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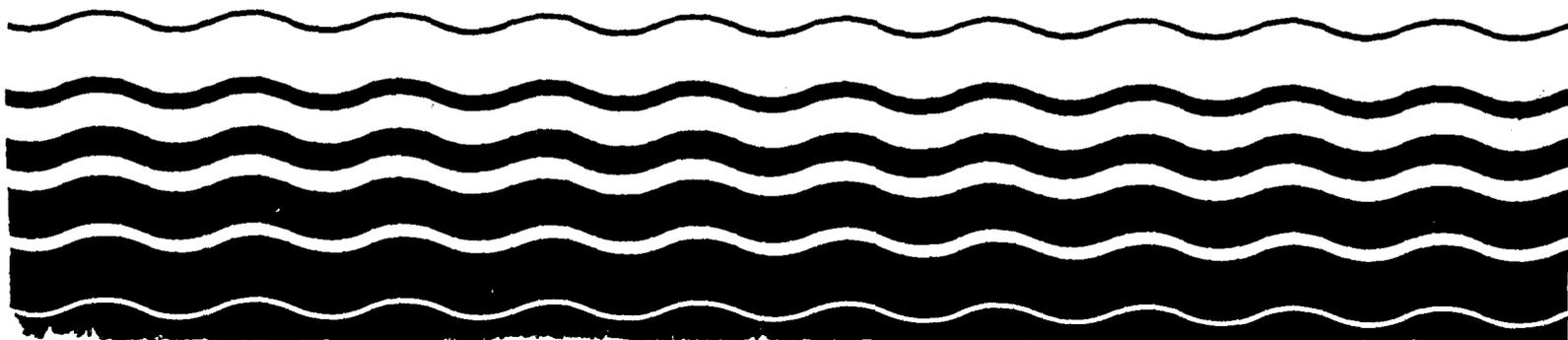
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# **Ambient Water Quality Criteria for**

## **Ammonia - 1984**



AMBIENT AQUATIC LIFE WATER QUALITY CRITERIA FOR  
AMMONIA

U.S. ENVIRONMENTAL PROTECTION AGENCY  
OFFICE OF RESEARCH AND DEVELOPMENT  
ENVIRONMENTAL RESEARCH LABORATORY  
DULUTH, MINNESOTA

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## FOREWORD

Section 304(a)(1) of the Clean Water Act of 1977 (P.L. 95-217) requires the Administrator of the Environmental Protection Agency to publish criteria for water quality accurately reflecting the latest scientific knowledge on the kind and extent of all identifiable effects on health and welfare which may be expected from the presence of pollutants in any body of water, including ground water. This document is a revision of proposed criteria based upon a consideration of comments received from other Federal agencies, State agencies, special interest groups, and individual scientists. The criteria contained in this document replace any previously published EPA aquatic life criteria.

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

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

Edwin L. Johnson  
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## CONTENTS

	<u>Page</u>
Foreword . . . . .	iii
Acknowledgments . . . . .	iv
Tables . . . . .	vi
Figures . . . . .	vii
Introduction . . . . .	1
Acute Toxicity to Aquatic Animals . . . . .	7
Chronic Toxicity to Aquatic Animals . . . . .	48
Toxicity to Aquatic Plants . . . . .	69
Bioaccumulation . . . . .	74
Other Data . . . . .	75
Unused Data . . . . .	86
Summary . . . . .	90
National Criteria . . . . .	94
Examples of Site-Specific Criteria . . . . .	100
References . . . . .	155

TABLES

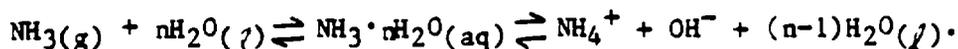
	<u>Page</u>
1. Acute Toxicity of Ammonia to Aquatic Animals . . . . .	105
2. Chronic Toxicity of Ammonia to Aquatic Animals . . . . .	129
3. Ranked Genus Mean Acute Values with Species Mean Acute-Chronic Ratios . . . . .	134
4. Toxicity of Ammonia to Aquatic Plants . . . . .	139
5. Other Data on Effects of Ammonia on Aquatic Organisms . . . . .	141

FIGURES

	<u>Page</u>
1. Acute NH <sub>3</sub> Toxicity at Different pH Values . . . . .	35
2. Acute NH <sub>3</sub> Toxicity at Different Temperatures . . . . .	38

## INTRODUCTION\*

In aqueous ammonia solutions, un-ionized ammonia exists in equilibrium with the ammonium ion and the hydroxide ion. The equation expressing this equilibrium can be written as:



As indicated in this equation, the dissolved ammonia molecule exists in hydrated form. The dissolved un-ionized ammonia is represented for convenience simply as  $\text{NH}_3$ . The ionized form is represented as  $\text{NH}_4^+$ . The term 'total ammonia' refers to the sum of these; i.e.,  $\text{NH}_3 + \text{NH}_4^+$ .

The toxicity of aqueous ammonia solutions to aquatic organisms is primarily attributable to the  $\text{NH}_3$  species, with the  $\text{NH}_4^+$  species being relatively less toxic (Chipman 1934; Wuhrmann et al. 1947; Wuhrmann and Woker 1948; Tabata 1962; Armstrong et al. 1978; Thurston et al. 1981c). It is, therefore, important to know the concentration of  $\text{NH}_3$  in any aqueous ammonia solution in order to determine what concentrations of total ammonia are toxic to aquatic life.

The concentration of  $\text{NH}_3$  is dependent on a number of factors in addition to total ammonia concentration (Skarheim 1973; Whitfield 1974; Emerson et al. 1975; Thurston et al. 1979; Messer et al. 1984). Most important among these are pH and temperature; the concentration of  $\text{NH}_3$  increases with increasing pH and with increasing temperature. The ionic strength is another important influence on this equilibrium. There is a

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\* An understanding of the "Guidelines for Deriving Numerical National Water Quality Criteria for the Protection of Aquatic Organisms and Their Uses" (Stephan, et al. 1985), hereafter referred to as the Guidelines, is necessary in order to understand the following text, tables, and calculations.

decrease in the percentage of un-ionized ammonia as the ionic strength increases in hard water or in saline water. In most natural freshwater systems the reduction of percent  $\text{NH}_3$  attributable to dissolved solids is negligible. In saline or very hard waters there will be small but measurable decreases in the percent  $\text{NH}_3$ .

A number of analytical methods are available for direct determination of total ammonia concentrations in aqueous solutions. Once total ammonia is measured, and the pH and temperature of the solution determined, the percent of total ammonia originally present as  $\text{NH}_3$  can be calculated based on the ammonia-water equilibrium. A review of analytical methods for ammonia in aqueous solution has been prepared by Richards and Healey (1984).

Emerson et al. (1975) carried out a critical evaluation of the literature data on the ammonia-water equilibrium system and published calculations of values of  $\text{pK}_a$  at different temperatures and of percent  $\text{NH}_3$  in ammonia solutions of zero salinity as a function of pH and temperature. The following table, reproduced from Emerson et al. (1975), provides values for percent  $\text{NH}_3$  at one-degree temperature intervals from 0 to 30 C, and pH intervals of 0.5 pH unit from pH 6.0 to 10.0. An expanded version of this percent  $\text{NH}_3$  table is provided in Thurston et al. (1979), which provides tabulated values of the  $\text{NH}_3$  fraction, expressed as percentage of total ammonia, at temperature intervals of 0.2 degree from 0.0 to 40.0 C, and pH intervals of 0.01 pH unit from pH 5.00 to 12.00. For salt water, reports by Whitfield (1974) and Skarheim (1973) provide calculations of  $\text{NH}_3$  as a function of pH, temperature, and salinity. Messer et al. (1984) indicate the impact of high total dissolved solids in fresh water.

Percent NH<sub>3</sub> in aqueous ammonia solutions for 0-30 C and pH 6-10.

Temp. (C)	pH								
	6.0	6.5	7.0	7.5	8.0	8.5	9.0	9.5	10.0
0	.00827	.0261	.0826	.261	.820	2.55	7.64	20.7	45.3
1	.00899	.0284	.0898	.284	.891	2.77	8.25	22.1	47.3
2	.00977	.0309	.0977	.308	.968	3.00	8.90	23.6	49.4
3	.0106	.0336	.106	.335	1.05	3.25	9.60	25.1	51.5
4	.0115	.0364	.115	.363	1.14	3.52	10.3	26.7	53.5
5	.0125	.0395	.125	.394	1.23	3.80	11.1	28.3	55.6
6	.0136	.0429	.135	.427	1.34	4.11	11.9	30.0	57.6
7	.0147	.0464	.147	.462	1.45	4.44	12.8	31.7	59.5
8	.0159	.0503	.159	.501	1.57	4.79	13.7	33.5	61.4
9	.0172	.0544	.172	.542	1.69	5.16	14.7	35.3	63.3
10	.0186	.0589	.186	.586	1.83	5.56	15.7	37.1	65.1
11	.0201	.0637	.201	.633	1.97	5.99	16.8	38.9	66.8
12	.0218	.0688	.217	.684	2.13	6.44	17.9	40.8	68.5
13	.0235	.0743	.235	.738	2.30	6.92	19.0	42.6	70.2
14	.0254	.0802	.253	.796	2.48	7.43	20.2	44.5	71.7
15	.0274	.0865	.273	.859	2.67	7.97	21.5	46.4	73.3
16	.0295	.0933	.294	.925	2.87	8.54	22.8	48.3	74.7
17	.0318	.101	.317	.996	3.08	9.14	24.1	50.2	76.1
18	.0343	.108	.342	1.07	3.31	9.78	25.5	52.0	77.4
19	.0369	.117	.368	1.15	3.56	10.5	27.0	53.9	78.7
20	.0397	.125	.396	1.24	3.82	11.2	28.4	55.7	79.9
21	.0427	.135	.425	1.33	4.10	11.9	29.9	57.5	81.0
22	.0459	.145	.457	1.43	4.39	12.7	31.5	59.2	82.1
23	.0493	.156	.491	1.54	4.70	13.5	33.0	60.9	83.2
24	.0530	.167	.527	1.65	5.03	14.4	34.6	62.6	84.1
25	.0569	.180	.566	1.77	5.38	15.3	36.3	64.3	85.1
26	.0610	.193	.607	1.89	5.75	16.2	37.9	65.9	85.9
27	.0654	.207	.651	2.03	6.15	17.2	39.6	67.4	86.8
28	.0701	.221	.697	2.17	6.56	18.2	41.2	68.9	87.5
29	.0752	.237	.747	2.32	7.00	19.2	42.9	70.4	88.3
30	.0805	.254	.799	2.48	7.46	20.3	44.6	71.8	89.0

[From Emerson et al. 1975; reproduced with permission from the Journal of the Fisheries Research Board of Canada.]

Concentrations of ammonia have been reported in the aquatic toxicity literature in terms of a variety of different forms, such as  $\text{NH}_3$ ,  $\text{NH}_4^+$ ,  $\text{NH}_3\text{-N}$ ,  $\text{NH}_4\text{OH}$ ,  $\text{NH}_4\text{Cl}$ , and others. The use in a literature article of the terms  $\text{NH}_3$ ,  $\text{NH}_3\text{-N}$ , or ammonia-nitrogen may not necessarily mean un-ionized ammonia, but may be the author's way of expressing total ammonia. The use of the term  $\text{NH}_3$  in this document always means un-ionized ammonia, and  $\text{NH}_3\text{-N}$  means un-ionized ammonia-nitrogen.

Throughout the following, all quantitative ammonia data have been expressed in terms of un-ionized ammonia, as mg/liter  $\text{NH}_3$ , for ease in discussion and comparison. Authors' ammonia concentration values are given as reported if authors provided data expressed as mg/liter  $\text{NH}_3$ . If authors reported only total ammonia values, or used concentration units other than mg/liter, these were used with the reported pH and temperature values to calculate mg/liter un-ionized  $\text{NH}_3$ . For calculations of  $\text{NH}_3$  in fresh water the table of Thurston et al. (1979) was used. For calculations in salt water the table of Skarheim (1973) was used.

Of the literature cited in this document, a significant number of papers provided insufficient pH and temperature data to enable calculation of  $\text{NH}_3$  concentrations; such papers were relegated to the section on "Unused Data" unless they provided useful qualitative or descriptive information. In some instances information missing in published papers on experimental conditions was obtained through correspondence with authors; data obtained in this manner are so indicated by footnotes.

Compounds used in the ammonia toxicity tests summarized here, and their formulas and Chemical Abstracts Services (CAS) Registry Numbers, are given below:

<u>Compound</u>	<u>Formula</u>	<u>CAS No.</u>
Ammonia	$\text{NH}_3$	7664417
Ammonium acetate	$\text{NH}_4\text{C}_2\text{H}_3\text{O}_2$	631618
Ammonium bicarbonate	$\text{NH}_4\text{HCO}_3$	1066337
Ammonium carbonate	$(\text{NH}_4)_2\text{CO}_3$	506876
Ammonium chloride	$\text{NH}_4\text{Cl}$	12125029
Ammonium hydrogen phosphate	$(\text{NH}_4)_2\text{HPO}_4$	7783280
Ammonium hydroxide	$\text{NH}_4\text{OH} (\text{NH}_3 \cdot \text{H}_2\text{O})$	1336216
Ammonium sulfate	$(\text{NH}_4)_2\text{SO}_4$	7783202

Papers stating use of other sources of ammonia were included if the source (e.g., excreted  $\text{NH}_3$  from fish) was deemed satisfactory. Papers using complex chemicals (e.g., ammonium ferricyanide, decyltrimethylammonium bromide) were not used. Finally, papers on ammonium compounds (e.g.,  $\text{NH}_4\text{F}$ ,  $(\text{NH}_4)_2\text{S}$ ) having anions that either might be themselves toxic or that would preclude calculation of  $\text{NH}_3$  concentration from the aqueous ammonia equilibrium relationship were not used.

A number of review articles or books dealing with ammonia as an aquatic pollutant are available. Water quality criteria for ammonia have been recommended in some of these. Liebmann (1960), McKee and Wolf (1963), Epler (1971), Becker and Thatcher (1973), Tsai (1975), Hampson (1976), Steffens (1976), Colt and Armstrong (1979), and Armstrong (1979) have published summaries of ammonia toxicity. Literature reviews including factors affecting ammonia toxicity and physiological effects of ammonia toxicity to aquatic organisms have been published by Lloyd (1961b), Lloyd and Herbert (1962), Warren (1962), Visek (1968), Lloyd and Swift (1976), and Kinne (1976). Literature reviews of ammonia toxicity information relating to criteria recommendations have been published by U.S. Federal Water Pollution

Control Administration (1968), European Inland Fisheries Advisory Commission (1970), National Academy of Sciences and National Academy of Engineering (1973), Willingham (1976), U.S. Environmental Protection Agency (1976, 1980), National Research Council (1979), Willingham et al. (1979), and Alabaster and Lloyd (1980).

The criteria presented herein supersede previous aquatic life water quality criteria for ammonia (U.S. Environmental Protection Agency 1976) because these new criteria were derived using more recent procedures and additional information. Whenever adequately justified, a national criterion may be replaced by a site-specific criterion (U.S. EPA, 1983a), which may include not only site-specific criterion concentrations (U.S. EPA, 1983b), but also site-specific durations of averaging periods and site-specific frequencies of allowed exceedences (U.S. EPA, 1985a). The latest literature search for information for this document was conducted in May, 1984; some newer information was also used; data from primary references only were used.

## ACUTE TOXICITY TO AQUATIC ANIMALS

### Freshwater Invertebrates

Acute toxicity of ammonia to freshwater invertebrate species has been much less studied than that to fishes. The preponderance of available invertebrate data is comprised of studies with arthropods, primarily crustaceans and insects. LC50s and EC50s are summarized in Table 1 for 12 species representing 14 families and 16 genera.

The acute toxicity of ammonia to Daphnia magna (Table 1) has been studied by several investigators, with reported 48-hour LC50s ranging from 0.53 to 4.94 mg/liter NH<sub>3</sub> (Parkhurst et al. 1979, 1981; Reinbold and Pescitelli 1982a; Russo et al. 1985).

Exposures (48 hours) of D. magna to NH<sub>4</sub>Cl in dilution water from two different sources were conducted by Russo et al. (1985). LC50s (Table 1) ranged from 2.4 to 2.8 mg/liter NH<sub>3</sub> in water of pH 7.95 to 8.15 and hardness 192 to 202 mg/liter as CaCO<sub>3</sub>, and from 0.53 to 0.90 mg/liter NH<sub>3</sub> in water of pH 7.4 to 7.5 and hardness 42 to 48 mg/liter as CaCO<sub>3</sub>. On an acute (48-hour LC50) basis, in dilution water from the same source, Ceriodaphnia acanthina, Simocephalus vetulus, and D. magna all exhibited similar sensitivities (Table 1) to ammonia (Mount 1982; Russo et al. 1985). West (1985) reported a LC50 of 2.29 mg/liter NH<sub>3</sub> for S. vetulus. The 48-hour LC50 (Table 1) of 1.16 mg/liter NH<sub>3</sub> reported by DeGraeve et al. (1980) for Daphnia pulicaria falls within the range of values reported for D. magna. Anderson (1948) reported a threshold toxicity value (Table 5) for D. magna of 2.4 to 3.6 mg/liter NH<sub>3</sub> in Lake Erie water. Threshold concentration was taken to mean the highest concentration that would just fail to immobilize the test animals under prolonged exposure (Anderson 1944). A minimum lethal concentration of 0.55 mg/liter NH<sub>3</sub> was reported for D.

magna by Malacea (1966), and a 24-hour LC50 of 1.50 mg/liter NH<sub>3</sub> was reported by Gyore and Olah (1980) for Moina rectirostris (Table 5).

Buikema et al. (1974) reported an EC50 (Table 5) for NH<sub>3</sub> toxicity to a bdelloid rotifer, Philodina acuticornis, to be 2.9-9.1 mg/liter NH<sub>3</sub> (calculated using reported pH values of 7.4 to 7.9). Tests of ammonia toxicity to a flatworm, Dendrocoelum lacteum (Procotyla fluviatilis), and tubificid worm, Tubifex tubifex, yielded LC50s (Table 1) of 1.4 and 2.7 mg/liter NH<sub>3</sub>, respectively (Stammer 1953).

Thurston et al. (1984a) conducted 25 flow-through toxicity tests with three mayfly, two stonefly, one caddisfly, and one isopod species; all tests were conducted with water of similar chemical composition. Ninety-six-hour LC50s ranged from 1.8 to 5.9 mg/liter NH<sub>3</sub> (Table 1). Results also indicated that a 96-hour test is not long enough to determine the acutely lethal effects of ammonia to the species tested; inasmuch as an asymptotic LC50 is not obtained within 96 hours. Percent survival data (Table 5) were reported for some mayfly, stonefly, and caddisfly tests in which LC50s were not obtained; 60 to 100 percent survival occurred at test concentrations ranging from 1.5 to 7.5 mg/liter NH<sub>3</sub>. Gall (1980) tested NH<sub>4</sub>Cl with Ephemerella sp. near excrucians. Organisms were exposed to ammonia for 24 hours, followed by 72 hours in ammonia-free water; mortality observations were made at the end of the overall 96-hour period. An EC50 (Table 5) of 4.7 mg/liter NH<sub>3</sub> was obtained. An LC50 (Table 1) of 8.00 mg/liter was reported for the beetle (Stenelmis sexlineata) by Hazel et al. (1979). West (1985) reported a 96-hour LC50 of 4.82 mg/liter NH<sub>3</sub> for the mayfly Callibaetis skokianus and 10.2 mg/liter NH<sub>3</sub> for the caddisfly Philarctus quaeris.

Ammonia toxicity tests conducted using dilution water from the Blue River in Colorado resulted in no mortalities of either scud (Gammarus lacustris) or D. magna after 96 hours' exposure to 0.08 mg/liter NH<sub>3</sub>. In a second test using river water buffered with sodium bicarbonate, 13 percent mortality occurred with scud at several concentrations tested, including the highest and lowest of 0.77 and 0.12 mg/liter NH<sub>3</sub>; seven and 13 percent mortality occurred with D. magna at the same concentrations (Miller et al. 1981).

Five freshwater mussel species (Amblema p. plicata, Anodonta imbecillis, Corbicula manilensis, Cyrtonaias tampicoensis, and Toxolasma texasensis) were exposed for 165 hours (Table 5) to a concentration of 0.32 mg/liter NH<sub>3</sub>; T. texasensis was most tolerant to ammonia, and A. p. plicata was most sensitive (Horne and McIntosh 1979). During the ammonia tolerance tests, the more tolerant species generally had their shells tightly shut, whereas the least tolerant species continued siphoning or had their mantles exposed. West (1985) reported 96-hour LC50s of 1.59 to 2.49 mg/liter NH<sub>3</sub> for the snail Physa gyrina, 2.76 mg/liter NH<sub>3</sub> for the snail Helisoma trivolvis, and 0.93 to 1.29 mg/liter NH<sub>3</sub> for the clam Musculium transversum.

Acute exposures of the freshwater crayfish (Orconectes nais) to NH<sub>4</sub>Cl gave LC50s of 3.15 and 3.82 mg/liter NH<sub>3</sub> (Evans 1979; Hazel et al. 1979). West (1985) reported LC50s of 22.8 mg/liter NH<sub>3</sub> for the crayfish Orconectes immunis, 1.63 to 5.63 mg/liter NH<sub>3</sub> for the amphipod Crangonyx pseudo-gracilis, and 4.95 mg/liter NH<sub>3</sub> for the isopod Asellus racovitzai.

## Freshwater Fishes

Acute toxicity tests with freshwater fish species have been conducted with 29 different species from 9 families and 18 genera, for which 96-hour LC50s are summarized in Table 1.

The acute toxicity of ammonia to rainbow trout (Salmo gairdneri) has been studied by many investigators, with reported 96-hour LC50s ranging from 0.16 to 1.1 mg/liter  $\text{NH}_3$  (Table 1).

Thurston and Russo (1983) conducted 71 toxicity tests with rainbow trout ranging in size from sac fry (<0.1 g) to 4-year-old adults (2.6 kg), in water of uniform chemical composition. LC50s (Table 1) ranged from 0.16 to 1.1 mg/liter  $\text{NH}_3$  for 96-hour exposures. Fish susceptibility to  $\text{NH}_3$  decreased with increasing weight over the range 0.06-2.0 g, but gradually increased above that weight range. LC50s for 12- and 35-day exposures (Table 5) were not greatly different from 96-hour values. No statistically significant differences in results were observed when different ammonium salts [ $\text{NH}_4\text{Cl}$ ,  $\text{NH}_4\text{HCO}_3$ ,  $(\text{NH}_4)_2\text{HPO}_4$ ,  $(\text{NH}_4)_2\text{SO}_4$ ] were used as the toxicants. Grindley (1946) also reported observing no appreciable difference in toxicity between toxicant solutions of  $\text{NH}_4\text{Cl}$  and  $(\text{NH}_4)_2\text{SO}_4$  with rainbow trout tests (Table 5).

LC50s (Table 1) ranging from 0.16 to 1.04 mg/liter  $\text{NH}_3$  for 96-hour exposures of rainbow trout to ammonia were reported by Calamari et al. (1977, 1981), Broderius and Smith (1979), Holt and Malcolm (1979), DeGraeve et al. (1980), Reinbold and Pescitelli (1982b), and West (1985). Ball (1967) reported an asymptotic (five-day) LC50 of 0.50 mg/liter  $\text{NH}_3$ . Acute exposures to ammonia of rainbow trout of life stages ranging from one to 345 days' post-fertilization (325 days post-hatch) were conducted by Calamari et al. (1977, 1981). They reported a tenfold increase in the speed of

intoxication processes between the embryonic and free larval stages; embryos and fingerlings (about one year old) were found to be less sensitive than the other life stages studied.

LC50s ranging from 0.49 to 0.70 mg/liter  $\text{NH}_3$  for 3-, 24-, and 48-hour exposures (Table 5) were reported by Herbert (1961, 1962), Herbert and Shurben (1964, 1965), and Herbert and Vandyke (1964). Rainbow trout (826 days old) subjected to 29.6 mg/liter  $\text{NH}_3$  reacted rapidly and strongly, overturned within two to three hours, and died within four hours (Corti 1951) (Table 5). Rainbow trout embryos and alevins were reported (Rice and Stokes 1975) to tolerate 3.58 mg/liter  $\text{NH}_3$  during 24-hour exposures; susceptibility increased during yolk absorption, with the 24-hour LC50 for 85-day-old fry being 0.068 mg/liter  $\text{NH}_3$  (Table 5). Nehring (1962-63) reported survival times of 1.3 and 3.0 hours at concentrations of 4.1 and 0.7 mg/liter  $\text{NH}_3$ , respectively (Table 5). Danecker (1964) reported survival times of 8 to 60 minutes at 0.4 to 4.0 mg/liter  $\text{NH}_3$ , respectively, with <0.2 mg/liter given as a no-mortality concentration (Table 5). Allan et al. (1958) reported a median survival time of 1000 minutes at 0.18 mg/liter  $\text{NH}_3$  (Table 5).

An acute value of 0.2 mg/liter  $\text{NH}_3$  attributed to Liebmann (1960) has been widely cited, in the EPA "Red Book" (U.S. Environmental Protection Agency 1976) and elsewhere, as being the lowest lethal concentration reported for salmonids. It is worthwhile to mention here a clarification and correction that was published in the American Fisheries Society's "Red Book Review" (Willingham et al. 1979): The research reported by Liebmann (1960) was that of Wuhrmann and Woker (1948); recomputation of the Wuhrmann and Woker data, using more accurate aqueous ammonia equilibrium tables, indicates

an effect level of approximately 0.32 mg/liter NH<sub>3</sub>, not 0.2 mg/liter NH<sub>3</sub> as cited by Liebmann.

A 96-hour LC50 of 0.44 mg/liter NH<sub>3</sub> was reported for rainbow trout in a test conducted using dilution water from the Blue River in Colorado (Miller et al. 1981). Pitts (1980) conducted toxicity tests using ammonium chloride and river water. Tests were conducted with rainbow trout, and LC50s ranged from 0.2 to 0.9 mg/liter NH<sub>3</sub> for 96-hour exposures at temperatures of 10 and 15 C.

Although acute toxicity studies with salmonids have been conducted preponderantly with rainbow trout, some data are also available for a few other salmonid species. Thurston et al. (1978) investigated the toxicity of ammonia to cutthroat trout (Salmo clarki), and reported 96-hour LC50s of 0.52 to 0.80 mg/liter NH<sub>3</sub> (Table 1). Thurston and Russo (1981) reported a 96-hour LC50 of 0.76 mg/liter NH<sub>3</sub> for golden trout (Salmo aguabonita) (Table 1). Taylor (1973) subjected brown trout (Salmo trutta) to 0.15 mg/liter NH<sub>3</sub> for 18 hours, resulting in 36 percent mortality (Table 5); when returned to ammonia-free water, the test fish recovered after nearly 24 hours. No mortalities occurred during a 96-hour exposure at 0.090 mg/liter NH<sub>3</sub>, although fish would not feed. Woker and Wuhrmann (1950) reported 0.8 mg/liter NH<sub>3</sub> was not acutely toxic to brown trout (Table 5). A 96-hour LC50 of 0.47 mg/liter NH<sub>3</sub> was reported for brown trout tested using dilution water from the Blue River in Colorado (Miller et al. 1981). Phillips (1950) reported that brook trout (Salvelinus fontinalis) evidenced distress within 1.75 hours at a concentration of 3.25 mg/liter NH<sub>3</sub> and within 2.5 hours at 5.5 mg/liter (Table 5). In replicated tests, Thurston and Mayn (1984) reported 96-hour LC50s (Table 1) of 0.60-0.70 mg/liter NH<sub>3</sub> for brown trout, 0.96-1.05 mg/liter NH<sub>3</sub> for brook trout, 0.40-0.48 mg/liter

# 4-Day Average Concentration for Ammonia<sup>1</sup>

Salmonids or Other Sensitive Coldwater Species Absent <sup>2</sup>

## Un-ionized Ammonia (mg/liter NH<sub>3</sub>)

pH	temperature 0°						
	0	5	10	15	20	25	30
6.50	0.0008	0.0011	0.0016	0.0022	0.0031	0.0031	0.0031
6.75	0.0014	0.0020	0.0028	0.0039	0.0055	0.0055	0.0055
7.00	0.0025	0.0035	0.0049	0.0070	0.0099	0.0099	0.0099
7.25	0.0044	0.0062	0.0088	0.0124	0.0175	0.0175	0.0175
7.50	0.0078	0.0111	0.0156	0.022	0.031	0.031	0.031
7.75	0.0129	0.0182	0.026	0.036	0.051	0.051	0.051
8.00	0.0149	0.021	0.030	0.042	0.059	0.059	0.059
8.25	0.0149	0.021	0.030	0.042	0.059	0.059	0.059
8.50	0.0149	0.021	0.030	0.042	0.059	0.059	0.059
8.75	0.0149	0.021	0.030	0.042	0.059	0.059	0.059
9.00	0.0149	0.021	0.030	0.042	0.059	0.059	0.059

## Total Ammonia (mg/liter NH<sub>3</sub>)

pH	temperature 0°						
	0	5	10	15	20	25	30
6.50	3.0	2.8	2.7	2.5	2.5	1.73	1.23
6.75	3.0	2.8	2.7	2.6	2.5	1.74	1.23
7.00	3.0	2.8	2.7	2.6	2.5	1.74	1.23
7.25	3.0	2.8	2.7	2.6	2.5	1.75	1.24
7.50	3.0	2.8	2.7	2.6	2.5	1.76	1.25
7.75	2.8	2.6	2.5	2.4	2.3	1.65	1.18
8.00	1.82	1.70	1.62	1.57	1.55	1.10	0.79
8.25	1.03	0.97	0.93	0.90	0.90	0.64	0.47
8.50	0.58	0.55	0.53	0.53	0.53	0.39	0.29
8.75	0.34	0.32	0.31	0.31	0.32	0.24	0.190
9.00	0.195	0.189	0.189	0.195	0.21	0.163	0.133

1 to convert these values to mg/liter N, multiply by 0.822

2 These values may be conservative, however, if a more refined criterion is desired, EPA recommends a site-specific criteria modification.

# 4-Day Average Concentration for Ammonia<sup>1</sup>

Salmonids or Other Sensitive Coldwater Species Present

		Un-ionized Ammonia (mg/liter NH <sub>3</sub> )						
		temperature 0°						
		0	5	10	15	20	25	30
pH	6.50	0.0008	0.0011	0.0016	0.0022	0.0022	0.0022	0.0022
	6.75	0.0014	0.0020	0.0028	0.0039	0.0039	0.0039	0.0039
	7.00	0.0025	0.0035	0.0049	0.0070	0.0070	0.0070	0.0070
	7.25	0.0044	0.0062	0.0088	0.0124	0.0124	0.0124	0.0124
	7.50	0.0018	0.0111	0.0156	0.022	0.022	0.022	0.022
	7.75	0.0129	0.0182	0.026	0.036	0.036	0.036	0.036
	8.00	0.0149	0.021	0.030	0.042	0.042	0.042	0.042
	8.25	0.0149	0.021	0.030	0.042	0.042	0.042	0.042
	8.50	0.0149	0.021	0.030	0.042	0.042	0.042	0.042
	8.75	0.0149	0.021	0.030	0.042	0.042	0.042	0.042
	9.00	0.0149	0.021	0.030	0.042	0.042	0.042	0.042

		Total Ammonia (mg/liter NH <sub>3</sub> )						
		temperature 0°						
		0	5	10	15	20	25	30
pH	6.50	3.0	2.8	2.7	2.5	1.76	1.23	0.87
	6.75	3.0	2.8	2.7	2.6	1.76	1.23	0.87
	7.00	3.0	2.8	2.7	2.6	1.76	1.23	0.87
	7.25	3.0	2.8	2.7	2.6	1.77	1.24	0.88
	7.50	3.0	2.8	2.7	2.6	1.78	1.25	0.89
	7.75	2.8	2.6	2.5	2.4	1.66	1.17	0.84
	8.00	1.82	1.70	1.62	1.57	1.10	0.78	0.56
	8.25	1.03	0.97	0.93	0.90	0.64	0.46	0.33
	8.50	0.58	0.55	0.53	0.53	0.38	0.28	0.21
	8.75	0.34	0.32	0.31	0.31	0.23	0.173	0.135
	9.00	0.195	0.189	0.189	0.195	0.148	0.116	0.094

<sup>1</sup> to convert these values to mg/liter N, multiply by 0.822



UNITED STATES ENVIRONMENTAL PROTECTION AGENCY  
WASHINGTON, D.C. 20460

JUL 30 1992

OFFICE OF  
WATER

MEMORANDUM

**SUBJECT:** Revised Tables for Determining Average Freshwater Ammonia Concentrations

**FROM:** Margarete Heber, Chief *Margarete Heber*  
Water Quality Criteria Section (WH-586)

Kent Ballentine, Chief *Kent Ballentine*  
Regulation and Policy Section (WH-585)

**TO:** Water Quality Standards Coordinators

The purpose of this memorandum is to provide you with a recalculation of the freshwater ammonia tables for Criteria Continuous Concentration, CCC (4 day average). These revised tables have been recalculated by removing the controversial white sucker data. Because the White Sucker was not one of the four most sensitive organisms, the Criteria Maximum Concentration remains the same as in the 1985 document.

Attached are the revised tables for determining the CCC for freshwater ammonia. The Final Acute Chronic Ratio (FACR) was calculated as in the 1985 ammonia criteria document except the Acute Chronic Ratio (ACR) for the white sucker was not used in determining the FACR. The white sucker data was removed because for many of the data there were not an adequate dose response. The FACR used is the geometric average of the ACR's of the channel catfish, bluegill, rainbow trout and fathead minnow. The result is that the FACR becomes 13.5 instead of the 16 as in the original tables.

The result of these changes should address concerns over the use of the white sucker data and result in a simplified freshwater ammonia criteria. If you have any questions regarding these changes please contact Margarete Heber at (202) 260-7144.

Attachments

NH<sub>3</sub> for chinook salmon (Oncorhynchus tshawytscha), and 0.14-0.47 mg/liter NH<sub>3</sub> for mountain whitefish (Prosopium williamsoni).

Toxicity tests (Tables 1, 5) on (NH<sub>4</sub>)<sub>2</sub>SO<sub>4</sub> with pink salmon (Oncorhynchus gorbuscha) at different stages of early life stage development (Rice and Bailey 1980) showed that late alevins near swim-up stage were the most sensitive (96-hour LC50 = 0.083 mg/liter NH<sub>3</sub>), and eyed embryos were the most tolerant, surviving 96 hours at >1.5 mg/liter NH<sub>3</sub>. Buckley (1978) reported a 96-hour LC50 of 0.55 mg/liter NH<sub>3</sub> for fingerling coho salmon, Oncorhynchus kisutch (Table 1). Herbert and Shurben (1965) reported a 24-hour LC50 (Table 5) of 0.28 mg/liter NH<sub>3</sub> for Atlantic salmon (Salmo salar). A comparison of relative susceptibilities of salmon smolts and yearling rainbow trout to 24-hour exposures to NH<sub>4</sub>Cl showed that the salmon were appreciably more susceptible than the trout in fresh water (Ministry of Technology, U.K. 1963).

Data are available on the acute toxicity of ammonia to a variety of non-salmonid fish species. Thurston et al. (1983) studied the toxicity of ammonia to fathead minnows (Pimephales promelas) of sizes ranging from 0.1 to 2.3 g; LC50s from 29 tests ranged from 0.75 to 3.4 mg/liter NH<sub>3</sub> (Table 1). Toxicity was not dependent upon test fish size or source. LC50s ranging from 0.73 to 2.35 mg/liter NH<sub>3</sub> (Tables 1,5) for fathead minnows were also reported by Sparks (1975), DeGraeve et al. (1980), Reinbold and Pescitelli (1982b), Swigert and Spacie (1983), and West (1985). Toxicity tests with fathead minnows using ammonium chloride and river water yielded 96-hour LC50s ranging from 0.6 to 2.4 mg/liter NH<sub>3</sub>; fathead minnows exposed to 0.12 mg/liter NH<sub>3</sub> in river water for 28 days incurred no mortalities (Pitts 1980).

LC50s (Table 1) for white sucker (Catostomus commersoni) exposed to ammonium chloride solutions for 96 hours (Reinbold and Pescitelli 1982c) were 1.40 and 1.35 mg/liter NH<sub>3</sub>; Swigert and Spacie (1983) determined a somewhat lower 96-hour LC50 of 0.79 mg/liter NH<sub>3</sub>, while West (1985) reported LC50s of 0.76 to 2.22 mg/liter NH<sub>3</sub> (Table 1). For mountain sucker (Catostomus platyrhynchus), Thurston and Meyn (1984) reported LC50s of 0.67-0.82 mg/liter NH<sub>3</sub> (Table 1).

Reported LC50s (Table 1) for 96-hour exposures of bluegill (Lepomis macrochirus) ranged from 0.26 to 4.60 mg/liter NH<sub>3</sub> (Emery and Welch 1969; Lubinski et al. 1974; Roseboom and Richey 1977; Reinbold and Pescitelli 1982b; Smith et al. 1983; Swigert and Spacie 1983). LC50s (Table 1) of 0.7 to 1.8 mg/liter NH<sub>3</sub> for smallmouth bass (Micropterus dolomieu) and 1.0 to 1.7 mg/liter NH<sub>3</sub> for largemouth bass (Micropterus salmoides) were reported by Broderius et al. (1985) and Roseboom and Richey (1977), respectively, for 96-hour exposures. Sparks (1975) reported 48-hour LC50s (in parentheses, as mg/liter NH<sub>3</sub>) for bluegill (2.30) and channel catfish (2.92), Dowden and Bennett (1965) reported a 24-hour LC50 for goldfish (Carassius auratus) (7.2), and Chipman (1934) reported lethal threshold values of 0.97 to 3.8 mg/liter NH<sub>3</sub> for goldfish (Table 5). Turnbull et al. (1954) reported a 48-hour LC50 for bluegill to be within the range 0.024 to 0.093 mg/liter NH<sub>3</sub> (Table 5); during the exposure they observed that the fish exhibited a lack of perception to avoid objects.

Reported 96-hour LC50s (Table 1) for channel catfish (Ictalurus punctatus) ranged from 0.5 to 4.2 mg/liter NH<sub>3</sub> (Colt and Tchobanoglous 1976; Roseboom and Richey 1977; Reinbold and Pescitelli 1982d; Swigert and Spacie 1983; West 1985). Vaughn and Simco (1977) reported a 48-hour LC50 for channel catfish of 1.24 to 1.96 mg/liter NH<sub>3</sub>, and Knepp and Arkin (1973)

reported one-week LC50s of 0.97 to 2.0 mg/liter  $\text{NH}_3$  (Table 5). From studies with bluegill, channel catfish, and largemouth bass, Roseboom and Richey (1977) reported that bluegill susceptibility was dependent upon fish weight, with 0.07-g fish being slightly more sensitive than either 0.22- or 0.65-g fish; size had little effect upon channel catfish or bass susceptibility.

LC50s (Table 1) were determined with two species of field-collected fishes indigenous to Kansas streams, orangethroat darter (Etheostoma spectabile) and red shiner (Notropis lutrensis) (Hazel et al. 1979); 96-hour LC50s were 0.90 and 1.07 mg/liter  $\text{NH}_3$  for darter and 2.83 for shiner. Commercially obtained largemouth bass, channel catfish, and bluegill (18 fish of each species) were also exposed for 96 hours to a concentration of 0.21 mg/liter  $\text{NH}_3$ , resulting in zero mortality for bluegill and channel catfish and one mortality (6 percent) among the largemouth bass tested. Reported LC50s for walleye (Stizostedion vitreum) range from 0.51 to 1.10 mg/liter  $\text{NH}_3$  (Reinbold and Pescitelli 1982a; West 1985).

LC50s (Table 1) ranging from 2.4 to 3.2 mg/liter  $\text{NH}_3$  for  $(\text{NH}_4)_2\text{CO}_3$ ,  $\text{NH}_4\text{Cl}$ ,  $\text{NH}_4\text{C}_2\text{H}_3\text{O}_2$ , and  $\text{NH}_4\text{OH}$ , in 96-hour exposures of mosquitofish (Gambusia affinis) in waters with suspended solids ranging from <25 to 1400 mg/liter were reported by Wallen et al. (1957). Susceptibility of mosquitofish to ammonia was studied by Hemens (1966) who reported a 17-hour LC50 of 1.3 mg/liter  $\text{NH}_3$  (Table 5); he also observed that male fish were more susceptible than females. Powers (1920) reported the relative susceptibilities of three fish species to ammonium chloride to be (most sensitive to least sensitive): straw-colored minnow (Notropis blennioides) > bluntnose minnow (Pimephales notatus) > goldfish.

Rubin and Elmaraghy (1976, 1977) tested guppy (Poecilia reticulata) fry and reported 96-hour LC50s (Table 1) averaging 1.50 mg/liter NH<sub>3</sub>; mature guppy males were more tolerant, with 100 percent survival for 96 hours at concentrations of 0.17 to 1.58 mg/liter NH<sub>3</sub>. LC50s (Table 1) of 0.15 and 0.20 mg/liter NH<sub>3</sub> at pH 6.0, and of 0.52 and 2.13 mg/liter NH<sub>3</sub> at pH 8.0, were reported by Stevenson (1977) for white perch (Morone americana). LC50s (96 hours) of 1.20 and 1.62 mg/liter NH<sub>3</sub> for spotfin shiner (Notropis spilopterus), and of 1.20 mg/liter NH<sub>3</sub> for golden shiner (Notemigonus crysoleucas), were reported by Rosage et al. (1979) and Baird et al. (1979), respectively. Swigert and Spacie (1983) determined 96-hour LC50s to be 0.72 mg/liter NH<sub>3</sub> for golden shiner, 1.35 mg/liter NH<sub>3</sub> for spotfin shiner, 1.25 mg/liter NH<sub>3</sub> for steelcolor shiner (Notropis whipplei), and 1.72 mg/liter NH<sub>3</sub> for stoneroller (Campostoma anomalum).

Jude (1973), Rainbold and Pescitelli (1982a), and McCormick et al. (1984) reported 96-hour LC50s ranging from 0.6 to 2.1 mg/liter NH<sub>3</sub> for green sunfish (Lepomis cyanellus) (Table 1). Pumpkinseed sunfish (Lepomis gibbosus) were tested by Jude (1973) and Thurston (1981), with reported 96-hour LC50s ranging from 0.14 to 0.86 mg/liter NH<sub>3</sub>. Mottled sculpin (Cottus bairdi) were tested by Thurston and Russo (1981), yielding a 96-hour LC50 of 1.39 mg/liter NH<sub>3</sub> (Table 1). Ball (1967) determined an asymptotic (six-day) LC50 (Table 5) of 0.44 mg/liter NH<sub>3</sub> for rudd (Scardinius erythrophthalmus). He compared the asymptotic LC50s for this species against that obtained within two days for rainbow trout. Although the trout had proven to be more sensitive to ammonia than had rudd during the first day of the tests, the asymptotic LC50 for both species showed little difference.

Rao et al. (1975) reported a 96-hour LC50 for carp (Cyprinus carpio) of 1.1 mg/liter NH<sub>3</sub> (Table 5). Carp exposed to 0.24 mg/liter NH<sub>3</sub> exhibited no adverse effects in 18 hours (Vamos 1963). Exposure to 0.67 mg/liter NH<sub>3</sub> caused gasping and equilibrium disturbance in 18 min, frenetic swimming activity in 25 min, then sinking to the tank bottom after 60 min; after 75 min the fish were placed in ammonia-free water and all revived. Similar effects were observed at a concentration of 0.52 mg/liter NH<sub>3</sub> (Table 5). Pre-treating fish orally with 12.5 mg Suprastin (N-dimethyl-aminoethyl-N-p-chlorobenzyl- $\alpha$ -aminopyridin hydrochlor), a chemical which reduces cell membrane permeability, somewhat reduced the toxic effect of ammonia.

A lethal concentration (Table 5) for carp was reported to be 7.5 mg/liter NH<sub>3</sub> (Kempinska 1968). Acute exposures (Table 5) to ammonium sulfate of bitterling (Rhodeus sericeus) and carp were conducted by Malacea (1966), who determined minimum lethal concentrations (i.e., after such exposure, fish placed in ammonia-free water were unable to recover) of 0.76 mg/liter NH<sub>3</sub> for bitterling and 1.4 mg/liter NH<sub>3</sub> for carp. Nehring (1962-63) reported survival times of carp to be 2.4 and 6.0 hours at NH<sub>3</sub> concentrations of 9.7 and 2.1 mg/liter NH<sub>3</sub>, respectively (Table 5). Danecker (1964) reported survival time for cench (Tinca tinca) to be 20 to 24 hours at 2.5 mg/liter NH<sub>3</sub> (Table 5). In a 24-hour exposure of creek chub (Semotilus atromaculatus) to NH<sub>4</sub>OH solution (Gilletce et al. 1952), the "critical range" below which all test fish lived and above which all died was reported to be 0.26 to 1.2 mg/liter NH<sub>3</sub> (Table 5).

In static exposures lasting 9 to 24 hours, with gradual increases in NH<sub>3</sub> content, lethal concentrations (Table 5) were determined for oscar (Astronotus ocellatus) (Magalhães Bastos 1954); mortalities occurred at 0.50 mg/liter NH<sub>3</sub> (4 percent) to 1.8 mg/liter (100 percent). Tests on oscar of

two different sizes (average weights 1.6 g for "small" fish and 22.5 g for "medium" fish) showed no difference in susceptibility related to fish size. A 72-hour LC50 (Table 5) of 2.85 mg/liter NH<sub>3</sub> was reported by Redner and Stickney (1979) for blue tilapia (Tilapia aurea).

#### Factors Affecting Acute Toxicity of Ammonia

There are a number of factors that can affect the toxicity of ammonia to aquatic organisms. These factors include effects of dissolved oxygen concentration, temperature, pH, previous acclimation to ammonia, fluctuating or intermittent exposures, carbon dioxide concentration, salinity, and presence of other toxicants. Almost all studies of factors affecting ammonia toxicity have been carried out using only acute exposures.

##### (a) Dissolved Oxygen

A decrease in dissolved oxygen concentration in the water can increase ammonia toxicity. Vamos and Tasnadi (1967) observed mortalities in carp ponds at ammonia concentrations lower than would normally be lethal, and attributed this to periodic low concentrations of oxygen. Based on research in warmwater (20-22 C) fish ponds, Selesi and Vamos (1976) projected a "lethal line" relating acute ammonia toxicity and dissolved oxygen, below which carp died. The line ran between 0.2 mg/liter NH<sub>3</sub> at 5 mg/liter dissolved oxygen and 1.2 mg/liter NH<sub>3</sub> at 10 mg/liter dissolved oxygen. Thurston et al. (1983) compared the acute toxicity of ammonia to fathead minnows at reduced and normal dissolved oxygen concentrations; seven 96-hour tests were conducted within the range 2.6 to 4.9 mg/liter dissolved oxygen, and three between 8.7 and 8.9 mg/liter. There was a slight positive trend between 96-hour LC50 and dissolved oxygen, although it was not shown to be statistically significant.

Alabaster et al. (1979) tested Atlantic salmon smolts in both fresh water and 30 percent salt water at 9.6-9.5 and 3.5-3.1 mg/liter dissolved oxygen. The reported 24-hour LC50s at the higher oxygen concentrations were about twice that at the lower. Recently, Alabaster et al. (1983) reported freshwater LC50s for Atlantic salmon in 10.2 and 3.1-3.2 mg/liter dissolved oxygen as 0.2 and 0.08 mg/liter  $\text{NH}_3$ , respectively.

Several studies have been reported on rainbow trout. Allan (1955) reported that below 0.12 mg/liter  $\text{NH}_3$  and at about 30 percent oxygen saturation, the median survival time was greater than 24 hours, but at the same concentration with oxygen saturation below 30 percent, the median survival time was less than 24 hours. Downing and Merkens (1955) tested fingerling rainbow trout at three different concentrations of  $\text{NH}_3$  at five different levels of dissolved oxygen. They reported, in tests lasting up to 17 hours, that decreasing the oxygen from 9.5 to 1.5 mg/liter shortened the periods of survival at all ammonia concentrations, and that a decrease in survival time produced by a given decrease in oxygen was greatest in the lowest concentration of  $\text{NH}_3$ . Merkens and Downing (1957), in tests which lasted up to 13 days, also reported that the effect of low concentrations of dissolved oxygen on the survival of rainbow trout was more pronounced at low concentrations of  $\text{NH}_3$ . Lloyd (1961a) found  $\text{NH}_3$  to be up to 2.5 times more toxic when dissolved oxygen concentration was reduced from 100 to about 40 percent saturation. Danecker (1964) reported that the toxicity of ammonia increased rapidly when the oxygen concentration decreased below two-thirds of the saturation value.

Thurston et al. (1981b) conducted 15 96-hour acute toxicity tests with rainbow trout over the dissolved oxygen range 2.6 to 8.6 mg/liter. They

reported a positive linear correlation between 96-hour LC50 and dissolved oxygen over the entire range tested.

Herbert (1956) reported on rainbow trout mortalities in a channel receiving sewage discharge containing 0.05 to 0.06 mg/liter  $\text{NH}_3$ . They found that at 25-35 percent dissolved oxygen saturation more than 50 percent of the fish died within 24 hours, compared with 50 percent mortality of test fish in the laboratory at 15 percent dissolved oxygen saturation. The difference was attributed to unfavorable water conditions below the sewage outflow, including ammonia, which increased the sensitivity of the fish to the lack of oxygen.

There is a reduction in fish blood oxygen-carrying capacity following ammonia exposure (Brockway 1950; Danecker 1964; Reichenbach-Klinke 1967; Körtting 1969a,b; Waluga and Flis 1971). Hypoxia would further exacerbate problems of oxygen delivery and could lead to the early demise of the fish.

(b) Temperature

Information in the literature on the effects of temperature on ammonia toxicity is varied. The concentration of  $\text{NH}_3$  increases with increasing temperature. Several researchers have reported an effect of temperature on the toxicity of the un-ionized ammonia species, independent of the effect of temperature on the aqueous ammonia equilibrium.

Hazel et al. (1971) tested ammonia with striped bass (Morone saxatilis) and stickleback (Gasterosteus aculeatus) and found little difference in toxicity between 15 and 23 C in fresh water, although both fish species were slightly more resistant at the lower temperature. Erickson (1985) noted, however, that Hazel et al. did not account for the effect of temperature on ammonia equilibrium; when corrected, their data indicate both species to be moderately more tolerant at the higher temperature. McCay and Vars (1931)

reported that it took three times as long for brown bullheads (Ictalurus nebulosus) to succumb to the toxicity of ammonia in water at 10-13 C than at 26 C. The pH of the tested water was not reported; however, within the probable range tested (pH 7-8), the percent  $\text{NH}_3$  at the higher test temperature is approximately three times that at the mean lower temperature. Powers (1920) reported the toxicity of ammonium chloride to goldfish, bluntnose minnow, and straw-colored minnow to be greater at high temperatures than at low; however, in that study also no consideration was given to the increase in relative concentration of  $\text{NH}_3$  as temperature increased.

Thurston et al. (1983) reported that the acute toxicity of  $\text{NH}_3$  to fathead minnows decreased with a rise in temperature over the range 12 to 22 C. Bluegill and fathead minnow were tested at low and high temperatures of 4.0 to 4.6 C and 23.9 to 25.2 C, respectively; rainbow trout were tested at 3.0 and 14.0 C (Reinbold and Pescitelli 1982b). All three species were more sensitive to un-ionized ammonia at the low temperatures, with toxicity being 1.5 to 5 times greater in the colder water; bluegill appeared to be the most sensitive of the three species to the effect of low temperature on ammonia toxicity.

Colt and Tchobanoglous (1976) reported that the toxicity of  $\text{NH}_3$  to channel catfish decreased with increasing temperature over the range 22 to 30 C. LC50s for bluegill, channel catfish, and largemouth bass at 28 to 30 C were approximately twice that at 22 C (Roseboom and Richey 1977). LC50s for channel catfish tested in Iowa River water were 0.49 mg/liter  $\text{NH}_3$  at 2.5 C and 0.56 mg/liter at 5.1 C (Miller and UNLV-EPA 1982). An effluent containing ammonia as a principal toxic component showed a marked decrease in toxicity to channel catfish over the temperature range 4.6 to 21.3 C (Cary 1976).

Herbert (1962) has reported that experiments with rainbow trout in his laboratory suggest that the effect of temperature on their susceptibility to  $\text{NH}_3$  toxicity is little if at all affected by temperature change; no details were provided. The Ministry of Technology, U.K. (1968), however, has reported that the toxicity of  $\text{NH}_3$  to rainbow trout was much greater at 5 C than at 18 C. Brown (1968) reported that the 48-hour LC50 for rainbow trout increased with an increase in temperature over the range 3 to 16 C; the reported increase in tolerance between ~12 to ~18 C was considerably less than that between ~3 to ~12 C. Thurston and Russo (1983) reported a relationship between temperature and 96-hour LC50 for rainbow trout over the temperature range 12 to 19 C; ammonia toxicity decreased with increasing temperature.

Lloyd and Orr (1969) investigated the effect of temperature over the range 10-20 C on urine flow rates of rainbow trout exposed to 0.30 mg/liter  $\text{NH}_3$ , and found no apparent temperature effect on the total diuretic response of the fish, although the relative increase in urine production was less at higher temperatures. From a study of the behavioral response of bluegill to gradients of ammonia chloride it was hypothesized that low temperatures increased the sensitivity of bluegill and interfered with their ability either to detect ammonia after a certain period of exposure or to compensate behaviorally for physiological stress caused by ammonia gradients (Lubinski 1979; Lubinski et al. 1980).

The European Inland Fisheries Advisory Commission (1970) has cautioned that at temperatures below 5 C the toxic effects of un-ionized ammonia may be greater than above 5 C. The basis for such a statement is not clearly documented in that report. Nevertheless, there is some merit to the argument that a decrease in temperature may increase the susceptibility of fish to

un-ionized ammonia toxicity. It is important that this relationship be further studied. The available evidence that temperature, independent of its role in the aqueous ammonia equilibrium, affects the toxicity of  $\text{NH}_3$  to fishes argues for further consideration of the effect of temperature on the toxicity of ammonia.

West (1985) investigated the seasonal variation of ammonia toxicity for five species of fish. Marked and generally steady increases of LC50s with temperature were observed for rainbow trout from 3.6 to 18.7 C and for channel catfish from 3.5 to 26 C. For fathead minnow, a similar trend was found for temperatures from 12 to 26 C, but at 3.4 C, the LC50 was higher than at 12 C. Similar trends were observed for walleye between 3.7 and 11 C and for white sucker between 3.6 and 15 C, but both these species showed a lower LC50 at a higher test temperature (19 C for walleye and 25 C for white sucker); in both cases, however, this apparent deviation from trends for other tests is confounded by different sizes of test organisms and, as with the other species, by seasonal changes other than temperature; also, for the white sucker test, the test at higher temperature suffered from low dissolved oxygen. West also examined the seasonal dependence of ammonia toxicity to three invertebrates (snail Physa gyrina, clam Musculium transversum, and amphipod Crangonyx pseudogracilis). For all species, the maximum LC50 was at intermediate temperature (12-15 C), with lower values at colder and warmer temperatures. For the two molluscs, the apparent variation with temperature was not great, the minimum LC50 being only about 30% less than the maximum. For the amphipod, the variation was two- to three-fold.

(c) pH

The toxicity to fishes of aqueous solutions of ammonia and ammonium compounds has been attributed to the un-ionized (undissociated) ammonia

present in the solution. Although there were observations in the early literature that ammonia toxicity was greater in alkaline solutions, the earliest reported thorough study of the pH dependence of ammonia toxicity was that of Chipman (1934). He concluded from experiments with goldfish, amphipods, and cladocerans that the toxicity was a function of pH and therefore of the concentration of undissociated ammonia in the solution.

Wuhrmann et al. (1947) discussed the importance of differentiating between  $\text{NH}_3$  and  $\text{NH}_4^+$  when considering ammonia toxicity. They summarized some unpublished experimental data indicating a correlation between solution pH and ammonia toxicity to fish (indicated by persistent loss of balance). Wuhrmann and Woker (1948) reported on the experiments referred to in Wuhrmann et al. (1947); these were conducted using ammonium sulfate solutions at different pH values on rainbow trout. Either four or six fish were tested at each of nine ammonium sulfate concentrations. The authors concluded from the experimental results that  $\text{NH}_3$  was much more toxic than  $\text{NH}_4^+$ . Downing and Merkens (1955) tested rainbow trout at different concentrations of ammonia at both pH 7 and 8. They reported a consistency of results when ammonia concentration was expressed as  $\text{NH}_3$ .

Tabata (1962) conducted 24-hour tests (Table 5) on ammonia toxicity to Daphnia (species not specified) and guppy at different pH values and calculated the relative toxicity of  $\text{NH}_3/\text{NH}_4^+$  to be 190 for guppy (i.e.,  $\text{NH}_3$  is 190 times more toxic than  $\text{NH}_4^+$ ) and 48 for Daphnia. From tests of the toxicity of ammonium chloride to juvenile coho salmon in flow-through bioassays within the pH range 7.0 to 8.5, the reported 96-hour LC50 for  $\text{NH}_3$  was approximately 60 percent less at pH 7.0 than at 8.5 (Robinson-Wilson and Seim 1975).

Armstrong et al. (1978) tested the toxicity of ammonium chloride to larvae of prawn (Macrobrachium rosenbergii) in six-day tests within the pH range 6.8 to 8.3; test solutions were renewed every 24 hours. They reported a 96-hour LC50 for NH<sub>3</sub> at pH 6.83 which was approximately 70 percent less than that for pH 8.34. They concluded that the toxicity of ammonia was not due solely to the NH<sub>3</sub> molecule, that in solutions of different pH and equal NH<sub>3</sub> concentrations survival was greatly reduced as NH<sub>4</sub><sup>+</sup> levels increased. Tomasso et al. (1980) tested the toxicity of ammonia at pH 7, 8, and 9 on channel catfish and reported that 24-hour NH<sub>3</sub> LC50s were significantly higher at pH 8 than at pH 7 or 9.

Thurston et al. (1981c) tested the toxicity of ammonia to rainbow trout and to fathead minnows in 96-hour flow-through tests at different pH levels within the range 6.5 to 9.0. Results showed that the toxicity of ammonia, in terms of NH<sub>3</sub>, increased at lower pH values. They concluded that NH<sub>4</sub><sup>+</sup> exerts some measure of toxicity, and/or that increased H<sup>+</sup> concentration increases the toxicity of NH<sub>3</sub>.

Acute (96-hour) exposures of green sunfish and smallmouth bass were conducted by McCormick et al. (1984) and Broderius et al. (1985) at four different pH levels over the range 6.5 to 8.7. For both species, NH<sub>3</sub> toxicity increased markedly with a decrease in pH, with LC50s at the lowest pH tested (6.6 for sunfish, 6.5 for bass) being 3.6 (sunfish) and 2.6 (bass) times smaller than those at the highest pH tested (8.7). LC50s found with rainbow trout for the ammoniacal portion (diammonium phosphate) of a chemical fire retardant at two different pH levels indicated greater NH<sub>3</sub> toxicity at lower pH (Blahm 1978).

(d) Acclimation and Fluctuating Exposures

The question of whether fish can acquire an increased tolerance to ammonia by acclimation to low ammonia concentrations is an important one. If fish had an increased ammonia tolerance developed due to acclimation or conditioning to low ammonia levels, they would perhaps be able to survive what otherwise might be acutely lethal ammonia concentrations.

Observations by McCay and Vars (1931) indicated that bullheads subjected to several successive exposures to ammonia, alternated with recovery in fresh water, acquired no immunity from the earlier exposures to the later ones. A greater number of researchers have reported that previous exposure of fishes to low concentrations of ammonia increases their resistance to lethal concentrations. Vámos (1963) conducted a single experiment in which carp which had been revived in fresh water for 12 hours after exposure to 0.67 or 0.52 mg/liter  $\text{NH}_3$  for 75 min were placed in a solution containing 0.7 mg/liter  $\text{NH}_3$ . The previously exposed fish exhibited symptoms of ammonia toxicity in 60-85 min, whereas control fish developed symptoms within 20 min. Redner and Stickney (1979) reported that blue tilapia acclimated for 35 days to 0.52 to 0.64 mg/liter  $\text{NH}_3$  subsequently survived 48 hours at 4.1 mg/liter; the 48-hour  $\text{LC}_{50}$  for unacclimated fish was 2.9 mg/liter.

Málácea (1968) studied the effect of acclimation of bitterling to ammonium sulfate solutions. A group of ten fish was held in an acclimation solution of 0.26 mg/liter  $\text{NH}_3$  for 94 hours, after which the fish were exposed to a 5.1 mg/liter  $\text{NH}_3$  solution for 240 min; a control group of ten was treated identically, except their acclimation aquarium did not contain added  $(\text{NH}_4)_2\text{SO}_4$ . The ratio of the mean survival times of "adapted" vs. "unadapted" fish was 1.13; mean survival times for the adapted and unadapted

fish were 78 and 88 minutes, respectively, indicating somewhat higher ammonia tolerance for adapted fish.

Fromm (1970) measured urea excretion rates of rainbow trout initially acclimated to either 5 or 0.5 mg/liter  $\text{NH}_3$ , then subjected to 3 mg/liter  $\text{NH}_3$ . Fish previously exposed to 5 mg/liter  $\text{NH}_3$  excreted slightly less urea than those exposed to the lower concentration. Lloyd and Orr (1969) conducted acclimation experiments with rainbow trout and found that the rate of urine excretion increased with a rise in the concentration of un-ionized ammonia to which the fish were exposed. They presented some evidence for acclimation of rainbow trout to sublethal levels of ammonia, although these levels may be as low as 12 percent of the "lethal threshold concentration". Acclimation was retained for 24 hours, but was not retained after three days. They also suggested that environmental factors which affect the water balance of fish may also influence susceptibility to ammonia toxicity. Fromm (1970) acclimated goldfish to low (0.5 mg/liter) or high (5.0 or 25.0 mg/liter) ambient  $\text{NH}_3$  for periods of 20 to 56 days and found that urea excretion rate in subsequent 24-hour exposures to concentrations ranging from 0.08 to 2.37 mg/liter was independent of the previous acclimation concentration or duration.

Schulze-Wiehenbrauck (1976) subjected two groups of rainbow trout (56 g and 110 g) which had been held for at least three weeks at sublethal ammonia concentrations to lethal ammonia concentrations. In the experiment with 110-g fish, the sublethal acclimation concentrations were 0.007 (control), 0.131, and 0.167 mg/liter  $\text{NH}_3$ ; the fish from these three tanks were then subjected to concentrations of 0.45, 0.42, and 0.47 mg/liter  $\text{NH}_3$ , respectively, for 8.5 hours. Fish from the two higher sublethal

concentrations had 100 percent survival after 8.5 hours in the 0.42 and 0.47 mg/liter  $\text{NH}_3$  solutions, whereas fish from the 0.007 mg/liter  $\text{NH}_3$  concentration had only 50 percent survival in 0.45 mg/liter  $\text{NH}_3$ . In the experiment with 56-g fish, the acclimation concentrations were 0.004 mg/liter  $\text{NH}_3$  (control) and 0.159 mg/liter  $\text{NH}_3$ ; these fish were placed in  $\text{NH}_3$  concentrations of 0.515 and 0.523 mg/liter, respectively, for 10.25 hours. There was 100 percent survival of the acclimated fish, and 85 percent survival of the control fish. The results of these experiments thus showed an increase in resistance of trout to high ammonia levels after prior exposure to sublethal ammonia levels.

Alabaster et al. (1979) determined 24-hour LC50s of  $\text{NH}_3$  for Atlantic salmon smolts under reduced dissolved oxygen test conditions. Fish acclimated to ammonia before oxygen reduction evidenced LC50s 38 and 79 percent higher than fish without prior ammonia acclimation.

Brown et al. (1969) tested rainbow trout in static tests in which fish were moved back and forth between tanks in which the ammonia concentrations were 0.5 and 1.5 times a previously determined 48-hour LC50. If fish were transferred on an hourly basis, the median period of survival for the fluctuating exposure was reported to be the same as that for constant exposure (>700 min). If the fish were transferred at two-hour intervals, the median survival time for the fluctuating exposure was reported to be less (370 min), indicating that the toxic effects from exposure to the fluctuating concentrations of ammonia was greater than those from exposure to the constant concentration.

Thurston et al. (1981a) conducted acute toxicity tests on rainbow trout and cutthroat trout in which fish were exposed to short-term cyclic

fluctuations of ammonia. Companion tests were also conducted in which test fish were subjected to ammonia at constant concentrations. LC50s in terms of both average and peak concentrations of ammonia for the fluctuating concentration tests were compared with LC50s for the constant concentration tests. Based on comparisons of total exposure, results showed that fish were more tolerant of constant concentrations of ammonia than of fluctuating concentrations. Fish subjected to fluctuating concentrations of ammonia at levels below those acutely toxic were subsequently better able to withstand exposure to higher fluctuating concentrations than fish not previously so acclimated.

In renewal exposures to ammonium chloride using river water as the dilution water, fathead minnows were reported (Pitts 1980) to survive for 28 days exposures fluctuating from 0.1 mg/liter  $\text{NH}_3$  for four days to 0.2 or 0.3 mg/liter  $\text{NH}_3$  for three days. Four-day excursions above 0.1 mg/liter to concentrations of 0.42, 0.48, and 0.52 mg/liter resulted in 80 to 100 percent mortality in 28 days, as did four-day excursions to 0.73 mg/liter. No constant exposure tests were conducted simultaneously for comparative purposes; however, constant exposure tests conducted approximately a year earlier yielded LC50s ranging from 0.6 to 2.4 mg/liter  $\text{NH}_3$ .

In summary, there is reasonable evidence that fishes with a history of prior exposure to some sublethal concentration of ammonia are better able to withstand an acutely lethal concentration, at least for some period of hours and possibly days. The relative concentration limits for both acclimation and subsequent acute response need better definition and a more complete explanation. Limited data on fluctuating exposures indicate that fish are more susceptible to fluctuating than to constant exposure with the same average  $\text{NH}_3$  concentrations. Much more research is needed to examine

further the effects of fluctuating and intermittent exposures under exposure regimes simulating actual field situations.

(e) Carbon Dioxide

An increase in carbon dioxide concentrations up to 30 mg/liter decreases total ammonia toxicity (Alabaster and Herbert 1954; Allan et al. 1958). CO<sub>2</sub> causes a decrease in pH, thereby decreasing the proportion of un-ionized ammonia in solution. Lloyd and Herbert (1960) found, however, that although total ammonia toxicity was reduced at elevated CO<sub>2</sub> levels, the inverse was true when considering un-ionized ammonia alone; more NH<sub>3</sub> is required in low CO<sub>2</sub>, high pH water to exert the same toxic effect as seen in fish in high CO<sub>2</sub>, low pH water. The explanation presented by Lloyd and Herbert (1960) for the decreased toxicity of NH<sub>3</sub> in low CO<sub>2</sub> water was that CO<sub>2</sub> excretion across the gills would reduce pH, and therefore NH<sub>3</sub> concentration, in water flowing over the gills.

The basic flaw in Lloyd and Herbert's (1960) hypothesis has been discussed in Broderius et al. (1977). CO<sub>2</sub> will only form protons very slowly in water at the tested temperature. The uncatalyzed CO<sub>2</sub> hydration reaction has a half-time of seconds or even minutes (e.g., at pH 8: 25 seconds at 25 C, 300 seconds at 0 C (Kern 1960)), and water does not remain in the opercular cavity for more than a few seconds, and at the surface of a gill lamella for about 0.5 to 1 second (Randall 1970; Cameron 1979). Thus the liberation of CO<sub>2</sub> will have little, if any, effect on water pH or, therefore, NH<sub>3</sub> levels while the water body is in contact with the gills. Hence the liberation of CO<sub>2</sub> across the gills can have little, if any, effect on the NH<sub>3</sub> gradient across the gills between water and blood. Szumski et al. (1982) hypothesized that in the course of its excretion CO<sub>2</sub> is converted in the gill epithelium to H<sup>+</sup> and HCO<sub>3</sub><sup>-</sup> which then pass

directly into the gill chamber where they cause an instantaneous pH reduction. Their interpretation of the published literature on fish respiratory physiology is questionable, and experimental evidence in support of their evaluation is required before it can be given serious consideration.

(f) Salinity

Herbert and Shurben (1965) reported that the resistance of yearling rainbow trout to ammonium chloride increases with salinity up to levels of 30-40 percent seawater; above that level, resistance appears to decrease. Katz and Pierro (1967) tested fingerling coho salmon at salinity levels of 20 to 30 parts per thousand (57 to 86 percent salt water) and found that toxicity of an ammonia-ammonium waste increased as salinity increased. These findings are in agreement at the levels tested with those of Herbert and Shurben (1965). Atlantic salmon were exposed to ammonium chloride solutions for 24 hours under both freshwater and 30 percent saltwater conditions; LC50s (Table 5) were 0.15 and 0.3 mg/liter  $\text{NH}_3$ , respectively, in the two different waters (Alabaster et al. 1979). For chinook salmon parr, Harader and Allen (1983) also found resistance to increase (by about 500%) as salinity increased to almost 30% seawater, with declines occurring as salinity increased even further.

As was discussed in Willingham et al. (1979), decreased  $\text{NH}_3$  toxicity with increased salinity may be partially explained, at least for low salinity levels, by the fact that there is a slight decrease in the  $\text{NH}_3$  fraction of total ammonia as ionic strength increases in dilute saline solutions (Thurston et al. 1979). At higher salinity levels, however, the toxicity to fishes of ammonia solutions must be attributable to some mechanism or mechanisms other than the changes in the  $\text{NH}_4^+/\text{NH}_3$  ratio. Further work

is needed to confirm results already reported and to clarify the observed mitigating effect of total dissolved solids.

(g) Presence of Other Chemicals

The presence of other chemicals may have an effect on ammonia toxicity, and some experimental work has investigated this topic. Herbert and Vandyke (1964), testing rainbow trout, determined the 48-hour LC50 for a solution of ammonium chloride and that for a solution of copper sulfate. They reported that a solution containing a mixture of one half of each of these LC50s was also the 48-hour LC50 for the two toxicants combined; i.e., the toxic response was simply additive. This information was also reported by the Ministry of Technology, U.K. (1964); it is not clear whether this was a separate study or the same study.

Shemchuk (1971) measured copper uptake in two-year old carp from solutions of  $\text{Cu}(\text{NH}_3)_4^{2+}$ ; copper uptake in various fish tissues was reported, but no information was provided about toxicity. Vamos and Tasnadi (1967) applied cupric sulfate to a "carp pond" to reduce the concentration of free ammonia and reported that this measure proved successful to reduce the toxic effect of ammonia; few details were provided.

Ministry of Technology, U.K. (1962, 1963) reported on the results of tests on rainbow trout in which 48-hour LC50s were determined for solutions of ammonium chloride, zinc sulfate and mixtures of these two salts. A fraction of each of those 48-hour LC50s, when combined in such a way that those fractions equalled unity, provided a mixture with a 48-hour LC50 equal to that of either of the two toxicants alone. Results were similar for tests conducted in waters with alkalinities of 240 and 50 mg/liter as  $\text{CaCO}_3$ .

Herbert (1962) studied the toxicity to rainbow trout of ammonia-phenol mixtures. The mixtures contained fractions of the 48-hour LC50s of phenol and of ammonia; the combined fractions equaled unity. The toxicity of the combined fractions approximated the toxicity of either phenol or ammonia when tested separately but under test conditions of similar water chemical characteristics. The same information was reported by Ministry of Technology, U.K. (1961); it is not clear whether this was a separate study or the same study.

Brown et al. (1969) conducted 48-hour tests on rainbow trout in mixtures of ammonia, zinc, and phenol; the mixture contained equal portions, by 48-hour LC50, of the three toxicants. They reported that each chemical nominally contributed equally to the toxicity. In a second series of three tests in which the mixture was adjusted to include approximately 75 percent of a 48-hour LC50 of one toxicant and the balance split equally between the other two, they reported that the principal toxicant contributed about three-fourths of the toxicity.

Broderius and Smith (1979), in 96-hour flow-through tests with rainbow trout, reported a synergistic effect for  $\text{NH}_3$  and HCN except at extremely low concentrations. Rubin and Elmaraghy (1976, 1977) estimated the individual and joint toxicities of ammonia and nitrate to guppy fry; the toxicities of the two in mixture were additive, except at very low ammonia-to-nitrate ratios. Tomasso et al. (1980) reported that elevated calcium levels increased the tolerance to ammonia of channel catfish.

## Derivation of the Final Acute Value for Fresh Water

### (a) pH Dependence of Acute Ammonia Toxicity

Erickson (1985) reviewed available data on the pH-dependence of un-ionized ammonia LC50s. For the pH 5 to 9 range, he noted that the principal feature of plots of  $\log(\text{LC50})$  versus pH was a declining slope with increasing pH, with the slope apparently approaching zero at the upper part of the range and approaching a constant value at the lower part of the range. He proposed the following empirical model for such behavior:

$$\text{LC50} = \frac{\text{LIM}}{1 + 10^{\text{SLP}(\text{PHT}-\text{pH})}} \quad (1)$$

where LIM = the asymptotic LC50 at high pH, SLP = the asymptotic slope at low pH, and PHT = a transition pH. The fit of this model to available data was found to generally be good, with the  $R^2$  varying from 60% to >99% for all data sets and residual errors being in the range of uncertainty for toxicity testing. Furthermore, for those data sets with certain minimum data requirements necessary for critically evaluating model fit (at least 6 observations spread over at least 4 distinct pHs with a range of at least 1.5), the fit was very good (Figure 1), with  $R^2$ s ranging from 96% to >99%. The parameter SLP was generally found to be similar among data sets and a pooled analysis estimated it to be 1.03, indistinguishable from 1.0 both for practical purposes and from a standpoint of statistical significance. The parameter PHT was also found to be similar among data sets, usually being in the pH 7 to 8 range.

This empirical model, however, did not incorporate indications in some data (Figure 1) that LC50s may be declining as pH increases over 8.5. To minimize possible errors associated with such behavior, the model was slightly modified for application here by requiring that LC50s are constant

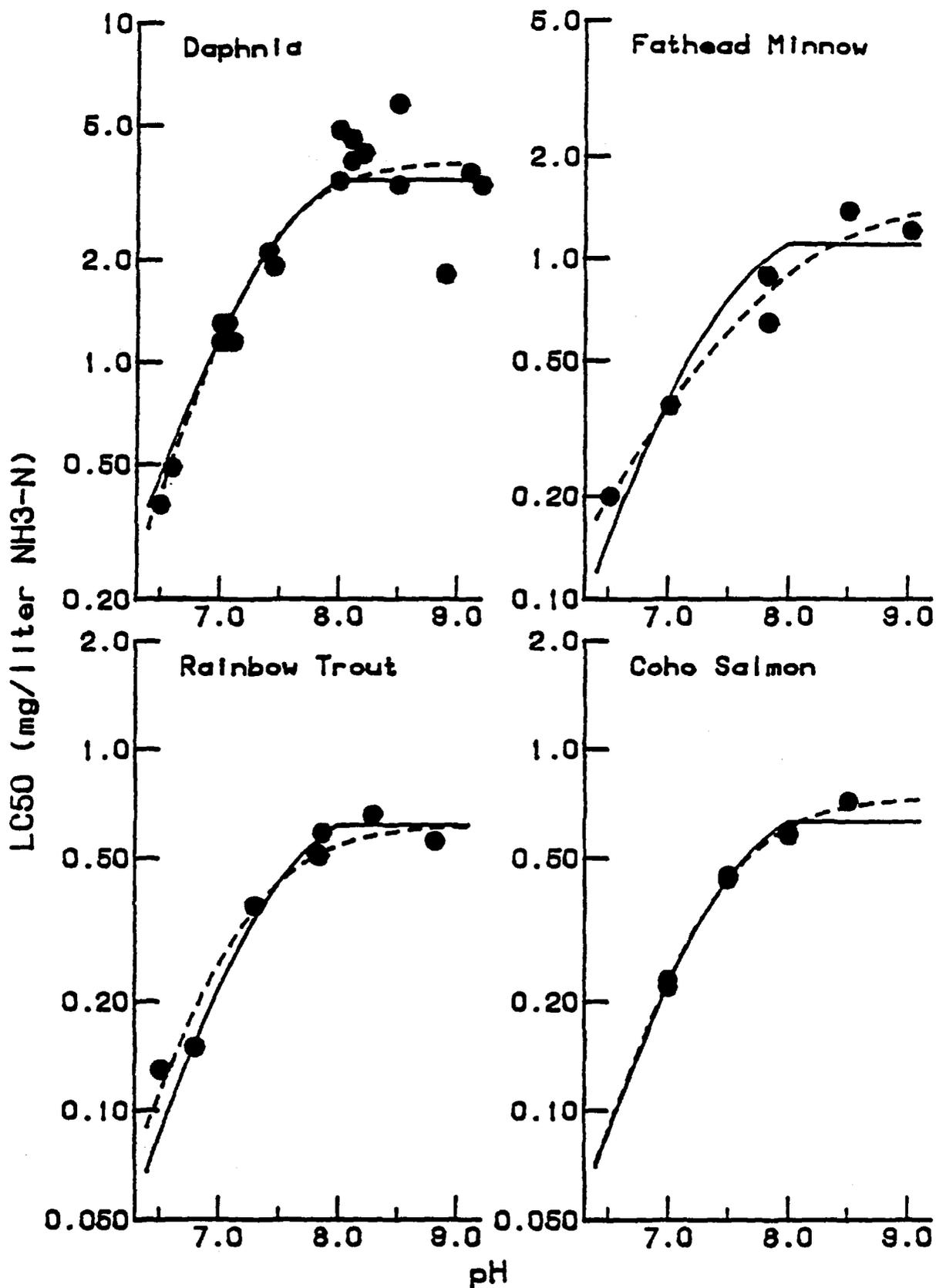


Figure 1. Acute NH<sub>3</sub> toxicity at different pH values (data from Tabata 1962, Thurston et al. 1981c, Robinson-Wilson and Seim 1975). Dashed lines = regression based on individual data set; solid lines = regression based on pooled data sets and modified model.

at pH 8 and above; this will tend to cause the fitted curve to pass slightly below the apparent peak at pH 8.5 and closer to the data near pH 9.0.

Based on the behavior of SLP noted above, the model was further modified by assuming the parameter SLP exactly equalled 1.0 and dropping it from the model. This is equivalent to assuming the pH dependence of ammonia toxicity is due to joint toxicity of  $\text{NH}_3$  and  $\text{NH}_4^+$ , but the interpretation here will remain strictly empirical and none of the ramifications of such a mechanism, such as temperature dependence of PHT, will be considered here due to absence of suitable data.

The modified model for pH dependence therefore was:

$$\begin{aligned} \text{LC50} &= \text{LC50}(\text{pH}=8) && ; \text{pH} \geq 8 \\ \text{LC50} &= \text{LC50}(\text{pH}=8) \frac{1 + 10^{\text{PHT}-8}}{1 + 10^{\text{PHT}-\text{pH}}} && ; \text{pH} < 8 \end{aligned} \quad (2)$$

where the parameter LIM has been replaced with  $\text{LC50}(\text{pH}=8) \cdot (1 + 10^{\text{PHT}-8})$ , thus adopting a reference pH of 8, where the imposed plateau begins.

Eventual application of this model to generating a criterion requires that it contain only one parameter dependent on test organism, since having more than one parameter would require that there be LC50s from multiple pHs for every test organism, when, in fact, such information is available for few of the tests in Table 1. Clearly,  $\text{LC50}(\text{pH}=8)$  is likely to be organism-dependent, since it represents the sensitivity under reference conditions. PHT must therefore be assumed to be constant among test organisms, at least until additional testing allows separate estimates for PHT for different taxa. This assumption is justified to some extent by the observed similarity of PHT among species noted above.

Using the modified model, a pooled regression analysis of the data in Figure 1 was conducted employing the procedures of Erickson (1985), resulting

in an estimate 7.4 for the parameter PHT. The resulting model fit was good, with an  $R^2$  for the pooled data set of 96%, little worse than achieved with individual analyses of each data set using the original model (Equation 1). The slight decrease in fit was solely due to using pooled estimates for PHT and SLP. The imposition of the plateau at  $\text{pH} > 8$  actually improved the fit slightly. The fit from this pooled analysis is indicated in Figure 1.

The final relationship adopted for the pH-dependence of acute ammonia toxicity therefore was:

$$\begin{aligned} \text{LC50} &= \text{LC50}(\text{pH}=8) && ; \text{pH} \geq 8 && (3) \\ \text{LC50} &= \frac{\text{LC50}(\text{pH}=8) \cdot 1.25}{1 + 10^{7.4-\text{pH}}} && ; \text{pH} < 8 \end{aligned}$$

Although the proposed relationship cannot be considered universally applicable or without error, the alternatives of using no pH relationship or of basing criteria only on species tested over a range of pHs are clearly less desirable. A relationship which can be applied with more confidence requires further experimentation. Of course, in site-specific applications, if evidence exists for significantly different pH relationships for species of importance to setting criteria, appropriate modifications should be considered.

#### (b) Temperature Dependence of Acute Ammonia Toxicity

Erickson (1985) reviewed available data on the temperature-dependence of un-ionized ammonia LC50s. For data sets with more than two tested temperatures, he noted that the principal feature was an approximately linear relationship of  $\log(\text{LC50})$  versus temperature (Figure 2). He noted some indication of declining slopes with increasing temperature, but due to the data uncertainty this trend could not be adequately verified or quantified.

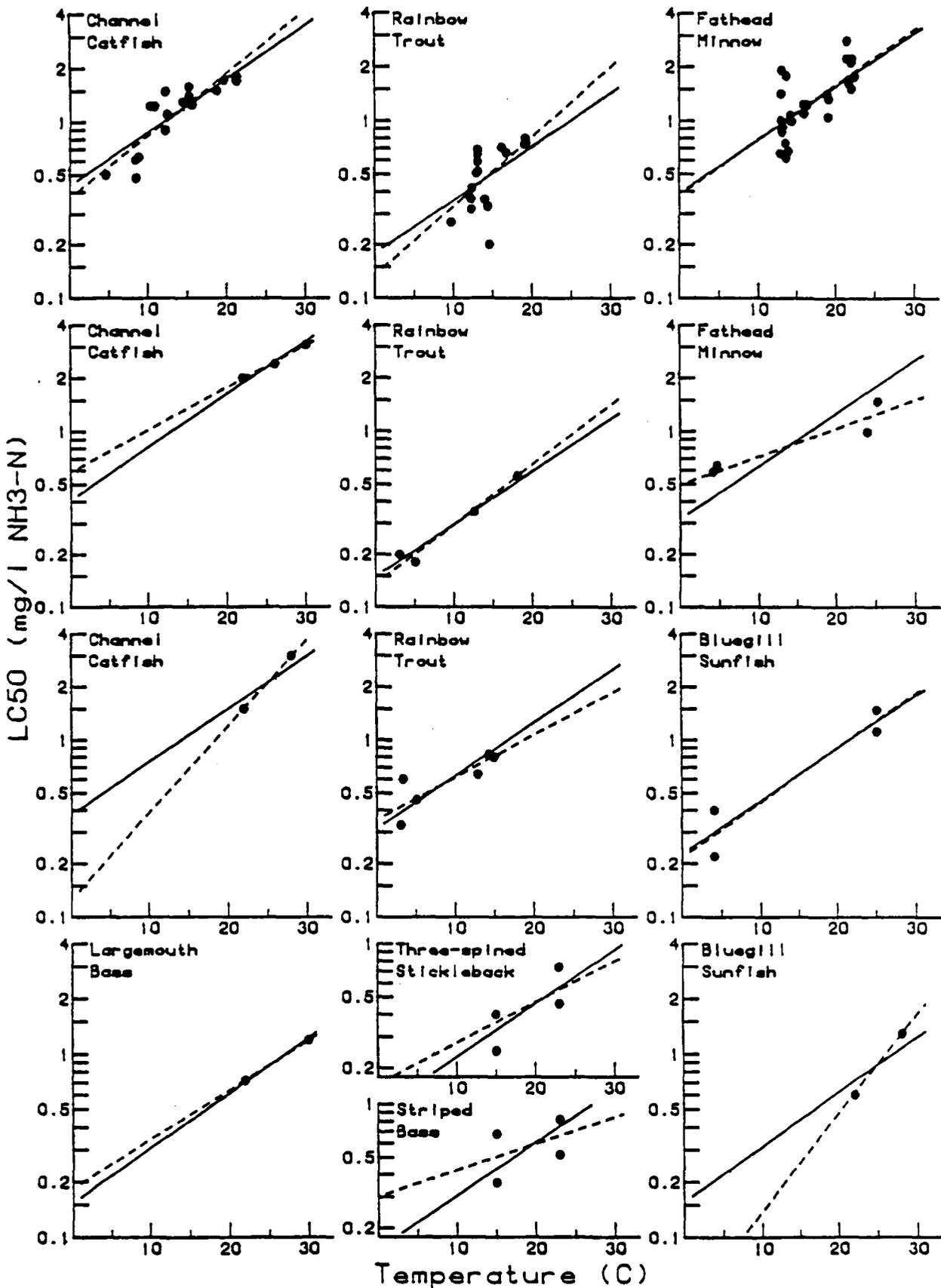


Figure 2. Acute NH<sub>3</sub> toxicity at different temperatures (data from Cary 1976, Thurston and Russo 1983, Thurston et al. 1983, Colt and Tchobanoulous 1976, Ministry of Technology 1968, Reinbold and Pescitelli 1982, Roseboom and Richey 1977, Hazel et al. 1971). Dashed lines indicate individual regressions; solid lines indicate pooled regression.

He therefore proposed the following empirical model for temperature dependence of ammonia toxicity:

$$LC50 = LCR \cdot 10^{SLT(T-20)} \quad (4)$$

where LCR is the LC50 at a reference temperature of 20 C and SLT is the slope of  $\log(LC50)$  versus temperature. Slope estimates were found to not vary significantly among data sets, which included variation in both organisms and temperature range tested. Slopes varied from 0.016 to 0.054, with an arithmetic mean of 0.03; interestingly, this is approximately equivalent to total ammonia being constant with temperature. The relationship adopted for the temperature dependence of acute ammonia toxicity in fish therefore was:

$$LC50 = LC50(T=20) \cdot 10^{0.03(T-20)} \quad (5)$$

where the parameter LCR has been replaced with the more descriptive term  $LC50(T=20)$ , consistent with the terminology adopted for the reference LC50 in the pH relationship. For invertebrates, no temperature relationship will be used; this assumption will cause little error because available data suggest that temperature effects are not as marked as in fish and because invertebrates are generally insensitive to ammonia and thus do not markedly influence the criteria.

However, this relationship cannot be applied without some limitations. As noted above, there is some indication of declining slopes as temperature increases. Also, available data sets were restricted to temperatures, at the high end, that were optimal or only marginally suboptimal. Thus, extrapolation of this relationship to high temperatures must be restricted. It can be used to adjust the data in Table 1 to reference conditions since the tests for each fish were rarely conducted under unfavorably high temperatures, but it should not be used for generating criteria at temperatures high enough to constitute a stress to an organism. Where criteria are necessary for such

high temperatures, it is recommended here that it be the same  $\text{NH}_3$  concentration as at the upper end of the temperature range considered favorable for the organism (i.e., SLT is assumed to be 0.00 rather than 0.03 between the upper end of the favorable range and higher temperatures). The final relationship adopted for the temperature dependence of the national criterion therefore was:

$$\begin{aligned} \text{LC50} &= \text{LC50}(T=20) \cdot 10^{0.03(\text{TCAP}-20)}; & T \geq \text{TCAP} & \quad (6) \\ \text{LC50} &= \text{LC50}(T=20) \cdot 10^{0.03(T-20)} & ; & T < \text{TCAP} \end{aligned}$$

For the purposes of the national criteria, when salmonid fish or other sensitive coldwater species are present, the temperature relationship will be applied only up to 20 C (TCAP = 20). Temperatures much higher than this are detrimental to coldwater species and data on the temperature dependence of ammonia toxicity for such species extends only up to 18 C. Thus, use of the temperature relationship above 20 C is of doubtful validity and un-ionized ammonia criteria at high temperatures will be assumed to be no higher than at 20 C. For sites without salmonids and other sensitive coldwater species, TCAP = 25 C will be used; a higher TCAP (30 C) may be justified on a site-specific basis when strictly warmwater species are present. The increase in the temperature cap should not be beyond where there is data to suggest that the tolerance of the most sensitive site genera continues to increase with temperature and should not result in a FAV at any temperature that is significantly greater than the AVs at the higher temperatures tested for the most sensitive genera at the site.

As for the pH relationship, this proposed temperature relationship is imperfect due to the limited database, but the alternatives of using no relationship or of restricting criteria to narrow temperature ranges where sufficient data is available are clearly less desirable. Of course, also as

for pH, where data for a species of importance to the setting of a criterion contradicts the above assumptions regarding temperature, appropriate modifications should be made.

(c) Application of pH and Temperature Relationships of Acute Ammonia Toxicity to Determination of Final Acute Values

A Species Mean Acute Value (SMAV) is the geometric average of the acute values (AVs), usually LC50s, available for a given species. A Genus Mean Acute Value (GMAV) is the geometric average of the SMAVs available for a given genus. A Final Acute Value (FAV) for a material is an estimate of the GMAV at the 0.05 cumulative proportion in the cumulative distribution of GMAVs for all genera tested for that material. These computations (see Guidelines) are not a subject of this discussion, but their application to pH and temperature dependent data is.

The existence of pH and temperature dependence in AVs requires that they be adjusted to a common reference pH and temperature basis before computing a FAV. After a FAV at this reference pH and temperature is computed, it can be applied to other pHs and temperatures using the same equations used to correct the AVs.

The reference pH and temperature are arbitrary insofar as final results are concerned. The reference temperature selected here was 20 C, as selected for equation 5, and the reference pH was 8, as selected for equation 2. These reference conditions furthermore are moderate and near those of most tests in Table 1, allowing more easy comparisons of values.

It is assumed here that the effects of pH and temperature are not significantly correlated. There are currently no data to contradict this assumption, much less mathematically model such a correlation. Equations 3 and 5 can then be combined as follows to provide a unified equation for

adjusting acute values measured at any pH and temperature to the reference conditions.

$$AV_{ref} = AV(pH, T) \cdot FT \cdot FPH \quad (7)$$

where:

$$\begin{aligned} FT &= 10^{0.03(20-T)} ; \text{ for fish} \\ &= 1 ; \text{ for invertebrates} \\ FPH &= 1 ; \text{ pH } \geq 8 \\ &= \frac{1 + 10^{7.4-pH}}{1.25} ; \text{ pH } < 8 \end{aligned}$$

Once all AVs available for establishing a criterion are adjusted to  $AV_{ref}$ s, the SMAV for each species at reference conditions ( $SMAV_{ref}$ ) can be computed as the geometric average of the  $AV_{ref}$ s for that species and the GMAV for each genus at reference conditions ( $GMAV_{ref}$ ) can be computed as the geometric average of the  $SMAV_{ref}$ s for that genus. The FAV at reference conditions ( $FAV_{ref}$ ) then can be computed from the  $GMAV_{ref}$ s available by the same procedures used for computing FAVs from GMAVs for any material. A FAV at a particular pH and temperature can finally be computed by reversing equation 7 (and also applying the restriction from equation 6 that  $FT = 10^{0.03(20-TCAP)}$  for  $T \geq TCAP$ ).

Application of these techniques to the data proceeded as follows. AVs from Table 1 were adjusted for temperature and pH and averaged to obtain the  $SMAV_{ref}$ s and  $GMAV_{ref}$ s reported in Table 3. The fifth percentile was estimated, by the Guidelines method, to be 0.70 mg/liter  $NH_3$ . However, the rainbow trout data in Table 1 indicate that sexually mature fish ( $\geq 1$  kg) are significantly more sensitive than the average of the tested fish. Since a species is not protected if each life stage is not protected, the

FAV<sub>ref</sub> was lowered to 0.52, the geometric average of the AV<sub>refs</sub> of rainbow trout in this size range. Thus, the equation for the FAV is:

$$FAV(pH, T) = 0.52/FT/FPH \quad (8)$$

where:

$$FT = 10^{0.03(20-TCAP)}; \quad TCAP \leq T \leq 30$$

$$= 10^{0.03(20-T)} \quad ; \quad 0 \leq T \leq TCAP$$

$$FPH = 1 \quad ; \quad 8.0 \leq pH \leq 9.0$$

$$= \frac{1 + 10^{7.4-pH}}{1.25} \quad ; \quad 6.5 \leq pH \leq 8.0$$

$$TCAP = 20 \text{ C; Salmonids present}$$

$$= 25 \text{ C; Salmonids absent}$$

(d) Application of the FAV to a Criterion to Protect Against Acute Toxicity

As specified in the Guidelines, the criterion to protect against acute toxicity will be based on requiring that 1-hour average concentrations not exceed, more often on the average than once every 3 years, one-half of the FAV specified in Equation 8 above. For ammonia there is considerable evidence that this short averaging period is justified, even though the FAV is based on tests with a typical duration of 96 hours. The acute response of some fish to ammonia can be very rapid. For example, McCormick et al. (1984) reported LC50s with green sunfish to be only 0% to 40% higher at 3 hours than at 96 hours for the pH range 7.2-8.7; furthermore, this did not take into consideration any delayed mortality at the shorter time, so the differences may be even smaller. Ball (1967) reported a 3-hour LC50 for rainbow trout to be just 50% greater than the asymptotic LC50, again not accounting for delayed mortality (other species, however, did not have such an extreme relationship). Effects of simple exposures of shorter duration are unknown, but LC50s for 1- to 2-hour periods quite possibly could also be just

marginally above that at 96 hours, especially if the 1- or 2-hour period is preceded and/or followed by concentrations which are not markedly lower.

Therefore, a criterion based on 96-hour LC50s cannot be treated as an average over any appreciable fraction of the test duration, since such averaging implicitly allows significant excursions over the criterion for an appreciable fraction of the averaging period and thus allows the occurrence of a time sequence of concentrations at lesser intervals that would have greater toxicity than is intended by the criterion. For example, in the case of the data cited above, even a 4-hour averaging period would allow concentrations of 2- to 3-hour duration that could have an impact greater than desired.

Experiments on the effects of fluctuating ammonia concentrations also support the use of extremely short averaging periods. Thurston et al. (1981a) exposed rainbow trout to ammonia concentrations that varied from virtually zero to a peak over a 12- to 24-hour cycle and reported LC50s based on peak concentrations to be only 16-39% higher than those based on 96-hour constant concentration tests and that LC50s based on the average of the fluctuating concentrations were 25-42% less than the 96-hour LC50s for all tests except those on large fish, which tolerated slightly higher peaks. Since concentrations were near or at the peak for only two hours, this suggests that, although some excursions above 96-hour LC50s are permissible at short durations, the allowable excursions are not large enough to allow averaging periods of more than a few hours. Brown et al. (1969) exposed rainbow trout to fluctuating concentrations with an average equal to the 48-hour constant concentration LC50, with the fluctuations varying between 1.5X and 0.5X the average over either a 2- or 4-hour cycle. They found that toxicity using the 2-hour cycle was similar to that under constant exposure,

but was markedly higher using the 4-hour cycle. This indicates that, even for a modest 50% excursion over the constant concentration LC50, an averaging period of longer than 2 hours is inappropriate. For more marked excursions, shorter periods may be necessary.

Thus, the 1-hour averaging period specified in the Guidelines is reasonable for ammonia. In fact, this duration may be too long if substantial excursions above the average occur within the hour. Therefore, it is further specified here that this 1-hour average criterion is not applicable to situations where concentrations exceed 1.5 times the average within the 1-hour period. The 1.5 factor was based on such an excursion being acceptable based on the fluctuating exposure studies discussed above, with no evidence that greater excursions are tolerable.

#### Saltwater Invertebrates

Data on acute toxicity of ammonia to saltwater invertebrate species are very limited. LC50s are summarized in Table 1 for five species representing five families. A 96-hour LC50 (Table 1) of 1.5 mg/liter  $\text{NH}_3$  was reported (Linden et al. 1979) for the copepod, Nitocra spinipes. Lethal effects of  $\text{NH}_4\text{Cl}$  on the quahog clam (Mercenaria mercenaria) and eastern oyster (Crassostrea virginica) were studied by Epifanio and Srna (1975) (Table 1). There was no observed difference in susceptibilities between juveniles and adults of the two species. Armstrong et al. (1978) conducted acute toxicity tests (6 days) on ammonium chloride using prawn larvae (Macrobrachium rosenbergii). LC50s (Tables 1, 5) were highly pH-dependent. Acute toxicity of  $\text{NH}_4\text{Cl}$  to penaeid shrimp was reported as a 48-hour composite LC50 of 1.6 mg/liter  $\text{NH}_3$  for seven species pooled, including the resident species Penaeus setiferus (Wickins 1976). The acute toxicity of  $\text{NH}_4\text{Cl}$  to the

caridean prawn, M. rosenbergii, was reported (Wickins 1976) as LT50s of 1700-560 minutes at concentrations of 1.74 to 3.41 mg/liter NH<sub>3</sub> (Table 5). Hall et al. (1978) measured the acute toxicity of NH<sub>4</sub>Cl to grass shrimp (Palaemonetes pugio) (Table 5). Catedral and coworkers (1977a,b) investigated the effect of NH<sub>4</sub>Cl on survival and growth of Penaeus monodon; larvae had lower tolerance to ammonia compared with postlarvae. Brown (1974) reported a time to 50 percent mortality of 106 min for nemertine worm (Cerebratulus fuscus) at 2.3 mg/liter NH<sub>3</sub> (Table 5).

Effects of NH<sub>4</sub>Cl solutions on American lobster (Homarus americanus) were studied by Delistracy et al. (1977). Their tests were performed on fourth stage larvae which they believed to be the most sensitive life stage, or nearly so. They reported a 96-hour LC50 (Table 1) of 2.2 mg/liter NH<sub>3</sub> and an incipient LC50 (Table 5) of 1.7 mg/liter NH<sub>3</sub>. A "safe" concentration of 0.17 mg/liter NH<sub>3</sub> was tentatively recommended.

### Saltwater Fishes

Very few acute toxicity data are available for saltwater fish species. Holland et al. (1960) reported the critical level for chinook salmon (Oncorhynchus tshawytscha) to be between 0.04 and 0.11 mg/liter NH<sub>3</sub> and for coho salmon to be 0.134 mg/liter NH<sub>3</sub>. A static test with coho salmon provided a 48-hour LC50 (Table 5) of 0.50 mg/liter NH<sub>3</sub> (Katz and Pierro 1967). Atlantic salmon smolts and yearling rainbow trout tested for 24 hours in 50 and 75 percent saltwater solutions exhibited similar sensitivities to ammonia (Ministry of Technology, U.K. 1963).

Holt and Arnold (1983) report a 96-hour LC50 of 0.47 mg/liter NH<sub>3</sub> (Table 1) for red drum (Sciaenops ocellatus). Venkataramiak (1981a) found

96-hour LC50s (Table 1) of 1.2-2.4 mg/liter  $\text{NH}_3$  for striped mullet (Mugil cephalus) and 0.69 mg/liter  $\text{NH}_3$  for planehead filefish (Monacanthus hispidus).

## CHRONIC TOXICITY TO AQUATIC ANIMALS

The following discussion of chronic and partial chronic ammonia toxicity includes both data used in the derivation of the Final Chronic Value (Table 2 data) and data that were not included in the criterion derivation, but that are important for an understanding of long-term lethal and sublethal effects of ammonia on aquatic organisms (Table 5 data).

### Freshwater Invertebrates

Few studies have been conducted on long-term exposure of freshwater invertebrates to ammonia, and life-cycle tests were conducted only for cladocerans.

The lowest concentrations affecting reproduction in two life-cycle tests (Table 2) with D. magna were 0.74 and 0.76 mg/liter NH<sub>3</sub> (Russo et al. 1985); a 28-day LC50 of 1.53 mg/liter NH<sub>3</sub> was reported. In a chronic test (Table 2) conducted by Reinbold and Pescitelli (1982a), reproduction and growth of D. magna were affected at a concentration of 1.6 mg/liter NH<sub>3</sub>. A life-cycle test (Table 2) with C. acanthina (Mount 1982) showed effects on reproduction at a concentration of 0.463 mg/liter NH<sub>3</sub>.

Two tests lasting 42 days were conducted by Anderson et al. (1978) on NH<sub>4</sub>Cl with the fingernail clam, Musculium transversum (Table 5). Significant mortalities (67 and 72 percent) occurred in both tests at a concentration of 0.7 mg/liter NH<sub>3</sub>. In one of the experiments, significant reduction in growth was observed after 14 days of exposure to 0.41 mg/liter NH<sub>3</sub>. Sparks and Sandusky (1981) reported that fingernail clams exposed to 0.23 and 0.63 mg/liter NH<sub>3</sub> incurred 36 and 23 percent mortality, respectively, in four weeks; after six weeks, 47 percent mortality occurred at 0.073 mg/liter NH<sub>3</sub>, and 83 percent mortality occurred at 0.23 and 0.63

mg/liter  $\text{NH}_3$ . No growth at all occurred in all test chambers (concentrations of 0.036 mg/liter  $\text{NH}_3$  and higher) other than the control after six weeks (Table 5).

Two partial chronic tests, of 24- and 30-days' duration, were conducted by Thurston et al. (1984a) with the stonefly Pteronarcella badia (Table 5). Adult stonefly emergence was delayed with increasing ammonia concentration, and little or no emergence occurred at concentrations exceeding 3.4 mg/liter  $\text{NH}_3$ . There was no significant relationship between food consumption rates of nymphs and concentrations up to 6.9 mg/liter  $\text{NH}_3$ . LC50s for 24- and 30-day exposures were 1.45 and 4.57 mg/liter  $\text{NH}_3$ , respectively.

#### Freshwater Fishes

A number of researchers have conducted long-term ammonia exposures to fishes, including complete life-cycle tests on rainbow trout and fathead minnows. Several kinds of endpoints have been studied, including effects on spawning and egg incubation, growth, survival, and tissues.

The effects of prolonged exposure (up to 61 days) to ammonia of pink salmon early life stages was studied by Rice and Bailey (1980). Three series of exposures were carried out, beginning at selected times after hatching: for 21 days prior to completion of yolk absorption, for 40 days up to 21 days before yolk absorption, and for 61 days up to yolk absorption. All test fish were sampled for size when the controls had completed yolk absorption.  $\text{NH}_3$  concentrations ranged from 0 (control) up to 0.004 mg/liter. For fry at the highest concentration of 0.004 mg/liter  $\text{NH}_3$  (Table 2), significant decreases in weight were observed for all three exposure groups. At a concentration of 0.0024 mg/liter  $\text{NH}_3$  (Table 2) the group of fry exposed for 40 and 61 days were significantly smaller, whereas a concentration of 0.0012

mg/liter had no significant effect on growth. Effects were consistently more adverse for the 61-day-exposed fish.

Thurston et al. (1984b) tested rainbow trout in a laboratory study in which adult fish exposed for five months to concentrations of ammonia from 0.01 to 0.07 mg/liter  $\text{NH}_3$  spawned of their own volition; baskets containing crushed rock served as the spawning substrate. There was no correlation between ammonia concentration and numbers of egg lots spawned, total numbers of eggs produced, or numbers of eggs subsequently hatched. Parental fish were exposed for 11 months, the first filial generation ( $F_1$ ) for four years, and the second filial generation ( $F_2$ ) for five months. Pathologic lesions were observed in both parental and  $F_1$  fish when ammonia concentrations reached and exceeded 0.04 mg/liter  $\text{NH}_3$  (Table 2). Measurements of blood ammonia concentrations in four-year-old  $F_1$  fish showed an increase when test water conditions reached or exceeded 0.04 mg/liter  $\text{NH}_3$ . Trout exposed for 52 months from day of hatching showed no relationship between growth and concentration at 10, 15, 21, and 52 months.

Burkhalter and Kaya (1977) tested ammonia at concentrations from 0.06 to 0.45 mg/liter  $\text{NH}_3$  on fertilized eggs and resultant sac fry of rainbow trout. Eggs were incubated at 12 C for 25 days in one test and at 10 C for 33 days in another; fry were maintained for 42 days. In neither test was there a concentration response on egg mortality or on incubation time. Retardation in early growth and development occurred at  $\text{NH}_3$  concentrations as low as 0.06 mg/liter  $\text{NH}_3$ , the lowest concentration they tested (Table 2). Fish exposed to 0.12 mg/liter  $\text{NH}_3$  (Table 2) required one week longer than controls to achieve a free-swimming state; fish at 0.34 and 0.45 mg/liter  $\text{NH}_3$  did not achieve a free-swimming state during a 42-day test period. A 21-day LC50 of 0.30 mg/liter  $\text{NH}_3$  was obtained (Table 5). For sac fry exposed for 42 days

after hatching, hypertrophy of secondary gill lamellae epithelium occurred at 0.23 mg/liter  $\text{NH}_3$ , and karyolysis and karyorrhexis in the secondary gill lamellae were observed after 28 days at 0.34 mg/liter  $\text{NH}_3$  and higher.

Calamari et al. (1977, 1981) exposed rainbow trout to ammonium chloride solutions for 72 days, beginning one day after fertilization and ending when fry were fed for 30 days. A 72-day LC50 of 0.056 mg/liter  $\text{NH}_3$  was calculated (Table 5); 23 percent mortality occurred at a concentration of 0.025 mg/liter  $\text{NH}_3$  (Table 2). Examination of 986 rainbow trout embryos at hatching stage after exposure to  $\text{NH}_3$  concentrations of 0.010 to 0.193 mg/liter for 24 days showed an increase in macroscopic malformations with increasing ammonia concentration. Kinds of deformities observed were varying degree of curvature from median body axis, which in extreme cases produced a complete spiral shape, and various kinds of malformations in the head region with a number of cases of double heads. At the highest concentration tested, 0.193 mg/liter  $\text{NH}_3$ , 60 percent of the observed fish were malformed. Microscopic examination at hatching of 128 larvae from the same exposure showed abnormalities on the epidermis and pronephros that correlated with ammonia concentrations. The epidermis was thickened with an irregular arrangement of the various layers of cells and an increase in the number and dimensions of mucous cells. The pronephros showed widespread vacuolization of the tubule cells, together with a thickening of the wall. Increasing abnormalities were observed after exposure to concentrations over 0.025 mg/liter  $\text{NH}_3$  for epidermis and 0.063 mg/liter  $\text{NH}_3$  for pronephros.

Broderius and Smith (1979) tested four-week-old rainbow trout fry for 30 days at concentrations of ammonia (reported graphically) ranging from ~0.06 to 0.32 mg/liter  $\text{NH}_3$  (Table 5). Growth rate at ~0.06 mg/liter  $\text{NH}_3$  was comparable to that of controls; above ~0.10 mg/liter  $\text{NH}_3$  growth rate

decreased, correlated with increased  $\text{NH}_3$  concentration. The survival at 0.32 mg/liter  $\text{NH}_3$  was reduced to 70 percent that of the controls. Schulze-Wiehenbrauck (1976) tested juvenile rainbow trout, approximately one-half-year-old but of different sizes, for periods of time from two to seven weeks, and at ammonia concentrations from 0.012 to 0.17 mg/liter  $\text{NH}_3$ . He concluded that 0.05 mg/liter  $\text{NH}_3$  caused a slight decrease in growth during the first 14-day interval on nonacclimatized fish, but that decrease was completely compensated in the next growth interval; exposure to 0.13 mg/liter  $\text{NH}_3$  (apparently for 3 or 4 weeks) did not affect growth, food consumption, or food conversion.

Smith (1972) and Smith and Piper (1975) reared young rainbow trout at three concentrations of ammonia (averaging 0.006, 0.012, and 0.017 mg/liter  $\text{NH}_3$ ) for a period of one year. There was no significant difference in fish growth reported among the three concentrations at four months. There was, however, a difference reported at 11 months; the fish at 0.012 and 0.017 mg/liter  $\text{NH}_3$  weighed 9 and 38 percent less than the fish at 0.006 mg/liter. Microscopic examination of tissues from fish exposed to the highest concentration, examined at 6, 9, and 12 months, showed severe pathologic changes in gill and liver tissues. Gills showed extensive proliferation of epithelium which resulted in severe fusion of gill lamellae which prevented normal respiration. Livers showed reduced glycogen storage and scattered areas of dead cells; these were more extensive as exposure time increased.

Ministry of Technology, U.K. (1968) reported on tests in which rainbow trout were exposed for three months to concentrations of 0.069, 0.14, and 0.28 mg/liter  $\text{NH}_3$ . The cumulative mortality of a control group (0.005 mg/liter  $\text{NH}_3$ ) was ~2 percent. Cumulative mortality at 0.069 and 0.14 mg/liter  $\text{NH}_3$  was ~5 percent, and that at 0.28 mg/liter was ~15 percent.

Reichenbach-Klinke (1967) performed a series of one-week ammonia tests on 240 fishes of nine species (including rainbow trout, goldfish, northern pike (Esox lucius), carp, and tench) at concentrations of 0.1 to 0.4 mg/liter  $\text{NH}_3$ . He observed swelling of and diminishing of the number of red blood cells, inflammations, and hyperplasia. Irreversible blood damage occurred in rainbow trout fry in un-ionized ammonia concentrations above 0.27 mg/liter  $\text{NH}_3$ . He also noted that low  $\text{NH}_3$  concentrations inhibited the growth of young trout and lessened their resistance to disease.

Smart (1976) exposed rainbow trout to 0.30 to 0.36 mg/liter  $\text{NH}_3$  (Table 5); 81 percent mortality occurred over the 36-day duration of the test, with most deaths occurring between days 14 and 21. Microscopic examination of the gills of exposed rainbow trout revealed some thickening of the lamellar epithelium and an increased mucous production. The most characteristic feature was a large proportion of swollen, rounded secondary lamellae; in these the pillar system was broken down and the epithelium enclosed a disorganized mass of pillar cells and erythrocytes. Gill hyperplasia was not a characteristic observation.

Fromm (1970) exposed rainbow trout to <0.0005 and 0.005 mg/liter  $\text{NH}_3$  for eight weeks. Subsequent examination of the gill lamellae of fish from the trace concentrations showed them to be long and slender with no significant pathology. Fish exposed to 0.005 mg/liter  $\text{NH}_3$  had shorter and thicker gill lamellae with bulbous ends; some consolidation of lamellae was noticed. Photomicrographs revealed that many filaments showed limited hyperplasia accompanied by the appearance of cells containing large vacuoles whose contents stained positive for protein. Other lamellae showed a definite hyperplasia of the epithelial layer, evidenced by an increase in the number of cell nuclei.

Thurston et al. (1978) studied the toxicity of ammonia to cutthroat trout fry in flow-through tests which lasted up to 36 days (Table 5). Results of duplicate tests on 1.0-g fish both showed 29- and 36-day LC50s of 0.56 mg/liter  $\text{NH}_3$ . Duplicate tests on 3.3-g fish provided 29-day LC50s of 0.37 and 0.34 mg/liter, slightly less than those of the 1.0-g fish. Tissues from heart, gastrointestinal tract, and thymus of cutthroat trout fry exposed to 0.34 mg/liter  $\text{NH}_3$  for 29 days were comparable to those of control fish. However, gills and kidneys of exposed fish showed degenerative changes. Gills showed hypertrophy of epithelium, some necrosis of epithelial cells, and separation of epithelium due to edema; kidneys showed mild hydropic degeneration and accumulation of hyaline droplets in renal tubule epithelium; reduced vacuolation was observed in livers. Daoust and Ferguson (1984) were unable to find rainbow trout gill lesions in  $\text{NH}_3$  concentrations of 0.2-0.4 mg/liter.

Samylin (1969) studied the effects of ammonium carbonate on the early stages of development of Atlantic salmon. The first set of experiments (temperature = 13 C) was conducted within the range 0.001 to >6.6 mg/liter  $\text{NH}_3$  beginning with the "formed embryo" stage; the experiment lasted 53 days. Accelerated hatching was observed with increasing  $(\text{NH}_4)_2\text{CO}_3$  concentrations, but concentrations  $\geq 0.16$  mg/liter  $\text{NH}_3$  were lethal in 12-36 hours to emerging larvae. Because  $(\text{NH}_4)_2\text{CO}_3$  was used as the toxicant, the pH in the test aquaria increased from 6.7 to 7.6 with increasing  $\text{NH}_3$  concentration. Growth inhibition was observed at 0.07 mg/liter  $\text{NH}_3$  (Table 2). Tissue disorders were observed in eyes, brains, fins, and blood of Atlantic salmon embryos and larvae exposed to concentrations from 0.16 to >6.6 mg/liter  $\text{NH}_3$ , with increased degree of symptom at increased ammonia concentrations. Effects observed included erosion of membranes of the eyes

and shedding of the crystalline lens, dilation of blood vessels in liver and brain, accumulation of blood in the occipital region and in intestines. Reaction to light and mechanical stimulation gradually disappeared with increased ammonia concentration, and the pulsebeat slowed. Morphological differences in development between experimental and control larvae were observed from the tenth day of exposure, including a lag in yolk resorption, decrease in growth of the skin fold, and contraction of skin pigment cells causing the skin color to become paler than it was after hatching. At concentrations up to 0.07 mg/liter  $\text{NH}_3$  no significant morphological differences were observed.

A second series of experiments (temperature = 16.5 C) was carried out in the 0.001 to 0.32 mg/liter  $\text{NH}_3$  concentration range, and began with larval salmon (Samylin 1969). Concentrations of 0.21 mg/liter  $\text{NH}_3$  and higher were lethal and caused weight loss in fry; 0.001 to 0.09 mg/liter  $\text{NH}_3$  caused a decrease in weight gain, although no differences in feeding activity, behavior, or development were observed in these concentrations compared to controls. Dissolved oxygen concentrations in this second series of experiments dropped as low as 3.5 mg/liter.

Burrows (1964) tested fingerling chinook salmon for six weeks in outdoor raceways into which ammonium hydroxide was introduced. Two experiments were conducted, one at 6.1 C and the other at 13.9 C, both at pH 7.8. In both cases fish were subsequently maintained in fresh water for an additional three weeks. A recalculation of Burrows reported un-ionized ammonia concentrations, based on more recent aqueous ammonia equilibrium tables, indicates that the concentrations at 6.1 C were 0.003 to 0.006 mg/liter  $\text{NH}_3$ , and at 13.9 C were 0.005 to 0.011 mg/liter  $\text{NH}_3$ . At both temperatures some fish at all ammonia concentrations showed excessive

proliferation and clubbing of the gill filaments; the degree of proliferation was progressive for the first four weeks, after which no measurable increase was discernible. Examination of a sample of the fish tested at 6.1 C after three weeks in fresh water indicated no recovery had taken place from the extensive proliferation. In the experiment with larger fish at 13.9 C a marked recovery from hyperplasia was noted after the three-week fresh water exposure period. In the first experiment the proliferated areas had consolidated; in the second they had not. Burrows postulated that continuous ammonia exposure is a precursor of bacterial gill disease.

Buckley et al. (1979) exposed duplicate groups (90 fish each) of hatchery-reared coho salmon for 91 days to "river-water" solutions of  $\text{NH}_4\text{Cl}$  at concentrations of 0.019 to 0.33 mg/liter  $\text{NH}_3$ ; these were compared with control groups reared at 0.002 mg/liter  $\text{NH}_3$ . Hemoglobin content and hematocrit readings were reduced slightly, but significantly, at the highest concentration tested, and there was also a greater percentage of immature erythrocytes at the highest concentration. Blood ammonia and urea concentrations were not significantly different after 91 days, regardless of concentration of ammonia to which the fish were exposed. Rankin (1979) conducted ammonia tests with embryos of sockeye salmon (Oncorhynchus nerka) from fertilization to hatching. Total embryo mortality occurred at concentrations of 0.49 to 4.9 mg/liter  $\text{NH}_3$ ; times to 50 percent mortality at these concentrations were 40 to 26 days. Mortality of the embryos exposed to 0.12 mg/liter  $\text{NH}_3$  was 30 percent, and time to 50 percent mortality was 66 days.

Two full life-cycle ammonia toxicity tests (354 and 379 days) were conducted with fathead minnows (Thurston et al., submitted). These tests began with newly hatched fry and were continued through their growth,

maturation and spawning stages; progeny were exposed from hatching through growth to 60 days of age. No statistically significant differences were observed based on spawning data (number of egg lots, egg lot size, egg lots per female, eggs per female per day) for concentrations up to 0.4 mg/liter  $\text{NH}_3$ , but large reductions occurred at 0.8-0.9 mg/liter  $\text{NH}_3$ . There was a substantial decrease of the percentage of fry hatching at concentrations of 0.19 mg/liter  $\text{NH}_3$  and higher (Table 2); no effect on hatching success was observed at concentrations of 0.09 mg/liter  $\text{NH}_3$  and lower. Also, there was some indication that length of time for incubation from spawning to hatching increased with increasing  $\text{NH}_3$  concentrations. No statistically significant effects on fish growth were observed for either parental fish or progeny after 30 and 60 days exposure and at exposure termination at concentrations up to 0.4 mg/liter  $\text{NH}_3$ , but parental fish growth was substantially reduced at 0.9 mg/liter  $\text{NH}_3$  after 30 days (at which concentration no progeny existed). Significant mortalities occurred among the parental generation at concentrations of 0.9 to 1.0 mg/liter  $\text{NH}_3$  after 30 and 60 days' exposure.

Head tissues from fathead minnows subjected to prolonged (up to 304 days) ammonia exposure were examined (Smich 1984). Growths, some massive, were observed on heads of several fish exposed to concentrations of 1.25 and 2.17 mg/liter  $\text{NH}_3$ , and swollen darkened areas were observed on heads of several fish held at 0.639 to 1.07 mg/liter. Similar lesions were noted by Thurston et al. (submitted) at lower concentrations, with swollen darkened areas on heads being observed on some fish held at concentrations of 0.22 mg/liter  $\text{NH}_3$  and growths being observed at concentrations as low as 0.43 mg/liter  $\text{NH}_3$ . Grossly and histologically the severity of the lesions, which varied from mild to severe, was positively correlated with ammonia concentration. Lesions appeared to be of a cell type originating from the

primitive meninx covering the brain. The hyperplastic tissue often completely surrounded the brain but was not observed around the spinal cord.

An early life-stage test initiated at the blastula stage of embryogenesis and extending through 39 days post-hatching was conducted with green sunfish by McCormick et al. (1984). Retardation of growth of green sunfish exposed from embryo through juvenile life stages was found at  $\text{NH}_3$  concentrations of 0.489 mg/liter and higher, but not at 0.219 mg/liter and less (Table 2). In a long-term test with green sunfish, Jude (1973) reported that for treatments greater than 0.17 mg/liter  $\text{NH}_3$ , mean fish weight increased less rapidly than controls after introduction of toxicant over the next four days. Thereafter, fish exposed to 0.26 and 0.35 mg/liter  $\text{NH}_3$  grew at an increasing rate while fish exposed to 0.68 and 0.64 mg/liter  $\text{NH}_3$  remained the same for 12 days before greater increases in growth occurred.

An early life-stage test with bluegill from embryo through 30 days post-hatch was conducted on ammonia by Smith et al. (1983). Significant retardation of growth due to ammonia exposure was observed at 0.136 mg/liter  $\text{NH}_3$ ; the no-observed-effect concentration was reported to be 0.063 mg/liter  $\text{NH}_3$  (Table 2).

Broderius et al. (1985) conducted four simultaneous early life-stage ammonia tests with smallmouth bass. These were carried out at four different pH levels, ranging from 6.6 to 8.7, to examine the effect of pH on chronic ammonia toxicity. Exposure to ammonium chloride solutions began with two- to three-day-old embryos and lasted for 32 days. The effect endpoint observed was growth, and ammonia was found to have a greater effect on growth at lower pH levels than at high.  $\text{NH}_3$  concentrations found to retard growth (Table 2) ranged from 0.0558 mg/liter at pH 6.60 to 0.865 mg/liter at pH 8.68.

Early life-stage tests (29-31 days' exposure) on ammonium chloride with channel catfish and white sucker were conducted by Reinbold and Pescitelli (1982a). No significant effect on percent hatch or larval survival was observed for channel catfish at concentrations as high as 0.583 mg/liter  $\text{NH}_3$  and for white sucker as high as 0.239 mg/liter  $\text{NH}_3$ . Significant retardation of growth, however, occurred for channel catfish at concentrations of 0.392 mg/liter  $\text{NH}_3$  and higher and for white sucker at 0.070 mg/liter  $\text{NH}_3$  and higher (Table 2). A delay in time to swim-up stage was also observed for both species at elevated (0.06 to 0.07 mg/liter  $\text{NH}_3$ ) ammonia concentrations.

Robinette (1976) cultured channel catfish fingerlings for periods of approximately one month at concentrations of 0.01 to 0.16 mg/liter  $\text{NH}_3$ . Growth at 0.01 and 0.07 mg/liter  $\text{NH}_3$  was not significantly different from that of control fish; growth retardation at 0.15 and 0.16 mg/liter  $\text{NH}_3$  was statistically significant. Colt (1978) and Colt and Tchobanglous (1978) reported retardation of growth of juvenile channel catfish during a 31-day-period of exposure to concentrations ranging from 0.058 to 1.2 mg/liter  $\text{NH}_3$ . Growth rate was reduced by 50 percent at 0.63 mg/liter  $\text{NH}_3$ , and no growth occurred at 1.2 mg/liter  $\text{NH}_3$ . The authors hypothesized that growth may be inhibited by high concentrations of  $\text{NH}_4^+$  and low concentrations of  $\text{Na}^+$  in solution, and/or the  $\text{NH}_4^+/\text{Na}^+$  ratio. Soderberg et al. (1984) found histopathological gill lesions in pond cultured channel catfish raised in  $\text{NH}_3$  concentrations from 0.02 to 0.067 mg/liter.

Early life-stage tests on ammonium chloride were conducted by Swigert and Spacie (1983) with channel catfish and fathead minnow (Table 2). For both species, growth at ca. 30 days was the most sensitive of reported responses to ammonia, significant reductions being observed at  $\geq 0.24$  mg/liter

NH<sub>3</sub> for channel catfish and at  $\geq 0.33$  mg/liter NH<sub>3</sub> for fathead minnow.

Ammonia exposure for 30 to 40 days of goldfish and tench resulted in lesions and diffuse necrosis of the caudal fin, causing it to degenerate progressively to the point of breaking off by degrees, ultimately leaving only a necrotized stump (Marchetti 1960).

Very little work has been done to investigate effects of different factors on chronic ammonia toxicity. The early life-stage tests at different pH levels conducted by Broderius et al. (1985) with smallmouth bass showed that NH<sub>3</sub> toxicity increased with decreasing pH. Mitchell and Cech (1983) reported that gill damage to channel catfish exposed to about 0.5 mg/l NH<sub>3</sub> occurred only in the presence of residual chlorine, apparently due to monochloramine being the proximate agent. Soderberg et al. (1983) suggested that, under wide diurnal variations of un-ionized ammonia, growth reductions of rainbow trout were better correlated with maximum daily concentrations rather than mean concentrations. Sousa et al. (1974) reduced chronic toxicity of ammonia to chinook salmon by reducing pH and increasing salinity.

#### Derivation of Final Chronic Value for Fresh Water

##### (a) pH and Temperature Dependence of Chronic Ammonia Toxicity

Only one data set exists (Broderius et al. 1985) for which the same investigator determined the chronic toxicity of ammonia to a fish over a suitable pH range. These data (for smallmouth bass) show pH trends qualitatively similar to those discussed earlier for acute toxicity, but suggest a greater relative change in the pH 6.5-7.5 range. Interestingly, total ammonia values were approximately constant at pH 7.8 and below. For Macrobrachium rosenbergii (a saltwater prawn), Armstrong et al. (1978) also found a more pronounced effect of pH on chronic toxicity than acute toxicity.

Chronic effect concentrations expressed as total ammonia were also constant at pH 7.6 and below.

The available data therefore do not adequately support the application of the acute ammonia toxicity pH relationship to chronic ammonia toxicity. Furthermore, the available data are not sufficient to support the derivation of a broadly applicable chronic pH relationship upon which even limited confidence can be placed. Temperature effects on chronic toxicity are totally lacking in the available data.

(b) Acute-Chronic Ratios

Acute-chronic ratios are available for ten species (Table 2). Because these ratios vary so widely (3-43), their dependence on species and physico-chemical factors should be evaluated so that they are properly applied.

The smallmouth bass data in Table 2 indicate that acute-chronic ratios increase with decreasing pH. This is consistent with the comment earlier that the effect of pH on chronic toxicity in the 6.5-7.5 pH range is greater than the effect of pH on acute toxicity. The large ratio for pink salmon also suggests such a pH dependence of the ratio, if it is assumed salmonids have similar ratios. The paucity of data makes firm conclusions impossible, but it is probably inappropriate to apply the pink salmon ratio (=43, measured at pH 6.4) and the largest smallmouth bass ratio (=18, measured at pH 6.6) to the pH range (>7.3) at which other ratios were measured. At pH greater than about 7.7, there is no clear indication that the pH dependence of chronic toxicity differs from that of acute toxicity; consequently, acute-chronic ratios are not expected to vary much, if any, in this pH range.

For temperature, no such clear effect exists. The highest ratio was measured at low temperature (43 for pink salmon at 4 C), but the high value

was probably in large part due to pH. The only other ratio at low temperature is not particularly high (14 for rainbow trout at 9 C). The second and third highest ratios were at higher temperatures (30 for white sucker at 19 C and 20 for fathead minnow at 24 C), but all the low ratios were also in or near this temperature range. In the absence of suitable data, it will be assumed here that ratios are not dependent on temperature.

The purpose of applying a ratio is to derive an estimate of a FCV from a FAV when there is insufficient chronic data available to derive a FCV directly. Since both a FAV and a FCV are estimates of the fifth percentiles of their respective data bases, it is necessary that the ratio be appropriate for applying to the lower part of the range of acute values to derive the lower part of the range of chronic values. When a wide range of ratios are present, this purpose requires a selection of those from an appropriate sensitivity range of acute and chronic values.

Consideration will first be given here to acute-chronic ratios at pH > 7.7, where ratios will be assumed here to be constant with pH. The procedure for selecting the ratios appropriate for determining a FCV from a FAV was as follows. In this pH range, chronic values and acute-chronic ratios are available for nine species in Table 2. Consideration was first restricted to those species with chronic toxicity less than or equal to the median, which included the channel catfish, rainbow trout, white sucker, bluegill, and fathead minnow. Species above the median (green sunfish, smallmouth bass, and two daphnids) had markedly higher chronic values (>0.3 mg/liter NH<sub>3</sub>) which are probably well above the range a FCV will assume, especially considering the diverse nature of the five species selected. The five acute chronic ratios so selected were 10 (channel catfish), 12 (bluegill), 14 (rainbow trout), 20 (fathead minnow), and 30 (white sucker), with a geometric

mean of 16. The higher ratio for the fathead minnow was used because it was for a whole life-cycle test which determined effects of ammonia on reproduction, apparently a more sensitive endpoint than the growth effects examined in other studies. The lower ratio for rainbow trout was used in part because it also was for a whole life-cycle test and in part because the other, higher ratio (=22) was for a pH slightly below the range of concern here.

However, before this average ratio is judged appropriate for deriving a FCV, greater scrutiny should be given to the data used in its derivation. As suggested above, an appropriate ratio is one which, when applied to a low percentile in the distribution of acute values, will produce the same percentile for chronic values. Thus, while it is appropriate to restrict consideration of acute-chronic ratios to species with chronic values less than the median, the relative acute values used should also be examined as to whether they are consistent, on the average, with the chronic values used. For example, the high ratio for the white sucker is apparently due to it being, relative to other species, more chronically sensitive than acutely sensitive. Other species have apparent biases in the other direction. What is important is whether the average relative acute and chronic values of the data used are consistent.

More specifically, the average percentile level of the acute data used for the ratios should approximately equal that of the chronic data used. Corrected to reference pH (8.0) and temperature (20 C), the geometric average of the acute values used for generating the five ratios above is 1.36. This corresponds to between ranks 12 and 13 in the data in Table 3, which, using the cumulative probability formula  $P = \text{Rank}/(N+1)$ , is equivalent to about the 35th percentile. Due to the small amount of data and the uncertain effect of

pH and temperature on chronic toxicity (and thus the relative chronic toxicity of the 11 species in Table 2), the average percentile level of the chronic data used is less easily estimated, but should lie between 30 and 40, most probably in the middle part of this range. Because of the similarity of the percentile levels so estimated and because more exact analysis cannot be supported by the limited database currently available, an acute-chronic ratio of 16 is recommended here as being most appropriate for the estimation of a FCV from a FAV when  $\text{pH} > 7.7$ .

For low pH, few data are present. At pH near 6.5, available ratios are 43 for the pink salmon and 18 for the smallmouth bass, with a geometric mean of 28. Even this ratio may be too low if the smallmouth bass is as relatively insensitive in chronic tests at low pH as it is at high pH. The higher acute-chronic ratio ( $\approx 22$ ) for rainbow trout at  $\text{pH} = 7.4$  may be indicative of somewhat higher ratios at moderate pH. No definite conclusion is made here about appropriate ratios at lower pHs, except that they are probably greater than 20 and will require further testing.

(c) Application of Acute-Chronic Ratios and pH Relationship of Chronic Ammonia Toxicity to Determination of Final Chronic Values

In the absence of sufficient data to directly compute final chronic values (FCVs), both with respect to the number and variety of chronic tests in Table 2 and to the inadequate data on the pH- and temperature-dependence of chronic toxicity, the following approach was adopted for setting FCVs:

- (1) To generate a FCV, an acute-chronic ratio must be applied to an appropriate FAV. The  $\text{FAV}_{\text{ref}}$  used for the 1-hour average criterion (0.52) is not appropriate since it is based on a life stage that is more sensitive than those used in generating the acute-chronic ratios. Furthermore, the fifth-percentile

FAV<sub>ref</sub> computed earlier (0.70) is also not appropriate since it is strongly influenced by the mountain whitefish data, which also was for a sensitive life stage. To compensate for this problem, the mountain whitefish SMAV<sub>ref</sub> was increased by 40%, from 0.56 to 0.78, based on the difference between the acute sensitivities of rainbow trout of the size of the cessed whitefish and of the size used for generating the acute-chronic ratio. The FAV<sub>ref</sub> was then recomputed to be 0.80, which will be used in subsequent calculations of FCVs.

- (2) Due to the lack of information on the effects of temperature on chronic toxicity for any organism and due to the lack of any chronic toxicity data for salmonids at temperatures above 15 C, the temperature relationship implicit in applying an acute-chronic ratio to a FAV will be capped at 15 C rather than 20 C as in Equation 8 for sites with salmonids or other sensitive coldwater species. For sites without salmonids and other sensitive coldwater species, TCAP will be 5 C higher, as for acute toxicity. This will result in the use of the following formula for the factor TCAP when computing a FCV:

$$\begin{aligned} \text{TCAP} &= 15 \text{ C ; salmonids present} & (9) \\ &= 20 \text{ C ; salmonids absent} \end{aligned}$$

These temperature caps are again placed here because the national criterion must be broadly protective and uncertainties require this restriction in order to guarantee protection for certain organisms. The cap may be raised in a site-specific analyses as warranted by the species present. The increase in the temperature cap should not be beyond where there is data that indicates the FCV will be

protective of the most sensitive genera present at the site; in particular, a FCV should not result at any temperature that is significantly greater than the CV at the highest temperature tested for any site genera.

- (3) At pH 7.7 and above, an acute-chronic ratio of 16 will be applied to FAV(pH,T) to calculate FCV(pH,T), this constant ratio being estimated as described above. The equation for FCV at pH > 7.7 therefore is:

$$FCV(pH,T) = \frac{0.80}{16 \cdot FT \cdot FPH} \quad (10)$$

where FPH is as in equation 8 and FT is as in equation 9.

- (4) At pH below 7.7, the FCV will be based on the observation made above that chronic toxicity has been found to be approximately constant on a total ammonia basis in this pH range. The FCV as un-ionized ammonia at any pH and temperature must therefore be set so that the corresponding total ammonia is the same as at pH = 7.7 and that temperature. The applicable equation is:

$$\begin{aligned} FCV(pH,T) &= FCV(7.7,T) \cdot \frac{1 + 10^{pK-7.7}}{1 + 10^{pK-pH}} \\ &= \frac{0.80}{19.2 \cdot FT} \cdot \frac{1 + 10^{pK-7.7}}{1 + 10^{pK-pH}} \end{aligned} \quad (11)$$

where pK is the ammonia speciation stability constant at T. This formula can be simplified by noting that, for the pHs and temperatures of concern,  $10^{pK-pH} \gg 1$ . The equation then approximately becomes:

$$FCV(pH,T) = \frac{0.80}{19.2 \cdot FT \cdot 10^{7.7-pH}} \quad (12)$$

This equation contains an implicit acute-chronic ratio equal to:

$$\text{RATIO}(\text{pH}) = \frac{19.2 \cdot 10^{7.7-\text{pH}}}{\text{FPH}} = \frac{24 \cdot 10^{7.7-\text{pH}}}{1+10^{7.4-\text{pH}}} \quad (13)$$

which varies from 16 at pH 7.7 to 42 at pH 6.5. This implicit ratio can probably be applied to site-specific calculations in this pH range.

It should be noted that the pH and temperature-dependent FCV so derived are within a factor of two of the chronic values for several species (Atlantic salmon, rainbow trout, fathead minnow, white sucker, and bluegill) in Table 2 if the temperature cap is ignored. It is also close to a chronic effect concentration for the clam Musculium transversum in Table 5. These relative differences give some indication that the criteria is approximately correct, since they are similar to the difference between the acute sensitivity of these species and the  $\text{FAV}_{\text{ref}}$  used and are not so large that markedly higher criteria are possible without impacting several species. That the relative margins do not appear to be markedly different for species tested at cold temperature than at warm temperature also provides some reassurance as to the appropriateness of applying the same slope for temperature dependence to chronic toxicity as to acute toxicity. This should not be taken, however, as strong evidence for the temperature relationship used; considerable uncertainty exists in the pH- and temperature-dependence for chronic toxicity, necessitating further research if more reliable criteria are to be developed.

(d) Application of the FCV to a Criterion to Protect Against Chronic Toxicity

As specified in the Guidelines, the criterion to protect against chronic toxicity of the types represented in Table 2 will be based on requiring that 4-day average concentrations not exceed, more often on the average than once

every 3 years, an average FCV based on Equations 10 and 12 above. In the typical situation, where flows, pHs, and temperatures fluctuate, the average FCV should not be obtained simply by applying the equations to the average flow, pH, and temperature, but should rather equal or approximate the arithmetic mean of a time series of FCVs reflective of the fluctuations.

Part of the intent of the short (4-day) averaging period, as opposed to a longer period (e.g., 30-day) more reflective of the duration of tests in Table 2, is to preclude time series of concentrations that would substantially exceed the criterion concentration for a substantial fraction of the longer period. A longer period will be allowed for some situations where limited variability of concentrations can be demonstrated. This matter is discussed in more detail in the Technical Support Document for Water Quality-Based Toxics Control (U.S. EPA, 1985).

#### Saltwater Animals

Little information is available on long-term effects of sublethal ammonia exposures on saltwater species, and no chronic data are available for any saltwater fish species.

Three-week exposure (Wickins 1976) of P. setiferus to  $\text{NH}_4\text{Cl}$  yielded an EC50 (Table 5), based on growth reduction, of 0.72 mg/liter  $\text{NH}_3$ . A six-week test (Table 5) with M. rosenbergii resulted in reduction in growth to 60-70 percent that of controls for prawn exposed to concentrations above 0.12 mg/liter  $\text{NH}_3$ . A "maximum acceptable level" was estimated to be 0.12 mg/liter  $\text{NH}_3$ . Armstrong et al. (1978) conducted growth tests (Table 5) on  $\text{NH}_4\text{Cl}$  using prawn larvae (M. rosenbergii). Retardation in growth was observed at sublethal concentrations (0.11 mg/liter  $\text{NH}_3$  at pH 6.83 and 0.63 mg/liter  $\text{NH}_3$  at pH 7.60), and this effect was greater at low pH.

## TOXICITY TO AQUATIC PLANTS

### Bacteria and Freshwater Plants

Ammonia is known to play an important part in the nitrogen metabolism of aquatic plants. In the aquatic environment, nitrogen plays an important role in determining the composition of phytoplankton and vascular plant communities and in some cases can act as a limiting nutrient in primary production. Ammonia can also be toxic at certain concentrations. Data concerning the toxicity of ammonia to freshwater vascular plants and phytoplankton are contained in Table 4. Few of the papers examined contained sufficient information to enable calculation of un-ionized ammonia concentrations, although total ammonia solutions were more toxic at high than at low pH, indicating that toxicity was likely due primarily to  $\text{NH}_3$  rather than  $\text{NH}_4^+$ . Some information on ammonia effects on bacteria is also included here.

The bacterial species Escherichia coli and Bacillus subtilis were found to be sensitive to  $\text{NH}_4\text{Cl}$  (Deal et al. 1975); 1100 mg/liter  $\text{NH}_3$  killed 90 percent of an E. coli population in 78 minutes. B. subtilis, an aerobic, spore-forming bacterium, was destroyed in less than two hours in 620 mg/liter  $\text{NH}_3$ .  $\text{NH}_3$  inhibition of the bacteria Nitrosomonas (that convert ammonium to nitrite) and the bacteria Nitrobacter (that convert nitrite to nitrate) was studied by Anthonisen et al. (1976) and Neufeld et al. (1980).  $\text{NH}_3$  inhibited the nitrification process at a concentration of 10 mg/liter (Neufeld et al. 1980). The  $\text{NH}_3$  concentrations that inhibited nitrosomonads (10 to 150 mg/liter) were greater than those that inhibited nitrobacters (0.1 to 1.0 mg/liter), and  $\text{NH}_3$ , not  $\text{NH}_4^+$ , was reported to be the inhibiting species (Anthonisen et al. 1976). Acclimation of the nitrifiers to  $\text{NH}_3$ , temperature, and the number of active nitrifying organisms are factors that may affect the inhibitory concentrations of  $\text{NH}_3$  in a nitrification system.

Langowska and Moskal (1974) investigated the inhibitory effects of 24-hour exposures to  $\text{NH}_3$  on pure cultures of ammonifying and denitrifying bacteria. Effects examined were based on ability of the bacteria to produce some specific metabolic processes, such as proteolysis, ammonification, denitrification, and nitrification. Ammonifying and denitrifying bacteria were most resistant to  $\text{NH}_3$ ; proteolytic and nitrifying bacteria were the most sensitive. Concentrations ranging from 0.8 to 170 mg/liter  $\text{NH}_3$  did not adversely affect denitrifying and ammonifying bacteria; 220 mg/liter caused reduction of the examined metabolic processes. Proteolytic bacteria were unaffected at 0.8 mg/liter  $\text{NH}_3$ , but were reduced to zero at 4.2 mg/liter; nitrifying bacteria were unaffected at 2.6 to 5.1 mg/liter and reduced to zero at 13 to 25 mg/liter.

Experimental data concerning the toxicity of ammonia to freshwater phytoplankton are limited. Przytocka-Jusiak (1976) reported ammonia effects (Table 4) on growth of Chlorella vulgaris with 50 percent inhibition in five days at 2.4 mg/liter  $\text{NH}_3$ , and complete growth inhibition in five days at 5.5 mg/liter. The  $\text{NH}_3$  concentration resulting in 50 percent survival of C. vulgaris after five days was found to be 9.8 mg/liter  $\text{NH}_3$ . In a separate study, Przytocka-Jusiak et al. (1977) were able to isolate a C. vulgaris strain with enhanced tolerance to elevated ammonia concentrations, by prolonged incubation of the alga in ammonium carbonate solutions. C. vulgaris was reported to grow well in solutions containing 4.4 mg/liter  $\text{NH}_3$ , but growth was inhibited at 7.4 mg/liter (Matusiak 1976). Tolerance to elevated concentrations of  $\text{NH}_3$  seemed to show a slight increase when other forms of nitrogen were available to the alga than when ammonia was the only form of nitrogen in the medium. The effects of ammonia on growth of the algal species Ochromonas sociabilis was studied by Bretthauer (1978). He

found that concentrations (assuming pH 6.5 and 30 C) of 0.6 mg/liter  $\text{NH}_3$  killed the organisms, and at 0.3 mg/liter development of the population was reduced. Concentrations of 0.06 to 0.15 mg/liter  $\text{NH}_3$  had insignificant effect on growth, and 0.015 to 0.03 mg/liter enhanced growth.

Effects of ammonia on four algal species (Table 4) were studied by Abeliovich and Azov (1976). Ammonia at concentrations over 2.5 mg/liter  $\text{NH}_3$  inhibited photosynthesis and growth of the algal species Scenedesmus obliquus and inhibited photosynthesis of the algae Chlorella pyrenoidosa, Anacystis nidulans, and Plectonema boryanum. Mosier (1978) reported that  $\text{NH}_3$  concentrations causing 50 percent reduction in oxygen production by the green alga Chlorella ellipsoidea and blue-green alga Anabaena subcylindrica were  $16.0 \times 10^{-8}$  and  $251.0 \times 10^{-8}$   $\mu\text{g NH}_3\text{-N/cell}$ , respectively.

The rate of photosynthesis in the blue-green alga P. boryanum was observed to be stimulated by  $\text{NH}_4^+$ , but inhibited by  $\text{NH}_3$  (Solomonson 1969); the magnitude of these effects was dependent on the sodium-potassium composition of the suspending media.  $\text{NH}_3$  inhibition of photosynthesis was associated with a conversion of inorganic polyphosphate stored in the cells to orthophosphate.

Champ et al. (1973) treated a central Texas pond with ammonia to a mean concentration of 25.6 mg/liter  $\text{NH}_3$ . A diverse population of dinoflagellates, diatoms, desmids, and blue-green algae were present before ammonia treatment. Twenty-four hours after treatment the mean number of phytoplankton cells/liter was reduced by 84 percent. By the end of two weeks ( $\text{NH}_3 = 3.6$  mg/liter) the original concentration of cells had been reduced by 95 percent.

Much of the work concerning the response of freshwater vegetation to high ammonia concentrations is only descriptive or is a result of research exploring the possible use of ammonia as an aquatic herbicide.

Champ et al. (1973) reported virtually complete eradication of rooted aquatic vegetation (water shield, Brasenia schreberi, and American lotus, Nelumbo sp.) in a central Texas pond within two weeks after treatment with anhydrous ammonia;  $\text{NH}_3$  concentration was 25.6 mg/liter 24 hours after ammonia addition, and 3.6 mg/liter two weeks later. In experiments with Potamogeton lucens, Litav and Lehrer (1978) observed that ammonia, which forms a readily available nitrogen source for the plant, can be toxic when present at high concentrations, with ammonia causing appreciable injury to detached branches. Ammonia inhibition of growth of Eurasian watermilfoil (Myriophyllum spicatum) affected length and weight similarly and affected roots and shoots similarly (Stanley 1974).

Litav and Agami (1976) studied changes in vegetation in two rivers subject to increased pollution from agricultural fertilizers, urban sewage, and industrial wastes, and attributed the changes in plant species composition primarily to ammonia and detergents. Agami et al. (1976) transplanted seven species of "clean water" macrophytes to various sections of river, and found that ammonia affected only Nymphaea caerulea. Use of high concentrations of ammonia to eradicate aquatic vegetation was described by Ramachandran (1960), Ramachandran et al. (1975), and Ramachandran and Ramaprabhu (1976).

### Saltwater Plants

Data concerning the toxicity of ammonia to saltwater phytoplankton are presented in Table 4. Ten species of estuarine benthic diatoms were cultured for ten days in synthetic media at a range of  $\text{NH}_3$  concentrations from 0.024 to 1.2 mg/liter  $\text{NH}_3$  (Admiraal 1977). A concentration of 0.24 mg/liter  $\text{NH}_3$  retarded the growth of most of the tested species (Table 4). Relative tolerance to ammonium sulfate of five species of chrysomonads was studied by

Pinter and Provasoli (1963). Coccolithus huxleyi was most sensitive, and Pavlova gyraus and Hymenomonas sp. were most tolerant, with intermediate tolerance exhibited by Syracosphaera sp. and Ochrosphaera neapolitana.

Shilo and Shilo (1953, 1955) reported that the euryhaline algae Prymnesium parvum was effectively controlled with applications of ammonium sulfate, which exerted a lytic effect. Laboratory and field tests showed that the concentration of ammonium sulfate necessary for cell lysis decreased with increasing pH, indicating that un-ionized ammonia and not the ammonium ion is responsible for the lytic activity of ammonium sulfate on P. parvum. Effect of ammonia on the dinoflagellate Amphidinium carterae was studied by Byerrum and Benson (1975), who reported that added ammonium ion at concentrations found to stimulate the photosynthetic rate also caused the algae to release up to 60 percent of fixed  $^{14}\text{CO}_2$  to the medium.

Natarajan (1970) found that the concentrations of fertilizer plant effluent toxic to natural phytoplankton (predominantly diatoms) in Cook Inlet, Alaska, were between 0.1 percent (1.1 mg/liter  $\text{NH}_3$ ) and 1.0 percent (11 mg/liter  $\text{NH}_3$ ). At 0.1 percent effluent concentration  $^{14}\text{C}$  uptake was reduced only 10 percent, whereas at 1.0 percent effluent concentration a 24-33 percent reduction in the relative  $^{14}\text{C}$  uptake was observed. Effects of ammonium sulfate on growth and photosynthesis of three diatom and two dinoflagellate species were reported by Thomas et al. (1980), who concluded that increased ammonium concentrations found near southern California sewage outfalls would not be inhibiting to phytoplankton in the vicinity. Provasoli and McLaughlin (1963) reported that ammonium sulfate was toxic to some marine dinoflagellates only at concentrations far exceeding those in seawater.

No data were found concerning the toxicity of ammonia to saltwater vegetation.

## BIOACCUMULATION

No data are available concerning the accumulation of ammonia by aquatic organisms.

## OTHER DATA

A number of investigators have studied effects of ammonia on behavior and various metabolic processes of exposed animals, or have conducted field studies. This research has dealt predominantly with freshwater fishes.

### Freshwater Invertebrates

The effect of ammonia (Table 5) on the ciliary beating rate of clam gills was investigated by Anderson et al. (1978). Concentrations of 0.036 to 0.11 mg/liter  $\text{NH}_3$  caused a reduction in ciliary beating rate of fingernail clams; the effect of these concentrations ranged from 50 percent reduction in beating rate to complete inhibition of cilia. Adult clams (>5 mm) were more sensitive than juveniles (<-5 mm); adults were also slightly more sensitive than the unionid mussel (Elliptio complanata) and the Asiatic clam (C. manilensis). Shaw (1960) investigated effects of ammonium chloride on sodium influx in the freshwater crayfish, Astacus pallipes. Ammonia produced an inhibition of sodium influx; a concentration of 18 mg/liter  $\text{NH}_4^+$  reduced the influx to about 20 percent of its normal value, and influx reduction was related to greater ammonia concentration. This effect was attributed to  $\text{NH}_4^+$  ions and not to any toxic effect exerted on the transporting cells by un-ionized ammonia.  $\text{NH}_4^+$  did not affect chloride influx nor the rate of sodium loss.

Ammonia was added to a Kansas stream at a 24-hour average concentration of 1.4 mg/liter  $\text{NH}_3$ , and a 24-hour drift net sampling was conducted (Liechti and Huggins 1980). No change in diel drift pattern was observed, but there was an increase in magnitude of drift, a shift in kinds of organisms present, and changes in benthic standing crop estimates; the ammonia concentration was concluded to be nonlethal.

## Freshwater Fishes

Herbert and Shurben (1963) investigated the effect on susceptibility to ammonium chloride solutions of rainbow trout forced to swim continuously against water currents of different velocities prior to ammonia exposure. Forcing rainbow trout to swim for one to two days at 85 percent of the maximum velocity they could sustain increased their susceptibility only slightly, corresponding to a 20 to 30 percent reduction in the 24- or 48-hour LC50.

The behavioral response of blacknose dace (Rhinichthys atratulus) to ammonium chloride solutions has been studied (Tsai and Fava 1975; Fava and Tsai 1976); the test fish did not avoid concentrations of 0.56 or 4.9 mg/liter  $\text{NH}_3$ , nor did these concentrations cause significant changes in activity. Avoidance studies were conducted by Westlake and Lubinski (1976) with bluegill using ammonium chloride solutions. Bluegill detected concentrations of approximately 0.01 to 0.1 mg/liter  $\text{NH}_3$ , and evidenced a decrease in general locomotor activity. No apparent avoidance of ammonia was observed, and there was some indication of an attraction. Behavioral responses of bluegill to a five-hour exposure to 0.040 mg/liter  $\text{NH}_3$ , although variable, were related to at least a small amount of physiological stress either at the gill or olfactory surfaces. At a concentration of 0.004 mg/liter  $\text{NH}_3$ , bluegill evidenced slight temporary increases in both activity and turning behavior; no preference or avoidance was demonstrated, with responses seemingly exploratory (Lubinski et al. 1978, 1980). Wells (1915) investigated the avoidance behavior of bluegill to ammonium hydroxide solutions and reported that fishes did not avoid ammonia prior to being killed by it. In a study of the repelling ability of chemicals to green sunfish, Summerfelt and Lewis (1967) concluded that concentrations of ammonia

high enough to repel fish would be rapidly fatal. In avoidance experiments with threespine stickleback, solutions of ammonia concentration 0.27 mg/liter  $\text{NH}_3$  elicited a positive (attraction) response from the test fish (Jones 1948).

Woltering et al. (1978), in tests with largemouth bass and mosquitofish, demonstrated that predator-prey interactions were sensitive to sublethal concentrations of  $\text{NH}_3$ . Ammonia concentrations of 0.63 and 0.86 mg/liter  $\text{NH}_3$  decreased prey consumption and bass growth; bass were reported to be more sensitive than mosquitofish to  $\text{NH}_3$ . The effect of ammonium chloride on consumption of juvenile chinook salmon by brook trout was studied by Hedtke and Norris (1980). At the lowest test concentration of 0.29 mg/liter  $\text{NH}_3$ , trout consumption rates decreased as much as 65 percent. As ammonia concentration increased, however, consumption of prey increased and was double that of controls at the highest tested concentration of 0.76 mg/liter  $\text{NH}_3$ . Increased consumption rate was related to both increased  $\text{NH}_3$  concentration and increased prey density. The magnitude of the effect of ammonia was not the same at all prey densities, having a greater effect on consumption rate at high than at low prey densities. Mortalities were observed among prey salmon at the highest  $\text{NH}_3$  levels, and these were attributed to the combined effect of  $\text{NH}_3$  and stress from presence of the predator. Brook trout exhibited toxic effects due to  $\text{NH}_3$ .

$\text{NH}_4\text{Cl}$  and  $\text{NH}_4\text{HCO}_3$  solutions were injected intraarterially into rainbow trout (Hillaby and Randall 1979). The same dose of each compound was required to kill fish, but there was a more rapid excretion of  $\text{NH}_3$  after  $\text{NH}_4\text{HCO}_3$  infusions, resulting in higher  $\text{NH}_3$  concentrations in blood, than after  $\text{NH}_4\text{Cl}$  infusions. Ammonium acetate solutions of different concentrations were injected intraperitoneally into three species of fishes

(Wilson 1968; Wilson et al. 1969). LD50s (mmoles/kg body weight) for channel catfish for one to four hours was 26.7 to 18.7, for goldfish for one hour were 29.3 and 29.6 in two separate tests, and for rainbow trout for one hour was 17.7. Goldfish was the most resistant species tested and rainbow trout the least resistant. Nehring (1964) compared toxicity of ammonia in the water to toxicity of ammonia administered orally and concluded that the threshold and lethal concentrations were considerably lower for ammonia in water than for ammonia administered orally.

Acute symptoms of  $\text{NH}_3$  toxicity to brown trout sac fry and 12-day-old fry were described by Penaz (1965), who exposed fry to concentrations ranging from 0.08 to 50.0 mg/liter  $\text{NH}_3$ . Symptoms caused by  $\text{NH}_3$  exposures were: rapid spasm-like movements at concentrations of 2.0 mg/liter  $\text{NH}_3$  and higher within 16-17 minutes of exposure; after 40 minutes these symptoms were also observed at 0.4 mg/liter  $\text{NH}_3$ . After 2.5 hours these abnormal movements ceased, and at 10 hours heart activity was decreased and fish lost movement ability at the higher ( $>2.0$  mg/liter  $\text{NH}_3$ ) concentrations. Other symptoms included inability to react to mechanical stimulation and disorders in rhythm of mouth movements culminating in the mouth's staying rigidly open. Thumann (1950), working with rainbow trout and brook (=brown?) trout, described observed symptoms of ammonia poisoning to fishes to be convulsions and frequent equilibrium and positional anomalies.

Smart (1978) reported that exposure of rainbow trout to an acutely lethal concentration of 0.73 mg/liter  $\text{NH}_3$  resulted in an increase in oxygen consumption, increase in ventilation volume, decrease in percent oxygen utilization, increase in respiratory frequency and amplitude (buccal pressure), decrease in dorsal aortic blood  $\text{P}_{\text{O}_2}$ , increase in dorsal aortic blood pressure, and increase in mean heart rate. Physiological parameters

not significantly affected by  $\text{NH}_3$  exposure were "cough" rate, dorsal aortic blood pH, blood P50, erythrocyte count, hematocrit, and hemoglobin concentration. Coho salmon exposed to concentrations ranging from 0.094 to 0.162 mg/liter  $\text{NH}_3$  (Sousa and Meade 1977) exhibited hyperexcitability, hyperventilation, ataxia, and progressive acidemia; methemoglobin concentrations in blood of exposed fish did not differ significantly from those of controls. Effects on trout (species not specified) blood with exposure to accumulated excreted  $\text{NH}_3$  were investigated by Phillips et al. (1949) and were reported to include an increase in blood carbon dioxide content and a decrease in oxygen content.

Arillo et al. (1979d) measured gill sialic acid content in rainbow trout exposed to  $\text{NH}_4\text{OH}$  or  $\text{NH}_4\text{Cl}$  solutions ranging from 0.05 to 0.5 mg/liter  $\text{NH}_3$ , and reported that increasing  $\text{NH}_3$  concentrations produced increasing gill sialic acid content. Elevated gill sialic acid levels were also produced by higher ammonium ion ( $\text{NH}_4^+$ ) concentrations at identical  $\text{NH}_3$  concentrations, and the authors concluded that  $\text{NH}_4^+$  was a stressor causing elevated sialic acid levels. Exposure of rainbow trout (14-cm length) for four hours to  $\text{NH}_4\text{Cl}$  and  $\text{NH}_4\text{OH}$  solutions of concentrations ranging from 0.094 to 0.50 mg/liter  $\text{NH}_3$  resulted in increased proteolytic activity and free amino acid levels in the fish livers, but no statistically significant change in fructose 1,6-biphosphatase enzyme activity (Arillo et al. 1978, 1979a). Renal renin activity was reported (Arillo et al. 1981b) to increase in rainbow trout exposed to concentrations of 0.043 to 0.61 mg/liter  $\text{NH}_3$ . A significant decrease in liver glycogen and increase in free glucose were observed in rainbow trout exposed to  $\text{NH}_4\text{Cl}$  solutions for four hours at a concentration of 0.048 mg/liter  $\text{NH}_3$ , and a decrease in total carbohydrates was observed at 0.12 mg/liter  $\text{NH}_3$  (Arillo et al. 1979b). For

trout similarly treated with ammonium hydroxide, significant decreases in glycogen and carbohydrates, and increase in glucose occurred at 0.097 mg/liter  $\text{NH}_3$ .

A statistically significant increase in rainbow trout liver concentrations of cyclic-3',5'-adenosine-monophosphate (cAMP) was reported by Arillo et al. (1979c) to be induced by a four-hour exposure to elevated ammonia concentrations of 0.011 to 0.124 mg/liter  $\text{NH}_3$ . Decreases in liver glycogen levels were also measured and were significantly different from controls only in the trout exposed to 0.048 mg/liter  $\text{NH}_3$ , the highest exposure used for glycogen measurements. The authors concluded that cAMP measurements provided a very sensitive means of discerning fish stress even at very low toxicant concentrations, although quantitative measurement of stress intensity was not possible. Lysosomal lability was also investigated as an indicator of stress in rainbow trout due to ammonia exposure (Arillo et al. 1980), and was reported to increase significantly for fish subjected to concentrations of 0.048 to 0.61 mg/liter  $\text{NH}_3$ . Exposure of rainbow trout for four to 48 hours to 0.024 to 0.61 mg/liter  $\text{NH}_3$  resulted in changes in various brain and liver metabolites; the magnitude of the changes was dependent on both exposure time and  $\text{NH}_3$  concentration (Arillo et al. 1981a).

Exposure of walking catfish (Clarias batrachus) to ammonia caused inhibition of fish brain cholinesterase and kidney peroxidase activity (Mukherjee and Bhattacharya 1974, 1975a). Plasma corticosteroid concentrations were measured (Tomasso et al. 1981) in channel catfish exposed to 1.1 mg/liter  $\text{NH}_3$  for 24 hours; corticosteroid levels increased initially, peaked after eight hours, then decreased. The overall increase was approximately tenfold over normal levels.

Korting (1969b) reported that carp exposed to 1 mg/liter  $\text{NH}_3$  exhibited an increase in number of blood erythrocytes, reaching an initial maximum

after several hours followed by a gradual decrease; after 50 hours the number was less than the average for non-exposed fish. Other blood changes from the ammonia exposure were: thickening of individual erythrocytes, reduction of osmotic resistance of erythrocytes, increase in concentrations of urea and lactic acid, and decrease in ATP concentration. Levi et al. (1974) reported that goldfish exposed for 24 hours to  $\text{NH}_4\text{Cl}$  solutions exhibited increases in cerebral and blood concentrations of glutamine and in other amino acids, with changes most pronounced in the brain. Concentrations of free amino acids in livers showed only slight increases of a few amino acids, including glutamine, and the concentration of lysine decreased. No change in concentrations of free amino acids was observed in kidneys. Rainbow trout exposed to 0.33 mg/liter  $\text{NH}_3$  had significantly higher packed cell volumes; exposures to concentrations of 0.24 mg/liter  $\text{NH}_3$  and higher resulted in significantly raised blood glucose and plasma cortisol concentrations (Swift 1981).

Diuretic response of rainbow trout exposed to concentrations of 0.09 to 0.45 mg/liter  $\text{NH}_3$  was studied by Lloyd and Orr (1969). After an initial lag period, urine production increased rapidly during exposure then returned to normal within a few hours after discontinuation of  $\text{NH}_3$  exposure. A no-observed-effect concentration was reported to be 0.046 mg/liter  $\text{NH}_3$ . Goldfish were exposed to solutions containing 1.0 to 1.9 mg/liter  $\text{NH}_3$  (Fromm 1970; Olson and Fromm 1971); onset of death was characterized by a gradual cessation of swimming movements and settling to the bottom of the tank. Some goldfish near death were returned to ammonia-free water in which they recovered to at least some degree. In similar experiments (Fromm 1970; Olson and Fromm 1971) rainbow trout were exposed to ambient total ammonia concentrations of 0.04 to 0.2 mg/liter  $\text{NH}_3$ . There was a decrease in total

nitrogen excreted with increase in ambient  $\text{NH}_3$ , and a concomitant decrease in the  $\text{NH}_3$  portion of total nitrogen excreted; urea and protein nitrogen excretion rates showed no changes as ambient  $\text{NH}_3$  increased. Onset of death for trout was characterized by violent thrashing movements.

Exposure of rainbow trout to solutions of  $\text{NH}_4\text{Cl}$  for 24 hours (Fromm and Gillette 1968; Fromm 1970) showed that an increase in ambient water  $\text{NH}_3$  concentration resulted in a corresponding increase in blood  $\text{NH}_3$  concentrations, and a decrease in total nitrogen and  $\text{NH}_3$  excretion. The decrease in  $\text{NH}_3$  excretion accounted for half or less of the total nitrogen excretion, depending on the water  $\text{NH}_3$  concentration, indicating that the reduction in  $\text{NH}_3$  excretion was to some extent compensated for by increased excretion of some other nitrogenous compound(s).

Young fry (2-20 days old) of loach (Misgurnus anguillicaudatus) and carp were exposed for five to 70 hours to  $^{15}\text{N}$ -labeled ammonium chloride solutions at six concentrations from 0.002 to 0.064 mg/liter  $\text{NH}_3$  (Ito 1976), and the proportion of  $^{15}\text{N}$  relative to total N in the fishes determined. Ammonia was shown to be directly absorbed by the fry; nitrogen conversion rate increased with increasing ammonia concentration and exposure time. Nitrogen conversion rates for carp fry decreased as fry age increased from 3 to 20 days. After 48 hours of exposure to 0.064 mg/liter  $\text{NH}_3$  followed by transfer to ammonia-free water, rapid excretion (15-20 percent) of the absorbed  $^{15}\text{N}$  occurred during the first hour in ammonia-free water. Excretion rate then slowed, with about 50 percent of the absorbed  $^{15}\text{N}$  being retained after 48 hours in ammonia-free water. Comparison of  $^{15}\text{N}$  absorption rates between live and sacrificed three-day-old carp fry showed one-third to one-half the uptake of  $^{15}\text{N}$  by dead fry compared with live,

indicating that the uptake of ammonia from water by live fish occurs not only by simple membrane permeation but also by metabolic action.

Flagg and Hinck (1978) reported that exposure to  $\text{NH}_3$  lowered the resistance of channel catfish to the pathogen Aeromonas hydrophila. In 17- and 28-day tests, increasing exposure concentrations from 0.02 to 0.04 mg/liter  $\text{NH}_3$  resulted in increasing numbers of bacteria in host livers. Schreckenbach et al. (1975) reported that ammonia in pond water leads to outbreaks of gill necrosis in carp, accompanied by an increase in ammonia concentration in serum of the fish. This is aggravated at elevated pH levels due to increasing inhibition of ammonia excretion at increasing pH levels, with ammonia excretion being almost totally blocked at pH values above 10.5. After investigating the possible role of parasites, bacteria, viruses, and other ultramicroscopic agents in causing gill necrosis, the authors concluded that pH-dependent intoxication or auto-intoxication with ammonia was the sole cause of the gill damage. Studies of the treatment and prophylaxis of gill necrosis using 28 different therapeutical preparations led to the conclusion that only those preparations that lowered the water-pH level and/or ammonia concentrations resulted in an improvement in clinical symptoms.

Increase in frequency of opercular rhythm in fishes was monitored as a means to measure fish response to sublethal concentrations of ammonia (Morgan 1976, 1977). Ammonia threshold detection concentration (Table 5) for largemouth bass was approximately 30 percent of the LC50 for that species. Increases in largemouth bass opercular rhythms and activity were electronically monitored (Morgan 1978, 1979) to determine threshold effect ammonia concentrations (Table 5); for a 24-hour exposure the effect concentration for opercular rhythms was 0.028 mg/liter  $\text{NH}_3$  and for activity was 0.0055 mg/liter. Lubinski et al. (1974) observed that ammonia stress apparently caused bluegill to consume more oxygen.

In field experiments in an Arizona mountain lake, mortalities of caged rainbow trout were attributed to high un-ionized ammonia concentrations and high pH levels; 20 to 100 percent of test fish died in 24 hours at  $\text{NH}_3$  concentrations of 0.109 to 0.225 mg/liter (Fisher and Ziebell 1980). Ammonia added to a Kansas stream at a 24-hour average concentration of 1.4 mg/liter  $\text{NH}_3$  resulted in fry of slender madtom (Noturus exilis), Notropis sp., and orangethroat darter being collected in large numbers in a 24-hour drift net sampling; these fishes are not normally found in drift net samples, and their presence was attributed to toxic effects of the ammonia (Liechti and Huggins 1980).

#### Saltwater Invertebrates

Sublethal toxicity of  $\text{NH}_4\text{Cl}$  to the quahog clam and eastern oyster was studied by Epifanio and Srna (1975) who measured the effect of ammonia over 20 hours on the rate of removal of algae (Isochrysis galbana) from suspension (clearing rate) by the clams and oysters. Concentrations of 0.06 to 0.2 mg/liter  $\text{NH}_3$  affected clearing; no difference was observed between juveniles and adults. The effect of ammonia on the ciliary beating rate of the mussel Mytilus edulis was studied by Anderson et al. (1978). Concentrations of 0.097 to 0.12 mg/liter  $\text{NH}_3$  resulted in a reduction in ciliary beating rate from 50 percent to complete inhibition (Table 5).

Exposure of unfertilized sea urchin (Lytechinus pictus) eggs to  $\text{NH}_4\text{Cl}$  resulted in stimulation of the initial rate of protein synthesis, an event that normally follows fertilization (Winkler and Grainger 1978).  $\text{NH}_4\text{Cl}$  exposure of unfertilized eggs of Strongylocentrotus purpuratus, L. pictus, and Strongylocentrotus drobachiensis was reported (Paul et al. 1976; Johnson et al. 1976) to cause release of "fertilization acid", more rapidly and in greater amounts than after insemination. Activation of unfertilized L. pictus eggs by  $\text{NH}_4\text{Cl}$  exposure was also evidenced by an increase in

intracellular pH (Shen and Steinhardt 1978; Steinhardt and Mazia 1973). Ammonia treatment was also reported to activate phosphorylation of thymidine and synthesis of histones in unfertilized eggs of the sea urchin S. purpuratus (Nishioka 1976). Premature chromosome condensation was induced by ammonia treatment of eggs of L. pictus and S. purpuratus (Epel et al. 1974; Wilt and Mazia 1974; Krystal and Poccia 1979). Ammonia treatment of S. purpuratus and S. drobachiensis fertilized eggs resulted in absence of the normal uptake of calcium following insemination, but did not inhibit calcium uptake if ammonia treatment preceded insemination (Paul and Johnston 1978).

The polychetous annelid (Nereis succinea), the channeled whelk (Busycon canaliculatum), and the brackish water clam (Rangia cuneata) were subjected to ammonia concentrations of 0.85, 0.37, and 2.7 mg/liter NH<sub>3</sub> and ammonia excretion measured (Mangum et al. 1978). The excretion of ammonia in these species was inhibited by non-lethal concentrations of ammonia; the authors concluded that ammonia crosses the excretory epithelium in the ionized form, and that the process is linked to the activity of the Na<sup>+</sup> + K<sup>+</sup> ATPases. When blue crab (Callinectes sapidus) were moved from water of 28 ppt salinity to water of 5 ppt, a doubling of ammonia excretion rate occurred; addition of excess NH<sub>4</sub>Cl to the low salinity water inhibited ammonia excretion and decreased net acid output (Mangum et al. 1976). The effect of gaseous NH<sub>3</sub> on hemoglobin from blood of the common marine bloodworm (Glycera dibrachiata) was examined (Sousa et al. 1977) in an attempt to determine whether there was competition between NH<sub>3</sub> and oxygen in binding to hemoglobin; such an NH<sub>3</sub>/O<sub>2</sub> relationship was not found.

#### Saltwater Fishes

No other data were found for saltwater fish species.

## UNUSED DATA

Many references cited in the References section were not used in the text or tables, for a variety of reasons. For those several cases where more than one reason applies to a given paper, it is listed only under the principal reason for its not being used.

The following references were not used because the research they reported was conducted using aquatic organisms not resident in North America: Alderson (1979), Arizzi and Nicotra (1980), Brown and Currie (1973), Brownell (1980), Chin (1976), Currie et al. (1974) Dockal and Varecha (1967), D'Silva and Verlencar (1976), Giussani et al. (1976), Greenwood and Brown (1974), Grygierek et al. (1978), Inamura (1951), Matias (1983), Nicotra and Arizzi (1980), Orzechowski (1974), Reddy and Menon (1979), Sadler (1981), Saha et al. (1956), Shaffi (1980b), Singh et al. (1967), Stroganov and Pozhitkov (1941), Thomas et al. (1976), Turoboyski (1960), Vailati (1979), Woker (1949), Wuhrmann (1952), Wuhrmann and Woker (1953), Wuhrmann and Woker (1955), Wuhrmann and Woker (1958), Yamagata and Niwa (1982).

The following references were not used because insufficient water chemical composition data were provided to permit calculation of  $\text{NH}_3$ : Belding (1927), Binstock and Lecar (1969), Bullock (1972), Chu (1943), Danielewski (1979), Das (1980), Ellis (1937), Hepher (1959), Joy and Sathyanesan (1977), Kawamoto (1961), Mukherjee and Bhattacharya (1978), Oshima (1931), Oya et al. (1939), Patrick et al. (1968), Rao and Ragothaman (1978), Roberts (1975), Rushton (1921), Scidmore (1957), Shelford (1917), Shevtsova et al. (1979), Sigel et al. (1972), Southgate (1950), Wolf (1957a), Wolf (1957b), Zgurovskaya and Kustenko (1968).

The following references were not used because the authors reported data published elsewhere which was cited in this document from the other

publication(s): Burkhalter (1975), Colt (1974), Dept. of Environment, U.K. (1972), Herbert (1955), Hillaby (1978), Larmoyeux and Piper (1973), Ministry of Technology, U.K. (1960), Ministry of Technology, U.K. (1966), Rice (1971), Smart (1975), Wilson (1974).

The following references were not used because they were foreign-language papers for which no translation was available, and no useful information could be obtained from the abstract: Desavelle and Hubault (1951), Fedorov and Smirnova (1978), Frahm (1975), Garcia-Romeu and Motais (1966), Guerra and Comodo (1972), Guseva (1937), Hubault (1955), Jocque and Persoone (1970), Kawamoto (1958), Korting (1976), Krauss (1937), Kuhn and Koecke (1956), Leclerc and Devlaminck (1950), Mamontova (1962), Oya et al. (1939), Pequignot and Moga (1975), Pora and Precup (1971), Revina (1964), Rosslenbroich and Dohler (1982), Saeki (1965), Schaperclaus (1952), Scheuring and Leopoldseder (1934), Schreckenbach and Spangenberg (1978); Steinmann and Surbeck (1922a), Steinmann and Surbeck (1922b), Svobodova (1970), Svobodova and Groch (1971), Teulon and Simeon (1966), Truelle (1956), Vamos and Tasnadi (1962a), Vamos and Tasnadi (1962b), Vamos et al. (1974), Yasunaga (1976), Yoshihara and Abe (1955).

The following references were not used because they relate more to ammonia metabolism in fishes, than to ammonia toxicity: Bartberger and Pierce (1976), Becker and Schmale (1978), Brett and Zala (1975), Cameron and Heisler (1983), Cowey and Sargent (1979), Creach et al. (1969), Cvancara (1969a), Cvancara (1969b), De and Bhattacharya (1976), De Vooy (1968), De Vooy (1969), Driedzic and Hochachka (1978), Fauconneau and Luquet (1979), Fechter (1973), Fellows and Hird (1979a), Fellows and Hird (1979b), Flis (1968a), Flis (1968b), Florkin and Duchateau (1943), Forster and Goldstein (1966), Forster and Goldstein (1969), Fromm (1963), Girard and Payan (1980),

Goldstein and Forster (1961), Goldstein and Forster (1965), Goldstein et al. (1964), Gordon (1970), Gregory (1977), Grollman (1929), Guerin-Ancey (1976a), Guerin-Ancey (1976b), Guerin-Ancey (1976c), Guerin-Ancey (1976d), Hays et al. (1977), Hoar (1958), Huggins et al. (1969), Janicki and Lingis (1970), Katz (1979), Kaushik and Luquet (1977), Kloppick et al. (1967), Kutty (1978), Lawrence et al. (1957), Lum and Hammen (1964), Maetz (1973), Maetz and Garcia-Romeu (1964), Makarewicz and Zydowo (1962), Mason (1979a), Mason (1979b), Matter (1966), McBean et al. (1966), McKhann and Tower (1961), Moore et al. (1963), Morii et al. (1978), Morii (1979), Morii et al. (1979), Mukherjee and Bhattacharya (1977), Nelson et al. (1977), Payan (1978), Payan and Maetz (1973), Payan and Matty (1975), Payan and Pic (1977), Pequin and Serfaty (1963), Pequin and Serfaty (1966), Pequin and Serfaty (1968), Pequin et al. (1969a), Pequin et al. (1969b), Raguse-Degener et al. (1980), Ray and Medda (1976), Read (1971), Rice and Stokes (1974), Rychley and Marina (1977), Savitz (1969), Savitz (1971), Savitz (1973), Savitz et al. (1977), Schooler et al. (1966), Smith (1929), Smith (1946), Smith and Thorpe (1976), Smith and Thorpe (1977), Storozhuk (1970), Sukumaran and Kutty (1977), Tandon and Chandra (1977), Thornburn and Matty (1963), Vellas and Serfaty (1974), Walton and Cowey (1977), Watts and Watts (1974), Webb and Brown (1976), Wood (1958), Wood and Caldwell (1978).

The following references were not used because the material the authors used was a complex compound or had an anion that might in itself be toxic: Blahm (1978), Curtis et al. (1979), Johnson and Sanders (1977), Kumar and Krishnamoorthi (1983), Simonin and Pierron (1937), Vallejo-Freire et al. (1954).

The following references were not used because they dealt with complex effluents or waste waters, of which ammonia was a primary component: Brown et al. (1970), Calamari and Marchetti (1975), Gupta et al. (1979), Iwan and Cella (1979), Janicke and Ludemann (1967), Lee et al. (1982), Lloyd and Jordan (1963), Lloyd and Jordan (1964), Martens and Servizi (1976), Matthews and Myers (1976), Mihnea (1978), Nedwell (1973), Okaichi and Nishio (1976), Parna (1971), Rosenberg et al. (1967), Ruffier et al. (1981), Sahai and Singh (1977), Shaffi (1980a), Vamos (1962), Vamos and Tasnadi (1972), Ward et al. (1982).

Three references consisted only of an abstract, providing insufficient information to warrant their use: Liebmann and Reichenbach-Klinke (1969), Mukherjee and Bhattacharva (1975b), Redner et al. (1980).

## SUMMARY

All concentrations used herein are expressed as un-ionized ammonia ( $\text{NH}_3$ ), because  $\text{NH}_3$ , not the ammonium ion ( $\text{NH}_4^+$ ) has been demonstrated to be the principal toxic form of ammonia. The data used in deriving the criteria are predominantly from flow-through tests in which ammonia concentrations were measured. Ammonia was reported to be acutely toxic to freshwater organisms at concentrations (uncorrected for pH) ranging from 0.53 to 22.8 mg/liter  $\text{NH}_3$  for 19 invertebrate species representing 14 families and 16 genera and from 0.083 to 4.60 mg/liter  $\text{NH}_3$  for 29 fish species from 9 families and 18 genera. Among fish species, reported 96-hour LC50s ranged from 0.083 to 1.09 mg/liter for salmonids and from 0.14 to 4.60 mg/liter  $\text{NH}_3$  for non-salmonids. Reported data from chronic tests on ammonia with two freshwater invertebrate species, both daphnids, showed effects at concentrations (uncorrected for pH) ranging from 0.304 to 1.2 mg/liter  $\text{NH}_3$ , and with nine freshwater fish species, from five families and seven genera, ranging from 0.0017 to 0.612 mg/liter  $\text{NH}_3$ .

Concentrations of ammonia acutely toxic to fishes may cause loss of equilibrium, hyperexcitability, increased breathing, cardiac output and oxygen uptake, and, in extreme cases, convulsions, coma and death. At lower concentrations ammonia has many effects on fishes including a reduction in hatching success, reduction in growth rate and morphological development, and pathologic changes in tissues of gills, livers, and kidneys.

Several factors have been shown to modify acute  $\text{NH}_3$  toxicity in fresh water. Some factors alter the concentration of un-ionized ammonia in the water by affecting the aqueous ammonia equilibrium, and some factors affect the toxicity of un-ionized ammonia itself, either ameliorating or exacerbating the effects of ammonia. Factors that have been shown to affect

ammonia toxicity include dissolved oxygen concentration, temperature, pH, previous acclimation to ammonia, fluctuating or intermittent exposures, carbon dioxide concentration, salinity, and the presence of other toxicants.

The most well-studied of these is pH; the acute toxicity of  $\text{NH}_3$  has been shown to increase as pH decreases. Sufficient data exist from toxicity tests conducted at different pH values to formulate a mathematical expression to describe pH-dependent acute  $\text{NH}_3$  toxicity. The very limited amount of data regarding effects of pH on chronic  $\text{NH}_3$  toxicity also indicate increasing  $\text{NH}_3$  toxicity with decreasing pH, but the data are insufficient to derive a broadly applicable toxicity/pH relationship. Data on temperature effects on acute  $\text{NH}_3$  toxicity are limited, and somewhat variable, but indications are that  $\text{NH}_3$  toxicity to fish is greater as temperature decreases. There is no information available regarding temperature effects on chronic  $\text{NH}_3$  toxicity.

Examination of pH- and temperature-corrected acute  $\text{NH}_3$  toxicity values among species and genera of freshwater organisms showed that invertebrates are generally more tolerant than fishes, a notable exception being the fingernail clam. There is no clear trend among groups of fish, the several most sensitive tested species and genera including representatives from diverse families (Salmonidae, Cyprinidae, Percidae, and Centrarchidae). Available chronic toxicity data for freshwater organisms also indicates invertebrates (cladocerans, one insect species) to be more tolerant than fishes, again with the exception of the fingernail clam. When corrected for the presumed effects of temperature and pH, there is also no clear trend among groups of fish for chronic toxicity values, the most sensitive species including representatives from five families (Salmonidae, Cyprinidae, Ictaluridae, Centrarchidae, and Catostomidae) and having chronic values

ranging by not much more than a factor or two. The range of acute-chronic ratios for ten species from six families was 3 to 43, and acute-chronic ratios were higher for the species having chronic tolerance below the median. Available data indicate that differences in sensitivities between warm and cold water families of aquatic organisms are inadequate to warrant discrimination in the national ammonia criterion between bodies of water with "warm" and "cold" water fishes; rather, effects of organism sensitivities on the criterion are most appropriately handled by site-specific criteria derivation procedures.

Data for concentrations of  $\text{NH}_3$  toxic to freshwater phytoplankton and vascular plants, although limited, indicate that freshwater plant species are appreciably more tolerant to  $\text{NH}_3$  than are invertebrates or fishes. The ammonia criterion appropriate for the protection of aquatic animals will therefore in all likelihood be sufficiently protective of plant life.

Available acute and chronic data for ammonia with saltwater organisms are very limited, and insufficient to derive a saltwater criterion. A few saltwater invertebrate species have been tested, and the prawn Macrobrachium rosenbergii was the most sensitive. The few saltwater fishes tested suggest greater sensitivity than freshwater fishes. Acute toxicity of  $\text{NH}_3$  appears to be greater at low pH values, similar to findings in freshwater. Data for saltwater plant species are limited to diatoms, which appear to be more sensitive than the saltwater invertebrates for which data are available.

More quantitative information needs to be published on the toxicity of ammonia to aquatic life. There are some key research needs that need to be addressed in order to provide a more complete assessment of ammonia toxicity. These are: (1) acute tests with additional saltwater fish species and saltwater invertebrate species; (2) life-cycle and early life-stage tests

with representative freshwater and saltwater organisms from different families, with particular attention to trends of acute-chronic ratios; (3) fluctuating and intermittent exposure tests with a variety of species and exposure patterns; (4) more complete tests of the individual and combined effects of pH and temperature, especially for chronic toxicity; (5) more histopathological and histochemical research with fishes, which would provide a rapid means of identifying and quantifying sublethal ammonia effects; (6) studies on effects of dissolved and suspended solids on acute and chronic toxicity.

## NATIONAL CRITERIA

The procedures described in the "Guidelines for Deriving Numerical National Water Quality Criteria for the Protection of Aquatic Organisms and Their Uses" indicate that, except possibly where a locally important species is very sensitive, freshwater aquatic organisms and their uses should not be affected unacceptably if:

- (1) the one-hour\* average concentration of un-ionized ammonia (in mg/liter  $\text{NH}_3$ ) does not exceed, more often than once every three years on the average, the numerical value given by  $0.52/\text{FT}/\text{FPH}/2$ ,

where:

$$\text{FT} = 10^{0.03(20-\text{TCAP})}; \text{TCAP} \leq T \leq 30$$

$$10^{0.03(20-T)} \quad ; \quad 0 \leq T \leq \text{TCAP}$$

$$\text{FPH} = 1 \quad ; \quad 8 \leq \text{pH} \leq 9$$

$$\frac{1 + 10^{7.4-\text{pH}}}{1.25} \quad ; \quad 6.5 \leq \text{pH} \leq 8$$

TCAP = 20 C; Salmonids or other sensitive coldwater species present

= 25 C; Salmonids and other sensitive coldwater species absent

(\*An averaging period of one hour may not be appropriate if excursions of concentrations to greater than 1.5 times the average occur during the hour; in such cases, a shorter averaging period may be needed.)

- (2) the 4-day average concentration of un-ionized ammonia (in mg/liter  $\text{NH}_3$ ) does not exceed, more often than once every three years on the average, the average\* numerical value given by  $0.80/\text{FT}/\text{FPH}/\text{RATIO}$ , where FT and FPH are as above and:

$$\begin{aligned} \text{RATIO} &= \cancel{16}^{13.5} && ; 7.7 \leq \text{pH} \leq 9 \\ &= \cancel{24}^{20} \cdot \frac{10^{7.7-\text{pH}}}{1 + 10^{7.4-\text{pH}}} && ; 6.5 \leq \text{pH} \leq 7.7 \end{aligned}$$

TCAP = 15 C; Salmonids or other sensitive coldwater species present

= 20 C; Salmonids and other sensitive coldwater species absent

(\*Because these formulas are nonlinear in pH and temperature, the criterion should be the average of separate evaluations of the formulas reflective of the fluctuations of flow, pH, and temperature within the averaging period; it is not appropriate in general to simply apply the formula to average pH, temperature and flow.)

The extremes for temperature (0, 30) and pH (6.5, 9) given in the above formulas are absolute. It is not permissible with current data to conduct any extrapolations beyond these limits. In particular, there is reason to believe that appropriate criteria at pH > 9 will be lower than the plateau given above between pH 8 and 9.

Criteria concentrations for the pH range 6.5 to 9.0 and the temperature range 0 C to 30 C are provided in the following tables. Total ammonia concentrations equivalent to each un-ionized ammonia concentration are also provided in these tables. There is limited data on the effect of temperature on chronic toxicity. EPA will be conducting additional research on the effects of temperature on ammonia toxicity in order to fill perceived data gaps. Because of this uncertainty, additional site-specific information should be developed before these criteria are used in wasteload allocation modelling. For example, the chronic criteria tabulated for sites lacking

(1) One-hour average concentrations for ammonia.\*

pH	0 C	5 C	10 C	15 C	20 C	25 C	30 C
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**A. Salmonids or Other Sensitive Coldwater Species Present**

Un-ionized Ammonia (mg/liter NH<sub>3</sub>)

6.50	0.0091	0.0129	0.0182	0.026	0.036	0.036	0.036
6.75	0.0149	0.021	0.030	0.042	0.059	0.059	0.059
7.00	0.023	0.033	0.046	0.066	0.093	0.093	0.093
7.25	0.034	0.048	0.068	0.095	0.135	0.135	0.135
7.50	0.045	0.064	0.091	0.128	0.181	0.181	0.181
7.75	0.056	0.080	0.113	0.159	0.22	0.22	0.22
8.00	0.065	0.092	0.130	0.184	0.26	0.26	0.26
8.25	0.065	0.092	0.130	0.184	0.26	0.26	0.26
8.50	0.065	0.092	0.130	0.184	0.26	0.26	0.26
8.75	0.065	0.092	0.130	0.184	0.26	0.26	0.26
9.00	0.065	0.092	0.130	0.184	0.26	0.26	0.26

Total Ammonia (mg/liter NH<sub>3</sub>)

6.50	35	33	31	30	29	20	14.3
6.75	32	30	28	27	27	18.6	13.2
7.00	28	26	25	24	23	16.4	11.6
7.25	23	22	20	19.7	19.2	13.4	9.5
7.50	17.4	16.3	15.5	14.9	14.6	10.2	7.3
7.75	12.2	11.4	10.9	10.5	10.3	7.2	5.2
8.00	8.0	7.5	7.1	6.9	6.8	4.8	3.5
8.25	4.5	4.2	4.1	4.0	3.9	2.8	2.1
8.50	2.6	2.4	2.3	2.3	2.3	1.71	1.28
8.75	1.47	1.40	1.37	1.38	1.42	1.07	0.83
9.00	0.86	0.83	0.83	0.86	0.91	0.72	0.58

**B. Salmonids and Other Sensitive Coldwater Species Absent**

Un-ionized Ammonia (mg/liter NH<sub>3</sub>)

6.50	0.0091	0.0129	0.0182	0.026	0.036	0.051	0.051
6.75	0.0149	0.021	0.030	0.042	0.059	0.084	0.084
7.00	0.023	0.033	0.046	0.066	0.093	0.131	0.131
7.25	0.034	0.048	0.068	0.095	0.135	0.190	0.190
7.50	0.045	0.064	0.091	0.128	0.181	0.26	0.26
7.75	0.056	0.080	0.113	0.159	0.22	0.32	0.32
8.00	0.065	0.092	0.130	0.184	0.26	0.37	0.37
8.25	0.065	0.092	0.130	0.184	0.26	0.37	0.37
8.50	0.065	0.092	0.130	0.184	0.26	0.37	0.37
8.75	0.065	0.092	0.130	0.184	0.26	0.37	0.37
9.00	0.065	0.092	0.130	0.184	0.26	0.37	0.37

Total Ammonia (mg/liter NH<sub>3</sub>)

6.50	35	33	31	30	29	29	20
6.75	32	30	28	27	27	26	18.6
7.00	28	26	25	24	23	23	16.4
7.25	23	22	20	19.7	19.2	19.0	13.5
7.50	17.4	16.3	15.5	14.9	14.6	14.5	10.3
7.75	12.2	11.4	10.9	10.5	10.3	10.2	7.3
8.00	8.0	7.5	7.1	6.9	6.8	6.8	4.9
8.25	4.5	4.2	4.1	4.0	3.9	4.0	2.9
8.50	2.6	2.4	2.3	2.3	2.3	2.4	1.81
8.75	1.47	1.40	1.37	1.38	1.42	1.52	1.18
9.00	0.86	0.83	0.83	0.86	0.91	1.01	0.82

\* To convert these values to mg/liter N, multiply by 0.822.

## (2) 4-day average concentrations for ammonia.\*

pH	0 C	5 C	10 C	15 C	20 C	25 C	30 C
<b>A. Salmonids or Other Sensitive Coldwater Species Present</b>							
Un-ionized Ammonia (mg/liter NH <sub>3</sub> )							
6.50	0.0007	0.0009	0.0013	0.0019	0.0019	0.0019	0.0019
6.75	0.0012	0.0017	0.0023	0.0033	0.0033	0.0033	0.0033
7.00	0.0021	0.0029	0.0042	0.0059	0.0059	0.0059	0.0059
7.25	0.0037	0.0052	0.0074	0.0105	0.0105	0.0105	0.0105
7.50	0.0066	0.0093	0.0132	0.0186	0.0186	0.0186	0.0186
7.75	0.0109	0.0153	0.022	0.031	0.031	0.031	0.031
8.00	0.0126	0.0177	0.025	0.035	0.035	0.035	0.035
8.25	0.0126	0.0177	0.025	0.035	0.035	0.035	0.035
8.50	0.0126	0.0177	0.025	0.035	0.035	0.035	0.035
8.75	0.0126	0.0177	0.025	0.035	0.035	0.035	0.035
9.00	0.0126	0.0177	0.025	0.035	0.035	0.035	0.035
Total Ammonia (mg/liter NH <sub>3</sub> )							
6.50	2.5	2.4	2.2	2.2	1.49	1.04	0.73
6.75	2.5	2.4	2.2	2.2	1.49	1.04	0.73
7.00	2.5	2.4	2.2	2.2	1.49	1.04	0.74
7.25	2.5	2.4	2.2	2.2	1.50	1.04	0.74
7.50	2.5	2.4	2.2	2.2	1.50	1.05	0.74
7.75	2.3	2.2	2.1	2.0	1.40	0.99	0.71
8.00	1.53	1.44	1.37	1.33	0.93	0.66	0.47
8.25	0.87	0.82	0.78	0.76	0.54	0.39	0.28
8.50	0.49	0.47	0.45	0.44	0.32	0.23	0.17
8.75	0.28	0.27	0.26	0.27	0.19	0.15	0.11
9.00	0.16	0.16	0.16	0.16	0.13	0.10	0.08
-----							
<b>B. Salmonids and Other Sensitive Coldwater Species Absent†</b>							
Un-ionized Ammonia (mg/liter NH <sub>3</sub> )							
6.50	0.0007	0.0009	0.0013	0.0019	0.0026	0.0026	0.0026
6.75	0.0012	0.0017	0.0023	0.0033	0.0047	0.0047	0.0047
7.00	0.0021	0.0029	0.0042	0.0059	0.0083	0.0083	0.0083
7.25	0.0037	0.0052	0.0074	0.0105	0.0148	0.0148	0.0148
7.50	0.0066	0.0093	0.0132	0.0186	0.026	0.026	0.026
7.75	0.0109	0.0153	0.022	0.031	0.043	0.043	0.043
8.00	0.0126	0.0177	0.025	0.035	0.050	0.050	0.050
8.25	0.0126	0.0177	0.025	0.035	0.050	0.050	0.050
8.50	0.0126	0.0177	0.025	0.035	0.050	0.050	0.050
8.75	0.0126	0.0177	0.025	0.035	0.050	0.050	0.050
9.00	0.0126	0.0177	0.025	0.035	0.050	0.050	0.050
Total Ammonia (mg/liter NH <sub>3</sub> )							
6.50	2.5	2.4	2.2	2.2	2.1	1.46	1.03
6.75	2.5	2.4	2.2	2.2	2.1	1.47	1.04
7.00	2.5	2.4	2.2	2.2	2.1	1.47	1.04
7.25	2.5	2.4	2.2	2.2	2.1	1.48	1.05
7.50	2.5	2.4	2.2	2.2	2.1	1.49	1.06
7.75	2.3	2.2	2.1	2.0	1.98	1.39	1.00
8.00	1.53	1.44	1.37	1.33	1.31	0.93	0.67
8.25	0.87	0.82	0.78	0.76	0.76	0.54	0.40
8.50	0.49	0.47	0.45	0.44	0.45	0.33	0.25
8.75	0.28	0.27	0.26	0.27	0.27	0.21	0.16
9.00	0.16	0.16	0.16	0.16	0.17	0.14	0.11

\* To convert these values to mg/liter N, multiply by 0.822.

† Site-specific criteria development is strongly suggested at temperatures above 20 C because of the limited data available to generate the criteria recommendation, and at temperatures below 20 C because of the limited data and because small changes in the criteria may have significant impact on the level of treatment required in meeting the recommended criteria.

salmonids are less certain at temperatures much below 20 C than those tabulated at temperatures near 20 C. Where the treatment levels needed to meet these criteria below 20 C may be substantial, use of site-specific criteria is strongly suggested. Development of such criteria should be based upon site-specific toxicity tests.

Data available for saltwater species are insufficient to derive a criterion for salt water.

The recommended exceedence frequency of three years is the Agency's best scientific judgment of the average amount of time it will take an unstressed system to recover from a pollution event in which exposure to ammonia exceeds the criterion. Stressed systems, for example, one in which several outfalls occur in a limited area, would be expected to require more time for recovery. The resilience of ecosystems and their ability to recover differ greatly, however, and site-specific criteria may be established if adequate justification is provided.

The use of criteria in designing waste treatment facilities requires the selection of an appropriate wasteload allocation model. Dynamic models are preferred for the application of these criteria. Limited data or other factors may make their use impractical, in which case one should rely on a steady-state model. The Agency recommends the interim use of 1Q5 or 1Q10 for Criterion Maximum Concentration (CMC) design flow and 7Q5 or 7Q10 for the Criterion Continuous Concentration (CCC) design flow in steady-state models for unstressed and stressed systems respectively. The Agency acknowledges that the CCC stream flow averaging period used for steady-state wasteload allocation modelling may be as long as 30 days in situations involving POTWs designed to remove ammonia where limited variability of effluent pollutant concentration and resultant concentrations in receiving waters can be

demonstrated. In cases where low variability can be demonstrated, longer averaging periods for the ammonia CCC (e.g., 30-day averaging periods) would be acceptable because the magnitude and duration of exceedences above the CCC would be sufficiently limited. These matters are discussed in more detail in the Technical Support Document for Water Quality-Based Toxics Control (U.S. EPA, 1985a).

## EXAMPLES OF SITE-SPECIFIC CRITERIA

National criteria are subject to modification, if appropriate, to reflect local conditions. One method provided in the Site-Specific Criteria Guidelines (U.S. Environmental Protection Agency 1982) for such modification is to base certain calculations only on those species that occur in the body of water of interest. As an example of how site-specific criteria for ammonia may differ from the national criteria, such recalculations were performed for several sites.

The sites were chosen on the basis of readily available information on the presence of fish and invertebrate species and on a reasonable diversity between sites. The sites were:

- (1) Naugatuck River, Waterbury, Connecticut  
(U.S. Environmental Protection Agency 1985a)
- (2) Five Mile Creek, Birmingham, Alabama  
(U.S. Environmental Protection Agency 1985b)
- (3) Piceance Creek, Colorado  
(Goettl and Edde 1978; Gray and Ward 1978)
- (4) Ottawa River, Lima, Ohio  
(Mount et al. 1984)

The calculations here are for pHs of 7 and 8 and temperatures of 10 and 20 C. This exercise is meant just to illustrate how variation in organisms among sites will result in different criteria formulations than the national criteria; specific design conditions for each site were not addressed.

For each site, available surveys of species occurrence were used to identify which of the genera tested for acute toxicity (Table 3) were present. Minimum data requirements for diversity of organisms were met except where inappropriate to a site (U.S. Environmental Protection Agency

1982). The national  $GMAV_{ref}$  (Table 3) was used for each genus, even if based in part or whole on species not occurring at the site. If a family was present at a site, but none of the site genera were tested or the site genera were not identified, the  $FMAV_{ref}$  for that family (the geometric mean of the  $GMAV_{ref}$ 's available for the family) was also used. The data so developed for each site are listed in the following table.

Sites 1 and 3 included salmonids, so the temperature caps (TCAP) for the log-linear temperature relationship for FAVs and FCVs were set as specified in the national criterion for sites with salmonids (20 C for FAVs, 15 C for FCVs). For sites 2 and 4, the TCAP was raised to 25 C for FAVs and 20 C for FCVs, as specified in the national criterion for sites lacking salmonids.

The Guidelines method for estimating the FAV as the fifth percentile of MAVs was applied to the set of  $GMAV_{ref}$ 's selected for each site. If the  $FAV_{ref}$ 's so computed exceeded the  $SMAV_{ref}$  of an important species at a site, or the  $MAV_{ref}$  of an important size class of an important species, the  $FAV_{ref}$  was lowered to the lowest such  $MAV_{ref}$ . The FAVs at each site were then computed by adjusting the  $FAV_{ref}$  to the specified temperature and pH using the relationship  $FAV_{ref}/FT/FPH$ , where FT and FPH are as specified for the national criterion. The one-hour average concentration criteria were set to one-half of the site FAVs.

The FCVs at each site at each particular temperature and pH were computed by the formula  $FAV_{ref}/FT/FPH/RATIO$ , where FT, FPH, and RATIO are as specified for the national criterion. If the  $FAV_{ref}$  was reduced for the 1-hour criteria to reflect an age/size class, it was restored for the above calculation to what it would be without such a reduction. If a resultant FCV at a site exceeded the chronic value of an important species present at the site, FCVs at all pHs and temperatures were proportionally

lowered until the chronic value was not exceeded. The 4-day average concentration criteria were set to the FCVs.

For site 3, which includes the mountain whitefish (*Prosopium*), the fifth percentile  $FAV_{ref}$  is about 15% lower than for the national criterion, due to the lower number of total genera causing the fifth percentile to be closer to the acute value for the whitefish. For the other sites, lacking the whitefish, the fifth percentile  $FAV_{ref}$  is above that for the national criterion, but by only several percent due to the presence of other sensitive genera and the lower number of total organisms partly compensating for the lack of the whitefish.

Only site 3 included rainbow trout, so only its  $FAV_{ref}$  was lowered to the  $MAV_{ref}$  of adult rainbow trout, as for the national criterion. As a consequence, the 1-hour average criterion for this site is identical to the national criterion. For the other three sites, the 1-hour average criterion is about 40% greater than the national number for a wide range of species composition. This greater value is not necessarily due to differences in general species sensitivities, but could reflect unavailability of information on the sensitivity of different age/size classes for species other than rainbow trout; these higher numbers should therefore be treated with caution as perhaps providing relatively less protection than the national criterion.

For the 4-day average concentration, the difference among sites and between sites and the national criterion are even less. The  $FAV_{ref}$ s used for computing the 4-day average concentration at the four sites are the same or slightly (<10%) less than for the national criterion. Consequently, the criterion for 4-day average concentrations at the sites are virtually the same as for the national criterion.

Some other sites may show higher site-specific criteria, but these increases will not be major. Due to the numerous, diverse genera with  $GMAV_{ref}$  in the 1.1-1.4 mg/liter  $NH_3$  range, few sites will have a  $FAV_{ref}$  greater than 1.1, resulting in 1-hour average concentration criteria little more than twice the national criteria at  $T < 20$  C and 4-day average criteria no more than 40% greater than the national criteria at  $T < 15$  C. At higher temperatures, greater increases are possible if temperature caps for the log-linear temperature relationship are raised, but at low temperatures additional increases are unlikely given current data on acute sensitivity and acute-chronic ratios.

Site-specific ammonia criteria examples for four sites

Site Number	1	2	3	4	
<u>Genus</u>			<u>GMAV<sub>ref</sub></u>		
Prosopium	a	a	0.56	a	
Notemigonus	0.76	0.76	a	0.76	
Etheostoma	0.88	0.88	a	0.88	
Salmo	1.10	a	1.10	a	
Musculium	a	a	1.10 <sup>b</sup>	a	
Lepomis	1.16	1.16	a	1.16	
Stizostedion	a	a	a	a	
Notropis	1.23	1.23	1.23	1.23	
Oncorhynchus	a	a	a	a	
Camptostoma	a	1.30	a	1.30	
Micropterus	1.34	1.34	a	1.34	
Dendrocoelium	1.40	1.40	a	1.40	
Poecilia	a	a	a	a	
Daphnia	1.49	b	a	b	
Ictalurus	1.63	1.63	1.63	1.63	
Morone	a	a	a	a	
Salvelinus	a	a	1.69	a	
Catostomus	1.79	1.79 <sup>b</sup>	1.79	1.79	
Simocephalus	1.89	1.79 <sup>b</sup>	a	1.79 <sup>b</sup>	
Physa	1.95 <sup>b</sup>	1.95	1.95	1.95	
Ceriodaphnia	1.96	b	a	b	
Pimephales	a	a	2.07	2.07	
Arcynopteryx	a	a	2.29 <sup>b</sup>	a	
Cottus	a	2.35	2.35	a	
Gambusia	a	2.48	a	a	
Tubifex	2.70 <sup>b</sup>	2.70	a	2.70	
Hellgram	2.76	a	2.76 <sup>b</sup>	a	
Crangonyx	a	a	3.12 <sup>b</sup>	a	
Calibaetis	3.18	3.18 <sup>b</sup>	3.18	3.18	
Asellus	4.02	4.02 <sup>b</sup>	4.02	a	
Ephemerella	a	a	5.25	a	
Stenelmis	a	8.00	8.00 <sup>b</sup>	8.00	
Orconectes	8.48	8.48	a	8.48	
Philarctus	11.4 <sup>b</sup>	a	11.4 <sup>b</sup>	a	
FAV <sub>ref</sub> (5th percentile)	0.75	0.73	0.60 <sup>c,d</sup>	0.72	
1-hour average concentration (mg/liter NH <sub>3</sub> )	pH=7; T=10:	0.067	0.065	0.046	0.064
	T=20:	0.133	0.130	0.093	0.127
	pH=8; T=10:	0.188	0.183	0.130	0.180
	T=20:	0.38	0.37	0.26	0.36
4-day average concentration (mg/liter NH <sub>3</sub> )	pH=7; T=10:	0.0039	0.0038	0.0042	0.37
	T=20:	0.0055	0.0076	0.0059	0.75
	pH=8; T=10:	0.023	0.023	0.025	0.22
	T=20:	0.032	0.046	0.034	0.45

(a) Genus and family absent from site.

(b) Genus absent or unidentified, but family present; FMAV<sub>ref</sub> used.

(c) For 1-hour average, FAV<sub>ref</sub> lowered to the MAV<sub>ref</sub> of adult rainbow trout (0.52).

(d) For 4-day average, FAV<sub>ref</sub> recalculated as 0.80 after GMAV<sub>ref</sub> of Prosopium raised to 0.78 to reflect impact of size on acute toxicity.

Table 1. Acute Toxicity of Ammonia to Aquatic Animals

<u>Species</u>	<u>Life Stage or Size</u>	<u>Chemical</u>	<u>Method<sup>a</sup></u>	<u>Effect<sup>b</sup></u>	<u>Concentration (mg/L NH<sub>3</sub>)</u>	<u>pH</u>	<u>Temperature (°C)</u>	<u>D.O. (mg/L)</u>	<u>Reference</u>
<u>FRESHWATER SPECIES</u>									
<u>Flatworm, Dendrocoelum lacteum (Procotyla fluviatilis)</u>	-	NH <sub>4</sub> Cl	S,U	LC50	1.4 <sup>d,f</sup>	8.2	18	-	Stammer 1953
<u>Tubificid worm, Tubifex tubifex</u>	-	NH <sub>4</sub> Cl	S,U	LC50	2.7 <sup>d,f</sup>	8.2	12	-	Stammer 1953
<u>Snail, Physa gyrina</u>	Adult	NH <sub>4</sub> Cl	FT,M	LC50	1.59	8.0	4.0	12.5	West 1985
<u>Snail, Physa gyrina</u>	Adult	NH <sub>4</sub> Cl	FT,M	LC50	2.09	8.2	5.5	12.3	West 1985
<u>Snail, Physa gyrina</u>	Adult	NH <sub>4</sub> Cl	FT,M	LC50	2.49	8.1	12.1	10.0	West 1985
<u>Snail, Physa gyrina</u>	Adult	NH <sub>4</sub> Cl	FT,M	LC50	2.16	8.2	12.8	9.5	West 1985
<u>Snail, Physa gyrina</u>	Adult	NH <sub>4</sub> Cl	FT,M	LC50	1.78	8.0	13.3	10.4	West 1985
<u>Snail, Physa gyrina</u>	Adult	NH <sub>4</sub> Cl	FT,M	LC50	1.71	8.0	24.9	7.1	West 1985
<u>Snail, Helisoma trivolvis</u>	Adult	NH <sub>4</sub> Cl	FT,M	LC50	2.76	8.2	12.9	9.5	West 1985
<u>Clam, Musculium transversum</u>	Adult	NH <sub>4</sub> Cl	FT,M	LC50	0.93	8.2	5.4	12.3	West 1985
<u>Clam, Musculium transversum</u>	Adult	NH <sub>4</sub> Cl	FT,M	LC50	1.29	8.1	14.6	9.6	West 1985
<u>Clam, Musculium transversum</u>	Adult	NH <sub>4</sub> Cl	FT,M	LC50	1.10	8.6	20.5	8.6	West 1985
<u>Cladoceran, Ceriodaphnia acanthina</u>	<2-h	NH <sub>4</sub> Cl	FT,M	LC50	0.770 <sup>d</sup>	7.06	24	4.8-5.3	Mount 1982

Table 1. (Continued)

<u>Species</u>	<u>Life Stage or Size</u>	<u>Chemical</u>	<u>Method<sup>a</sup></u>	<u>Effect<sup>b</sup></u>	<u>Concentration (mg/L NH<sub>3</sub>)</u>	<u>pH</u>	<u>Temperature (°C)</u>	<u>D.O. (mg/L)</u>	<u>Reference</u>
<u>Cladoceran, Daphnia magna</u>	Mixed ages	NH <sub>4</sub> Cl	S,M	LC50	2.08	8.2	25	7.0-8.5	Parkhurst et al. 1979, 1981 <sup>d</sup>
<u>Cladoceran, Daphnia magna</u>	<24-h old	NH <sub>4</sub> Cl	S,M	LC50	2.45	7.95	22.0	-	Russo et al. 1985
<u>Cladoceran, Daphnia magna</u>	<24-h old	NH <sub>4</sub> Cl	S,M	LC50	2.69	8.07	19.6	7.4	Russo et al. 1985
<u>Cladoceran, Daphnia magna</u>	<24-h old	NH <sub>4</sub> Cl	S,M	LC50	2.50	8.09	20.9	6.8	Russo et al. 1985
<u>Cladoceran, Daphnia magna</u>	<24-h old	NH <sub>4</sub> Cl	S,M	LC50	2.77	8.15	22.0	-	Russo et al. 1985
<u>Cladoceran, Daphnia magna</u>	<24-h old	NH <sub>4</sub> Cl	S,M	LC50	2.38	8.04	22.8	-	Russo et al. 1985
<u>Cladoceran, Daphnia magna</u>	<24-h old	NH <sub>4</sub> Cl	S,M	LC50	0.75	7.51	20.1	7.6	Russo et al. 1985
<u>Cladoceran, Daphnia magna</u>	<24-h old	NH <sub>4</sub> Cl	S,M	LC50	0.90	7.53	20.1	8.0	Russo et al. 1985
<u>Cladoceran, Daphnia magna</u>	<24-h old	NH <sub>4</sub> Cl	S,M	LC50	0.53	7.4	20.6	8.0	Russo et al. 1985
<u>Cladoceran, Daphnia magna</u>	<24-h old	NH <sub>4</sub> Cl	S,M	LC50	0.67	7.5	20.3	8.0	Russo et al. 1985
<u>Cladoceran, Daphnia magna</u>	<24-h old	NH <sub>4</sub> Cl	FT,M	LC50	4.94 <sup>c</sup>	8.11-8.58	19.7	95% Saturated	Reinbold & Pescitelli 1982a
<u>Cladoceran, Daphnia pulicaria</u>	-	NH <sub>4</sub> Cl	FT,M	LC50	1.16	8.0-8.1	14	7.2-7.4	DeGraeve et al. 1980
<u>Cladoceran, Simocephalus vetulus</u>	<24-h old	NH <sub>4</sub> Cl	FT,M	LC50	0.613 <sup>d</sup>	7.06	24	4.8-5.3	Mount 1982
<u>Cladoceran, Simocephalus vetulus</u>	Adult	NH <sub>4</sub> Cl	FT,M	LC50	2.29	8.3	17.0	9.5	West 1985

Table 1. (Continued)

<u>Species</u>	<u>Life Stage or Size</u>	<u>Chemical</u>	<u>Method<sup>a</sup></u>	<u>Effect<sup>b</sup></u>	<u>Concentration (mg/L NH<sub>3</sub>)</u>	<u>pH</u>	<u>Temperature (°C)</u>	<u>D.O. (mg/L)</u>	<u>Reference</u>
<u>Isopod, Asellus racovitzai</u>	12 mm	NH <sub>4</sub> Cl	FT,M	LC50	2.94	7.81	11.9	9.1	Thurston et al. 1983a
<u>Isopod, Asellus racovitzai</u>	Adult	NH <sub>4</sub> Cl	FT,M	LC50	4.95	8.0	4.0	12.6	West 1985
<u>Amphipod, Crangonyx pseudogracilis</u>	Adult	NH <sub>4</sub> Cl	FT,M	LC50	2.76	8.0	4.0	12.6	West 1985
<u>Amphipod, Crangonyx pseudogracilis</u>	Adult	NH <sub>4</sub> Cl	FT,M	LC50	5.63	8.0	12.1	10.1	West 1985
<u>Amphipod, Crangonyx pseudogracilis</u>	Adult	NH <sub>4</sub> Cl	FT,M	LC50	3.56	8.2	13.0	9.5	West 1985
<u>Amphipod, Crangonyx pseudogracilis</u>	Adult	NH <sub>4</sub> Cl	FT,M	LC50	3.29	8.0	13.3	10.4	West 1985
<u>Amphipod, Crangonyx pseudogracilis</u>	Adult	NH <sub>4</sub> Cl	FT,M	LC50	1.63	8.0	24.9	7.1	West 1985
<u>Crayfish, Orconectes nalis</u>	2.78 cm	NH <sub>4</sub> Cl	FT,M	LC50	3.15	7.6-9.0	26-27	7.8-8.2	Evans 1979
<u>Crayfish, Orconectes immunis</u>	Adult	NH <sub>4</sub> Cl	FT,M	LC50	22.8	8.2	4.6	12.4	West 1985
<u>Mayfly, Callibaetis sp. near montanus</u>	10 mm	NH <sub>4</sub> Cl	FT,M	LC50	1.80	7.81	11.9	9.1	Thurston et al. 1984a
<u>Mayfly, Callibaetis skoklanus</u>	Nymph	NH <sub>4</sub> Cl	FT,M	LC50	4.82	7.9	13.3	10.3	West 1985
<u>Mayfly, Ephemerella grandis</u>	11 mm	NH <sub>4</sub> Cl	FT,M	LC50	4.96	7.84	12.8	8.3	Thurston et al. 1984a
<u>Mayfly, Ephemerella grandis</u>	11 mm	NH <sub>4</sub> Cl	FT,M	LC50	5.88	7.85	12.0	8.8	Thurston et al. 1984a
<u>Mayfly, Ephemerella grandis</u>	10 mm	NH <sub>4</sub> Cl	FT,M	LC50	3.86	7.84	13.2	8.4	Thurston et al. 1984a

Table 1. (Continued)

<u>Species</u>	<u>Life Stage or Size</u>	<u>Chemical</u>	<u>Method<sup>a</sup></u>	<u>Effect<sup>b</sup></u>	<u>Concentration (mg/L NH<sub>3</sub>)</u>	<u>pH</u>	<u>Temperature (°C)</u>	<u>D.O. (mg/L)</u>	<u>Reference</u>
<u>Stonefly, Arcynopteryx parallela</u>	19 mm	NH <sub>4</sub> Cl	FT,M	LC50	2.06	7.76	13.8	8.5	Thurston et al. 1984a
<u>Stonefly, Arcynopteryx parallela</u>	19 mm	NH <sub>4</sub> Cl	FT,M	LC50	2.00	7.81	13.1	8.9	Thurston et al. 1984a
<u>Caddisfly, Philarctus quærns</u>	Larvae	NH <sub>4</sub> Cl	FT,M	LC50	10.2	7.8	13.3	10.4	West 1985
<u>Beetle, Stenelmis sexlineata</u>	2.8 mm	NH <sub>4</sub> Cl	FT,M	LC50	8.00 <sup>c</sup>	8.7	25	8.0	Hazel et al. 1979
<u>Pink salmon, Oncorhynchus gorbuscha</u>	Late alevins	(NH <sub>4</sub> ) <sub>2</sub> SO <sub>4</sub>	S,M	LC50	0.083	6.3-6.5	3.7-4.8	-	Rice & Bailey 1980
<u>Pink salmon, Oncorhynchus gorbuscha</u>	Fry	(NH <sub>4</sub> ) <sub>2</sub> SO <sub>4</sub>	S,M	LC50	0.1	6.3-6.5	3.7-4.8	-	Rice & Bailey 1980
<u>Coho salmon, Oncorhynchus kisutch</u>	Juvenile	NH <sub>4</sub> Cl	FT,M	LC50	0.272	7.0	15	-	Robinson-Wilson & Seim 1975
<u>Coho salmon, Oncorhynchus kisutch</u>	Juvenile	NH <sub>4</sub> Cl	FT,M	LC50	0.280	7.0	15	-	Robinson-Wilson & Seim 1975
<u>Coho salmon, Oncorhynchus kisutch</u>	Juvenile	NH <sub>4</sub> Cl	FT,M	LC50	0.550	7.5	15	-	Robinson-Wilson & Seim 1975
<u>Coho salmon, Oncorhynchus kisutch</u>	Juvenile	NH <sub>4</sub> Cl	FT,M	LC50	0.528	7.5	15	-	Robinson-Wilson & Seim 1975
<u>Coho salmon, Oncorhynchus kisutch</u>	Juvenile	NH <sub>4</sub> Cl	FT,M	LC50	0.712	8.0	15	-	Robinson-Wilson & Seim 1975
<u>Coho salmon, Oncorhynchus kisutch</u>	Juvenile	NH <sub>4</sub> Cl	FT,M	LC50	0.700	8.0	15	-	Robinson-Wilson & Seim 1975
<u>Coho salmon, Oncorhynchus kisutch</u>	Juvenile	NH <sub>4</sub> Cl	FT,M	LC50	0.880	8.5	15	-	Robinson-Wilson & Seim 1975
<u>Coho salmon, Oncorhynchus kisutch</u>	6. g	NH <sub>4</sub> Cl	FT,M	LC50	0.55 <sup>c</sup>	8.04-8.20	14.7-	>80% Saturated	Buckley 1978

Table 1. (Continued)

<u>Species</u>	<u>Life Stage or Size</u>	<u>Chemical</u>	<u>Method<sup>a</sup></u>	<u>Effect<sup>b</sup></u>	<u>Concentration (mg/L NH<sub>3</sub>)</u>	<u>pH</u>	<u>Temperature (°C)</u>	<u>D.O. (mg/L)</u>	<u>Reference</u>
<u>Chinook salmon, Oncorhynchus tshawytscha</u>	15.3 g	NH <sub>4</sub> Cl	FT,M	LC50	0.476	7.82	12.2	7.78	Thurston & Meyn 1984
<u>Chinook salmon, Oncorhynchus tshawytscha</u>	18.1 g	NH <sub>4</sub> Cl	FT,M	LC50	0.456	7.84	12.3	7.87	Thurston & Meyn 1984
<u>Chinook salmon, Oncorhynchus tshawytscha</u>	14.4 g	NH <sub>4</sub> Cl	FT,M	LC50	0.399	7.87	13.5	7.26	Thurston & Meyn 1984
<u>Golden trout, Salmo aquabonita</u>	0.09 g	NH <sub>4</sub> Cl	FT,M	LC50	0.755	8.06	13.2	8.9	Thurston & Russo 1981
<u>Cutthroat trout, Salmo clarki</u>	1.0 g	NH <sub>4</sub> Cl	FT,M	LC50	0.80	7.81	13.1	8.6	Thurston et al. 1978
<u>Cutthroat trout, Salmo clarki</u>	1.0 g	NH <sub>4</sub> Cl	FT,M	LC50	0.66	7.80	12.8	8.4	Thurston et al. 1978
<u>Cutthroat trout, Salmo clarki</u>	3.3 g	NH <sub>4</sub> Cl	FT,M	LC50	0.62	7.80	12.4	8.2	Thurston et al. 1978
<u>Cutthroat trout, Salmo clarki</u>	3.4 g	NH <sub>4</sub> Cl	FT,M	LC50	0.52	7.78	12.2	8.3	Thurston et al. 1978
<u>Rainbow trout, Salmo gairdneri</u>	1-d-old sac fry	NH <sub>4</sub> Cl	FT,M	LC50	>0.486	7.4	14.5	>80% Saturated	Calamari et al. 1977, 1981
<u>Rainbow trout, Salmo gairdneri</u>	5-d-old sac fry	NH <sub>4</sub> Cl	FT,M	LC50	>0.486	7.4	14.5	>80% Saturated	Calamari et al. 1977, 1981
<u>Rainbow trout, Salmo gairdneri</u>	13-d-old	NH <sub>4</sub> Cl	FT,M	LC50	0.325	7.4	14.4	>80% Saturated	Calamari et al. 1977, 1981
<u>Rainbow trout, Salmo gairdneri</u>	17-d-old	NH <sub>4</sub> Cl	FT,M	LC50	0.370	7.4	14.5	>80% Saturated	Calamari et al. 1977, 1981
<u>Rainbow trout, Salmo gairdneri</u>	51-d-old, 1.7-1.9 cm	NH <sub>4</sub> Cl	FT,M	LC50	0.160	7.4	14.5	>80% Saturated	Calamari et al. 1977, 1981
<u>Rainbow trout, Salmo gairdneri</u>	325-d-old, 8-10 cm	NH <sub>4</sub> Cl	FT,M	LC50	0.440	7.4	14.5	>80% Saturated	Calamari et al. 1977, 1981

Table 1. (Continued)

<u>Species</u>	<u>Life Stage or Size</u>	<u>Chemical</u>	<u>Method<sup>a</sup></u>	<u>Effect<sup>b</sup></u>	<u>Concentration (mg/L NH<sub>3</sub>)</u>	<u>pH</u>	<u>Temperature (°C)</u>	<u>D.O. (mg/L)</u>	<u>Reference</u>
<u>Rainbow trout, <i>Salmo gairdneri</i></u>	1.48 g	NH <sub>4</sub> Cl	FT,M	LC50	0.697	7.95	10	80% Saturated	Broderius & Smith 1979
<u>Rainbow trout, <i>Salmo gairdneri</i></u>	-	NH <sub>4</sub> Cl	S,M	LC50	0.4	7.5	15	Aerated	Holt & Malcolm 1979
<u>Rainbow trout, <i>Salmo gairdneri</i></u>	-	NH <sub>4</sub> Cl	FT,M	LC50	0.77	8.0-8.1	14	7.2-7.4	DeGraeve et al. 1980
<u>Rainbow trout, <i>Salmo gairdneri</i></u>	0.06 g	NH <sub>4</sub> Cl	FT,M	LC50	0.436	7.90	12.7	8.8	Thurston & Russo 1983
<u>Rainbow trout, <i>Salmo gairdneri</i></u>	0.06 g	NH <sub>4</sub> Cl	FT,M	LC50	0.446	7.90	13.4	8.6	Thurston & Russo 1983
<u>Rainbow trout, <i>Salmo gairdneri</i></u>	0.06 g	NH <sub>4</sub> Cl	FT,M	LC50	0.478	7.91	13.0	8.6	Thurston & Russo 1983
<u>Rainbow trout, <i>Salmo gairdneri</i></u>	0.08 g	NH <sub>4</sub> Cl	FT,M	LC50	0.291	7.91	13.1	8.5	Thurston & Russo 1983
<u>Rainbow trout, <i>Salmo gairdneri</i></u>	0.12 g	NH <sub>4</sub> Cl	FT,M	LC50	0.232	7.88	12.8	9.2	Thurston & Russo 1983
<u>Rainbow trout, <i>Salmo gairdneri</i></u>	0.14 g	NH <sub>4</sub> Cl	FT,M	LC50	0.336	7.88	12.9	8.8	Thurston & Russo 1983
<u>Rainbow trout, <i>Salmo gairdneri</i></u>	0.15 g	NH <sub>4</sub> Cl	FT,M	LC50	0.347	7.87	12.9	8.8	Thurston & Russo 1983
<u>Rainbow trout, <i>Salmo gairdneri</i></u>	0.15 g	NH <sub>4</sub> Cl	FT,M	LC50	0.474	7.95	12.5	9.0	Thurston & Russo 1983
<u>Rainbow trout, <i>Salmo gairdneri</i></u>	0.18 g	NH <sub>4</sub> Cl	FT,M	LC50	0.440	7.87	13.0	8.9	Thurston & Russo 1983
<u>Rainbow trout, <i>Salmo gairdneri</i></u>	0.18 g	NH <sub>4</sub> Cl	FT,M	LC50	0.392	7.87	12.9	8.9	Thurston & Russo 1983
<u>Rainbow trout, <i>Salmo gairdneri</i></u>	0.23 g	NH <sub>4</sub> Cl	FT,M	LC50	0.426	7.88	13.4	8.9	Thurston & Russo 1983

Table 1. (Continued)

<u>Species</u>	<u>Life Stage or Size</u>	<u>Chemical</u>	<u>Methods<sup>a</sup></u>	<u>Effect<sup>b</sup></u>	<u>Concentration (mg/L NH<sub>3</sub>)</u>	<u>pH</u>	<u>Temperature (°C)</u>	<u>D.O. (mg/L)</u>	<u>Reference</u>
<u>Rainbow trout, Salmo gairdneri</u>	0.23 g	NH <sub>4</sub> Cl	FT,M	LC50	0.400	7.87	13.1	8.9	Thurston & Russo 1983
<u>Rainbow trout, Salmo gairdneri</u>	0.33 g	NH <sub>4</sub> Cl	FT,M	LC50	0.497	7.86	13.4	9.0	Thurston & Russo 1983
<u>Rainbow trout, Salmo gairdneri</u>	0.33 g	NH <sub>4</sub> Cl	FT,M	LC50	0.421	7.86	13.0	9.0	Thurston & Russo 1983
<u>Rainbow trout, Salmo gairdneri</u>	0.36 g	NH <sub>4</sub> Cl	FT,M	LC50	0.758	8.08	12.8	9.4	Thurston & Russo 1983
<u>Rainbow trout, Salmo gairdneri</u>	0.47 g	NH <sub>4</sub> Cl	FT,M	LC50	0.572	7.86	12.7	9.0	Thurston & Russo 1983
<u>Rainbow trout, Salmo gairdneri</u>	0.47 g	NH <sub>4</sub> Cl	FT,M	LC50	0.570	7.85	12.5	9.0	Thurston & Russo 1983
<u>Rainbow trout, Salmo gairdneri</u>	0.61 g	NH <sub>4</sub> Cl	FT,M	LC50	0.673	7.85	13.1	8.7	Thurston & Russo 1983
<u>Rainbow trout, Salmo gairdneri</u>	1.01 g	NH <sub>4</sub> Cl	FT,M	LC50	1.09	8.06	13.2	8.8	Thurston & Russo 1983
<u>Rainbow trout, Salmo gairdneri</u>	1.02 g	NH <sub>4</sub> Cl	FT,M	LC50	0.641	7.85	12.3	8.7	Thurston & Russo 1983
<u>Rainbow trout, Salmo gairdneri</u>	1.7 g	NH <sub>4</sub> Cl	FT,M	LC50	0.696	7.79	12.4	8.6	Thurston & Russo 1983
<u>Rainbow trout, Salmo gairdneri</u>	1.7 g	NH <sub>4</sub> Cl	FT,M	LC50	0.772	7.86	14.1	8.8	Thurston & Russo 1983
<u>Rainbow trout, Salmo gairdneri</u>	1.8 g	NH <sub>4</sub> Cl	FT,M	LC50	0.683	7.84	13.8	9.0	Thurston & Russo 1983
<u>Rainbow trout, Salmo gairdneri</u>	2.3 g	NH <sub>4</sub> Cl	FT,M	LC50	0.812	7.80	12.4	8.6	Thurston & Russo 1983
<u>Rainbow trout, Salmo gairdneri</u>	2.5 g	NH <sub>4</sub> Cl	FT,M	LC50	0.632	7.85	13.1	8.7	Thurston & Russo 1983

Table 1. (Continued)

<u>Species</u>	<u>Life Stage or Size</u>	<u>Chemical</u>	<u>Methods<sup>a</sup></u>	<u>Effect<sup>b</sup></u>	<u>Concentration (mg/L NH<sub>3</sub>)</u>	<u>pH</u>	<u>Temperature (°C)</u>	<u>D.O. (mg/L)</u>	<u>Reference</u>
<u>Rainbow trout, <i>Salmo gairdneri</i></u>	2.6 g	NH <sub>4</sub> Cl	FT,M	LC50	0.618	7.87	12.1	9.2	Thurston & Russo 1983
<u>Rainbow trout, <i>Salmo gairdneri</i></u>	4.0 g	NH <sub>4</sub> Cl	FT,M	LC50	0.410	7.71	11.4	8.3	Thurston & Russo 1983
<u>Rainbow trout, <i>Salmo gairdneri</i></u>	4.3 g	NH <sub>4</sub> Cl	FT,M	LC50	0.390	7.71	11.5	8.3	Thurston & Russo 1983
<u>Rainbow trout, <i>Salmo gairdneri</i></u>	4.3 g	NH <sub>4</sub> Cl	FT,M	LC50	0.752	7.84	13.0	8.4	Thurston & Russo 1983
<u>Rainbow trout, <i>Salmo gairdneri</i></u>	4.6 g	NH <sub>4</sub> Cl	FT,M	LC50	0.662	7.83	13.5	8.6	Thurston & Russo 1983
<u>Rainbow trout, <i>Salmo gairdneri</i></u>	5.7 g	NH <sub>4</sub> Cl	FT,M	LC50	0.763	7.80	13.3	7.7	Thurston & Russo 1983
<u>Rainbow trout, <i>Salmo gairdneri</i></u>	6.3 g	NH <sub>4</sub> Cl	FT,M	LC50	0.250	7.44	12.8	8.6	Thurston & Russo 1983
<u>Rainbow trout, <i>Salmo gairdneri</i></u>	6.7 g	NH <sub>4</sub> Cl	FT,M	LC50	0.449	7.84	12.2	8.1	Thurston & Russo 1983
<u>Rainbow trout, <i>Salmo gairdneri</i></u>	7.0 g	NH <sub>4</sub> Cl	FT,M	LC50	0.392	7.87	12.2	7.9	Thurston & Russo 1983
<u>Rainbow trout, <i>Salmo gairdneri</i></u>	7.9 g	NH <sub>4</sub> Cl	FT,M	LC50	0.464	7.90	11.9	8.2	Thurston & Russo 1983
<u>Rainbow trout, <i>Salmo gairdneri</i></u>	8.0 g	NH <sub>4</sub> Cl	FT,M	LC50	0.243	7.50	14.5	8.1	Thurston & Russo 1983
<u>Rainbow trout, <i>Salmo gairdneri</i></u>	8.0 g	NH <sub>4</sub> Cl	FT,M	LC50	0.635	7.82	13.2	7.5	Thurston & Russo 1983
<u>Rainbow trout, <i>Salmo gairdneri</i></u>	8.1 g	NH <sub>4</sub> Cl	FT,M	LC50	0.510	7.75	12.3	6.9	Thurston & Russo 1983
<u>Rainbow trout, <i>Salmo gairdneri</i></u>	9.0 g	NH <sub>4</sub> Cl	FT,M	LC50	0.623	7.84	12.9	7.9	Thurston & Russo 1983

Table 1. (Continued)

<u>Species</u>	<u>Life Stage or Size</u>	<u>Chemical</u>	<u>Methods<sup>a</sup></u>	<u>Effect<sup>b</sup></u>	<u>Concentration (mg/L NH<sub>3</sub>)</u>	<u>pH</u>	<u>Temperature (°C)</u>	<u>D.O. (mg/L)</u>	<u>Reference</u>
<u>Rainbow trout, <i>Salmo gairdneri</i></u>	9.3 g	NH <sub>4</sub> Cl	FT,M	LC50	0.833	7.90	13.0	6.6	Thurston & Russo 1983
<u>Rainbow trout, <i>Salmo gairdneri</i></u>	9.5 g	NH <sub>4</sub> Cl	FT,M	LC50	0.432	7.70	13.9	8.0	Thurston & Russo 1983
<u>Rainbow trout, <i>Salmo gairdneri</i></u>	9.7 g	NH <sub>4</sub> Cl	FT,M	LC50	0.796	7.90	13.0	6.1	Thurston & Russo 1983
<u>Rainbow trout, <i>Salmo gairdneri</i></u>	11.1 g	NH <sub>4</sub> Cl	FT,M	LC50	0.714	7.87	13.0	7.8	Thurston & Russo 1983
<u>Rainbow trout, <i>Salmo gairdneri</i></u>	11.2 g	NH <sub>4</sub> Cl	FT,M	LC50	0.326	7.80	9.7	9.2	Thurston & Russo 1983
<u>Rainbow trout, <i>Salmo gairdneri</i></u>	12.3 g	NH <sub>4</sub> Cl	FT,M	LC50	0.404	7.65	14.3	7.3	Thurston & Russo 1983
<u>Rainbow trout, <i>Salmo gairdneri</i></u>	14.8 g	NH <sub>4</sub> Cl	FT,M	LC50	0.389	7.67	14.0	7.4	Thurston & Russo 1983
<u>Rainbow trout, <i>Salmo gairdneri</i></u>	15.1 g	NH <sub>4</sub> Cl	FT,M	LC50	0.375	7.62	14.4	7.2	Thurston & Russo 1983
<u>Rainbow trout, <i>Salmo gairdneri</i></u>	18.9 g	NH <sub>4</sub> Cl	FT,M	LC50	0.364	7.64	13.1	7.2	Thurston & Russo 1983
<u>Rainbow trout, <i>Salmo gairdneri</i></u>	22.6 g	NH <sub>4</sub> Cl	FT,M	LC50	0.382	7.66	13.6	7.0	Thurston & Russo 1983
<u>Rainbow trout, <i>Salmo gairdneri</i></u>	22.8 g	NH <sub>4</sub> Cl	FT,M	LC50	0.367	7.65	13.2	7.3	Thurston & Russo 1983
<u>Rainbow trout, <i>Salmo gairdneri</i></u>	23.6 g	NH <sub>4</sub> Cl	FT,M	LC50	0.392	7.69	13.4	6.8	Thurston & Russo 1983
<u>Rainbow trout, <i>Salmo gairdneri</i></u>	24.5 g	NH <sub>4</sub> Cl	FT,M	LC50	0.281	7.60	12.9	7.3	Thurston & Russo 1983
<u>Rainbow trout, <i>Salmo gairdneri</i></u>	25.8 g	NH <sub>4</sub> Cl	FT,M	LC50	0.456	7.75	11.8	7.9	Thurston & Russo 1983

Table 1. (Continued)

<u>Species</u>	<u>Life Stage or Size</u>	<u>Chemical</u>	<u>Methods<sup>a</sup></u>	<u>Effect<sup>b</sup></u>	<u>Concentration (mg/L NH<sub>3</sub>)</u>	<u>pH</u>	<u>Temperature (°C)</u>	<u>D.O. (mg/L)</u>	<u>Reference</u>
<u>Rainbow trout, Salmo gairdneri</u>	26.0 g	NH <sub>4</sub> Cl	FT,M	LC50	0.432	7.66	12.8	7.2	Thurston & Russo 1983
<u>Rainbow trout, Salmo gairdneri</u>	28.0 g	NH <sub>4</sub> Cl	FT,M	LC50	0.268	7.60	13.0	7.3	Thurston & Russo 1983
<u>Rainbow trout, Salmo gairdneri</u>	29.6 g	NH <sub>4</sub> Cl	FT,M	LC50	0.307	7.63	12.9	7.2	Thurston & Russo 1983
<u>Rainbow trout, Salmo gairdneri</u>	29.8 g	NH <sub>4</sub> Cl	FT,M	LC50	0.351	7.59	12.7	7.3	Thurston & Russo 1983
<u>Rainbow trout, Salmo gairdneri</u>	38.0 g	NH <sub>4</sub> Cl	FT,M	LC50	0.448	7.68	13.0	7.1	Thurston & Russo 1983
<u>Rainbow trout, Salmo gairdneri</u>	42.0 g	NH <sub>4</sub> Cl	FT,M	LC50	0.552	7.77	13.6	6.2	Thurston & Russo 1983
<u>Rainbow trout, Salmo gairdneri</u>	48.6 g	NH <sub>4</sub> Cl	FT,M	LC50	0.580	7.86	10.2	8.8	Thurston & Russo 1983
<u>Rainbow trout, Salmo gairdneri</u>	52.1 g	NH <sub>4</sub> Cl	FT,M	LC50	0.484	7.88	10.0	9.4	Thurston & Russo 1983
<u>Rainbow trout, Salmo gairdneri</u>	152 g	NH <sub>4</sub> Cl	FT,M	LC50	0.297	7.69	10.7	8.3	Thurston & Russo 1983
<u>Rainbow trout, Salmo gairdneri</u>	248 g	NH <sub>4</sub> Cl	FT,M	LC50	0.327	7.74	10.4	7.7	Thurston & Russo 1983
<u>Rainbow trout, Salmo gairdneri</u>	380 g	NH <sub>4</sub> Cl	FT,M	LC50	0.289	7.76	10.0	7.6	Thurston & Russo 1983
<u>Rainbow trout, Salmo gairdneri</u>	513 g	NH <sub>4</sub> Cl	FT,M	LC50	0.262	7.66	9.8	7.6	Thurston & Russo 1983
<u>Rainbow trout, Salmo gairdneri</u>	558 g	NH <sub>4</sub> Cl	FT,M	LC50	0.312	7.64	10.0	6.9	Thurston & Russo 1983
<u>Rainbow trout, Salmo gairdneri</u>	1122 g	NH <sub>4</sub> Cl	FT,M	LC50	0.201	7.69	10.4	7.1	Thurston & Russo 1983

Table 1. (Continued)

<u>Species</u>	<u>Life Stage or Size</u>	<u>Chemical</u>	<u>Methods<sup>a</sup></u>	<u>Effect<sup>b</sup></u>	<u>Concentration (mg/L NH<sub>3</sub>)</u>	<u>pH</u>	<u>Temperature (°C)</u>	<u>D.O. (mg/L)</u>	<u>Reference</u>
<u>Rainbow trout, <i>Salmo gairdneri</i></u>	1140 g	NH <sub>4</sub> Cl	FT,M	LC50	0.234	7.69	10.7	7.0	Thurston & Russo 1983
<u>Rainbow trout, <i>Salmo gairdneri</i></u>	1496 g	NH <sub>4</sub> Cl	FT,M	LC50	0.249	7.64	9.8	7.2	Thurston & Russo 1983
<u>Rainbow trout, <i>Salmo gairdneri</i></u>	1698 g	NH <sub>4</sub> Cl	FT,M	LC50	0.192	7.65	9.8	6.6	Thurston & Russo 1983
<u>Rainbow trout, <i>Salmo gairdneri</i></u>	2596 g	NH <sub>4</sub> Cl	FT,M	LC50	0.163	7.62	7.9	7.7	Thurston & Russo 1983
<u>Rainbow trout, <i>Salmo gairdneri</i></u>	1.7 g	NH <sub>4</sub> HCO <sub>3</sub>	FT,M	LC50	0.677	8.10	13.9	8.8 <sup>h</sup>	Thurston & Russo 1983
<u>Rainbow trout, <i>Salmo gairdneri</i></u>	1.8 g	NH <sub>4</sub> HCO <sub>3</sub>	FT,M	LC50	0.662	8.12	13.6	9.1 <sup>h</sup>	Thurston & Russo 1983
<u>Rainbow trout, <i>Salmo gairdneri</i></u>	1.7 g	(NH <sub>4</sub> ) <sub>2</sub> HPO <sub>4</sub>	FT,M	LC50	0.636	7.94	12.8	9.2 <sup>h</sup>	Thurston & Russo 1983
<u>Rainbow trout, <i>Salmo gairdneri</i></u>	2.1 g	(NH <sub>4</sub> ) <sub>2</sub> HPO <sub>4</sub>	FT,M	LC50	0.694	7.98	12.5	9.1 <sup>h</sup>	Thurston & Russo 1983
<u>Rainbow trout, <i>Salmo gairdneri</i></u>	1.8 g	(NH <sub>4</sub> ) <sub>2</sub> SO <sub>4</sub>	FT,M	LC50	0.764	7.89	12.4	9.2 <sup>h</sup>	Thurston & Russo 1983
<u>Rainbow trout, <i>Salmo gairdneri</i></u>	2.1 g	(NH <sub>4</sub> ) <sub>2</sub> SO <sub>4</sub>	FT,M	LC50	0.921	7.94	12.5	9.0 <sup>h</sup>	Thurston & Russo 1983
<u>Rainbow trout, <i>Salmo gairdneri</i></u>	9.4 g	NH <sub>4</sub> Cl	FT,M	LC50	0.856	7.85	16.1	6.6	Thurston & Russo 1983
<u>Rainbow trout, <i>Salmo gairdneri</i></u>	11.9 g	NH <sub>4</sub> Cl	FT,M	LC50	0.801	7.88	16.7	6.3	Thurston & Russo 1983
<u>Rainbow trout, <i>Salmo gairdneri</i></u>	7.1 g	NH <sub>4</sub> Cl	FT,M	LC50	0.897	7.91	19.0	7.1	Thurston & Russo 1983
<u>Rainbow trout, <i>Salmo gairdneri</i></u>	10.1 g	NH <sub>4</sub> Cl	FT,M	LC50	0.942	7.91	19.1	6.2	Thurston & Russo 1983

Table 1. (Continued)

<u>Species</u>	<u>Life Stage or Size</u>	<u>Chemical</u>	<u>Methods<sup>a</sup></u>	<u>Effect<sup>b</sup></u>	<u>Concentration (mg/L NH<sub>3</sub>)</u>	<u>pH</u>	<u>Temperature (°C)</u>	<u>D.O. (mg/L)</u>	<u>Reference</u>
<u>Rainbow trout, <i>Salmo gairdneri</i></u>	8.6 g	NH <sub>4</sub> Cl	FT,M	LC50	0.931	7.96	19.2	6.4	Thurston & Russo 1983
<u>Rainbow trout, <i>Salmo gairdneri</i></u>	10.6 g	NH <sub>4</sub> Cl	FT,M	LC50	0.158 <sup>C</sup>	6.51	14.1	7.9	Thurston et al. 1981c <sup>1</sup>
<u>Rainbow trout, <i>Salmo gairdneri</i></u>	9.0 g	NH <sub>4</sub> Cl	FT,M	LC50	0.184 <sup>C</sup>	6.80	14.1	7.9	Thurston et al. 1981c <sup>1</sup>
<u>Rainbow trout, <i>Salmo gairdneri</i></u>	8.2 g	NH <sub>4</sub> Cl	FT,M	LC50	0.454 <sup>C</sup>	7.30	14.0	8.0	Thurston et al. 1981c <sup>1</sup>
<u>Rainbow trout, <i>Salmo gairdneri</i></u>	9.0 g	NH <sub>4</sub> Cl	FT,M	LC50	0.799 <sup>C</sup>	8.29	14.1	7.8	Thurston et al. 1981c <sup>1</sup>
<u>Rainbow trout, <i>Salmo gairdneri</i></u>	10.0 g	NH <sub>4</sub> Cl	FT,M	LC50	0.684 <sup>C</sup>	8.82	13.9	8.1	Thurston et al. 1981c <sup>1</sup>
<u>Rainbow trout, <i>Salmo gairdneri</i></u>	10.4 g	NH <sub>4</sub> Cl	FT,M	LC50	0.648 <sup>C</sup>	9.01	14.5	7.4	Thurston et al. 1981c <sup>1</sup>
<u>Rainbow trout, <i>Salmo gairdneri</i></u>	4.0 g	NH <sub>4</sub> Cl	FT,M	LC50	0.683	7.83	12.8	7.57	Thurston et al. 1981b
<u>Rainbow trout, <i>Salmo gairdneri</i></u>	5.7 g	NH <sub>4</sub> Cl	FT,M	LC50	0.704	7.79	12.9	7.37	Thurston et al. 1981b
<u>Rainbow trout, <i>Salmo gairdneri</i></u>	5.7 g	NH <sub>4</sub> Cl	FT,M	LC50	0.564	7.75	12.5	6.60	Thurston et al. 1981b
<u>Rainbow trout, <i>Salmo gairdneri</i></u>	5.0 g	NH <sub>4</sub> Cl	FT,M	LC50	0.610	7.76	12.5	6.57	Thurston et al. 1981b
<u>Rainbow trout, <i>Salmo gairdneri</i></u>	4.6 g	NH <sub>4</sub> Cl	FT,M	LC50	0.497	7.75	12.7	5.66	Thurston et al. 1981b
<u>Rainbow trout, <i>Salmo gairdneri</i></u>	3.2 g	NH <sub>4</sub> Cl	FT,M	LC50	0.643	7.75	13.0	5.47	Thurston et al. 1981b
<u>Rainbow trout, <i>Salmo gairdneri</i></u>	18.1 g	NH <sub>4</sub> Cl	FT,M	LC50	0.56 <sup>C</sup>	8.10-8.57	5.0	51-88% Saturated	Reinbold & Pescitelli 1982b

Table 1. (Continued)

<u>Species</u>	<u>Life Stage or Size</u>	<u>Chemical</u>	<u>Methods<sup>a</sup></u>	<u>Effect<sup>b</sup></u>	<u>Concentration (mg/L NH<sub>3</sub>)</u>	<u>pH</u>	<u>Temperature (°C)</u>	<u>D.O. (mg/L)</u>	<u>Reference</u>
<u>Rainbow trout, Salmo gairdneri</u>	20.6 g	NH <sub>4</sub> Cl	FT,M	LC50	0.79 <sup>c</sup>	8.02-8.55	12.8	47-85% Saturated	Reinbold & Pescitelli 1982b
<u>Rainbow trout, Salmo gairdneri</u>	0.61 g	NH <sub>4</sub> Cl	FT,M	LC50	0.40	8.30-8.56	3.0	86-100% Saturated	Reinbold & Pescitelli 1982b
<u>Rainbow trout, Salmo gairdneri</u>	0.86 g	NH <sub>4</sub> Cl	FT,M	LC50	1.02	8.03-8.29	14.2	76-93% Saturated	Reinbold & Pescitelli 1982b
<u>Rainbow trout, Salmo gairdneri</u>	0.76 g	NH <sub>4</sub> Cl	FT,M	LC50	0.77	8.45-8.76	3.3	74-95% Saturated	Reinbold & Pescitelli 1982b
<u>Rainbow trout, Salmo gairdneri</u>	1.47 g	NH <sub>4</sub> Cl	FT,M	LC50	0.97	8.32-8.69	14.9	74-87% Saturated	Reinbold & Pescitelli 1982b
<u>Rainbow trout, Salmo gairdneri</u>	10.9 g	NH <sub>4</sub> Cl	FT,M	LC50	0.26	7.7	3.6	12.4	West 1985
<u>Rainbow trout, Salmo gairdneri</u>	14.0 g	NH <sub>4</sub> Cl	FT,M	LC50	0.61	7.7	9.8	9.6	West 1985
<u>Rainbow trout, Salmo gairdneri</u>	10.3 g	NH <sub>4</sub> Cl	FT,M	LC50	0.59	7.9	11.3	8.7	West 1985
<u>Rainbow trout, Salmo gairdneri</u>	22.4 g	NH <sub>4</sub> Cl	FT,M	LC50	0.43	7.9	16.2	7.3	West 1985
<u>Rainbow trout, Salmo gairdneri</u>	3.3 g	NH <sub>4</sub> Cl	FT,M	LC50	1.04	8.3	18.7	7.3	West 1985
<u>Brown trout, Salmo trutta</u>	1.17 g	NH <sub>4</sub> Cl	FT,M	LC50	0.701	7.86	13.8	8.65	Thurston & Meyn 1984
<u>Brown trout, Salmo trutta</u>	0.91 g	NH <sub>4</sub> Cl	FT,M	LC50	0.677	7.82	14.2	8.99	Thurston & Meyn 1984
<u>Brown trout, Salmo trutta</u>	1.20 g	NH <sub>4</sub> Cl	FT,M	LC50	0.597	7.85	13.2	9.28	Thurston & Meyn 1984
<u>Brook trout, Salvelinus fontinalis</u>	3.40 g	NH <sub>4</sub> Cl	FT,M	LC50	1.05	7.94	10.6	8.48	Thurston & Meyn 1984
<u>Brook trout, Salvelinus fontinalis</u>	3.12 g	NH <sub>4</sub> Cl	FT,M	LC50	0.962	7.83	13.6	8.65	Thurston & Meyn 1984

Table 1. (Continued)

<u>Species</u>	<u>Life Stage or Size</u>	<u>Chemical</u>	<u>Methods<sup>a</sup></u>	<u>Effect<sup>b</sup></u>	<u>Concentration (mg/L NH<sub>3</sub>)</u>	<u>pH</u>	<u>Temperature (°C)</u>	<u>D.O. (mg/L)</u>	<u>Reference</u>
<u>Mountain whitefish, <i>Prosopium williamsoni</i></u>	56.9 g	NH <sub>4</sub> Cl	FT,M	LC50	0.473	7.84	12.4	7.74	Thurston & Meyn 1984
<u>Mountain whitefish, <i>Prosopium williamsoni</i></u>	63.0 g	NH <sub>4</sub> Cl	FT,M	LC50	0.358	7.80	12.3	7.69	Thurston & Meyn 1984
<u>Mountain whitefish, <i>Prosopium williamsoni</i></u>	177 g	NH <sub>4</sub> Cl	FT,M	LC50	0.143	7.68	12.1	6.19	Thurston & Meyn 1984
<u>Golden shiner, <i>Notemigonus crysoleucas</i></u>	8.7 g	NH <sub>4</sub> Cl	FT,M	LC50	0.72 <sup>c</sup>	7.5	24.5	7.7	Swigert & Spacie 1983
<u>Red shiner, <i>Notropis lutrensis</i></u>	0.43 g	NH <sub>4</sub> Cl	FT,M	LC50	2.83 <sup>c</sup>	8.2-8.4	24	7.6-8.2	Hazel et al. 1979
<u>Red shiner, <i>Notropis lutrensis</i></u>	0.40 g	NH <sub>4</sub> Cl	FT,M	LC50	3.16 <sup>c</sup>	9.0-9.2	24	7.5-8.0	Hazel et al. 1979
<u>Spotfin shiner, <i>Notropis spilopterus</i></u>	31-85 mm	NH <sub>4</sub> Cl	FT,M	LC50	1.20 <sup>c</sup>	7.7-8.2	26.5	81-89% Saturated	Rosage et al. 1979
<u>Spotfin shiner, <i>Notropis spilopterus</i></u>	41-78 mm	NH <sub>4</sub> Cl	FT,M	LC50	1.62 <sup>c</sup>	7.8-8.5	26.5	86-91% Saturated	Rosage et al. 1979
<u>Spotfin shiner, <i>Notropis spilopterus</i></u>	0.5 g	NH <sub>4</sub> Cl	FT,M	LC50	1.35 <sup>c</sup>	7.9	25.7	7.3	Swigert & Spacie 1983
<u>Steelcolor shiner, <i>Notropis whipplei</i></u>	0.5 g	NH <sub>4</sub> Cl	FT,M	LC50	1.25 <sup>c</sup>	7.9	25.7	7.3	Swigert & Spacie 1983
<u>Stoneroller, <i>Campostoma anonalum</i></u>	2.1 g	NH <sub>4</sub> Cl	FT,M	LC50	1.72 <sup>c</sup>	7.8	25.7	6.4	Swigert & Spacie 1983
<u>Fathead minnow, <i>Pimephales promelas</i></u>	-	NH <sub>4</sub> Cl	FT,M	LC50	1.59	8.0-8.1	14	7.2-7.4	DeGraeve et al. 1980
<u>Fathead minnow, <i>Pimephales promelas</i></u>	0.09 g	NH <sub>4</sub> Cl	FT,M	LC50	1.50	7.91	16.3	8.1	Thurston et al. 1983
<u>Fathead minnow, <i>Pimephales promelas</i></u>	0.09 g	NH <sub>4</sub> Cl	FT,M	LC50	1.10	7.89	13.1	8.7	Thurston et al. 1983

Table 1. (Continued)

<u>Species</u>	<u>Life Stage or Size</u>	<u>Chemical</u>	<u>Methods<sup>a</sup></u>	<u>Effect<sup>b</sup></u>	<u>Concentration (mg/L NH<sub>3</sub>)</u>	<u>pH</u>	<u>Temperature (°C)</u>	<u>D.O. (mg/L)</u>	<u>Reference</u>
<u>Fathead minnow, Pimephales promelas</u>	0.13 g	NH <sub>4</sub> Cl	FT,M	LC50	0.754	7.64	13.6	8.8	Thurston et al. 1983
<u>Fathead minnow, Pimephales promelas</u>	0.19 g	NH <sub>4</sub> Cl	FT,M	LC50	0.908	7.68	13.5	8.8	Thurston et al. 1983
<u>Fathead minnow, Pimephales promelas</u>	0.22 g	NH <sub>4</sub> Cl	FT,M	LC50	2.73	8.03	22.1	7.6	Thurston et al. 1983
<u>Fathead minnow, Pimephales promelas</u>	0.22 g	NH <sub>4</sub> Cl	FT,M	LC50	2.59	8.06	22.0	7.6	Thurston et al. 1983
<u>Fathead minnow, Pimephales promelas</u>	0.26 g	NH <sub>4</sub> Cl	FT,M	LC50	0.832	7.67	13.9	8.5	Thurston et al. 1983
<u>Fathead minnow, Pimephales promelas</u>	0.31 g	NH <sub>4</sub> Cl	FT,M	LC50	2.33	8.05	13.0	9.0	Thurston et al. 1983
<u>Fathead minnow, Pimephales promelas</u>	0.31 g	NH <sub>4</sub> Cl	FT,M	LC50	2.17	8.05	13.6	8.9	Thurston et al. 1983
<u>Fathead minnow, Pimephales promelas</u>	0.35 g	NH <sub>4</sub> Cl	FT,M	LC50	1.61	7.94	19.1	7.8	Thurston et al. 1983
<u>Fathead minnow, Pimephales promelas</u>	0.42 g	NH <sub>4</sub> Cl	FT,M	LC50	1.27	7.76	19.0	8.2	Thurston et al. 1983
<u>Fathead minnow, Pimephales promelas</u>	0.42 g	NH <sub>4</sub> Cl	FT,M	LC50	0.775	7.66	13.4	8.8	Thurston et al. 1983
<u>Fathead minnow, Pimephales promelas</u>	0.47 g	NH <sub>4</sub> Cl	FT,M	LC50	1.51	7.87	15.8	8.3	Thurston et al. 1983
<u>Fathead minnow, Pimephales promelas</u>	0.47 g	NH <sub>4</sub> Cl	FT,M	LC50	1.85	7.83	22.0	7.1	Thurston et al. 1983
<u>Fathead minnow, Pimephales promelas</u>	0.50 g	NH <sub>4</sub> Cl	FT,M	LC50	1.73	7.91	18.9	7.6	Thurston et al. 1983
<u>Fathead minnow, Pimephales promelas</u>	0.8 g	NH <sub>4</sub> Cl	FT,M	LC50	1.22	7.77	14.3	8.6	Thurston et al. 1983
<u>Fathead minnow, Pimephales promelas</u>	1.0 g	NH <sub>4</sub> Cl	FT,M	LC50	1.31	7.77	14.1	8.6	Thurston et al. 1983

Table 1. (Continued)

<u>Species</u>	<u>Life Stage or Size</u>	<u>Chemical</u>	<u>Methods<sup>a</sup></u>	<u>Effect<sup>b</sup></u>	<u>Concentration (mg/L NH<sub>3</sub>)</u>	<u>pH</u>	<u>Temperature (°C)</u>	<u>D.O. (mg/L)</u>	<u>Reference</u>
<u>Fathead minnow, Pimephales promelas</u>	1.4 g	NH <sub>4</sub> Cl	FT,M	LC50	2.16	8.04	22.4	6.7	Thurston et al. 1983
<u>Fathead minnow, Pimephales promelas</u>	1.4 g	NH <sub>4</sub> Cl	FT,M	LC50	2.73	8.08	21.4	6.8	Thurston et al. 1983
<u>Fathead minnow, Pimephales promelas</u>	1.4 g	NH <sub>4</sub> Cl	FT,M	LC50	3.44	8.16	21.4	6.8	Thurston et al. 1983
<u>Fathead minnow, Pimephales promelas</u>	1.4 g	NH <sub>4</sub> Cl	FT,M	LC50	2.04	7.88	21.7	6.3	Thurston et al. 1983
<u>Fathead minnow, Pimephales promelas</u>	1.4 g	NH <sub>4</sub> Cl	FT,M	LC50	1.23	7.68	12.9	8.9	Thurston et al. 1983
<u>Fathead minnow, Pimephales promelas</u>	1.4 g	NH <sub>4</sub> Cl	FT,M	LC50	1.10	7.63	13.2	8.7	Thurston et al. 1983
<u>Fathead minnow, Pimephales promelas</u>	1.5 g	NH <sub>4</sub> Cl	FT,M	LC50	1.73	7.76	12.9	8.8	Thurston et al. 1983
<u>Fathead minnow, Pimephales promelas</u>	1.7 g	NH <sub>4</sub> Cl	FT,M	LC50	2.03	7.84	21.7	6.2	Thurston et al. 1983
<u>Fathead minnow, Pimephales promelas</u>	2.1 g	NH <sub>4</sub> Cl	FT,M	LC50	1.09	7.76	13.1	9.0	Thurston et al. 1983
<u>Fathead minnow, Pimephales promelas</u>	2.2 g	NH <sub>4</sub> Cl	FT,M	LC50	0.796	7.74	12.8	9.0	Thurston et al. 1983
<u>Fathead minnow, Pimephales promelas</u>	2.3 g	NH <sub>4</sub> Cl	FT,M	LC50	1.34	7.91	15.9	8.0	Thurston et al. 1983
<u>Fathead minnow, Pimephales promelas</u>	1.8 g	NH <sub>4</sub> Cl	FT,M	LC50	0.240 <sup>c</sup>	6.51	13.0	9.3	Thurston et al. 1981 <sup>c</sup>
<u>Fathead minnow, Pimephales promelas</u>	2.0 g	NH <sub>4</sub> Cl	FT,M	LC50	0.452 <sup>c</sup>	7.01	13.8	9.6	Thurston et al. 1981 <sup>c</sup>
<u>Fathead minnow, Pimephales promelas</u>	2.0 g	NH <sub>4</sub> Cl	FT,M	LC50	1.08 <sup>c</sup>	7.82	12.0	9.9	Thurston et al. 1981 <sup>c</sup>
<u>Fathead minnow, Pimephales promelas</u>	1.8 g	NH <sub>4</sub> Cl	FT,M	LC50	0.793 <sup>c</sup>	7.83	11.8	9.6	Thurston et al. 1981 <sup>c</sup>

Table 1. (Continued)

<u>Species</u>	<u>Life Stage or Size</u>	<u>Chemical</u>	<u>Methods<sup>a</sup></u>	<u>Effect<sup>b</sup></u>	<u>Concentration (mg/L NH<sub>3</sub>)</u>	<u>pH</u>	<u>Temperature (°C)</u>	<u>D.O. (mg/L)</u>	<u>Reference</u>
<u>Fathead minnow, Pimephales promelas</u>	2.0 g	NH <sub>4</sub> Cl	FT,M	LC50	1.68 <sup>c</sup>	8.51	13.5	9.8	Thurston et al. 1981c <sup>i</sup>
<u>Fathead minnow, Pimephales promelas</u>	1.8 g	NH <sub>4</sub> Cl	FT,M	LC50	1.47 <sup>c</sup>	9.03	13.2	9.5	Thurston et al. 1981c <sup>i</sup>
<u>Fathead minnow, Pimephales promelas</u>	0.030 g	NH <sub>4</sub> Cl	FT,M	LC50	0.73 <sup>c</sup>	8.21-8.70	4.1	87-96% Saturated	Reinbold & Pescitelli 1982b
<u>Fathead minnow, Pimephales promelas</u>	0.032 g	NH <sub>4</sub> Cl	FT,M	LC50	1.24 <sup>c</sup>	7.86-8.18	23.9	73-79% Saturated	Reinbold & Pescitelli 1982b
<u>Fathead minnow, Pimephales promelas</u>	0.063 g	NH <sub>4</sub> Cl	FT,M	LC50	0.80 <sup>c</sup>	8.13-8.38	4.6	88-96% Saturated	Reinbold & Pescitelli 1982b
<u>Fathead minnow, Pimephales promelas</u>	0.066 g	NH <sub>4</sub> Cl	FT,M	LC50	1.65 <sup>c</sup>	8.01-8.32	25.2	73-79% Saturated	Reinbold & Pescitelli 1982b
<u>Fathead minnow, Pimephales promelas</u>	0.2 g	NH <sub>4</sub> Cl	FT,M	LC50	1.75 <sup>c</sup>	7.78	25.9	7.1	Swigert & Spacie 1983
<u>Fathead minnow, Pimephales promelas</u>	0.5 g	NH <sub>4</sub> Cl	FT,M	LC50	1.87 <sup>c</sup>	7.8	25.6	7.2	Swigert & Spacie 1983
<u>Fathead minnow, Pimephales promelas</u>	1.9 g	NH <sub>4</sub> Cl	FT,M	LC50	2.41	7.9	3.4	12.4	West 1985
<u>Fathead minnow, Pimephales promelas</u>	1.8 g	NH <sub>4</sub> Cl	FT,M	LC50	1.83	8.1	12.1	9.8	West 1985
<u>Fathead minnow, Pimephales promelas</u>	1.6 g	NH <sub>4</sub> Cl	FT,M	LC50	1.97	8.0	17.1	8.0	West 1985
<u>Fathead minnow, Pimephales promelas</u>	1.7 g	NH <sub>4</sub> Cl	FT,M	LC50	2.55	8.1	26.1	6.3	West 1985
<u>White sucker, Catostomus commersoni</u>	6.3 g	NH <sub>4</sub> Cl	FT,M	LC50	1.40 <sup>c</sup>	8.07-8.26	15.0	93% Saturated	Reinbold & Pescitelli 1982c
<u>White sucker, Catostomus commersoni</u>	6.3 g	NH <sub>4</sub> Cl	FT,M	LC50	1.35 <sup>c</sup>	8.00-8.28	15.4	88% Saturated	Reinbold & Pescitelli 1982c
<u>White sucker, Catostomus commersoni</u>	11.4 g	NH <sub>4</sub> Cl	FT,M	LC50	0.79 <sup>c</sup>	7.8	22.5	7.4	Swigert & Spacie 1983

Table 1. (Continued)

<u>Species</u>	<u>Life Stage or Size</u>	<u>Chemical</u>	<u>Methods<sup>a</sup></u>	<u>Effect<sup>b</sup></u>	<u>Concentration (mg/L NH<sub>3</sub>)</u>	<u>pH</u>	<u>Temperature (°C)</u>	<u>D.O. (mg/L)</u>	<u>Reference</u>
<u>White sucker, <i>Catostomus commersoni</i></u>	5.6 g	NH <sub>4</sub> Cl	FT,M	LC50	0.76	7.8	3.6	12.5	West 1985
<u>White sucker, <i>Catostomus commersoni</i></u>	5.2 g	NH <sub>4</sub> Cl	FT,M	LC50	1.87	8.1	11.3	9.4	West 1985
<u>White sucker, <i>Catostomus commersoni</i></u>	6.1 g	NH <sub>4</sub> Cl	FT,M	LC50	1.73	8.2	12.6	9.2	West 1985
<u>White sucker, <i>Catostomus commersoni</i></u>	9.6 g	NH <sub>4</sub> Cl	FT,M	LC50	2.22	8.2	15.3	9.7	West 1985
<u>Mountain sucker, <i>Catostomus platyrhynchus</i></u>	63.3 g	NH <sub>4</sub> Cl	FT,M	LC50	0.819	7.67	12.0	6.68	Thurston & Meyn 1984
<u>Mountain sucker, <i>Catostomus platyrhynchus</i></u>	47.8 g	NH <sub>4</sub> Cl	FT,M	LC50	0.708	7.73	11.7	7.45	Thurston & Meyn 1984
<u>Mountain sucker, <i>Catostomus platyrhynchus</i></u>	45.3 g	NH <sub>4</sub> Cl	FT,M	LC50	0.668	7.69	13.2	6.59	Thurston & Meyn 1984
<u>Channel catfish, <i>Ictalurus punctatus</i></u>	50-70 mm	NH <sub>4</sub> Cl	S,U	LC50	2.4	8.6-8.8	22	Near saturation	Colt & Tchobanoglous 1976
<u>Channel catfish, <i>Ictalurus punctatus</i></u>	50-76 mm	NH <sub>4</sub> Cl	S,U	LC50	2.9	8.6-8.8	26	Near saturation	Colt & Tchobanoglous 1976
<u>Channel catfish, <i>Ictalurus punctatus</i></u>	50-76 mm	NH <sub>4</sub> Cl	S,U	LC50	3.8	8.6-8.8	30	Near saturation	Colt & Tchobanoglous 1976
<u>Channel catfish, <i>Ictalurus punctatus</i></u>	20.3 g	NH <sub>4</sub> Cl	FT,M	LC50	1.95 <sup>c</sup>	8.34-8.44	28	7.6	Colt & Tchobanoglous 1978
<u>Channel catfish, <i>Ictalurus punctatus</i></u>	7.1-12.7 g	NH <sub>4</sub> Cl	FT,M	LC50	2.1 <sup>k</sup>	7.77-8.41	22	80-89% Saturated	Roseboom & Richey 1977
<u>Channel catfish, <i>Ictalurus punctatus</i></u>	4.5-8.3 g	NH <sub>4</sub> Cl	FT,M	LC50	4.2 <sup>k</sup>	7.91-8.25	28	80-90% Saturated	Roseboom & Richey 1977
<u>Channel catfish, <i>Ictalurus punctatus</i></u>	12.8 g	NH <sub>4</sub> Cl	FT,M	LC50	1.76 <sup>c</sup>	7.75-8.20	23.8	89% Saturated	Reinbold & Pescitelli 1982b
<u>Channel catfish, <i>Ictalurus punctatus</i></u>	12.8 g	NH <sub>4</sub> Cl	FT,M	LC50	1.75 <sup>c</sup>	7.77-8.12	23.8	88% Saturated	Reinbold & Pescitelli 1982d

Table 1. (Continued)

<u>Species</u>	<u>Life Stage or Size</u>	<u>Chemical</u>	<u>Methods<sup>a</sup></u>	<u>Effect<sup>b</sup></u>	<u>Concentration (mg/L NH<sub>3</sub>)</u>	<u>pH</u>	<u>Temperature (°C)</u>	<u>D.O. (mg/L)</u>	<u>Reference</u>
<u>Channel catfish, Ictalurus punctatus</u>	0.5 g	NH <sub>4</sub> Cl	FT,M	LC50	1.45 <sup>c</sup>	7.8	25.7	7.1	Swigert & Spacie 1983
<u>Channel catfish, Ictalurus punctatus</u>	5.8 g	NH <sub>4</sub> Cl	FT,M	LC50	0.50	8.0	3.5	12.7	West 1985
<u>Channel catfish, Ictalurus punctatus</u>	6.4 g	NH <sub>4</sub> Cl	FT,M	LC50	0.98	8.0	14.6	9.2	West 1985
<u>Channel catfish, Ictalurus punctatus</u>	3.6 g	NH <sub>4</sub> Cl	FT,M	LC50	1.91	8.1	17.0	8.1	West 1985
<u>Channel catfish, Ictalurus punctatus</u>	3.5 g	NH <sub>4</sub> Cl	FT,M	LC50	1.29	7.8	19.6	7.9	West 1985
<u>Channel catfish, Ictalurus punctatus</u>	7.4 g	NH <sub>4</sub> Cl	FT,M	LC50	2.26	8.0	26.0	4.5	West 1985
<u>Mosquitofish, Gambusia affinis</u>	Adult females	(NH <sub>4</sub> ) <sub>2</sub> CO <sub>3</sub>	S,U	LC50	2.4 <sup>d</sup>	7.9-8.5	17-22	-	Wallen et al. 1957
<u>Mosquitofish, Gambusia affinis</u>	Adult females	NH <sub>4</sub> Cl	S,U	LC50	3.2 <sup>d</sup>	7.4-8.1	17-21	-	Wallen et al. 1957
<u>Mosquitofish, Gambusia affinis</u>	Adult females	NH <sub>4</sub> OH	S,U	LC50	2.4 <sup>d</sup>	8.2-8.8	20-26	-	Wallen et al. 1957
<u>Mosquitofish, Gambusia affinis</u>	Adult females	NH <sub>4</sub> C <sub>2</sub> H <sub>3</sub> O <sub>2</sub>	S,U	LC50	2.6 <sup>d</sup>	7.6-8.4	23-25	-	Wallen et al. 1957
<u>Guppy, Poecilia reticulata</u>	8.0 mm	NH <sub>4</sub> Cl	S,M	LC50	1.47 <sup>c</sup>	6.95-7.50	25	6.8-8.2	Rubin & Elmaraghy 1976, 1977
<u>Guppy, Poecilia reticulata</u>	8.2 mm	NH <sub>4</sub> Cl	S,M	LC50	1.59 <sup>c</sup>	7.40-7.50	25	6.6-8.2	Rubin & Elmaraghy 1976, 1977
<u>Guppy, Poecilia reticulata</u>	8.7 mm	NH <sub>4</sub> Cl	S,M	LC50	1.45 <sup>c</sup>	7.40-7.50	25	7.1-8.2	Rubin & Elmaraghy 1976, 1977
<u>White perch, Morone americana</u>	76 mm	NH <sub>4</sub> Cl	S,M	LC50	0.15	6.0	16	-	Stevenson 1977
<u>White perch, Morone americana</u>	76 mm	NH <sub>4</sub> Cl	S,M	LC50	0.52	8.0	16	-	Stevenson 1977

Table 1. (Continued)

<u>Species</u>	<u>Life Stage or Size</u>	<u>Chemical</u>	<u>Methods<sup>a</sup></u>	<u>Effect<sup>b</sup></u>	<u>Concentration (mg/L NH<sub>3</sub>)</u>	<u>pH</u>	<u>Temperature (°C)</u>	<u>D.O. (mg/L)</u>	<u>Reference</u>
<u>Green sunfish, Lepomis cyanellus</u>	8.4 g	NH <sub>4</sub> Cl	FT,M	LC50	0.61 <sup>d</sup>	7.84	12.3	8.3	Jude 1973
<u>Green sunfish, Lepomis cyanellus</u>	9-d old	NH <sub>4</sub> Cl	FT,M	LC50	1.08 <sup>c</sup>	8.09-8.46	26.2	88% Saturated	Rainbold & Pescitelli 1982a
<u>Green sunfish, Lepomis cyanellus</u>	63.1 mg	NH <sub>4</sub> Cl	FT,M	LC50	0.59	6.6	22.4	8.1	McCormick et al. 1984
<u>Green sunfish, Lepomis cyanellus</u>	63.1 mg	NH <sub>4</sub> Cl	FT,M	LC50	1.29	7.2	22.4	8.1	McCormick et al. 1984
<u>Green sunfish, Lepomis cyanellus</u>	63.1 mg	NH <sub>4</sub> Cl	FT,M	LC50	1.64	7.7	22.4	8.1	McCormick et al. 1984
<u>Green sunfish, Lepomis cyanellus</u>	63.1 mg	NH <sub>4</sub> Cl	FT,M	LC50	2.11	8.7	22.4	8.1	McCormick et al. 1984
<u>Pumpkinseed, Lepomis gibbosus</u>	4.5 g	NH <sub>4</sub> Cl	FT,M	LC50	0.14 <sup>d</sup>	7.77	12.0	8.4	Jude 1973
<u>Pumpkinseed, Lepomis gibbosus</u>	16.7 g	NH <sub>4</sub> Cl	FT,M	LC50	0.78	7.77	14.5	8.37	Thurston 1981
<u>Pumpkinseed, Lepomis gibbosus</u>	18.0 g	NH <sub>4</sub> Cl	FT,M	LC50	0.86	7.77	14.0	8.36	Thurston 1981
<u>Pumpkinseed, Lepomis gibbosus</u>	18.9 g	NH <sub>4</sub> Cl	FT,M	LC50	0.61	7.71	15.7	7.16	Thurston 1981
<u>Bluegill, Lepomis macrochirus</u>	22.0-55.2 mm	NH <sub>4</sub> Cl	FT,M	LC50	0.89	7.96-8.26	18.5	9.1	Emery & Welch 1969
<u>Bluegill, Lepomis macrochirus</u>	41.0-67.1 mm	NH <sub>4</sub> Cl	FT,M	LC50	2.97	7.95-8.54	18.5	9.1	Emery & Welch 1969
<u>Bluegill, Lepomis macrochirus</u>	35.3-65.5 mm	NH <sub>4</sub> Cl	FT,M	LC50	2.57	8.50-9.00	18.5	9.1	Emery & Welch 1969
<u>Bluegill, Lepomis macrochirus</u>	0.072 g	NH <sub>4</sub> Cl	FT,M	LC50	0.55 <sup>k</sup>	8.01-8.13	22	95% Saturated	Roseboom & Richey 1977
<u>Bluegill, Lepomis macrochirus</u>	0.217 g	NH <sub>4</sub> Cl	FT,M	LC50	0.68 <sup>k</sup>	7.89-8.12	22	95% Saturated	Roseboom & Richey 1977

Table 1. (Continued)

<u>Species</u>	<u>Life Stage or Size</u>	<u>Chemical</u>	<u>Methods<sup>a</sup></u>	<u>Effect<sup>b</sup></u>	<u>Concentration (mg/L NH<sub>3</sub>)</u>	<u>pH</u>	<u>Temperature (°C)</u>	<u>D.O. (mg/L)</u>	<u>Reference</u>
<u>Bluegill, Lepomis macrochirus</u>	0.646 g	NH <sub>4</sub> Cl	FT,M	LC50	1.1 <sup>k</sup>	7.89-7.97	22	93% Saturated	Roseboom & Richey 1977
<u>Bluegill, Lepomis macrochirus</u>	0.342 g	NH <sub>4</sub> Cl	FT,M	LC50	1.8 <sup>k</sup>	8.12-8.28	28	91% Saturated	Roseboom & Richey 1977
<u>Bluegill, Lepomis macrochirus</u>	0.078 g	NH <sub>4</sub> Cl	FT,M	LC50	0.50 <sup>c</sup>	8.32-8.47	4.0	73-100% Saturated	Reinbold & Pescitelli 1982b
<u>Bluegill, Lepomis macrochirus</u>	0.111 g	NH <sub>4</sub> Cl	FT,M	LC50	1.98 <sup>c</sup>	7.98-8.25	25.0	74-83% Saturated	Reinbold & Pescitelli 1982b
<u>Bluegill, Lepomis macrochirus</u>	0.250 g	NH <sub>4</sub> Cl	FT,M	LC50	0.26 <sup>c</sup>	8.06-8.26	4.5	87-97% Saturated	Reinbold & Pescitelli 1982b
<u>Bluegill, Lepomis macrochirus</u>	0.267 g	NH <sub>4</sub> Cl	FT,M	LC50	1.35 <sup>c</sup>	7.98-8.20	24.8	74-89% Saturated	Reinbold & Pescitelli 1982b
<u>Bluegill, Lepomis macrochirus</u>	49.2 mg	NH <sub>4</sub> Cl	FT,M	LC50	0.94	7.60	21.7	7.89	Smith et al. 1983
<u>Bluegill, Lepomis macrochirus</u>	0.9 g	NH <sub>4</sub> Cl	FT,M	LC50	1.35 <sup>c</sup>	7.8	24.2	6.4	Swigert & Spacie 1983
<u>Bluegill, Lepomis macrochirus</u>	0.9 g	NH <sub>4</sub> Cl	FT,M	LC50	1.75 <sup>c</sup>	7.6	26.5	7.0	Swigert & Spacie 1983
<u>Bluegill, Lepomis macrochirus</u>	1.2 g	NH <sub>4</sub> Cl	FT,M	LC50	1.76 <sup>c</sup>	7.8	26.6	7.2	Swigert & Spacie 1983
<u>Smallmouth bass, Micropterus dolomieu</u>	265 mg	NH <sub>4</sub> Cl	FT,M	LC50	0.694	6.53	22.3	7.93	Broderius et al. 1985
<u>Smallmouth bass, Micropterus dolomieu</u>	265 mg	NH <sub>4</sub> Cl	FT,M	LC50	1.01	7.16	22.3	7.90	Broderius et al. 1985
<u>Smallmouth bass, Micropterus dolomieu</u>	265 mg	NH <sub>4</sub> Cl	FT,M	LC50	1.20	7.74	22.3	7.97	Broderius et al. 1985
<u>Smallmouth bass, Micropterus dolomieu</u>	265 mg	NH <sub>4</sub> Cl	FT,M	LC50	1.78	8.71	22.3	8.00	Broderius et al. 1985
<u>Largemouth bass, Micropterus salmoides</u>	2.0-6.3 g	NH <sub>4</sub> Cl	FT,M	LC50	1.0 <sup>k</sup>	7.82-8.11	22	85-94% Saturated	Roseboom & Richey 1977

Table 1. (Continued)

<u>Species</u>	<u>Life Stage or Size</u>	<u>Chemical</u>	<u>Methods<sup>a</sup></u>	<u>Effect<sup>b</sup></u>	<u>Concentration (mg/L NH<sub>3</sub>)</u>	<u>pH</u>	<u>Temperature (°C)</u>	<u>D.O. (mg/L)</u>	<u>Reference</u>
<u>Largemouth bass, <i>Micropterus salmoides</i></u>	0.09-0.32 g	NH <sub>4</sub> Cl	FT,M	LC50	1.7 <sup>k</sup>	7.98-8.10	28	83-88% Saturated	Roseboom & Richey 1977
<u>Orangethroat darter, <i>Etheostoma spectabile</i></u>	0.78 g	NH <sub>4</sub> Cl	FT,M	LC50	0.90 <sup>c</sup>	8.4	21	7.6-8.1	Hazel et al. 1979
<u>Orangethroat darter, <i>Etheostoma spectabile</i></u>	0.71 g	NH <sub>4</sub> Cl	FT,M	LC50	1.07 <sup>c</sup>	7.7-8.5	22	7.5-8.1	Hazel et al. 1979
<u>Walleye, <i>Stizostedion vitreum vitreum</i></u>	6-d old	NH <sub>4</sub> Cl	FT,M	LC50	0.85 <sup>c</sup>	7.84-8.31	18.2	97% Saturated	Reinbold & Pescitelli 1982a
<u>Walleye, <i>Stizostedion vitreum</i></u>	22.6 g	NH <sub>4</sub> Cl	FT,M	LC50	0.52	7.9	3.7	11.7	West 1985
<u>Walleye, <i>Stizostedion vitreum</i></u>	19.4 g	NH <sub>4</sub> Cl	FT,M	LC50	1.10	7.7	11.1	9.0	West 1985
<u>Walleye, <i>Stizostedion vitreum</i></u>	13.4	NH <sub>4</sub> Cl	FT,M	LC50	0.51	8.3	19.0	6.9	West 1985
<u>Mottled sculpin, <i>Cottus bairdi</i></u>	1.8 g	NH <sub>4</sub> Cl	FT,M	LC50	1.39	8.02	12.4	8.9	Thurston & Russo 1981
<u>SALTWATER SPECIES</u>									
<u>Sargassum shrimp, <i>Latreutes fucorum</i></u>	0.045 g	NH <sub>4</sub> Cl	S,M	LC50	0.936	8.07	23.4	6.7	Venkataramlak et al. 1981a
<u>Prawn, <i>Macrobrachium rosenbergii</i></u>	3-8 days old	NH <sub>4</sub> Cl	R,M	LC50	1.3 <sup>f</sup>	8.34	28	7.3	Armstrong et al. 1978
<u>Prawn, <i>Macrobrachium rosenbergii</i></u>	3-8 days old	NH <sub>4</sub> Cl	R,M	LC50	0.95 <sup>f</sup>	7.60	28	7.3	Armstrong et al. 1978
<u>Prawn, <i>Macrobrachium rosenbergii</i></u>	3-8 days old	NH <sub>4</sub> Cl	R,M	LC50	0.38 <sup>f</sup>	6.83	28	7.3	Armstrong et al. 1978

Table 1. (Continued)

<u>Species</u>	<u>Life Stage or Size</u>	<u>Chemical</u>	<u>Methods<sup>a</sup></u>	<u>Effect<sup>b</sup></u>	<u>Concentration (mg/L NH<sub>3</sub>)</u>	<u>pH</u>	<u>Temperature (°C)</u>	<u>D.O. (mg/L)</u>	<u>Reference</u>
<u>Eastern oyster, Crassostrea virginica</u>	46-62 mm	NH <sub>4</sub> Cl	S,M	LC50	24-37 <sup>d,e</sup>	7.70-8.23	20	7.0-8.2	Epifanio & Srna 1975
<u>Eastern oyster, Crassostrea virginica</u>	13-17 mm	NH <sub>4</sub> Cl	S,M	LC50	8.3-13 <sup>d,e</sup>	7.70-8.23	20	7.0-8.2	Epifanio & Srna 1975
<u>Quahog clam, Mercenaria mercenaria</u>	28-32 mm	NH <sub>4</sub> Cl	S,M	LC50	3.2-5.0 <sup>d,e</sup>	7.70-8.23	20	7.0-8.2	Epifanio & Srna 1975
<u>Quahog clam, Mercenaria mercenaria</u>	4.7-5.2 mm	NH <sub>4</sub> Cl	S,M	LC50	4.6-7.2 <sup>d,e</sup>	7.70-8.23	20	7.0-8.2	Epifanio & Srna 1975
<u>Copepod, Nitocra spinipes</u>	3-6 wk old	NH <sub>3</sub>	S,U	LC50	1.5 <sup>d</sup>	7.8	21	>5	Linden et al. 1979
<u>American lobster, Homarus americanus</u>	22-63 mg	NH <sub>4</sub> Cl	S,M	LC50	2.2 <sup>d</sup>	8.1	21.9	6.9	Dellstraty et al. 1977
<u>Red drum, Sciaenops ocellatus</u>	larva	(NH <sub>4</sub> ) <sub>2</sub> SO <sub>4</sub>	S,M	LC50	0.47	8.0-8.2	25-26	5.4-6.4	Holt and Arnold, 1983
<u>Striped mullet, Mugil cephalus</u>	0.4 g	NH <sub>4</sub> Cl	S,M	LC50	1.23	8.08	21.0	7.9	Venkataramlak, 1981a
<u>Striped mullet, Mugil cephalus</u>	0.7 g	NH <sub>4</sub> Cl	S,M	LC50	1.19	8.14	22.0	7.8	Venkataramlak, 1981a
<u>Striped mullet, Mugil cephalus</u>	1.8 g	NH <sub>4</sub> Cl	S,M	LC50	1.63	7.99	23.3	7.6	Venkataramlak, 1981a
<u>Striped mullet, Mugil cephalus</u>	10.0 g	NH <sub>4</sub> Cl	S,M	LC50	2.38	8.00	23.3	7.5	Venkataramlak, 1981a
<u>Planehead filefish, Monacanthus hispidus</u>	0.7 g	NH <sub>4</sub> Cl	S,M	LC50	0.690	8.07	23.4	6.7	Venkataramlak, 1981a

Table 1. (Continued)

- <sup>a</sup> FT = flow-through, S = static, R = renewal, M = measured, U = unmeasured.
- <sup>b</sup> Duration of exposure for Invertebrates either 48 h or 96 h; duration of exposure for fishes 96 h.
- <sup>c</sup> Recalculated from authors'  $\text{NH}_3\text{-N}$  values.
- <sup>d</sup> Recalculated from authors' total ammonia values.
- <sup>e</sup> pH data used in  $\text{NH}_3$  calculation obtained from: Epifanio, C. E., personal communication.
- <sup>f</sup> 96-h LC50 or EC50 estimated from authors' graph.
- <sup>g</sup> EC50: 50% of test animals motionless.
- <sup>h</sup> Dissolved oxygen data obtained from: Thurston, R.V., personal communication.
- <sup>i</sup> Dissolved oxygen and fish size data obtained from: Thurston, R.V., personal communication.
- <sup>j</sup> Information on test conditions obtained from: Parkhurst, B., personal communication.
- <sup>k</sup> Recalculated from authors'  $\text{NH}_3\text{-N}$  values with re-correction for percent  $\text{NH}_3$  per authors' text.

Table 2. Chronic Toxicity of Ammonia to Aquatic Animals

<u>Species</u>	<u>Method<sup>a</sup></u>	<u>pH</u>	<u>Temperature (°C)</u>	<u>D.O. (mg/L)</u>	<u>Limits (mg/L NH<sub>3</sub>)</u>	<u>Chronic Value (mg/L NH<sub>3</sub>)</u>	<u>Reference</u>
<u>FRESHWATER SPECIES</u>							
<u>Cladoceran, Ceriodaphnia acanthina</u>	LC	7.0- 7.5	24.0- 25.0	5.7- 6.4	0.199-0.463 <sup>g</sup>	0.304	Mount 1982
<u>Cladoceran, Daphnia magna</u>	LC	8.09	22.1	6.9	0.378-0.735	0.527	Russo et al. 1985
<u>Cladoceran, Daphnia magna</u>	LC	7.6	20.2	7.7	0.53-0.76	0.63	Russo et al. 1985
<u>Cladoceran, Daphnia magna</u>	LC	7.63- 8.16	17.8- 20.8	88-91% Saturated	0.96-1.6 <sup>b</sup>	1.2	Reinbold & Pescitelli 1982a
<u>Pink salmon, Oncorhynchus gorbuscha</u>	ELS	6.3- 6.5	4	-	0.0024-0.004	0.0031	Rice & Bailey 1980
<u>Pink salmon, Oncorhynchus gorbuscha</u>	ELS	6.3- 6.5	4	-	0.0012-0.0024	0.0017	Rice & Bailey 1980
<u>Pink salmon, Oncorhynchus gorbuscha</u>	ELS	6.3- 6.5	4	-	0.0012-0.0024	0.0017	Rice & Bailey 1980
<u>Rainbow trout, Salmo gairdneri</u>	ELS	7.4	14.5	>80% Saturated	0.010-0.025	0.016	Calamari et al. 1977 1981
<u>Rainbow trout, Salmo gairdneri</u>	LC	7.69- 7.72	9.3	7.3- 7.6	0.0221-0.0439	0.0311	Thurston et al. 1984b
<u>Rainbow trout, Salmo gairdneri</u>	ELS	7.4- 7.6	10- 12	>8	<0.06	<0.06 <sup>c</sup>	Burkhalter & Kaya 1977
<u>Rainbow trout, Salmo gairdneri</u>	ELS	7.4- 7.6	10- 12	>8	0.06-0.12	0.085	Burkhalter & Kaya 1977
<u>Atlantic salmon, Salmo salar</u>	ELS	6.7- 7.5	13	10	0.002-0.07 <sup>g</sup>	0.01	Samylin 1969
<u>Fathead minnow, Pimephales promelas</u>	LC	8.01	24.0	6.3	0.088-0.188	0.13	Thurston et al. (Submitted)
<u>Fathead minnow, Pimephales promelas</u>	LC	7.99	24.2	6.5	0.092-0.187	0.13	Thurston et al. (Submitted)

Table 2. (Continued)

<u>Species</u>	<u>Method<sup>a</sup></u>	<u>pH</u>	<u>Temperature (°C)</u>	<u>D.O. (mg/L)</u>	<u>Limits (mg/L NH<sub>3</sub>)</u>	<u>Chronic Value (mg/L NH<sub>3</sub>)</u>	<u>Reference</u>
<u>Fathead minnow, Pimephales promelas</u>	ELS	7.63- 8.13	22.7- 26.3	6.6- 7.8	0.15-0.34	0.22	Swigert & Spacie 1983
<u>White sucker, Catostomus commersoni</u>	ELS	8.01- 8.65	16.9- 20.5	68-74% Saturated	0.048-0.070 <sup>b</sup>	0.058	Reinbold & Pescitelli 1982a
<u>Channel catfish, Ictalurus punctatus</u>	ELS	7.6- 7.8	25.1- 25.3	5.1	0.073-0.146 <sup>b</sup>	0.103	Robinette 1976
<u>Channel catfish, Ictalurus punctatus</u>	J <sup>1</sup>	8.30- 8.44	27.8- 28.0	7.4- 7.8	<0.25 <sup>b</sup>	<0.25 <sup>c</sup>	Coit & Tchobanoglous 1978
<u>Channel catfish, Ictalurus punctatus</u>	J <sup>1</sup>	7.53- 8.37	24.8- 28.4	70-76% Saturated	0.205-0.392 <sup>b</sup>	0.283	Reinbold & Pescitelli 1982a
<u>Channel catfish, Ictalurus punctatus</u>	ELS	7.34- 7.95	23.5- 28.0	3.6- 6.7	0.13-0.24 <sup>b</sup>	0.18	Swigert & Spacie 1983
<u>Green sunfish, Lepomis cyanellus</u>	ELS	7.9	22	7.9	0.22-0.49	0.33	McCormick et al. 1984
<u>Bluegill, Lepomis macrochirus</u>	ELS	7.74	22	7.05	0.063-0.136	0.0926	Smith et al. 1984
<u>Smallmouth bass, Micropterus dolomieu</u>	ELS	6.60	22.5	7.69	0.0342-0.0558	0.0437	Broderius et al. 1985
<u>Smallmouth bass, Micropterus dolomieu</u>	ELS	7.25	22.2	7.68	0.120-0.182	0.148	Broderius et al. 1985
<u>Smallmouth bass, Micropterus dolomieu</u>	ELS	7.83	22.3	7.72	0.472-0.760	0.599	Broderius et al. 1985
<u>Smallmouth bass, Micropterus dolomieu</u>	ELS	8.68	22.2	7.78	0.433-0.865	0.612	Broderius et al. 1985

Acute-Chronic Ratio

<u>Species</u>	<u>Acute Value<sup>1</sup> (mg/L NH<sub>3</sub>)</u>	<u>Chronic Value (mg/L NH<sub>3</sub>)</u>	<u>Ratio</u>
<u>Cladoceran, Ceriodaphnia acanthina</u>	1.05	0.304	3.5

Table 2. (Continued)

<u>Species</u>	<u>Acute-Chronic Ratio</u>		<u>Ratio</u>
	<u>Acute Value<sup>l</sup></u> <u>(mg/L NH<sub>3</sub>)</u>	<u>Chronic Value</u> <u>(mg/L NH<sub>3</sub>)</u>	
<u>Cladoceran,</u> <u>Daphnia magna</u>	2.68	0.527	5.1
<u>Cladoceran,</u> <u>Daphnia magna</u>	0.87 <sup>o</sup>	0.63	1.4
<u>Cladoceran,</u> <u>Daphnia magna</u>	4.6	1.2	3.9
<u>Pink salmon,</u> <u>Oncorhynchus gorbuscha</u>	0.090 <sup>d</sup>	0.0017	53
<u>Pink salmon,</u> <u>Oncorhynchus gorbuscha</u>	0.090 <sup>d</sup>	0.0017	53
<u>Pink salmon,</u> <u>Oncorhynchus gorbuscha</u>	0.090 <sup>d</sup>	0.0031	29
<u>Rainbow trout,</u> <u>Salmo gairdneri</u>	0.422 <sup>f</sup>	0.0311	14
<u>Rainbow trout,</u> <u>Salmo gairdneri</u>	0.35 <sup>o</sup>	0.016	22 <sup>h</sup>
<u>Fathead minnow,</u> <u>Pimephales promelas</u>	2.54 <sup>f</sup>	0.13	20
<u>Fathead minnow,</u> <u>Pimephales promelas</u>	2.56 <sup>f</sup>	0.13	20
<u>Fathead minnow,</u> <u>Pimephales promelas</u>	1.75 <sup>o</sup>	0.22	8.0 <sup>h</sup>
<u>White sucker,</u> <u>Catostomus commersoni</u>	1.75 <sup>o</sup>	0.058	30
<u>Channel catfish,</u> <u>Ictalurus punctatus</u>	2.42	0.103	15 <sup>j</sup>
<u>Channel catfish,</u> <u>Ictalurus punctatus</u>	1.95	<0.25	8-34 <sup>k</sup>

Table 2. (Continued)

<u>Species</u>	<u>Acute-Chronic Ratio</u>		<u>Ratio</u>
	<u>Acute Value<sup>1</sup></u> <u>(mg/L NH<sub>3</sub>)</u>	<u>Chronic Value</u> <u>(mg/L NH<sub>3</sub>)</u>	
<u>Channel catfish,</u> <u>Ictalurus punctatus</u>	2.12 <sup>9</sup>	0.283	7.5
<u>Channel catfish,</u> <u>Ictalurus punctatus</u>	1.58	0.18	8.8
<u>Green sunfish,</u> <u>Lepomis cyanellus</u>	2.05	0.33	6.3
<u>Bluegill,</u> <u>Lepomis macrochirus</u>	1.08	0.0926	12
<u>Smallmouth bass,</u> <u>Micropterus dolomieu</u>	0.81	0.0437	19
<u>Smallmouth bass,</u> <u>Micropterus dolomieu</u>	1.14	0.148	7.7
<u>Smallmouth bass,</u> <u>Micropterus dolomieu</u>	1.30	0.599	2.2
<u>Smallmouth bass,</u> <u>Micropterus dolomieu</u>	1.77	0.612	2.9

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Geometric mean of acute-chronic ratios for *Daphnia magna* = 3.0  
 for pink salmon = 43  
 for fathead minnow = 20 (15 if ELS study included)  
 for smallmouth bass = 5.4 (3.6 for pH  $\geq$  7.25)  
 for channel catfish = 10  
 for rainbow trout = 14 (18 if ELS study included)

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**Table 2. (Continued)**

- <sup>a</sup> LC = life cycle, ELS = early life stage, J = juvenile.
- <sup>b</sup> Recalculated from author's  $\text{NH}_3\text{-N}$  values.
- <sup>c</sup> Lowest concentration tested, above control, affected growth ( $P < 0.05$ ).
- <sup>d</sup> Estimated from authors' graph.
- <sup>e</sup> Acute value geometric mean of acute tests in same waters as used for respective chronic tests.
- <sup>f</sup> Acute value geometric mean of acute tests with juveniles in same water as used for chronic test.
- <sup>g</sup> Recalculated from author's total ammonia values.
- <sup>h</sup> Value not used in criteria calculations because results are available from life cycle test with same species (see Guidelines).
- <sup>i</sup> Value corrected to pH of chronic value.
- <sup>j</sup> Acute value is for 24 hours, acute-chronic ratio multiplied by 0.65 = average ratio of 96-hour LC50 to 24-hour LC50 for several acute studies on channel catfish (range = 0.50 - 0.75).
- <sup>k</sup> Upper limit for ratio based on control concentration (0.06 mg/liter  $\text{NH}_3$ ).
- <sup>l</sup> Juvenile tests included because same or greater sensitivity shown as for embryo-larval ELS tests.

Table 3. Ranked Genus Mean Acute Values with Species Mean Acute/Chronic Ratios

<u>Rank<sup>a</sup></u>	<u>Reference Genus Mean Acute Value (mg/L NH<sub>3</sub>)<sup>b</sup></u>	<u>Species</u>	<u>Reference Species Mean Acute Value (mg/L NH<sub>3</sub>)<sup>b</sup></u>	<u>Species Mean Acute-Chronic Ratio</u>
<u>FRESHWATER SPECIES</u>				
34	11.40	<u>Caddisfly, Philarctus</u>	11.4	-
33	8.48	<u>Crayfish, Orconectes nalis</u>	3.15	-
		<u>Crayfish, Orconectes immunis</u>	22.8	-
32	8.00	<u>Beele, Stenelmis sexlineata</u>	8.00	-
31	5.25	<u>Mayfly, Ephemereilla grandis</u>	5.25	-
30	4.02	<u>Isopod, Asellus racovitzai</u>	4.02	-
29	3.18	<u>Mayfly, Callibaetis sp. near montanus</u>	2.00	-
		<u>Mayfly, Callibaetis skoklanus</u>	5.07	-
28	3.12	<u>Amphipod, Cragonyx pseudogracilis</u>	3.12	-
27	2.76	<u>Snail, Helisoma trivolvis</u>	2.76	-
26	2.70	<u>Tubificid worm, Tubifex tubifex</u>	2.70	-
25	2.48	<u>Mosquitofish, Gambusia affinis</u>	2.48	-
24	2.35	<u>Mottled sculpin, Cottus bairdi</u>	2.35	-
23	2.29	<u>Stonefly, Arcynopteryx parallela</u>	2.29	-

Table 3. (Continued)

Rank <sup>a</sup>	Reference Genus Mean Acute Value (mg/L NH <sub>3</sub> ) <sup>b</sup>	Species	Reference Species Mean Acute Value (mg/L NH <sub>3</sub> ) <sup>b</sup>	Species Mean Acute-Chronic Ratio
22	2.07	Fathead minnow, <u>Pimephales promelas</u>	2.07	20
21	1.96	Cladoceran, <u>Ceriodaphnia acanthina</u>	1.96	3.5
20	1.95	Snail, <u>Physa gyrina</u>	1.95	-
19	1.89	Cladoceran, <u>Simocephalus vetulus</u>	1.89	-
18	1.79	White sucker, <u>Catostomus commersoni</u>	2.15	30
		Mountain sucker, <u>Catostomus platyrhynchus</u>	1.49	-
17	1.69	Brook trout, <u>Salvelinus fontinalis</u>	1.69	-
16	1.68	White perch, <u>Morone americana</u>	1.68	-
15	1.63	Channel catfish, <u>Ictalurus punctatus</u>	1.63	7.5
14	1.49	Cladoceran, <u>Daphnia magna</u>	1.91	3.1
		Cladoceran, <u>Daphnia pulicaria</u>	1.16	-
13	1.48	Guppy, <u>Poecilia reticulata</u>	1.48	-

Table 3. (Continued)

<u>Rank<sup>a</sup></u>	<u>Reference Genus Mean Acute Value (mg/L NH<sub>3</sub>)<sup>b</sup></u>	<u>Species</u>	<u>Reference Species Mean Acute Value (mg/L NH<sub>3</sub>)<sup>b</sup></u>	<u>Species Mean Acute-Chronic Ratio<sup>c</sup></u>
12	1.40	Flatworm, <u>Dendrocoelum lacteum</u> ( <u>Procotyla fluviatilis</u> )	1.40	-
11	1.34	Smallmouth bass, <u>Micropterus dolomieu</u>	1.92	5.4
		Largemouth bass, <u>Micropterus salmoides</u>	0.93	-
10	1.30	Stoneroller, <u>Campostoma anomalum</u>	1.30	-
9	1.24	Pink salmon, <u>Oncorhynchus gorbuscha</u>	2.37	43
		Coho salmon, <u>Oncorhynchus kisutch</u>	1.02	-
		Chinook salmon, <u>Oncorhynchus tshawytscha</u>	0.80	-
8	1.23	Red shiner, <u>Notropis lutrensis</u>	2.27	-
		Spotfin shiner, <u>Notropis spilopterus</u>	0.92	-
		Steelcolor shiner, <u>Notropis whippiei</u>	0.89	-
7	1.16	Green sunfish, <u>Lepomis cyanellus</u>	1.57	6.3
		Pumpkinseed, <u>Lepomis gibbosus</u>	0.85	-
		Bluegill, <u>Lepomis macrochirus</u>	1.16	12

Table 3. (Continued)

<u>Rank<sup>a</sup></u>	<u>Reference Genus Mean Acute Value (mg/L NH<sub>3</sub>)<sup>b</sup></u>	<u>Species</u>	<u>Reference Species Mean Acute Value (mg/L NH<sub>3</sub>)<sup>b</sup></u>	<u>Species Mean Acute-Chronic Ratio</u>
6	1.10	Clam, <u>Musculium transversum</u>	1.10	-
5	1.10	Golden trout, <u>Salmo aguabonita</u>	1.21	-
		Cutthroat trout, <u>Salmo clarki</u>	1.20	-
		Rainbow trout, <u>Salmo gairdneri</u>	0.93	14
		Brown trout, <u>Salmo trutta</u>	1.10	-
4	1.07	Walleye, <u>Stizostedion vitreum vitreum</u>	1.07	-
3	0.88	Orangethroat darter, <u>Etheostoma spectabile</u>	0.88	-
2	0.76	Golden shiner, <u>Notemigonus crysoleucas</u>	0.76	-
1	0.56	Mountain whitefish, <u>Prosopium williamsoni</u>	0.56	-
<u>SALTWATER SPECIES</u>				
9	18.3	Eastern oyster, <u>Crassostrea virginica</u>	18.3	-
8	5.01	Quahog clam, <u>Mercenaria mercenaria</u>	5.01	-

Table 3. (Continued)

<u>Rank<sup>a</sup></u>	<u>Reference Genus Mean Acute Value (mg/L NH<sub>3</sub>)<sup>b</sup></u>	<u>Species</u>	<u>Reference Species Mean Acute Value (mg/L NH<sub>3</sub>)<sup>b</sup></u>	<u>Species Mean Acute-Chronic Ratio</u>
7	1.93	American lobster, <u>Homarus americanus</u>	2.20	-
6	1.56	Copepod, <u>Nitocra spinipes</u>	1.68	-
5	0.76	Prawn, <u>Macrobrachium rosenbergii</u>	1.32	-
4	1.31	Striped mullet, <u>Mugil cephalus</u>	1.31	-
3	2.13	Sargassum shrimp, <u>Latreutes fucorum</u>	0.94	-
2	0.55	Planehead filefish, <u>Monacanthus hispidus</u>	0.55	-
1	0.32	Red drum, <u>Sciaenops ocellatus</u>	0.32	-

<sup>a</sup> Ranked from least sensitive to most sensitive based on Genus Mean Acute Values.

<sup>b</sup> See text for discussion of reference conditions. Mid-range pH and temperature values used where given as a range in test results from Table 1.

Freshwater FAV<sub>ref</sub> = 0.70 mg/L NH<sub>3</sub> (calculated from GMAV<sub>ref</sub>'s).

Freshwater FAV<sub>ref</sub> = 0.52 mg/L NH<sub>3</sub> (lowered to protect rainbow trout - see text).

Table 4. Toxicity of Ammonia to Aquatic Plants

<u>Species</u>	<u>Chemical</u>	<u>pH</u>	<u>Temperature (°C)</u>	<u>Effect</u>	<u>Concentration (mg/L NH<sub>3</sub>)</u>	<u>Reference</u>
<u>FRESHWATER SPECIES</u>						
Alga, <u>Scenedesmus obliquus</u>	NH <sub>4</sub> Cl	8.8	30	EC50, oxygen evolution inhibition	11 <sup>a,b</sup>	Abellovich & Azov 1976
Alga, <u>Scenedesmus obliquus</u>	NH <sub>4</sub> Cl	7.9	30	10% reduction in CO <sub>2</sub> photoassimilation rate	5.1 <sup>a</sup>	Abellovich & Azov 1976
Alga, <u>Scenedesmus obliquus</u>	NH <sub>4</sub> Cl	9.0	30	88% reduction in CO <sub>2</sub> photoassimilation rate	38 <sup>a</sup>	Abellovich & Azov 1976
Alga, <u>Anacystis nidulans</u>	NH <sub>4</sub> Cl	7.0	30	10% reduction in CO <sub>2</sub> photoassimilation rate	0.68 <sup>a</sup>	Abellovich & Azov 1976
Alga, <u>Anacystis nidulans</u>	NH <sub>4</sub> Cl	9.0	30	77% reduction in CO <sub>2</sub> photoassimilation rate	38 <sup>a</sup>	Abellovich & Azov 1976
Alga, <u>Plectonema boryanum</u>	NH <sub>4</sub> Cl	7.0	30	16% reduction in CO <sub>2</sub> photoassimilation rate	0.68 <sup>a</sup>	Abellovich & Azov 1976
Alga, <u>Plectonema boryanum</u>	NH <sub>4</sub> Cl	9.0	30	92% reduction in CO <sub>2</sub> photoassimilation rate	38 <sup>a</sup>	Abellovich & Azov 1976
Alga, <u>Chlorella pyrenoidosa</u>	NH <sub>4</sub> Cl	7.0	30	11% reduction in CO <sub>2</sub> photoassimilation rate	0.68 <sup>a</sup>	Abellovich & Azov 1976
Alga, <u>Chlorella pyrenoidosa</u>	NH <sub>4</sub> Cl	9.0	30	79% reduction in CO <sub>2</sub> photoassimilation rate	38 <sup>a</sup>	Abellovich & Azov 1976
Alga, <u>Chlorella vulgaris</u>	(NH <sub>4</sub> ) <sub>2</sub> CO <sub>3</sub>	7.0	26	LC50	9.8 <sup>a</sup>	Przytocka-Juslak 1976
Alga, <u>Chlorella vulgaris</u>	(NH <sub>4</sub> ) <sub>2</sub> CO <sub>3</sub>	7.0	26	EC50, growth inhibition	2.4 <sup>a</sup>	Przytocka-Juslak 1976

Table 4. (Continued)

<u>Species</u>	<u>Chemical</u>	<u>pH</u>	<u>Temperature (°C)</u>	<u>Effect</u>	<u>Concentration (mg/L NH<sub>3</sub>)</u>	<u>Reference</u>
<u>SALTWATER SPECIES</u>						
Diatom, <u>Navicula arenaria</u>	NH <sub>4</sub> Cl	8.0	12	25% reduction in chlorophyll <u>a</u>	0.24 <sup>a</sup>	Admiraal 1977
Diatom, <u>Nitzschia c.f. dissipata</u>	NH <sub>4</sub> Cl	8.0	12	62% reduction in chlorophyll <u>a</u>	0.24 <sup>a</sup>	Admiraal 1977
Diatom, <u>Nitzschia dubiformis</u>	NH <sub>4</sub> Cl	8.0	12	73% reduction in chlorophyll <u>a</u>	0.24 <sup>a</sup>	Admiraal 1977
Diatom, <u>Nitzschia closterium</u>	NH <sub>4</sub> Cl	8.0	12	77% reduction in chlorophyll <u>a</u>	1.2 <sup>a</sup>	Admiraal 1977
Diatom, <u>Amphiprora c.f. paludosa</u>	NH <sub>4</sub> Cl	8.0	12	46% reduction in chlorophyll <u>a</u>	0.24 <sup>a</sup>	Admiraal 1977
Diatom, <u>Stauroneis constricta</u>	NH <sub>4</sub> Cl	8.0	12	33% reduction in chlorophyll <u>a</u>	0.24 <sup>a</sup>	Admiraal 1977
Diatom, <u>Navicula cryptocephala</u>	NH <sub>4</sub> Cl	8.0	12	14% reduction in chlorophyll <u>a</u>	0.24 <sup>a</sup>	Admiraal 1977
Diatom, <u>Navicula salinarum</u>	NH <sub>4</sub> Cl	8.0	12	18% reduction in chlorophyll <u>a</u>	0.24 <sup>a</sup>	Admiraal 1977
Diatom, <u>Gyrosigma spencerii</u>	NH <sub>4</sub> Cl	8.0	12	66% reduction in chlorophyll <u>a</u>	0.24 <sup>a</sup>	Admiraal 1977
Diatom, <u>Nitzschia sigma</u>	NH <sub>4</sub> Cl	8.0	12	66% reduction in chlorophyll <u>a</u>	0.24 <sup>a</sup>	Admiraal 1977

<sup>a</sup> Recalculated from authors' total ammonia values.

<sup>b</sup> Estimated from authors' graph.

Table 5. Other Data on Effects of Ammonia on Aquatic Organisms

<u>Species</u>	<u>Chemical</u>	<u>pH</u>	<u>Temperature (°C)</u>	<u>Duration</u>	<u>Effect</u>	<u>Concentration (mg/L NH<sub>3</sub>)</u>	<u>Reference</u>
<u>FRESHWATER SPECIES</u>							
<u>Rotifer, Philodina acuticornis</u>	NH <sub>4</sub> Cl	7.4- 7.9	20	96 h	EC50 (No re- sponse to light)	2.9-9.1 <sup>a</sup>	Bulkema et al. 1974
<u>Mussel, Elliptio complanata</u>	NH <sub>4</sub> Cl	7.5	18	<1 h	50% reduction in ciliary beating rate	0.073 <sup>b</sup>	Anderson et al. 1978
<u>Mussel, Elliptio complanata</u>	NH <sub>4</sub> Cl	7.5	18	<1 h	90% reduction in ciliary beating rate	0.11 <sup>b</sup>	Anderson et al. 1978
<u>Mussel, Elliptio complanata</u>	NH <sub>4</sub> Cl	7.5	18	<1 h	Complete in- hibition of cilia	0.11-0.12 <sup>b</sup>	Anderson et al. 1978
<u>Mussel, Amblema p. plicata</u>	NH <sub>4</sub> HCO <sub>3</sub>	7.8- 8.2	24- 26	165 h	33% Mortality	0.32 <sup>b</sup>	Horne & McIntosh 1979
<u>Mussel, Anodonta imbecillis</u>	NH <sub>4</sub> HCO <sub>3</sub>	7.8- 8.2	24- 26	165 h	56% Mortality	0.32 <sup>b</sup>	Horne & McIntosh 1979
<u>Mussel, Cyrtonaias tampicoensis</u>	NH <sub>4</sub> HCO <sub>3</sub>	7.8- 8.2	24- 26	165 h	70% Mortality	0.32 <sup>b</sup>	Horne & McIntosh 1979
<u>Mussel, Toxolasma texasensis</u>	NH <sub>4</sub> HCO <sub>3</sub>	7.8- 8.2	24- 26	165 h	80% Mortality	0.32 <sup>b</sup>	Horne & McIntosh 1979
<u>Asiatic clam, Corbicula manilensis</u>	NH <sub>4</sub> Cl	7.5	18	<1 h	50% reduction in ciliary beating rate	0.061 <sup>b</sup>	Anderson et al. 1978
<u>Asiatic clam, Corbicula manilensis</u>	NH <sub>4</sub> Cl	7.5	18	<1 h	90% reduction in ciliary beating rate	0.073 <sup>b</sup>	Anderson et al. 1978
<u>Asiatic clam, Corbicula manilensis</u>	NH <sub>4</sub> Cl	7.5	18	<1 h	Complete in- hibition of cilia	0.11-0.12 <sup>b</sup>	Anderson et al. 1978
<u>Asiatic clam, Corbicula manilensis</u>	NH <sub>4</sub> HCO <sub>3</sub>	7.8- 8.2	24- 26	165 h	62% Mortality	0.32 <sup>b</sup>	Horne & McIntosh 1979

Table 5. (Continued)

<u>Species</u>	<u>Chemical</u>	<u>pH</u>	<u>Temperature (°C)</u>	<u>Duration</u>	<u>Effect</u>	<u>Concentration (mg/L NH<sub>3</sub>)</u>	<u>Reference</u>
<u>Fingernail clam, Musculium transversum</u>	NH <sub>4</sub> Cl	8.09- 8.20	23.5	42 d	67% Mortality <sup>c</sup>	0.72 <sup>b</sup>	Anderson et al. 1978
<u>Fingernail clam, Musculium transversum</u>	NH <sub>4</sub> Cl	8.08- 8.18	22.9	42 d	72% Mortality <sup>c</sup>	0.73 <sup>b</sup>	Anderson et al. 1978
<u>Fingernail clam, Musculium transversum</u>	NH <sub>4</sub> Cl	8.08- 8.18	22.9	14 d	Reduction in growth	0.41 <sup>b</sup>	Anderson et al. 1978
<u>Fingernail clam, Musculium transversum</u>	NH <sub>4</sub> Cl	7.5	18	<1 h	50% reduction in ciliary beating rate of >5 mm clams	0.036 <sup>b</sup>	Anderson et al. 1978
<u>Fingernail clam, Musculium transversum</u>	NH <sub>4</sub> Cl	7.5	18	<1 h	50% reduction in ciliary beating rate of <5 mm clams	0.073- 0.085 <sup>b</sup>	Anderson et al. 1978
<u>Fingernail clam, Musculium transversum</u>	NH <sub>4</sub> Cl	7.5	18	<1 h	90% reduction in ciliary beating rate of >5 mm clams	0.049 <sup>b</sup>	Anderson et al. 1978
<u>Fingernail clam, Musculium transversum</u>	NH <sub>4</sub> Cl	7.5	18	<1 h	90% reduction in ciliary beating rate of <5 mm clams	0.097 <sup>b</sup>	Anderson et al. 1978
<u>Fingernail clam, Musculium transversum</u>	NH <sub>4</sub> Cl	7.5	18	<1 h	Complete in- hibition of cilia of >5 mm clams	0.061- 0.073 <sup>b</sup>	Anderson et al. 1978
<u>Fingernail clam, Musculium transversum</u>	NH <sub>4</sub> Cl	7.5	18	<1 h	Complete in- hibition of cilia of <5 mm clams	0.097- 0.11 <sup>b</sup>	Anderson et al. 1978
<u>Fingernail clam, Musculium transversum</u>	NH <sub>4</sub> Cl	7.75- 7.85	21.7- 21.9	6 wk	47% Mortality	0.073 <sup>b</sup>	Sparks & Sandusky 1981
<u>Fingernail clam, Musculium transversum</u>	NH <sub>4</sub> Cl	7.75- 7.85	21.7- 21.9	4 wk	36% Mortality	0.23 <sup>b</sup>	Sparks & Sandusky 1981

Table 5. (Continued)

<u>Species</u>	<u>Chemical</u>	<u>pH</u>	<u>Temperature (°C)</u>	<u>Duration</u>	<u>Effect</u>	<u>Concentration (mg/L NH<sub>3</sub>)</u>	<u>Reference</u>
<u>Fingernail clam, Musculium transversum</u>	NH <sub>4</sub> Cl	7.75- 7.85	21.7- 21.9	4 wk	23% Mortality	0.63 <sup>b</sup>	Sparks & Sandusky 1981
<u>Fingernail clam, Musculium transversum</u>	NH <sub>4</sub> Cl	7.75- 7.85	21.7- 21.9	6 wk	Complete growth Inhibi- tion	0.036 <sup>b</sup>	Sparks & Sandusky 1981
<u>Cladoceran, Daphnia magna</u>	NH <sub>4</sub> Cl	8.2- 8.4	25	64 h	Threshold value	2.4-3.6	Anderson 1948
<u>Cladoceran, Daphnia magna</u>	(NH <sub>4</sub> ) <sub>2</sub> SO <sub>4</sub>	8	19	2 d	Minimum lethal concentration	0.55	Măiăcea 1966
<u>Cladoceran, Daphnia magna</u>	NH <sub>4</sub> Cl	7.9	22	50 h	LC50	2.0 <sup>a,f</sup>	Dowden & Bennett 1965
<u>Cladoceran, Daphnia magna</u>	NH <sub>4</sub> Cl	8.09	22.1	28 d	LC50	1.53	Russo et al. (In prep.)
<u>Cladoceran, Moina rectirostris</u>	-	8.3	25	24 h	LC50	1.50	Györe & Oláh 1980
<u>Daphnia (sp. not specified)</u>	NH <sub>4</sub> Cl	6.0	25	24 h	LC50	0.17 <sup>a,c</sup>	Tabata 1962
<u>Daphnia (sp. not specified)</u>	NH <sub>4</sub> Cl	7.0	25	24 h	LC50	1.4 <sup>a,c</sup>	Tabata 1962
<u>Daphnia (sp. not specified)</u>	NH <sub>4</sub> Cl	8.0	25	24 h	LC50	5.1 <sup>a,c</sup>	Tabata 1962
<u>Mayfly, Ephemera daddsi</u>	NH <sub>4</sub> Cl	7.85	12.0	96 h	60% Survival	6.20	Thurston et al. 1984a
<u>Mayfly, Ephemera daddsi</u>	NH <sub>4</sub> Cl	7.91	13.6	96 h	80% Survival	5.46	Thurston et al. 1984a
<u>Mayfly, Ephemera daddsi</u>	NH <sub>4</sub> Cl	7.83	10.7	96 h	100% Survival	2.64	Thurston et al. 1984a
<u>Mayfly, Ephemera daddsi</u>	NH <sub>4</sub> Cl	7.91	11.0	96 h	90% Survival	2.20	Thurston et al. 1984a

Table 5. (Continued)

<u>Species</u>	<u>Chemical</u>	<u>pH</u>	<u>Temperature (°C)</u>	<u>Duration</u>	<u>Effect</u>	<u>Concentration (mg/L NH<sub>3</sub>)</u>	<u>Reference</u>
<u>Mayfly, EphemereUa grandis</u>	NH <sub>4</sub> Cl	8.06	13.5	96 h	90% Survival	4.66	Thurston et al. 1984a
<u>Mayfly, EphemereUa grandis</u>	NH <sub>4</sub> Cl	7.83	10.7	96 h	60% Survival	2.64	Thurston et al. 1984a
<u>Mayfly, EphemereUa grandis</u>	NH <sub>4</sub> Cl	7.91	11.0	96 h	60% Survival	2.20	Thurston et al. 1984a
<u>Mayfly, EphemereUa grandis</u>	NH <sub>4</sub> Cl	7.68	10.5	96 h	80% Survival	1.54	Thurston et al. 1984a
<u>Mayfly, EphemereUa sp. near excrucians</u>	NH <sub>4</sub> Cl	8.53	20	24 h + 72 h recovery	EC50 (Mortality at 96 h after 24-h exposure)	4.7 <sup>b</sup>	Gall 1980
<u>Stonefly, Pteronarcella badia</u>	NH <sub>4</sub> Cl	7.66- 7.91	10.7- 13.3	96 h	100% Survival	1.35-7.49	Thurston et al. 1984a
<u>Stonefly, Pteronarcella badia</u>	NH <sub>4</sub> Cl	8.04	12.1	30 d	LC50	4.57	Thurston et al. 1984a
<u>Stonefly, Pteronarcella badia</u>	NH <sub>4</sub> Cl	8.04	12.1	30 d	Inhibition of emergence	3.7	Thurston et al. 1984a
<u>Stonefly, Pteronarcella badia</u>	NH <sub>4</sub> Cl	7.81	13.2	24 d	LC50	1.45	Thurston et al. 1984a
<u>Stonefly, Pteronarcella badia</u>	NH <sub>4</sub> Cl	7.81	13.2	24 d	Inhibition of emergence	3.4	Thurston et al. 1984a
<u>Stonefly, Arcynopteryx parallela</u>	NH <sub>4</sub> Cl	7.88	13.3	96 h	100% Survival	7.49	Thurston et al. 1984a
<u>Stonefly, Arcynopteryx parallela</u>	NH <sub>4</sub> Cl	7.84	12.8	96 h	80% Survival	6.24	Thurston et al. 1984a
<u>Stonefly, Arcynopteryx parallela</u>	NH <sub>4</sub> Cl	7.95	13.3	96 h	90% Survival	4.05	Thurston et al. 1984a
<u>Stonefly, Arcynopteryx parallela</u>	NH <sub>4</sub> Cl	7.88	13.6	96 h	60% Survival	3.03	Thurston et al. 1984a

Table 5. (Continued)

<u>Species</u>	<u>Chemical</u>	<u>pH</u>	<u>Temperature (°C)</u>	<u>Duration</u>	<u>Effect</u>	<u>Concentration (mg/L NH<sub>3</sub>)</u>	<u>Reference</u>
<u>Caddisfly, Arctopsyche grandis</u>	NH <sub>4</sub> Cl	7.88	13.3	96 h	90% Survival	7.49	Thurston et al. 1984a
<u>Caddisfly, Arctopsyche grandis</u>	NH <sub>4</sub> Cl	7.92	13.8	96 h	80% Survival	4.19	Thurston et al. 1984a
<u>Pink salmon, Oncorhynchus gorbuscha</u>	(NH <sub>4</sub> ) <sub>2</sub> SO <sub>4</sub>	6.3- 6.5	3.7 4.8	96 h	No harm to eyed embryos	>1.5	Rice & Bailey 1980
<u>Coho salmon, Oncorhynchus kisutch</u>	NH <sub>4</sub> OH	8.0	14.2	72 h	critical level	0.13 <sup>a</sup>	Holland et al. 1960
<u>Sockeye salmon, Oncorhynchus nerka</u>	NH <sub>4</sub> Cl	8.42	10	62 d	30% Mortality	0.12 <sup>b</sup>	Rankin 1979
<u>Sockeye salmon, Oncorhynchus nerka</u>	NH <sub>4</sub> Cl	8.45	10	62 d	100% Mortality	0.49 <sup>b</sup>	Rankin 1979
<u>Chinook salmon, Oncorhynchus tshawytscha</u>	NH <sub>4</sub> OH	7.6	15.3	72 h	critical level	0.04- 0.11 <sup>a</sup>	Holland et al. 1960
<u>Chinook salmon, Oncorhynchus tshawytscha</u>	NH <sub>4</sub> Cl	7.59- 7.90	11.7	24 h	LC50	0.36	Harader and Allen 1983
<u>Cutthroat trout, Salmo clarki</u>	NH <sub>4</sub> Cl	7.81	13.1	36 d	LC50	0.56	Thurston et al. 1978
<u>Cutthroat trout, Salmo clarki</u>	NH <sub>4</sub> Cl	7.80	12.8	36 d	LC50	0.56	Thurston et al. 1978
<u>Cutthroat trout, Salmo clarki</u>	NH <sub>4</sub> Cl	7.80	12.4	29 d	LC50	0.37	Thurston et al. 1978
<u>Cutthroat trout, Salmo clarki</u>	NH <sub>4</sub> Cl	7.78	12.2	29 d	LC50	0.34	Thurston et al. 1978
<u>Rainbow trout, Salmo gairdneri</u>	(NH <sub>4</sub> ) <sub>2</sub> SO <sub>4</sub>	7.55	14	360 min	time to death	0.32 <sup>h</sup>	Muhrmann & Woker 1948
<u>Rainbow trout, Salmo gairdneri</u>	NH <sub>4</sub> OH	9.42	13.5	3.5 h	activity ceased	29.6 <sup>a</sup>	Corti 1951
<u>Rainbow trout, Salmo gairdneri</u>	NH <sub>4</sub> Cl	7.2	15.2	1000 min	median survival time	0.18 <sup>b</sup>	Allan et al. 1958

Table 5. (Continued)

<u>Species</u>	<u>Chemical</u>	<u>pH</u>	<u>Temperature (°C)</u>	<u>Duration</u>	<u>Effect</u>	<u>Concentration (mg/L NH<sub>3</sub>)</u>	<u>Reference</u>
<u>Rainbow trout, Salmo gairdneri</u>	NH <sub>4</sub> Cl	7.80	17.5	48 h	LC50	0.60 <sup>a</sup>	Herbert 1961
<u>Rainbow trout Salmo gairdneri</u>	NH <sub>4</sub> Cl	-	17.5	3 h	LC50	0.49 <sup>b</sup>	Herbert 1962
<u>Rainbow trout, Salmo gairdneri</u>	Urea	8.55	16-18	1.28 h	time to death	4.1	Nehring 1962-63
<u>Rainbow trout, Salmo gairdneri</u>	Urea	8.1	16-18	3.05 h	time to death	0.7	Nehring 1962-63
<u>Rainbow trout, Salmo gairdneri</u>	NH <sub>4</sub> Cl	7.8	17.5	48 h	LC50	0.61 <sup>a</sup>	Herbert & Shurben 1964
<u>Rainbow trout, Salmo gairdneri</u>	NH <sub>4</sub> Cl	7.8	17.0	48 h	LC50	0.63 <sup>a</sup>	Herbert & Vandyke 1964
<u>Rainbow trout, Salmo gairdneri</u>	Manure leachate	-	15	8-60 min	time to death	0.4-4.0	Danecker 1964
<u>Rainbow trout, Salmo gairdneri</u>	Manure leachate	-	15-17	"Unlimited"	no observed effect	<0.2	Danecker 1964
<u>Rainbow trout, Salmo gairdneri</u>	NH <sub>4</sub> Cl	7.81	13.6	24 h	LC50	0.70 <sup>a</sup>	Herbert & Shurben 1965
<u>Rainbow trout, Salmo gairdneri</u>	NH <sub>4</sub> Cl	7.86- 8.22	10.5- 11.6	5 d	LC50	0.50 <sup>b</sup>	Ball 1967
<u>Rainbow trout, Salmo gairdneri</u>	NH <sub>4</sub> Cl	6.9	18	27.3 min	time to overturning	2.71 <sup>a</sup>	Grindley 1946
<u>Rainbow trout, Salmo gairdneri</u>	NH <sub>4</sub> Cl	7.7	18	>1000 min	time to overturning	0.85 <sup>a</sup>	Grindley 1946
<u>Rainbow trout, Salmo gairdneri</u>	(NH <sub>4</sub> ) <sub>2</sub> SO <sub>4</sub>	7.1	18	29.8 min	time to overturning	4.3 <sup>a</sup>	Grindley 1946
<u>Rainbow trout, Salmo gairdneri</u>	(NH <sub>4</sub> ) <sub>2</sub> SO <sub>4</sub>	8.1	10.5	1-4 d	Diuresis	0.081	Lloyd & Orr 1969

Table 5. (Continued)

<u>Species</u>	<u>Chemical</u>	<u>pH</u>	<u>Temperature (°C)</u>	<u>Duration</u>	<u>Effect</u>	<u>Concentration (mg/L NH<sub>3</sub>)</u>	<u>Reference</u>
<u>Rainbow trout, Salmo gairdneri</u>	Endogenous NH <sub>3</sub> -N	7.75	10	12 mon	Histopatho- logical effects with juveniles	0.0155	Smith & Piper 1975
<u>Rainbow trout, Salmo gairdneri</u>	(NH <sub>4</sub> ) <sub>2</sub> SO <sub>4</sub>	8.3	10	24 h	LC50	0.068	Rice & Stokes 1975
<u>Rainbow trout, Salmo gairdneri</u>	-	7.6	15	36 d	81% Mortality	0.30-0.36 <sup>b</sup>	Smart 1976
<u>Rainbow trout (embryo), Salmo gairdneri</u>	NH <sub>4</sub> Cl	7.4	14.5	96 h	LC50	>0.486	Calamari et al. 1977, 1981
<u>Rainbow trout, Salmo gairdneri</u>	NH <sub>4</sub> Cl	7.4	14.5	72 d	LC50	0.056	Calamari et al. 1977, 1981
<u>Rainbow trout, Salmo gairdneri</u>	NH <sub>4</sub> Cl	7.4- 7.6	10-12	21 d	LC50	0.30 <sup>b</sup>	Burkhalter & Kaya 1977
<u>Rainbow trout, Salmo gairdneri</u>	NH <sub>4</sub> Cl	7.95	10	30 d	Reduced growth	≥0.10	Broderius & Smith 1979
<u>Rainbow trout, Salmo gairdneri</u>	NH <sub>4</sub> Cl	7.85	13.1	12 d	LC50	0.490	Thurston & Russo 1983
<u>Rainbow trout, Salmo gairdneri</u>	NH <sub>4</sub> Cl	7.90	11.9	12 d	LC50	0.464	Thurston & Russo 1983
<u>Rainbow trout, Salmo gairdneri</u>	NH <sub>4</sub> Cl	7.9	13	12 d	LC50	0.684	Thurston & Russo 1983
<u>Rainbow trout, Salmo gairdneri</u>	NH <sub>4</sub> Cl	7.66	9.8	12 d	LC50	0.262	Thurston & Russo 1983
<u>Rainbow trout, Salmo gairdneri</u>	NH <sub>4</sub> Cl	7.64	10.0	12 d	LC50	0.312	Thurston & Russo 1983
<u>Rainbow trout, Salmo gairdneri</u>	NH <sub>4</sub> Cl	7.81	13.0	35 d	LC50	0.483	Thurston & Russo 1983
<u>Rainbow trout, Salmo gairdneri</u>	NH <sub>4</sub> Cl	7.89	12.6	35 d	LC50	0.598	Thurston & Russo 1983

Table 5. (Continued)

<u>Species</u>	<u>Chemical</u>	<u>pH</u>	<u>Temperature (°C)</u>	<u>Duration</u>	<u>Effect</u>	<u>Concentration (mg/L NH<sub>3</sub>)</u>	<u>Reference</u>
<u>Rainbow trout, Salmo gairdneri</u>	NH <sub>4</sub> Cl	7.69	13.2	35 d	LC50	0.426	Thurston & Russo 1983
<u>Rainbow trout, Salmo gairdneri</u>	NH <sub>4</sub> Cl	7.69	13.2	35 d	LC50	0.322	Thurston & Russo 1983
<u>Rainbow trout, Salmo gairdneri</u>	NH <sub>4</sub> Cl	7.9	13	35 d	LC50	0.659	Thurston & Russo 1983
<u>Rainbow trout, Salmo gairdneri</u>	NH <sub>4</sub> Cl	7.82- 8.06	8-12	90 d	No gill lesion	0.2- 0.4	Daoust & Ferguson 1984
<u>Rainbow trout, Salmo gairdneri</u>	NH <sub>4</sub> Cl	7.7	9.3	5 yr	Histopath- ological effects in parental and juveniles	>0.04	Thurston et al. 1984b
<u>Atlantic salmon, Salmo salar</u>	NH <sub>4</sub> Cl	7.81	13.6	24 h	LC50	0.28 <sup>a</sup>	Herbert & Shurben 1965
<u>Atlantic salmon, Salmo salar</u>	NH <sub>4</sub> Cl	7.69	12.0	24 h	LC50 in freshwater	0.15	Alabaster et al. 1979
<u>Atlantic salmon, Salmo salar</u>	NH <sub>4</sub> Cl	7.92	12.0	24 h	LC50 in 30‰ seawater	0.3	Alabaster et al. 1979
<u>Atlantic salmon, Salmo salar</u>	NH <sub>4</sub> Cl	8.12	10.7	24 h	LC50 (10 mg/L dissolved oxygen)	0.2	Alabaster et al. 1983
<u>Atlantic salmon, Salmo salar</u>	NH <sub>4</sub> Cl	8.05	11.5	24 h	LC50 (3.2 mg/L dissolved oxygen)	0.08	Alabaster et al. 1983
<u>Brown trout, Salmo trutta</u>	NH <sub>4</sub> OH	7.8	11	18 h	36% mortality	0.15 <sup>a</sup>	Taylor 1973
<u>Brown trout, Salmo trutta</u>	-	7-8	15	"Un- limited"	No observed effect	0.8	Woker & Wuhrmann 1950
<u>Brook trout, Salvelinus fontinalis</u>	NH <sub>3</sub> excreted from fish	7.0	12.8	1.75 h	distress	3.25	Phillips 1950

Table 5. (Continued)

<u>Species</u>	<u>Chemical</u>	<u>pH</u>	<u>Temperature (°C)</u>	<u>Duration</u>	<u>Effect</u>	<u>Concentration (mg/L NH<sub>3</sub>)</u>	<u>Reference</u>
<u>Brook trout, Salvelinus fontinalis</u>	NH <sub>3</sub> excreted from fish	7.0	15	2.5 h	distress	5.5	Phillips 1950
<u>Goldfish, Carassius auratus</u>	NH <sub>4</sub> Cl	7.9	22	24 h	LC50	7.2 <sup>a,f</sup>	Dowden & Bennett 1965
<u>Goldfish, (sp. not specified)</u>	NH <sub>4</sub> Cl	7.65	18.8- 20.5	15 d	Lethal threshold	1.4-1.5 <sup>a</sup>	Chipman 1934
<u>Goldfish, (sp. not specified)</u>	(NH <sub>4</sub> ) <sub>2</sub> SO <sub>4</sub>	7.60	18.80 20.5	15 d	Lethal threshold	0.97-1.1 <sup>a</sup>	Chipman 1934
<u>Goldfish, (sp. not specified)</u>	(NH <sub>4</sub> ) <sub>2</sub> CO <sub>3</sub>	8.0	18.80- 20.5	15 d	Lethal threshold	3.4-3.8 <sup>a</sup>	Chipman 1934
<u>Carp, Cyprinus carpio</u>	Urea	8.75	16-18	2.42 h	time to death	9.7	Nehring 1962-63
<u>Carp, Cyprinus carpio</u>	Urea	8.35	16-18	6.0 h	time to death	2.1	Nehring 1962-63
<u>Carp, Cyprinus carpio</u>	(NH <sub>4</sub> ) <sub>2</sub> SO <sub>4</sub>	7.8	24.5	4 d	Minimum lethal concentration	1.4 <sup>a</sup>	Malacea 1966
<u>Carp, Cyprinus caprio</u>	NH <sub>4</sub> Cl	7.4	28	96 h	LC50	1.1	Rao et al. 1975
<u>Carp, (sp. not specified)</u>	Manure leachate	-	15-17	"Unlimited"	No observed effect	<1.5	Danecker 1964
<u>Carp, (sp. not specified)</u>	-	-	-	4 h	death	7.5 <sup>b</sup>	Kemplnska 1968
<u>Carp, (sp. not specified)</u>	(NH <sub>4</sub> ) <sub>2</sub> SO <sub>4</sub>	7.8	18	18 h	Not lethal	0.24	Vamos 1963
<u>Carp, (sp. not specified)</u>	(NH <sub>4</sub> ) <sub>2</sub> SO <sub>4</sub>	8.2	25	17 min	Loss of equilibrium	0.67	Vamos 1963
<u>Carp, (sp. not specified)</u>	(NH <sub>4</sub> ) <sub>2</sub> SO <sub>4</sub>	-	22	45 min	Loss of equilibrium	0.52	Vamos 1963
<u>Fathead minnow, Pimephales promelas</u>	NH <sub>4</sub> Cl	7.59- 7.82	21.6- 21.9	72 h	LC50	1.68	Sparks 1975

Table 5. (Continued)

<u>Species</u>	<u>Chemical</u>	<u>pH</u>	<u>Temperature (°C)</u>	<u>Duration</u>	<u>Effect</u>	<u>Concentration (mg/L NH<sub>3</sub>)</u>	<u>Reference</u>
<u>Fathead minnow, <i>Pimephales promelas</i></u>	NH <sub>4</sub> Cl	8.0	25	304 d	Histopath- ological intracerebral lesions	>0.639	Smith 1984
<u>Bitterling, <i>Rhodeus sericeus</i></u>	(NH <sub>4</sub> ) <sub>2</sub> SO <sub>4</sub>	7.8	24.5	4 d	Minimum lethal concentration	0.76 <sup>a</sup>	Malacea 1966
<u>Rudd, <i>Scardinius erythrophthalmus</i></u>	NH <sub>4</sub> Cl	8.05- 8.30	12.2- 13.2	6 d	asymptotic LC50	0.44 <sup>b</sup>	Ball 1967
<u>Creek chub, <i>Semotilus atromaculatus</i></u>	NH <sub>4</sub> OH	8.3	15-21	24 h	*critical range <sup>a</sup>	0.26-1.2 <sup>a</sup>	Gillette et al. 1952
<u>Tench, <i>Tinca tinca</i></u>	Manure leachate	-	18	20-24 h	time to death	2.5	Danecker 1964
<u>Channel catfish, <i>Ictalurus punctatus</i></u>	NH <sub>3</sub> excreted from fish	7.7	21.1	1 wk	LC50	0.974 <sup>a</sup>	Knepp & Arkin 1973
<u>Channel catfish, <i>Ictalurus punctatus</i></u>	NH <sub>3</sub> excreted from fish	7.8	21.7	1 wk	LC50	1.27 <sup>a</sup>	Knepp & Arkin 1973
<u>Channel catfish, <i>Ictalurus punctatus</i></u>	NH <sub>3</sub> excreted from fish	7.8	22.8	1 wk	LC50	1.41 <sup>a</sup>	Knepp & Arkin 1973
<u>Channel catfish, <i>Ictalurus punctatus</i></u>	NH <sub>3</sub> excreted from fish	8.0	22.8	1 wk	LC50	1.97 <sup>a</sup>	Knepp & Arkin 1973
<u>Channel catfish, <i>Ictalurus punctatus</i></u>	NH <sub>4</sub> Cl	7.73- 8.16	19.8- 20.0	48 h	LC50	2.92	Sparks 1975
<u>Channel catfish, <i>Ictalurus punctatus</i></u>	-	-	-	48 h	LC50	1.24- 1.96	Vaughn & Simco 1977
<u>Channel catfish, <i>Ictalurus punctatus</i></u>	NH <sub>4</sub> Cl	7.0	21- 25	24 h	LC50	1.69 <sup>b</sup>	Tomasso et al. 1980
<u>Channel catfish, <i>Ictalurus punctatus</i></u>	NH <sub>4</sub> Cl	7.0	21- 25	24 h	LC50	2.17 <sup>b</sup>	Tomasso et al. 1980

Table 5. (Continued)

<u>Species</u>	<u>Chemical</u>	<u>pH</u>	<u>Temperature (°C)</u>	<u>Duration</u>	<u>Effect</u>	<u>Concentration (mg/L NH<sub>3</sub>)</u>	<u>Reference</u>
<u>Channel catfish, Ictalurus punctatus</u>	NH <sub>4</sub> Cl	8.0	21- 25	24 h	LC50	2.21 <sup>b</sup>	Tomasso et al. 1980
<u>Channel catfish, Ictalurus punctatus</u>	NH <sub>4</sub> Cl	9.0	21- 25	24 h	LC50	1.81 <sup>b</sup>	Tomasso et al. 1980
<u>Channel catfish, Ictalurus punctatus</u>	NH <sub>3</sub> Excreted by fish	-	-	7 mo	Histopath- ological gill lesions in pond cultures	0.020- 0.067	Soderberg et al. 1984
<u>Mosquitofish, Gambusia affinis</u>	NH <sub>4</sub> Cl	7.8	21.8	17 h	LC50	1.3	Hemans 1966
<u>Guppy, Poecilia reticulata</u>	NH <sub>4</sub> Cl	7.0	25	24 h	LC50	0.8 <sup>a</sup>	Tabata 1962
<u>Guppy, Poecilia reticulata</u>	NH <sub>4</sub> Cl	8.0	25	24 h	LC50	1.4 <sup>a</sup>	Tabata 1962
<u>Green sunfish, Lepomis cyanellus</u>	NH <sub>4</sub> Cl	7.82- 8.56	23.3- 27.3	31 d	Larval mortality	0.80	Reinbold & Pescitelli 1982a
<u>Bluegill, Lepomis macrochirus</u>	NH <sub>4</sub> Cl	7.72- 8.00	21.9- 22.1	48 h	LC50	2.30	Sparks 1975
<u>Bluegill, Lepomis macrochirus</u>	NH <sub>4</sub> Cl	7.9	22	96 h	LC50	8.1 <sup>a,f</sup>	Dowden & Bennett 1965
<u>Bluegill, Lepomis macrochirus</u>	NH <sub>4</sub> OH	6.9- 7.5	20	48 h	LC50	0.024- 0.093 <sup>a</sup>	Turnbull et al. 1954
<u>Largemouth bass, Micropterus salmoides</u>	-	7.0	22	24 h	Opercular rhyth- m frequency increase	0.028	Morgan 1976, 1977
<u>Largemouth bass, Micropterus salmoides</u>	-	7.0	22	5 d	Threshold value (Increase in activity)	0.0055 <sup>a</sup>	Morgan 1978, 1979

Table 5. (Continued)

<u>Species</u>	<u>Chemical</u>	<u>pH</u>	<u>Temperature (°C)</u>	<u>Duration</u>	<u>Effect</u>	<u>Concentration (mg/L NH<sub>3</sub>)</u>	<u>Reference</u>
<u>Largemouth bass, Micropterus salmoides</u>	-	7.0	22	5 d	Threshold value (Increase in opercular rhythm)	0.028 <sup>a</sup>	Morgan 1978, 1979
<u>Oscar, Astronotus ocellatus</u>	-	8.4	25.5	9-24 h	4-100% mortality	0.50- 1.8 <sup>a</sup>	Magalhaes Bastos 1954
<u>Oscar, Astronotus ocellatus</u>	-	8.5	25.5	13 d	20% mortality	1.4 <sup>a</sup>	Magalhaes Bastos 1954
<u>Blue tilapia, Tilapia aurea</u>	NH <sub>4</sub> Cl	7.3- 7.4	25	72 h	LC50	2.85 <sup>b</sup>	Redner & Stickney 1979
<u>SALTWATER SPECIES</u>							
<u>Nemertine worm, Cerebratulus fuscus</u>	NH <sub>4</sub> NO <sub>3</sub>	7.9	15	106 min	LT50	2.3 <sup>a</sup>	Brown 1974 <sup>9</sup>
<u>Mussel, Mytilus edulis</u>	NH <sub>4</sub> Cl	7.5	18	<1 h	50% reduction in ciliary	0.097 <sup>b</sup>	Anderson et al. 1978
<u>Mussel, Mytilus edulis</u>	NH <sub>4</sub> Cl	7.5	18	<1 h	90% reduction in ciliary beating rate	0.11 <sup>b</sup>	Anderson et al. 1978
<u>Mussel, Mytilus edulis</u>	NH <sub>4</sub> Cl	7.5	18	<1 h	Complete In- hibition of cilia	0.11- 0.12 <sup>b</sup>	Anderson et al. 1978
<u>Copepod, Eucalanus elongatus</u>	NH <sub>4</sub> Cl	8.1	20	96 h	LC50	>0.66	Venkataramlak et al. 1981b
<u>Copepod, Eucalanus plleatus</u>	NH <sub>4</sub> Cl	8.2	20	96 h	LC50	>0.65	Venkataramlak et al. 1981b
<u>Prawn, Penaeus setiferus</u>	NH <sub>4</sub> Cl	-	28	3 wk	EC50 <sup>d</sup>	0.72 <sup>b,e</sup>	Wicklins 1976

Table 5. (Continued)

<u>Species</u>	<u>Chemical</u>	<u>pH</u>	<u>Temperature (°C)</u>	<u>Duration</u>	<u>Effect</u>	<u>Concentration (mg/L NH<sub>3</sub>)</u>	<u>Reference</u>
<u>Prawn, Macrobrachium rosenbergii</u>	NH <sub>4</sub> Cl	7.0	29.2	1700 min.	LT50	1.7 <sup>b</sup>	Wicklins 1976
<u>Prawn, Macrobrachium rosenbergii</u>	NH <sub>4</sub> Cl	7.0	29.2	1400 min.	LT50	2.7 <sup>b</sup>	Wicklins 1976
<u>Prawn, Macrobrachium rosenbergii</u>	NH <sub>4</sub> Cl	7.0	29.2	560 min.	LT50	3.4 <sup>b</sup>	Wicklins 1976
<u>Prawn, Macrobrachium rosenbergii</u>	NH <sub>4</sub> Cl	-	28	6 wk	30-40% Growth reduction	0.12 <sup>b</sup>	Wicklins 1976
<u>Prawn, Macrobrachium rosenbergii</u>	NH <sub>4</sub> Cl	6.83	28	24 h	LC50	0.66	Armstrong et al. 1978
<u>Prawn, Macrobrachium rosenbergii</u>	NH <sub>4</sub> Cl	6.83	28	144 h	LC50	0.26	Armstrong et al. 1978
<u>Prawn, Macrobrachium rosenbergii</u>	NH <sub>4</sub> Cl	7.60	28	24 h	LC50	2.10	Armstrong et al. 1978
<u>Prawn, Macrobrachium rosenbergii</u>	NH <sub>4</sub> Cl	7.60	28	144 h	LC50	0.80	Armstrong et al. 1978
<u>Prawn, Macrobrachium rosenbergii</u>	NH <sub>4</sub> Cl	8.34	28	24 h	LC50	3.58	Armstrong et al. 1978
<u>Prawn, Macrobrachium rosenbergii</u>	NH <sub>4</sub> Cl	8.34	28	144 d	LC50	1.35	Armstrong et al. 1978
<u>Prawn, Macrobrachium rosenbergii</u>	NH <sub>4</sub> Cl	6.83	28	7 d	Reduction in growth rate	0.11	Armstrong et al. 1978
<u>Prawn, Macrobrachium rosenbergii</u>	NH <sub>4</sub> Cl	7.60	28	7 d	Reduction in growth rate	0.63	Armstrong et al. 1978
<u>Grass shrimp, Palaemonetes pugio</u>	NH <sub>4</sub> Cl	8.0- 8.2	20	48 h	LC50	0.34- 0.53 <sup>c</sup>	Hall et al. 1978

Table 5. (Continued)

<u>Species</u>	<u>Chemical</u>	<u>pH</u>	<u>Temperature (°C)</u>	<u>Duration</u>	<u>Effect</u>	<u>Concentration (mg/L NH<sub>3</sub>)</u>	<u>Reference</u>
Lobster, <u>Homarus americanus</u>	NH <sub>4</sub> Cl	8.1	21.9	8 d	LC50	1.7 <sup>b</sup>	Dellstraty et al. 1977
Coho salmon, <u>Oncorhynchus kisutch</u>	NH <sub>4</sub> Cl	-	15.5- 16	48 h	LC50	0.50	Katz & Pierro 1967
Atlantic salmon, <u>Salmo salar</u>	NH <sub>4</sub> Cl	7.46- 7.90	11.9- 13.8	24 h	LC50	0.14- 0.26	Alabaster et al. 1983

<sup>a</sup> Recalculated from authors' total ammonia values.

<sup>b</sup> Recalculated from authors' NH<sub>3</sub>-N values.

<sup>c</sup> Estimated from authors' graphs.

<sup>d</sup> EC50 based on reduction in growth.

<sup>e</sup> Author's calculated NH<sub>3</sub>-N values could be increased by up to 11% due to imprecision of pH measurements (Wickins 1976).

<sup>f</sup> pH and temperature data from: Freeman 1953.

<sup>g</sup> pH data obtained from: Brown, A., personal communication.

<sup>h</sup> Recalculated from authors' NH<sub>3</sub> value (see text).

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