Canadian Water Quality Guidelines for the Protection of Aquatic Life

POLYCYCLIC AROMATIC HYDROCARBONS (PAHs)

Polycyclic aromatic hydrocarbons (PAHs) are a group of organic compounds that contain two or more benzene rings in their structure. Present in the environment mainly as a result of incomplete combustion of forest fires, internal combustion engines, wood stoves, and coal coking, etc., PAHs are also constituents of petroleum and its derivatives (Neff 1979). Oil spills and refinery effluents are major sources of PAH contamination of freshwater and marine environments. Domestic sewage, stormwater runoff, landfills, the wood preservative industry (e.g., creosote), and waste disposal sites are further contributors of anthropogenic PAHs to the environment. Neff (1985) reported that PAHs were released by aluminium smelters using Soderberg electrodes. PAHs of natural origin are produced at very low rates (Blumer 1976).

PAHs are ubiquitous in terrestrial, atmospheric, and aquatic environments throughout the world and have been detected in rivers, lakes, groundwaters, sediments, soils, and biota throughout Canada.

PAHs are nonpolar, hydrophobic compounds that do not ionize. Volatilization, photolysis, hydrolysis, microbial degradation, and adsorption and subsequent sedimentation determine the fate of PAHs in the environment (Southworth 1979). Sorption to sediment substrates plays an important role in PAH transport and distribution (Smith et al. 1978; USEPA 1982b; Broman et al. 1991). PAHs tend to adsorb onto solid phases in aquatic environments because of their hydrophobic nature and low water solubilities (Neff 1979; NRCC 1983; Eisler 1987; Slooff et al. 1989). The association of PAHs with the solid phase depends on their molecular weight and octanol–water partitioning coefficient (K_{ow}). Up to 88% of benzo*(a)*pyrene in aquatic systems, for instance, was associated with particulate matter, while 13% of fluorene and 20% of pyrene were associated with particulate (Broman et al. 1991). PAHs may be retained in the water column in the presence of dissolved organics such as humic acids, which increase the solubility of the compound (Slooff et al. 1989; Pinal et al. 1990).

Photodegradation is an important degradation pathway in aquatic systems for high molecular weight PAHs (Suess 1976). Photooxidation can chemically transform PAHs, and the resulting products may be more carcinogenic and toxic than the parent compounds (Suzuki et al. 1982; USEPA 1982b, 1982c; NRCC 1983).

Particle-bound PAHs or PAHs adsorbed to water-suspended materials are more resistant to photo-degradation (McGinnes and Snoeyink 1974; Korfmacher et al. 1980a, 1980b). Other researchers, however, have found that PAHs attached to particulate matter are more susceptible to photolysis than PAHs in solution (Neff 1979; Moore and Ramamoorthy 1984). Zepp and Schlotzhauer (1979) also reported that the partitioning of high molecular weight PAHs to sediment decreases the rate of photooxidation. Smith et al. (1978) reported that the photooxidation half-lives of some PAHs in natural waters are 20–60% longer than those in laboratory solutions.

Volatilization plays an important role in the removal of low molecular weight PAHs from aquatic systems (USEPA 1982a, 1982b, 1982c). Naphthalene has the highest vapour pressure of the PAHs, and volatilization from aquatic environments is probably the most important removal mechanism for this compound (Callahan et al. 1979; Southworth 1979; USEPA 1982a). Based on their Henry's law constants, acenaphthene, anthracene,

Table 1. Water quality guidelines for polycyclic aromatic hydrocarbons for the protection of aquatic life (Environment Canada 1998).

Aquatic life		Guideline value ($\mu g \cdot L^{-1}$)
Freshwater	Acenaphthene	5.8*
	Acridine	4.4*
	Anthracene	0.012*
	Benz*(a)*anthracene	0.018*
	Benzo*(a)*pyrene	0.015*
	Chrysene	NRG†
	Fluoranthene	0.04*
	Fluorene	3.0*
	Naphthalene	1.1*
	Phenanthrene	0.4*
	Pyrene	0.025*
	Quinoline	3.4*
Marine	Naphthalene	1.4*

*Interim guideline.

†No recommended guideline.

fluorene, and phenanthrene have moderate volatility (Coover and Sims 1987). Park et al. (1990), however, suggested that volatilization was insignificant for PAHs with three or more aromatic rings.

PAHs are subject to biodegradation by various microorganisms such as bacteria, fungi, and certain algae that live in soils, in sediment substrate, or are suspended in the water column (Gibson et al. 1975; Gibson 1976).

Microbial degradation of PAHs is one of the main processes responsible for removing these substances from bottom sediments and the water column. Biodegradation of PAHs depends on such factors as the number of aromatic rings and type of ring fusion (Walker et al. 1975; Herbes and Schwall 1978; Lee et al. 1978; USEPA 1982b; Wild et al. 1991). Herbes and Schwall (1978) found that the turnover times (1/rate constant) of PAHs exposed to sediment-associated microorganisms increased 30–100 times per additional aromatic ring. It has also been observed that many two- and three-ringed PAHs, such as naphthalene, phenanthrene, and anthracene, are readily degraded and may be used as primary substrates by PAH-degrading organisms (Herbes and Schwall 1978; Gardner et al. 1979; Sims and Overcash 1983; Uthe 1991). Higher molecular weight compounds, such as pyrene and benzo*(a)*pyrene, degrade more slowly. Some degradation-resistant PAHs are inadequate sources of carbon and are thought to degrade mainly by cometabolism, where one hydrocarbon acts as a substrate for growth while a second, which cannot act as a growth substrate, is degraded by the same process (Neff 1979; NRCC 1983).

In animals, the mixed-function oxygenase (MFO) enzyme systems are responsible for the biotransformation of PAHs. Detoxification of PAHs is not a simple process. Before formation of nontoxic and harmless end products by various enzymatic and nonenzymatic reactions, PAHs are converted to arene oxide intermediates followed by formation of derivatives of trans-dihydrodiols, phenols, and quinones. These intermediate products are known to be toxic, carcinogenic, and/or mutagenic (Moore and Ramamoorthy 1984) and are further broken down to less toxic products by various enzymatic and nonenzymatic reactions (Neff 1979).

Aquatic organisms may remove a significant fraction of PAHs from a body of water. Pelagic organisms may take up PAHs directly from the water column. Benthic organisms may absorb these substances from contact with bottom sediments and the overlying water. Uptake of these compounds, however, tends to occur much more rapidly in the solubilized form. At a high concentration and in a short exposure situation, therefore, pelagic organisms may actually be more at risk than their benthic counterparts.

Aquatic organisms can accumulate PAHs from water, sediment, and food. The literature suggests that PAH uptake by aquatic organisms depends on several factors: (a) physical and chemical properties of the PAH (e.g., molecular weight, octanol–water partition coefficient, etc.); (b) environmental variables (e.g., suspended matter, dissolved organic matter, bioavailability, temperature, presence of other contaminants, biodegradation, etc.); and (c) biological factors (e.g., PAH metabolism and depuration rates, feeding characteristics of organisms, fat content of tissue, life stage, etc.) (McElroy et al. 1989).

The bioconcentration data from the literature exhibit a high degree of variability between species, PAH compounds, as well as within species and over time (Neff 1979; USEPA 1982a, 1982b, 1982c; NRCC 1983). The ability of different organisms to metabolize PAHs appears to play a major role in the potential for bioaccumulation and bioconcentration. Algae, mollusks, and other species, for example, which cannot metabolize PAHs rapidly, exhibit the highest BCFs, while fish and many crustaceans, which readily metabolize PAHs, generally obtain lower whole body residues (Eisler 1987; Neff 1982; Landrum and Scavia 1983).

Water Quality Guideline Derivation

The interim Canadian water quality guidelines for PAHs for the protection of aquatic life were developed based on the CCME protocol (CCME 1991). For more information, see the supporting document (Environment Canada 1998).

Freshwater Life

Acenaphthene

Acute toxicity data were available for five species of freshwater fish, with 96-h LC_{50}s ranging from 580 $\mu g \cdot L^{-1}$ for brown trout *(Salmo trutta)* to 1730 $\mu g \cdot L^{-1}$ for juvenile fathead minnows *(Pimephales promelas)* (Holcombe et al. 1983; Geiger et al. 1985). Cairns and Nebeker (1982) exposed fathead minnow embryos to acenaphthene for 32–35 d and reported LOECs of 495 $\mu g \cdot L^{-1}$ for growth and 682 $\mu g \cdot L^{-1}$ for survival. Lemke (1983) conducted an interlaboratory comparison to evaluate the sensitivity of fathead minnow embryos to acenaphthene. The 28-d

NOECs from seven laboratories ranged from 4 to 420 $\mu g \cdot L^{-1}$.

Acceptable data for invertebrates was limited. The 48-h LC_{50} and NOEC for *Daphnia magna* were 41 000 and 600 $\mu g \cdot L^{-1}$, respectively (LeBlanc 1980). A 96-h LC_{50} of >2040 $\mu g \cdot L^{-1}$ was reported for the snail *Aplexa hypnorum* (Holcombe et al. 1983).

Bastian and Toetz (1982) reported that a 14-d exposure to 2427 $\mu g \cdot L^{-1}$ of acenaphthene increased the biomass of a blue–green algae culture *(Anabaena flos-aquae)* by 26%. A 2-h exposure to acenaphthene levels of 421–4619 $\mu g \cdot L^{-1}$ had no effect on nitrogen fixation by *A. flos-aquae* (Bastian and Toetz 1985).

The interim water quality guideline for acenaphthene for the protection of freshwater life is 5.8 $\mu g \cdot L^{-1}$. It was derived by multiplying the 96-h LC_{50} of 580 $\mu g \cdot L^{-1}$ for brown trout (Holcombe et al. 1983) by a safety factor of 0.01 (CCME 1991). Because the LOEC values were near the LC_{50} values, it was deemed that deriving the guideline from a chronic endpoint would not ensure that the whole range of sensitivities would be covered. The acute 96-h LC_{50} with a higher safety factor was, therefore, chosen in preference to the chronic LOEC for growth (Cairns and Nebeker 1982). Acenaphthene was considered to be a persistent substance, as its half-life in water is 12 d to 14 weeks (SRC 1989).

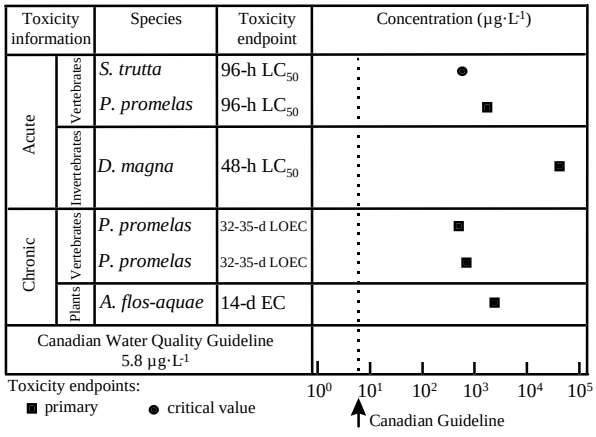

Figure 1. Select freshwater toxicity data for acenaphthene.

Acridine

Chronic toxicity data were available for two freshwater fish species. Freshly fertilized eggs from both rainbow trout *(Oncorhynchus mykiss)* and largemouth bass *(Micropterus salmoides)* were treated with acridine until 4 d after hatching (Black et al. 1983; Millemann et al. 1984). The average hatching times were 23 d for rainbow trout and 3 d for largemouth bass. Black et al. (1983) reported that 4 d after hatching, the 27-d and 7-d LC_{50}s were 320 and 1020 $\mu g \cdot L^{-1}$, respectively. Millemann et al. (1984) used an identical protocol to Black et al. (1983) and reported 27-d (4-d posthatch) and 7-d (4-d posthatch) LC_{50}s of 300 and 910 $\mu g \cdot L^{-1}$ for rainbow trout and largemouth bass, respectively.

Several PAHs are acutely toxic only in the presence of solar UV radiation. Oris and Giesy (1987) reported that exposing fathead minnows simultaneously to 525 $\mu g \cdot L^{-1}$ acridine and UV radiation resulted in 50% mortality in 4.3 h. The 96-h exposure in complete darkness at the above concentration was not toxic.

Acute toxicity data ranged from a 48-h LC_{50} of 1860 $\mu g \cdot L^{-1}$ for *Chironomus tentans* (Millemann et al. 1984) to a 48-h LC_{50} of 2300 $\mu g \cdot L^{-1}$ for *D. magna* (Parkhurst et al. 1981a).

D. magna were exposed to acridine for 28 d in full life cycle toxicity tests (Parkhurst et al. 1981a, 1981b). The total number of young produced per female, the number of broods produced per female, and the number of young per brood were assessed. The NOECs for all three endpoints were 400 $\mu g \cdot L^{-1}$, and the LOECs were 800 $\mu g \cdot L^{-1}$.

Newsted and Giesy (1987) reported an LT_{50} of 53.8 min for *D. magna* simultaneously exposed to 440.1 $\mu g \cdot L^{-1}$ acridine and simulated sunlight.

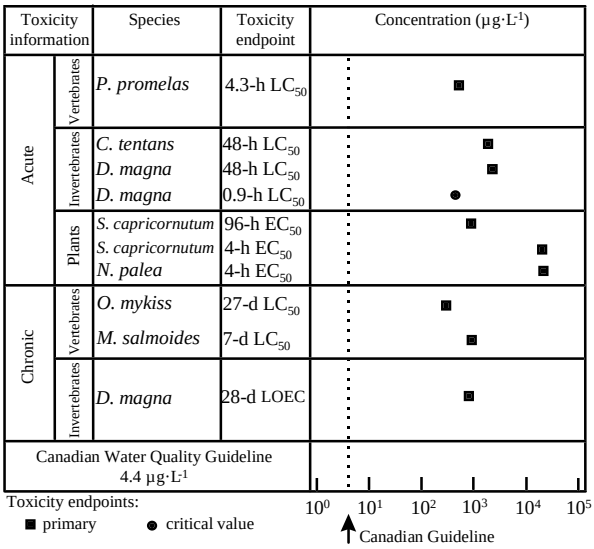

Figure 2. Select freshwater toxicity data for acridine.

Phytotoxicity data are limited. Blaylock et al. (1985) studied the effect of acridine on growth in *Selenastrum capricornutum* and found a 96-h EC_{50} of 900 $\mu g \cdot L^{-1}$. Millemann et al. (1984) reported 4-h EC_{50}s of 20 000 and 20 800 $\mu g \cdot L^{-1}$ for *S. capricornutum* and *Nitzschia palea*, respectively.

The interim water quality guideline for acridine for the protection of freshwater life is 4.4 $\mu g \cdot L^{-1}$. It was derived by multiplying the most sensitive acute concentration of 440.1 $\mu g \cdot L^{-1}$ for *D. magna* (LT_{50} = 0.9 h) by a safety factor of 0.01 (CCME 1991). Although appropriate data were not available from the literature, acridine was considered to be a persistent chemical because it has properties (e.g., K_{oc}, molecular weight, and phototoxicity) similar to other PAHs in its group (PAHs with three benzene rings, e.g., anthracene). The 0.9-h LT_{50} of 440.1 $\mu g \cdot L^{-1}$ for daphnia (Newsted and Giesy 1987) was chosen as the starting point over the 27-d LC_{50} of 300 $\mu g \cdot L^{-1}$ for rainbow trout (Millemann et al. 1984) for two reasons: (1) photoinduced toxicity is relatively more severe than an acute or a chronic toxicity effect in the absence of UV light; and (2) a guideline based on phototoxic effect will be protective of all adverse effects, including photo-induced toxicity. It is assumed that the experimental exposure to either simulated or natural sunlight can resemble the potential exposure to UV radiation in the field.

Anthracene

Anthracene was not acutely toxic to bluegill sunfish *(Lepomis macrochirus)* at saturation concentrations under conditions of artificial light (gold fluorescent light at 500 nm), shade, or darkness (Spacie et al. 1983). However, in the presence of solar UV radiation, anthracene is extremely toxic. The acute toxicity of anthracene to bluegill sunfish depends on the amount of time an animal is exposed to solar UV radiation. Oris and Giesy (1986) reported 96-h LC_{50} values ranging from 4.5 $\mu g \cdot L^{-1}$ for a 24-h light/0-h dark photoperiod to 46 $\mu g \cdot L^{-1}$ for a 6-h light/18-h dark photoperiod.

Invertebrates are also very sensitive to anthracene in the presence of solar radiation. *D. pulex* were exposed to anthracene levels of 1.2, 7.5, and 32.7 $\mu g \cdot L^{-1}$ under laboratory lighting for 24 h (Allred and Giesy 1985). None of these treatments were toxic. When the animals were subsequently exposed to solar radiation, there was 100% immobilization within 2 min at 32.7 $\mu g \cdot L^{-1}$ and within 10 min at 7.5 $\mu g \cdot L^{-1}$. At the lowest treatment level (1.2 $\mu g \cdot L^{-1}$), 50% of the treated daphnids were immobilized within 15 min. The affected organisms did not recover when returned to freshwater and laboratory lighting.

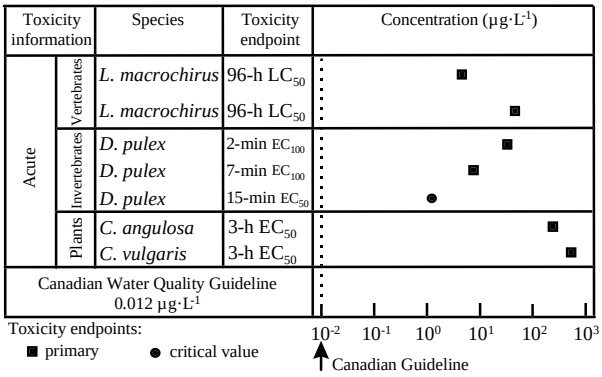

Figure 3. Select freshwater toxicity data for anthracene.

Hutchinson et al. (1980) reported 3-h EC_{50}s of 239 and 535 $\mu g \cdot L^{-1}$, respectively, for the green algae *Chlamydomonas angulosa* and *Chlorella vulgaris*. Gala and Giesy (1993) suggested that the carotenoid pigments provided algae *(S. capricornutum)* with greater resistance to the photoinduced toxicity of anthracene relative to aquatic animals.

The interim water quality guideline for anthracene for the protection of freshwater life is 0.012 $\mu g \cdot L^{-1}$. It was derived by multiplying the acute value (~15 min LT_{50}) of 1.2 $\mu g \cdot L^{-1}$ (Allred and Giesy 1985) for *D. pulex* by a safety factor of 0.01 (CCME 1991). Anthracene was considered to be a persistent substance, as its half-life in water is longer than 8 weeks (SRC 1989). It is assumed that the experimental exposure to either simulated or natural sunlight can resemble the potential exposure to UV radiation in the field.

Benz(a)anthracene

Data for benz*(a)*anthracene toxicity in the freshwater environment are very limited. Brown et al. (1975) reported 87% mortality of bluegill sunfish exposed to 1000 $\mu g \cdot L^{-1}$ of benz*(a)*anthracene for 6 months. The concentrations used by these investigators in their study, however, were much higher than the aqueous solubility of the PAH (11 $\mu g \cdot L^{-1}$). More recently, Oris and Giesy (1987) found that 50% of fathead minnows *(P. promelas)* died in about 65 h when exposed to 1.8 $\mu g \cdot L^{-1}$ benz*(a)*anthracene in UV light (simulated sunlight).

A 48-h LC_{50} of 10 $\mu g \cdot L^{-1}$ was reported for *D. pulex* exposed to benz*(a)*anthracene (Trucco et al. 1983). In another study, Newsted and Giesy (1987) observed 50%

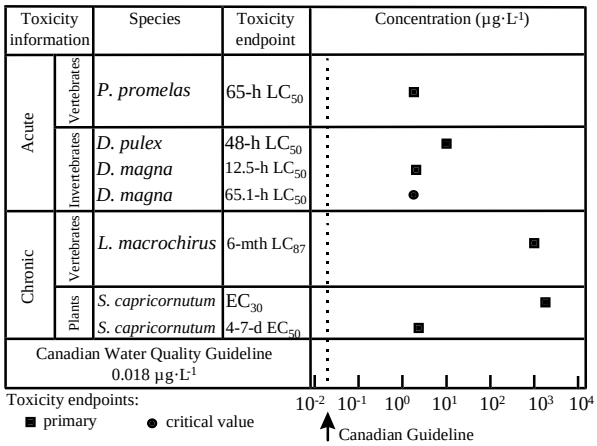

Figure 4. Select freshwater toxicity data for benz*(a)*anthracene.

mortality in *D. magna* exposed to benz*(a)*anthracene concentrations of 2 and 1.8 $\mu g \cdot L^{-1}$ after 12.51 and 65.1 h of UV exposure (in simulated sunlight), respectively.

A 30% reduction in growth was reported for the green alga *S. capricornutum* following an exposure to 1830 $\mu g \cdot L^{-1}$ (Schoeny et al. 1988). Cody et al. (1984) observed a 50% decrease in cell growth during a 4- to 7-d exposure to 2.3–22 800 $\mu g \cdot L^{-1}$ benz*(a)*anthracene.

The interim water quality guideline for benz*(a)*anthracene for the protection of freshwater life is 0.018 $\mu g \cdot L^{-1}$. It was derived by multiplying the acute value of 1.8 $\mu g \cdot L^{-1}$ for *D. magna* (Newsted and Giesy 1987) by a safety factor of 0.01 (CCME 1991). Benz*(a)*anthracene was considered to be a persistent substance, as its half-life in water is longer than 8 weeks (MacKay et al. 1992). It is assumed that the experimental exposure to either simulated or natural sunlight can resemble the potential exposure to UV radiation in the field. The interim Canadian water quality guideline for benz*(a)*anthracene was proposed even though there was insufficient information according to the CCME (1991) protocol (there was a lack of data on coldwater fish such as trout and invertebrates other than daphnia). The reasons in favour of an interim guideline were: (a) fathead minnows, found in a wide range of geographic locations that extend from the southern United States to the Northwest Territories in Canada, can be considered as a coldwater fish; and (b) daphnia are one of the key indicator species that are commonly used to assess toxicity of contaminants.

Benzo(a)pyrene

Chronic effects, including morphological abnormalities and necrosis of brain and spine, have been reported in rainbow trout eggs and alevins exposed to 0.08–0.21 $\mu g \cdot L^{-1}$ benzo*(a)*pyrene (Hannah et al. 1982; Hose et al. 1984). Oris and Giesy (1987) noted that 50% of the fathead minnows *(P. promelas)* exposed to 5.6 $\mu g \cdot L^{-1}$ benzo*(a)*pyrene and UV radiation died in 40 h. In the absence of UV radiation, however, a 96-h exposure to benzo*(a)*pyrene at a concentration of 5.6 $\mu g \cdot L^{-1}$ was not toxic.

Invertebrates were very sensitive to benzo*(a)*pyrene. Trucco et al. (1983) reported a 96-h LC_{50} of 5 $\mu g \cdot L^{-1}$ for *D. pulex*. Newsted and Giesy (1987) exposed *D. magna* to 1.5 $\mu g \cdot L^{-1}$ benzo*(a)*pyrene in the presence of solar UV radiation and reported an LT_{50} of only 4.4 h. Kagan and Kagan (1986) reported a 30-min LC_{50} of 8 $\mu g \cdot L^{-1}$ for mosquitoes *(A. agypti)* exposed to benzo*(a)*pyrene in the presence of UV radiation.

The green alga *S. capricornutum* was exposed to benzo*(a)*pyrene for 4–7 d using different light regimens (Cody et al. 1984). Under cool-white fluorescent light, a 30% inhibition of algal growth occurred at 25.2 $\mu g \cdot L^{-1}$; however, under fluorescent black light (high UV radiation), a complete inhibition of growth occurred at 16 $\mu g \cdot L^{-1}$.

The interim water quality guideline for benzo*(a)*pyrene for the protection of freshwater life is 0.015 $\mu g \cdot L^{-1}$. It was

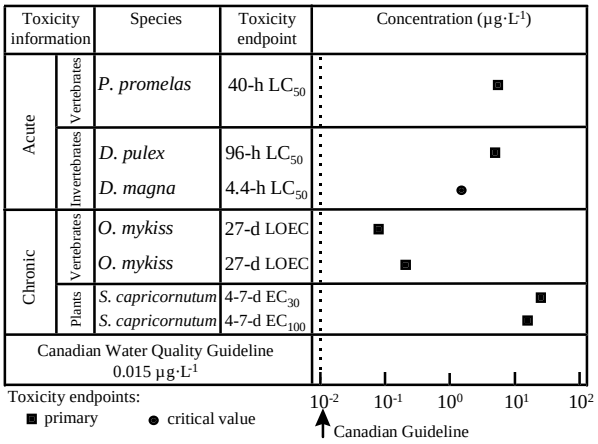

Figure 5. Select freshwater toxicity data for benzo*(a)*pyrene.

derived by multiplying the acute (~4-h LC_{50}) concentration of 1.5 $\mu g \cdot L^{-1}$ for *D. magna* (Newsted and Giesy 1987) by a safety factor of 0.01 (CCME 1991). Benzo*(a)*pyrene was considered to be a persistent substance, as its half-life in water is longer than 8 weeks (SRC 1989). It is assumed that the experimental exposure to either simulated or natural sunlight can resemble the potential exposure to UV radiation in the field. Twenty-seven day LOECs of 0.08 (Hannah et al. 1982) and 0.21 $\mu g \cdot L^{-1}$ (Hose et al. 1984) were also reported for morphological abnormalities in the early life stages of rainbow trout *(O. mykiss)*. The results of Hannah et al. (1982) and Hose et al. (1984), however, were not used in the guideline derivation because they were obtained in the presence of benzo*(a)*pyrene-contaminated sediment/sand in contact with the test water and it was not clear whether sediment toxicity was a factor in the effects.

Chrysene

Data were insufficient to derive a freshwater quality guideline for chrysene. A mortality rate of 50% was observed for *D. magna* exposed to 0.7 $\mu g \cdot L^{-1}$ chrysene and UV light for almost 24 h (the lowest reported effect level) (Newsted and Giesy 1987). Bastian and Toetz (1985) reported a 17% decrease in the rate of nitrogen fixation by blue–green algae (*A. flos-aquae*) exposed to 13.9 $\mu g \cdot L^{-1}$ chrysene. In an earlier experiment, these investigators observed a 35% reduction in the cell growth of the same alga exposed to 1.9 $\mu g \cdot L^{-1}$ chrysene. No other data for chrysene were found in the literature.

Fluoranthene

Kagan et al. (1985) found that 50% of fathead minnows (*P. promelas*) died in 24 h when exposed to 200 $\mu g \cdot L^{-1}$ fluoranthene in UV light for 30 min.

Newsted and Giesy (1987) and Kagan et al. (1985) reported a 50% mortality for *D. magna* exposed for 10.8 h to UV light and a fluoranthene concentration of 9 $\mu g \cdot L^{-1}$. Kagan et al. (1985) reported 1-h LC_{50}s of 4 and 12 $\mu g \cdot L^{-1}$ for *D. magna* and *Aedes aegypti*, respectively, after 1 h irradiation with UV light.

A 38% inhibition in growth of the blue–green alga *A. flos-aqua* was observed after a 14-d exposure to 38 $\mu g \cdot L^{-1}$ fluoranthene (Bastian and Toetz 1982). Complete inhibition of cell growth was observed following exposure to 417 $\mu g \cdot L^{-1}$ fluoranthene. Bastian and Toetz (1985) observed 20–28% inhibition of nitrogen fixation rate after a 2-h exposure of the alga to 434 $\mu g \cdot L^{-1}$ fluoranthene.

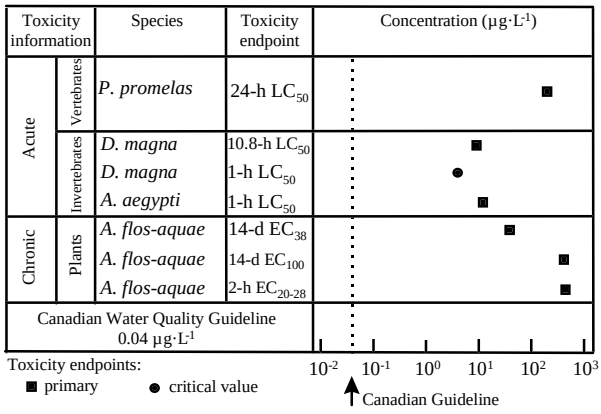

Figure 6. Select freshwater toxicity data for fluoranthene.

The interim water quality guideline for fluoranthene for the protection of freshwater life is 0.04 $\mu g \cdot L^{-1}$. It was derived by multiplying the acute 1-h LC_{50} of 4 $\mu g \cdot L^{-1}$ for *D. magna* exposed to UV light (Kagan et al. 1985) by a safety factor of 0.01 (CCME 1991). Fluoranthene was considered to be a persistent substance, as its half-life in water is longer than 8 weeks (MacKay et al. 1992). It is assumed that the experimental exposure to either simulated or natural sunlight can resemble the potential exposure to ultraviolet radiation in the field. The interim Canadian water quality guideline for fluoranthene was proposed even though the CCME requirement (CCME 1991) minimum data set was not met. (There was lack of data on coldwater fish such as trout.) The reasons in favour of the proposed interim guideline are the same as those suggested for benz*(a)*anthracene.

Fluorene

Finger et al. (1985) reported significant reductions in survival and growth of juvenile bluegill sunfish *(L. macrochirus)* at fluorene concentrations of 500 and 250 $\mu g \cdot L^{-1}$, respectively. Bluegill sunfish exposed to 62 $\mu g \cdot L^{-1}$ struck more frequently at food, but captured fewer prey. Such a reduction in feeding efficiency could translate into decreases in growth and reproductive capacity. These investigators also reported 96-h LC_{50}s of 820 and 910 $\mu g \cdot L^{-1}$, respectively, for rainbow trout *(O. mykiss)* and bluegill sunfish exposed to fluorene. Both species of fish suffered a loss of equilibrium at fluorene levels of 320 $\mu g \cdot L^{-1}$.

Finger et al. (1985) exposed *D. magna* to fluorene levels of 125 µg·L^{-1} and found reduced reproduction following 14 d (44% lower fecundity than control). The authors noted that measured fluorene concentrations in the chronic tests were 76% lower than the nominal concentrations. Finger et al. (1985) also reported that the emergence of larval midges *(Chironomus riparius)* was reduced following a 30-d exposure to fluorene at a concentration of 600 µg·L^{-1}.

There is considerable intraspecific variation in the sensitivities of algae to fluorene. A 20% decrease in nitrogen fixation was reported in the blue–green alga *A. flos-aquae* exposed to 612 µg·L^{-1} fluorene for 2 h (Bastian and Toetz, 1985). Finger et al. (1985) reported a 96-h EC_{50} (reduction in photosynthesis) of 3400 µg·L^{-1} for the alga *S. capricornutum* and a 21-d EC_{50} (production) of 20 000 µg·L^{-1} for the macrophyte *Chara* sp.

The interim water quality guideline for fluorene for the protection of freshwater life is 3.0 µg·L^{-1}. It was derived by multiplying the 14-d LOEC (a nominal chronic value) of 125 µg·L^{-1} reported for *D. magna* (Finger et al. 1985) by a safety factor of 0.1 (CCME 1991). The result, thus obtained, was then multiplied by a correction factor of 0.24 to derive the proposed guideline. This correction was required since the actual (or measured) fluorene concentration during chronic tests with daphnids was, on average, 24% of the reported nominal LOEC of 125 µg·L^{-1}.

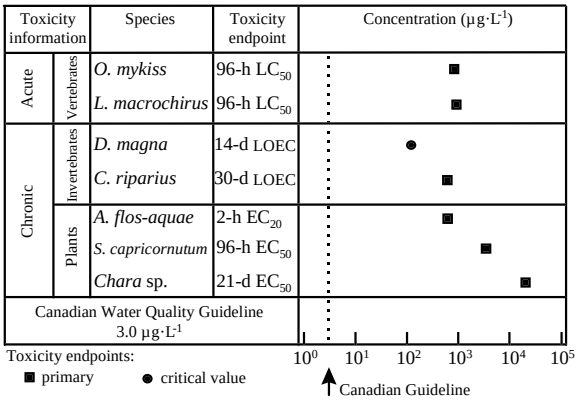

Figure 7. Select freshwater toxicity data for fluorene.

Naphthalene

Black et al. (1983) and Millemann et al. (1984) examined the acute toxicity of naphthalene to early life stages of rainbow trout and largemouth bass *(M. salmoides)*. Freshly fertilized eggs from both species were treated with naphthalene until 4 d after hatching. The average hatching times were 23 d for rainbow trout and 3 d for largemouth bass. Black et al. (1983) reported LC_{50}s at the time of hatching of 120 and >240 µg·L^{-1}, and 4-d posthatch LC_{50} values of 110 and 510 µg·L^{-1}, respectively, for rainbow trout and largemouth bass. These results were supported by Millemann et al. (1984) who found 4-d posthatch LC_{50}s of 120 and 680 µg·L^{-1}, respectively, for the same two species. Black et al. (1983) reported chronic values for rainbow trout *(O. mykiss)* larvae of 8, 15, and 46 µg·L^{-1} (~11 µg·L^{-1} is the geometric mean of the two lower values). These chronic values represented control-adjusted survival of 97, 91, and 84%, respectively, of the trout 4 d after hatching (at the embryo–larval stages).

Several studies have reported 96-h LC_{50} values for fathead minnows *(P. promelas)* exposed to naphthalene: 7900 µg·L^{-1} (DeGraeve et al. 1982), 6080 µg·L^{-1} (Holcombe et al. 1984), 1990 µg·L^{-1} (Millemann et al. 1984), and 6140 µg·L^{-1} (Geiger et al. 1985).

The acute sensitivity of daphnids to naphthalene has been assessed by several studies. For instance, 48-h LC_{50}s of 3400 µg·L^{-1} (Geiger and Buikema 1981) and 4663 µg·L^{-1} (Smith et al. 1988) were quoted for *D. pulex*. Similarly, 48-h LC_{50}s of 4100 µg·L^{-1} (Crider et al. 1982) and 2160 µg·L^{-1} (Millemann et al. 1984) were found for *D. magna*. Trucco et al. (1983) reported a 96-h LC_{50} of 1000 µg·L^{-1} for *D. pulex*; however, the study was conducted under a combination of fluorescent and natural light; therefore, it is possible that the increased sensitivity was due to photoenhanced effects.

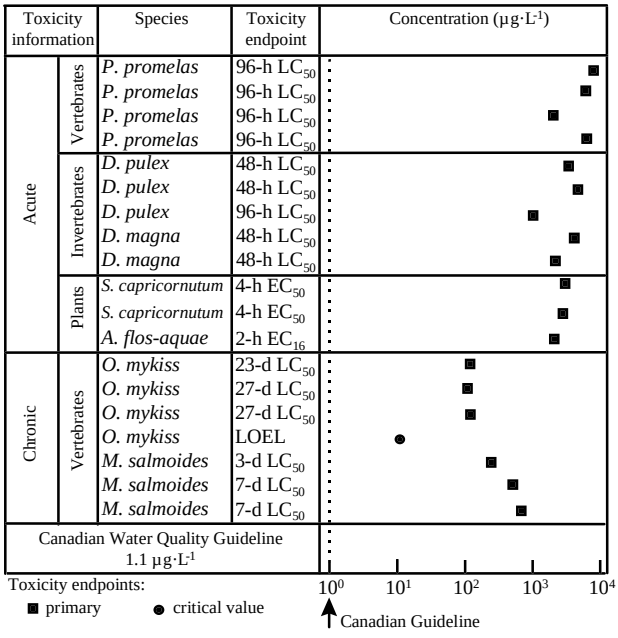

Figure 8. Select freshwater toxicity data for naphthalene.

Millemann et al. (1984) determined 4-h EC_{50}s (photosynthesis) of 2960 and 2820 $\mu g \cdot L^{-1}$, respectively, for the green alga *S. capricornutum* and the diatom *N. palea*. Bastian and Toetz (1985) reported a 16% decrease in nitrogen fixation for the blue–green alga *A. flos-aquae* following a 2-h exposure to 2071 $\mu g \cdot L^{-1}$.

The interim water quality guideline for naphthalene for the protection of freshwater life is 1.1 $\mu g \cdot L^{-1}$. It was derived by multiplying the chronic LOEL of 11 $\mu g \cdot L^{-1}$, which is the geometric mean of the lowest two of three chronic values, namely, 8, 15, and 46 $\mu g \cdot L^{-1}$ corresponding to the 97, 91, and 84% survival success in rainbow trout embryo–larval stages (Black et al. 1983), by a safety factor of 0.1 (CCME 1991).

Phenanthrene

Black et al. (1983) and Millemann et al. (1984) treated freshly fertilized eggs of rainbow trout *(O. mykiss)* and largemouth bass *(M. salmoides)* with phenanthrene until 4 d after hatching. The average hatching times were 23 d for rainbow trout and 3 d for largemouth bass. The early life stages of rainbow trout were more sensitive than that of bass. Black et al. (1983) reported LC_{50}s at the time of hatching of 40 $\mu g \cdot L^{-1}$ for the trout and >70 $\mu g \cdot L^{-1}$ for the bass and 4-d posthatch LC_{50}s of 40 and 180 $\mu g \cdot L^{-1}$, respectively. These authors also reported 93 and 82% control-adjusted survival of the trout (4-d posthatching at the embryo–larval stages) exposed to 4 and 8 $\mu g \cdot L^{-1}$ phenanthrene. Millemann et al. (1984) found 4-d posthatch LC_{50}s of 30 and 250 $\mu g \cdot L^{-1}$, respectively, for the trout and bass. Call et al. (1986) also conducted chronic tests with rainbow trout embryos exposed to phenanthrene. Several endpoints were examined, including hatching efficiency, teratogenic and dead fry at hatch, wet weight, and length, however, the most sensitive endpoint was mortality. The LOEC and NOEC for mortality were 8 and 5 $\mu g \cdot L^{-1}$, respectively, resulting in a SMATC (geometric mean of NOEC and LOEC) of 6 $\mu g \cdot L^{-1}$. The same study found 96-h LC_{50}s of 375 and 234 $\mu g \cdot L^{-1}$ and 96-h EC_{50}s (loss of equilibrium) of 50 and 49 $\mu g \cdot L^{-1}$ for rainbow trout and bluegill sunfish, respectively.

Call et al. (1986) assessed reproductive performance in phenanthrene-exposed *D. magna*. The 21-d LOEC and NOEC values were 163 and 57 $\mu g \cdot L^{-1}$, respectively, resulting in a SMATC of 96 $\mu g \cdot L^{-1}$. The same study examined the toxicity of phenanthrene to several invertebrate species. A 48-h EC_{50} (immobilization) of 117 $\mu g \cdot L^{-1}$ was reported for *D. magna*. These authors also reported 96-h EC_{50}s of 96 $\mu g \cdot L^{-1}$ (shortened tentacles and body column) for hydroids (*Hydra* sp.) and 126 $\mu g \cdot L^{-1}$ (immobilization) for amphipods *(Gammarus pseudolimnaeus)* exposed to phenanthrene. The Call et al. (1986) data suggest that fish are more sensitive to phenanthrene than invertebrates. Several studies have subjected daphnids to acute exposure to phenanthrene. For *D. pulex*, the reported endpoints ranged from a 96-h LC_{50} of 100 $\mu g \cdot L^{-1}$ (Trucco et al. 1983) to a 48-h LC_{50} of 1140 $\mu g \cdot L^{-1}$ (Geiger and Buikema 1981).

Acute phytotoxicity data for phenanthrene are available for blue–green algae *(A. flos-aquae)*, green algae *(C. vulgaris, C. angulosa,* and *S. capricornutum)*, duckweed *(Lemna minor)*, and the diatom *N. palea*. *A. flos-aquae* was the most sensitive, with Bastian and Toetz (1985) reporting that nitrogen fixation was decreased by 40% following a 2-h exposure to 134 $\mu g \cdot L^{-1}$ phenanthrene.

The interim water quality guideline for phenanthrene for the protection of freshwater life is 0.4 $\mu g \cdot L^{-1}$. It was derived by multiplying the chronic LOEL of 4 $\mu g \cdot L^{-1}$ for rainbow trout (corresponding to the control-adjusted 93% survival of the trout) (Black et al. 1983) by a safety factor of 0.1 (CCME 1991).

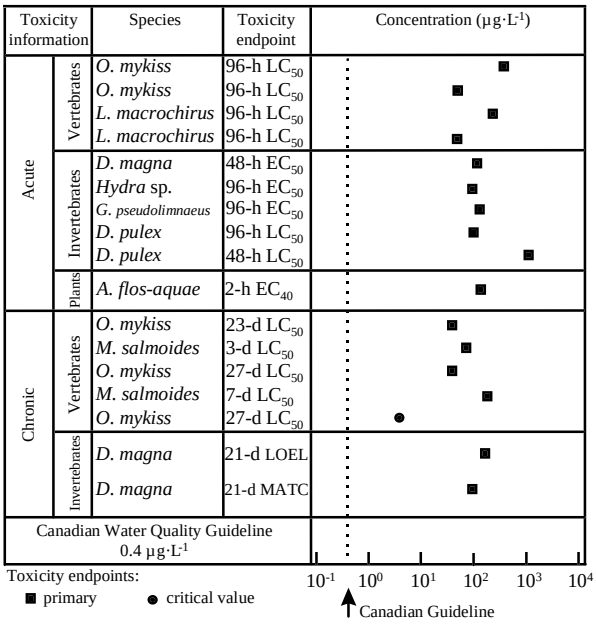

Figure 9. Select freshwater toxicity data for phenanthrene.

Pyrene

Oris and Giesy (1987) exposed juvenile fathead minnows *(P. promelas)* to pyrene in the presence of solar UV radiation and reported an LT_{50} of 3.2 h at 26 $\mu g \cdot L^{-1}$. Kagan et al. (1985) reported a 30-min LC_{50} of 220 $\mu g \cdot L^{-1}$ for fathead minnows exposed to pyrene in UV light (13 W $\cdot m^{-2}$). Kagan et al. (1985) also studied the phototoxicity of pyrene in leopard frog tadpoles *(Rana pipiens)*. The 1-h LC_{50} in the presence of sunlight was 140 $\mu g \cdot L^{-1}$.

The phototoxicity of pyrene to first instar mosquito larvae *(A. aegypti)* was also examined by Kagan and Kagan (1986). Exposure to 30 $\mu g \cdot L^{-1}$ pyrene for 12 h in the absence of a UV light, followed by a further 30 min in UV light, resulted in 100% mortality of mosquitos. No adverse effects of pyrene were observed in the absence of UV radiation for 12.5 h. The LC_{50}s immediately after the irradiation and 24 h later were 12 and 9 $\mu g \cdot L^{-1}$. If the larvae were allowed to develop through to adult emergence, then the LC_{50} was 2.5 $\mu g \cdot L^{-1}$. Kagan et al. (1985) exposed *D. magna* to pyrene for 1 h under laboratory conditions. It was followed by a 30-min exposure of the organisms to UV light (13 W$\cdot m^{-2}$). The investigators reported a 90-min LC_{50} of 20 $\mu g \cdot L^{-1}$ pyrene for *D. magna*. Increasing the initial exposure time (i.e., under laboratory light) to 2 and 12 h resulted in 2.5- and 12.5-h LC_{50}s of 15 and 12 $\mu g \cdot L^{-1}$, respectively. However, doubling the time of UV light exposure from 30 min to 1 h resulted in a 2-h LC_{50} of 4 $\mu g \cdot L^{-1}$, a five-fold increase in sensitivity. Newsted and Giesy (1987) reported that the daphnids treated with the toxicant under laboratory lights for 24 h, followed by a 24-h UV light exposure, displayed a 50% mortality in 208.6 min at a pyrene concentration of 5.7 $\mu g \cdot L^{-1}$.

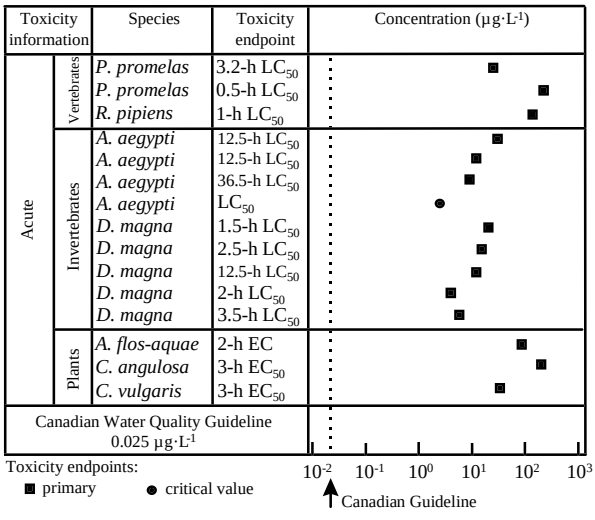

Figure 10. Select freshwater toxicity data for pyrene.

Toxicity data for freshwater algae are limited. Bastian and Toetz (1985) found that nitrogen fixation was elevated in *A. flos-aquae* following a 2-h pyrene treatment of 85 $\mu g \cdot L^{-1}$. Hutchinson et al. (1980) reported that pyrene reduced photosynthetic activity in green algae. For *C. angulosa* and *C. vulgaris*, 3-h EC_{50}s of 202 and 332 $\mu g \cdot L^{-1}$, respectively, were found.

The interim water quality guideline for pyrene for the protection of freshwater life is 0.025 $\mu g \cdot L^{-1}$. It was derived by multiplying the acute value (LC_{50}) of 2.5 $\mu g \cdot L^{-1}$ for mosquito larvae *(A. aegypti)* (Kagan and Kagan 1986) by a safety factor of 0.01 (CCME 1991). Pyrene was considered to be a persistent substance, as its half-life in water is longer than 8 weeks (SRC 1989). It is assumed that the experimental exposure to either simulated or natural sunlight can resemble the potential exposure to ultraviolet radiation in the field.

Quinoline

Black et al. (1983) and Millemann et al. (1984) conducted chronic toxicity tests with freshly fertilized rainbow trout *(O. mykiss)* and largemouth bass *(M. salmoides)* eggs exposed to quinoline until 4 d after hatching. The average hatching times for largemouth bass and rainbow trout were 3 and 23 d, respectively. Millemann et al. (1984) reported 4-d posthatch LC_{50}s of 7420 and 11 500 $\mu g \cdot L^{-1}$, respectively, for the bass and trout. Black et al. (1983) found very similar values with LC_{50}s at the time of hatching of 10 800 and >10 800 $\mu g \cdot L^{-1}$ for the trout and bass and 4-d posthatch LC_{50}s of 11 000 and 7500 $\mu g \cdot L^{-1}$, respectively. In a chronic toxicity test with quinoline, Black et al. (1993) also found that the control-adjusted survival of rainbow trout *(O. mykiss)* (4-d posthatch at the embryo–larval stages) decreased to 95% at 13 $\mu g \cdot L^{-1}$, 89% at 90 $\mu g \cdot L^{-1}$, and 82% at 370 $\mu g \cdot L^{-1}$. The geometric mean of the lowest two chronic values is calculated to be 34 $\mu g \cdot L^{-1}$. Millemann et al. (1984) reported a 96-h LC_{50} for juvenile fathead minnows *(P. promelas)* of 440 $\mu g \cdot L^{-1}$.

Exposing pond snails *(Physa gyrina)* to quinoline for 17–22 d delayed hatching at concentrations of 12 500 $\mu g \cdot L^{-1}$ and reduced embryogenesis at 25 000 $\mu g \cdot L^{-1}$ (Millemann and Ehrenberg 1982). The 48-h LC_{50} of 183 000 $\mu g \cdot L^{-1}$ for *P. gyrina* was considerably higher than the nonlethal end points (Millemann et al. 1984). Millemann et al. (1984) also reported 48-h LC_{50}s of 34 500, 40 900, and

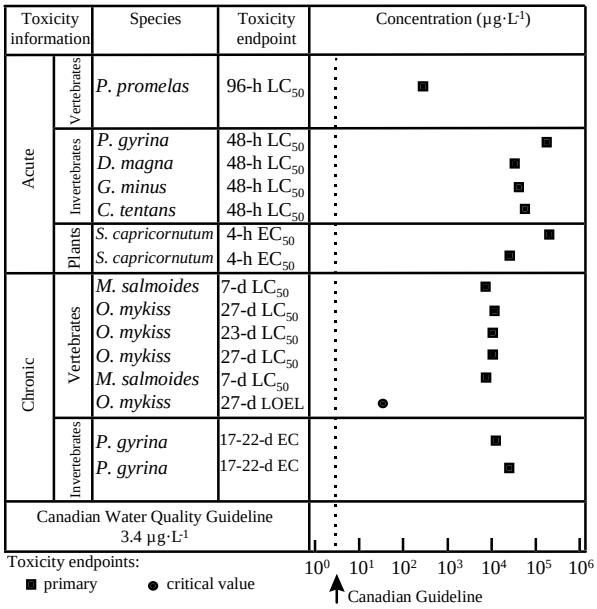

Figure 11. Select freshwater toxicity data for quinoline.

56 800 $\mu g \cdot L^{-1}$ for the water flea *(D. magna)*, scud *(Gammarus minus)*, and midge (*C. tentans*), respectively.

Millemann et al. (1984) reported a 4-h EC_{50} for reduced photosynthetic activity of 202 000 $\mu g \cdot L^{-1}$ in the green alga *S. capricornutum*. Similarly, a 4-h EC_{50}, for reduced photosynthesis in *S. capricornutum* of 25 000 $\mu g \cdot L^{-1}$ was also reported by Giddings et al. (1983).

An interim water quality guideline for quinoline for the protection of freshwater life is 3.4 $\mu g \cdot L^{-1}$. It was derived by multiplying the chronic LOEC of 34 $\mu g \cdot L^{-1}$ for rainbow trout by a safety factor of 0.1 (CCME 1991). Black et al. (1983) observed that the survival of rainbow trout (*O. mykiss*) larvae exposed to quinoline was 95% at 13 $\mu g \cdot L^{-1}$, 89% at 90 $\mu g \cdot L^{-1}$, and 82% at 370 $\mu g \cdot L^{-1}$. The chronic LOEL of 34 $\mu g \cdot L^{-1}$ is the geometric mean of 13 and 90 $\mu g \cdot L^{-1}$. In this case, the geometric mean was chosen as it was assumed to be more environmentally relevant than the lowest effect level (95% survival rate) alone.

Marine Life

Naphthalene

Moles and Rice (1983) reported a 96-h LC_{50} of 1200 $\mu g \cdot L^{-1}$ for juvenile pink salmon *(O. gorbuscha)* exposed to naphthalene. Following 40-d exposures, LOEC and NOEC values (body weight) of 380 and 120 $\mu g \cdot L^{-1}$, respectively, were reported. A 96-h LC_{50} of 1240 $\mu g \cdot L^{-1}$ for pink salmon (Korn et al. 1979) and a 24-h LC_{50} of 2400 $\mu g \cdot L^{-1}$ for the sheepshead minnow *(C. variegatus)* (Anderson et al. 1974) have been reported.

Ott et al. (1978) exposed female copepods *(Eurotemora affinis)* carrying their first egg sacs to 14.2 $\mu g \cdot L^{-1}$ of naphthalene until their deaths (29 d). Lifespan, total eggs per female, mean brood size, and rate of egg production were all significantly decreased by naphthalene treatment. Korn et al. (1979) exposed the marine shrimp *Pandalus goniurus* to naphthalene and reported 96-h LC_{50}s ranging from 971 $\mu g \cdot L^{-1}$ at 12°C to 2160 $\mu g \cdot L^{-1}$ at 4°C. The increase in temperature was thought to elevate the sensitivity of the shrimp by changing the naphthalene uptake and metabolic rate.

Thursby et al. (1985) reported a 50% reduction in growth for the red alga *Champia parvula* over an 11- to 14-d exposure at a concentration of 695 $\mu g \cdot L^{-1}$.

The interim water quality guideline for naphthalene for the protection of marine life is 1.4 $\mu g \cdot L^{-1}$. It was derived by multiplying the lowest chronic value of 14.2 $\mu g \cdot L^{-1}$ for the calanoid copepod (Ott et al. 1978) by a safety factor of 0.1 (CCME 1991).

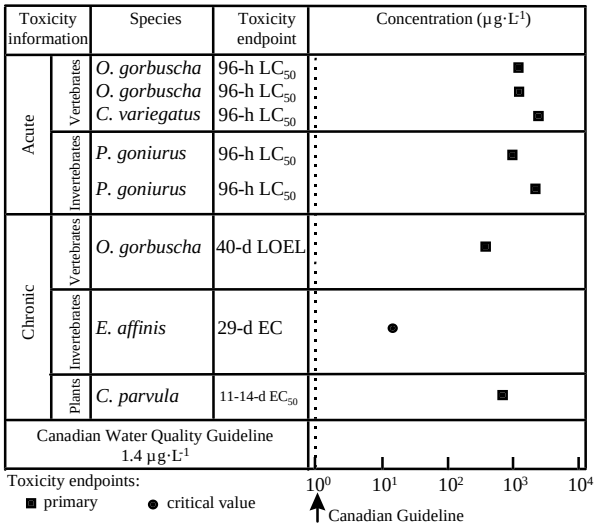

Figure 12. Select marine toxicity data for naphthalene.

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For further scientific information, contact:

Environment Canada
Guidelines and Standards Division
351 St. Joseph Blvd.
Hull, QC K1A 0H3
Phone: (819) 953-1550
Facsimile: (819) 953-0461
E-mail: ceqg-rcqe@ec.gc.ca
Internet: http://www.ec.gc.ca

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