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Boron Hazards to Fish, Wildlife, and Invertebrates: A Synoptic Review

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ABSTRACT. Ecological and toxicological aspects of boron (B) in the environment are reviewed, with emphasis on natural resources. Subtopics covered include environmental chemistry, background concentrations, effects, and current recommendations for the protection of living resources.

The United States is the major producer of boron compounds and supplies about 70% of the annual global demand. Although boron is ubiquitous in the environment, human activities such as mining, coal burning, drainwater disposal, and use of borax laundry detergents have resulted in elevated boron loadings in the atmosphere and in irrigation waters. The chemistry of boron is complex and rivals that of carbon in its diversity. However, most boron compounds enter or degrade in the environment to B-0 compounds (borates)such as borax and boric acid--and these are considered to be the most significant ecologically.

Boron is an essential trace element for the growth of terrestrial crop plants and for some species of fungi, bacteria, and algae, but excess boron is phytotoxic. Representative species of aquatic organisms, including plants, invertebrates, fishes, and amphibians, usually tolerated up to 10 mg B/L of medium for extended periods without harm. In waterfowl, growth was adversely affected at dietary levels of 30 to 100 mg B/kg fresh weight, tissue boron concentrations were elevated at 100 to 300 mg B/kg diet, and survival was reduced at dietary levels of 1,000 mg B/kg; all of these dietary levels currently exist near agricultural drainwater disposal sites in the western United States. Boron is not now considered essential in mammalian nutrition, although low dietary levels protect against fluorosis and bone demineralization. Excessive consumption (i.e., > 1,000 mg B/kg diet, > 15 mg B/kg body weight daily, > 1.0 mg B/L drinking water, or > 210 mg B/kg body weight in a single dose) adversely affects growth, survival, or reproduction in sensitive mammals. Boron and its compounds are potent teratogens when applied directly to the mammalian embryo, but there is no evidence of mutagenicity or carcinogenicity. Boron's unique affinity for cancerous tissues has been exploited in neutron capture radiation therapy of malignant human brain tumors.

Current boron criteria recommended for the protection of sensitive species include < 0.3 mg B/L in crop irrigation waters, < 1.0 mg B/L for aquatic life, < 5.0 mg B/L in livestock drinking waters, < 30 mg B/kg in waterfowl diets, and < 100 mg B/kg in livestock diets.

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TABLES

Introduction

Borax ($Na_2B_40_7$ .10 H_20) was the first of the boron (B) minerals to be traded by the Babylonians more than 4,000 years ago for use in the working and welding of gold (Greenwood and Thomas 1973). Borax has been known as a cleaning agent since the days of the ancient Greek and Roman empires and was used as a food preservative in Europe and America, although its use for the latter purpose has been discontinued (Weir and Fisher 1972). Boron and its compounds were used in Egyptian and Roman eras to prepare borosilicate glass. Borax glazes were known from about the year 200; by 1556, borax was widely used throughout Europe as a flux (Greenwood and Thomas 1973). Boric acid (H_3BO_3) was first synthesized in 1707 (Greenwood and Thomas 1973). Boric acid and borates are the main boron compounds of ecological significance; other boron compounds usually degrade or are transformed to borates or boric acid (Sprague 1972).

Boron is an essential trace element for the growth and development of higher plants, although the range between insufficiency and excess is generally narrow, varying with the plant; boron is not required in fungi and animals (Sprague 1972; Weir and Fisher 1972; Birge and Black 1977; Goldbach and Amberger 1986). In the southwestern United States, naturally elevated boron concentrations in surface waters used for irrigation may be sufficiently high to cause toxicity to plants of commercial importance (Benson et al. 1984). Another major source of boron entering ground and surface waters results from the use of borax-containing laundry products coupled with ineffective removal of boron by conventional sewage processes (Benson et al. 1984). Agricultural drainwaters contaminated with boron are considered potentially hazardous to waterfowl and other wildlife populations throughout areas of the western United States (Smith and Anders 1989).

Medical and household uses of boric acid solutions as antiseptics have led to numerous accidental poisonings by ingestion or absorption through abraded skin, particularly in infants (Environmental Protection Agency 1975; Dixon et al. 1976; Landolph 1985; Siegel and Wason 1986). Poisonings have been reported in English children consuming milk containing 0.7 g boric acid/L, and in burn patients treated topically with saturated boric acid solutions (National Academy of Sciences 1980). In the 1940's, topical preparations of boric acid became a popular remedy for diaper rash in England. By 1953, at least 60 fatal cases of boric acid poisoning had been reported in English infants (O'Sullivan and Taylor 1983). Inhalation of boranes, especially diborane (B_2H_6), pentaborane (B_5H_9), and decaborane ($B_{IO}H_{14}$)--which is used as a rocket propellant--is toxic to exposed workers (Dixon et al. 1976; NAS 1980). Boron compounds, especially boric acid, can also accumulate in animal tissues and produce a reduction in fertility, an increase in developmental abnormalities--especially those involving the skeletal system--stillbirth, and death (Weir and Fisher 1972; Lee et al. 1978; Landolph 1985). At present, there seems to be a reasonable margin between a toxic dose in humans and other vertebrates and in boron levels that may occur as incidental residues from the use of borax and boric acid in agriculture and industry (Weir and Fisher 1972). Additional information on ecological and toxicological aspects of boron in the environment is presented in reviews by Sprague (1972), Environmental Protection Agency (EPA; 1975), National Academy of Sciences (NAS; 1980), Anonymous (1983), Klasing and Pilch (1988), and Butterwick et al. (1989).

In this report, I summarize available data on boron in the environment, with emphasis on fishery and wildlife resources. It is part of a continuing series of brief reviews on chemical contaminants and natural resources that are prepared in response to informational requests from environmental specialists of the U.S. Fish and Wildlife Service.

Environmental Chemistry

General

The United States supplies about 70% of the global boron demand, and Turkey supplies 18%. Of the total annual United States production of about 500,000 tons, 45% is used in the manufacture of glass and glassware, 15% in laundry products, 10% in enamels and glazes, and 8% in agricultural chemicals. It is estimated that boron compounds enter the North American environment at a rate of 32,000 tons annually as a result of human activities, primarily from laundry products, irrigation drainwater, agricultural chemicals, coal combustion, and mining and processing (Table 1). Boron compounds tend to accumulate in aquatic ecosystems because of the relatively high water solubility of these compounds (EPA 1975).

The chemistry of boron is exceedingly complex and rivals that of carbon in its diversity. Most boron compounds, however, enter or degrade in the environment to borates (B-0 compounds), such as borax and boric acid, and these are considered to be the most significant ecologically.

Toxicosis in animals has resulted from ingestion of boric acid or borax solutions, from topical applications of boric acid solutions to damaged skin, and from inhalation of boranes; the exact mechanisms of action are not understood. Boron and its compounds are potent teratogens when applied directly to the embryo, but there is no evidence of mutagenicity or carcinogenicity. Boron's unique affinity for cancerous tissues has been exploited in neutron capture radiation therapy of malignant human brain tumors.

Sources and Uses

Boron is a dark brown element that is widespread in the environment but occurs naturally only in combined form, usually as borax, colemanite ($Ca_2B_60_{11} \cdot 5H_20$), boronatrocalcite ($CaB_40_7NaBO_2 \cdot 8H_20$), and boracite ($Mg_7Cl_2B_{16}O_{30}$; EPA 1975; NAS 1980). In the United States, boron deposits in the form of borax are concentrated in the desert areas of southern California, especially near Boron, California (EPA 1975). Proven deposits of sodium tetraborates--from which borax is prepared and from which boron can be isolated--also exist in Nevada, Oregon, Turkey, Russia, and China (Sprague 1972; NAS 1980). The United States supplies about 70% of the world boron demand, and Turkey supplies 18%; the most common commercial compounds are boric acid and borax (Sprague 1972; Butterwick et al. 1989).

The majority of the 555,000 tons of boron produced annually in the United States--of which half is usually exported--is in the form of sodium tetraborate compounds. Of the total production, about 42% occurs as anhydrous borax ($Na_2B_40_7$), 29% as borax pentahydrate ($Na_2B_40_7 \cdot 5H_20$), 10% as borax decahydrate or borax, and 16% as boric acid or boric oxide (B_20_3; Sprague 1972; EPA 1975). Boron and its compounds are used in the manufacture of glassware (40 to 45%); soaps and cleansers (15%); enamels, frits, and glazes (10%); fertilizers (5%); and herbicides (2 to 3%); 22 to 28% goes for other uses (Sprague 1972; EPA 1975; NAS 1980). Borates have some toxicity to insects and, in relatively high concentrations, can control cockroaches, woodboring insects, gypsy moths, and larvae of flies in manure piles and in dog runs (Sprague 1972; EPA 1975). Some organoboron compounds are used to sterilize fuel distribution and storage systems against fungi and bacteria (Sprague 1972). Radioboron-10 is widely used in radiation therapy against brain tumors, especially in Japan (Hatanaka 1986). Various boron compounds are also used widely as thermal protection materials in space probes, in fireproofing of fabrics and wood, in leather manufacture, in numerous pharmaceuticals and hygienic products, in steel hardening, in deoxidation of bronze, as a high-energy fuel, as neutron-absorbing shielding near atomic reactors, and as water softeners, pH adjusters, emulsifiers, neutralizers, stabilizers, buffers, and viscosifiers (NAS 1980; Parry and Kodama 1980; Schillinger et al. 1982; Siegel and Wason 1986).

Most boron ends up in the aquatic environment because of the relatively high water solubility of all boron compounds, especially B-containing laundry products and sewage (EPA 1975). Conventional sewage treatment removes little or no boron (EPA 1975). Of the total boron in coal, as much as 71% may be lost to the atmosphere upon combustion; more than 50% of the boron found in coal ash is readily water soluble (Pagenkopf and Connolly 1982). The release of boron from coal fly ash to leachate water is dependent on the ash to water ratio: at 1 g ash/L up to 90% of the boron is soluble; at 50 g/L only 40% is released; at 100 g/L less than 30% is released. Coating of coal ash with aluminum solution reduces boron solubility by about 90% because of the formation of an insoluble aluminum-borate complex (Pagenkopf and Connolly 1982).

Table 1. Environmental sources of domestic boron (EPA 1975).

Source	Metric tons, annually
Laundry products	14,000
Agricultural chemicals and fertilizers	7,000
Coal combustion	4,000
Mining and processing	3,000
Glass and ceramics	1,500
Miscellaneous	2,500
Total	32,000

Boron compounds listed in the "Commodity List of Explosives and Other Dangerous Articles" are boron trichloride ($BC1_3$), boron trifluoride (BF_3), decaborane ($B_{10}H_{14}$), and pentaborane (B_5H_9; EPA 1975). Boron trichloride is a corrosive liquid; the maximum quantity allowed in containers by rail is 1 L, and by air only one container is permitted per aircraft. Boron trifluoride is a nonflammable gas restricted to 140 kg in one outside container by rail, and to 140 kg in cargo planes only. Decaborane is a flammable solid, and transport by rail or air is limited to 12 kg. Pentaborane is a flammable liquid and is prohibited for transport by air or rail. Diborane (B_2H_6) and higher boranes are unstable and are classified as dangerous articles in transport; no more than 0.1 kg can be shipped in a cylinder (EPA 1975). Organic boron-oxygen compounds readily hydrolyze and should be stored and transferred in an inert atmosphere; usually, glass containers are used for shipping small quantities, and steel containers or tank cars are used for bulk items. Hazardous atmospheric conditions resulting from high concentrations of boron compounds are localized and are not considered a serious environmental problem (EPA 1975).

Chemical Properties

The element boron has an atomic number of 5, a molecular weight of 10.811, an oxidation state of 3 for simple compounds (but other oxidation states for carboranes and other polyhedral cage boron compounds), a specific gravity of 2.34, a melting point of 2,300 °C, sublimation at 2,550 °C, and is almost insoluble in water. Boron exists as B-10 (19.78%) and B-11 (80.22%) isotopes, and it contributes about 0.001% to the Earth's crust, although it does not occur free in nature (EPA 1975; Smith 1985). The chemistry of boron is exceedingly complex and rivals that of carbon in diversity. Reviews on boron's chemistry are especially abundant and include those by Steinberg and McCloskey (1964), Brotherton and Steinberg (1970a, b), Greenwood and Thomas (1973), Grimes (1982), Evans and Sparks (1983), Smith (1985), Emin et al. (1986), Heller (1986), Niedenzu and Trofimenko (1986), and Hermanek (1987).

Most boron compounds degrade in the environment to B-0 (borate) compounds, and these are the boron compounds of ecological significance-especially borax and boric acid (Sprague 1972; Antia and Cheng 1975; Thompson et al. 1976). Sodium tetraborate decahydrate (borax) has a melting point of 75°C, a boiling point of 320°C, and is soluble in water to 20 g/L at O°C and to 1,700 g/L at 100°C (EPA 1975). Boric acid has a melting point of 169°C, a boiling point of 300°C and, like borax, is exceedingly soluble in water: 63.5 g/L at 30°C and 276 g/L at 100°C (EPA 1975).

Boron exists in several forms in the soil (EPA 1975), and in soil solution it exists largely as the undissociated weak monobasic acid that accepts hydroxyl groups (Gupta and Macleod 1982). Most plant-available boron in soils is associated with soil organic matter (Gupta and Macleod 1982), with the hot-water soluble boron fraction (Hingston 1986), and with soil solution pH ranges of 5.5 to 8.5 and 10 to 11.5 (Goldberg and Glaubig 1986). It is assumed that boron adsorbs to soil particles and aluminum and iron oxide minerals (Goldberg and Glaubig 1986).

The predominant boron species in seawater is boric acid (Thompson et al. 1976); concentrations are higher at higher salinities and in proximity to industrial waste discharges (Liddicoat et al. 1983; Narvekar et al. 1983). In seawater, borate or boric acid occurs naturally at 4.5 to 5.5 mg/L. About 76% of the total inorganic boron in seawater occurs as undissociated boric acid [$B(OH)_3$], and the remainder is identified as the borate ion

$[B(OH)_4^-]$. Of the total borate ion, 44% seems to be complexed with sodium, magnesium, and calcium (Antia and Cheng 1975). Other evidence suggests additional complexation of borate with ferric ions and polyhydroxylated organic compounds (Antia and Cheng 1975).

Mode of Action

The complexing ability of the boron atom is considered to be the key explanation of why it is essential to higher plants (EPA 1975), although the exact mechanism of action is still unknown. Boron's complexing ability is thought to beneficially influence transport of sugars and other organic compounds, production of plant growth regulators, biosynthesis of nucleic acids and phenolic acid, carbohydrate metabolism, respiration, and pollen germination (EPA 1975; Nielson 1986).

Boron poisoning in animals is primarily an experimental phenomenon, although livestock in certain regions may be exposed to high concentrations in drinking water--up to 80 mg B/L--that have not been shown to be toxic (NAS 1980). Toxicosis in humans has resulted from ingestion of boric acid or borax solutions, topical applications of boric acid solutions to burn-damaged skin, and inhalation of boranes (NAS 1980). In mammals, boron is thought to regulate parathyroid function through metabolism of phosphorus, magnesium, and especially calcium. Boron has a close relation with calcium metabolism, most likely at the cell membrane level (Nielsen 1986).

Dietary boron at nontoxic concentrations, as sodium borate or boric acid, is rapidly and almost completely absorbed from the gastrointestinal tract, doesn't seem to accumulate in healthy tissues, and is excreted largely in urine, usually within hours; similar patterns are evident for humans, dogs, cows, rabbits, and guinea pigs (NAS 1980; Benson et al. 1984; Nielsen 1986; Siegel and Wason 1986). Boric acid poisoning in animals, regardless of route of administration, is characterized by the following signs: generalized erythema (boiled lobster appearance) starting in the axillary, inguinal, and facial regions, eventually covering the entire body with conjunctival redness, followed by massive desquamation 2 to 3 days later; acute gastroenteritis, including nausea and vomiting; diarrhea; anorexia; cardiac weakness; excessive urinary excretion of riboflavin; decreased oxygen uptake by the brain; hypoacidity; altered enzyme activity levels; impaired growth and reproduction; and death from circulatory collapse and shock, usually within 5 days (Dani et al. 1971; Sprague 1972; EPA 1975; NAS 1980; Schillinger et al. 1982; Settimi et al. 1982; Siegel and Wason 1986).

Boron hydrides or boranes, such as B_2H_6, B_4H_{10}, and B_5H_9, from chemical processes produce acute central nervous system pulmonary damage and lung disease through inhalation (NAS 1980; Klaassen et al. 1986). Boranes produce toxic effects by creating embolisms of hydrogen gas as they react with tissue, and by depleting biogenic amines of the central nervous system and inhibiting aminotransferases and other pyridoxol-dependent enzymes (Korty and Scott 1970; EPA 1975). Boranes produce similar effects in humans and animals, and these are generally ascribable to central nervous system depression and excitation (Naeger and Leibman 1972; Smith 1985). Symptoms of borane intoxication include pulmonary irritation, headache, chills, fatigue, muscular weakness and pain, cramps, dizziness, chest tightness, and pneumonia (NAS 1980). Boranes may adversely affect male reproductive capacity (Klaassen et al. 1986), but this requires verification. Decaborane ($B_{l0}H_{14}$), as one example, is a highly lipid-soluble compound that can enter the body through inhalation, ingestion, or the skin. In water, decaborane is rapidly transformed into intermediate products that are eventually degraded to boric acid. The intermediate products, but not decaborane or boric acid, reduce phosphomolybdic acid and inhibit glutamic-oxaloacetic transaminase; treatment of intermediates with pyridoxol phosphase tends to reverse the inhibitory activity (Naeger and Leibman 1972). Low decaborane doses cause behavioral effects such as depression, catatonia, and convulsions (EPA 1975).

Inorganic borates are quite toxic, seemingly complexing hydroxy compounds and interfering with protein synthesis (EPA 1975). Organoborate compounds exert physiological effects on the central nervous system and peripheral nervous system, acting as spasmolytics, sedatives, and convulsants, depending on their structure (EPA 1975). Boron trihalides, such as BBr_3, $BC1_3$, and BF_3, are corrosive to the eyes, skin, and mucous membranes, and will cause burns on the skin seemingly because of the hydrolysis of the trihalides to their halogen acids, and not to boron (EPA 1975; Smith 1985).

Boron is a potent teratogen when applied directly to the embryo. Boric acid injected into chicken and amphibian embryos produced abnormal development of the neural tube, notochord, tail, and limbs, perhaps through complexing polyhydroxy compounds and interfering with riboflavin metabolism (Landauer 1952, 1953a, 1953b, 1953c; Landauer and Clark 1964; EPA 1975; Settimi et al. 1982). Boron and its compounds, however, are neither mutagenic nor carcinogenic (Landolph 1985). Nonmutagenicity is based on results of the *Salmonella typhimurium*-mammalian microsome mutagenicity assay; boron neither enhances nor inhibits the activity of benzo(a)pyrene, a known mutagen (Anonymous 1983; Benson et al. 1984). There is no evidence that boron is a possible carcinogen, although long-term, selective uptake of boron by tumors has been reported (EPA 1975).

Boron seems to have an affinity for cancerous tumors, and this property has been exploited in radiation therapy (Hamada et al. 1983; Hatanaka 1986). Boron-10 has been used in neutron capture therapy to cure malignant sarcomas implanted in the hind legs of mice, as well as spontaneous malignant melanomas in pigs (Slatkin et al. 1986). The sulfhydral borane monomer $(B_{12}H_{11}SH)^{2-}$ is used as a B-10 carrier in neutron therapy of malignant human brain tumors and seems to be most effective at 30 µ*g* B-10/kg tissue (Hatanaka 1986). Polyhedral boranes attached to monoclonal antibodies that are tumor-specific may become useful in tumor therapy by neutron irradiation (Parry and Kodama 1980). It is possible, however, that uptake of boron may be a nonspecific attribute of tumors and of a variety of normal tissues that lack a blood-brain barrier. Thus, the potential usefulness of selected B-10 carriers for treating extracranial neoplasms seems questionable (Slatkin et al. 1986).

Background Concentrations

General

Terrestrial plants are normally rich sources of boron. Levels in meat and fish are usually low. These generalizations, however, are based on extremely limited data. Boron is ubiquitous in the environment, although human activities such as mining, coal burning, and use of borax laundry detergents have resulted in elevated boron loadings in the atmosphere and in irrigation waters. Comparatively high levels of boron were recorded in fish, insects, and aquatic plants at Kesterson National Wildlife Refuge, which was contaminated by agricultural drainwater; the significance of the contamination is under investigation.

Nonbiological

Boron is distributed widely in the environment (Ahl and Jonsson 1972; EPA 1975). Naturally elevated boron levels are usually associated with marine sediments, thermal springs, large deposits of boron minerals, seawater, and certain groundwaters (Table 2). Human activities, however, have resulted in elevated boron concentrations near coal-fired plants, in mine drainage waters, in municipal wastes, and in agricultural drainage waters. In one instance, agricultural drainwater practices in western California produced boron concentrations in local rivers, groundwaters, and surface waters that exceeded the established limits for the protection of crops and aquatic life (Table 2; Schuler 1987; Klasing and Pilch 1988).

Coal-fired power plants are major sources of atmospheric boron contamination; at least 30% of boron in coal is lost in this manner (Cox et al. 1978; Gladney et al. 1978). The seemingly large amounts of boron lost to the environment through stack emissions may be directly related to the organic content of coal (Table 2; Gladney et al. 1978). Also, disposal of B-laden drainage waters from boron mines is a major problem in certain geographic areas. In Turkey, for example, which possesses about 60% of the world's boron reserves--localized in a rectangular area about 100 x 200 km near the Simav River--drainage waters discharged from the mines as a result of borate production have elevated boron concentrations in the Simav River to levels unsuitable for crop irrigation. About 68,000 ha of agricultural land irrigated by the Simav River are now threatened by boron pollution (Okay et al. 1985). In the United States, laundry detergents originating from household use may contribute as much as 50% of the boron loadings in effluents discharged into aquatic environments; lesser amounts are contributed by soil minerals, rainfall, and industry and sewage effluents (EPA 1975).

Table 2. Boron concentrations in selected nonbiological materials. Concentrations are in mg B/kg fresh weight (FW), dry weight (DW), or ash weight (AW).

Material	Concentration (mg/kg or mg/L)	Reference[a]
Coal-fired power plants		
Chalk Point Power Plant, New Mexico		
Coal	13 AW	1
Bottom slag	19 AW	1
Fly ash	33 AW	1
Four Corners Power Plant, New Mexico		
Coal	92 AW	1
Bottom slag	120 AW	1
Fly ash	240 AW	1
Coal ash		
Anthracite	90 AW	1
Volatile bituminous		
Low	123 AW	1
Medium	218 AW	1
High	770 AW	1
Lignite	1,020 AW	1
Coal ash	5–200 DW	2, 15
Sediments		
Nonmarine clays	<10 DW	3
Postglacial marine	Max. 500 DW	3
Mine drainage waters		
Turkish boron mines		
Avsar mine	16 FW	4
Simav mine	260 FW	4
Yenikoy mine	390 FW	4
Soil		
Worldwide	Usually 45–124 DW, range 4–200, mostly as biologically unavailable tourmaline	5, 6
United States	30 (10–300) DW	7
Thermal springs, Greece	43 FW	4
Surface fresh waters		
Worldwide	0.0001–<0.5 FW	8, 9, 10, 16
Norway, 1970	Usually <0.004 FW, median 0.013, range 0.001–1.05 FW	3
Sweden, 1970	0.12 (0.001–1.0) FW	3, 7
Southeastern United States 1969–70		
Streams, swamps, ponds	Usually <0.1 FW	11
Reservoirs	0.007 (<0.001–0.09) FW	11
In regions where marine deposits are common	>0.006 FW	3
United States	Generally <0.1 FW	12
United States	0.1–0.3 FW	8
Nevada		
Humboldt River	0.2 FW	6
Borax Flat	Up to 80 FW	6
Turkey		
Uncontaminated	<0.5 FW	4
Contaminated with boron mine wastes	4 (Max. 7) FW	4
Western United States	Sometimes 5–15 FW	10, 12

Japan	1–15 FW	7
Seawater		
British Columbia		
Surface	3.5 (0.2–4.7) FW	13
Depth 5 m	3.9 FW	13
Open ocean	4.5 FW	8
Coastal	4.6 FW	8
Total inorganic	4.5–5.5 FW	14
As undissociated boric acid	3.4–4.2 FW	14
As borate ion, $B(OH)_4^-$	1.1–1.3 FW	14
As complex with sodium, magnesium, and calcium	0.5–0.6 FW	14
Groundwaters		
Worldwide	Usually <0.5 FW	9
Greece	2.3–5.4 FW	4
United States	Max. 5.0 FW	12
Sewage waters, Scandinavia	0.4 (Max. 0.7) FW	3
Well water, India	0.08–0.5 FW	7
Rain		
Sweden	0.002 FW	7
France	0.002–0.004 FW	7
United States		
Mississippi	Usually <0.01 FW	11
Florida	~0.01 FW	7, 8
India	0.03 (0.002–0.007) FW	11
England	0.08 FW	7
Japan	0.1 FW	7
Affected by agricultural drainage waters		
Western San Joaquin Valley, California		
River waters	Median 1.1 FW	16
Surface waters	Median 3.1 (Max. 83.0) FW	16
Groundwaters	Median 7.4 (Max. 70.0) FW	16
Kesterson National Wildlife Refuge, 1984		
Subsurface waters	20 (12–41) FW	17
Sediments	20 (10–71) DW	17

[a]1, Gladney et al. 1978; 2, Pagenkopf and Connolly 1982; 3, Ahl and Jonsson 1972; 4, Okay et al. 1985; 5, Gupta and Macleod 1982; 6, NAS 1980; 7, Sprague 1972; 8, EPA 1975; 9, Benson et al. 1984; 10, Lewis and Valentine 1981; 11, Boyd and Walley 1972; 12, Birge and Black 1977; 13, Thompson et al. 1976; 14, Antia and Cheng 1975; 15, Cox et al. 1978; 16, Klasing and Pilch 1988; 17, Schuler 1987.

Biological

Boron occurred at high concentrations in plants, insects, and fish at Kesterson National Wildlife Refuge in California--the recipient of contaminated agricultural drainwater--when compared to a nearby control area (Table 3; Ohlendorf et al. 1986; Schuler 1987). These authors indicated that little is known about the effect of boron ingestion on bird reproduction, although both boric acid and borax produced mortality and teratogenic development when injected into eggs. Recent studies on effects of boron on waterfowl growth, physiology (Hoffman et al. 1990), and reproduction (Smith and Anders 1989) are discussed later.

Table 3. Boron concentrations in field collections of selected species of plants and animals. Concentrations are in mgB/kg fresh weight (FW), dry weight (DW), or ash weight (AW).

Ecosystem, organism, and other variables	Concentration (mg/kg)	Reference[a]
Terrestrial plants		
Cereal grains	1–5 DW	1, 2
Box thorn, *Lycium andersonii*		
Stem 7–26 DW	8	
Leaf 26–163 DW	8	
Root 25–74 DW	8	
Prunes, raisins, dates	9–27 DW	2
Tropical fruits	Usually <10 FW	1
Nuts 16–23 DW	2	
Vegetables	Usually <13 DW	2
Angiosperms	Mean 50 DW	8
Gymnosperms	Mean 63 DW	8
Pteridophytes	Mean 77 DW	8
Big sagebrush, *Artemisia tridentata*		
On high B soil		
Whole	Max. 250 DW	8
Leaf Max. 156 DW	8	
Stem Max. 54 DW	8	
Apple, pear, tomato, red pepper	440–1,250 DW	2
Freshwater organisms		
Lake trout, *Salvelinus namaycush*, muscle	0.2–0.6 FW	8
Cattail, *Typha latifolia*, whole	15–30 DW	8
Aquatic macrophytes		
22 species	Usually <20 DW; mean 11.3 (1.2–100) DW	4
Macrophytes, various	2–19 DW	5
Waterweed, *Elodea* sp., whole	18–44 DW	8
Pondweed, *Potamogeton* sp., whole	18–170 DW	8
Yellow pond lily, *Nuphar* sp., whole	23–31 DW	8
Watermilfoil, *Myriophyllum* sp., whole	25–54 DW	8
Kesterson National Wildlife Refuge, California, contaminated with irrigation drainwater		
1983		
Aquatic plants	382 (270–510) DW	3
Aquatic insects	45 (36–54) DW	3
Mosquitofish, *Gambusia affinis*, whole	11 (8–20) DW	3
1984		
Widgeongrass, *Ruppia maritima*		
Whole	371 (120–780) DW	9
Seeds	1,860 (450–3,500) DW	9
Filamentous algae	501 (390–787) DW	9
Aquatic insects	43 to 186 (22–340) DW	9
Volta Wildlife area, California, control area		
1983		
Aquatic plants	34 DW	3
Aquatic insects	13 (6–35) DW	3
Mosquitofish, whole	2.8 (Max. 3.6) DW	3
1984		
Widgeongrass		

Whole	100 (37–540) DW	9
Seeds	36 (32–43) DW	9
Filamentous algae	85 (64–140) DW	9
Aquatic insects	12 to 32 (7–47) DW	9
Western San Joaquin Valley, California, contaminated with irrigation drainwater		
Vegetation and seeds	Max. 3,390 DW	10
Clams, 2 species, muscle	Max. 9.3 FW	10
Bluegill, *Lepomis macrochirus*, whole	<0.8–1.9 FW, Max. 3.9 FW	10
Common carp, *Cyprinus carpio*, whole	0.5–5.7 FW, Max. 6.2 FW	10
Marine organisms		
Seaweeds, whole, Japan, 41 species	106 (16–319) DW 762 (231–3,308) AW	6
Marine algae	4–120 DW	8
Zooplankton	18–216 DW	7
Ctenophore, *Beroe cucumis*	115 AW	6
Corals, 34 species		
Deep open ocean	50–85 DW	6
Shallow open ocean	65–100 DW	6
Shallow coastal zone	40–110 DW	6
Tunicate, *Salpa fusiformis*, whole	50 AW	6
Chaetognath, *Sagitta elegans*	130 AW	6
Dungeness crab, *Cancer magister*, whole	1.8 (0.9–3.3) FW	6, 7
Molluscs, bivalves		
Soft parts, 11 species	1.6–4.5 FW	6
Soft parts, British Columbia		
Clams, 8 species	0.9–5.3 FW	7
Oysters, 2 species	3.1–4.0 FW	7
Mussels, 2 species	2.0–5.5 FW	7
Octopus, *Polypus bimaculatus*, whole	1.3 FW	7
Sockeye salmon, *Oncorhynchus nerka*		
Soft tissues	0.5–0.7 FW	6, 7
Bone	1.5 (1.1–4.4) FW	6, 7
Anchovetta, *Cetengraulis mysticetus*, whole	3.3–3.8 AW	8
Yellowfin tuna, *Thunnus albacares*		
Muscle	39.0 AW	8
Whole	9.0 AW	8
Eyeball	5.6 AW	8
Spleen	3.3 AW	8
Gill	1.8 AW	8
Heart	1.5 AW	8
Harbor seal, *Phoca vitulina*		
Blood	2.0 FW	8
Spleen	0.5 FW	8
Muscle	0.3 FW	8
Liver	0.2 FW	8
Heart	0.1 FW	8
Kidney	0.01 FW	8
Mammals, terrestrial		
Humans		
Teeth	18.2 (0.5–69) DW	1
Rib	6.2–10.2 AW	1
Kidney, lung, lymph nodes	0.6 FW	1, 2
Blood	0.1–0.4 FW	1, 2

Serum	0.2 FW	2
Muscle	0.1 FW	1, 2
Testes	0.09 FW	2
Milk	0.06–0.08 FW	2
Brain	0.06 FW	1, 2
Animal meat for human consumption	0.2 DW	2
Animal muscle and organs	0.5–1.5 DW	1
Milk, cow	0.5–1.0 FW	1
Dairy products	1.1 DW	2

[a]1, NAS 1980; 2, Nielsen 1986; 3, Ohlendorf et al. 1986; 4, Boyd and Walley 1972; 5, Ahl and Jonsson 1972; 6, Eisler 1981; 7, Thompson et al. 1976; 8, Jenkins 1980; 9, Schuler 1987; 10, Klasing and Pilch 1988.

Terrestrial plants, especially nuts, some fruits, and vegetables, are rich sources of boron (Table 3). Honey is another good source of boron, and concentrations up to 7.2 mg/kg dry weight (DW) have been reported (Nielsen 1986). Boron concentrations are also elevated in marine plants, zooplankton, and corals, but are low in fish and certain marine invertebrates (Table 3). No data are available on boron levels in terrestrial mammalian wildlife. Data for humans and domestic animals indicate that boron levels are elevated in bony tissues, but are always less than 0.6 mg B/kg fresh weight (FW) or 1.5 mg/ kg DW in other tissues examined (Table 3).

Effects

General

Boron is essential for the growth of higher plants and has been applied to B-deficient soils for at least 50 years to improve yields of many crops. Phytotoxic levels of B occur usually as a result of human activities, such as B-contaminated irrigation waters and excess applications of B-rich fertilizers, sewage sludges, and fly ashes. Boron compounds at comparatively high concentrations are used to control pestiferous insects through direct biocidal action, through enhancement of disease sensitivity, or through use as a chemosterilant.

Representative species of aquatic plants, invertebrates, fishes, and amphibians can usually tolerate up to 10 mg B/L medium for extended periods without adverse effects, although it has been suggested that concentrations >0.l mg B/L may ultimately affect reproduction in rainbow trout (*Oncorhynchus mykiss*), and >0.2 mg/L may impair survival of other fish species.

In waterfowl, diets that contain 30 or 100 mg B/kg FW adversely affect growth rate. Elevated tissue residues were recorded in ducks fed diets containing between 100 and 300 mg B/kg, and reduced survival occurred at dietary levels of 1,000 mg B/kg. Boron is a potent avian teratogen when injected directly into embryos during the first 96 h of development.

In mammals, the lethal dose of boron, as boric acid, varies according to species, and usually ranges between 210 and 603 mg B/kg body weight (BW); early developmental stages are especially sensitive. Excessive boron consumption adversely affects growth and reproduction in sensitive species of mammals (i.e., > 1,000 mg B/kg diet, > 15 mg B/kg BW daily, >1.0 mg B/L drinking water, or >3 g B/kg BW single dose on the first day of pregnancy). Boron is not considered essential for mammalian growth, but does protect against fluorosis and bone demineralization.

Terrestrial Plants

The role of boron in nutrition and toxicity of terrestrial crops has been reviewed extensively by Eaton (1944), EPA (1975), Gupta (1979, 1983), Gupta and Macleod (1982), Pilbeam and Kirkby (1983), and Gupta et al. (1985). It is generally agreed that boron is essential for the growth of higher plants and some species of fungi, bacteria, and algae, and that excess B is phytotoxic. It is also agreed that plants vary greatly in their sensitivity to B toxicity (Boyd and Walley 1972; EPA 1975; Birge and Black 1977; Goldberg and Glaubig 1986; Dear and Lipsett 1987). The exact mode of action of boron is unknown; however, its complexing ability facilitates the movement of sugars and other materials, and it is involved in cell wall bonding, conversion of glucose-l-phosphate to starch, and metabolism of nucleic acids (Sprague 1972; Gupta et al. 1985; Goldbach and Amberger 1986). Boron level in plants depends on the content and availability of soil boron, season, disease

state, inherent species or variety differences, and interactions with other substances (EPA 1975; Gestring and Soltanpour 1987). Most of the plant-available boron comes from the decomposition of soil organic matter and from boron adsorbed and precipitated onto soil surface particles; however, soil solution boron is the most important form, and plants take it up directly from this source (Gupta et al. 1985). Boron availability to plants is strongly associated with the hot-water-soluble fraction. This usually ranges from 0.4 to 4.7% of the total boron; the highest percentage occurs in fine-textured soils, and the lowest occurs in coarse-textured soils (Gupta and Macleod 1982). Uptake of boron by plants is about x 4 higher at pH 4 than at pH 9, highest in the temperature range 10 to 30°C, and higher with increased light intensity (Sprague 1972).

For the past 50 years, boron has been applied to B-deficient soils to improve crop yields of grains, fruits, vegetables, legumes, pine trees, tobacco, cotton, sunflowers, and peanuts (EPA 1975; Gupta 1979; Lipsett et al. 1979; Shorrocks and Nicholson 1980; Hopmans and Flinn 1984; Gupta and Cutcliffe 1985; Willett et al. 1985; Combrink and Davies 1987; Dear and Lipsett 1987; Mozafar 1987; Nuttall et al. 1987; Rerkasem et al. 1988). Boron is unique among the essential micronutrients because it is the only element normally present in soil solution as a nonionized molecule over the pH range suitable for plant growth (Gupta 1979). Boron deficiency in plants is widespread and has been reported in one or more crops in at least 43 States, almost all Canadian Provinces, and many countries (Gupta 1979). Boron deficiency in crops is more widespread than that of any other micronutrient (Gupta et al. 1985). It is more likely to occur in light-textured acid soils in humid regions because of boron's tendency to leach; however, deficiency may also occur in heavy-textured soils with high pH because boron is readily adsorbed under these conditions (Gupta et al. 1985). Deficiency signs include browning and spotting of leaves, chlorosis, abnormal thickening of cell walls, increased production of indoleacetic acid, accumulation of polyphenolic compounds, changes in membrane permeability, necrosis, and finally death (EPA 1975; Gupta 1979). Visible signs of deficiency in corn are accentuated by calcium deficiency, and are least evident when calcium is added to excess. Under conditions of boron and calcium deficiency combined, yields are low, and starch phosphorylase activity in corn leaves increases markedly, as does that of ribonuclease and polyphenol oxidase (Chatterjee et al. 1987). Interaction effects were also measured between B and potassium in alfalfa (Walker et al. 1987). Boron deficiency is usually corrected by application of 0.5 to 3 kg B/ha, depending on crop and formulation (Gupta 1979). Adding boron promotes the translocation rate of photosynthetic products and increases $C0_2$ incorporation into free amino acids (Gupta 1979).

Boron toxicity has been reported in many species of grasses, fruits, vegetables, grains, trees, and other terrestrial plants (Table 4; Gupta and Macleod 1982; Dye et al. 1983; Glaubig and Bingham 1985; Francois 1986; Nicholaichuk et al. 1988). Toxic levels generally do not occur on agricultural lands unless boron compounds have been added in excessive quantities, such as with fertilizer materials, irrigation water sewage sludge, or coal ash (Gupta and Macleod 1982; Gestring and Soltanpour 1987). Boron-contaminated irrigation water is one of the main causes of boron toxicity to plants. The continued use and concentration of boron in the soil due to evapotranspiration is the reason for eventual toxicity problems (Gupta et al. 1985). Borates have also been used as herbicides for complete kill of vegetation at application rates of 2,244 kg/ha (equivalent to 2,000 pounds/acre; Sprague 1972). Borates are frequently applied at elevated concentrations (i.e., >2 g/kg soil) in combination with organic pesticides to produce bacteriostatic effects; the resultant B-produced reduction in microbial degradation of the pesticide effectively extends the pesticide's biocidal properties (Sprague 1972). In some instances, cooling tower drift from geothermal steam containing boron may cause foliar boron toxicity near generating units (Glaubig and Bingham 1985; Sage et al. 1989).

Boron poisoning in plants is characterized by stunted growth, leaf malformation, browning and yellowing, chlorosis, necrosis, increased sensitivity to mildew, wilting, and inhibition of pollen germination and pollen tube growth (EPA 1975; Glaubig and Bingham 1985; Mitchell et al. 1987). In barley (*Hordeum vulgare*), for example, excess boron caused decreased growth and grain yield, elevated residues in leaves, and increased rate of leaf senescence (Table 4; Riley 1987). Barley grown on zinc-deficient soils tended to accumulate boron up to x 2.5 within 7 days; a similar pattern was evident for excess phosphorus (Graham et al. 1987). Thus, under conditions of marginally high boron in the rooting zone, low zinc, and high phosphorus, boron may accumulate to toxic levels in plants (Graham et al. 1987). Toxic effects in plants—including leaf injury—were observed in 26% of plants at or below substrate concentrations that resulted in greatest growth, indicating considerable overlap between injurious and beneficial effects of boron in plants (Eaton 1944). In general, deficiency effects in plants were evident when boron concentrations in soil solution were <2 mg/L; optimal growth occurred at 2 to 5 mg/L; and toxic effects were evident at 5 to 12 mg B/L (Gupta et al. 1985). However, considerable variation

exists in resistance to boron between species (Table 5). Sensitive species are known to include citrus, stone fruits, and nut trees; semitolerant species include cotton, tubers, cereals, grains, and olives; tolerant species usually include most vegetables (Gupta et al. 1985).

Table 4. Boron toxicity to some terrestrial plants.

Species, dose, and other variables	Effect	Reference[a]
Bigleaf maple, *Acer macrophyllum*		
0.9–5.4 mg B/L in saturated soil extracts	Reduced growth; >25% foliar damage; leaf residues of 76–324 mg B/kg ash weight (AW)	1
Madrone, *Arbutus menziesii*		
2.2–5.4 mg B/L in saturated soil extracts	Growth inhibition; >25% foliar damage; leaf residues of 216–540 mg B/kg AW	1
Beet, *Beta vulgaris*		
Soil B solutions		
5 mg/L	Optimal growth	2
15 mg/L	Injury evident	2
Broccoli, *Brassica oleracea italica*		
Grown in nutrient solutions containing		
0.08 mg B/L	Chlorophyll levels and net photosynthetic rates were significantly lower than those for plants grown in 0.41–0.81 mg B/L solutions	3
4.1 and 8.1 mg B/L	Leaf damage evident; lower chlorophyll levels and lower net photosynthetic rate than 0.4 and 0.8 mg B/L groups	3
Rhodes grass, *Chloris gayana*		
Grown in fly ash containing 3 mg hot-water-soluble B/L	Toxic. Residues >149 mg/kg dry weight (DW)	4
Lemon, *Citrus limonia osbeck*		
Soil B concentrations		
0.03–0.04 mg/L	Optimal growth	2
1 mg/L	Injury evident	2
Soybean, *Glycine max*		
Grown in soils amended with scrubber sludge residues (4.1 g B/kg) from coal-fired power plant for 2–3 years	Higher sludge B levels of 2 mg B/kg soil surface at year 1, and 1.2 mg B/kg at year 2 produced signs of B toxicity, including decreased growth and elevated residues in leaf (>83 mg/kg DW) and in seeds (>47 mg/kg DW)	5
Sunflower, *Helianthus annuus*		
50 mg B/L growth medium	Adversely affects phospholipid composition and synthesis in roots and microsomes from seedlings by inhibition of choline phosphotransferasse	6
10 mg B/L growth medium	Tolerated level	6

Barley, *Hordeum vulgare*		
Residues, in mg B/kg DW		
0.5–1.0 in soil	Residues of 46–100 mg/kg DW in leaves	7
30 in shoots	Damage to older leaves	8
50–70 in shoots	Reduction of 10% in DW of shoots	7
60–80 in leaf	Toxicity evident	8
80–120 in shoots	Toxic signs, but no yield reductions	8
120–130 in shoots	Grain yield reduced 10%	8
Alfalfa, *Madicago sativa*		
850–975 mg B/kg DW plant	Reduced yield	9
Rice, *Oryza sativa*		
Whole plant B residues		
38 mg/kg DW	No signs of toxicity	10
43–55 mg/kg DW	Signs of toxicity evident	10
Soil waters		
2.5–5 mg B/L	Toxic	10
French bean, *Phaseolus vulgaris*		
Grown in fly ash containing 3 mg hot-water-soluble B/L	Toxic. Residues >209 mg/kg DW	4
Residues in whole plant, in mg B/kg DW		
9–12	Slow flowering and pod formation; general yellowing of tips	11
>125	Reduced growth; burned older leaves dark brown	11
Digger pine, *Pinus sabiniana*, seedlings	Growth reduction; foliar damage >25%; needle	
13–17 mg B/L in saturated soil extracts	residues 1,242–1,512 mg B/kg AW	1
Pear, *Pyrus communis*		
82–164 kg B/ha applied to soil around pear trees in a nonirrigated orchard over a 6-year period	Toxicity observed during application and during 4 years postapplication. Toxicity was associated with residues, in mg B/kg DW, of 90–115 in blossom clusters and 45–55 in fruit. Within 5 years postapplication, soil B levels were <2 mg/kg, and all visible signs of toxicity had disappeared	12
Vegetation, various species		
2,244 kg borates/ha (2,000 lbs/acre)	Total kill of most species	2
Soil B concentrations		
1 mg/L	Optimal growth	2
5 mg/L	Injury evident	2
Plant residues		
>98 mg B/kg DW	Marginal burning and dark brown tips of older leaves	11

[a]1, Glaubig and Bingham 1985; 2, Sprague 1972, 3, Petracek and Sams 1987; 4, Aitken and Bell 1985; 5, Ransome and Dowdy 1987; 6, Belver and Donaire 1987; 7, Riley 1987; 8, Kluge and Podlesak 1985; 9, Gestring and Soltanpour 198; 10, Cayton 1985; 11, Gupta 1983; 12, Crandall et al. 1981.

Table 5. Boron concentrations in soil water associated with optimal growth and plant injury (from Sprague 1972).

Plant category	Boron concentration in soil water (mg/L) — Optimal growth	Boron concentration in soil water (mg/L) — Plant injury (usually)
Sensitive species	Trace to 1	1–5
Semitolerant species	Usually 1–5	5–15
Tolerant species	Usually 5–10	5–25

Table 6. Lethal and sublethal effects of boron on terrestrial invertebrates.

Organism, dose, and other variables	Effect	Reference[a]
Fruit fly, *Anastrepha ludens*		
Baits containing cottonseed hydrolysate and borax	Reduced infestation in oranges by 68%, and in mangoes by 98%	1
Honey bee, *Apis mellifera*		
8.7 mg B/L syrup (50 mg boric acid/L)	No effect on survival	2
17.5 mg B/L syrup (100 mg boric acid/L)	Fatal to about 50%	2
German cockroach, *Blattella germanica*		
Baits containing 25% boric acid plus honey	Population reduction of 50% in about 5 days, 80% in 4 weeks, and 98% in 6 to 9 months	3
Sugar diet containing		
11% boric acid	44% dead in 72 h	1
25% boric acid	79% dead in 72 h	1
50% boric acid	80% dead in 72 h	1
100% boric acid	91% dead in 72 h	1
Baits containing 20% boric acid	88% population reduction in 2 weeks; 92 to 95% reduction in 4 to 12 weeks	4
Gypsy moth, *Lymantria dispar*, larvae		
0.25% boric acid solution (436 mg B/L)	No effect on gypsy moth nucleopolyhedrosis virus (NPV)	5
0.5% boric acid	Enhanced NPV activity by x2	5
1% boric acid	Enhanced NPV activity by x11	5
Houseflies, *Musca domestica*		
250–5,000 mg B/kg diet, as boric acid	Inhibits reproduction	2
Isobornyl thiocyanoacetate		
27.3 µg/fly	LD50	1
Aerosols, >2%	50% knockdown in 6 min	1
American cockroach, *Periplaneta americana*		
Baits containing 1.5% boric acid	All dead in 6 days	6

Woodboring insects		
Common houseborer		
430 mg boric acid/m^3 wood	Adequate wood protection	2
Termites, 3 species		
>10,000 mg boric acid/m^3 wood	Required for wood protection	2

[a]1, EPA 1975; 2, Sprague 1972; 3, Gupta and Parrish 1984; 4, Wright and Dupree 1982; 5, Shapiro and Bell 1982; 6, Lizzio 1986.

Terrestrial Invertebrates

Relatively high concentrations of boron compounds are used to control fruit flies, cockroaches, gypsy moth larvae, houseflies, and woodboring insects (Table 6; Sprague 1972; EPA 1975). Boric acid is an effective stomach poison for several insect species (including German cockroaches [*Blattella germanica*], which are unable to detect the presence of boric acid (EPA 1975). Insect infestation of wood and other substrates can be prevented by pretreatment with boric acid or borax at doses of 0.25 to 0.55 kg/m^3 of wood (EPA 1975). Boric acid and other boron compounds are effective chemosterilants of the cotton boll weevil (*Anthonomus grandis*) and houseflies (EPA 1975).

Aquatic Organisms

Boron effects on aquatic plants are highly species-specific (Rao 1981; Table 7). Borate, like silicate, is an essential micronutrient for the growth of aquatic plants, such as diatoms, and it seems that a chemical combination of both nutrients in the form of silicoborate may be required by certain diatoms (Antia and Cheng 1975). In aquatic plants, boron affects nucleic acid metabolism, carbohydrate biosynthesis and transport, membrane integrity, and it interacts with growth substances (Frick 1985). Diatoms (*Cylindrotheca fusiformis*) cultured under B-deficient conditions stop dividing and swell in size despite increased photosynthetic rates. Boron-deficient diatoms accumulate rubidium, phenolic compounds, nitrates, and phosphates, and they show increased activity of various enzymes, especially glucose-6-phosphate dehydrogenase; however, respiratory adjustment is negligible until nutrient stress becomes irreversible in about 48 h (Smyth and Dugger 1980, 1981). Boron, under conditions of excess, alleviates nutrient deficiency in some phytoplankters and may cause temporal variations of phytoplankton composition in coastal waters (Rao 1981). Phytoplankton can tolerate up to 10 mg inorganic B/L in the absence of stress from pH adversity and nutrient deficiency, although higher borate concentrations up to 100 mg/L are expected to cause species redistribution by favoring the growth of some species and suppressing that of others (Table 7; Antia and Cheng 1975).

Data are limited for aquatic invertebrates and boron, although those data available suggest that the no-observable-effect levels were 13.6 mg B/L for freshwater organisms and 37 mg B/L for marine biota (Table 7). Juvenile Pacific oysters (*Crassostrea gigas*) accumulated boron in relation to availability, but showed no prolonged retention following cessation of exposure (Thompson et al. 1976). At current industrial discharge levels of about 1.0 mg B/L, no hazard is clear to oysters and aquatic vertebrates (Thompson et al. 1976).

The most sensitive aquatic vertebrates tested for which data are available were coho salmon (*Oncorhynchus kisutch*), with an LC50 (16-day) value of 12 mg B/L in seawater, and sockeye salmon (*O. nerka*), showing elevated tissue residues after exposure for 3 weeks in seawater containing 10 mg B/L (Table 7). Boron concentrations between 0.001 and 0.1 mg/L had little effect on survival of rainbow trout embryos after exposure for 28 days (Table 7). These low levels may represent a reduction in reproductive potential of rainbow trout, and >0.2 mg B/L may impair survival of other fish species, according to Birge and Black (1977); however, additional data are needed to verify these speculations. Birge and Black (1977) reported that concentrations of 100 to 300 mg B/L killed all species of aquatic vertebrates tested, that embryonic mortality and teratogenesis were greater in hard water than in soft water, but that larval mortality of fish and amphibians was higher in soft water than in hard water, and that boron compounds were more toxic to embryos and larvae than to adults. Moreover, they found no measurable effect of boron toxicity to aquatic vertebrates in water temperature in the range of 13 to 29°C, dissolved oxygen between 6.4 and 10.3 mg/L, and pH between 7.5 and 8.5.

Table 7. Lethal and sublethal effects of boron on aquatic organisms.

Taxonomic group, organism, compound, dose, and other variables	Effect	Reference[a]
Aquatic plants		
Blue green alga, *Anacystis nidulans*, boric acid, H_3BO_3		
0.01–4.0 mg B/L	Grows well in B-deficient media; growth neither stimulated nor inhibited at higher levels	1, 15
50 mg B/L	No effect on growth or organic constituents	2
75–100 mg B/L	Growth and chlorophyll content reduced; at 72 h, photosynthetic pigments depleted	2
100 mg B/L	Decrease in protein content causing inhibition in nitrate uptake and nitrate reductase activity. Decreased chlorophyll content and photosynthesis inhibition within 72 h	2, 15
Green alga, *Chlorella pyrenoidosa*, boric acid		
10 mg B/L	No effect on growth or cell composition. Bioconcentration factor (BCF) of x4 after 7 days	3
50 mg B/L	BCF of x5 after 7 days	3
50–100 mg B/L	Altered cell division and amino acid activity after 72 h; reversible photosynthesis inhibition. Giant cells formed with increased nitrate and protein	4
100 mg B/L	BCF of x4.8 after 7 days	3
>100 mg B/L	Totally inhibitory for cell division and biomass synthesis in 72 h	4
Duckweed, *Lemna minor*, boric acid		
Control media, 10–20 mg B/L, pH 5.0	Normal growth	5
100 mg B/L, pH 5.0	Growth inhibited; recovery on transfer to control media	5
20 mg B/L, pH 4.0	Residues of 93 mg B/kg fresh weight (FW) v. 63 in controls	5
20 mg B/L, pH 7.0	Growth inhibited. Residues of 257 mg/kg FW v. 49 in controls	5
Marine algae, 19 species, boric acid		
5–10 mg B/L	No inhibitory effect on growth rate in 60 days; stimulatory to some species	6
10–50 mg B/L	Prolonged survival of peak populations of certain diatoms after growth cessation: *Bellerochea polymorpha* at 10 mg B/L, *Skeletonema costatum* at 50 mg B/L	6
50 mg B/L	Growth inhibition in 26% of species tested; adaptation and recovery by most species	6
100 mg B/L	Growth inhibition in 12 of 19 species tested; 8 species did not recover and died	6
Marine phytoplankton		
30 mg B/L, high nitrates, phosphates, silicates, and low temperatures	Increased primary production and carbon assimilation	7
30 mg B/L, low nutrients, high temperatures	Photosynthesis inhibited up to 62%	7

30 mg B/L, unialgal cultures, 5-days-old	Photosynthesis inhibition	7
As above, 14-days-old	Enhanced photosynthesis in certain species	7
Invertebrates		
Sea urchin, *Anthocidaris crassispina*, embryos, boric acid		
37 mg B/L	Normal development	8
75 mg B/L	Fatal	8
Cladoceran, *Daphnia magna*, boric acid		
6.4 mg B/L	Highest concentration tested in 21-day exposure producing no measurable effect	9, 10
13.6 mg B/L	Lowest concentration tested in 21-day exposure causing reduction in number of broods, total young produced, mean brood size, and mean size	9, 10
27 mg B/L	LC14 (21 days)	10
53 mg B/L	LC50 (21 days)	10
54–200 mg B/L	No deaths (48 h)	9, 10
106 mg B/L	LC100 (21 days)	10
115–246 mg B/L	LC50 (48 h)	9, 10
420 mg B/L	LC100 (48 h)	9
Mosquito larvae, 3 species, boric acid, mg/L		
250 (43.7 mg B/L)	LC97-LC99 through hatching	11
4,000 (700 mg B/L)	LC100 (48 h), freshly hatched	11
3,000 (524 mg B/L)	LC100 (48 h), second instar	11
10,000 (1,748 mg B/L)	LC100 (48 h), third instar	11
16,000 (2,797 mg B/L)	LC100 (48 h), pupae	11
Vertebrates		
Fowler's toad, *Bufo fowleri*, embryos, through day 4 posthatch		
Boric acid		
Soft water, 50 mg $CaCO_3$/L		
25 mg B/L	LC1 (7.5 days)	12
145 mg B/L	LC50 (7.5 days)	12
Hard water, 200 mg $CaCO_3$/L		
5 mg B/L	LC1 (7.5 days)	12
123 mg B/L	LC50 (7.5 days)	12
Toad, *Bufo vulgaris*, embryos		
874 mg B/L, as boric acid. Exposure for 24 h from 2-cell stage to tailbud stage	Malformations included edema, microcephalia, short tail, and suppressed forebrain development	11
Goldfish, *Carassius auratus*, embryos, through day 4 posthatch		
Boric acid		
Soft water		
0.6 mg B/L	LC1 (7 days)	12
46 mg B/L	LC50 (7 days)	12
Hard water		
0.2 mg B/L	LC1 (7 days)	12
75 mg B/L	LC50 (7 days)	12
Borax, $Na_2B_4O_7 \cdot 10H_2O$		
Soft water		
0.5 mg B/L	LC1 (7 days)	12

65 mg B/L	LC50 (7 days)	12
Hard water		
0.9 mg B/L	LC1 (7 days)	12
59 mg B/L	LC50 (7 days)	12
Mosquitofish, *Gambusia affinis*, adults		
Boric acid		
5,600 mg/L (979 mg B/L)	LC50 (96 h)	12
Sodium borate		
3,600 mg/L	LC50 (96 h)	12
Channel catfish, *Ictalurus punctatus*, embryos, through day 4 posthatch		
Boric acid		
Soft water		
0.5 mg B/L	LC1 (9 days)	12
155 mg B/L	LC50 (9 days)	12
Hard water		
0.2 mg B/L	LC1 (9 days)	12
22 mg B/L	LC50 (9 days)	12
Borax		
Soft water		
5.5 mg B/L	LC1 (9 days)	12
155 mg B/L	LC50 (9 days)	12
Hard water		
1.7 mg B/L	LC1 (9 days)	12
71 mg B/L	LC50 (9 days)	12
Bluegill, *Lepomis macrochirus*		
Boron trifluoride, BF_3		
15,000 mg B/L	LC50 (24 h)	12
Dab, *Limnada limnada*		
74.0 mg B/L	LC50 (96 h)	13
88.3 mg B/L	LC50 (24 h)	13
Coho salmon, *Oncorhynchus kisutch*, underyearlings		
12 mg B/L	LC50 (283–384 h), seawater	14
113 mg B/L	LC50 (283–552 h), fresh water	14
Rainbow trout, *Oncorhynchus mykiss*, embryos, through day 4 posthatch		
Boric acid		
Soft water		
0.1 mg B/L	LC1 (28 days)	12
100 mg B/L	LC50 (28 days)	12
Hard water		
0.001 mg B/L	LC1 (28 days)	12
79 mg B/L	LC50 (28 days)	12
Borax		
Soft water		
0.07 mg B/L	LC1 (28 days)	12
27 mg B/L	LC50 (28 days)	12
Hard water		
0.07 mg B/L	LC1 (28 days)	12
54 mg B/L	LC50 (28 days)	12
Adults		
339 mg B/L	LC50 (48 h)	10, 12, 16
350 mg B/L	No effect in 30 min	16
3,500 mg B/L	All alive after 30 min, but in obvious distress	16
14,000 mg B/L	After exposure for 30 min, all recovered if	16

	placed in flowing B-free water	
Sockeye salmon, *Oncorhynchus nerka*		
10 mg B/L, exposure in seawater for 3 weeks	Maximum residues, in mg/kg FW, were 17 in bone, 12 in kidney, 10 in gill, 9 in liver, and 8 in muscle. Maximum control values were always <1.0, except bone, which was 4.4 mg/kg FW	14
Leopard frog, *Rana pipiens*, embryos, through day 4 posthatch		
Boric acid		
Soft water		
13 mg B/L	LC1 (7.5 days)	12
130 mg B/L	LC50 (7.5 days)	12
Hard water		
22 mg B/L	LC1 (7.5 days)	12
135 mg B/L	LC50 (7.5 days)	12
Borax		
Soft water		
5 mg B/L	LC1 (7.5 days)	12
47 mg B/L	LC50 (7.5 days)	12
Hard water		
3 mg B/L	LC1 (7.5 days)	12
54 mg B/L	LC50 (7.5 days)	12

[a]1, Martinez et al. 1986b; 2, Martinez et al. 1986a; 3, Fernandez et al. 1984; 4, Maeso et al. 1985; 5, Frick 1985; 6, Antia and Cheng 1975; 7, Rao 1981; 8, Kobayashi 1971; 9, Gerisch 1984; 10, Lewis and Valentine 1981; 11, EPA 1974; 12, Birge and Black 1977; 13, Taylor et al. 1985; 14, Thompson et al. 1976; 15, Mateo et al 1987; 16, Sprague 1972.

Birds

Boron is a potent teratogen to domestic chicken embryos when injected into eggs. Injection of boron into the yolk sac of chicken embryos during the first 96 h of development with 1.0 to 2.5 mg of boric acid--equivalent to 3.2 to 8.0 mg B/kg FW egg (55 g egg)--produced a wide range of developmental abnormalities (Table 8). Several compounds are known to counteract B-induced avian developmental abnormalities, or to reduce the frequency of malformations, although the mode of action is unclear. These compounds include sodium pyruvate, to counteract rumplessness (Landauer 1952); nicotinamide, to decrease frequency of facial defects (Landauer 1952) and melanin formation (Landauer 1953c); and riboflavin, which greatly reduced the teratogenic effects of boric acid (Landauer 1952, 1953a, 1953b; Landauer and Clark 1964). Other polyhydroxy compounds, such as D-ribose, pyridoxine hydrochloride, and D-sorbitol hydrate, also reduced or abolished boric acid-induced teratogencity in chick embryos (Landauer 1953b).

High concentrations of boron have been found in the San Joaquin Valley of California in irrigation drainwater and in aquatic plants consumed by waterfowl. Measured boron concentrations in that locale exceeded 20 mg/L in subsurface agricultural drainage waters, 400 mg/kg DW in widgeongrass (*Ruppia maritime*) and algae, 150 mg/kg DW in aquatic insects, 1,860 mg/kg DW in some aquatic plants, and up to 3,390 mg/kg DW in seeds consumed by waterfowl (Schuler 1987; Klasing and Pilch 1988; Smith and Anders 1989; Hoffman et al. 1990). At present, only selenium has been implicated as the cause of abnormal development among waterfowl in western areas affected by irrigation drainwaters (Ohlendorf et al. 1986; Hoffman et al. 1988, 1990). However, recent studies by Smith and Anders (1989) and Hoffman et al. (1990) with mallards demonstrate that dietary boron concentrations well below levels that can occur in the environment represent a toxicological hazard that has not been considered in the management of agricultural drainwater. For example, dietary concentrations of 300 to 400 mg B/kg of feed FW--substantially lower than boron levels reported near some western wildlife refuges contaminated by agricultural drainwater--adversely affect mallard growth, behavior, and brain biochemistry and are often associated with elevated tissue boron levels (Table 8). Dietary levels of 100 mg B/kg FW resulted in reduced growth of female mallard ducklings (Hoffman et al. 1990), and diets containing as little

as 30 mg B/kg FW fed to mallard adults adversely affected growth rate of their ducklings (Smith and Anders 1989). Resource managers must now consider boron, as well as selenium, and their possible interactions, as a toxic hazard to wildlife populations throughout areas of the western United States (Smith and Anders 1989).

Table 8. Lethal and sublethal effects of boron on birds.

Species, dose, and other variables	Effect	Reference[a]
Domestic chicken, *Gallus domesticus*		
Embryo, yolk injection		
Boric acid		
0.01 mg B/kg body weight (BW)	LD1	1
1.0 mg B/kg BW	LD50	1
1.0 mg at 28 h of development	Developmental abnormalities	2
2.0 mg at 28 h of development	Malformations of nervous system, eyes, and spinal cord	3
2.5 mg at 24 h of development	Rumplessness	7
2.5 mg at 84 h of development	Skeletal deformities, cleft palate, missing toes, eye deformities	4, 5, 6
15.8 mg B/kg egg at 96 h of development	LD50 (96 h). Most (70 to 85%) of the survivors at age 18 days had edema, inhibited feather growth, pale body coloration, and reduced BW	10
Borax		
0.01 mg B/kg BW	LD1	1
0.5 mg B/kg BW	LD50	1
Adult		
875 mg B/kg diet, as boric acid, for 6 days	Egg production ceased; production normal 14 days after B withdrawn	1
Mallard, *Anas platyrhynchos*		
Adults fed diets containing various concentrations of B, as boric acid, for 3 weeks, then mated. Resultant ducklings continued on same diets for 21 days. Data collected on reproduction, survival, residues, and histopathology when ducklings were age 21 days		
8 mg B/kg diet fresh weight (FW) (controls). Diets contained about 10% moisture	Boron residues in egg, liver, and brain of adults and ducklings were always <3 mg B/kg dry weight (DW)	8
30 mg B/kg diet FW	Duckling weight gain reduced compared to controls. Residues in egg and duckling liver and brain about 3–4 mg B/kg DW; residues <3 in adult liver and brain	8
300 mg B/kg FW	Duckling BW at hatch significantly lower than controls; duckling weight gain reduced. Mean residues, in mg B/kg DW, were 13 in egg, 15 in adult liver (Max. 24), 17 in duckling liver (Max. 36), 14 in adult brain (Max. 24), and 19 in duckling brain (Max. 44)	8
1,000 mg B/kg diet FW	No observable effect on adults. No effect on egg fertility or shell thickness. Significantly reduced hatching success; duckling mortality through age 7 days significantly greater than controls; body weight lower. Total number of 21-day-old	8

	ducklings produced per female, and brain to BW ratios were significantly higher than controls. Mean B residues, in mg/kg DW, were 49 in egg, 33 in adult liver (Max. 74), 51 in duckling liver (Max. 89), 41 in adult brain (Max. 89), and 66 in duckling brain (Max. 110). No histopathology evident in liver, brain, kidney, or heart	
Ducklings, age 1 day, 2-week dietary exposure		
1,000 mg B/kg FW diet, as boric acid	Adverse effects on growth	9
5,000 mg B/kg diet, as boric acid	Some deaths	9
Ducklings, age 1 day, 10-week dietary exposure to boric acid		
Controls, 13 mg B/kg FW diet. Diets contained 12–14% moisture	Brain B concentration of 2 mg/kg DW	9
100 mg B/kg FW diet	Delayed growth of females, plasma triglyceride levels evaluated, abnormal liver metabolism, brain residue of 4 mg/kg DW	
400 mg B/kg FW diet	Delayed growth of females, plasma triglyceride elevated, brain B residue of 5 mg/kg DW, decrease in brain ATP, altered duckling behavior in bathing and resting	9
1,600 mg B/kg FW diet	Some deaths (10%), delayed growth, decreased food consumption, plasma triglyceride elevated, brain B residue of 51 mg/kg DW (Max. 99), decrease in brain calcium and ATP, reduction in time spent bathing and standing, increase in time spent resting, increased serum calcium, lower hematocrit and hemoglobin; no histopathology of brain, liver, or kidney	9

[a]1, Birge and Black 1977; 2, Schowing and Cuevas 1975; 3, Schowing et al. 1976; 4, Landauer 1953a; 5, Landauer 1953b; 6, Landauer 1953c; 7, Landauer 1952; 8, Smith and Anders 1989; 9, Hoffman et al. 1990; 10, Ridgeway and Karnofsky 1952.

Mammals

Epidemics and sporadic cases of oral intoxication in humans are often due to inadvertent addition of boric acid to infant formulas (Siegel and Wason 1986). Pacifiers and some other products containing boron compounds have been sold in Ireland despite a recommendation from the Pharmaceutical Society of Great Britain that these products should not be sold because of hazards to infants (O'Sullivan and Taylor 1983). Fatal cases of boron poisoning have involved misuse of boron compounds in hospitals, either from accidental substitution of boric acid solution for water in infant formula or from accidental use of boric acid as a diapering powder (EPA 1975). In an adult fatality, the victim died after inundation by borax solution (EPA 1975). In one case, a 12-month-old girl developed violent vomiting, coughing, irritability, tremors, seizures, and a delirious reaction after accidentally swallowing a mixture containing 3 g of boric acid and 300 mg of cinchocaine chloride prescribed for a painful dental protrusion (Egfjord et al. 1988). Her plasma boric acid level 6 h later was 26 mg/L; the half-time persistence (Tb l/2) for boric acid in plasma is about 7 h (Egfjord et al. 1988). The lethal dose of boric acid varies according to the species. In mammals it ranges from 210 to 603 mg B/kg BW, and death is due to central nervous system paralysis and gastrointestinal irritation (Table 9; NAS 1980). Human newborns are especially sensitive, and accidental deaths have been recorded at doses between 50 and 140 mg B/kg BW (Table 9).

Table 9. Lethal and sublethal effects of boron on mammals.

Organism, route of administration, dose, and other variables	Effect	Reference[a]
Cattle, *Bos* spp.		
Drinking water		
Supplemented with 15, 30, 60, or 120 mg B/L for 10 days	Boron levels in plasma rose from 2.7 mg/L in controls to 4.4 (15 mg/L group), 5.3 (30 mg/L group), 8.3 (60 mg/L group), and 13.4 mg/L in the 120 mg B/L drinking water supplement	5
29 mg B/L, and higher	When given a choice, cattle preferred tap water to drinking water supplemented with B compounds	1
120 mg B/L, as borax, for 10 days	No effect on feed or water consumption; no overt signs of toxicosis	2
150 mg B/L, as borax, for 30 days equivalent to 15.3 mg B/kg body weight (BW) daily	Decreased feed consumption, weight loss, edema, inflammation of legs, daily elevated plasma B levels of 1.2 mg/L v. 0.5 in controls, abnormal blood chemistry	1, 2, 3, 4, 5
Diet		
Consumed feed containing 157 mg B/kg, as borax, for 42 days	No adverse effects	3
Fed 2 to 2.5 g of boron daily as borax, for 40 days	No observable adverse effects; all B excreted, mostly in urine	6
Fed 20 g of borax daily	Milk B residues increased from <1.0 mg/L to >3 mg/L	5
Ingested total dose of 100–300 g of boron equivalent to 200–600 mg B/kg BW	Toxic dose	7
Found dead after consuming 1 kg of borax, or about 250 g of B	Residues in mg B/kg fresh weight (FW) were 1,300 in ruminal fluids, 1,900 in abomasal fluids, 24 in liver, 19 in rumen, and 21 in abomasums	7
Dog, *Canis familiaris*		
Diet		
350 mg B/kg feed, 2 years	Tolerated	8
1,540 mg borax/kg or 3,000 mg boric acid/kg, chronic study (174–524 mg B/kg diet)	No adverse effects	6, 9
1,170 mg B/kg, 38 weeks	Testicular degeneration, spermatogenesis cessation	5, 8
Inhalation		
92 mg pentaborane/m^3 for 15 min	LC50	9
Guinea pig, *Cavia* sp.		
Inhalation		
0.018 mg decaborane/m^3, 6 h daily, 5–6 exposures	Eye inflammation, listlessness, emaciation, convulsions	3
Human, *Homo sapiens*		
Dermal		
7-month-old infant treated for dermatitis with 3% boric acid powder	Fatal. Boron concentrations elevated in bile, intestinal contents, and spleen	9

Adult administered about 645 g of boric acid dermally	Toxicosis observed	
Inhalation		
Borax dust, 1.1–14.4 mg/m^3, occupational exposure for at least 5 years	At 14.4 mg/m^3, 33% of workers noted dryness of mouth, nose, or throat; 28% had eye irritation problems; 15% had nosebleeds and cough; 13% had sore throat or shortness of breath and ches tightness. At 4.0 and 1.1 mg/m^3, no symptoms except eye irritation were noted by more than 5 and 3% of exposed participants	11
Boranes, various	Pulmonary irritation, headache, nausea, fatigue, muscular weakness, liver and kidney pathology	3
Oral		
3 mg B daily for 119 days in diet containing 0.25 mg B	Reduction in urinary excretion of calcium and magnesium by postmenopausal women	12
20 mg B daily	Normal adult intake	6
Solutions >88 mg B/L or >500 mg boric acid/L	Fatal to infants	13
1–3 g boric acid, or 0.3–0.8 g/kg BW	Lethal to newborns	14
2–4.5 g boric acid or 0.5–1.2 g/kg BW	Nonfatal to infants, but serum levels elevated from 20–150 mg borate/L	14
>3.5 g boric acid daily	Probably harmful or lethal to infants and newborns	10
4 g boric acid or borates daily	No toxicosis in adults	6, 9
4.5–15 g of boric acid, equivalent to 1.25–4.2 g/kg BW, in accidentally contaminated formula in newborn nursery	Death preceded by severe symptomology; serum levels of 400–1,000 mg borate/L	13, 14
5–6 of borates, or 0.7 g/kg BW	Fatal to infants	14, 15
15–20 g of boric acid, equivalent to 0.25–0.3 g/kg BW	Fatal to adults	9, 14, 15
Infants, age 6–16 weeks		
Given pacifiers dipped in a proprietary borax (107 g/L) and honey compound. Dose during 1-month-exposure period estimated at 3–9 g borax	Some developed seizure disorders characterized by vomiting, loose stools, irritability, diarrhea; elevated blood B values of 2.6–8.5 mg B/L v. <0.6 in controls. When preparation witheld, seizures stopped and children remained well for at least 5 years	13
Injections, intravenous		
Adult males, age 22–28 years, given single infusion of 562–611 mg boric acid equivalent to 8.0–8.7 mg B/kg BW	Boric acid half-time persistence was 21 h. Most was excreted in urine 24 h, 94% in 96 h, and ~99% in 120 h; plasma boric acid concentration after infusion was about 16 mg/L v. 0.5 at start; no discomfort during or after infusion	16
Adults given dose of 20 g boric acid	No permanent adverse effects	9
Monkey, *Macaca* sp.		
Inhalation		
Pentaborane, 640 mg/m^3, 2 min	LC50	9
Intraperitoneal injection		
Decaborane, 1 mg/kg BW daily, multiple	Altered brain wave activity	9

injections		
Decaborane, 6 mg/kg BW, single injection	LC50	9
Mice, *Mus* sp.		
Drinking water		
5 mg B/L, lifetime exposure	No effect on growth, longevity, or tumor incidence	2, 5
Ingestion		
3 g B/kg BW, first day of pregnancy	94% of embryos failed to develop past blastocyst stage v. 9% in controls	9
Diet		
1,500 mg boric acid/kg (262 mg B/kg)	All dead within 10 days	17
Injection, intravenous		
1.32 g sodium borate/kg BW, single dose	LD50	18
Injection, intraperitoneal		
25.2 mg decaborane/kg BW, single dose	LD50	9
44.7 mg decaborane/kg BW, single dose, prior treatment for 8 days at 250 mg/kg BW with pyridoxine hydrochloride	LD50	9
2,817 mg sodium borate/kg BW, single dose	LD50	18
Inhalation		
Pentaborane		
0.011 mg/m^3 for 4 h	LC50	3
50 mg/m^3, 15 min	LC50	9
342 mg/m^3, 2 min	LC50	9
1,034 mg/m^3, 30 s	LC50	9
Rabbit, *Oryctolagus* sp.		
Diet		
Equivalent to 800–1,000 mg borates/kg BW daily for 4 days	Growth retardation	18
Intragastric route		
Daily dose of 100 mg calcium borate 4 months	Altered serum chemistry	9
Intravenous injection		
Single dose of 800–900 mg boric acid/kg BW	LD50	18
Intraperitoneal injection		
30 mg decaborane/kg BW	Death within 24 h	3
Dermal		
25–200 mg boric acid/kg BW daily	Nonirritative and nontoxic when applied to intact skin	18
Sodium borate solutions of 50,000 or 100,000 mg borates/L applied to skin	Mildly or moderately irritating	18
Boron oxide dust	Application to skin produced erythema that lasted 2–3 days; instillation in eyes produced immediate conjunctivitis as a result of exothermic hydration of boron oxide to boric acid	19
Inhalation		
120–150 mg calcium borate/m^3, 2 h daily, 10-week exposure	Respiratory tract pathology, growth inhibition, enlarged liver	9
Rat, *Rattus* sp.		

Drinking water		
Free access for 90 days to drinking water containing 0.3, 1.0, or 6.0 mg B/L	Rats refused to drink water at 1.0 or 6.0 mg/L	15
0.3 mg boric acid/L for 6 months	No effect on gonadotoxicity	20
1.0 mg boric acid/L for 6 months, equivalent to 0.05 mg B/kg BW daily	Decreased spermatozoid count, reduction in spermatozoid activity	20
6 mg B/L, 90 days	No toxic effect on male reproductive system, blood chemistry, or growth	5, 15
6 mg B/L for 6 months, equivalent to 0.3 mg B/kg BW daily	Gonadotoxicity in male rats; altered enzyme activity levels in blood and liver	20, 21
75 mg B/L, as borax, for 45 days	No effect on growth or reproduction	3
100 mg B/L for 21 days	Tissue B levels in kidney, liver, brain, and blood increased for first 9 days but returned to normal by day 21 except for blood, which continued to rise	
150 mg/L for 70 days, or 170 mg B/L for 25 days	Slight reduction in growth rate	
>150 mg B/L for extended periods	Adverse effects probable	5
300 mg B/L for 49–70 days	Growth rate reduced 21% but no change in food consumption; coarse coat; atrophied scrotal sacs	4
440 mg B/L for 25 days	Growth inhibition	4
3 g sodium tetraborate/L for 10–14 weeks	Increase in activity of cerebral succinic dehydrogenase, brain acid proteinase, and in brain RNA concentration; decrease in liver cytochrome P-450 activity	22
Diet		
0.09–1.71 mg boric acid/kg BW daily for 6 months (0.015–0.3 mg B/kg BW daily)	Adverse changes in testes	18
350 or 525 mg B/kg diet, as borax or boric acid, for 2 years	No observable adverse effects on fertility, lactation, litter size, weight, or appearance	6
500, 1,000, or 2,000 mg B/kg diet, as borax, for 30–60 days, equivalent to 12, 25, or 50 mg B ingested daily	No adverse effects at 500 mg B/kg diet for 60 days. At 1,000 and 2,000 mg B/kg, adverse effects measured on male reproductive capacity, including germinal aplasia and infertility; effects persisted for at least 8 months following B exposure at highest dose	23
525 mg B/kg diet for 90 days	Tolerated	8
1,000 mg boric acid or borax/kg BW daily	Weight loss after 1 week on borax diet or 2 weeks on boric acid diet; toxic signs after 3 weeks on both diets	24
1,050 mg B/kg diet, as borax or boric acid, for 2 years	Testicular degeneration	6
1,060 mg B/kg diet, as sodium borate, chronic exposure	Growth retardation and testicular atrophy	18
1,170 mg B/kg diet, 2 months	Coarse coat, scaly tails, hunched position, bloody discharge from eyes, depressed hemoglobin and hematocrit	5
1,170 mg B/kg diet, as borax or boric acid 2 years	Sterility in males and females	6, 8
1,750 mg B/kg diet, 25 days	Reduction of 50% in growth rate	4
1,750 mg B/kg diet, as sodium borate, chronic	Severe testicular atrophy	18

Oral, single dose		
450 mg B/kg BW	No effect on male fertility	15
510–690 mg B/kg BW, as borax	LD50	8, 9, 24
550–710 mg B/kg BW, as boric acid	LD50	8, 9, 24
600 mg B/kg BW	LD50	2
3.45–5.14 g sodium borate/kg BW	LD50	18
5.1 g boric acid/kg BW	LD50	6
6.1 g borax/kg BW	LD50	6
Injection subcutaneous		
1.4 g boric acid/kg BW	LD50	18
Injection, intravenous		
5–75 mg boric acid/kg BW	Slight reduction in arterial blood pressure	21
Injection, intraperitoneal		
42 mg sodium borate/kg BW, single injection	Tissue residues after 30 min, in mg B/kg FW, were 25 in blood, 30 in liver, and 50 in kidney v. <5 in all control tissues. After 3 months, residues were 20 mg B/kg FW in brain, 45 in heart, 60 in liver, and 75 in kidney	21
Inhalation, boron trifluoride		
2, 6, or 17 mg BF_3/m^3, 6 h daily, 5 days weekly, 13 weeks	At 17 mg/m^3, altered proximal tubluar epithelium of kidney and abnormal serum chemistry. At 6 mg/m^3, elevated fluoride levels in urine, serum, and bone, but no toxic response. No difference from controls at 2 mg/m^3	26
24 or 66 mg/m^3, 6 h daily, 9 days	Clinical signs of respiratory irritation, nasal discharge, weight loss, increased lung weight, depressed liver weight, kidney pathology at 66 but not 24 mg/m^3	26
55 mg/m^3, 4–7 h daily, 5 days weekly, 6 weeks	Some deaths in rats and other rodent species tested, but no deaths in nonrodent species	26
180 mg/m^3, 6 h daily, consecutive days	All dead prior to sixth exposure	26
259 mg/m^3, 4–7 h daily, 2 days	All dead. Mortality was lower for guinea pigs, dogs, rabbits, mice, and cats. Lung and kidney damage in all species	26
1,210 mg/m^3, 4 h	50% dead	26
Inhalation, boron oxide		
470 mg/m^3, 10 weeks	Reddish exudates from nose, but no deaths or signs of lung damage	19
470 mg/m^3, 24 weeks	No signs of toxicosis	9
Inhalation, decaborane		
20 mg/m^3, 6 h daily, 5 days weekly	Tremors, convulsions, nervousness, restlessness, weight loss, belligerency	3
36 mg/m^3, 4 h	LC50	3
Inhalation, pentaborane		
3 mg/m^3, 6 h daily, 5 days weekly	Extreme belligerency, tremors, weight loss	3
18 mg/m^3, 4 h	LC50	3

[a]1, Green and Weeth 1977; 2, Weeth et al. 1981; 3, NAS 1980; 4, Seal and Weeth 1980; 5, Nielsen 1986; 6, Sprague 1972; 7, Brockman et al. 1985; 8, Weir and Fisher 1972; 9, EPA 1975; 10, Gupta and Parrish 1984; 11, Garabrant et al. 1985; 12, Nielsen et al. 1987; 13, O'Sullivan and Taylor 1983; 14, Siegel and Wasson 1986; 15,

Dixon et al. 1976; 16, Jansen et al. 1984; 17, Lizzio 1986; 18, Anonymous 1983; 19, Garabrant et al. 1984; 20, Krasovskii et al. 1976; 21, Magour et al. 1982; 22, Settimi et al. 1982; 23, Lee et al. 1978; 24, Dani et al. 1971; 25, Benson et al. 1984; 26; Rusch et al. 1986.

Table 10. Proposed boron criteria for the protection of natural resources and human health.

Resource and other variables	Criterion	Reference[a]
Crops		
Irrigation waters		
Sensitive crops	0.3–1.25 mg B/L	1, 2, 3
Semitolerant crops	0.67–2.5 mg B/L	1, 2, 3
Tolerant crops	1–4 mg B/L	1, 2, 3
Maximum safe concentration	4 mg B/L	2
Residues in crops		
Boron deficiency	<15 mg B/kg dry weight (DW) plant	4, 5
Toxicosis	>200 mg B/kg DW plant	4, 5
Aquatic organisms		
Nonhazardous levels in water		
Fish, oysters	1 mg B/L	6
Aquatic plants	4 mg B/L	2
Fish	5 mg B/L	2
"Safe" levels in water		
Largemouth bass, *Micropterus salmoides*	<30 mg B/L	1
Bluegill, *Lepomis macrochirus*	<33 mg B/L	1, 7
Adverse effects, sensitive species	10–12 mg/L	6
Waterfowl		
Diet		
No observed adverse effect	<13 mg/kg fresh weight (FW)	17
Adverse effects	30–100 mg/kg FW	17, 18
Fatal	1,000 mg/kg FW	18
Livestock		
Diet		
Boron deficiency	<0.4 mg B/kg DW	5
Toxic signs probable	>100 mg B/kg DW	5
Maximum tolerable level, as borax	150 mg B/kg DW	4, 5
Total dose, toxic	100–300 g of B (equivalent to 200–600 mg B/kg body weight)	9
Drinking water		
Maximum allowable	5 mg B/L	4, 8, 10, 11
Maximum tolerated	40 mg B/L	10
"Safe"	40–150 mg B/L	11
Adverse effects	>150 mg B/L	5
Pesticide applications		
Boric acid, 99% powder	Effective for control of household cockroaches, ants, and fleas	2
Boric acid, 8% solution	Fungicide for vegetables, fruits, and trees	12
Human health		
Daily intake		
Worldwide	Range 0.3–41 mg B, means usually 10–20 mg B	4, 5, 13
Finland	1.7 mg B	5
England	2.8 mg B	5
United States	3 mg B	4

No effect level	4 g boric acid	14
Adverse effect level		
Chronic intoxication	4–5 g boric acid	14
Lethal to infants and small children	5–6 g boric acid	14
Lethal to adults	18–20 g boric acid, single dose	14
Drinking water		
Recommended	<0.3 mg B/L	15
USSR	<0.5 mg B/L	10
United States	<1.0 mg B/L	4, 11
"Safe"	<20 mg B/L	2, 10
No toxic effects	20–30 mg B/L	2
Dermal, ocular		
Sodium borate and boric acid	Safe as cosmetic ingredients at <5% concentrations; not recommended on infant skin or injured skin	12, 16
Air, Threshold Limit Value (8 h daily, 5 days weekly)		
Pentaborane	0.01 mg/L	4
Dioborane	0.1 mg/L	4
Decaborane	0.5 mg/L	4
Sodium borate	1–5 mg/m^3	16, 17
Boron trifluoride	3 mg/m^3	3
Calcium borate	4–6 mg/m^3	3
Boron tribromide	10 mg/m^3	3
Boron oxide	10 mg/m^3	3

[a]1, Sprague 1972; 2, Papchristou et al. 1987; 3, EPA 1975; 4, NAS 1980; 5, Nielsen 1986; 6, Thompson et al. 1976; 7, Birge and Black 1977; 8, Weeth et al. 1981; 9, Brockman et al. 1985; 10, Seal and Weeth 1980; 11, Green and Weeth 1977; 12, Siegel and Wason 1986; 13, Benson et al. 1984; 14, Schillinger et al. 1982; 15, Krasovskii et al. 1976; 16, Anonymous 1983; 17, Hoffman et al. 1989; 18, Smith and Anders 1989.

In mammals, excessive boron consumption results in a reduced growth rate and sometimes loss in body weight; these may not be entirely due to reduced feed and water consumption (Table 9; Seal and Weeth 1980). Growth retardation has been reported in cattle Even 150 mg B/L drinking water (about 15 mg B/kg BW daily), in dogs consuming diets containing 1,760 mg B/kg, in rabbits eating rations equivalent to > 140 mg B/kg BW daily, and in rats given 150 mg B/L drinking water or 1,060 mg B/kg diet (Table 9). In some instances, animals will avoid B-contaminated drinking water if given a choice. Rats, for example, will reject drinking water containing as little as 1.0 mg B/L (Dixon et al. 1976), and cattle will avoid water containing > 29 mg B/L (Green and Weeth 1977).

Male workers engaged in boric acid production showed weakened sexual activity, decreased seminal volume, low sperm count and motility, and increased seminal fructose (EPA 1975). Adverse effects on reproduction of laboratory animals have been reported in sensitive species fed diets containing more than 1,000 mg B/kg, given drinking water containing 1.0 mg B/L (equivalent to about 0.3 mg B/kg BW daily), or given a single oral dose of 3,000 mg B/kg BW on the first day of pregnancy (Table 9).

Volatile boron compounds, especially boranes, are usually more toxic than boric acid or soluble borates (Table 9; NAS 1980). However, there is little commercial production of synthetic boranes, except for sodium borohydride—one of the least toxic boranes (Sprague 1972). Boron trifluoride is a gas used as a catalyst in several industrial systems, but on exposure to moisture in air it reacts to form a stable dihydride (Busch et al. 1086). For boric oxide dusts, occupational exposures to 4.1 mg/m^3 (range 1.2 to 8.5), are associated with eye irritation; dryness of mouth, nose and throat; sore throat; and cough (Garabrant et al. 1984).

No requirement for boron in mammals is known. Boron may accumulate in tissues because of slow excretion rates, although the significance of elevated residues is largely unknown (EPA 1975). Boron dietary

supplements to postmenopausal women age 48 to 82 years induced changes consistent with the prevention of calcium loss and bone demineralization (Nielson et al. 1987). In cattle, increases in boron ingestion were associated with elevated boron levels in plasma and urine, increased boron excretion, decreased plasma phosphate concentrations, and increased renal and urinary clearance of phosphates (Weeth et al. 1981). Boron accumulations in rat testes were associated with progressive germ cell depletion that persisted long after toxic exposure to boron had occurred (Lee et al. 1978).

Boron effectively counteracts symptoms of fluoride intoxication in humans (Zhou et al. 1987) and in experimentally poisoned rabbits (Elsair et al. 1980a, 1980b, 1981). Men suffering from skeletal fluorosis experienced 50 to 80% improvement after drinking solutions containing 300 to 1,100 mg of borax/L daily, 3 weeks a month for 3 months (Zhou et al. 1987). Boron enhances sequestration of fluoride from bone and excretion through kidneys and possibly the intestinal tract (Elair et al. 1980a, 1981).

Recommendations

Many boron criteria have been proposed for the protection of crops, aquatic life, waterfowl, livestock, and human health (Table 10).

Boron concentrations in contaminated industrial effluents seldom exceed 1.0 mg B/L, a level considered nonhazardous to aquatic life (Table 10; Thompson et al. 1976). However, future accumulations of boron in groundwaters through wider uses of B-containing cleansing agents may adversely affect aquatic organisms and other species of plants and animals, as now occurs in areas where natural boron deposits exist (EPA 1975). Long-term monitoring of groundwaters and surface waters for boron levels seems warranted.

Results of chronic feeding studies using mallards demonstrate that diets containing 13 mg B/kg FW produce no adverse effects, but those containing 30 or 100 mg B/kg FW are associated with elevated tissue boron residues and growth reduction, and diets containing 1,000 mg B/kg are fatal (Table 10). More research is needed on the fate and effects of boron on waterfowl and raptors, especially in those areas where high dietary boron loadings are encountered as a result of agricultural drainwater disposal practices.

Minimum concentrations of dietary boron needed to maintain animal health are not known with certainty. However, diets containing < 0.4 mg B/kg FW may adversely affect metabolism of rats and chicks; accordingly, animal diets should contain >0.3 mg B/kg FW until necessary feeding data become available (Nielsen 1986). Also, the defensible boron maximum for livestock drinking water may be considerably higher than 5 mg/L (Table 10) because several “safe” water sources in Nevada exceeded this upper maximum and approached 80 mg B/L (Green and Weeth 1977). Data are unavailable on boron effects on terrestrial wildlife. Until these data become available, it seems reasonable to apply the same criteria proposed for livestock protection (Table 10) to mammalian wildlife; that is, diets should contain more than 0.4 mg B/kg DW but less than 100 mg/kg, and drinking water < 5 mg/L.

Medicinal use of boric acid and borax for babies has resulted in anorexia, nausea, vomiting, diarrhea, marked cardiac weakness, a red eruption over the entire body, and (rarely) death (NAS 1980). The medical community has abandoned the use of boric acid solutions as irritants and antiseptics (Siegel and Wason 1486), abandoned all medical uses in Denmark (Egfjord et al. 1988), and severely limited availability (prescription only) in Ireland (O'Sullivan and Taylor 1983). Increased use of boric acid as a household pesticide should be viewed with concern, especially in households where children have access to nonsafety-capped boric acid containers (Siegel and Wason 1986).

The fact that boron is essential to plants is firmly established (NAS 1980). However, when boron concentrations in irrigation waters exceed 2 mg/L, extensive plant toxicity should be expected (Pagenkopf and Connolly 1982). High boron concentrations in some potential irrigation waters in the western United States (at levels capable of causing crop damage) have prompted implementation of boron criteria for irrigation waters (Table 10), although no legally enforceable boron standards have been promulgated (EPA 1975). Information is needed on crop plants in the following subjects: interaction of boron with other elements in the soil and its effects on boron availability to plants, the role of boron on pollination as it affects seed yield and sugar content of crops, and distinguishing signs of boron deficiency in plants from similar signs of molybdenum deficiency (Gupta and Macleod 1982).

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