WATER QUALITY GUIDELINE SERIES

Lead Water Quality Guidelines (Reformatted Guideline from 1987)
Technical Appendix

Ministry of Environment and Climate Change Strategy Water Protection & Sustainability Branch

No. WQG-12

BRITISH COLUMBIA

The Water Quality Guideline Series is a collection of British Columbia (B.C.) Ministry of Environment and Climate Change Strategy water quality guidelines. Water quality guidelines are developed to protect a variety of water values and uses: aquatic life, drinking water sources, recreation, livestock watering, irrigation, and wildlife. The Water Quality Guideline Series focuses on publishing water quality guideline technical reports and guideline summaries using the best available science to aid in the management of B.C.'s water resources. For additional information on B.C.'s approved water quality parameter specific guidelines, visit:

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Notes on Reformatted Version:

Sections of this report on industrial water use, drinking water and recreation have been removed. B.C. adopts Health Canada drinking water and recreation guidelines and no longer develops or supports guidelines for industrial water use. Fish tissue guidelines for human consumption have been removed due to out-of-date derivation methods. The current recommendation is to use a risk-based approach to develop human health-based fish/shellfish tissue guidelines for human consumption; these guidelines are referred to as screening values (SVs). Screening values are derived using Health Canada's general equation for calculating the ingested contaminant dose via consumption of contaminated food (Health Canada 2010).

Cover Photograph:

Location: Buttle Lake, B.C.

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Ms. L.D. Rounds and Ms. G. Rhodes, typists, Resource Quality Section.

CONTENTS

LIST OF TABLES

LIST OF FIGURES

1. INTRODUCTION

Lead is a non-essential element for plant, animal, and human nutrition; yet it is ubiquitous in our environment. This document discusses the effects of lead on various water uses which include drinking water, aquatic life, wildlife, livestock water supply, irrigation, recreation and aesthetics, and industrial water supplies. Since aquatic organisms are particularly sensitive to excessive lead levels in water, a large portion of this document focuses on lead toxicity to aquatic life. Standards, objectives, and criteria from various jurisdictions are reviewed, which, in conjunction with other information available from the literature, provide a basis for criteria recommended to protect water uses in British Columbia from anthropogenic lead.

Because of the extensive amount of literature on lead, a large portion of the information presented in this document has been extracted· from recent reviews on the subject. The purpose of this document was not to re-review the original literature already addressed in recent publications, but instead, to focus on the most applicable information which could be used to formulate defensible criteria for British Columbia waters.

2. FORMS AND TRANSFORMATIONS IN THE ENVIRONMENT

Naturally occurring lead (Pb) has three oxidation states: metal, Pb(II), and Pb (IV). In water, lead primarily exists as Pb(II). Pb(IV) may exist only in extreme conditions, outside the environmental pH and oxidation-reduction potential (Eh) range. However, Pb(IV) compounds are produced artificially and are discharged into the environment. One such compound, tetraethyl lead, is widely used as an antiknock agent in gasoline; lead emissions from gasoline and waste oil combustion are the principal sources of anthropogenic lead (Nriagu, 1986a).

The relative abundance of Pb(II) speci.es in the aqueous environment is pH dependent. In acidic (pH 7) waters, Pb^{2+}, $PbSO_4$, and $PbCl^+$, are the dominant species, whereas $PbCO_3$ and lead hydroxide complexes are common in alkaline (pH>7) waters. Other forms of lead include those (a) complexed with organic matter giving soluble, colloidal, and particulate compounds, and (b) sorbed on the suspended matter in water. In acidic media, humic acid sorbs lead more strongly than clay; whereas at pH 6.5 soluble lead-humate complexes are formed and clay seems to compete strongly with soluble lead-humates for retention on the solid phase. The presence of anions such as phosphate and sulphate also reduces aqueous metal ion concentration through precipitation of lead salts of low solubility. In general terms, the solubility of lead in water decreases with increased alkalinity.

Lead forms complex molecules with organic and suspended sediment fractions of water, and therefore is rapidly removed from surface and ocean waters (Goldberg, 1976; Ter Haar, 1975), The possibility of conversions of Pb^{2+} (as lead acetate), dimethyl lead, and trimethyl lead to volatile tetramethyl lead in flooded soils and lake sediments has been reported in the laboratory (Schmidt and Huber, 1976; Dumas et al., 1977; Huber et al., 1978; Wong et al., 1975, Jarvie et al., 1975; O'Hare et al., 1977; Chau and Wong, 1978). Lead methylation was dependent upon the pH, temperature, and microbial activity of the medium. Similar lead methylation in the environment is still controversial (Chau, 1986). Organolead compounds are relatively unstable and disappear rapidly from the environment through volatilization and degradation. However, alkyllead compounds are found in fish (Chau et al., 1985). Generally, organolead compounds are more toxic than the inorganic ones, and among the alkylated lead compounds the tetra-alkylated lead compounds are the most toxic (Chau and Wong, 1978; Chau et al., 1979).

Hart and Davies (1981) found that in the freshwater section of the Yarra River, Australia, about 45% of the total lead was present in particulate forms. As a comparison, 47 to 72% of the total lead was found

associated with particulates in the Susquehanna River, USA (McDuffie et al., 1976) and the Rhine River, FRG (DeGroot et al., 1976). In estuaries, the particulate lead fractions could be even higher due partially to resuspension of sediments (Hart and Davies, 1981). In Buttle Lake, British Columbia, the dissolved to total lead ratio ranged from 0.01 to 1.0 (Clark and Morrison, 1982).

3. OCCURRENCE IN THE ENVIRONMENT

3.1 Natural Sources

The most common ores of lead are the oxides, carbonate, and sulphate ores. Galena or lead glance (PbS) is, however, the most important commercial ore. In general, Pb is found in all the rocks of the earth's crust. The lead content of igneous rocks ranges from 8 mg/kg (for basic rocks such as basalt and gabro) to 20 mg/kg (for acid rocks such as granite and rhyolite). Similar levels were found in sedimentary rocks; from 7 mg Pb/kg for sandstones and limestones to 20 mg/kg for shales (Aubert and Pinta 1977, Demayo et al., 1980). The average concentration of Pb in the lithosphere is about 16 mg/kg (Swaine, 1955; Vinogradov, 1959; Turekian and Wedepohl, 1961).

Soils tend to be richer in lead than the rocks from which they are derived. The usual range for Pb content in soils has been estimated at 2 to 200 mg/kg (Bowen, 1966), with an average value between 10 to 25 mg/kg. In the U.S.A., a range of <10 to 700 mg/kg was reported by Shacklette al. (1971) for the Pb concentration in soils. Agricultural soils in British Columbia had an average lead concentration in the top 16 cm of 10.4 mg/kg (John, 1975). The mean lead concentrations in various horizons of Podzols in New Brunswick ranged from 13 to 73 mg/kg, whereas the mean concentrations above a sulphide deposit varied from 752 to 1 431 mg/kg (Presant and Tupper, 1965).

Depending upon weathering rates of various rocks and their average lead content, it was estimated that 152 000 to 162 000 tonnes pe year of lead enters surface waters naturally world-wide (Eriksson, 1960). These estimates agree with those of 180 000 tonnes of Pb/year being naturally mobilized and discharged into oceans by rivers (Bryce-Smith, 1971). Bertine and Goldberg (1971) estimated that weathering mobilizes about 21 000 to 110 000 tonnes of lead each year. More recently Nriagu (1986a) concluded that about 24 500 tonnes of lead are emitted annually from natural sources, a large fraction (65%) of which is derived from wind-blown dusts. These calculations assume that the dust also contains anthropogenic lead, so that emissions in pre-technological times should have been much smaller.

3.2 Anthropogenic Sources

The atmospheric fallout (wet and dry) originating from such activities as: (a) mining and milling of lead ores, (b) smelting and refining of primary and secondary lead, (c) manufacturing of lead chemicals, batteries, and other products, (d) production of other metals, cement, and combustion of fossil fuel, (e) consumption of lead products, notably leaded gasoline, and (f) incineration of refuse and sewage containing trace amounts of lead, is by far the major source of the anthropogenic lead in watercourses. The atmospheric lead deposited on the land reaches watercourses via surface runoff (e.g., urban runoff). Other sources include effluent generated from lead-related operations (e.g., mining, milling, and smelting, etc.), leachate from landfills, and effluent from sewage treatment plants, agricultural runoff.

Kemp and Thomas (1976) estimated that the man-made lead input to Lakes Ontario, Erie, and Huron ranged from 47% to 93% of the total input (anthropogenic + natural) at a given location. Distribution of Pb in core samples extracted from Lake Michigan suggested that natural input into the southern basin of the lake was about 1.6 mg/m^2, whereas the 1972 anthropogenic input from burning of coal and leaded gasoline was estimated at 13 mg/m^2 (Eddington and Robbins, 1976). More recently, it was estimated that

industrial activities released about 10 440 tons of lead into the Canadian atmosphere in 1980; about 80% of the lead emissions came from automobile tail pipes, and about 17% was contributed by the mining and smelting of non-ferrous metals (Nriagu, 1986a).

Lead chromate paints used on the inside of steel storage tanks or on old structures, lead pipes which may exist in some old houses and buildings, and lead solders connecting copper tubing used for distribution of water in homes and buildings may contribute significant amounts of lead to drinking water, depending upon the hardness of the water.

3.3 Levels in Water, Sediment, And Biota

3.3.1 *Water*

The lead concentration of all raw waters (rivers, lakes, wells, or groundwater) in Canada, between 1972 and 1977, was <1.0 µg/L to about 50 µg/L for dissolved lead, and from <1.0 µg/L to 100 µg/L for extractable lead. In a few cases, the results exceeded the upper limits of the test method of 50 µg/L (dissolved) and 100 µg/L (extractable) (Environment Canada, 1978). In areas where limestone and galena ores are found, waters may contain lead in solution up to 800 µg/L (Kopp and Kroner, 1967).

Most background waters in British Columbia contained lead levels ranging from <1.0 to 21 µg/L for total lead and <1.0 to 15 µg/L for dissolved lead, with median values of 3.0 and <1.0 µg/L, respectively. Less than 5% of background water samples contained total lead levels 5.0 µg/L. Elevated levels of lead in freshwater were determined in the vicinity of some mines, smelters, and landfills. In the Fraser River estuary (British Columbia), the median values for dissolved lead and total lead were 1 and 2 µg/L, respectively (Fraser River Estuary Study, 1979). The maximum levels in the Columbia River, about 0.8 km downstream from the Cominco Pb-Zn smelter, in British Columbia, were 3 µg/L for dissolved lead and 200 µg/L for total lead. The concentrations of lead in water were highest during low flow (Kootenay Air and Water Quality Study Phase II, 1979).

Neri, Schreiber, and Fortescue (1973) reported that the lead concentration in drinking water from 247 locations in Canada, ranged from 0 to 320 µg/L with a mean value of 11.8 µg/L. More recently, lead levels in drinking water in several Canadian locations were found to range from 0.25 to 71 µg/L with an average value of 8.8 µg/L (Dabeka, 1986).

Bowen (1966) and Preston (1973) estimated the Pb concentration in sea water at 0.02 to 0.03 µg/L. In British Columbia, on the west coast of Canada, the seawater lead concentrations were less than 0.05 µg/L except near large lead sources (Stukas and Wong, 1981). In some areas of the Mediterranean Sea and the Pacific Ocean, lead contents of about 0.20 µg/L and 0.35 µg/L, respectively, were found (NAS, 1972). Values up to 2.3 µg/L have been reported in the British coastal waters (Preston, 1973). The particulate matter collected from the surface of the oceans (0 to 5 m) had an average lead content of 58 µg/kg, probably largely located in plankton (Chester and Stoner, 1975).

3.3.2 *Sediment*

Lead forms complex molecules with organic and suspended sediment fractions of water, and therefore is rapidly removed from surface and ocean waters (Goldberg, 1976; Ter Haar, 1975). In the Saline Branch watershed, Getz et al. (1977) noted that the lead concentration in filtered (0.45 µm) water samples, unfiltered water samples, and the top 5 cm of bottom sediments in urban areas, ranged from 15 µg/L, 15 to 200 µg/L, and 2 330 to 6 300 µg/g, respectively. Lead contents of up to 3 200 µg/g, were found in the sediments from Wapato Lake (Washington), which received urban storm water with no treatment (Wisseman and Cook, 1977). The sediment lead concentrations of 390 µg/g near a sewage treatment plant, and 1344 µg/g near a snow dump exceeded by far the average concentrations in the Ottawa (26

µg/g) and the Rideau (42 µg/g) Rivers (Oliver and Kinrade, 1972). The lead concentration in sediments from the Great Lakes ranged up to 274 µg/g (Hutchinson and Fitchko, 1974).

Background concentrations of lead in most fresh and coastal water sediments ranged from <10 to <20 µg/g (Garrett, 1985). Elevated levels were, however, determined near several mines, smelters, and certain areas of the Brunette and Fraser River systems. The sediment lead content of 866 µg/g in the Columbia River, about 0.8 km downstream from the Cominco Pb-Zn smelter, was about 45 times higher than the background levels (19 µg/g, Kootenay Air and Water Quality Study Phase II, 1979). Lead levels in the Buttle Lake sediments ranged from 42 to 559 µg/g (Clark and Morrison, 1982).

The lead content in deep-sea sediments was estimated at 9 µg/g for clay and 80 µg/g for the carbonate type of materials (Turekian and Wedepohl, 1961). Chester and Stoner (1975) recorded 20 µg/g associated with nearshore sediments and 45 µg/g in deep-sea sediments.

In marine areas of British Columbia, maximum levels of lead in sediment, ranging from 2 to about 5 500 µg/g, were detected at mining operations in Alice Arm, the Alcan smelter at Kitimat, several pulp mills, in Victoria and Vancouver Harbours, certain ocean dump sites, False Creek, Comox, and the Esquimalt Harbour Armed Forces Base (Garrett, 1985). As indicated above, background lead levels in coastal water sediments ranged from <10 to <20 µg/g (Garrett, 1985).

3.3.3 *Biota*

Uthe and Bligh (1971) reported that fish {headless dressed fish samples ground and thoroughly mixed) from Great Lakes had less than 0.5 µg/g (wet weight). Lead concentrations in the liver of several species of fish (148 samples) from 62 British Columbia Lakes, ranged from 0.1 to 5.3 µg/g·(wet weight) with an average value of about 0.7 µg/g (Peterson et al., 1970). In another survey, the lead concentration was measured in the livers of large-scale suckers and rainbow trout upstream and downstream from a Pb-Zn smelter at Trail, B.C. on the Columbia River. The samples taken upstream from the smelter averaged 0.98 and less than 0.5 µg/g (dry weight) for four large-scale suckers and one rainbow trout, respectively; the samples taken downstream from the smelter averaged 1.5 and 1.4 µg/g for one largescale sucker and two rainbow trout, respectively (Kootenay Air and Water Quality Study Phase II, 1979). Lead levels (<0.49 µg/g wet wt.) in the livers of Fraser River fish were generally less than those taken from other British Columbia waters (Singleton, 1983).

In a more recent report (Smith, 1987), average lead levels in Columbia River fish muscle were reported to range from 0.016-0.49 µg/g-wet wt. (or 0.075-2.2 µg/g-dry wt.). Lead levels in some largescale suckers caught downstream from Cominco were elevated (average value = 0.78 µg/g-wet wt.) over the background (0.17 µg/g-wet wt.); the highest lead levels downstream were in excess of 1.0 µg/g-wet wt. Smith (1987) also noted that lead levels were higher in fish liver than in fish muscle. For instance, the average lead level in the liver of largescale suckers at the upstream (background) sites was 0.39 µg/g-wet wt., whereas at the downstream (from Cominco) sites it ranged from 2.5-2.6 µg/g-wet wt.

Brown and Chow (1977) found average concentrations of lead in fish muscle (wet weight) of 0.19 µg/g and 1.78 µg/g from Baie du Dore (Lake Huron) and Toronto Harbour, respectively. Also, there was close agreement between the distribution of metals in sediments and in non-migratory fish. Bottom-feeding fish, e.g., suckers (Catostomidae), accumulated more tissue lead in contaminated ecosystems than did centrarchids feeding in the water column at the same location; both groups were exposed to the same levels of waterborne lead, but suckers feed on organisms living in contaminated sediments (Hodson et al., 1983; Ney and Van Hassel, 1983; Czarnezki, 1985).

The transfer of lead from water and sediment into food webs (e.g., fish) is not a simple function of the lead concentration in the environment. Chemical speciation of lead in water, chemical partitioning of lead

in sediments, and the nature of biota affect lead uptake. Laboratory studies have shown that lead uptake by fish is reduced when dissolved organic carbon concentrations are elevated (Wiener and Giesy, 1979), when phosphate concentrations are increased (Freedman et al., 1980), or when pH is elevated (Merlini and Pozzi, 1977). Studies in natural systems have also shown reduced concentrations of lead in organisms where sediments are high in organic carbon (Schierup and Larsen, 1981) or high in amorphous iron oxides (Louma and Bryan, 1978; Tessier et al., 1984).

4. AQUATIC LIFE

The distribution and effects of lead on the aquatic environment have been reviewed by Demayo et al., (1 980), Moore and Ramamoorthy (1984) and U.S. EPA (1985a). The distribution of lead through the ecosystem suggests that it does not biomagnify in higher trophic levels; lead concentrations are highest in sediments and decrease in the order: sediments > plankton > benthos > fish (Namminga, et al., 1974; Getz, et al., 1977; Patrick and Loutit, 1977).

4.1 Effects on Algae

4.1.1 *Freshwater Algae*

Several factors influence lead toxicity to aquatic plants under laboratory conditions; these include water chemistry, availability of nutrients, complexing capacity of water, species, and forms of lead. Lead-induced toxicity under field conditions has not been reported in the literature. Lead inhibits photosynthesis and adenosine triphosphate (ATP) synthesis and thus structural protein formation (Silverberg, 1975).

The effects of pH and nutrient (phosphate) availability on lead toxicity to algae were studied by Monahan (1976). The growth of *Selenastrum capricornutum* in a phosphate-limiting acidic medium (pH 6.2) containing 500 µg Pb/L was inhibited by 50% compared to the control. In an alkaline (pH 8.0) medium, however, growth inhibition did not become apparent until 3 000 µg Pb/L, either with or without phosphate as the growth-limiting factor. The same study also indicated that the different algal species showed different sensitivities to lead under differing water qualities (e.g., pH), and that phosphate may have been involved in the lead inhibition of algae.

Manganese and copper may off-set lead-induced inhibition in aquatic plants, thus reflecting competition for active sites on enzymes (Pietilainen, 1975). A severe depression in algal growth to below 40% of control growth was noted in lake water of low complexing capacity (0.75 µmol Cu/L) containing 500 µg Pb/L. In contrast, water of high complexing capacity enabled at least 50% of control growth to be maintained in the presence of 500 µg Pb/L (Chau and Wong, 1976).

Some species are more tolerant of lead than others. No detectable effects of Pb (NO3) 2 and (CH 3 C00) 2 Pb were found on either *Chlorella* or *Chlamydomonas* at concentrations of 5 000 and 50 000 µg Pb/L, respectively (Hutchinson, 1973). Algae producing extracellular polysaccharides and polypeptides can withstand large amounts of lead (Wolk, 1973; Jones and Stewart, 1969; Kushner, 1974). The green algae *Microthamnion kuetzingianum* tolerated concentrations of more than 100 000 µg Pb/L (Lorch, 1974).

4.1.2 *Marine Algae*

The saltwater species of algae are more sensitive to lead than the freshwater species. The following discussion is based on tests conducted for a duration of 2 to 12 days by various investigators.

Steele and Thurs by (1983) noted reduced growth (final dry weight) of the females, reduced tetrasporangia production, and inhibition of sexual reproduction in the saltwater alga, *Champia parvula*, at 20.3, 23.3, and 20.3 µg Pb/L of lead nitrate in water, respectively. A reduction of 65% in growth was noted in *Dunaliella salina* when exposed to a lead concentration of 900 µg Pb/L of lead nitrate (Pace et

al., 1977). In tests with organolead compounds, Marchetti (1978) reported that the saltwater alga *Dunaliella tertiolecta* was ten times more sensitive to tetraethyl lead (96-h EC50 = 150 µg Pb/L) than tetramethyl lead (96-h EC50 = 1 650 µg Pb/L).

Saltwater diatoms are also quite sensitive to lead in water. The EC50 for two species of diatoms, *Ditylurn brightwelli* and *Astrionella japonica,* were noted to be 40 µg Pb/L of P bC1 2 and 207 µg Pb/L of Pb (NO3) 2, respectively (Canterford and Canterford, 1980; Fisher and Jones, 1981). In tests with *Skeletonema costatum* exposed to lead nitrate *fn* 12 days, a 50% reduction in growth rate and maximum yield of the organisms (EC50) was caused by 3.7 and 5.1 µg Pb/L in water, respectively (Rivkin, 1979). A reduction of 25-50% in photosynthetic and respiration activities was reported in *Phaedactylum tricarnutum* subjected to 100 µg Pb/L for 48 to 72 hours (Woolery and Lewin, 1976); however, Hannan and Patouillet (1972) observed no adverse effect in the growth of the same species of diatom exposed to 1 000 µg Pb/L for 72 hours.

4.2 Effects on Invertebrates

4.2.1 *Freshwater Invertebrates*

(a) Acute Toxicity

Different species of invertebrates show different sensitivities to lead. Acute effects are usually reported at concentrations of 100 to 100 000 µg Pb/L.

Among various factors, hardness of water is considered to have a major effect on lead toxicity to a given species; the toxicity is lower in hard waters. However, the effect of hardness of water is indirect and probably due to one or more of a number of interrelated ions; e.g., hydroxide, carbonate, calcium, and magnesium. Chapman et al. (U.S. E.P.A., 1985a) reported that the LC50's for *Daphnia magna* were 612, 952, and 1 910 µg Pb/L in water of hardness 54, 110, and 152 mg/Las $CaCO_3$. Using the same species of *Daphnia*, another investigator reported a 48-h LC50 of 450 µg Pb/L at a water hardness of 44 mg/L as $CaCO_3$ (Biesinger and Christensen, 1972).

Acute toxicity tests with amphipods suggested that these micro-crustaceans are much more sensitive to lead than other freshwater animal species. The LC50's of 124 µg Pb/L (Spehar et al., 1978) and 140 µg Pb/L (Call et al., 1983) for *Gammarus pseudolimnaeus* in water of hardness 4 and 48 mg/L as $CaCO_3$, respectively, were the lowest reported in the literature (U.S. EPA, 1985a).

Aquatic insects are also important fish-food organisms, but these organisms are much less sensitive to lead toxicity. Using $PbSO_4$ as a source of lead, Warnick and Bell (1969) reported the 7-d LC50's of 16 000 and 32 000 µg Pb/L in soft water (44 mg/L as $CaCO_3$) for the mayfly *(Ephemerella subvaria)* and the caddisfly (*Hydropsyche betteni*), respectively. In the same study, *Acroneuria lycorias* (stonefly) had a 14-d LC50 of 64 000 µg Pb/L. For other species of mayfly (*Ephemerella grandis*) and stonefly (*Pteronarcys californica*), the 14-d LC50's in soft water (50 mg/Las $CaCO_3$) were 3 500 µg Pb/Land over 19 200 µg Pb/L, respectively (Nehring, 1976). The LC50 of 224 000 µg Pb/L of Pb $(NO_3)_2$ for *Tanytarsus dissimilis* (midge) in water of hardness 48 mg/L as $CaCO_3$ (Call et al., 1983) suggests that they are very tolerant to lead contamination. However, the 10-d LC50 in soft water (47 mg/L as $CaCO_3$) for the same species of midge at the embryo-3rd instar stage was much lower (258 µg Pb/L; Anderson et al., 1980).

Aquatic snails also differ widely in their response to lead. As with aquatic insects, embryos are much more sensitive to lead than the adult snails. Cairns et al. (1 976) reported the 48-h LC50 of 71 000 µg Pb/L and

14 000 µg Pb/L of lead acetate. for *Goniobasis livescens* and *Lymnaea emarginata*, respectively, in water of hardness 131 to 171 mg/L as $CaCO_3$. Ravera (1977) noted that no embryos of the snail *Biomphalaria glabrata* survived exposure to 1 000 and 500 µg Pb/L of lead nitrate; also, 41% of the embryos hatched at 100 µg Pb/L, but all hatched snails died after 15 days.

Aquatic organisms, including invertebrates, can adapt to high levels of lead. The tolerance of invertebrates, taken from lead-affected rivers, to high lead levels was demonstrated by Fraser et al. (1978) and Brown (1976 and 1978). In Brown's (1 976) study, the 48-h LC50 of 3 500 µg Pb/L for *Asellus meridianus* from River Gannell (with 190 µg Pb/L in water, 6 614 mg Pb/kg in dry sediment, and water hardness of 25 mg/Las $CaCO_3$) in a lead mining area was much higher than the 280 µg Pb/L for the same species taken from a control area of Bradwell Brook (with <100 µg Pb/L in water, 56 mg Pb/kg in dry sediment, and water hardness of 25 mg/L as $CaCO_3$). During acute toxicity tests, Brown (1976) also found that the. tolerance of *Asellus meridianus* taken from copper-tin contaminated areas of the River Hayle was intermediate (2 800 µg Pb/L) between that of the crustaceans from the lead-polluted River Gannel and the unaffected river Bradwell Brook; the difference in toxicity was assumed to be due to the different tolerances of the populations. Metal storage and/or metal detoxification were postulated as possible mechanisms for this tolerance (Brown, 1977).

The presence of certain metals can induce tolerance to other metals in the aquatic environment (Brown, 1978). The uptake of copper by *A. meridianus* from a river where the levels of copper were higher than lead was considerably reduced by addition of increasing levels of lead. The lead was taken up readily. When experimental conditions were reversed, and the organisms were exposed to a constant level of lead and increasing copper, the uptake of lead was reduced at higher copper concentrations. There was evidence that lead is more readily bound than copper and competes with copper for the same storage sites.

(b) Chronic Toxicity

In studying chronic toxicity of lead to *Daphnia magna*, Chapman et al. (U.S. EPA, 1985a) found that daphnids were 11 times more sensitive to lead in soft water than in hard water. The chronic toxicity limits found by these investigators in a life-cycle test, ranged between 9 to 16.7, 78 to 181, and 85 to 193 µg Pb/L of lead nitrate at water hardnesses of 52, 102, and 151 mg/L as $CaCO_3$, respectively. In another test, Biesinger and Christensen (1972) reported a 16% impairment in reproduction of *D. magna* exposed to 30 µg Pb/L of PbC1 2 in soft water (45 mg/L $CaCO_3$) for 21 days.

In a life-cycle test with the snails, *Lymnaea palustris*, in hard water (139 mg/L as $CaCO_3$), Borgmann et al. (1978) found no observed effect on survival of these organisms at 12 µg Pb/L of lead nitrate, and nearly complete mortality at 54 µg Pb/L. Based on this information, a chronic value (geometric mean of the lower and upper limits) for snails was established at 25.5 µg Pb/L (U.S. EPA, 1985a).

4.2.2 *Marine Invertebrates*

(a) Acute Toxicity

The lead-induced acute toxicity to marine invertebrates varies with species and its life stage. An LC50 of 27 000 µg/L was noted for the adult soft-shell clam, *Mya arenaria* (Eisler, 1977); however, the acute values obtained with larvae of the blue mussel (*Mytilus edulis*), pacific oyster (*Crassostrea gigas*), eastern oyster (*Crassostrea virginica*), and quahog clam (*Mercenaria mercenaria*) were only 476, 758, 2 450, and 780 µg/L, respectively (Martin et al., 1981; Calabrese et al., 1973, and Calabrese and Nelson, 1974). The LC50 values for some other species of marine invertebrates, as reported in the literature, are: Copepod (*Acartia clausti*), 668 µg/L (Gentile, 1982); Dungeness crab (*Cancer magister*), 575 µg/L (Martin et al., 1981).

(b) Chronic Toxicity

Mysidopsis bahia (mysid) appears to be the only species which was subjected to chronic toxicity tests. In a life-cycle test with the mysid, lead levels ranging from 17 to 37 µg/L (with a chronic value of 25.1 µg Pb/L defined as the geometric mean of the lower and upper limits) caused reduced spawning (Lussier et al., as quoted in U.S. EPA, 1985a).

4.3 Effects On Fish

4.3.1 *Freshwater Fish*

(a) Acute Toxicity

As with other aquatic organisms, lead is more toxic to fish in soft water than in hard water. The 96-h LC50 for total lead varies widely. In very hard waters (350 mg/Las $CaCO_3$), it may exceed 400 000 µg/L (Moore and Ramamoorthy, 1984).

Pickering and Henderson (1966) reported 96-h LC50's for the fathead minnow (*Pimephales promelas*) and bluegill (*Lepomis macrochirus*) of 5 580 to 7 330 and 23 800 µg Pb/L of PbC1 2, respectively, in water of hardness of 20 mg/Las $CaCO_3$. However, when the same tests were conducted in hard water (360 mg/L as $CaCO_3$), the 96-h LC50 for the two types of fish increased to 482 000 and 442 000 µg Pb/L, respectively. Similar effects were shown with rainbow trout (*Salmo gairdneri*) with 96-h LC50 values of 170 and 471 000 µg Pb/L of total lead nitrate at water hardness of 32 and 290 mg/L $CaCO_3$, respectively (Davies et al., 1976). The 96-h LC50 for brook trout (*Salvelinus fontinalis*) was reported to be 4 100 µg Pb/L (total) and 3 362 µg Pb/L (dissolved) of lead nitrate in soft water (44 mg/L as $CaCO_3$, Holcombe et al., 1976).

(b) Chronic Toxicity

In early life-stage (embryo to 19 months after hatching) tests with rainbow trout (*Salmo gairdneri*) in soft water (28 mg/L as $CaCO_3$), Davies et al. (1976) suggested that chronic effects may appear at 4.1 to 7.6 µg Pb/L of lead nitrate during pre-hatch and 7.2 to 14.6 µg Pb/L in post-hatch fry. The incidence of chronic effects (e.g., black-coloured tails, spinal deformities, etc.) at these concentrations was, however, low (0.7 to 4.7%). Spinal deformities (curvature) were noted in 32% of the fish when the lead concentration in water was increased to 27 µg Pb/L.

The chronic effects of lead are more severe in soft water than in hard water. This trend was demonstrated by Davies et al. (1976) while studying the long-term effects on rainbow trout (*Salmo gairdneri*) fry and fingerlings exposed to various concentrations of lead for 19 months. The results of these tests indicated that in hard water (353 mg/L as $CaCO_3$) 0 and 10% of the fish developed spinal deformities at measured concentrations of 190 and 380 µg Pb/L, respectively. However, in soft water (28 mg/L as $CaCO_3$) 44% and 97% of the fish developed spinal deformities at concentrations of 31 and 62 µg Pb/L, respectively.

Spinal deformities due to lead were also noted in a life-cycle test of three generations with brook trout (Holcombe et al., 1976), and in an early life-stage test (2-month duration) with rainbow trout (Sauter et al., 1976). The chronic values obtained by these investigators were 58 to 119 µg Pb/L (total), or 39 to 84 µg Pb/L (dissolved), for brook trout in water of hardness 44 mg/L as $CaCO_3$; and 48 to 83 µg Pb/L for rainbow trout at water hardness of 34 mg/L as $CaCO_3$, respectively.

Exposure of rainbow trout (*Salmo gairdneri*) to lead as low as 13 µg/L (free lead) in water of hardness of 135 mg/L as $CaCO_3$ caused significant (i) increase in red blood cell (RBC) numbers, (ii) decline in RBC volumes and cellular iron content, and (iii) decrease in RBC δ-amino levulinic acid dehydratase (δ-ALAD) activity (Hodson, 1976; Hodson et al., 1978). Since the next lowest concentration was 3 µg/L (the control),

harmful effects of lead may also occur between 3 and 13 µg/L (Hodson, 1976). The degree of o-ALAD inhibition, however, varied between fish species; little effect was caused by 50 to 60 µg/L in brook trout, and by 90 µg/L in sunfish (Hodson et al., 1977).

4.3.2 *Marine Fish*

(a) Acute Toxicity

Four species of marine fish, namely sheepshead minnow (*Cyprinodon variegatus*), mummichog (*Fundulus heteroclitus*), inland silverside (*Menidia beryllina*), and Atlantic silverside (*Menidia menidia*) have been tested for acute toxicity of lead. *Fundulus heteroclitus* was the most sensitive with an LC50 of 315 µg Pb/L (Dorfman, 1977 in U.S. EPA, 1985a). In fact, of the 11 saltwater genera (including four species of fish indicated above) for which acute values were available, the most sensitive genus, *Fundulus*, was 85 times more sensitive than the most resistant, *Mya* (*Mya arenaria* or soft-shell clam) (Eisler, 1977; U.S. EPA, 1985a). In flow-through toxicity tests with Sheepshead minnow (*Cyprinodon variegatus*) and inland silversides (*Menidia beryllina*), less than 50% of the test organisms were killed at 3 140 µg Pb/L, which is the solubility of lead in sea water under the test conditions.

(b) Chronic Toxicity

No data for marine fish were available.

4.4 Toxicity of Metal Mixtures

The presence of other metals may reduce or enhance the toxic effects of lead on aquatic organisms. Pietilainen (1975) reported a synergistic effect on primary production of phytoplankton in solutions when the concentration of cadmium (1 000 µg Cd/L) was greater than that of lead (100 µg Pb/L). (Effects are termed synergistic when the cumulative effect of metals A and B together is more than the sum of the effects of each metal alone). However, when the lead (1 000 µg Pb/L) was present in a greater concentration than cadmium (100 µg Cd/L), the cumulative effect of lead and cadmium mixture on primary production of phytoplankton was smaller than the sum of effects of lead and cadmium alone, suggesting antagonism.

Ozoh (1979) studied the effects of lead and copper ions (using concentrations of 36 and 72 µg/L for both metals) on fish (*Brachydanio rerio*) eggs in distilled water. Both lead and copper reduced hatching, and a high concentration of lead in the Pb-Cu mixture antagonized copper toxicity.

4.5 Toxicity of Organolead Compounds

Lead is methylated by bacterial action. Methylated lead (0.5 mg/L as volatile tetramethyl lead) has been shown to be more toxic to the growth of *Scenedesmus quadricauda*, *Chlorella pyrenoidosa*, and *Ankistrodesmus falcatus* than inorganic lead. Twenty times more lead nitrate will be required to have the same effect on these species as methylated lead (Silverberg et al., 1976 and 1977; Chau and Wong, 1978). In a continuous-flow dosing system with water maintained at a constant lead level, a tetramethyl lead (Me_4Pb) level as low as 3.5 µg/L was found toxic to *Salmo gairdneri* (Chau et al., 1979; Wong et al., 1981). Alkyllead compounds are also toxic to marine organisms (Marchetti, 1978; Maddock and Taylor, 1980). However, organolead compounds are volatile and disappear rapidly from the environment. The half-life of tetraethyl lead (Et_4Pb) ranges from about 1/2 day in seawater to about 2 days in freshwater, while trialkyl lead compounds (e.g. Me_3Pbx and Et_3Pbx and products of tetraalkyl lead compounds) are more stable (Grove, 1980; Roderer, 1980). The evidence of production of organolead compounds is lacking under field conditions; nevertheless, these compounds are produced industrially in large quantities and

have the potential to escape into the environment. The presence of alkyllead compounds in water so far has been reported in one case only (Hodson, 1986).

4.6 Criteria from the Literature

4.6.1 *Criteria from Other Jurisdictions*

Criteria, objectives, and standards to protect aquatic life from harmful effects of lead are shown in Tables 2 and 3. The recommended lead criteria varied with jurisdiction. For freshwater aquatic life, the recommended criteria ranged from a value of 2 µg Pb/L (total) to a value which depended upon the hardness of water. The limits for the marine environment ranged from 4 to 140 µg Pb/L.

Due to insufficient information regarding chronic effects and the effect of hardness on lead toxicity to aquatic organisms, earlier criteria for lead were expressed as a single value and disregarded the influence of water hardness (U.S. EPA, 1972). In later publications, chronic toxicity was determined by multiplying acute toxicity values (96-h LC50) by an application factor (U.S. EPA, 1976; Hart, 1974). The use of the application factor was, however, criticized by the American Fisheries Society (1979) on the grounds that an application factor could not predict safe concentrations of lead in waters of different quality, when based on total lead analyses determined by atomic absorption spectrophotometry.

Table 1. Lead Criteria for Freshwater Aquatic Life

Criteria Statements	Criteria Values (µg Pb/L)	Jurisdiction	Date	Reference
To protect aquatic life the lead concentration in water should not exceed. 0.03 mg/L at any time or place	30	U.S. EPA	1972	U.S. EPA (1972)*
Total lead in excess of 20 µg/L in water should be investigated	≤20 (Total lead)	Australia	1974	Hart (1974)
Maximum concentration for lead in surface water= 0.05 mg/L	50	Saskatchewan	1975	SSWQO (1975)**
0.01 times the 96-h LC50 value using the receiving or comparable water as diluent and soluble lead measurements (using 0.45 µm filters), for sensitive freshwater resident species	0.01 times the 96-h LC50 (soluble lead)	U.S. EPA	1976	U.S. EPA (1976)*
Maximum concentration for lead in surface water= 0.05 mg/L	50	Alberta	1977	ASWQO (1977)+
To protect aquatic life, the total lead concentration in unfiltered water samples should not exceed: Hardness mg/L $CaCO_3$ Lake Superior 44 Lake Huron 94 Other Great Lakes >119	 Total Lead 10 20 25	International Joint Commission	1977	IJC (1977)++
Total lead concentration at given alkalinity should not exceed the following Alkalinity (mg/L as $CaCO_3$) up to 20 20-80 40-80 >80	 Total lead 5 10 20 25	Ontario	1984	OME (1984)+++
To protect aquatic life, recommended criteria for total lead in water should be as follows Hardness (mg/L $CaCO_3$) 0-30 30-100 100-300 >300	 Total lead 4 25 50 100	America Fisheries Society	1979	AFS (1979)[1]
To protect aquatic life, the total lead concentration in unfiltered water samples should not exceed:		International Joint Commission	1980	IJC (1980)++

Criteria Statements	Criteria Values (µg Pb/L)	Jurisdiction	Date	Reference
Hardness mg/L $CaCO_3$ Lake Superior 44 Lake Huron 94 Lake Ontario 135	Total Lead 2 3 5			
For protection of aquatic life, the maximum 24-h average for total recoverable lead (µg/L) at a given hardness of water (mg/Las $CaCO_3$) should not exceed exp(2.35 ln (hardness) -9.48), or the maximum concentration at any time should not exceed exp (1.22 ln (hardness) -0.47). For example; Hardness (mg/L $CaCO_3$) 50 100 200	Total Recoverable Lead 24-h Av. Max. 0.75 74 3.8 172 20.0 400	U.S. EPA	1980	U.S. EPA (1980)*
In presence of sensitive species, recommend objective of 0.005 mg/L total lead in soft water (45 mg/L as $CaCO_3$), and 0.01 mg/L total lead in hard water (>95 mg/L as $CaCO_3$); recommended level of total lead in soft as well as hard water where sensitive fish are absent = 30 µg/L	5 (soft) 10 (hard) 30 (soft or hard) (Total lead)	Inland Waters Directorate	1980	Demayo et al. (1980)
For protection of aquatic life, the maximum concentration of total lead (µg/L) at a given hardness of water (mg/L as $CaCO_3$) should not exceed exp(2.35 ln (hardness)-9.48) Hardness (mg/L $CaCO_3$) 50 100 200	Total lead 0.75 3.8 20.0	Manitoba	1983	MDEWSH (1983)[2]
Average concentration for protection of freshwater fish Hardness (mg/L $CaCO_3$) <50 50-100 >150	Total lead Salmonid Waters / Coarse Fish Waters 4 50 10 125 20 250	U.K.	1984	Mance et al., 1984
To protect aquatic organisms, except possibly where a local species is very sensitive, 4-day average concentration		U.S. EPA	1985	U.S. EPA (1985a)*

Criteria Statements	Criteria Values (µg Pb/L)	Jurisdiction	Date	Reference
(µg/L) of acid-soluble lead at a given hardness of water (mg/ L as $CaCO_3$) should not exceed exp (1.273 ln (hardness)-4.705) more than once every three years on the average, and 1-h average concentration (µg/L) should not exceed exp(1.273 ln (hardness)-1.460) more than once every three years on the average. For example: Hardness (mg/L $CaCO_3$) 50 100 200	Total Recoverable Lead 4-d-Av. / 1-h Av. 1.3 / 34 3.2 / 82 7.7 / 200 (Total recoverable lead recommended until acid-soluble techniques can be developed)			
Concentration of total lead should not exceed value as given below Hardness (mg/L $CaCO_3$) 0-60 (soft) 60-120 (medium) 120-180 (hard) >180 (very hard)	Total lead 1 2 4 7	CCREM	1987	Canadian Council of Resource and Environment Ministers, 1987

* U.S. Environmental Protection Agency
** Saskatchewan Surface water Quality Objectives
\+ Alberta Surface Water Quality Objectives
++ International Joint Commission
+++ Ontario Ministry of Environment
[1] American Fisheries Society
[2] Manitoba Department of Environment and Workplace Safety and Health

Table 2. Lead Criteria for Marine Aquatic Life

Criteria Statements	Criteria Values (µg Pb/L)	Jurisdiction	Date	Reference
Concentration of lead in sea water should not exceed 0.02 of the 96-h LC50 for the most sensitive species, and the 24-h average concentration should not exceed 0.01 of the 96-h LC50. Concentrations of lead 0.05 mg/L constitute a hazard and levels <0.01 mg/L present minimum risk. Special efforts should be made to reduce lead levels even further in oyster growing areas	0.02×96-h LC50 0.01×96-h LC50 50 10	U.S. EPA	1972	U.S. EPA
Recommended criteria for marine =0.004 mg/L of total lead until more information is available on the long-term effects of lead on sensitive marine organisms	4	America Fisheries Society	1979	AFS (1979)[1]
Except possibly where a locally important species is very sensitive, saltwater aquatic organisms should not be affected unacceptably if 4-d average concentration of lead does not exceed 5.6 µg/L more than once every three years on the average, and if the 1-h average concentration does not exceed 140 µg/L more than once every three years on the average	5.6 (4-d Average) 140 (1-h Average)	U.S. EPA	1985	U.S. EPA (1985a)*

* U.S. Environmental Protection Agency

[1] American Fisheries Society

The criterion of 50 µg Pb/L for surface water adopted by Alberta and Saskatchewan is the same as that for drinking water supply proposed by Health and Welfare Canada (1978) and does not appear to be adequately protective for aquatic life.

Criteria for lead based on a sliding scale of water hardness was first recommended by the IJC (1977) for the Great Lakes and then by the American Fisheries Society (1979) in a review of the U.S. EPA (1976) document. Ontario Ministry of Environment (1979) also adopted criteria based on a relationship between water hardness and lead toxicity to aquatic organisms. However, the scale of hardness used in defining lead criteria varied between the jurisdictions.

More recently the U.S. EPA (1980) proposed a relationship, based on statistical analysis of available data, between water hardness and lead levels to protect freshwater aquatic life. The relationship was updated in 1985 in view of additional information available in the literature (U.S. EPA, 1985a). Two averaging periods, 1 h for acute and 4 d for chronic effects, were chosen.

The U.S.E.P.A. (1985a,b) criteria were designed to protect 95% of a group of diverse genera from the harmful effects of lead. The 'Final Acute Value (FAV) ', which is an integral part of the procedure for deriving water quality criteria for aquatic life, is an estimate of the fifth percentile of a statistical population represented by the sets of 'Genus Mean Acute Value (GMAV)'. The GMAV is defined as the concentration of the material, e.g., lead, that causes a specified level of acute toxicity to aquatic organisms in some taxonomic group. In determining the FAV, it is assumed that the measured GMAV's are (i) randomly selected, and (ii) follow a log triangular distribution. Since the assumption of 'log triangular distribution' is unlikely over the entire data range, FAV estimation is based on a subset of the data near the fifth percentile (i.e., cumulative probability = 0.05). Figure 1 indicates that both the acute and chronic

lead criteria recommended by the U.S. EPA (based on FAV calculations) are well below experimentally observed acute and chronic toxic levels, respectively, and may therefore be overprotective, especially at low water hardness. For instance, Davies et al. (1 976) observed that the maximum acceptable toxicant concentration for *Salmo gairdneri* (the most sensitive species to lead) in water of hardness 28 mg/L $CaCO_3$ ranged from 4 to 7.6 µg Pb/L, with a geometric mean value of 5.5 µg Pb/L (Table 4). According to the U.S. EPA, the 4-day average (chronic value) at that hardness is only 0.6 µg Pb/L. This overprotectiveness of the U.S. EPA criteria is primarily the result of a lack of data and a statistical procedure which extrapolated the sparse data to low levels for which there were no data, resulting in very low criteria. For instance: (i) only ten GMAV is were considered in FAV estimation, (ii) the lowest GMAV thus corresponded to a ninth percentile (cumulative probability = 0.09) instead of the desirable fifth percentile (cumulative probability = 0.05), (iii) the FAV at the fifth percentile was then extrapolated from GMAV's with cumulative probabilities ranging from 0.09 to 0.36. A set of GMAV's with such a wide range of probabilities may not satisfy the assumption of a log triangular distribution.

Instead of a two-number (acute and chronic) approach, a single maximum value was adopted by CCREM (1987). The maximum value was based on a long-term 'no-effect' concentration rather than on an acute concentration. The reasons for adopting this approach were: (i) a concentration in water continuously just below the acute value could cause chronic effects and eventually harm or eliminate a resident population, (ii) a single sample does not yield information about conditions that may continue for hours, days, or even weeks, (iii) lack of a generally accepted and scientifically sound basis for the duration of the averaging period, and (iv) fluctuating concentrations are considered more toxic than unvarying concentrations even though the average may be the same.

The criterion proposed by CCREM is a step {instead of a continuous) function of water hardness and ranges from 1 µg Pb/L at water hardness <60 mg/L $CaCO_3$ to 7 µg/ Pb/L at hardness >180 mg/L $CaCO_3$. It essentially follows the U.S. EPA 4-day average (chronic) criterion (see Figure 1) and may therefore be overprotective for the reasons discussed above with regard to the U.S. EPA (1985a) criteria.

The Inland Waters Directorate (Demayo et al., 1980) also recognized the importance of water hardness to lead toxicity, but only in water where sensitive species of aquatic organisms were present. Where sensitive species of fish (e.g., rainbow trout) were absent, the suggested criteria for lead (30 µg Pb/L) were the same for both soft and hard waters. The criterion of 30 µg Pb/L (total) in hard waters was set under the notion that it would provide an added safety factor. Nevertheless, in view of the decrease in lead toxicity with an increase in hardness of water, the recommended level of 30 µg Pb/L in hard waters, in the absence of sensitive species of organisms, could be overprotective.

The criteria for marine life (Table 2) were independent of water hardness because of the high and relatively constant hardness of marine waters. Only the American Fisheries Society (1979) and U.S. EPA (1985a) have put forward lead criteria for marine aquatic life. The procedures used by U.S. EPA (1985a) to derive lead criteria were the same for both freshwater and marine situations.

4.6.2 *Forms of Lead for Criteria*

Aquatic life criteria for lead have been expressed in terms of total, total recoverable, soluble, free, and acid-soluble lead. The total lead and total recoverable lead measurements include all forms of inorganically and organically bound, dissolved, and particulate lead in unfiltered samples, depending upon the completeness of the digestion step. The acid-soluble lead (defined as the lead that passes through 0.45 µm membrane filter after the sample is acidified with nitric acid to pH 1.5 to 2) measures forms of lead that are considered toxic to aquatic life or can be readily converted to toxic forms under natural conditions; these forms include carbonate and hydroxide precipitates/salts of lead in effluent that may be dissolved in receiving waters, and soluble and complexed forms of lead (e.g., EDTA complex of lead). This

measurement, however, does not include forms of lead that are occluded in minerals, clays and sand, or strongly sorbed to particulate matter. In the case of soluble lead, an unacidified sample is passed through a 0.45 µm membrane filter, and the metal analysis is carried out on the filtrate after acidification with nitric acid. As a result, it may contain a portion of the colloidal lead that may pass through the filter, but will exclude lead that may have been released from the unacidified particulate matter retained on the filter. Free lead is measured by pulse polarography and represents the dissolved fraction of the heavy metal.

Free forms of lead (Davies and Everhart, 1973; Davies et al., 1976) and soluble forms (Carpenter 1927, Wallen et al., 1957) have been considered more important in determining lead toxicity to aquatic life than total lead. Nevertheless, Ellis (1937) found that finely divided lead sulfide (insoluble) was lethal to fish in two months, suggesting that the suspended forms of lead may also be toxic. It is thought that particles of suspended (organic or inorganic colloids) matter may be retained or 'stuck' on gills of fish or other animals. The pH decreases in the surface layers of water on the gill, due to increased CO_2 levels from respiration, and may dissolve the lead, which could diffuse into the gill tissue. Also, some invertebrates are more susceptible to metals complexed by amino acids than to free metals, simply because these organisms ingest dissolved organic material as a nutrient (George, 1980; Borgmann and Charlton, 1984).

More recently, according to the U.S. EPA (1985a), acid-soluble lead was considered to be the most important measurement with regard to lead toxicity to aquatic life. However, the method for the measurement of· acid-soluble lead has not yet been developed or approved, and the U.S. EPA has recommended the use of total recoverable lead until an approved acid-soluble lead method is available. Understandably, a correlation between acid-soluble lead and toxicity to aquatic organisms will be a prerequisite for adoption of such forms of lead as criteria.

4.6.3 *Measurement of Complexing Capacity*

Another technique which may be used to estimate biologically available trace metals is the measurement of the complexing capacity of a waterbody. A number of methods have been used to measure complexing capacity. In almost all cases, copper has been the metal ion used, mainly because it forms relatively stable complexes with naturally occurring ligands (Hart, 1981). Since lead also forms complexes, as does copper, the conclusions provided by Hart (1981) in his review of trace metal complexing capacity of natural waters should also apply to lead. One of the major conclusions drawn by Hart related to the uncertainty in the analytical techniques currently used for measurement of metal complexing capacity. According to Hart, methods used to determine complexing capacity should ideally include the following characteristics:

(a) selectivity for the metal ion added.

(b) a capacity to measure the metal, or a related quantity at very low concentrations.

(c) a capacity to provide information on both metal-binding ligand concentration and conditional formation constant.

(d) no addition of other reagents that may disturb the equilibrium or bind with the added metal.

(e) usefulness over a wide range of pH values

(f) relative rapidity.

For a detailed discussion on measurement of trace metal complexing capacity, refer to Hart (1981). Because of the lack of lead-specific research, further discussion on the subject is not warranted at this time.

4.6.4 *Other Criteria*

The Canadian Food and Drug Directorate (1979) suggested a tolerance limit of 0.5 µg Pb/g (wet weight) in fish protein, but is now using site-specific assessments to predict human health risks from metals in fish (Health and Welfare Canada, 1987b).

The maximum allowable concentration in fresh fish and shellfish for human consumption recommended by the United Kingdom is 2 µg Pb/g (wet weight) and 10 µg Pb/g (wet weight), respectively (Mance et al., 198¼).

4.6.5 *Lead Mixed with Other Metals*

The following discussion is summarized from Singleton (1987) and deals with the effects of copper in mixtures with other metals. Although synergistic (more than additive) or antagonistic (le s than additive) effects have been shown for some mixtures of toxicants, it is generally agreed that, for most metal mixtures and other commonly-occurring constituents of sewage and industrial wastes, an additive model is adequate to describe the joint effect of toxicants in the mixture.

The IJC (1981) has outlined an approach to deal with mixtures of metals in the Great Lakes, using an additive model as follows:

The sum of the ratios of each metal concentration (Mi) to its respective objective concentration (Oi).should not exceed 1.0, i.e.:

$$\sum \frac{Mi}{Oi} \leq 1.0$$

While the IJC points out that this criterion is scientifically indefensible, its application to surveillance data analysis may serve as an early warning signal of harmful effects to aquatic life. Also, it is unlikely that adequate information required to formulate a scientifically defensible criterion will be available in the foreseeable future.

The EIFAC (1980) and Alabaster and Lloyd (1982) agree with the concentration addition concept for metal mixtures; however, they concluded that because concentrations lower than EIFAC-recommended values for single metals do not appear to contribute to the toxicity of mixture of metals, there is no need to adjust these values downward in such situations. Neither EIFAC nor Alabaster and Lloyd have recommended criteria for lead at this time.

4.7 Recommended Criteria

Criteria to protect aquatic life from acute and chronic effects of lead are based on information presented in U.S. EPA (1 985a), Davies et al. (1976), and Rivkin (1979).

4.7.1 *Freshwater Aquatic Life*

(a) The average concentration of total lead in water over a 30-day period (based on a minimum of 5 weekly samples) at an average hardness of water >8 mg/L as $CaCO_3$ should not exceed a value given by the expression: 3.31+exp (1.273 ln (average hardness)−4.705). Examples based on this relationship are as follows:

Average Water Hardness (mg/L $CaCO_3$)	30-day Average Lead Criterion (µg/L)
20	4.0
30	4.0
40	4.0
50	5.0
80	6.0
100	6.0
200	11.0
300	16.0

Criteria for intermediate values of hardness may be obtained from the above relationship.

In addition, not more than 20% (e.g., 1 in 5) of the values in a 30-day period should exceed 1.5 times the 30-day average criterion.

The 30-day average criterion does not apply at hardness of water ≤8 mg/L as $CaCO_3$.

(b) The maximum concentration of total lead in water at a water hardness ≤8.0 mg/L as $CaCO_3$ is set at 3.0 µg/L, Above water hardness of 8.0 mg/L as $CaCO_3$, the maximum concentration at any time is given by the expression exp(1.273 ln(hardness)−1.460), as shown below:

Water Hardness (mg/L $CaCO_3$)	Maximum Lead Criterion (µg/L)
≤8	3
20	10
30	18
40	25
50	34
80	61
100	82
200	197
300	330

(c) If natural (i.e., non-anthropogenic) lead levels in water exceed (a) or (b) above, then the increase in total lead to be allowed, if any, should be based on site-specific conditions.

4.7.2 *Marine and Estuarine Aquatic Life*

(a) The average concentration of total lead in water over a 30-day period (based on a minimum of 5 weekly samples) should not exceed 2 µg/L at any time. Not more than 20% (e.g., 1 in 5) of the values in a 30-day period should exceed 3 µg/L.

(b) The maximum concentration of total lead in water at any time should not exceed 140 µg/L.

(c) If natural levels exceed (a) or (b) above, then the increase in total lead to be allowed, if any, should be based on site-specific conditions.

4.7.3 *Application of Criteria*

This section outlines aspects that need consideration in the application of the recommended criteria for the purposes of assessing existing water quality and setting water quality objectives.

4.7.3.1 *Forms of Lead*

Toxicity of lead has been expressed in terms of total as well as soluble forms of lead (e.g., dissolved lead, free lead, acid-soluble lead, etc.). However, total lead is recommended for assessing water quality or setting water quality objectives for a given waterbody. The advantages of total lead are several fold: (i) all the lead that may potentially be toxic is included in the measurement. If the total lead concentration in water is within the criteria limits, then it is safe to conclude that no lead pollution exists; (ii) for comparison purposes, there is sometimes a considerable amount of historical background data available for total lead; and (iii) total lead measurement is routine and relatively inexpensive. Dissolved lead (field filtered and acidified) should also be measured when investigating lead contamination problems.

The main disadvantage of using total lead to assess water quality is that a large fraction of total lead may be in a form that is biologically unavailable (e.g., organically and inorganically complexed lead, lead sorbed by suspended matter, etc.). Therefore, total lead may over-estimate toxicity, especially in waters with high complexing capacity.

4.7.3.2 *Meeting the 3O-day Average Criterion*

Conceivably, the lead concentration in water may vary widely but still meet the 30-day average criterion. The converse may also be true. To illustrate this, the 30-day average lead levels in water based on five assumed measurements collected over a 30-day period are shown in Table 3. In one hypothetical situation that could be common, Case E, lead levels in water fluctuated up and down in the vicinity of the recommended average criterion, but did not exceed 1.5 times the recommended average levels at a given hardness of water. Yet the 30-day average lead level based on all five observations did not meet the criterion in this instance.

Table 3. Evaluation of 30-Day Average Levels Based on Five Observations Collected Over a 30-Day Period

	Case A		**Case B**		**Case C**		**Case D**		**Case E**	
Observation	H*	Pb**	H*	Pb**	H*	Pb**	H*	Pb**	H*	Pb**
1	20	1.0	20	1.0	7	1.0	150	8.0	80	7.0
2	30	2.0	30	2.0	10	1.5	80	5.0	75	6.0
3	40	2.5	25	6.5	15	2.0	50	4.0	85	8.0
4	25	7.0	35	3.0	5	3.0	120	7.0	100	8.5
5	35	3.0	40	7.5	8	4.5	100	11.0.	90	7.5
30-d Av. level	30	3.1	30	4.0	9	2.4	100	7.0	86	7.4
30-d Av. Criterion	30	4.0	30	4.0	9	3.0	100	6.0	86	6.0
Max. Criterion	20	10	20	10	8	3.0	50	34	75	57
Comment	Meets criterion: Only one of five observations exceeds 1.5 times the 30-d average criterion at given hardness and average does not exceed 30-d		Does not meet criterion: Two of five observations exceed 1.5 times the 30-d average criterion at given hardness.		Does not meet criterion: No observation exceeds 1.5 times the average criterion, but one exceeds the maximum criterion.		Does not meet criterion: Only one of five observations exceeds 1.5 times the 30-d criterion at given hardness, but the average exceeds the 30-d average criterion.		Does not meet criterion: No observation exceeds 1.5 times the 30-d criterion at given hardness, but the average exceeds the 30-d average criterion.	

	average criterion.				

*H = hardness of water in mg/L as $CaCO_3$
**Pb = concentration of lead in water in µg/L

In Case A, lead concentrations in water again fluctuated around the recommended average criterion levels; in only one observation (at hardness of water = 25 mg/L $CaCO_3$) out of the five the lead concentration exceeded 1.5 times the 30-day average criterion level. The 30-day average lead level, however, met the recommended 30-day average criterion.

Case D is similar to Case A, and only one observation (at hardness of water = 100 mg/L $CaCO_3$) out of the five exceeded 1.5 times the recommended average level. However, although the other four observations were closer to the recommended average criterion levels, the 30-day average level did not meet the criterion.

The 30-day average lead levels in Case B and Case C did not meet the recommended 30-day average criterion. The reasons being that, in Case B, two observations (at hardness of water = 25 and 40 mg/L $CaCO_3$) out of the five exceeded 1.5 times the recommended 30-day average criterion level, and in Case C, one observation (at hardness of water = 8 mg/L Ca C0 3) exceeded the maximum criterion level. Note that in both cases (Case B and Case C) the 30-day average lead levels (based on all five observations) appear to meet the criterion.

4.7.3.3 *Assessment of Existing Water Quality*

The criteria recommended in this document are primarily based on laboratory bioassays, which usually have been performed using soluble lead and dilution waters of low complexing capacities. The criteria are therefore likely to be over-protective for many waterbodies, especially those in which lead complexes may form. Based on total lead measurements, it cannot be concluded with certainty that lead pollution exists in a waterbody if the measurements exceed the criteria. However, if the measurements exceed the criteria levels, and if lead is anthropogenically generated, then a more intensive investigation of the site in question using other methods may be warranted. Other methods may include one or more assessment techniques such as measurement of complexing capacity, long-term bioassays on sensitive resident species using local water, and population studies on biota. Because of the complexity and cost of these alternative methods, they should be reserved for waterbodies with high fisheries values, which are threatened by a controllable lead source.

4.7.3.4 *Setting Water Quality Objectives*

Natural (i.e., non-anthropogenic) concentrations in a waterbody may not always meet the criteria. In a case where natural lead concentrations in water equal or exceed the criteria, the objective should be based on the natural levels and any increase in total lead to be allowed should be based on site-specific investigations. Atmospheric deposition is a major source of anthropogenic lead, and thus determining whether lead levels are natural or anthropogenic may be difficult.

When natural concentrations of total lead in undeveloped waterbodies are less than the criteria levels, then the criteria, or more stringent values if justified, should apply. In some cases, socio-economic factors may justify objectives which are less stringent than the criteria. Site-specific impact studies would be required in such cases.

4.8 Rationale

4.8.1 *Freshwater Aquatic Life*

The average (chronic) and the maximum (acute) criteria for lead recommended here are shown in Figure in relation to those recommended in the literature. The sources of data points along with other details of experiments, e.g., chemicals used, species tested, and effects observed are tabulated in Tables 6 and 7.

The 30-day average criterion based on a minimum of 5 weekly samples was designed to protect aquatic life from chronic effects of lead in water. The information presented in the U.S. EPA (1985a) and Davies et al. (1976) provided the basis for this criterion. According to the U.S. EPA (1985a), the recommended four-day average (chronic) lead criterion is a function of water hardness (mg/L as $CaCO_3$) and is given by the expression exp (1.273 ln (hardness)−4.705). However, this relationship yields chronic lead values which appear to be over-restrictive (see section 4.2.1). Davies et al. (1976) noted that the maximum acceptable lead concentration for rainbow trout (*Salmo gairdneri*) in 19-month tests carried out with soft water (28 mg/L as $CaCO_3$) ranged between 4.1 and 7.6 µg/L. Other investigators have shown agreement with Davies et al. results (Goettl et al., 1976; Hodson, 1976; Davies and Everhart, 1973). In designing chronic lead criteria in this document, the 30-day average lead concentration in water of hardness 30 mg/L as $CaCO_3$ was set at 4.0 µg/L (the lower limit of the maximum acceptable toxic concentration range given by Davies et al.). In order to express the criteria as a function of water hardness, the 30-day average criterion was increased or decreased in accordance with the relationship (the four-day average lead concentration = exp(1.273 ln(hardness)-4.705)) developed by the

U.S. EPA (1985a). The final relationship between 30-day average lead criterion and water hardness is shown below:

30-day Average Lead Criterion (µg/L) = 3.31+exp(1.273 ln(average hardness)−4.705).

Note that the lowest concentration of lead in water causing chronic effects in aquatic organisms was reported for Lake Ontario (Wilson, 1982 - as reported in Hodson et al., 1984). Whereas no significant effects of lead concentration up to 3.3 µg/L were observed, Wilson reported a 41% reduction in the phototactic response for *Diaptomus sicilis* at 3.8 µg/L lead in Lake Ontario water at 15°C and hardness of water = 135 mg/L $CaCO_3$. These data, however, were not considered in defining chronic lead criteria for B.C. for the following reasons: (a) the lethal or effective concentration of 3.3 µg/L lead at water hardness = 135 mg/L $CaCO_3$ would lie well below any data point in Figure 1, and (b) the chronic lead criteria proposed by all jurisdictions including the IJC (1980; 5 µg/L for Lake Ontario) where the results of Wilson's work (in progress at the time) were first reported, are well above the no effect level of 3.3 µg/L lead in water of hardness 135 mg/L $CaCO_3$ (Figure 1). Both (a) and (b) suggest that more work is required in support of Wilson's (1982) observations.

The maximum concentration of lead permitted at any time is designed to protect aquatic life from acute (short-term lethal) effects of lead in water. This criterion was adopted from the U.S. EPA (1985a) acute (one-hour average) criterion, which *is* expressed in terms of water hardness (measured in units of mg/L as $CaCO_3$) as exp(1.273 ln(hardness)−1.460) and was intended to protect 95% of a group of diverse genera, unless a commercially or recreationally important species is very sensitive. However, instead of the one-hour average, the acute criteria in this document were expressed in terms of a maximum concentration. Also, this relationship was applied to waters of hardness >8.0 mg/L $CaCO_3$.

Because the recommended relationships for the average and maximum criteria intersect at hardness of about 8 mg/L as $CaCO_3$, it is recommended that the lead criterion at hardness of water <8 mg/L as $CaCO_3$

be determined by the maximum concentration. The maximum concentration of lead in waters of hardness <8 mg/L as $CaCO_3$ is arbitrarily set at 3.0 µg/L.

Both the 30-day average and the maximum lead criteria are expressed in terms of total lead in unfiltered water. This choice was made because (i) both soluble and insoluble forms of lead can cause toxicity to aquatic organisms (section 4.1.2), and (ii) the method for the acid-soluble lead has yet to be developed. The U.S. EPA (1985a) recommended that total recoverable lead (= total lead) be used until an approved method for acid-soluble lead is developed.

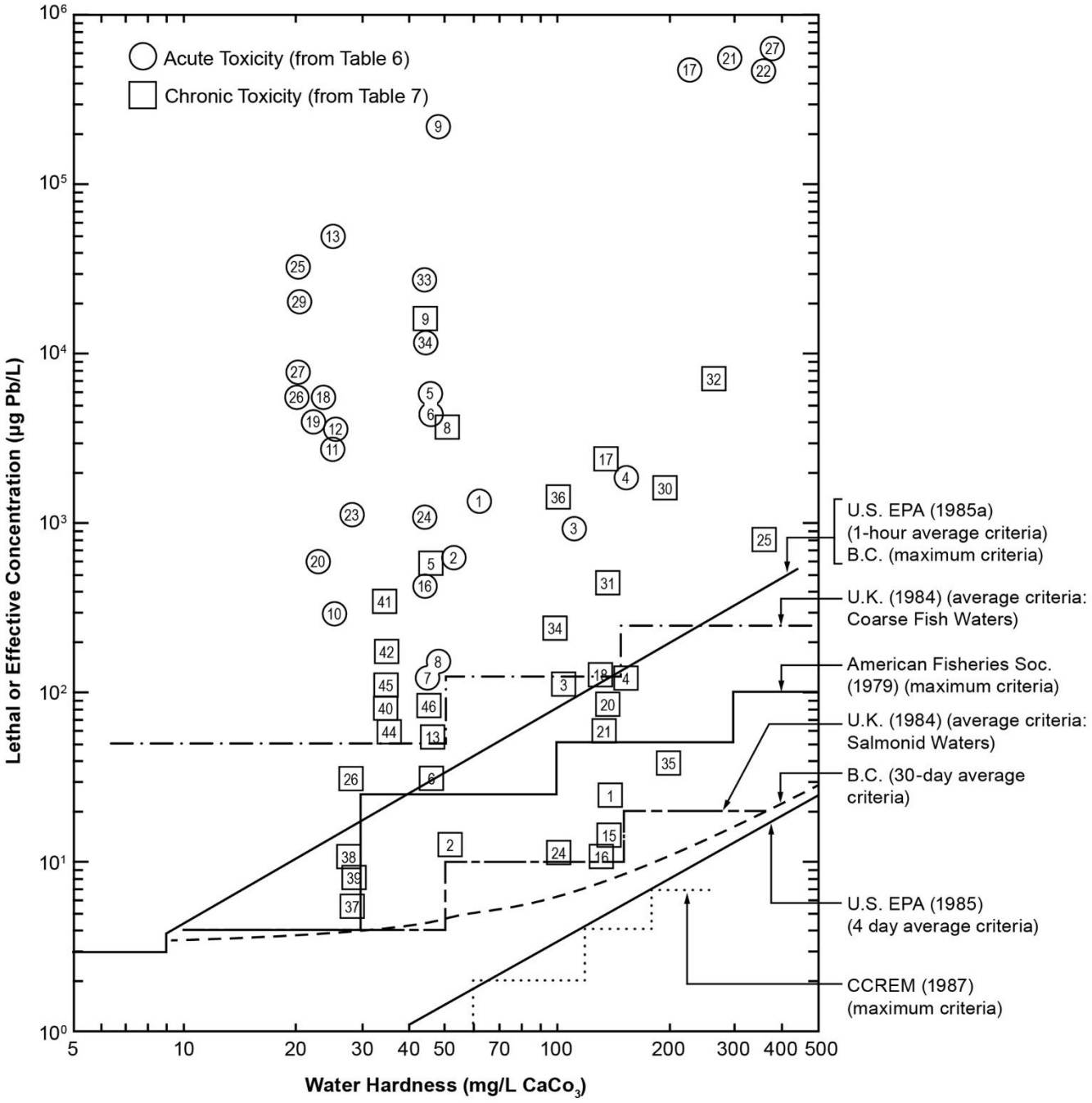

Figure 1. Acute and Chronic Toxicity of Lead to Freshwater Organisms and Selected Water Quality Criteria

As indicated above, the U.S. EPA (1985a) recommended one-hour and four-day averaging periods for defining short-term (acute) and long-term (chronic) toxicity of lead to aquatic life. Since concentrations maintained above an acceptable level for a long enough period of time, and substantial fluctuations in the concentrations may enhance the adverse effects of pollutants on aquatic organisms, these periods were chosen (a) to restrict fluctuations in the pollutant concentration in receiving waters, and (b) to restrict the length of time that the concentration in the receiving water can be continuously above a criterion value (U.S. EPA, 1985b). The problem with these averaging periods is that very frequent monitoring would be required to determine whether the criteria were being met. Monitoring this frequently is costly and normally impractical. Consequently, we have recommended a maximum (replacing the one-hour average) and a 30-day average (based on a minimum of 5 weekly samples) to allow for the collection of a more reasonable number of samples. In addition, restrictions have been placed on the size and frequency of fluctuations above the average criterion (as outlined below) to address the concerns that led the U.S. EPA to specify a 4-day averaging period.

In order to protect ecosystems from frequent excursions above the criteria, the U.S. EPA (1985a,b) also recommended that the one-hour and four-day average concentrations must not exceed the criteria more than once every three years. Most aquatic systems were considered by the U.S. EPA to recover from most pollution events in about three years. However, (a) the long-term ecological consequences of periodic stress conditions are yet unknown, and (b) some ecosystems may take more than three years to recover from such stresses (U.S. EPA, 1985a,b). Excursions in lead levels above the criteria every three years may mean that the aquatic system will be in a perpetual state of recovery. For these reasons, the concept of ecosystem recovery period was not adopted in this document.

It is, however, necessary to guard against fluctuations above the 30-day average level, which do not exceed either the maximum or the 30-day average criteria, but which could cause significant effects if they persisted for a long time. For instance, it is theoretically possible for the lead concentration to be twice the 30-day level for 15 days, or four times the 30-day level for 7.5 days, and still meet the 30-day average criterion, provided the concentration for the remaining time in the 30-day period is zero. In very soft water (hardness 10 mg/L Ca C0 3), the ratio between the maximum and 30-day average criteria is 1.5 and thus the maximum effectively limits the magnitude of fluctuations above the 30-day average level. In water of hardness >10 mg/L $CaCO_3$, the ratio between the maximum and average criteria is relatively high and ranges from 1.5 to 33 (at hardness = 350 mg/L $CaCO_3$); the maximum criterion in this case will not provide an effective upper limit for fluctuations in lead levels. Consequently, it is recommended that for water of hardness >10 mg/L $CaCO_3$, the lead concentration should not exceed 1.5 times the 30-day average criterion in more than 20% of the samples (e.g., one in five, 2 in 10, etc.) collected in a 30-day period. Figure 1 suggests that exceeding the 30-day average criterion by. more than 1.5 times (for a prolonged period) may result in significant effects on aquatic life.

It would be unrealistic to apply the recommended toxicity criteria to waterbodies where natural lead levels exceed the criteria. In those circumstances, therefore, it is recommended that little or no increase in lead levels above the natural background should be permitted; the amount of any increase would depend upon site-specific characteristics.

CCREM (1987) guidelines for lead to protect freshwater aquatic life were expressed as maximum values (depending upon water hardness), and thus differed from a two-number approach used by the U.S. EPA and by British Columbia in this document. CCREM (1987) based their maximum guidelines on U.S. EPA (1985a) 4-day average (chronic) lead criteria, except that CCREM (1987) guidelines are expressed as a step, rather than a continuous function of water hardness (see Figure 1). Consequently, the CCREM (1987) guidelines are overprotective because: (i) the U.S. EPA 4-day average criteria are overprotective as outlined in Section 4.6.1, and (ii) U.S. EPA averages are used as maximums. The adoption of U.S. EPA

average (chronic) criteria as maximums by CCREM results in criteria that are simple to use, but overrestrictive because they do not allow for short-term excursions above the chronic level which can frequently occur in receiving water, but do not harm aquatic life.

4.8.2 *Marine Aquatic Life*

The criteria for the protection of marine aquatic life were based on information presented in U.S. EPA (1985a). The one-hour (acute) criterion recommended by the U.S. EPA was 140 µg/L. This value is less than half (0.44) of the lowest marine LC50 of 315 µg/L noted for the mummichog (*Fundulus heteroclitus*; Dorfman, 1977), and is accepted in this document as the maximum criterion in marine water. It is expected that this concentration would cause little or no mortality even if it persisted for four days. Research by the U.S. EPA (1985a) suggests that 0.44 of the 96-h LC50 would be ≤96-h LC1 in about 75% of cases.

The lowest level (a 12-day EC50 of 3.7 µg/L) causing long-term (chronic) effects (50% reduction in growth rate) was reported by Rivkin (1979) for the diatom *Skeletonema costatum*. In view of these results, the chronic (four-day average) criterion of 5.6 µg/L recommended by the U.S. EPA (1985a) was modified. In order to provide adequate protection from long-term effects, a 30-day average (chronic) criterion of 2 µg/L of lead (i.e., one-half of the lowest chronic effect level, based on a minimum of 5 weekly samples) is recommended in this document. Data on effects of fluctuations in lead levels in water on marine aquatic life are rare. It is believed, however, that the effects will be the same in both fresh and marine waters. It is, therefore, further recommended that no more than 20% of the samples collected over a 30-day period should exceed 1.5 times the recommended 30-day average criterion, or 3 µg/L.

Since the hardness of marine waters was assumed to be high and relatively constant, the above criteria do not vary with hardness.

The CCREM (1987) did not recommend guidelines for marine aquatic life.

4.8.3 *Monitoring Strategy*

Although soluble forms of lead are considered more important in determining lead toxicity to aquatic life, insoluble forms have also been shown to be toxic (Carpenter, 1927; Wallen et al., 1957; Davies and Everhart, 1973; Davies et al., 1976; Ellis, 1937). In an aqueous environment, the solubility of lead is controlled by pH, partial pressure of CO_2 (g), and the characteristics of the solid phase. Both pH and CO_2 (g) concentrations are subject to large fluctuations due to fish respiration. Also, no present technique seems to provide a suitable means of separating the truly dissolved lead from the suspended (colloidal) lead. It is therefore recommended that both dissolved and total lead be measured in water in order to assess the impact of lead on aquatic life.

4.8.4 *Lead Mixed with Other Metals*

In view of the discussion presented in section 4.6.5 no separate criterion for lead mixed with other metals has been recommended here. It is believed that lead in mixtures will behave like other heavy metals (e.g., copper), and at the levels recommended in section 4.6 should not contribute to the toxicity of a mixture of toxicants, especially at chronic levels.

However, if multiple toxicity in a waterbody with high fisheries values is suspected for some reason, then bioassays could be used to assess the situation.

Table 4. Acute Toxicity of Lead to Freshwater Organisms (From: U.S. EPA, 1985a and Demayo et al., 1980)

Data Point (Fig. 1)	Species	Chemical	Hardness (mg/L as $CaCO_3$)	LC50 or EC50 (µg Pb/L)	Reference*
1	Snail, *Aplexa hypnorum*	Lead nitrate	61	1 340	Call et al., 1983
2	Cladoceran, *Daphnia magna*	Lead nitrate	54	612	Chapman et al., 1985?
3	Cladoceran, *Daphnia magna*	Lead nitrate	110	952	Chapman et al., 1985?
4	Cladoceran, *Daphnia magna*	Lead nitrate	152	1 910	Chapman et al., 1985?
5	Cladoceran, *Daphnia pulex*	Lead nitrate	45	5 100	Mount & Norberg , 1984
6	Cladoceran, *Simocephalus vetulus*	Lead nitrate	45	4 500	Mount & Norberg , 1984
7	Amphipod, *Gammarus pseudolimnaeus*	Lead nitrate	46	124	Spehar et al., 1978
8	Amphipod, *Gammarus pseudolimnaeus*	Lead nitrate	48	140	Call et al., 1983
9	Midge, *Tanytarsus dissimilis*	Lead nitrate	48	224 000	Call et al., 1983
10	*Asellus meridianus* (non-tolerant from control river)	Unknown	25	280	Brown, 1976
11	*Asellus meridianus* (from Sn− and Cu− contaminated river)	Unknown	25	2 800	Brown, 1976
12	*Asellus meridianus* (tolerant from Pb contaminated river)	Unknown	25	3 500	Brown, 1976
13	Rotifer, *Philodina acuticornis*	Lead-chloride	25	50 500 & 50 400	Buikema et al., 1974
14	Snail, *Goniobasis livescens*	Lead acetate	137-171	71 000	Cairns et al., 1974
15	Snail, *Lymnaea emarginata*	Lead acetate	137-171	14 000	Cairns et al., 1974
16	Cladoceran, *Daphnia magna*	Lead chloride	44	450	Biesinger and Christensen, 1972
17	Tubificid worm, *Tubifex tubifex*	Lead nitrate	224	450 000	Qureshi et al., 1980
18	*Cyclops abyssorum*	Lead acetate	23	5 500	Baudouin and Scoppa, 1974
19	*Eudiaptomus padanus*	Lead acetate	23	4 000	Baudouin and Scoppa, 1974
20	*Daphnia hyalina*	Lead acetate	23	600	Goettl et al., 1972; Davies &Everhart, 1973; Davies et al., 1976
21	Rainbow trout, *Salmo gairdneri*	Lead nitrate	290	542 000	Goettl et al., 1972; Davies &Everhart, 1973; Davies et al., 1976
22	Rainbow trout, *Salmo gairdneri*	Lead nitrate	353	471 000	Goettl et al., 1972; Davies &Everhart, 1973; Davies et al., 1976

23	Rainbow trout, *Salmo gairdneri*	Lead nitrate	28	1 170	Holcombe et al., 1976
24	Brook trout, (18 mos.), *Salvelinus fontinalis*	Lead nitrate	44	4 100	Pickering & Henderson, 1966
25	Goldfish, *Carassius auratus*	Lead chloride	20	31 500	Pickering & Henderson, 1966
26	Fathead minnow, *Pimephales promelas*	Lead chloride	20	5 580	Pickering & Henderson, 1966
27	Fathead minnow, *Pimephales promelas*	Lead chloride	20	7 330	Pickering & Henderson, 1966
28	Fathead minnow, *Pimephales promelas*	Lead chloride	360	482 000	Pickering & Henderson, 1966
29	Guppy (6 mos.), *Poecilla reticulata*	Lead chloride	20	20 600	Pickering & Henderson, 1966
30	Bluegill, *Lepomis macrochirus*	Lead chloride	20	23 800	Pickering & Henderson, 1966
31	Bluegill, *Lepomis macrochirus*	Lead chloride	360	442 000	Pickering & Henderson, 1966
32	Fathead minnow, *Pimephales promelas*	Lead acetate	20	7 480	Pickering & Henderson, 1966
33	Fathead minnow, *Pimephales promelas*	Lead acetate	44	27 800	Curtis and Ward, 1981
34	Fathead minnow, *Pimephales promelas*	Lead fluoroborate	44	1 200	Curtis and Ward, 1981
35	Channel catfish, *Ictalurus punctatus*	Lead arsenate	45	>100 000	Johnson & Finley, 1980

*As quoted in U.S. EPA (1985a) and Demayor et al. (1980).

Table 5. Chronic Toxicity of Lead to Freshwater Aquatic Organisms (From: U.S. EPA, 1985a and Demayo et al., 1980)

Data Point (Fig. 1)	Species	Chemical	Hardness (mg/L as $CaCO_3$)	Effect	Result* (µg Pb/L)	Reference**
1	Snail, *Lymnaea palustris*	Lead nitrate	139	-	12-54 (25.4)	Borgmann et al., 1978
2	Cladoceran, *Daphnia magna*	Lead nitrate	52	-	9-16.7 (12.3)	Chapman et al., 1985?
3	Cladoceran, *Daphnia magna*	Lead nitrate	102	-	78-181 (118.8)	Chapman et al., 1985?
4	Cladoceran, *Daphnia magna*	Lead nitrate	151	-	85-193 (128.1)	Chapman et al., 1985?
5	Snail, *Physa integra*	Lead nitrate	46	no effect on survival	565	Spehar et al., 1978
6	Cladoceran, *Daphnia magna*	Lead chloride	45	reproductive impairment	30	Biesinger & Christensen, 1972
7	Amphipod, *Gammarus pseudolimnaeus*	Lead nitrate	46	LC50	28.4	Spehar et al., 1978
8	Mayfly, *Ephemerella grandis*	Lead nitrate	50	LC50	3500	Nehring, 1976
9	Mayfly, *Emphemerella subvaria*	Lead sulfate	44	LC50	16000	Warnick & Bell, 1969
10	Stonefly, *Pteronarcys dorsata*	Lead nitrate	46	no effect on survival	565	Spehar et al., 1978
11	Stonefly, *Acroneuria lycorias*	Lead suphate	44	-	64000	Warnick & Bell, 1969
12	Caddisfly, *Hydropsyche betteni*	Lead nitrate	44	LC50	32000	Warnick & Bell, 1969
13	Caddisfly, *Brachycentrus* sp.	Lead nitrate	46	no effect on survival	565	Spehar et al., 1978
14	Midge, (embryo-3rd instar), *Tanytarsus dissimilis*	Lead nitrate	47	LC50	258	Anderson et al., 1980
15	Rainbow trout, *Salmo gairdneri*	Lead nitrate	135	inhibition of ALA-D activity	13	Hodson, 1976
16	Rainbow trout, (12 mos), *Salmo gairdneri*	Unknown	135	inhibition of ALA-D activity	10	Hodson et al., 1977

Data Point (Fig. 1)	Species	Chemical	Hardness (mg/L as $CaCO_3$)	Effect	Result* (µg Pb/L)	Reference**
17	Rainbow trout, *Salmo gairdneri*	Lead nitrate	135	LC50	2400	Hodson et al., 1978a
18	Rainbow trout, *Salmo gairdneri*	Lead nitrate	135	black-tails in 3 of 10 remaining fish	120	Hodson et al., 1978a; Sippel et al., 1983
19	Rainbow trout, *Salmo gairdneri*	Lead nitrate	135	affected RBC, iron content, & ALA-D in blood	13	Hodson et al., 1978a
20	Rainbow trout, *Salmo gairdneri*	Unknown	135	all fish had black tails & decrease in ALA-D in blood	87	Hodson et al., 1979a, 1980
21	Rainbow trout, *Salmo gairdneri*	Lead nitrate	135	64% inhibition of ALA-D activity and black tails in 88% of fish	65	Hodson et al., 1979b
22	Rainbow trout, *Salmo gairdneri*	Lead nitrate	135	45% inhibition of ALA-D activity	25	Hodson et al., 1983b
23	Rainbow trout, (embryo, larva), *Salmo gairdneri*	Lead chloride	101	EC50 (death and deformity)	220	Birge et al., 1980
24	Rainbow trout, (embryo, larva), *Salmo gairdneri*	Lead chloride	101	EC1 (death and deformity)	10.3	Birge et al., 1980 and 1981
25	Rainbow trout, (fingerling), *Salmo gairdneri*	Lead nitrate	353	Lordoscoliosis	850	Goettl et al., 1972; Davies et al., 1976
26	Rainbow trout, (sac fry), *Salmo gairdneri*	Lead nitrate	28	Lordoscoliosis	31	Goettl et al., 1972; Davies et al., 1976
27	Brook trout, (12 mos.), *Salvelinus fontinalis*	Lead nitrate	135	inhibition of ALA-D activity	90	Hodson et al., 1977
28	Brook trout, (embryo-21 days), *Salvelinus fontinalis*	Lead chloride	44	elevation of ALP and ACH activity	525	Christensen et al., 1975
29	Brook trout, (12 mos.) *Salvelinus fontinalis*	Lead chloride	44	decrease of hemoglobin and inhibition of GOT activity	58	Christensen et al., 1977

Data Point (Fig. 1)	Species	Chemical	Hardness (mg/L as $CaCO_3$)	Effect	Result* (µg Pb/L)	Reference**
30	Goldfish (embryo, larva), *Carassius auratus*	Lead chloride	195	EC50 (death and deformity)	1660	Birge, 1978
31	Goldfish (<12 mos.), *Carassius auratus*	Lead nitrate	135	inhibition of ALA-D activity	470	Hodson et al., 1977
32	Commom carp (embryo), *Cyprinus carpio*	Lead acetate	360	EC50 (hatch)	7293	Kapur & Yadav, 1982
33	Pumpkinseed (>12 mos), *Lepomis gibbosus*	Lead nitrate	135	inhibition of ALA-D activity	90	Hodson et al., 1977
34	Largemouth bass, (embryo, larva), *Micropterus salmoides*	Lead chloride	99	EC50 (death and deformity)	240	Birge et al., 1978
35	Narrow-mouthed toad, (embryo, larva), *Gastrophryne carolinenis*	Lead chloride	195	EC50 (death and deformity)	40	Birge, 1978
36	Marbled salamander, (embryo, larva), *Ambystoma opacum*	Lead chloride	99	EC50 (death and deformity)	1460	Birge et al., 1978
37	Rainbow trout, (pre-hatch fry), *Salmo gairdneri*	Lead nitrate	28	MATC	4-7.6 (5.5)	Davies et al., 1976
38	Rainbow trout, (post-hatch fry), *Salmo gairdneri*	Lead nitrate	28	MATC	7.2-14.6 (10.2)	Davies et al., 1976
39	Rainbow trout, *Salmo gairdneri*	Lead nitrate	28	MATC	6-11.9 (8.4)	Davies & Everhart, 1973
40	Bluegill, *Lepomis macrochirus*	Lead nitrate	34	-	70-120 (91.7)	Sauter et al., 1976
41	Northern pike, *Esox lucius*	Lead nitrate	34	-	253-483 (350)	Sauter et al., 1976
42	White sucker, *Catostomus commersoni*	Lead nitrate	34	-	119-253 (174)	Sauter et al., 1976

Data Point (Fig. 1)	Species	Chemical	Hardness (mg/L as $CaCO_3$)	Effect	Result* (µg Pb/L)	Reference**
43	Channel catfish, Ictalurus *punctatus*	Lead nitrate	34	-	75-136 (101)	Sauter et al., 1976
44	Lake trout, *Salvelinus namaycush*	Lead nitrate	34	-	48-83 (63.1)	Sauter et al., 1976
45	Rainbow trout, *Salmo gairdneri*	Lead nitrate	35	-	71-146 (101.8)	Sauter et al., 1976
46	Brook trout, *Salvelinus fontinalis*	Lead nitrate	44	-	58-119 (83.1)	Holcombe et al., 1976

* Values in parenthesis are geometric means of the observed results.

** As quoted in U.S. EPA (1985a) and Demayo et al. (1980).

5. Wildlife

5.1 Effects

Accidental ingestion of lead shot mistaken for seed or grit appears to be the major cause of lead poisoning in waterfowl. In hunting areas of marshlands, Bellrose (1959) estimated that the number of lead pellets per acre of marsh bottom ranged from 25 000 to over 30 000. Anderson (1975) reported that lesser scaup (a species of duck) containing 1 to 10 lead pellets in the gizzard lost an average of 30 to 35% of their body weight before dying as compared to healthy birds.

Different species of waterfowl show different tendencies to ingest lead shot. For instance, gadwall (*Anas strepera*), teal (*Amus* sp.) and shoveler (*Spatula clypeata*) show lower incidence of ingesting lead shot than redhead (*Aythya americana*), canvas back (*Aythya valisneria*), and ringnecked ducks (*Aythya collaris*). Mallards are by far the most susceptible to lead ingestion.

The toxicity of lead in waterfowl varies with species, sex, and their food intake characteristics (i.e., quantity and quality). Female waterfowl are about twice as sensitive to lead poisoning as males (Jordan, 1952). Mallards dosed with lead were less affected when maintained on a balanced diet than the birds fed on a diet low in nutrients (Finley et al., 1976a). Some green plants, e.g., *Ceratophyllum demersum* (coontail), are thought to alleviate the effects of lead poisoning in waterfowl (Locke et al., 1966). Analyses revealed that lead levels in livers of poisoned waterfowl ranged from 9 to 27 mg/kg in Canada geese (Adler, 1944), 18 to 37 mg/kg in whistling swans (Chupp and Dalke, 1964), and 6 to 20 mg/kg in mallards (Longcore et al., 1974). These levels are 6 to about 40 times higher than background, which is about 1.0 mg/kg of the wet weight of liver (Bagley and Locke, 1967).

The activity of the enzyme δ-aminolevulinic acid dehydratase (ALA-D) was inhibited by 40% after 3 and 12 weeks in mallards fed with a diet containing 25 and 5 mg Pb/kg, respectively. The inhibition of ALA-D activity by one pellet of lead was 88% (Finley et al., 1976b). Dieter et al., (1976) indicated that a blood lead level of 0.2 mg/L was dangerous for wildfowl as it caused 75% inhibition of ALA-D activity; 75% inhibition of ALA-D has been used to indicate dangerous accumulation of lead in humans.

Plants may be another source of lead for ducks. The food of ducks ranges from 10-to 100% plant material (Martin et al., 1961), and industrial and automotive lead could contribute to the lead burden of marsh plants in areas exposed to these sources.

Flynn et al. (1975) noted that the amount of lead in moose peaked in June and July, and was related to the onset of new browse growth and not the levels of lead in water. However, no correlation was found between lead concentrations in plants and herbivorous muskrats (Everett and Anthony, 1976).

5.2 Criteria from the Literature

No water quality criteria specifically for wildlife were found in the literature.

5.3 Recommended Criterion

Due to the lack of sufficient relevant information in the literature, specific water quality criteria to protect wildlife from the harmful effects of lead were not developed.

The criterion to protect wildlife from harmful effects of lead in water is the same as that specified for livestock watering in section 6.3.

The concentration of total lead in water for wildlife use should not exceed 100 µg/L at any time.

5.4 Rationale

The use of livestock criteria for wildlife in waters devoid of sensitive or desirable aquatic life is based on the fact that, in all likelihood, the safe concentration of lead for both groups of animals is similar in magnitude. As an example, the toxic dose of 12 mg Pb/kg body weight/d for waterfowl is well within the toxic range of 2.4 to 320 mg/kg body weight/d for livestock as noted in the joint publication of Agriculture Canada and B. C. Ministry of Agriculture (Puls, 1981). The rationale for the-livestock watering criteria is presented in section 6.4.

For waters inhabited by aquatic life, the criteria recommended to protect relatively sensitive aquatic life appear more than adequate to protect wildlife.

The CCREM (1987) did not recommend criteria for wildlife; however, the livestock criterion that we are recommending for wildlife is the same as the CCREM (1987) guideline for livestock watering.

6. LIVESTOCK WATER SUPPLY

6.1 Effects

The accidental ingestion of lead-containing products such as lead-based paints (discarded surplus paint or flakes of lead paints on old walls), discarded oil, linoleum, plumber's lead, putty, discarded lead-acid batteries, and pastures contaminated by industrial lead operations are the most common causes of lead poisoning in domestic animals (Christian and Tryphonas, 1971; Hatch and Funnell, 1969; NAS, 1972; Schmitt et al., 1971). Lead in effluent from lead mining and processing may also be a hazard.

As in humans, the bone of domestic animals acts as a sink for lead thereby providing an important detoxification mechanism. However, as the bone reaches its saturation point, the level of lead in soft tissue and blood increases and leads to symptoms of lead poisoning (Hatch, 1977). In a review of lead poisoning with laboratory animals, Tornabene et al. (1977) concluded that the decrease of ALA-D enzyme (which regulates hemoglobin production) was the most significant effect of lead poisoning of rats. Another important biochemical effect of lead poisoning in chick embryos was the suppression of the production of antibodies normally caused by certain viral and bacterial agents (King et al., 1978).

Several factors determine lead toxicity to domestic animals. These include species, age (young animals are more susceptible), reproduction stage and health of the animals, quality of animal nutrition, rate of lead ingestion, and form of lead (Hatch, 1977),

Excessive amounts of lead from ingested forage were found to be the cause of a chronic debilitating disorder in six young horses near the smelter at Trail, British Columbia (Schmitt et al., 1971). Lead in oven-dried, unwashed forage varied from 6 to 264 mg/kg dry matter within 8 km of the smelter; air contained 3 to 6 µg/m^3 lead. Horses receiving 2.4 mg Pb/kg b.w. (body weight)/d from hay died, whereas 6 to 7 mg Pb/kg b.w./d was estimated to be the minimum lethal dose for cattle (Hammond and Aronson, 1964). Pregnant ewes fed at a rate of 3 mg Pb/kg b.w./d of lead acetate showed no adverse effects, but died of severe emaciation due to lead poisoning when fed at a rate of 5.7 mg Pb/kg b.w./d (James et al., 1966). A value of 6 mg Pb/kg b.w./d was suggested as the threshold toxic level for non-pregnant ewes (Allcroft and Blaxter, 1950). The toxic cumulative and single oral doses suggested by Agriculture Canada and the B.C. Ministry of Agriculture (Puls, 1981) are as shown below:

	Cattle	Dogs	Horses	Poultry	Sheep
			- mg Pb/kg b.w./d -		
Toxic Cumulative Dose	5-7	3-30	2.4-7	320	3.0
Toxic Single Oral Dose	200-800	600-1000	-	-	60-80

In more recent studies, doses of 5 mg Pb/kg b.w./d for 7 days, or 2.7 mg Pb/kg b.w./d for 21 days were highly toxic to young calves fed a whole milk diet (Bratton et al., 1981; Zmudzki et al., 1983). *A* dose of 1 mg Pb/kg b.w./d could be lethal in approximately 60 days to calves consuming a milk diet (Zmudzki et al., 1985).

Morrison et al. (1977) reported that lambs fed 400 mg Pb/kg of a diet low in calcium and sulphur (but adequate in other minerals) died within five weeks. The diet of 400 mg Pb/kg is equivalent to about 12 mg Pb/kg b.w./d, assuming an average feed requirement for a lamb of 0.03 kg d.m. (dry matter)/kg b.w./d. However, the animals survived up to 10 months when fed a diet containing all minerals or a low phosphorus diet. A decrease in lead toxicity to lambs as a result of sulphate sulphur in the diet was reported by Quarterman et al. (1977). Bratton and Zmudzki (1984) noted that the calves on a milk diet accumulated 12 (in bone) to 25 (in kidneys) times more lead than calves of the same age fed a diet of grain and hay.

The addition of 2 and 5 mg Pb/kg as lead acetate or lead oxide to the ration had no effect on the mortality of chickens and hatching results in a 329-d laying test. Also, in a 198-d supplementary test, the addition of 80 mg Pb/kg of feed did not increase the lead levels of the yolks and whites (Vogt et al., 1977). A decrease in growth and an increase in incidence of perosis in broiler chickens, was reported by Chah et al. (1976) at lead concentrations of 300 mg Pb/kg of diet (or ≥9.0 mg Pb/kg b.w./d, assuming average daily feed requirement for broilers = 0.03 kg dry matter/kg b.w.). Dietary lead concentrations greater than 630 mg Pb/kg of diet sharply reduced the egg production of hens (Hermayer et al., 1977).

Prenatal exposure of lambs to blood-lead levels of 0.34 mg Pb/L caused behaviour (visual discrimination) problems in the animals, while lambs exposed to blood-lead levels of 0.17 mg Pb/L did not differ from the controls. Diets of 4.5 mg Pb/kg b.w./d and 2.3 mg Pb/kg b.w./d were needed to maintain blood-lead levels of 0.34 and 0.17 mg Pb/L, respectively in the pregnant ewes (Carson et al., 1974a,b). Hapke (1973) noted that the ingestion of more than 0.5 mg Pb/kg b.w./d of lead by sheep inhibited the activity of ALA-D in blood and increased the ·excretion of δ-aminolevulinic acid (δ-ALA) in urine. Sheep with lead intake **of** up to 3 mg Pb/d did not retain any of the ingested lead (Jones and Clement, 1972).

Williams (1939) reported chronic lead poisoning among animals by 0.18 mg Pb/Lin soft water, while Pierse (1938) noted occurrence of chronic lead poisoning in cows with water containing 2.4 mg Pb/L. Chronic poisoning of horses due to lead in spring and stream water and grasses was reported by Singer (1976). The lead concentration in water ranged between 0.01 and 16 mg/L, with most of the samples having a lead concentration in the range of 0.5 to 1.0 mg/L; grasses contained 5 to 20 mg/kg (dry basis) of lead.

6.2 Criteria from the Literature

Lead criteria for livestock water supply from various jurisdictions are shown in Table 6. The lowest lead criterion of 50 µg/L was reported by Alberta and Saskatchewan, Both Alberta and Saskatchewan did not establish water use categories while setting water quality criteria, but based the criteria on the most sensitive water use. Thus, it is probable that the 50 µg/L criterion is for a more sensitive water use than livestock watering, such as drinking water or aquatic life.

Considering horses to be the most sensitive animals to lead poisoning, the Inland Waters Directorate (Demayo et al., 1980) recommended two values for lead criteria in the livestock water supply; one (500 µg/L) where horses are present and the other (1000 µg/L) for other livestock. However, these criteria exceed the criterion for lead of 100 µg/L recommended by U.S. EPA (1972), Ontario Ministry of Environment (1984), and CCREM (1987).

6.3 Recommended Criterion

The criterion to protect livestock from lead in water is based on the susceptibility of horses to lead poisoning. Horses appear to be as sensitive, if not more, than cattle or sheep (Puls, 1981).

The concentration of total lead in livestock drinking water supply should not exceed 100 µg/L at any time.

6.4 Rationale

The rationale for the criteria recommended in this document for the protection of livestock was based on information presented in Singer (1976). The logic followed is shown below:

(1) Singer (1976) reported cases of lead poisoning in horses feeding on hay containing 5 to 20 mg/kg dry matter and water with lead concentrations mostly ranging from 0.5 to 1.0 mg/L (the actual lead content of water ranged from 0.1 to 16 mg/L).

Table 6. Lead Criteria for Livestock Water Supply

Criteria Statement	Criteria Value (µg Pb/L)	Jurisdiction	Date	Reference
Recommended upper limit of 0.1 mg/L for lead in livestock waters	100	U.S. EPA	1972	U.S. EPA (1972)*
The derived working level for lead in stock drinking water is 0.5 mg/L	500	Australia	1974	Hart (1974)
Maximum concentration for lead in water = 0.05 mg/L	50	Saskatchewan	1975	SSWQO (1975)**
Maximum concentration for lead in water = 0.05 mg/L	50	Alberta	1977	ASWQO (1977)+
Lead in water used for livestock should not exceed 0.1 mg/L	100	Ontario	1984	OME (1984)#
Recommended water quality objectives (as total lead) in livestock water of 0.5 mg/L where horses are present, and 1.0 mg/L where horses are absent	500 (horses present) 1000 (horses absent)	Inland Waters Directorate	1980	Demayo et al. (1980b)
Desirable annual average concentration in livestock water = 0.1 mg/L	100	U.K.	1984	Mance et al. (1984)
Maximum acceptable concentration of total lead in water of 0.5 mg/L	500	Manitoba	1983	MDEWSH (1983)++
Maximum recommended concentration of lead in water for livestock should be 0.1 mg/L	100	CCREM	1987	CCREM (1987)##

* U.S. Environmental Protection Agency
** Saskatchewan Surface Water Quality Objectives
\+ Alberta Surface Water Quality Objectives
++ Manitoba Department of Environment and Workspace Safety and Health
\# Ontario Ministry of Environment
Canadian Council of Resource and Environment Ministers

(2) Assuming a dry feed requirement of 0.03 kg d.m./kg b.w./d and a water requirement of 3 L/kg d.m., the amount of lead consumed by a horse from both water and food sources collectively in step 1, is calculated to range from 200 to 700 µg/kg b.w./d.
(3) Thus, from step 2, a daily intake of 200 to 700 µg Pb/kg b.w., with an average value of 450 µg Pb/kg b.w., may be considered unsafe for horses.
(4) Lead levels in vegetation collected in the vicinity of well-travelled highways and lead-related industry in British Columbia may be well above the 20 mg Pb/kg d.m. noted in step 1, and above the normal levels of 1.0 mg Pb/kg d.m. in the diet of a horse (Puls, 1981; Garrett, 1985).
(5) In lead-affected areas, horses consuming water containing 500 µg Pb/L may, therefore, have a lead intake exceeding the critical limits as noted in step 3.
(6) The above analyses suggest that 500 µg Pb/L in livestock water supply, as recommended by the Inland Waters Directorate (Demayo et al., 1980), may not be safe for the livestock. Chronic poisoning among animals due to lead in water at a concentration of 180 µg Pb/L, has been reported in the literature (Williams, 1939).
(7) To protect livestock from lead, it is therefore recommended that the maximum concentration of lead in livestock drinking water be 100 µg/L.

Although the recommended lead level in step 7 is perhaps overprotective, especially for relatively more resistant species of livestock, it does not restrict an area to only the more tolerant species.

The recommended criterion is the same as that recommended by the CCREM (1987).

7. IRRIGATION

7.1 Effects

Lead is not an essential element for plant growth. However, it is ubiquitous in soil-water-plant systems. In general, lead is tightly held in soils by sorption and/or by forming complexes with both inorganic and organic components of the soil. As a result, only a small amount of the total lead content of soil is available to plants. A study carried out at the University of Guelph (Ontario) showed that the first crop of rye grass removed only 0.004 to 0.017 kg Pb/ha from a soil supplied with 1.5 to 116 kg Pb/ha through an application of sewage sludge (Bates et al., 1975). In British Columbia, the ratio of lead between lettuce and soil supporting the crop was 0.015; the lead concentration in lettuce was 3 mg/kg, while in the soil it was 200 mg/kg (John, 1975). Wilson and Cline (1966) estimated that only 0.003% to 0.005% of the total lead in soil was taken up by barley plants. Since lead tends to accumulate near the soil surface, shallow-rooted crops are exposed to relatively higher lead concentrations than the deep-rooted crops (Walsh et al., 1976; John, 1975).

Lead can enter the plant through the root system or the leaves. Besides the form and concentration of the metal in the environment, several factors related to soil (pH, cation exchange capacity, texture, temperature, moisture content, organic matter content, etc.), crop (species, rooting depth, anatomy, etc.), and climate (precipitation, temperature, etc.) determine lead uptake by plants.

Lagerwerff (1971) noted that the lead (also zinc and cadmium) concentration in plants was lower when the soil pH was increased from 5.9 to 7.2. A reduction in the availability of lead to plants as a result of the formation of lead compounds (e.g., organic complexes, hydroxides, phosphates, etc.) of very low solubility, was reported by MacLean et al. (1969) and Walsh et al. (1976) in response to the addition of organic matter, lime, and phosphate to soils. Foliar absorption of lead in lettuce and radish plants from foliar application of Pb $(NO_3)_2$ solution has been demonstrated by Hemphill and Rule (1975).

The total and soluble lead content of soils has been quoted to range from 2 to 200 mg/kg with a mean of 16 mg/kg, and from 0.05 to 5 mg/kg, respectively (Demayo et al., 1980). Agricultural soils in British Columbia have been shown to contain an average of 10.4 mg/kg (total lead) in the top 16 cm of soil (John, 1975). In a study concerning the lead content of vegetables and soils in British Columbia, Warren et al. (1970) found no correlation between soil (total) and plant lead contents. In this study, the vegetable (lettuce, cabbage, carrot, beet, potato, tomato, peas, spinach, etc.) lead content ranged from 0.4 to 1.9 mg/kg (dry weight), and soils supporting the crop showed a range of 6 to 230 mg/ kg. Plant lead uptake increased little in response to an increase in total lead concentration of soils (Ter Haar, 1970; McIntyre, et al., 1977; Dowdy and Larson, 1975; Lagerwerff, 1972; John and Van Laerhoven, 1972; Andersson and Nilsson, 1972). Other investigations, however, suggested that soluble or extractable lead was a better indicator of the availability of lead to plants (MacLean et al., 1969; Kerin, 1975; Dudas and Pawluk, 1975). Although a strong positive correlation was found between total and extractable lead in heavily contaminated soils (extractable lead >90 mg/kg), this correlation was not evident in uncontaminated areas (Kerin, 1975). Cole (1977) noted that the addition of 1 800 mg Pb/kg to soil of various lead salts resulted in almost identical concentrations of soluble lead immediately after the addition of the lead salt or after 48 hours. These soluble lead levels ranged from 0.14% to 0.58% of the total lead added.

Different parts of plants accumulate lead to a different degree. In general, the fruiting and flowering parts accumulate the smallest amounts of lead. In a greenhouse experiment, Motto et al. (1970) noted that the edible parts of carrot, tomato, corn, lettuce, and potato plants grown in soils containing 76 to 164 mg Pb/kg had lead levels of 1.3 (corn kernels) to 16 mg/kg (carrots). The lead concentrations were relatively higher in the leaves (up to 74 mg/kg in corn leaves) than other parts of these plants.

When the same experiments were conducted with sand containing 1 to 4 mg Pb/kg, the edible parts of these plants contained lead levels of 0.6 to 21 mg /kg. However, the maximum levels of lead (up to 764 mg/kg) were associated with the roots of tomato, potato, and lettuce plants. Similar observations were made by Jones et al. (1973) while working with perennial rye grass in solution cultures containing 1 mg Pb/L as Pb $(NO_3)_2$; the plants removed 95% of the lead from the nutrient solution, and the roots contained 57% to 80% of the added lead.

Toxicity of lead to plants differs with the plant species. Beans grown in soil containing 820 mg Pb/kg (dry weight) showed poor growth and spotty discolouration, whereas peanuts were unaffected (Berg, 1970). A significant difference in the yield of oats and red clover grown in pots could occur at soil lead concentration above 50 mg/kg (Von Hodenberg and Finck, 1975); however, John and Van Laerhoven (1972) reported no effect on the yield of oats in response to an addition of 1000 mg Pb/kg as $Pb(NO_3)_2$, $PbCl_2$, or $PbCO_3$. Lead chloride concentrations of 125 mg Pb/kg have been reported to decrease the Ca, Mg, K, and P uptake by corn plants and reduce their growth in greenhouse experiments (Walker et al., 1977).

Cole (1977) reported that lead compounds added to soil modified soil biological activity; a reduction in amylase synthesis was initiated at concentrations of 450 mg Pb/kg (as $PbCl_2$ and PbS) of soil. Debosz et al. (1985) noted that the addition of 500 or 1 000 mg Pb/kg to natural or clay amended soils did not have an appreciable adverse effect on the extent of carbon mineralization after 16 days of incubation.

7.2 Criteria from the Literature

Table 7 shows the criteria from various jurisdictions to protect crops from harmful effects of lead in irrigation water. The limits for lead in irrigation water are strikingly similar in magnitude among most jurisdictions. This similarity in lead criteria suggests a common source and perhaps a lack of new information with regard to lead toxicity in the soil-plant-water system.

Table 7. Lead Criteria for Irrigation

Criteria Statement	Criteria Value (µg Pb/L)	Jurisdiction	Date	Reference
Recommended maximum concentrations of lead in irrigation water are: 5.0 mg/L for continuous use on all soils; 10.0 mg/L for a 20-year period on neutral and alkaline fine textured soils	5 000 10 000	U.S. EPA	1972	U.S. EPA (1972)*
Recommended working level for lead in irrigation water = 5. 0 mg/L	5 000	Australia	1974	Hart (1974)
Recommended maximum concentrations of lead for irrigation water: used continuously on all soils=5.0 mg/L; for up to 20 years on fine textured soils of pH 6.0 to 8.0 = 10.0 mg/L	5 000 10 000	Ontario	1984	OME (1984)+
Recommended maximum concentrations of lead in irrigation waters: for continuous ·use 5.0 mg/L; for intermittent use = 10.0 mg/L	5 000 10 000	Inland Waters Directorate	1980	Demayo et al. (1980b)
Maximum average concentration for the normal irrigation period = 2 mg/L	2 000	U.K.	1984	Mance et al. (1984)
Maximum acceptable concentration of total lead in irrigation water = 10.0 mg/L	10 000	Manitoba	1983	MDEWSH++ (1983)
Concentration of total lead in irrigation water should not exceed 0.2 mg/L for continuous use on all soils, and 2.0 mg/L for use on neutral and alkaline fine textured soils for up to 20 years	200 2 000	CCREM	1987	CCREM# (1987)

* U.S. Environmental Protection Agency
\+ Ontario Ministry of Environment;
++ Manitoba Department of Environment and Workplace Safety and Health
\# Canadian Council of Resource and Environment Ministers

The Canadian (CCREM, 1987) and the U.K. (Mance et al., 1984) water quality, guidelines recommended much lower lead levels in irrigation water. Both these guidelines were aimed at limiting the accumulation of lead in agricultural soils to acceptable levels. The U.K. guidelines assumed an irrigation rate of 500 mm annually for 50 years, whereas CCREM based their calculations on an irrigation rate of 1000 mm/y for 100 years. Assuming soil bulk density of 1 500 kg/m^3 and accumulation depth for applied lead of 0.15 m, the Canadian guidelines allow a build-up in agricultural soils of about 100 (in acid soils) to 200 mg/kg (in neutral and alkaline soils), and the U.K. guidelines allow a buildup of 225 mg/kg (dry weight) in agricultural soils. It would appear that the Canadian (CCREM) guidelines were much more conservative than all other jurisdictions in Table 7.

The justification for safe lead levels of 5 mg/Land 10 mg/Lin irrigation water, recommended by the Inland Waters Directorate (IWD), was based upon accumulation of added lead in the top 10 cm of a soil with bulk density of 1 500 kg/m^3. It was assumed that 1.0 m^3 of water was needed to irrigate 1.0m^2 of agricultural land in a year, and that losses of lead due to plant uptake or leaching through the profile were negligible.

Based on these assumptions, Demayo et al. (1980, IWD) concluded that the maximum lead con centration of 5 mg/Lin irrigation water would increase the lead content of soil by 3.3 mg/kg/y. However, this increase ls in error, and instead should be 33.3 mg/kg/y as shown below:

Mass of 1 m^2 of 10 cm deep soil 1.0 m^2 × 0.1 m × 1500 kg/m^3 = 150 kg

Amount of Pb added to soil (1.0 m^2 × 0.1 m) by irrigation water (5 mg Pb/L) @ 1.0 m^3/ y

= 5 mg Pb/L × 1.0 m^3/ y × 10^3L/m^3 × 1/150 kg soil

= 33.3 mg Pb/kg/y

7.3 Recommended Criteria

It is recommended that (a) for neutral and alkaline fine textured soils the maximum total lead concentration in irrigation water should not exceed 400 µg Pb/L at any time; (b) the concentration of total lead in irrigation water for continuous use on all other soils should not exceed 200 µg/L at any time.

7.4 Rationale

Criteria to protect vegetation from adverse effects of lead in irrigation water are based on information presented in CCREM (1987), Motto et al. (1970), and Tornabene et al. (1977). Laboratory experiments suggest that detrimental effects of lead on plants may occur at soil lead concentrations well below 100 mg/kg (Van Hodenberg and Finck, 1975). On the other hand it has been suggested that a concentration of at least 000 mg Pb/kg of dry weight must be present in soils (except sandy soils) before any effects of lead can be observed (Tornabene et al., 1977). On the average, lead in edible portions of plants excluding roots (Table 8) and in pastures (Table 9) grown in soils containing 1 000 mg Pb/kg (dry weight) is 24. 3 and 15.7 mg/kg (dry matter), respectively (similar values of lead in animal feed were used in establishing a lead criterion for livestock water supply in Chapter 7).

Sands containing lower lead levels (1.0 to 4.0 mg/kg) tend to contribute as much lead to the edible parts of vegetables grown in them as soils containing higher lead levels (76 to 164 mg/kg; Motto et al., 1970). This may have been the consequence of the lower cation exchange capacity (CEC) of the sandy soils (e.g., CEC of fine sandy loam = 5-10 meq/ 100 g) compared to that of fine textured soils (e.g., CEC of clay loam = 15-30 meq/100 g). In view of these facts, the critical level of lead in sandy soils must be reduced at least by a factor of 3. Further reduction in soil lead level may be desirable in acidic sandy soils, because lead uptake by plants increases at low pH's. The effects of pH and CEC factors suggest that the safe concentration of lead in soil (causing no adverse effects) should be less than 300 mg/kg as opposed to 1 000 mg/kg suggested by Tornabene et al. (1977) above.

As with the Canadian water quality guidelines (CCREM, 1987), therefore, the lead levels of 100 mg Pb/kg (dry weight) for acid and 200 mg Pb/kg (dry weight) for neutral and alkaline agricultural soils were considered to be safe levels in this document. The criterion proposed by CCREM (1987) for acidic soils was, therefore, adopted. However, the 20-year irrigation period for neutral and alkaline soils was considered too short to provide long-term protection to soils. Instead, the criterion for neutral and alkaline soils was derived for a long-term (100 years) or continuous use of land, based on assumptions specified in the CCREM (1987). Consequently, the recommended criteria are the same or consistent with the CCREM (1987) guidelines.

An example of calculations involved in the derivation of criteria in this document is shown below:

Given (i) the bulk density of a soil to be 1 500 k g/ m^3, (ii) the concentration of lead in irrigation water at 400 µg/L, (iii) the irrigation rate of 1.0 $m^3/m^2/y$. and (iv) the lead in irrigation water to be retained in the top 15 cm of the soil, the soil in question will accumulate lead at the rate of:

$$0.4\ \frac{mg\ Pb}{L} \times 1.0\ \frac{m^3}{m^2 y} \times \frac{1}{0.15m} \times \frac{m^3}{1500\ kg\ soil} \times 1000 \frac{L}{m^3} = 1.8 \frac{mg\ Pb}{kg - soil\ y}$$

provided there is no loss of lead from the soil. At this rate, it will take over 100 years for an alkaline fine textured soil to accumulate lead to the recommended safe level of 200 mg/kg.

In practice, lead levels of this magnitude are unlikely to be encountered in irrigation water, and for the majority of cases the much lower criteria for the protection of aquatic life will apply to waters being used for irrigation.

Table 8. Mean Values of Lead in Various Parts of Seven Edible Plants at Three Rates of Soil Contamination in British Columbia (John and Van Laerhoven, 1972)

Plant		Plant Pb (mg/kg dry weight)		
Name	**Part**	**Control**	**200 mg Pb/kg**	**1 000 mg Pb/kg**
Leaf lettuce	leaves	2.5 a[a]	3.0 a	54.2 b
	roots	5.8 a	84.5 b	867.7 c
Spinach	leaves	0.7 a	7.9 b	39.2 c
	roots	4.7 a	73.3 a	unavailable
Broccoli	leaves	7.2 a	8.4 a	18.4 b
	roots	6.5 a	83.0 a	745.6 b
Cauliflower	leaves	5.3 a	6.3 a	11.8 b
	roots	2.5 a	55. 1 b	532.2 c
Oats	grains	3.2 a	4.4 a	4.9 a
	husks	11. 1 a	11 .8 a	1 6.4 a
	leaves	6.0 a	6.8 a	20. 1 a
	stalks	1.6 a	2.5 a	9.2 a
	roots	4.5 a	82.o a	396.6 b
Radish	tops	3.7 a	9.9 a	1 4.3 a
	tubers	6.3 a	7.0 a	44.6 b
Carrot	tops	2.3 a	8.0 b	17.6 c
	tubers	1.9 a	5.3 a	41.0 b
	roots	8.9 a	241.7 b	561.4 c

[a]Values within a row of means, followed by the same letter do not differ significantly at the 5% level.

Table 9. Pb in Herbage from Upland and Lowland Pastures in Wales (Alloway and Davies, 1971)

Sample Number	Site	Pb in plants (mg/kg dry matter)	Dominant plant species[a]	Soil Pb Content (mg/kg)	Soil pH
1a	Frongoch	2.6	R, T, M, Cl		
1b	Frongoch	1.9	R, T, M	60	5.7
1c	Frongoch	5.4	Cl		
2	Ceunant	17.7	R, T, C, Cl	2 400	5.7
3	Ceunant	1. 5	R, T, C, Cl	630	5.0
4	Ceunant	10.2	R, T, C, Cl	-	4.8
5	Cwm Ystwyth	74.2	Fo, S	3 600	4.0
6	Clarach	17.0	Fo, S	3 680	5.2
7	Parys Mt.	66.4	C	890	5.1
8	Parys Mt.	40.9	B		

Sample Number	Site	Pb in plants (mg/kg dry matter)	Dominant plant species[a]	Soil Pb Content (mg/kg)	Soil pH
9	Parys Mt.	6.1	C	220	5.0
10	Parys Mt.	2.5	C, R, T	340	5.1
11	Llanafan	21.1	R, M, T, Cl	2 050	5.0
		Av. = 15.7			

[a]Plant species:
R = ryegrass (*L. perenne*) M = meadow fescue (*F. pratensis*);
B = bent (*A. tenuis*);
T = timothy (*P. pralense*);
Fo = sheep fescue (*F. ovina*);
S = sedges (*Carex* spp.);
Cl = clovers (*Trifolium* spp.);
C = cocksfoot (*D. glomerata*).

8. RESEARCH AND DEVELOPMENT NEEDS

Several research needs, as noted below, were identified during preparation of this document.

1. Since man's lead intake comes from food, water, and air and since all intakes vary greatly and independently, it is not correct to correlate only one intake with blood lead values or other effects. A knowledge of lead intakes from all sources is lacking in detail and needs further refinement. Epidemiological studies so far have produced low correlation between, for instance, lead in water and blood-lead levels. Further studies need to define more carefully critical blood-lead levels and their relationship to lead uptake from all sources.
2. Research to date has established that hardness of water influences lead toxicity to aquatic life. Both chronic and acute toxicity data in very soft waters (hardness <20 mg/L $CaCO_3$) are scant. The toxic effects of lead in very soft water need to be evaluated; there are a large number of fresh bodies of water in British Columbia with hardness less than 20 mg/L as Ca C0 3, but which contain valuable fisheries resources.
3. Organolead compounds are highly toxic to aquatic life even in small concentrations. Although organolead compounds are rarely found under natural environmental conditions, these compounds are produced industrially in large quantities and inevitably escape into the environment. The sublethal effects due to chronic exposure of freshwater biota to organolead compounds, and the fate of organolead compounds in the aquatic environment need to be examined.
4. The use of 'total lead' is still perceived as a practical and conservative basis for water quality criteria and objectives. This is perhaps due to the variety of forms of lead and lack of definite information about their relative toxicities. Identification of the chemical forms of lead (including their interconversion), knowledge of their toxicities to aquatic life, and the development of suitable analytical methodology are needed.
5. Objectives/criteria developed to date have addressed the biological effects of single compounds. This is largely due to a lack of data dealing with interactions among multiple contaminants. Research is needed to examine the significance of metal mixtures found in the environment at or less than objective/criteria levels and to correlate their toxicity with effects found in the environment.
6. The behaviour of lead in natural waters depends upon precipitation equilibria and complexing with inorganic and organic ligands. Quantitative relationships between lead complexing capacity and toxic potency need to be investigated. A method for measuring complexing capacity needs to be developed in British Columbia.

7. Minimum daily intakes of lead causing harmful effects in livestock have not been well defined in the literature. Investigations with low doses of lead are needed to define the minimum dose causing clinical and sub-clinical toxicosis in livestock.
8. There is little information on the toxicity of lead to marine organisms. Acute and chronic bioassays are needed on marine species frequenting British Columbia coastal waters.

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