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AMBIENT WATER QUALITY CRITERIA FOR THE PROTECTION OF HUMAN HEALTH

Acrylonitrile

DRAFT

Ambient Water Quality Criteria for the Protection of Human Health: Acrylonitrile

NOTE TO READER

The Agency is intending to develop streamlined criteria documents which focus on critical toxicological and exposure-related studies only. This is a departure from the past format in which all existing toxicological and exposure studies were presented and evaluated in the 1980 criteria documents, with equal emphasis placed on exposure, pharmacokinetics, toxicological effects, and criterion formulation. Due to limited resources and a need to update criteria as quickly as possible, EPA has decided to develop more abbreviated versions of criteria documents with an emphasis on using existing risk assessments (on IRIS or other EPA health assessment documents) where available and still relevant, and focusing to a greater extent on pertinent exposure and toxicological studies which may influence the development of a criterion (e.g., critical effects studies which form the basis of RfD development or cancer assessment). EPA will continue to conduct a comprehensive review of the literature for the latest studies, but will not provide a summary or an evaluation of those studies in the criteria documents which are deemed less significant in the criteria development process. Where there is a significant amount of literature on an area of study (for instance, pharmacokinetics), EPA, to the extent possible, will reference the information or cite existing documents (e.g., IRIS or other existing EPA risk assessment documents) which discuss the information in greater detail.

The overall objective of this change in philosophy is to allow EPA to update 1980 AWQC at a greater frequency, while still maintaining the scientific rigor which EPA requires when developing an AWQC. EPA believes these "new" criteria documents will be just as informative as previous criteria documents and will continue to serve as the key scientific basis for State and Tribal standards. EPA also believes the documents will provide the necessary scientific content and scope to allow a State or Tribe to come to an appropriate technical and/or policy decision with regard to setting water quality standards.

EPA requests that commenters identify any relevant information missing from this criteria document which may result in a different criteria calculation or scientific interpretation. EPA also requests comments on the change in criteria document format. This criteria document has undergone extensive external peer review.

1. BACKGROUND

Criteria for acrylonitrile were set in 1980 based on non-threshold carcinogenic effects (45 FR 79318). Because of these carcinogenic effects, the levels of acrylonitrile in water should ideally be zero. However, because the zero level may not be attainable, the following criteria were set based on three incremental increases of cancer risk:

Risk Level	Criterion (µg/L)	
	Ingestion of Water and Aquatic Organisms	Ingestion of Aquatic Organisms Only
10^{-5}	0.58	6.5
10^{-6}	0.058	0.65
10^{-7}	0.006	0.065

Under the National Toxics Rule (USEPA, 1992), criteria were updated based on a new cancer potency factor from IRIS. At the 10^{-6} risk level, criteria for water and organisms and organisms only were set at 0.059 and 0.66 µg/L, respectively.

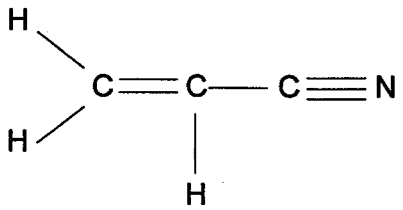
This criteria document updates national criteria for acrylonitrile using new methods and information described in the *Federal Register* notice (USEPA, 1998a) and Technical Support Document (TSD) (USEPA, 1998b) to calculate ambient water quality criteria. These new methods include updated approaches to determine toxicity dose-response relationships for both carcinogenic and non-carcinogenic effects, updated information for determining exposure factors (e.g., values for fish consumption), new exposure assumptions used in the calculation, and new procedures to determine bioaccumulation factors.

In addition to new methods for deriving AWQC values, new data on the toxicity, exposure, and bioaccumulation of acrylonitrile are also included in the criterion calculation. Based on the most sensitive end point (cancer), the proposed criterion to protect against ingestion from water and aquatic organisms is 0.055 µg/L, and the criterion to protect against ingestion of aquatic organisms and incidental water exposure is 4.0 µg/L. Both of these values are calculated based on a lifetime risk of 10^{-6}. The calculation is based on adults in the general population.

The following sections include the toxicological, exposure, and bioaccumulation factor evaluations, the calculation of the criterion, and a discussion of site-specific adjustments to the criterion.

2. CHEMICAL NAME AND FORMULA

The AWQC is being derived for acrylonitrile (CAS No. 107-13-1). The chemical formula is C_3H_3N, and the chemical structure is



Synonyms include the following: 2-propenenitrile, acrylon, carbacryl, cyanoethene, cyanoethylene, Acritet, Fumigrain, propenenitrile, VCN, Ventox, vinyl cyanide (U.S. EPA, 1983; Budavari, 1989).

Physical and chemical properties (U.S. EPA, 1983; NTP, 1998)

Chemical Formula	C_3H_3N
Molecular Weight	53.06
Physical State (25°C)	Clear colorless liquid
Boiling Point	77.3°C
Density (20°C)	0.8060 g/mL
Vapor Pressure (20°C)	80 mm Hg
Specific Gravity (20°C)	0.8060
Water Solubility (20°C)	7.35 weight %
Log Octanol Water Partition	-0.92
Conversion Factor	

3. SUMMARY OF PHARMACOKINETICS

Pharmacokinetic data were obtained from animal, but not human studies. The data obtained from animal studies show that acrylonitrile is extensively absorbed from the gastrointestinal tract. Approximately 95% was absorbed in male rats receiving a single oral dose of 0.1 or 10 mg/kg (Young et al., 1977). Acrylonitrile and its metabolites are distributed throughout the body with the highest accumulation in the gastrointestinal tract, skin, and red blood cells. These targets are maintained regardless of the route of delivery or level of exposure. Accumulation also occurs in the liver, lung, kidney, mucosa, and adrenal cortex. (Ahmed et al., 1982; Sandberg and Slanina, 1980; Silver et al., 1987; Young et al., 1977).

Acrylonitrile undergoes various biotransformation and conjugation reactions. A major product of metabolism is cyanide and many of the short-term toxic effects (acute toxicity) are

due to cyanide formation. Acrylonitrile binds to nucleic acids and proteins and conjugates with glutathione. The conjugation product is metabolized by glutathionase to form cyanoethylated mercapturic acid which is excreted in urine (Dahm, 1977; Gut et al., 1975; Hashimoto and Kanai, 1965; Langvardt et al., 1979). Alternatively, acrylonitrile is converted by the microsomal mixed function oxidase (MFO) system to 2-cyanoethylene oxide, a reactive intermediate. This is, in turn, metabolized by several pathways including covalently binding with macromolecules or conjugation with glutathione. The extent of conversion of acrylonitrile to cyanide and thiocyanate is route and species dependent (Gut et al., 1975).

A biphasic elimination dynamic has been observed in rats, indicative of a two-compartment open model. The half-lives observed were 3.5 to 5.8 hours in the faster phase and 50 to 77 hours in the slower phase (Young et al., 1977).

4. TOXICOLOGICAL BASIS FOR CRITERIA

4.1 Noncancer Data and Previous Evaluations

4.1.1 Oral Exposure

Human chronic exposure data are not available regarding oral exposure to acrylonitrile (there are human inhalation exposure data, discussed in the following section). Oral exposure effects are reported in numerous animal studies. An oral RfD was developed in 1993 by the RfD Work Group, but is not yet available on the Integrated Risk Information System (IRIS). The following summary of the oral non-carcinogenic effects reported in the literature is based on the Work Group report (USEPA, 1993).

A number of long-term animal studies have been carried out on oral exposure to acrylonitrile:

Tandon et al. (1988) studied effects on the male reproductive system in a gavage study in mice. They found degenerative changes in reproductive structures (seminiferous tubules), as well as direct effects on sperm including decreases in the number of spermatozoa. Absence of effects was observed at the lowest dose tested (1 mg/kg-day for 60 days).

Chronic exposure studies by Biodynamics (1980a and b) in male and female rats found numerous blood disorders, food and water intake, body weight, and organ-specific weight disturbances, epithelial hyperplasia and hyperkeratosis in the stomach, gliosis and perivascular cuffing of the brain, and mammary gland hyperplasia. There were problems in these studies with premature mortality, as discussed below in the cancer section.

A study of beagle dogs by Quast et al. (1975) for six months at doses of 10, 16, and 17 mg/kg-day in males and 8, 17, and 19 mg/kg-day in females found high toxicity at the

two highest dose levels with associated high mortality rates. The following effects were observed in the animals that died prematurely: lethargy, weakness, emaciation, and respiratory distress. No effects were observed at the lowest dose level.

Bigner et al. (1986) observed neurological effects in a lifetime rat study with administration via drinking water at doses of 8 and 40 mg/kg-day in males and 8 and 50 mg/kg-day in females. These effects were observed at both dose levels and brain tumors were reported in the high dose group. Mortality was also observed in both dose groups.

Beliles et al. (1980) observed rats over three generations dosed with 8 and 40 mg/kg-day in males and 8 and 50 mg/kg-day in females via drinking water. Effects observed included reduced weight gain, food intake, and water consumption at both doses, reduced pup viability and reduced lactation. Microscopic evaluation of tissues was not done.

Murray et al. (1978) evaluated developmental toxicity in rats exposed to 10, 25, and 65 mg/kg-day via gavage on days 6 through 15 of gestation. Maternal effects included hyperactivity, stomach abnormalities, reduced incidence of pregnancy, reduced body weight gain, and increased liver weights. Offspring effects included: missing vertebrae, shortened trunk, imperforate anus, and minor skeletal variants. The authors identified a LOAEL of 25 mg/kg-day. These findings are supported by an intraperitoneal study that also found skeletal abnormalities (Willhite et al., 1981).

Additional studies have suggested effects on steroidogenesis (Szabo et al., 1984), however, the implications of these observations are not clear (Szalay et al., 1987).

The RfD Work Group identified the Tandon et al. (1988) study as the principal study with a NOAEL of 1 mg/kg-day. The critical effects include seminiferous tubule degeneration and a decrease in the sperm counts. An uncertainty factor of 1000 was applied, with a factor of 10 used to account for extrapolation from mice to humans, 10 to account for sensitive human sub-populations, and 10 for the use of a sub-chronic study and data base deficiencies. These deficiencies include the lack of a chronic mouse study and the lack of adequate reproductive toxicity studies. The calculated RfD for acrylonitrile from this study is 1×10^{-3} mg/kg-day (USEPA, 1993). Confidence in the oral RfD is medium due to the small sample size of the critical study and because only two dose levels were tested.

4.1.2 Inhalation Exposure

Acrylonitrile causes central nervous system depression and respiratory irritation. Exposure has been reported to cause headaches, nausea, fatigue, and weakness in workers chronically exposed via inhalation (USEPA, 1994b). It also causes dermal irritation with skin contact (Bakker, 1991). Numerous animal inhalation studies are available for acrylonitrile; summaries of these studies are available on IRIS. A reference concentration (RfC) for inhalation exposure of 2×10^{-3} mg/m^3 was developed by EPA based upon degeneration and inflammation of

nasal respiratory epithelium and hyperplasia of mucous secreting cells. Results were obtained from a 2 year rat inhalation study (Quast et al., 1980b, as reported in IRIS), with no NOAEL and an adjusted LOAEL of 1.9 mg/m^3. The uncertainty factors applied were: 10 to protect unusually sensitive individuals, 3 to adjust from a minimally adverse LOAEL to a NOAEL, 3 for interspecies variability, and 10 due to an incomplete data base including the lack of an inhalation bioassay in a second species and lack of reproductive data by the inhalation route (with the presence of reproductive effects in an oral study). Confidence in the RfC is medium. The critical study was confounded by the early sacrifice of rats with large mammary gland tumors and limited examination of the target organ (nasal turbinates) at the end of the study. Other tumor data are also reported in the study (IRIS).

4.2 Cancer Evaluation

4.2.1 Human Data

Several epidemiological studies have reported an increased incidence of lung cancer mortality in workers exposed to acrylonitrile (Thiess et al., 1980; Werner and Carter, 1981; Delzel and Manson, 1982; O'Berg et al., 1980, 1985; Zhou and Wang, 1991 in Rothman, 1994), but these excesses were not observed in other studies (Chen et al. 1987; Collins et al., 1989; Swaen et al., 1992). Most of the available studies have limitations, such as exposure levels not measured, exposure to other carcinogens, and/or lack of information on confounding factors such as cigarette smoking. In addition, increased incidence of prostate cancer were reported in several cohorts (O'Berg et al., 1985; Collins et al., 1989; Chen et al., 1987; Swaen et al., 1992). A comprehensive review of the earlier health-related studies is available in a document entitled "Health Assessment Document for Acrylonitrile" (USEPA, 1983).

4.2.1.1 Oral Exposure

Human data are not available regarding chronic oral exposure to acrylonitrile.

4.2.1.2 Inhalation Exposure

Cancers observed in humans, associated primarily with inhalation exposure, included lung cancer. The O'Berg study (O'Berg, 1980) was used as the basis for the estimation of the inhalation cancer potency in IRIS. In this study, 1345 male textile workers exposed to 5 to 20 ppm (0.011 to 0.04 μg/m^3) acrylonitrile for an average of 9 years were followed for 10 or more years. A maximum age at the end of the observation period of 60 years was assumed to calculate an age-adjusted standardized mortality ratio in the cohort of 113. An exposure response trend was observed with increased duration of exposure and follow-up associated with increased cancer incidence. A statistically significant increase in respiratory cancer was observed and the analysis included controls for the contribution to cancer from smoking. Later, an update of the study is reported (O'Berg et al., 1985). Thus, the IRIS information needs to be updated as well.

Other studies have also found statistically significant associations between respiratory cancer and acrylonitrile (Delzell and Monson, 1982; Thiess et al., 1980; Werner and Carter, 1981; Monson, 1978). These studies have problems associated with exposure quantification and/or confounding exposures such as cigarette smoking and as a result are not appropriate for use in dose-response analysis.

4.2.2. Animal Data

There is substantial evidence that acrylonitrile causes multiple tumor types following oral and inhalation exposure to several strains of rats. Details are not provided here on animal inhalation exposure studies, because human data are available on inhalation exposure, and because the AWQC derivation focuses on the oral route rather than inhalation.

4.2.2.1 Oral Exposure

Animal tumors induced via oral exposure to acrylonitrile included stomach, tongue, small intestine, mammary gland, and Zymbal gland (ear canal), and astrocytomas (the CNS) (Maltoni et al., 1977; Biodynamics, 1980a; Biodynamics, 1980b; Quast et al., 1980a; Quast et al., 1980a; Bigner et al., 1986). The study by Quast et al., (1980a) which provides data from a lifetime study with large group sizes, is most applicable in this evaluation. In the study, acrylonitrile was administered in drinking water at doses of 35, 100, and 300 ppm (3.42, 8.53, and 21.18 mg/kg-day respectively) to 48 served as controls. A statistically significant increase in tumors was observed in the CNS (astrocytomas), ear canal (Zymbal gland), stomach, tongue, and small intestine of both male and female animals and in the mammary gland of females. In general, the increase was dose-dependent.

The tumor data from the Quast et al. (1980a) study are listed in Table 4.2.1 with the estimated equivalent human doses. The human equivalent dose was calculated two ways: using the new proposed approach of scaling dose in proportion to body weight raised to the 3/4 power and the current approach of scaling dose in proportion to body weight raised to the 2/3 power. The tumor incidence indicates the number of animals bearing tumors that were statistically significant at any site divided by the total number of animals for the dose group.

Exposure and follow-up periods were less than a lifetime for some animals in Biodynamics (1980 a and b) and for all animals in Maltoni (1977). In the case of the Biodynamics studies, interim sacrifices were carried out on thirty percent of the animals. Premature mortality was also observed. It should be noted that the cancer potency factors obtained from Biodynamics (1980a,b) were two times smaller than the factor obtained by Quast et al. (1980a). The Maltoni et al. (1977) study was planned as a one-year study. In all these studies, the shortened study duration (for some animals in the Biodynamics studies and all animals in the Maltoni study) does not provide optimal information for determining the total lifetime tumor risk by acrylonitrile. The Bigner et al. (1986) study does not provide clear dose-response data.

Table 4.2.1: Human Equivalent Doses and Combined Tumor Incidence of a Lifetime Drinking Water Study*.

Administered Dose (mg/kg-day)	Human Equivalent Dose using Body Weight$^{3/4}$ Scaling (mg/kg-day)	Human Equivalent Dose using Body Weight$^{2/3}$ Scaling (mg/kg-day)	Tumor Incidence
0.00	0.00	0.00	4/80
3.42	0.90	0.58	18/47
8.53	2.27	1.46	36/48
21.18	5.63	3.62	45/48

* The Administered dose and the Combined Tumor Incidence data are from Quast et al. (1980a); the human equivalent doses are computed (For detail, See the Draft FR and the Technical Support Document in USEPA, 1998a,b).

4.2.3 Other Information Relevant to the Cancer Evaluation

4.2.3.1 Mutagenicity

Mutagenic studies of acrylonitrile are generally positive. Acrylonitrile causes mutations in both *Salmonella typhimurium* (Venitt et al., 1977) and *Escherichia coli* (De Meester et al., 1978). It has been shown to bind to DNA (Guengerich et al., 1981). It did induce an increase in sister-chromatid exchange in CHO cells (Ved Brat and Williams, 1982). Acrylonitrile has been shown to transform Syrian hamster embryo cells and to enhance transformation of these cells infected with an oncogenic virus (Parent and Casto, 1979). On the other hand, a study has reported negative results in *Salmonella typhimurium* (Ashby et al., 1985). In an *in vivo* test, DNA adducts were identified in rat liver but not in rat brain (Hogy and Guengerich, 1986). Acrylonitrile did not cause chromosomal aberrations in bone marrow cells of rats and mice (Rabello-Gay and Ahmed, 1980; Leonard et al., 1981), nor in peripheral blood lymphocytes of exposed workers (Thiess and Fleig, 1978; IRIS, 1996).

4.2.3.2 Summary of Cancer Data on Structural Analogue and Metabolite

Like vinyl chloride, a metabolite of acrylonitrile, 2,3-epoxy-propionitrile, is mutagenic in *Salmonella* (Kier, 1982).

4.2.3.3 Mode of Action

The current scientific consensus is that there is virtually complete correspondence between the ability of an agent to have a direct DNA activity and carcinogenicity. The data on short-term studies as a whole support a mutagenic mode of action. Based on this assumption and a lack of information supporting a non-linear approach for this chemical, a default low dose linearity is assumed. (See further discussion of the use of mode of action data in USEPA, 1996 and USEPA, 1998a,b).

4.2.4 Previous Cancer Risk Evaluation

Acrylonitrile has been classified as a B1 carcinogen (a probable human carcinogen) based on the observation of a statistically significant increase in incidence of lung cancer in exposed workers and observation of tumors, generally astrocytomas in the brain, in studies in two strains of rats exposed by various routes (drinking water, gavage, and inhalation) (IRIS, 1996).

4.2.4.1 Oral Exposure

The oral cancer potency, entered into IRIS in 1987, was calculated by taking the geometric mean of the individual cancer potencies obtained from three drinking water studies: Biodynamics, 1980a; Biodynamics, 1980b, and Quast et al., 1980a. The overall risk was determined from the number of animals having tumors that were statistically significant at any site (i.e., the pooled tumor incidence of brain and spinal cord astrocytomas, Zymbal gland carcinomas and stomach papillomas/carcinomas). The human equivalent dose was calculated using the scaling approach of body weight raised to the 2/3 power (shown in Table 4.2.1). The linearized multistage model was used to calculate the individual cancer potency factors for each study. The geometric mean was calculated by taking the cube root of the product of the three cancer potency factors obtained from the these studies. The resulting factor, reported in IRIS, is 5.4×10^{-1} $(mg/kg\text{-}day)^{-1}$. The approach used in the IRIS file is based on the 1986 Cancer Guidelines (USEPA, 1986).

4.2.4.2 Inhalation Exposure

The unit risk was calculated from an average relative risk model adjusted for smoking and based on a continuous lifetime equivalent of occupational exposure using information from O'Berg (1980) as discussed in section 4.2.1.2 above. The unit risk obtained was 6.8×10^{-5} $(\mu g/m^3)^{-1}$ (IRIS, 1996).

4.2.5 Cancer Risk Evaluation Using the New Proposed Methodology

The proposed revision of the methodology for deriving ambient water quality criteria is consistent with the principles of the proposed cancer guidelines to evaluate/describe the carcinogenicity of chemicals (as described in USEPA, 1996, and USEPA, 1998a,b).

Based on sufficient evidence from animal studies (multiple tumor types in several strains of rats by several routes) and limited evidence from human studies (lung tumor in workers), positive mutagenicity, acrylonitrile is considered as a likely human carcinogen by any route. A linear approach is used for the low dose extrapolation.

4.2.5.1 Rationale for Selecting the Cancer Assessment Approach

In the new scheme of cancer classification, acrylonitrile is considered a likely human carcinogen based on sufficient evidence in animal studies and limited evidence in human studies. See the Proposed Cancer Guidelines (USEPA, 1996) for additional information on the classification scheme. Based on the mutagenic mode of action of acrylonitrile, and the lack of information that would support using a non-linear approach to this chemical, a linear low dose approach is used.

4.2.5.2 Calculation of the Cancer Potency Factor Using the New Linear Method[1]

The cancer potency factor for oral exposure to acrylonitrile was determined by following the steps outlined in the FR notice (USEPA, 1998a) and the related Technical Support Document (USEPA, 1998b). The Quast et al. (1980a) study, which provides data from a lifetime drinking water study in rats with large group sizes, is used for the calculation of the slope factor. The Biodynamics (1980a,b) studies are not included in the calculation because of interim sacrifice which would underestimate the risk (see section 4.2.2 under *Oral Exposure* and section 4.2.6 under *Discussion of Confidence* for more detail). The following steps are carried out in the calculations:

1) The quantal polynomial model (using Global 86 multistage model software) was used to model the Quast et al. (1980) dose response (tumor) data in the observed range using $BW^{3/4}$ as the dose scaling factor (see Table 4.2.1 for detail). The LED_{10}[2] (the lower 95th percent

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[1]This section contains a discussion of the derivation of a cancer potency value based on oral exposure to acrylonitrile. The focus of this criterion document is on waterborne exposure and the development of AWQC. While the contribution to cancer risk from air sources may be important, it is not the primary subject of this analysis. Consequently, the inhalation cancer potency value listed in IRIS, and described above, is not re-examined in this analysis.

[2]Use of the LED_{10} as the point of departure is recommended with this methodology, as it is with the Proposed Cancer Guidelines. Public comments were requested on the use of the LED_{10}, ED_{10}, or other points. EPA is currently evaluating these comments, and any changes in the Cancer Guidelines will be reflected in the final AWQC Methodology.

confidence limit on the dose at which 10 percent of the animals responded in significantly elevated tumor incidence) was calculated to be 0.16 mg/kg-day.

2) linear extrapolation was carried out from the LED_{10} to the origin (the zero dose, zero response point). The slope of this line, *m* (i.e., $\Delta y/\Delta x$), was estimated using the following

$$m = \frac{0.10}{LED_{10}}$$

(Equation 4.2.1)

The variable "*m*" is the low dose cancer potency value and was calculated to be 6.3×10^{-1} $(mg/kg\text{-}day)^{-1}$.

3) The risk specific dose (RSD) was calculated for the specific targeted lifetime cancer risk (i.e., 10^{-4}, 10^{-5} or 10^{-6}), using the equation:

$$RSD = \frac{Target\ Incremental\ Cancer\ Risk}{m}$$

(Equation 4.2.2)

where:

RSD	=	risk specific dose (mg/kg-day),
Target Risk	=	10^{-6} (lifetime incremental risk), and
m	=	cancer potency factor of 6.3×10^{-1} $(mg/kg\text{-}day)^{-1}$

The calculated RSD is 1.6×10^{-6} mg/kg-day for a 10^{-6} lifetime cancer risk. When the RSD (1.6×10^{-6}) is substituted into Equation 7.1.1. in Section 7.1, an AWQC of 0.055 µg/L or 4.0 µg/L is calculated for ingestion of drinking water and aquatic organisms, or ingestion of aquatic organisms alone (including incidental water ingestion from recreational activities), respectively, for a lifetime risk of 10^{-6}.

4.2.6 Discussion of Confidence

A large number of animals were evaluated in the Quast et al. (1980) study that serves as the basis for the oral exposure cancer evaluation. This increases confidence in the values

obtained. However, there are no human data regarding oral intake of acrylonitrile. Human studies with exposure via the oral route could provide greater certainty regarding the carcinogenicity and carcinogenic potency of acrylonitrile. Balancing this, the human evidence, based on inhalation studies in workers, indicates that acrylonitrile is carcinogenic via that route, causing lung cancers. The animal studies are also persuasive regarding the carcinogenic potential of acrylonitrile in humans, with multiple tumor types observed in multiple studies.

A problem with the Biodynamics (1980a,b) studies is related to the use of interim necropsy results at 6, 12, and 18 months. The results from these animals were included with the final sacrifice values and they are counted in the overall incidence denominator, even though they were not followed for the full lifespan. This leads to an underestimate of incidence and risk. The Quast et al. (1980a) study, which was not terminated prematurely, obtained a higher incidence of tumors (e.g., 75% tumor response at 8 mg/kg-day versus 34% at the same dose in Biodynamics (1980b). In addition, there were problems with premature mortality in both Biodynamics (1980a,b) studies. As a result of the deficiencies in the Biodynamics studies, it was determined that the Quast et al. (1980a) study should serve as the single source of dose-response data for the cancer potency estimate and AWQC calculation.

5. EXPOSURE ASSUMPTIONS

5.1 RSC Analysis

When an ambient water quality criterion is set based on non-carcinogenic effects, or carcinogenic effects evaluated by the margin of exposure (MOE) approach, anticipated exposures from non-occupational sources (e.g., food, air) are taken into account. The amount of exposure attributed to each source compared to total exposure is called the relative source contribution (RSC) for that source. The allowable dose (typically, the RfD) is then allocated via the RSC approach to ensure that the criterion is protective enough, given the other anticipated sources of exposure. Thus, accounting for non-water exposure sources results in a more stringent ambient water quality criterion than if these sources were not considered. The method of accounting for non-water exposure sources is described in more detail in the Federal Register Notice (USEPA, 1998a) and in the Technical Support Document (USEPA, 1998b). Available information on exposure sources is discussed below. However, because the criterion is based on the linear approach used to assess carcinogenicity, the information is not used to determine an RSC for acrylonitrile.

5.1.1 Overview of Potential for Exposure

Acrylonitrile occurrence in environmental media is not well-documented. Several regional and local drinking water surveys were found and one limited study analyzed ambient air samples. Limited information is also available on acrylonitrile migration into foods from packaging materials. All of these studies are described in section 5.1.2 below.

Acrylonitrile is largely used in the manufacture of copolymers for the production of acrylic and modacrylic fibers. Other major uses include the manufacture of acrylonitrile-butadiene-styrene (ABS) and styrene acrylonitrile (SAN) (used in production of plastics), and nitrile elastomers and latexes. It is also used in the synthesis of antioxidants, pharmaceuticals, dyes, and surface-active agents. Table 5.1.1 lists location of plants where acrylonitrile is manufactured and the types of products made.

According to the U.S. Environmental Protection Agency's (EPA) Toxic Release Inventory, the total release of acrylonitrile into the environment in 1990 by manufacturers was 8,077,470 pounds. The two largest pathways of release were underground injection, which accounted for 61% (or 4,925,276 pounds) of the total release, and emissions into the air, which accounted for 39% (or 3,148,049 pounds) of the total release. Release of acrylonitrile into water bodies was reported at 3,877 pounds and release onto land was reported at 268 pounds.

5.1.2 Occurrence in Environmental Media

The following sections describe studies that measured concentrations of acrylonitrile in environmental media.

5.1.2.1 Exposure from Public Drinking Water Systems

Acrylonitrile samples were taken from drinking water supplies in three different surveys, two of which analyzed samples from groundwater supplies. In the early 1980's, 1,174 community wells and 617 private wells in Wisconsin were screened for 98 volatile organic chemicals (VOCs), including acrylonitrile. Wells chosen were either located near known or potential sources of VOC contamination or in areas of soil and geologic formation that would permit transport of contaminants to the ground water. There were no detections of acrylonitrile; minimum reporting limits ranged from 0.1 to 3.0 µg/L (Krill and Sonzogni, 1986). In a more recent survey, Ellingson and Redding (1988) reportedly sampled 40 randomly selected public supply wells throughout Arizona for a variety of contaminants. Samples were collected from July to September 1986 and wells were then re-sampled in January of 1987. Acrylonitrile was not detected above the detection limit of 8.1 µg/L.

Table 5.1.1: Acrylonitrile Manufacturing Locations and Products

Company	Manufacturing Site	Product
American Cyanamid	New Orleans, LA	Acrylonitrile
	Linden, NJ	Acrylonitrile
American Cyanamid	Avondale, LA	Acrylonitrile
Borg-Warner	Washington, WV	ABS, SAN resins
BP Chemicals	Lima, OH	Acrylonitrile
BP Chemicals	Green Lake, TX	Acrylonitrile
Copolymer Chemical & Rubber	Baton Rouge, LA	Nitrile rubber
DuPont	Lugoff, SC	Acrylic fibers
DuPont	Waynesboro, VA	Acrylic, modacrylic fibers
DuPont	Beaumont, TX	Acrylonitrile
Goodrich	Louisville, KY	Nitrile elastomers, ABS,
Goodyear	Houston, TX	Nitrile latex
Goodyear	Houston, TX	Nitrile rubber
Ketema	Odenton, MD	ABS monofilament
Miles, Inc.	Orange, TX	Nitrile rubber
Monsanto	Alvin, TX	Acrylonitrile
Monsanto	Texas City, TX	Acrylonitrile
Monsanto	Decatur, AL	Acrylic, modacrylic fibers
Monsanto	Addyston, OH	ABS,SAN resins
Uniroyal	Painesville, OH	Nitrile elastomers
Uniroyal	Painesville, OH	Nitrile rubber
Vistron	Lima, OH	Acrylonitrile, Acrylamide
Zeon Chemicals	Louisville, KY	Nitrile rubber
Zeon Chemicals	Pasadena, TX	Nitrile rubber

Source: SRI 1992; Going et al., 1978

The third study analyzed samples from surface water supplies. In a study of publicly-owned treatment works (POTWs) in the R.M. Clayton sewage district near Atlanta, Georgia, tap water samples were taken. Acrylonitrile was not detected in six daily composite samples based on a reporting limit of 1 μg/L (Levins et al., 1979).

In two ambient water studies, samples were taken and analyzed for acrylonitrile. The first collected samples were from the Potomac River at Quantico, Virginia. Acrylonitrile was not found based on a detection limit of 10 μg/L (Hall et al., 1987). The second study examined levels of acrylonitrile in surface water around eleven industrial sites that manufactured acrylonitrile, acrylamide, acrylic and modacrylic fibers, ABS and SAN resin, and nitrile elastomers. If possible, the liquid wastes of the manufacturing facilities were sampled. Otherwise, the water was sampled at any observable uprising in the water body. The detection limits ranged from 0.1 to 1.3 μg/L . The results for 30 samples varied from <0.1 to 4,300 μg/L as seen in Table 5.1.2. The two high values, 3,500 and 4,300 μg/L, were samples of the effluent from the manufacturing facilities (Going et al., 1978).

STORET, operated by the EPA, is a computerized data base comprised of water quality data collected from states, EPA Regional offices, and other government agencies. It contains over 130 million observations for over 700,000 sampling sites throughout the United States. It is important to note that there are limitations in using STORET data to estimate representative concentrations of contaminants in public water systems. The data in STORET were collected from an array of studies conducted for various purposes. Analyses were conducted in different laboratories employing different methodologies with a range of detection limits. In many cases the detection limits were not reported. A search of the data base located no positive detections of acrylonitrile in ambient water (USEPA, 1992b).

Table 5.1.2: Acrylonitrile Sampling in Ambient Water (Surface) for 11 Industrial Sites

Manufacturing Site	Product	Acrylonitrile (Surface Water)	
		No. of Samples	Range (ug/L)
American Cyanamid New Orleans, LA	Acrylonitrile	2	<0.1
American Cyanamid Linden, NJ	Acrylamide	1	0.8
Monsanto Texas City, TX	Acrylonitrile	3	<0.1-0.4
Monsanto Decatur, AL	Acrylic, modacrylic fibers	4	<0.1-3,500
DuPont Lugoff, SC	Acrylic fibers	4	<0.1-19.7
DuPont Waynesboro, VA	Acrylic, modacrylic fibers	3	<1.3
Borg-Warner Washington, WV	ABS, SAN resins	2	1.4-1.9
Goodrich Louisville, KY	Nitrile elastomers, ABS, SAN resins	2	<1.4-2.0
Monsanto Addyston, OH	ABS, SAN resins	2	<1.4-8.0
Uniroyal Painesville, OH	Nitrile elastomers	3	9.3-4,300
Vistron Lima, OH	Acrylonitrile, Acrylamide	4	<1.3

Source: Going et al., 1978

5.1.2.2 Dietary and Fish Exposures

Acrylonitrile can migrate from food packaging into foods. The Chemistry Review Branch (CRB) of the Department of Health and Human Services reviewed information submitted by the Society of the Plastics Industry, Inc. (SPI) who modeled the migration of acrylonitrile into foods for all currently regulated uses of acrylonitrile copolymers (DHHS, 1996).

Information submitted included diffusion of acrylonitrile from a refrigerator into air and subsequently into food, and from plastic dishes, bottles, and other containers into foods. From these migrations, SPI calculated daily intakes. Based on a CRB review of these intakes, a revised estimate of 20 μg/person/day was determined to be the dietary intake. This estimate is based on reasonably conservative assumptions about modeling the migration into the food.

Information on concentrations of acrylonitrile in fish tissues was not found.

5.1.2.3 Respiratory Exposures

Going et al. (1978) collected air samples from 11 industrial sites, chosen based on the high probability that there would be sources of acrylonitrile emissions. As part of this study, a total of 104 samples (24-hour composites) were collected from the perimeter of each site. Concentrations reportedly ranged from < 0.1 to 325 $\mu g/m^3$. The limit of detection for the analytical method was estimated to be 0.3 $\mu g/m^3$; however, lower limits were achieved during the study.

One of 16 studies included in a screening analysis of Urban Area Sources monitored for ambient air concentrations of acrylonitrile. The study, conducted in Houston and Southeast Texas, reported acrylonitrile concentrations at a minimum detection level of 2.0 $\mu g/m^3$ at 12 monitoring sites (USEPA, 1994a).

5.2 Exposure Data Adequacy and Estimate Uncertainties

As stated in the overview of exposure, environmental sampling for acrylonitrile is not well-documented. The few studies that sampled public drinking water supplies indicated that acrylonitrile was not present at levels above detection limits. Similarly, in one ambient water study, acrylonitrile was not detected, although the detection limit was relatively high (10 μg/L). However, in surface waters near sites that manufacture acrylonitrile, levels were found and some had very high concentrations. In general, based on rather limited data, it appears that acrylonitrile is not a commonly occurring contaminant at significant levels in drinking water supplies or ambient waters. However, more monitoring studies are needed to decrease the uncertainty associated with this possible exposure route given the large amount of releases by underground injection and into water bodies. The amount of information on acrylonitrile in either fish or other dietary foods is more limited than that for the water medium. The only estimate available is from the Society of the Plastics Industry, Inc., which indicates a conservative daily intake of 20 μg/person/day. More information is needed to adequately assess the potential for exposure to acrylonitrile from the diet. Additionally, the amount of data on concentrations in ambient air is limited. Two available studies indicate the potential for exposure from air near industrial sites and from two urban air locations. The latter study could represent levels that persons living in urban areas may be exposed to. However, more information is necessary to adequately assess the likelihood and potential range of exposure to acrylonitrile from ambient air, especially given the large amount of emissions into the air.

The exposure parameters used for national criteria for acrylonitrile reflect exposures for the general adult population. Sufficient information on the toxicological susceptibility of specific high risk populations (i.e., pregnant women and children) is not available for acrylonitrile. In addition, it is not clear whether a particular population is likely to be more highly exposed than another population. Although infants and children have a higher rate of water and food consumption per body weight compared to adults (USEPA, 1994c), the cancer estimates are based on lifetime exposures and, therefore, the criterion for acrylonitrile is evaluated using exposure factors applicable to adults, because individuals are adults over most of the course of a lifetime. Also, although certain water bodies may support populations of sport fishers and subsistence fishers who eat more fish than the general population, these national criteria are derived to protect the majority of the general population.

5.3 Exposure Intake Parameters

Exposure parameters (e.g., fish intake, drinking water intake, and body weight) used in the Ambient Water Quality Criterion equation should reflect the population to be protected. Default exposure parameters are available for the general population of adults as well as several specific populations that may be highly exposed or more toxicologically susceptible to a given chemical. A full discussion of these exposure parameters is included in the Federal Register notice (USEPA, 1998a) and Technical Support Document (USEPA, 1998b).

The exposure parameters and values for the general population of adults are as follows:

Fish intake (FI)	0.01780 kg/day
Drinking water intake (DI)	2 L/day (used for drinking water sources)
Incidental ingestion (II)	0.01 L/day (used for non-drinking water sources)
Body Weight (BW)	70 kg

6. BIOACCUMULATION FACTORS

This section describes the procedures and data sources used to calculate the bioaccumulation factor (BAF) used for deriving an ambient water quality criterion for acrylonitrile. Details and the scientific basis of U.S. EPA's recommended methodology for deriving BAFs are described in USEPA (1998a) and USEPA (1998b). When determining a BAF for use in deriving ambient water quality criteria (AWQC) for nonpolar organic chemicals, two general steps are required. The first step consists of calculating baseline BAFs for organisms at appropriate trophic levels using available data from field and laboratory studies of the bioaccumulation or bioconcentration of the chemical of interest. Since baseline BAFs are normalized by important factors shown to affect bioaccumulation (e.g., the lipid content of aquatic organisms on which they are based, the freely dissolved concentration of the chemical in water), they are more universally applied to different sites compared to BAFs not adjusted for

these factors. Once baseline BAFs have been calculated for the appropriate trophic levels, the second step involves adjusting the baseline BAFs to reflect the expected conditions at the sites that are applicable to the AWQC (e.g., lipid content of consumed organisms and the freely dissolved fraction of the chemical in the site water). Application of both of these steps to the derivation of a BAF for acrylonitrile is described below:

6.1 Baseline BAF

Different procedures are recommended by EPA for determining the baseline BAF depending on the type of bioaccumulation data available. As described in USEPA (1998b), the data preference for deriving a BAF for non-polar organics is (in order of preference):

1. Calculation of a baseline BAF from a reliable field-measured BAF,
2. Calculation of a baseline BAF from a reliable field-measured biota-sediment accumulation factor (BSAF),
3. Calculation of a baseline BAF from a laboratory-measured bioconcentration factor (BCF) and food-chain multiplier (FCM), and
4. Calculation of a baseline BAF from a predicted BCF and FCM.

For acrylonitrile, no acceptable measured BAF, BSAF, or BCF was found. Given the low K_{ow} for this chemical, lack of field bioaccumulation data is not unexpected. Therefore, method 4 above was chosen for determining the baseline BAF. This method is described further in USEPA (1998b). According to this method, the baseline BAF is determined for each trophic level as:

$$\text{Baseline } BAF_{\ell}^{fd} = (BCF_{\ell}^{fd}) \cdot (FCM) = (K_{ow}) \cdot (FCM)$$

(Equation 6.1.1)

where:

Baseline BAF_{ℓ}^{fd}	=	predicted baseline BAF (L/kg-lipid) that if measured, would reflect the lipid-normalized concentration in the biota divided by the freely-dissolved concentration in the water for aquatic organisms occupying a designated trophic level,
BCF_{ℓ}^{fd}	=	baseline BCF expressed on a freely-dissolved and lipid-normalized basis
FCM	=	food-chain multiplier reflecting biomagnification at the designated trophic level (unitless), and
K_{ow}	=	octanol-water partition coefficient.

Fish consumption rates determined from the USDA's Continuing Survey of Food Intakes by Individuals (CSFII) indicate that on a national, average per capita basis, individuals in the United States consume significant quantities of fish and shellfish at trophic levels two (e.g., clams, oysters), three (e.g., crab, shrimp, flounder) and four (e.g., trout, pike, certain catfish species) (USEPA, 1998c). Therefore, the national AWQC for acrylonitrile requires that baseline BAFs be derived to reflect bioaccumulation in aquatic organisms at each of these three trophic levels.

For acrylonitrile, a baseline BAF of 1.5 was calculated for organisms at trophic levels two, three and four using Equation 6.1.1. These calculations are shown below for each trophic level.

Trophic Level Two:

$$\text{Baseline } BAF_{\ell}^{fd} = (K_{ow})\bullet(FCM2)$$
$$= (10^{0.17})\bullet(1.000)$$
$$= 1.5 \text{ L/kg-lipid (expressed as 2 significant digits for convenience)}$$

Trophic Level Three:

$$\text{Baseline } BAF_{\ell}^{fd} = (K_{ow})\bullet(FCM3)$$
$$= (10^{0.17})\bullet(1.000)$$
$$= 1.5 \text{ L/kg-lipid (expressed as 2 significant digits for convenience)}$$

Trophic Level Four:

$$\text{Baseline } BAF_{\ell}^{fd} = (K_{ow})\bullet(FCM4)$$
$$= (10^{0.17})\bullet(1.000)$$
$$= 1.5 \text{ L/kg-lipid (expressed as 2 significant digits for convenience)}$$

The calculated baseline BAFs do not differ at each trophic level because the relatively low K_{ow} of acrylonitrile (1.5 or $\log_{10} K_{ow}$ of 0.17) results in predicted FCMs of 1.000 at each trophic level. A log K_{ow} of 0.17 was selected as a typical value for acrylonitrile based on the mean of two measured log K_{ow} values (0.09 from Tanii and Hashimoto, 1984; and 0.25 from Pratesi et al., 1979). Values of 1.000 were selected as the FCMs at trophic levels two, three, and four according to FCMs recommended in the Technical Support Document (TSD) for the AWQC Methodology Revisions for organic chemicals with a log K_{ow} of 2.0 or less (USEPA, 1998b).

6.2 AWQC BAF

After the derivation of trophic level-specific baseline BAFs for acrylonitrile (described in the previous section), the next step is to calculate BAFs that will be used in the derivation of AWQC. This step is necessary to adjust the baseline BAFs to conditions that are expected to affect the bioavailability of acrylonitrile at the sites applicable to the AWQC. Derivation of the AWQC BAF requires information on: (1) the baseline BAF at appropriate trophic levels, (2) the

percent lipid of the aquatic organisms consumed by humans at the site(s) of interest (trophic level specific), and (3) the freely dissolved fraction of the chemical in ambient water at the site(s) of interest. For each trophic level, the equation for deriving a BAF to used in deriving AWQC is:

$$\text{BAF for AWQC}_{(TL\ n)} = [(\text{Baseline BAF}_{\ell}^{fd})_{TL\ n} \cdot (f_{\ell})_{TL\ n} + 1] \cdot (f_{fd})$$

(Equation 6.2.1)

where:

$\text{BAF for AWQC}_{(TL\ n)}$	=	BAF at trophic level "n" used to derive AWQC based on site conditions for lipid content of consumed aquatic organisms for trophic level "n" and the freely dissolved fraction in the site water
$\text{Baseline BAF}_{\ell}^{fd}{}_{(TL\ n)}$	=	BAF expressed on a freely dissolved and lipid-normalized basis for trophic level "n"
$f_{\ell\ (TL\ n)}$	=	Fraction lipid of aquatic species consumed at trophic level "n"
f_{fd}	=	Fraction of the total chemical in water that is freely dissolved

Each of the equation components is discussed below.

6.2.1 Baseline BAFs (Baseline BAF_{ℓ}^{fd})

The derivation of baseline BAFs at specific trophic levels is described in Section 6.1. For acrylonitrile, a baseline BAF of 1.5 was derived for aquatic organisms at trophic levels two, three and four.

6.2.2 Lipid Content of Consumed Aquatic Species (f_{ℓ})

Accumulation of nonpolar organic chemicals in aquatic organisms has repeatedly been shown to be a function of lipid content (e.g., Mackay, 1982; Connolly and Pedersen, 1988; Thomann, 1989). Therefore, baseline BAFs, which are lipid normalized for comparative purposes, need to be adjusted to reflect the lipid content of aquatic organisms consumed by the target population. As discussed in USEPA (1998a, 1998b), EPA recommends that where possible, lipid content of consumed aquatic species be determined on a consumption-weighted average basis.

For the purposes of deriving national ambient water quality criteria, EPA has established national default, consumption-weighted lipid content values of 2.3% at trophic level two, 1.5% at trophic level three, and 3.1% at trophic level four. These national default lipid content values are based on a national survey of fish and shellfish consumption rates and information on their lipid

content (see USEPA (1998a, 1998b) for details of the determination of national default lipid content values). As discussed in USEPA (1998a, 1998b), EPA considers the use of national default lipid values as being appropriate in situations where local or regional data on lipid content and consumption rates are unavailable for the site(s) applicable to the AWQC. However, if local or regional data are available for the site(s) of interest, EPA recommends that States and Tribes use the local or regional data instead of the national default values because the type and quantity of consumed aquatic organisms and their lipid content may vary from one location to another.

6.2.3 Freely-Dissolved Fraction Applicable to AWQC

Information on the freely-dissolved fraction of the chemical expect at the site(s) applicable to the AWQC is important because the freely dissolved form of nonpolar organic chemicals is considered to represent the most bioavailable form in water and thus, the form that best predicts bioaccumulation (USEPA 1998a, 1998b). Freely dissolved chemical is defined as the portion of the chemical dissolved in water, excluding the portion sorbed on to particulate and dissolved organic carbon. The freely-dissolved fraction is estimated from the octanol-water partition coefficient and the dissolved and particulate organic carbon concentrations as shown below.

$$f_{fd} = \frac{1}{[1 + (POC \cdot K_{ow}) + (DOC \cdot \frac{K_{ow}}{10})]}$$

(Equation 6.2.2)

where:

f_{fd}	=	freely-dissolved fraction of chemical in water applicable to the AWQC
POC	=	concentration of particulate organic carbon applicable to the AWQC (kg/L)
DOC	=	concentration of dissolved organic carbon applicable to the AWQC (kg/L)
K_{ow}	=	n-octanol water partition coefficient for the chemical

In this equation, the terms "K_{ow}" and "$K_{ow}/10$" are used to estimate the partition coefficients to POC and DOC, respectively, which have units of L/kg, the scientific basis of which is explained in USEPA (1998b). Based on national default values of 2.9 mg/L for DOC, 0.48 mg/L for POC, and 1.5 for the K_{ow} (Log K_{ow} of 0.17), the freely dissolved concentration of acrylonitrile is calculated to be 1.000 (expressed as four significant digits for convenience). Calculation of the default freely dissolved concentration is provided below.

$$f_{fd} = \frac{1}{[1 + (POC \cdot K_{ow}) + (DOC \cdot \frac{K_{ow}}{10})]}$$

$$f_{fd} = \frac{1}{[1 + (4.8 \times 10^{-7}\ kg/L \cdot 1.5\ L/kg) + (2.9 \times 10^{-6}\ kg/L \cdot \frac{1.5}{10}\ L/kg)]}$$

$f_{fd} = 1.000$

The national default values for POC and DOC used here are based on the median value of POC and DOC concentrations observed in numerous water bodies across the United States and are described further in USEPA (1998a,b). For the purposes of deriving national AWQC, EPA believes that the use of national default values is appropriate. In addition, EPA considers the use of national default values of POC and DOC as being appropriate in situations where local or regional data on POC and DOC are unavailable for the site(s) applicable to the AWQC. However, if local or regional data are available for the site(s) of interest, EPA recommends that States and Tribes use the local or regional data instead of the national default values because the POC and DOC can vary on a local basis, thus affecting the freely dissolved fraction.

6.2.4 Calculation of AWQC BAFs

Using equation 6.2.1 above, BAFs appropriate for calculating national AWQC for acrylonitrile are: 1.03, 1.02, and 1.05 for organisms at trophic levels two, three and four, respectively (expressed as three significant digits for convenience). These BAFs were derived using 1.5 L/kg for the baseline BAF at all three trophic levels, percent lipid content values of 2.3%, 1.5%, and 3.1% at trophic levels two, three, and four, respectively, and a freely dissolved fraction of 1.000. Calculation of the AWQC BAFs are shown below.

$$\text{BAF for AWQC}_{(TL\ n)} = [(\text{Baseline BAF}_{\ell}^{fd})_{TL\ n} \cdot (f_{\ell})_{TL\ n} + 1] \cdot (f_{fd})$$

AWQC BAF for Trophic Level Two

= [(1.5 L/kg-lipid)•(0.023) +1] • (1.000)

= 1.03 L/kg-tissue (expressed as three significant digits for convenience)

AWQC BAF for Trophic Level Three

= [(1.5 L/kg-lipid)•(0.015) +1] • (1.000)

= 1.02 L/kg-tissue (expressed as three significant digits for convenience)

AWQC BAF for Trophic Level Four

= [(1.5 L/kg-lipid)•(0.031) +1] • (1.000)

= 1.05 L/kg-tissue (expressed as three significant digits for convenience)

7. AWQC CALCULATION

7.1 For Ambient Waters Used as Drinking Water Sources

The cancer-based AWQC was calculated using the RSD and other input parameters listed below:

$$AWQC = RSD \times \left(\frac{BW}{DI + \sum_{i=2}^{4} (FI_i \times BAF_i)} \right)$$

(Equation 7.1.1)

where:

RSD = Risk specific dose (1.6×10^{-6} mg/kg-day at 10^{-6} lifetime risk, see Section 4.2.5.2)

BW = Human body weight assumed to be 70 kg

DI = Drinking water intake assumed to be 2 L/day

FI_i = Fish intake at trophic level i, i=2, 3, and 4; total intake assumed to be 0.01780 kg/day[3]

BAF_i = Bioaccumulation factor equal to 1.03 L/kg-tissue for trophic level two, 1.02 L/kg-tissue for trophic level three, and 1.05 L/kg-tissue for trophic level four.

This yields a concentration of 5.5×10^{-5} mg/L (or 0.055 µg/L) for a 10^{-6} lifetime cancer risk.

[3] Fish intake rates for each trophic level are: TL2=0.0011 kg/day; TL3=0.0115 kg/day; and TL4=0.0052 kg/day (presented as four significant figures for convenience). For further information, see Section 2.4.8 of the TSD.

7.2 For Ambient Waters Not Used as Drinking Water Sources

When the water body is to be used for recreational purposes and not as a source of drinking water, the drinking water value (DI above) is eliminated from the equation and it is substituted with an incidental ingestion value (II). The incidental intake is assumed to occur from swimming and other activities. The fish intake value is assumed to remain the same. The default value for incidental ingestion is 0.01 L/day. When the above equation is used to calculate the AWQC with the substitution of an incidental ingestion of 0.01 L/day an AWQC of 4.0×10^{-3} mg/L (or 4.0 µg/L, rounded from 3.95 µg/L) is obtained for a 10^{-6} lifetime cancer risk.

8. SITE-SPECIFIC OR REGIONAL ADJUSTMENTS TO CRITERIA

Several parameters in the AWQC equation can be adjusted on a site-specific or regional basis to reflect regional or local conditions and/or specific populations of concern. These include fish consumption, incidental water consumption as related to regional/local recreational activities, BAF (including factors used to derive BAFs such as POC/DOC, percent lipid of fish consumed by target population, and species representative of given trophic levels), and the relative source contribution. States and Tribes are encouraged to make adjustments using the information and instructions provided in the Technical Support Document (USEPA, 1998b).

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