are available concerning the toxic effects of fluoranthene produced by repeated administration. These are limited to reports of mortality produced in mice by repeated dermal application or subcutaneous injection. Pertinent data from these studies are summarized in Table 9.

TABLE 9

Toxicity of Fluoranthene by Repeated Administration to Mice

Species	No.	Sex	Preparation and Dose	Route of Administration	Effect	Reference
Mouse (Strain A)	14	M&F	10 mg crystalline fluoranthene in glycerol, repeated 4 times	subcutaneous injection in the left flank	6 mice survived for 18 months; experiment terminated at 19 months	Shear, 1938
Mouse	10	?	0.3 % solution of fluoranthene in benzene applied twice weekly	dermal application to interscapular region	3 of 10 alive after 6 months; 3 of 10 alive after 1 year; last mouse died after 501 days	Barry, et al. 1935
Mouse	10	?	0.3 % solution of fluoranthene in benzene applied twice weekly	dermal application to interscapular region	4 of 10 alive after 6 months; 1 of 10 alive after 1 year; last mouse died after 379 days	Barry, et al. 1935
Mouse (Ha/ICR/Mil Swiss Albino)	20	F	50 µl of 1.0% fluoranthene solution in acetone applied 3 times weekly for 12 months	dermal application	No mortality after 15 months	Hoffman, et al. 1972

Synergism and/or Antagonism

Because fluoranthene is normally encountered in the environment as part of a complex mixture of PAH, concern has often been expressed over its interactive toxic effects. In this regard, Pfeiffer (1973,1977) tested ten noncarcinogenic PAH found in automobile exhaust in combination with benzo(a)pyrene (3-100 μg) and dibenz(a,h)anthracene (2-75 μg) by subcutaneous injection in groups of female NMRI mice. The ten noncarcinogens tested were: benzo(e)pyrene (2-70 μg); benz(a)anthracene (3-100 μg); phenanthrene (125-4,000 μg); anthracene (31-1,000 μg); pyrene (62-2,100 μg); chrysene (3-100 μg); perylene (0.2-7.0 μg); benzo(g,h,i)perylene (12.8 - 410 μg); coronene (3-100 μg); and fluoranthene (28-900 μg). The tumor incidence resulting from all 12 compounds being administered together could be attributed to the presence of dibenz(a,h)anthracene, with little influence from benzo(a)pyrene or the other ten chemicals. No inhibitory effect of the ten noncarcinogens was evident; moreover, an increased tumor yield resulted from injection of mixtures containing increasing amounts of the components. This effect, however, was less dramatic than if benzo(a)pyrene were administered alone, and paralleled the dose-response curve of dibenz(a,h)anthracene acting alone.

Similar experiments were conducted by Schmahl and coworkers (1977) involving the dermal application of mixtures containing carcinogenic and noncarcinogenic PAH to mice. They concluded that the tumorigenic response obtained with PAH mixtures that included fluoranthene could be attributed almost entirely to the presence of

the carcinogenic PAH (benzo(a)pyrene, dibenz(a,h)anthracene, benz-(a)anthracene, benzo(b)fluoranthene) in the mixture.

There is evidence that fluoranthene may considerably enhance the tumorigenic response produced by benzo(a)pyrene. These studies are discussed in the Carcinogenicity section of this report.

Teratogenicity

There is no information available concerning the possible teratogenic effects of fluoranthene in animals or man. Furthermore, only limited data are available regarding the teratogenic effects of other PAH in experimental animals.

Benzo(a)pyrene had little effect on fertility or embryonic development in several mammalian and nonmammalian species (Rigdon and Rennels, 1964; Rigdon and Neal, 1965). On the other hand, 7,12-dimethylbenz(a)anthracene and its hydroxymethyl derivatives apparently possess considerable teratogenic potency in the rat (Currie, et al. 1970; Bird, et al. 1970).

Mutagenicity

The concept that carcinogenesis is an expression of an alteration in the genetic material of a cell (i.e., somatic mutation) implies that a formal relationship exists between mutagenesis and carcinogenesis (Nery, 1976; Miller, 1978). The results obtained with several *in vitro* mutagenesis test systems, particularly the Ames *Salmonella typhimurium* assay, support the belief that most carcinogenic chemicals are mutagenic as well. For PAH, the Ames assay has been very effective in detecting parent structures and their biotransformation products that possess carcinogenic activity (McCann, et al. 1975; Teranishi, et al. 1975; McCann and Ames,

1976; Sugimura, et al. 1976; Wislocki, et al. 1976; Wood, et al. 1976).

Tokiwa and coworkers (1977) employed the Ames assay to search for mutagenic activity in a series of PAH, including fluoranthene, which were detected in the particulate fraction of urban air pollutants. Salmonella strain TA 98 in the presence of rat liver S-9 fraction (to provide bioactivation) was employed. Under these test conditions, fluoranthene displayed no mutagenic activity.

In a comparative study of the mutagenic activity, tumor initiating activity, and complete carcinogenicity of several PAH, fluoranthene was also found to be inactive towards both tester strains TA 98 and TA 100 in the presence of Araclor 1254-induced rat liver homogenate (LaVoie, et al. 1978).

No reports are available regarding the potential mutagenicity of fluoranthene in other test systems, either in vitro or in vivo.

Carcinogenicity

Among the unsubstituted polycyclic hydrocarbons containing a nonaromatic ring, a number of active carcinogens are known. The most prominent examples of this type of compound are: cholanthrene; 11,12-ace-benz(a)anthracene; 6,7-ace-benz(a)anthracene; 8,9-cyclopentabenz(a)anthracene; acenaphthanthracene; 1,2,5,6-tetrahydrobenzo(j)cyclopenta(f,g)aceanthrylene, and "angular" stearanthrene (Arcos and Argus, 1974). In addition, alkyl substitution of partially and fully aromatic condensed ring systems may also add considerable carcinogenic activity. The best examples of this type of activation are 3-methylcholanthrene, a highly potent carcinogen, 2-methylfluoranthene, and 5-methylchrysene.

Fluoranthene was first tested for carcinogenic activity more than four decades ago (Barry, et al. 1935). The results from that investigation, and from several studies conducted since that time, indicate that fluoranthene has virtually no activity as a complete carcinogen. The conditions employed and results obtained in these studies are summarized in Table 10. Both dermal application and subcutaneous injection in mice have been employed for the bioassay of fluoranthene.

Despite the fact that fluoranthene shows no activity as a complete carcinogen in the mouse, a number of fluoranthene derivatives are active carcinogens. These include 2-methylfluoranthene (Hoffmann, et al. 1972) and several benzofluoranthenes and dibenzofluoranthenes (IARC, 1973; Arcos and Argus, 1974).

Investigations in which polycyclic carcinogens were applied to the skin of mice have shown the two-stage theory of skin carcinogenesis (Van Duuren, 1976). The first stage, initiation, results from the ability of a carcinogen to effect a permanent change within a cell or cell population following a single application. The measure of carcinogenic potency is often regarded as the capacity for tumor initiation. However, some weak or inactive complete carcinogens can be active as tumor initiators (e.g., dibenz(a,c)anthracene, 1-methylchrysene, benz(a)anthracene). The second stage, promotion, is a prolonged process which does not necessarily require the presence of a carcinogen, but nevertheless a chemical stimulus must be supplied (e.g., by croton oil). A complete carcinogen is one that is capable of producing tumors when applied alone in sufficient quantity.

TABLE 10

Activity of Fluoranthene as a Complete Carcinogen in Mice

Species	No.	Sex	Preparation and Dose	Route of Administration	Results	Reference
Mouse	10	?	0.3% solution of fluoranthene in benzene, applied twice weekly	dermal application to interscapular region	70% mortality after 6 months; no tumors by 1 year	Barry, et al. 1935
Mouse	10	?	0.3% solution of fluoranthene in benzene, applied twice weekly	dermal application to interscapular region	60% mortality after 6 months; no tumors by 1 year	Barry, et al. 1935
Mouse (Strain A)	14	M&F	10 mg crystalline fluoranthene in glycerol, repeated 4 times	subcutaneous injection in the left flank	6 mice survived for 18 months; no tumors by 19 months	Shear, 1938
Mouse (CAF, Jackson)	25-50	M&F	10% solution of fluoranthene in acetone 3 times weekly	dermal application to the back	No papillomas or carcinomas found by 13 months	Suntzeff, et al. 1957
Mouse (Swiss, Millerton)	25-50	M&F	10% solution of fluoranthene in acetone applied 3 times weekly	dermal application to the back	No papillomas or carcinomas found by 13 months	Suntzeff, et al. 1957
Mouse	20	M&F	not specified	subcutaneous injection	No sarcomas produced	Buu-hoi, 1964

TABLE 10 (Continued)

Species	No.	Sex	Preparation and Dose	Route of Administration	Results	Reference
Mouse (Ha/ICR/Mil Swiss Albino)	20	F	50 µl of 1.0% fluoranthene solution in acetone applied 3 times weekly for 12 months	dermal application	No tumors observed after 15 months; no mortality encountered	Hoffmann, et al. 1972
Mouse (C3H)	15	M	50 mg fluoranthene as an 0.5% solution in decalin applied 2 times each week for 82 weeks	dermal application	No skin tumors observed; 13 of 15 mice were alive at 52 weeks	Horton and Christian, 1974
Mouse (C3H)	15	M	50 mg fluoranthene as an 0.5% solution in 50:50 decalin-n-dodecane applied 2 times each week for 82 weeks	dermal application	No skin tumors observed; 12 of 15 mice were alive at 52 weeks	Horton and Christian, 1974
Mouse (ICR/Ha Swiss)	50	F	40 µg fluoranthene in acetone applied 3 times weekly for 440 days	dermal application	No skin tumors observed	Van Duuren and Goldschmidt, 1976

It has been established for many years that fluoranthene is inactive as a complete carcinogen. In recent years fluoranthene has also been tested for tumor initiating and promoting activity (Hoffmann, et al. 1972; Van Duuren and Goldschmidt, 1976).

Fluoranthene was applied repeatedly to the shaved backs of mice and followed by application of croton oil (a known tumor promoter) to test for initiating activity (Hoffmann, et al. 1972). As indicated in Table 11, fluoranthene displayed no significant capacity for tumor initiation.

In related studies conducted by Van Duuren and Goldschmidt (1976) fluoranthene was tested as a tumor promoter in a two-stage carcinogenesis test system. Their results were equivocal and indicated that, at best, fluoranthene was only a very weak tumor promoter in comparison to the action of classical tumor promoting chemicals such as phorbol myristate acetate (PMA) (the active component of croton oil) (Table 12).

The most remarkable aspect of the biological activity of fluoranthene is its potency as a cocarcinogen. The designation of a cocarcinogen is here intended to denote a compound that on repeated application to mouse skin together with low doses of a complete carcinogen such as benzo(a)pyrene, produces a considerable enhancement in carcinogenic effect (Van Duuren, 1976). It should be noted that, by this definition, a cocarcinogen need not necessarily possess either tumor initiating or tumor promoting activity in the two-stage carcinogenesis system.

It was first recognized by Hoffmann and Wynder (1963) in studies on the components of gasoline engine exhaust that fluoranthene

TABLE 11

Tumor Initiating Activity of Fluoranthene[a,*]

Species	No.	Sex	Dose and Preparation	Tumors After 20 weeks
Mouse (Swiss-Albino Ha/ICR/Mil)	30	F	0.1 mg fluoranthene in 50 µl acetone applied every 2nd day for 10 applications	1[b](1)[c]29[d]
Mouse	30	F	10 applications at 5 µg benzo(a)-pyrene[e]	19(67)29

[a]Ten days after last application of fluoranthene, the tumor promoter 2.5% croton oil in acetone, average dose 3.8 mg, was applied for 20 weeks

[b]Tumor-bearing mice

[c]Number in parenthesis = total number of tumors

[d]Surviving mice

[e]Positive control

*Source: Hoffmann, et al. 1972

TABLE 12

Two-stage Carcinogenesis: Tumor-Promoting Activity of Cocarcinogens and Inactive Analogues[a,*]

Secondary treatment[b] (dose)	Days of testing	Median Survival time in days	Days to first papilloma	Mice with papillomas/total papillomas[c]
Pyrene (40 µg)	448	448	414	1/1 (1)
Fluoranthene (40 µg)	448	448	401	1/1 (1)
Catechol (2 mg)	448	448	---	0
Resorcinol (10 mg)	449	449	---	0
Hydroquinone (5 mg)	409	409	---	0
Pyrogallol (5 mg)	449	449	328	1/1 (0)
PMA (2.5 µg)	449	357	54	43/155 (18)
Anthralin (80 µg)	434	434	85	9/14 (2)
Acetone	450	450	---	0
No treatment[d]	443	427	---	0
PMA alone (2.5 µg)	368	?	174	5/5 (0)

[a]50 female ICR/Ha Swiss mice per group, except for the anthralin experiment in which 20 mice were used.

[b]150 ug B(a)P/0.1 ml acetone applied to dorsal skin once by micropipette. For the anthralin experiment, the initiating dose was 100 ug B(a)P. For the duration of the test, the promoters were applied to the dorsal skin 3 times weekly in 0.1 ml acetone beginning 14 days after initiator. For data on the application of promoting compounds, see Table 2.

[c]Numbers in parentheses are numbers of mice with squamous carcinoma.

[d]100 mice.

*Source: Van Duuren and Goldschmidt, 1976

could enhance the yield of benzo(a)pyrene-induced skin carcinomas in mice. These results are depicted in Figures 1 and 2. Although the details of their experimental protocol were not reported, the authors concluded that the potential interaction of components in complex environmental mixtures dictates the need for caution in interpretation of results. In particular, the extrapolation of results from animal bioassays with single chemicals may not provide a realistic estimate of human risk resulting from exposure to these chemicals in combination.

A more detailed investigation of the cocarcinogenic activity of fluoranthene was undertaken by Van Duuren and Goldschmidt (1976). In that study, fluoranthene not only increased the total number of papillomas and carcinomas produced by benzo(a)pyrene on mouse skin, but also decreased the number of days to the appearance of the first tumor as compared to mice treated with benzo(a)pyrene only (Table 13). Among all the cocarcinogens tested in this study, only fluoranthene caused a marked decrease in the tumor latency period. These results led the authors to conclude that fluoranthene possesses potent cocarcinogenic activity.

The mechanism of action for cocarcinogenic compounds is not understood. Since both aliphatic and aromatic compounds have displayed cocarcinogenic activity, the elucidation of structure-activity relationships is difficult. Van Duuren and coworkers (1978) have proposed a number of possibilities to explain the effects of cocarcinogens. These include: (a) ability to alter the rate of absorption and disappearance of the carcinogen, (b) ability to alter metabolic pathways for the carcinogen, and (c) metal-chelating

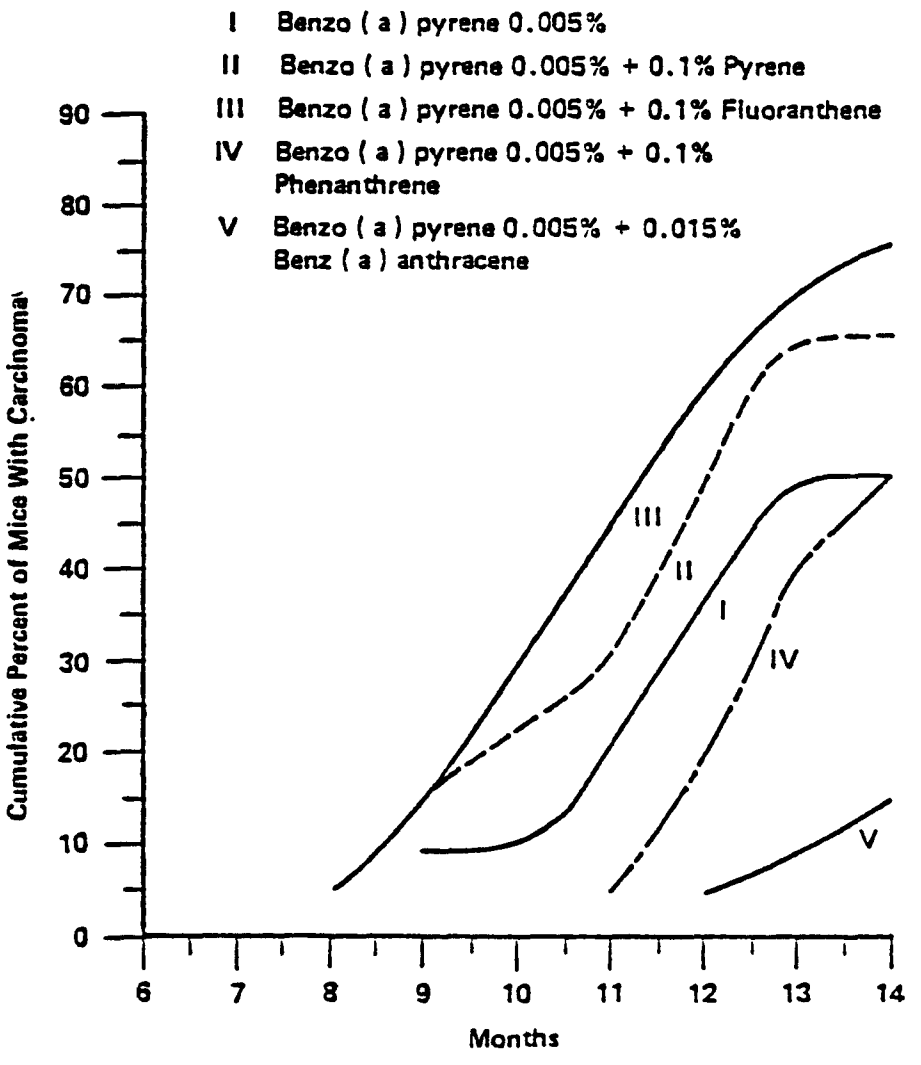

FIGURE 1

Effect of Fluoranthene on Production of Skin Carcinomas in Mice

Source: Hoffmann and Wynder, 1963

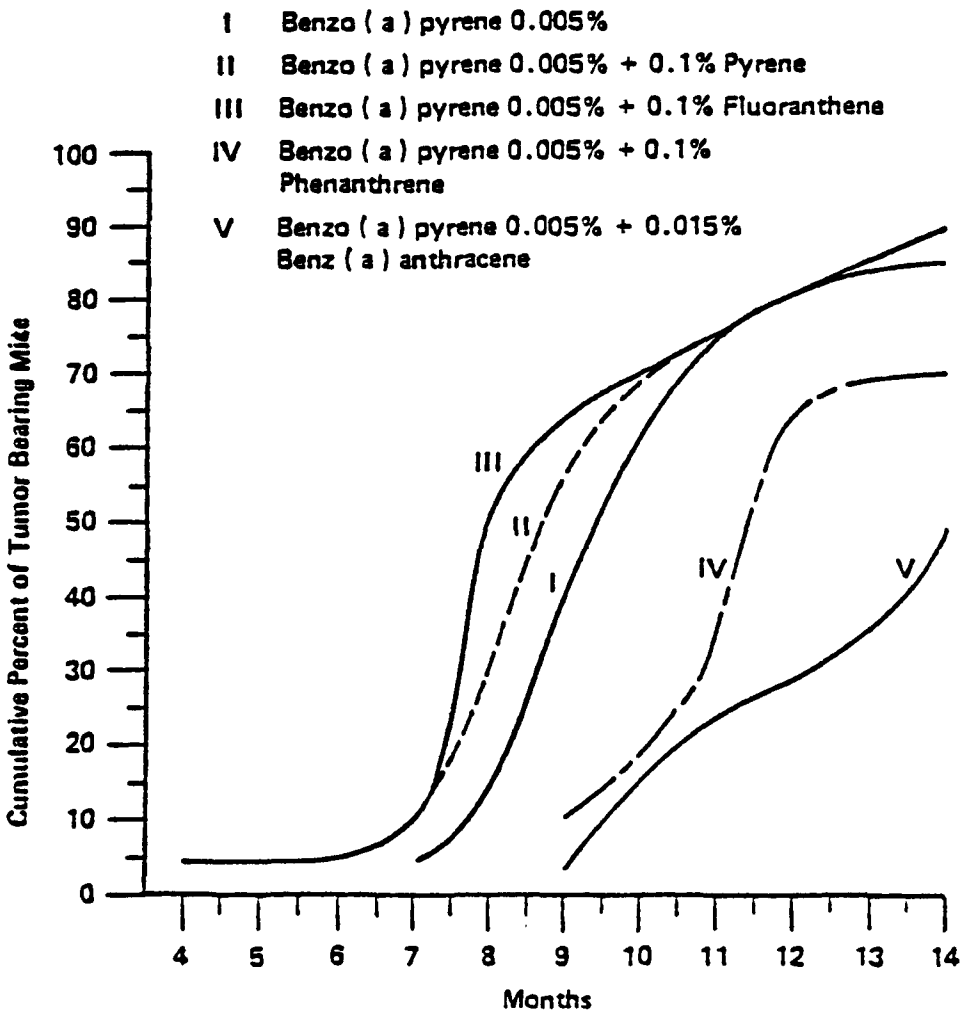

FIGURE 2

Effect of Fluoranthene on Production of Skin Tumors in Mice

Source: Hoffmann and Wynder, 1963

TABLE 13

Cocarcinogenesis: Bioassay in Mouse Skin[a,*]

Carcinogen[b]	Cocarcinogen (dose)	Days of testing	Days to first papilloma	Mice with papillomas/total papillomas[c]
Benzo(a)pyrene	Fluoranthene (40 µg)	440	99	39/126 (37)
none	Fluoranthene (40 µg)	440	___	0
Benzo(a)pyrene	Acetone	440	210	16/26 (12)
none	Acetone	440	___	0
none	none[d]	440	___	0

[a]50 female ICR/Ha Swiss mice used per group.

[b]Benzo(a)pyrene was applied in the same solution as the cocarcinogen (5 µg/0.1 ml acetone) three times weekly to the dorsal skin.

[c]Numbers in parentheses are numbers of mice with squamous carcinoma.

[d]100 mice, as stated by the source.

*Source: Van Duuren and Goldschmidt, 1976

ability. However, none of these possibilities is considered acceptable as a general mechanism of action for all compounds displaying cocarcinogenic activity. Furthermore, there is not enough information available to determine the importance of the cocarcinogenic activity of fluoranthene to human health.

There is no information available concerning the carcinogenicity of fluoranthene to humans.

CRITERION FORMULATION

Existing Guidelines and Standards

There have been no standards developed for fluoranthene in air, water, food, or the workplace. The only existing standard that takes fluoranthene into consideration is a drinking water standard for PAH. The 1970 World Health Organization European Standards for Drinking Water recommends a concentration of PAH not exceeding 0.2 µg/l. This recommended standard is based upon the analysis of the following six PAH in drinking water:

Fluoranthene
Benzo(a)pyrene
Benzo(g,h,i)perylene
Benzo(b)fluoranthene
Benzo(k)fluoranthene
Indeno(1,2,3-cd)pyrene

The designation of the above six PAH for analytical monitoring of drinking water was not made on the basis of potential health effects or bioassay data on these compounds (Borneff and Kunte, 1969). It should not be assumed that these six compounds have special significance in determining the likelihood of adverse health effects resulting from absorption of any particular PAH. They are considered to be a useful indicator for the presence of PAH pollutants. Borneff and Kunte (1969) found that PAH were present in ground water at concentrations as high as 50 ng/l, and in drinking water at concentrations as high as 100 ng/l. Based on these data they suggested that water containing more than 200 ng/l should be rejected. However, as data from a number of U.S. cities indicate (see Ingestion from water section), levels of PAH in raw and finished waters are typically less than the 0.2 µg/l criterion recommended by WHO (1970).

Current Levels of Exposure

Quantitative estimates of human exposure to fluoranthene require numerous assumptions concerning routes of exposure, extent of absorption, lifestyle, and variables relating to specifics of geography, sex, and age. Nevertheless, working with estimates developed for PAH as a class, certain extrapolations are possible to arrive at a crude estimate of fluoranthene exposure.

An estimate of fluoranthene intake from drinking water may be derived from data obtained in a survey of 16 U.S. cities (Basu and Saxena, 1977; Basu and Saxena, 1978). By arbitrarily assigning the lower limit of detectability for fluoranthene to those samples where none was detected, and using the measured values of fluoranthene in the four positive samples found, the estimated average fluoranthene level in drinking water would be 8.6 ng/l. Thus the daily intake of fluoranthene in drinking water may be calculated:

$$8.6 \text{ ng/l} \times 2 \text{ liters/day} = 17.2 \text{ ng/day}$$

Borneff (1977) estimates that the daily dietary intake of PAH is about 8-11 ug/day. As a check on this estimate, fluoranthene intake may be calculated based upon reported concentrations of fluoranthene in various foods (see Distribution section of the document), and the per capita estimates of food consumption by the International Commission on Radiological Protection (1974). Taking a range from 1 to 10 ppb as a typical concentration for fluoranthene in various foods, and 1,600 g/day as the total daily food consumption by man from all types of foods (i.e., fruits, vegetables, cereals, dairy products, etc.), the intake of fluoranthene from the diet would be in the range of 1.6-16 μg/day.

It has recently been reported that fluoranthene concentrations in ambient air average about 4 µg/1,000 m^3 (Santodonato, et al. 1978). If it is assumed that 100 percent of the fluoranthene which is inhaled is absorbed, and that the average amount of air inhaled by a human each day is about 10-20 m^3, then fluoranthene intake via the air would be in the range of 40-80 ng/day. However, in certain indoor environments, particularly in the presence of sidestream tobacco smoke, PAH exposure from inhaled air may be considerably higher (Grimmer, et al. 1977).

In summary, a crude estimate of total daily exposure to fluoranthene would be as follows:

Source	Estimated Exposure
Water	0.017 µg/day
Food	1.6 - 16 µg/day
Air	0.040 - 0.080 µg/day

The above cited figures show that foods are the greatest source of fluoranthene to humans. Accordingly, the present levels of fluoranthene in drinking water would be expected to contribute little to the total human intake.

It should be noted that two factors in the above estimates are not taken into account. First, it is known that tobacco smoking can contribute greatly to fluoranthene exposure in man. It is estimated that smoking one cigarette will increase exposure to fluoranthene via the lungs by about 0.26 µg (Hoffmann, et al. 1972). The sum of methylfluoranthene in the smoke of a nonfiltered cigarette is about 0.18 ng (Hoffmann, et al. 1972). Second, it is assumed that dermal absorption of fluoranthene contributes only a negligible amount to the total exposure. It is expected that only in

certain occupational situations would dermal exposure be a quantitatively important route of exposure.

Special Groups at Risk

Individuals living in areas which are heavily industrialized, and in which large amounts of fossil fuels are burned, would be expected to have greatest exposure from ambient sources of fluoranthene. In addition, certain occupations (e.g., coke oven workers, steelworkers, roofers, automobile mechanics) would also be expected to have greater exposure than the general population.

Exposure to fluoranthene will be considerably increased among tobacco smokers or those who are exposed to smokers in closed environments (i.e., indoors).

Basis and Derivation of Criteria

The attempt to develop a valid drinking water criterion for fluoranthene is hindered by several gaps in the scientific data base:

(1) There have been no chronic dose-response studies conducted with fluoranthene in animals.

(2) There are no chronic animal toxicity studies involving oral exposure to fluoranthene.

(3) There are no human data concerning the effects of exposure to fluoranthene.

From a survey of PAH in U.S. drinking waters using the same criteria for analysis as recommended by the World Health Organization, it is possible to calculate the amount of fluoranthene relative to other PAH in the same sample (Saxena, et al. 1977; Basu and Saxena, 1977,1978). These data indicate that in drinking water samples where fluoranthene was detected, it represented about 58.9 percent of the total PAH. Therefore, the drinking water standard

recommended by WHO (1970) for PAH of 0.2 µg/l would be equivalent to a drinking water standard for fluoranthene of:

$$0.2\ \mu g/l \times 0.589 = 0.12\ \mu g/l$$

Attempts to develop a water quality criterion based upon animal toxicity data are seriously hindered by an inadequate data base. The only study available which shows a no-effect level for fluoranthene (in terms of chronic mortality) was reported by Hoffmann, et al. (1972). This study involved dermal administration of fluoranthene to mice, and necessitates the assumption that 100 percent of the applied dose was absorbed. Their data can be used to develop a water quality criterion based on the method employed by the U.S. EPA in formulating national interim primary drinking water regulations (Saxena, et al. 1977).

Calculation of the criterion is summarized in Table 14. The approach took into consideration the contribution of dietary and airborne sources of fluoranthene. Once these factors are accounted for, this procedure leads to the conclusion that 42 µg/l of fluoranthene in drinking water would represent an acceptable level of exposure. It must be emphasized, however, that the criterion is based on chronic toxicity data with mortality being the endpoint, and applies only to situations where exposure occurred to fluoranthene alone.

Because of the limitations in the data base concerning fluoranthene toxicity, it is considered necessary to apply an uncertainty factor of 1,000 in the calculation of an exposure criterion. The animal study upon which the criterion is derived involved only 20 mice, which received dermal applications of fluoranthene at one

TABLE 14

Derivation of Criterion for Fluoranthene in Water

Species	Chronic No-effect Level (Hoffman, et al. 1972)	mg/kg body weight/day[a]	Calculated Maximum Safe Levels			Intake from:		Maximum Permissible Intake from Water	Recommended[d] Limit
			Safety Factor	mg/kg/day (x)	mg/man/day[b]	Diet mg/man/day[c]	Air mg/man/day[c]	mg/day	µg/l
Mouse (Ha/ICR/Mil Swiss Albino	50 µl of 1.0% fluoranthene in acetone applied 3 times weekly for 12 months	6.12	1/1000	0.006	0.420	0.016	0.0001	0.400	42.0

[a]Assume weight of mouse = 35 grams

[b]Assume average weight of human adult = 70 kg

[c]Calculated as described

[d]Assume average daily intake of water for man = 2 liters.

Calculated as follows:

$$\frac{0.400 \text{ mg}}{2 \text{ liter} + (0.0065 \times 1150)} = 0.042 \text{ mg/l} = 42.0 \text{ µg/l.}$$

where: 0.0065 = average daily consumption of fish and shellfish in kilograms
1150 = bioconcentration factor for fluoranthene

dose level three times weekly for 12 months. Thus, the use of these data for calculation of a criterion relating to ingestion of fluoranthene in humans will admittedly be imprecise. To justify the use of an uncertainty factor of less than 1,000, however, valid results of long-term feeding studies in one or more species of experimental animal would be required. In environmental situations, it is well established that fluoranthene is found in the presence of numerous PAH; a situation having important implications for potential toxic interactions.

Several studies have clearly shown that fluoranthene possesses no carcinogenic activity, and is neither a tumor initiator nor a tumor promoter (see Carcinogenicity section). However, two carefully conducted studies have shown that fluoranthene, when applied to mouse skin together with much smaller quantities of benzo(a)pyrene, could act as a cocarcinogen to increase tumorigenic response. These data do not permit a quantitative estimation of health risks incurred by this type of biological phenomenon. Nevertheless, because fluoranthene is present in environmental mixtures together with other PAH (including several carcinogens) it may pose an additional risk to the population exposed. In view of the cocarcinogenic and anticarcinogenic properties of several environmental PAH, the degree of added risk, if one exists, cannot be easily determined on the basis of our present scientific knowledge. At least one study (Pfeiffer, 1977) has demonstrated that when fluoranthene was administered together with 11 other PAH (carcinogenic and noncarcinogenic) by cutaneous injection to mice, it had no enhancing effect on tumor incidence. However, because of the close

association between fluoranthene and the other PAH, some of which are known carcinogens, it would seem prudent to temporarily limit the level of fluoranthene in drinking water to no more than the acceptable concentration of all non-fluoranthene PAH. In any event, the adoption of the recommended water quality criterion for PAH as a class would undoubtedly result in levels of fluoranthene in water which are below the 42 µg/l criterion derived in Table 14.

Inadequacies in the current scientific data base prevent the formulation of a water quality criterion for fluoranthene based on potential cocarcinogenicity. In addition, since environmental exposures to fluoranthene will almost certainly involve concomitant exposure to carcinogenic PAH, their potential interaction should be considered in future research and health criteria development.

In summary, based on the use of chronic mouse toxicological data and an uncertainty factor of 1,000, the criterion level of fluoranthene corresponding to an acceptable daily intake of 0.4 mg, is 42 µg/l. Drinking water contributes 21 percent of the assumed exposure while eating contaminated fish products accounts of 79 percent. The criterion level can similarly be expressed as 54 µg/l if exposure is assumed to be from the consumption of fish and shellfish products alone.

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