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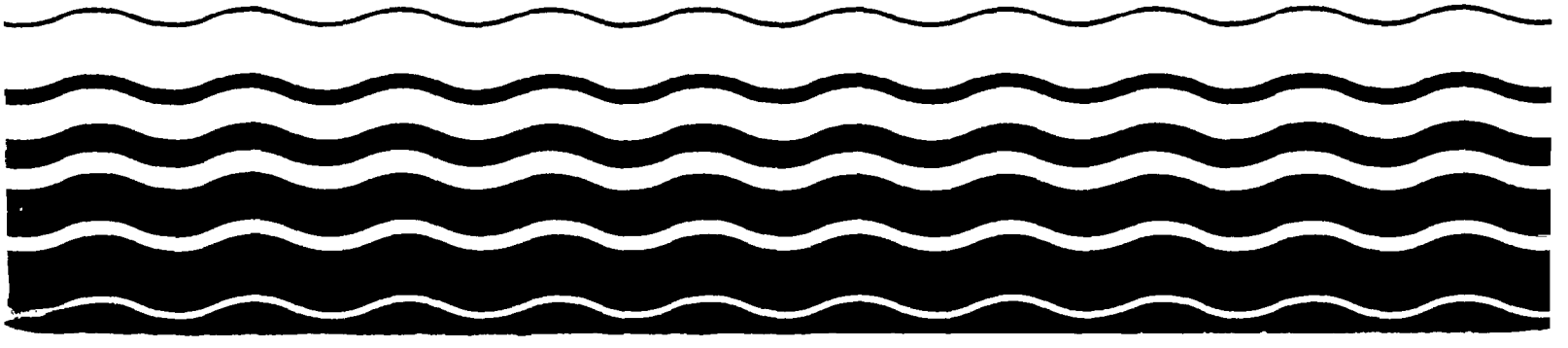
# Ambient Water Quality Criteria for Chromium



# AMBIENT WATER QUALITY CRITERIA FOR

# CHROMIUM

Prepared By
U.S. ENVIRONMENTAL PROTECTION AGENCY

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# FOREWORD

Section 304 (a)(1) of the Clean Water Act of 1977 (P.L. 95-217), requires the Administrator of the Environmental Protection Agency to publish criteria for water quality accurately reflecting the latest scientific knowledge on the kind and extent of all identifiable effects on health and welfare which may be expected from the presence of pollutants in any body of water, including ground water. Proposed water quality criteria for the 65 toxic pollutants listed under section 307 (a)(1) of the Clean Water Act were developed and a notice of their availability was published for public comment on March 15, 1979 (44 FR 15926), July 25, 1979 (44 FR 43660), and October 1, 1979 (44 FR 56628). This document is a revision of those proposed criteria based upon a consideration of comments received from other Federal Agencies, State agencies, special interest groups, and individual scientists. The criteria contained in this document replace any previously published EPA criteria for the 65 pollutants. This criterion document is also published in satisifaction of paragraph 11 of the Settlement Agreement in Natural Resources Defense Council, et. al. vs. Train, 8 ERC 2120 (D.D.C. 1976), modified, 12 ERC 1833 (D.D.C. 1979).

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

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

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CHROMIUM

## CRITERIA

### Aquatic Life

For total recoverable hexavalent chromium the criterion to protect freshwater aquatic life as derived using the Guidelines is 0.29 µg/l as a 24-hour average and the concentration should not exceed 21 µg/l at any time.

For freshwater aquatic life the concentration (in µg/l) of total recoverable trivalent chromium should not exceed the numerical value given by $e^{(1.08[\ln(\text{hardness})]+3.48)}$ at any time. For example, at hardnesses of 50, 100, and 200 mg/l as $CaCO_3$ the concentration of total recoverable trivalent chromium should not exceed 2,200, 4,700, and 9,900 µg/l, respectively, at any time. The available data indicate that chronic toxicity to freshwater aquatic life occurs at concentrations as low as 44 µg/l and would occur at lower concentrations among species that are more sensitive than those tested.

For total recoverable hexavalent chromium the criterion to protect saltwater aquatic life as derived using the Guidelines is 18 µg/l as a 24-hour average and the concentration should not exceed 1,260 µg/l at any time.

For total recoverable trivalent chromium, the available data indicate that acute toxicity to saltwater aquatic life occurs at concentrations as low as 10,300 µg/l, and would occur at lower concentrations among species that are more sensitive than those tested. No data are available concerning the chronic toxicity of trivalent chromium to sensitive saltwater aquatic life.

## Human Health

For the protection of human health from the toxic properties of chromium (III) ingested through water and contaminated aquatic organisms, the ambient water criterion is determined to be ~~170~~ 57 mg/l.

For the protection of human health from the toxic properties of chromium (III) ingested through contaminated aquatic organisms alone, the ambient water criterion is determined to be ~~3,433~~ 1,200 mg/l.

The ambient water quality criterion for chromium (VI) is recommended to be identical to the existing water standard for total chromium which is 50 µg/l. Analysis of the toxic effects data resulted in a calculated level which is protective of human health against the ingestion of contaminated water and contaminated aquatic organisms. The calculated value is comparable to the present standard. For this reason a selective criterion based on exposure solely from consumption of 6.5 grams of aquatic organisms was not derived.

# INTRODUCTION

Chromium is a metallic element which can exist in several valence states. However, in the aquatic environment it virtually is always found in valence states +3 or +6. Hexavalent chromium is a strong oxidizing agent which reacts readily with reducing agents such as sulfur dioxide to give trivalent chromium. Cr (III) oxidizes slowly to Cr (VI), the rate increasing with temperature. Oxidation progresses rapidly when Cr (III) adsorbs to MnO, but is interfered with by Ca (II) and Mg (II) ions. Thus, accumulation would probably occur in sediments where chemical equilibria favor the formation of Cr (III), while Cr (VI), if favored, would presumably dissipate in soluble forms. Hexavalent chromium exists in solution as a component of an anion, rather than a cation, and therefore, does not precipitate from alkaline solution. The three important anions are: hydrochromate, chromate, and dichromate. The proportion of hexavalent chromium present in each of these forms depends on pH. In strongly basic and neutral solutions the chromate form predominates. As pH is lowered, the hydrochromate concentration increases. At very low pH the dichromate species predominates. In the pH ranges encountered in natural waters the proportion of dichromate ions is relatively low. In the acid portion of the environmental range, the predominant form is hydrochromate ion (63.6 percent at pH 6.0 to 6.2) (Trama and Benoit, 1960). In the alkaline portion of the range, the predominant form is chromate ion (95.7 percent at pH 8.5 to 7.8) (Trama and Benoit, 1960).

Trivalent chromium in solution forms numerous types of hexa-coordinate complexes (Cotton and Wilkinson, 1962). The best known and one of the most stable of these is the amine class (complexes include aquo- ions, acido- complexes (which are anionic), and poly-nuclear complexes. Complex formation can prevent precipitation of the hydrous oxide or other insoluble forms at pH values at which it would otherwise occur.

Chromium salts are used extensively in the metal finishing industry as electroplating, cleaning agents, and as mordants in the textile industry. They also are used in cooling waters, in the leather tanning industry, in catalytic manufacture, in pigments and primer paints, and in fungicides and wood preservatives. Kopp (1969) reported a mean surface water concentration in the United States of 9.7 µg/l, based on 1,577 samples. Trivalent chromium is recognized as a essential trace element for humans. Hexavalent chromium in the workplace is suspected of being carcinogenic.

# REFERENCES

Cotton, F.A. and G. Wilkinson. 1962. Advanced Inorganic Chemistry. Interscience Publishers, John Wiley and Sons, Inc., New York.

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Aquatic Life Toxicology*

## INTRODUCTION

Chromium is a chemically complex metal which occurs in valence states ranging from -2 to +6. The hexavalent and trivalent chromium compounds are the biologically and environmentally significant forms of the element, but they have very different chemical characteristics. Hexavalent chromium is very soluble in natural water. Although it is a strong oxidizing agent in acidic solutions, hexavalent chromium is relatively stable in most natural waters. Trivalent chromium tends to form stable complexes with negatively charged organic or inorganic species and thus its solubility and toxicity vary with water quality characteristics such as hardness and alkalinity. Most of the trivalent chromium species are either cationic or neutral and the hexavalent species are anionic.

Information on the toxic effects of chromium on freshwater organisms is relatively extensive, but the data base for hexavalent chromium is greater than that for trivalent chromium. The data indicate that water hardness has an insignificant influence on the toxicity of hexavalent chromium in fresh water; thus, it is not necessary to develop a criterion as a function of water quality. On the other hand, the freshwater data indicate that water hardness has a significant influence on the acute toxicity of trivalent chromium.

Most of the saltwater acute and chronic toxicity data are for hexavalent chromium. Only a few studies have been conducted on the effects of triva

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*The reader is referred to the Guidelines for Deriving Water Quality Criteria for the Protection of Aquatic Life and Its Uses in order to better understand the following discussion and recommendation. The following tables contain the appropriate data that were found in the literature, and at the bottom of each table are calculations for deriving various measures of toxicity as described in the Guidelines.

lent chromium on saltwater organisms, probably because of the low solubility of trivalent chromium in saltwater. The kinetics of precipitation of trivalent chromium in saltwater systems are complex but regardless of its form, trivalent chromium may still be ingested and bioconcentrated by filter or deposit feeding bivalve mollusc and polychaete species.

Of the analytical measurements currently available, water quality criteria for trivalent chromium and for hexavalent chromium are probably best stated in terms of total recoverable trivalent chromium and total recoverable hexavalent chromium, respectively, because of the variety of forms of chromium that can exist in bodies of water and the various chemical and toxicological properties of these forms. The forms of chromium that are commonly found in bodies of water and are not measured by the total recoverable procedure, such as the chromium that is a part of minerals, clays, and sand, probably are forms that are less toxic to aquatic life and probably will not be converted to the more toxic forms very readily under natural conditions. On the other hand, forms of chromium that are commonly found in bodies of water and are measured by the total recoverable procedure, such as the free ion, and the hydroxide, carbonate, and sulfate salts, probably are forms that are more toxic to aquatic life or can be converted to the more toxic forms under natural conditions. Because the criterion is derived on the basis of tests conducted on soluble inorganic salts of chromium, total chromium and total recoverable chromium concentrations in the tests will probably be about the same and a variety of analytical procedures will produce about the same results. Except as noted, all concentrations reported herein are expected to be essentially equivalent to total recoverable trivalent or hexavalent chromium concentrations. All concentrations are expressed as chromium, not as the compound.

## EFFECTS

### Acute Toxicity

#### Hexavalent Chromium

As shown in Table 1, the freshwater data base available for hexavalent chromium has numerous acute values for thirteen species from ten different families. Acute values have been reported for six freshwater invertebrate species from five families. These acute values range from 67 µg/l for a scud to 59,900 µg/l for a midge. The scud Gammarus pseudolimnaeus was by far the most sensitive species tested with an $LC_{50}$ value about one-fiftieth of the next lower acute value. Invertebrate species are generally more sensitive to hexavalent chromium than fish species. As shown in Table 3, the species mean acute values for five of the six invertebrate species are less than that of any fish species. The rotifer Philodina roseola was about three times as sensitive to chromium at 35°C as at 5°C (Schaffer and Pipes, 1973).

Table 1 also lists acute values for seven freshwater fish species, of which more than 70 percent of the values are for the goldfish and fathead minnow. The 96-hour $LC_{50}$ values range from 17,600 µg/l for the fathead minnow to 249,000 µg/l for the goldfish. Static tests with unmeasured concentrations and flow-through tests with measured concentrations gave similar results (Pickering, 1980).

Wallen, et al. (1957) studied the toxicity of hexavalent chromium to mosquitofish in turbid water using potassium and sodium salts of both dichromate and chromate (Table 6). Based on chromium, both dichromate salts were more toxic than the chromate salts. The geometric means of the two values were 95,000 µg/l and 120,000 µg/l for the dichromate and chromate, respectively. Trama and Benoit (1960) studied the toxicity of chromium to

the bluegill using potassium dichromate and potassium chromate. The 96-hour $LC_{50}$ values were 110,000 µg/l for the dichromate salt and 170,000 µg/l for the chromate salt. They attributed the lower $LC_{50}$ value of the dichromate salt to its greater acidity, because chromium is slightly more toxic at lower pH values.

The toxicity of hexavalent chromium to the bluegill in soft and hard water was tested at 18°C and 30°C (Academy of Natural Sciences of Philadelphia, 1960). At 18°C the 96-hour $LC_{50}$ values were 113,000 µg/l in soft water and 135,000 µg/l in hard water. Similar results were obtained at 30°C with the 96-hour $LC_{50}$ values being 113,000 µg/l in soft water and 130,000 µg/l in hard water.

Pickering and Henderson (1966) tested the toxicity of potassium dichromate to the fathead minnow and bluegill in soft and hard water. The 96-hour $LC_{50}$ values for the fathead minnow in soft and hard water were 17,600 and 27,300 µg/l, respectively. The corresponding values for the bluegill were 118,000 µg/l and 133,000 µg/l.

The data from Adelman and Smith (1976) shown in Tables 1 and 6 indicate that the threshold lethal concentration for hexavalent chromium does not occur within 96 hours. They found that for 16 tests, the average ratio of 11-day to 96-hour values was 0.37 for the fathead minnow and 0.27 for the goldfish.

The Freshwater Final Acute Value for hexavalent chromium, derived from the species mean acute values listed in Table 3 using the calculation procedures described in the Guidelines, is 21.2 µg/l.

Acute toxicity data for hexavalent chromium and twenty saltwater fish and invertebrate species have been reported (Table 1). Acute toxicity values ranged from 2,000 µg/l for a polychaete worm and mysid shrimp to

105,000 µg/l for the mud snail. The $LC_{50}$ values for fish species range from 12,400 µg/l for the Atlantic silverside to 91,000 µg/l for the mummichog. The most sensitive species were the polychaete annelids (2,000–8,000 µg/l), the mysid shrimp (2,000–4,400 µg/l), and two copepods (3,650 and 6,600 µg/l). The $LC_{50}$ values for hexavalent chromium and bivalve molluscs range from 57,000 µg/l for the soft shell clam to 14,000 µg/l for the brackish water clam. The sensitivity of the latter was salinity dependent with acute toxicity values of 35,000 µg/l and 14,000 µg/l at salinities of 22 g/kg and 5.5 g/kg, respectively. Adult starfish were insensitive with an $LC_{50}$ value of 32,000 µg/l. A Saltwater Final Acute Value of 1,260 µg/l was obtained for hexavalent chromium using the species mean acute values in Table 3 and the calculation procedures described in the Guidelines.

Trivalent Chromium

As shown in Table 1, the data base for acute toxicity of trivalent chromium to freshwater organisms includes 28 values for 19 animal species from 14 different families. Although the total number of values is smaller, more species have been tested with trivalent chromium than with hexavalent chromium.

Thirteen acute values for trivalent chromium have been reported for eight invertebrate species (Table 1). These values range from 2,000 µg/l for Daphnia magna and the mayfly to 64,000 µg/l for the caddisfly, all three of which were determined in soft water. Chapman, et al. (Manuscript) studied the effects of three levels of water hardness on the toxicity of trivalent chromium to Daphnia magna. They reported 48-hour acute values that ranged from 16,800 µg/l in soft water to 58,700 µg/l in hard water.

Table 1 also includes data for the acute toxicity of trivalent chromium to freshwater fish species. Fifteen 96-hour $LC_{50}$ values have

been reported for 11 fish species from eight families. These values ranged from 3,330 µg/l for the guppy in soft water to 71,900 µg/l for the bluegill in hard water. There are comparative data on the influence of water hardness on toxicity for the fathead minnow and the bluegill. The 96-hour $LC_{50}$ values for the fathead minnow tested in soft and hard water are 5,070 and 67,400 µg/l, respectively. The corresponding values for the bluegill are 7,460 and 71,900 µg/l.

The comparative data from Pickering and Henderson (1966) indicate that in soft water trivalent was more toxic than hexavalent chromium to four fish species. In hard water trivalent chromium was less toxic to the fathead minnow and more toxic to the bluegill than hexavalent chromium.

An exponential equation was used to describe the observed relationship of the acute toxicity of trivalent chromium to hardness in fresh water. A least square regression of the natural logarithms of the acute values on the natural logarithms of hardness produced slopes of 1.64, 0.83, and 0.78, respectively, for Daphnia magna, fathead minnow, and bluegill (Table 1). The first two slopes were significant, but the last could not be tested because only two values were available. The arithmetic mean slope (1.08) was used with the geometric mean toxicity value and hardness for each species to obtain a logarithmic intercept for each of the nineteen freshwater species for which acute values are available for trivalent chromium. The species mean acute intercept, calculated as the exponential of the logarithmic intercept, was used to compare the relative acute sensitivities (Table 3). Both the most sensitive and the least sensitive species are invertebrates. A freshwater final acute intercept of 32.3 µg/l was obtained for trivalent

chromium using the species mean acute intercepts listed in Table 3 and the calculation procedures described in the Guidelines. Thus, the Final Acute Equation is $e^{(1.08[\ln(\text{hardness})]+3.48)}$.

The few data that are available on the toxicity of trivalent chromium to saltwater species (Table 1) indicate that, probably because of precipitation, a large amount of trivalent chromium must be added to saltwater to kill aquatic organisms.

Chronic Toxicity

Hexavalent Chromium

The chronic data base for hexavalent chromium and freshwater species (Table 2) contains data for three fish species. Benoit (1976) studied the effects of hexavalent chromium in the chronic tests with brook trout and rainbow trout. The limits of 200 and 350 µg/l, with a chronic value of 265 µg/l, were established on the basis of survival for both species. Growth in weight during the first eight months was retarded at all test concentrations. However, this was a temporary effect on growth and was not used to establish the chronic limits.

Sauter, et al. (1976) also used the rainbow trout in a chronic study. The limits for this early life stage exposure were 51 and 105 µg/l with a chronic value of 73 µg/l. These values were established on the basis of a reduction of growth after 60 days post-hatch exposure. This chronic value of 73 µg/l was about one-fourth of the chronic value of 265 µg/l from the chronic test reported by Benoit (1976).

The acute-chronic ratios for brook trout and rainbow trout, calculated from the data of Benoit (1976) are 220 and 260, respectively (Table 2). Sauter, et al. (1976) provided no acute data in their study with which to calculate acute-chronic ratios.

The limits of 1,000 and 3,950 µg/l in a life-cycle test with the fathead minnow (Pickering, 1980) were based on survival. In this exposure also an early retardation of growth was only temporary. The chronic value of 1,990 µg/l is much higher than that for the trout but the acute-chronic ratio of 19 is much lower.

No chronic values are available for hexavalent chromium with any freshwater invertebrate species.

Results of life-cycle studies with the saltwater polychaete, Neanthes arenaceodentata, and the mysid shrimp Mysidopsis bahia are reported in Table 2. Other life cycle data on the polychaetes, Capitella capitata and Ophryotrocha diadema, (Table 6) were not included here because exposure concentrations were not adequately defined. Hexavalent chromium was chronically toxic to the polychaete at 25 µg/l and to the mysid at 132 µg/l and both of these species were among the most acutely saensitive to hexavalent chromium (Table 1). The acute-chronic ratios were 120 for the polychaete and 15 for the mysid. These ratios, while quite different, are consistent with those for freshwater fish species.

The geometric mean of the five acute-chronic ratios for three freshwater fish species and two saltwater invertebrate species is 72. The Freshwater Final Acute Value of 21.2 µg/l divided by the acute-chronic ratio of 72 results in a Freshwater Final Chronic Value for hexavalent chromium of 0.29 µg/l. Similarly, the Saltwater Final Chronic Value for hexavalent chromium is 17.5 µg/l.

Trivalent Chromium

The freshwater chronic data base for trivalent chromium (Table 2) contains data for a life-cycle test with Daphnia magna in soft water and a life-cycle test with the fathead minnow in hard water. In hard water the

chronic value of 1,020 µg/l for the fathead minnow is greater than the chronic value of 66 µg/l for Daphnia magna. Trivalent chromium appeared to be more toxic to Daphnia magna in hard water than in soft water. The chronic value in soft water was 66 µg/l (Table 2), but in hard water the lowest tested concentration (44 µg/l) inhibited reproduction (Table 6). Chapman, et al. (Manuscript) speculated that ingested precipitated chromium contributed to the toxicity in hard water. Biesinger and Christensen (1972) also conducted a life-cycle test with Daphnia magna but the test concentrations were not measured; the data are included in Table 6. The acute-chronic ratio is 27 for the fish and 250 for Daphnia magna.

No data on the chronic effects of trivalent chromium on saltwater species are available.

Plant Effects

Hexavalent Chromium

The data for four species of freshwater algae and Eurasian watermilfoil (Table 4) indicate that algae are sensitive to hexavalent chromium. The effect concentrations of chromium range from 10 µg/l for reduction in growth of a green alga to 1,900 µg/l for root weight inhibition of Eurasian watermilfoil. Growth of the green alga, Chlamydomonas reinhardi, was reduced at a concentration of 10 µg/l in BOLD's basal medium.

Toxicity of hexavalent chromium to the diatom, Navicula seminulum, was tested at three temperatures in both soft and hard waters (Academy of Natural Sciences of Philadelphia, 1960). The geometric mean of the concentrations causing a 50 percent reduction in growth was 245 µg/l in soft waters and 335 µg/l in hard water. The diatom was more sensitive to chromium at 22°C than at 30°C.

The data indicate that green algae are quite sensitive to hexavalent chromium. However, chromium concentrations were not measured in any of the exposures listed in Table 4, so a Freshwater Final Plant Value is not available for hexavalent chromium.

Toxicity studies were performed with the saltwater macroalga, Macrocystis pyrifera, to investigate the effect of hexavalent chromium on photosynthesis (Table 4). The 96-hour $EC_{50}$ reported by Clendenning and North (1959) was 5,000 µg/l, whereas 20 percent inhibition was noted after five days at 1,000 µg/l (Bernhard and Zattera, 1975). These data indicate that the plants were among the most sensitive species to chromium. Again, because no chromium concentrations were measured, no Saltwater Final Plant Value can be stated.

Trivalent Chromium

Toxicity data are available for only one freshwater plant species (Table 4). Root weight was inhibited at a trivalent chromium concentration of 9,900 µg/l (Stanley, 1974). Exposure concentrations were not measured, so a Freshwater Final Plant Value for trivalent chromium is not available.

No saltwater plant species have been tested with trivalent chromium.

Residues

Hexavalent Chromium

Data are available from two studies with the rainbow trout and hexavalent chromium, and the bioconcentration factor is about one (Table 5). Data on bioconcentration of hexavalent chromium and saltwater species is limited to one polychaete species and the oyster and blue mussel (Table 4). The bioconcentration factors are in the range of 125 to 200.

Trivalent Chromium

Data are not available concerning the bioconcentration of trivalent chromium by freshwater organisms.

Uptake of trivalent chromium by the blue mussel, soft shell clam, and oyster has been studied and the bioconcentration factors range from 86 to 153 (Table 5). These results are similar to those for hexavalent chromium.

Miscellaneous

Hexavalent Chromium

Table 6 includes data for other effects on freshwater species that were not included in the first five tables. The data base for hexavalent chromium is more extensive than that for trivalent chromium.

The data in this table indicate that Daphnia magna is a very sensitive species. Debalka (1975) reported 72-hour $EC_{50}$ values that ranged from 31 to 81 µg/l. In addition, Trabalka and Gehrs (1977) studied the chronic toxicity of hexavalent chromium to Daphnia magna. They found a significant effect on both life span and fecundity at all test concentrations including the lowest of 10 µg/l. Because a lower limit was not obtained, this datum is included in Table 6 instead of Table 2. This value certainly supports the Final Chronic Value.

Algae also appear to be sensitive to chromium. Zarafonetis and Hampton (1974) reported inhibition of photosynthesis of a natural population of river algae exposed to 20 µg/l.

Data in Table 6 also indicate that low concentrations of hexavalent chromium have a deleterious effect on the growth of fishes. Olson and Foster (1956) reported a statistically significant effect on growth of chinook salmon at 16 µg/l and on rainbow trout at 21 µg/l. At these concentra-

tions, growth in weight was reduced about ten percent. As noted earlier, Benoit (1976) and Pickering (1980) also reported effects on growth of fishes exposed to low concentrations. However, in these life-cycle tests the effect was temporary and was not used to establish chronic limits.

Chronic mortality of the saltwater polychaete, Neanthes arenaceodentata, resulted in 59-day $EC_{50}$ value for hexavalent chromium of 200 µg/l compared to the 96-hour $LC_{50}$ of 3,100 µg/l and the chronic value of 25 µg/l. Sublethal effects reported for this species show inhibition of tube building at 79 µg/l.

Holland, et al. (1960) reported toxicity to silver salmon at a concentration of 31,800 µg/l which is similar to the species mean acute values (Table 1) reported for the speckled sanddab (30,500) but twice as high as that reported for the Atlantic silverside (15,000).

The effect of salinity and temperature on hexavalent chromium toxicity to grass shrimp is reported by Fales (1978). At fixed salinities of 10 and 20 g/kg toxicity increased with increasing temperature between 10 to 25°C. At fixed temperatures toxicity decreased with increasing salinity from 10 to 20 g/kg.

Trivalent Chromium

Embryos of a freshwater snail are rather insensitive to trivalent chromium (Table 6).

Mearns, et al. (1976) were able to kill a saltwater polychaete worm with trivalent chromium by adding 50,400 µg/l, probably because the pH dropped to 4.5 due to the extensive precipitation. When the pH was raised to about 7.9 by adding sodium hydroxide, the worms not only survived for at least 160 days, but also reproduced (Table 6).

## Summary

### Hexavalent Chromium

Acute data for hexavalent chromium are available for thirteen freshwater animal species from ten different families which include a wide variety of animals that perform a spectrum of ecological functions. Data indicate that water hardness has an insignificant influence on toxicity. Most invertebrate species are more sensitive than most fish, and a scud is the most acutely sensitive species.

Long-term tests with brook trout and rainbow trout both gave chronic values of 265 µg/l which are much lower than the chronic value of 1,990 µg/l for the fathead minnow. No chronic values are available for freshwater invertebrate species.

The data for freshwater plants indicate that green algae are sensitive to hexavalent chromium and the bioconcentration factor for rainbow trout is about one.

Other data reveal more sensitive effects. The growth of chinook salmon was reduced at a measured concentration of 16 µg/l. In chronic tests with brook trout, rainbow trout, and fathead minnows a temporary adverse affect on growth occurred at low concentrations. In a life-cycle test with Daphnia magna the lowest test concentration of 10 µg/l reduced life span and fecundity.

The acute toxicity of hexavalent chromium to twenty saltwater vertebrate and invertebrate species ranges from 2,000 µg/l for polychaete annelids and a mysid shrimp, to 105,000 µg/l for the mud snail. Polychaetes and microcrustaceans are the most acutely sensitive taxa. The chronic values for polychaetes and a mysid shrimp are 25 and 132 µg/l, respectively, and the acute-chronic ratios are 120 and 15, respectively. Toxicity to macroalgae was reported at 1,000 and 5,000 µg/l.

Data for bioconcentration factors for hexavalent chromium range from 125 to 200 for bivalves and polychaetes.

Trivalent Chromium

Acute data for trivalent chromium are available for 19 freshwater animal species from 14 different families. The data indicate that water hardness has a significant influence on toxicity, with trivalent chromium being more toxic in soft water. In soft water the sensitivity of fish and invertebrate species is comparable.

One life-cycle test with Daphnia magna in soft water gave a chronic value of 66 µg/l, but another gave a chronic value of 445 µg/l. In a chronic test in hard water the lowest test concentration of 44 µg/l inhibited reproduction of Daphnia magna, but this effect may have been due to ingested precipitated chromium. In a life-cycle test with the fathead minnow in hard water the chronic value was 1,020 µg/l. Toxicity data are available for only one freshwater plant species. A concentraton of 9,900 µg/l inhibited growth of roots of Eurasian watermilfoil. No bioconcentration factors are available for trivalent chromium and freshwater organisms.

The available acute values for trivalent chromium in saltwater are both above 10,000 µg/l, probably because trivalent chromium has a low solubility in saltwater. Bioconcentration factors for saltwater organisms and trivalent chromium range from 86 to 153. This is similar to the bioconcentration factors for hexavalent chromium and saltwater species.

## CRITERIA

For total recoverable hexavalent chromium the criterion to protect freshwater aquatic life as derived using the Guidelines is 0.29 µg/l as a 24-hour average and the concentration should not exceed 21 µg/l at any time.

For freshwater aquatic life the concentration (in µg/l) of total recoverable trivalent chromium should not exceed the numerical value given by $e^{(1.08[\ln(\text{hardness})]+3.48)}$ at any time. For example, at hardnesses of 50, 100, and 200 mg/l as $CaCO_3$ the concentration of total recoverable trivalent chromium should not exceed 2,200, 4,700, and 9,900 µg/l, respectively, at any time. The available data indicate that chronic toxicity to freshwater aquatic life occurs at concentrations as low as 44 µg/l and would occur at lower concentrations among species that are more sensitive than those tested.

For total recoverable hexavalent chromium the criterion to protect saltwater aquatic life as derived using the Guidelines is 18 µg/l as a 24-hour average and the concentration should not exceed 1,260 µg/l at any time.

For total recoverable trivalent chromium, the available data indicate that acute toxicity to saltwater aquatic life occurs at concentrations as low as 10,300 µg/l, and would occur at lower concentrations among species that are more sensitive than those tested. No data are available concerning the chronic toxicity of trivalent chromium to sensitive saltwater aquatic life.

Table 1. Acute values for chromium

| Species | Method* | Chemical | Hardness (mg/l as $CaCO_3$) | LC50/EC50** (µg/l) | Species Mean Acute Value** (µg/l) | Reference |
|---|---|---|---|---|---|---|
| | | | Hexavalent Chromium | | | |
| | | | FRESHWATER SPECIES | | | |
| Rotifer, *Philodina acuticornis* | S, U | Potassium dichromate | 25 | 3,100 | - | Bulkema, et al. 1974 |
| Rotifer, *Philodina acuticornis* | S, U | Potassium dichromate | 81 | 15,000 | 6,800 | Bulkema, et al. 1974 |
| Rotifer, *Philodina roseola* | S, M | Sodium chromate | - | 12,000 | - | Schaffer & Pipes, 1973 |
| Rotifer, *Philodina roseola* | S, M | Sodium chromate | - | 8,900 | - | Schaffer & Pipes, 1973 |
| Rotifer, *Philodina roseola* | S, M | Sodium chromate | - | 7,400 | - | Schaffer & Pipes, 1973 |
| Rotifer, *Philodina roseola* | S, M | Sodium chromate | - | 5,500 | - | Schaffer & Pipes, 1973 |
| Rotifer, *Philodina roseola* | S, M | Sodium chromate | - | 4,400 | 7,200 | Schaffer & Pipes, 1973 |
| Snail, *Physa heterostropha* | S, U | Potassium chromate | 45 | 17,300 | - | Academy of Sciences, 1960 |
| Snail, *Physa heterostropha* | S, U | Potassium chromate | 45 | 17,300 | - | Academy of Sciences, 1960 |
| Snail, *Physa heterostropha* | S, U | Potassium chromate | 171 | 40,600 | - | Academy of Sciences, 1960 |
| Snail, *Physa heterostropha* | S, U | Potassium chromate | 171 | 31,600 | 25,000 | Academy of Sciences, 1960 |
| Cladoceran, *Daphnia magna* | S, U | Potassium dichromate | - | 6,400 | 6,400 | Dowden & Bennett, 1965 |
| Scud, *Gammarus pseudolimnaeus* | FT, M | Potassium chromate | 45 | 67 | 67 | U.S. EPA, 1980a |

Table 1. (Continued)

| Species | Method* | Chemical | Hardness (mg/l as $CaCO_3$) | LC50/EC50** (µg/l) | Species Mean Acute Value** (µg/l) | Reference |
|---|---|---|---|---|---|---|
| Midge, *Tanytarsus dissimilis* | FT, M | Potassium chromate | 44 | 59,900 | 59,900 | U.S. EPA, 1980a |
| Rainbow trout, *Salmo gairdneri* | FT, M | Sodium dichromate | 45 | 69,000 | 69,000 | Benoit, 1976 |
| Brook trout, *Salvelinus fontinalis* | FT, M | Sodium dichromate | 45 | 59,000 | 59,000 | Benoit, 1976 |
| Goldfish, *Carassius auratus* | FT, M | Potassium dichromate | 220 | 123,000 | - | Adelman & Smith, 1976 |
| Goldfish, *Carassius auratus* | FT, M | Potassium dichromate | 220 | 123,000 | - | Adelman & Smith, 1976 |
| Goldfish, *Carassius auratus* | FT, M | Potassium dichromate | 220 | 90,000 | - | Adelman & Smith, 1976 |
| Goldfish, *Carassius auratus* | FT, M | Potassium dichromate | 220 | 125,000 | - | Adelman & Smith, 1976 |
| Goldfish, *Carassius auratus* | FT, M | Potassium dichromate | 220 | 109,000 | - | Adelman & Smith, 1976 |
| Goldfish, *Carassius auratus* | FT, M | Potassium dichromate | 220 | 135,000 | - | Adelman & Smith, 1976 |
| Goldfish, *Carassius auratus* | FT, M | Potassium dichromate | 220 | 110,000 | - | Adelman & Smith, 1976 |
| Goldfish, *Carassius auratus* | FT, M | Potassium dichromate | 220 | 129,000 | - | Adelman & Smith, 1976 |
| Goldfish, *Carassius auratus* | FT, M | Potassium dichromate | 220 | 98,000 | - | Adelman & Smith, 1976 |
| Goldfish, *Carassius auratus* | FT, M | Potassium dichromate | 220 | 133,000 | - | Adelman & Smith, 1976 |
| Goldfish, *Carassius auratus* | FT, M | Potassium dichromate | 220 | 102,000 | - | Adelman & Smith, 1976 |

Table 1. (Continued)

| Species | Method* | Chemical | Hardness (mg/l as $CaCO_3$) | LC50/EC50** (µg/l) | Species Mean Acute Value** (µg/l) | Reference |
|---|---|---|---|---|---|---|
| Goldfish, Carassius auratus | FT, M | Potassium dichromate | 220 | 133,000 | - | Adelman & Smith, 1976 |
| Goldfish, Carassius auratus | FT, M | Potassium dichromate | 220 | 126,000 | - | Adelman & Smith, 1976 |
| Goldfish, Carassius auratus | FT, M | Potassium dichromate | 220 | 126,000 | - | Adelman & Smith, 1976 |
| Goldfish, Carassius auratus | FT, M | Potassium dichromate | 220 | 133,000 | - | Adelman & Smith, 1976 |
| Goldfish, Carassius auratus | FT, M | Potassium dichromate | 220 | 126,000 | - | Adelman & Smith, 1976 |
| Goldfish, Carassius auratus | FT, M | Potassium dichromate | 220 | 124,000 | - | Adelman & Smith, 1976 |
| Goldfish, Carassius auratus | S, U | Sodium dichromate | 100 | 249,000 | - | Dowden & Bennett, 1965 |
| Goldfish, Carassius auratus | S, U | Potassium dichromate | 20 | 37,500 | 120,000 | Pickering & Henderson, 1966 |
| Fathead minnow, Pimephales promelas | FT, M | Potassium dichromate | 220 | 56,000 | - | Adelman & Smith, 1976 |
| Fathead minnow, Pimephales promelas | FT, M | Potassium dichromate | 220 | 51,000 | - | Adelman & Smith, 1976 |
| Fathead minnow, Pimephales promelas | FT, M | Potassium dichromate | 220 | 53,000 | - | Adelman & Smith, 1976 |
| Fathead minnow, Pimephales promelas | FT, M | Potassium dichromate | 220 | 49,000 | - | Adelman & Smith, 1976 |
| Fathead minnow, Pimephales promelas | FT, M | Potassium dichromate | 220 | 48,000 | - | Adelman & Smith, 1976 |
| Fathead minnow, Pimephales promelas | FT, M | Potassium dichromate | 220 | 60,000 | - | Adelman & Smith, 1976 |

Table 1. (Continued)

| Species | Method* | Chemical | Hardness (mg/l as $CaCO_3$) | LC50/EC50** (µg/l) | Species Mean Acute Value** (µg/l) | Reference |
|---|---|---|---|---|---|---|
| Fathead minnow, Pimephales promelas | FT, M | Potassium dichromate | 220 | 50,000 | - | Adelman & Smith, 1976 |
| Fathead minnow, Pimephales promelas | FT, M | Potassium dichromate | 220 | 53,000 | - | Adelman & Smith, 1976 |
| Fathead minnow, Pimephales promelas | FT, M | Potassium dichromate | 220 | 49,000 | - | Adelman & Smith, 1976 |
| Fathead minnow, Pimephales promelas | FT, M | Potassium dichromate | 220 | 37,000 | - | Adelman & Smith, 1976 |
| Fathead minnow, Pimephales promelas | FT, M | Potassium dichromate | 220 | 66,000 | - | Adelman & Smith, 1976 |
| Fathead minnow, Pimephales promelas | FT, M | Potassium dichromate | 220 | 55,000 | - | Adelman & Smith, 1976 |
| Fathead minnow, Pimephales promelas | FT, M | Potassium dichromate | 220 | 38,000 | - | Adelman & Smith, 1976 |
| Fathead minnow, Pimephales promelas | FT, M | Potassium dichromate | 220 | 34,000 | - | Adelman & Smith, 1976 |
| Fathead minnow, Pimephales promelas | FT, M | Potassium dichromate | 220 | 29,000 | - | Adelman & Smith, 1976 |
| Fathead minnow, Pimephales promelas | FT, M | Potassium dichromate | 220 | 34,000 | - | Adelman & Smith, 1976 |
| Fathead minnow, Pimephales promelas | FT, M | Potassium dichromate | 220 | 26,000 | - | Adelman & Smith, 1976 |
| Fathead minnow, Pimephales promelas | FT, M | Sodium dichromate | 235 | 33,200 | - | Broderius & Smith, 1979 |
| Fathead minnow, Pimephales promelas | S, U | Potassium dichromate | 209 | 39,700 | - | Pickering, 1980 |
| Fathead minnow, Pimephales promelas | S, U | Potassium dichromate | 208 | 32,700 | - | Pickering, 1980 |

Table 1. (Continued)

| Species | Method* | Chemical | Hardness (mg/l as $CaCO_3$) | LC50/EC50** (µg/l) | Species Mean Acute Value** (µg/l) | Reference |
|---|---|---|---|---|---|---|
| Fathead minnow, Pimephales promelas | FT, M | Potassium dichromate | 209 | 37,700 | - | Pickering, 1980 |
| Fathead minnow, Pimephales promelas | FT, M | Potassium dichromate | 209 | 37,000 | - | Pickering, 1980 |
| Fathead minnow, Pimephales promelas | FT, M | Potassium dichromate | 209 | 35,900 | - | Pickering, 1980 |
| Fathead minnow, Pimephales promelas | S, U | Potassium dichromate | 20 | 17,600 | - | Pickering & Henderson, 1966 |
| Fathead minnow, Pimephales promelas | S, U | Potassium dichromate | 360 | 27,300 | - | Pickering & Henderson, 1966 |
| Fathead minnow, Pimephales promelas | S, U | Potassium chromate | 20 | 45,600 | - | Pickering & Henderson, 1966 |
| Fathead minnow, Pimephales promelas | FT, M | - | - | 52,000 | - | Ruesink & Smith, 1975 |
| Fathead minnow, Pimephales promelas | FT, M | - | - | 37,000 | 43,100 | Ruesink & Smith, 1975 |
| Guppy, Poecilia reticulata | S, U | Potassium dichromate | 20 | 30,000 | 30,000 | Pickering & Henderson, 1966 |
| Striped bass, Morone saxatilis | S, U | Potassium dichromate | 35 | 35,000 | - | Hughes, 1971 |
| Striped bass, Morone saxatilis | S, U | Potassium dichromate | 35 | 26,500 | 30,400 | Hughes, 1971 |
| Bluegill, Lepomis macrochirus | S, U | Potassium dichromate | 20 | 118,000 | - | Pickering & Henderson, 1966 |
| Bluegill, Lepomis macrochirus | S, U | Potassium dichromate | 360 | 133,000 | - | Pickering & Henderson, 1966 |

Table 1. (Continued)

| Species | Method* | Chemical | Hardness (mg/l as $CaCO_3$) | LC50/EC50** (µg/l) | Species Mean Acute Value** (µg/l) | Reference |
|---|---|---|---|---|---|---|
| Bluegill, *Lepomis macrochirus* | S, U | Potassium dichromate | 45 | 110,000 | - | Trama & Benoit, 1960 |
| Bluegill, *Lepomis macrochirus* | S, U | Potassium chromate | 45 | 170,000 | - | Trama & Benoit, 1960 |
| Bluegill, *Lepomis macrochirus* | S, U | Sodium dichromate | 120 | 213,000 | - | Turnbull, et al. 1954 |
| Bluegill, *Lepomis macrochirus* | S, U | Potassium dichromate | 44 | 113,000 | - | Academy of Sciences, 1960 |
| Bluegill, *Lepomis macrochirus* | S, U | Potassium dichromate | 44 | 113,000 | - | Academy of Sciences, 1960 |
| Bluegill, *Lepomis macrochirus* | S, U | Potassium dichromate | 171 | 135,000 | - | Academy of Sciences, 1960 |
| Bluegill, *Lepomis macrochirus* | S, U | Potassium dichromate | 171 | 130,000 | 134,000 | Academy of Sciences, 1960 |
| | | SALTWATER SPECIES | | | | |
| Polychaete worm (larva), *Capitella capitata* | S, U | Chromium trioxide | - | 8,000 | - | Reish, et al. 1976 |
| Polychaete worm (adult), *Capitella capitata* | S, U | Chromium trioxide | - | 5,000 | 6,300 | Reish, et al. 1976 |
| Polychaete worm, *Ctenodrilus serratus* | S, U | Chromium trioxide | - | 4,300 | 4,300 | Reish & Carr, 1978 |
| Polychaete worm, *Neanthes arenaceodentata* | S, M | Potassium dichromate | - | 3,100 | 3,100 | Mearns, et al. 1976 |
| Polychaete worm, *Nereis virens* | S, U | Potassium chromate | - | 2,000 | 2,000 | Eisler & Hennekey, 1977 |

Table 1. (Continued)

| Species | Method* | Chemical | Hardness (mg/l as $CaCO_3$) | LC50/EC50** (µg/l) | Species Mean Acute Value** (µg/l) | Reference |
|---|---|---|---|---|---|---|
| Polychaete worm, *Ophryotrocha diadema* | S, U | Chromium trioxide | - | 7,500 | 7,500 | Reish & Carr, 1978 |
| Soft shell clam, *Mya arenaria* | S, U | Potassium chromate | - | 57,000 | 57,000 | Eisler & Hennekey, 1977 |
| Brackish water clam, *Rangia cuneata* | S, U | Potassium dichromate | - | 14,000 | - | Olson & Harrel, 1973 |
| Brackish water clam, *Rangia cuneata* | S, U | Potassium dichromate | - | 35,000 | 22,000 | Olson & Harrel, 1973 |
| Mud snail, *Nassarius obsoletus* | S, U | Potassium chromate | - | 105,000 | 105,000 | Eisler & Hennekey, 1977 |
| Copepod, *Acartia clausi* | S, U | Potassium dichromate | - | 6,600 | 6,600 | U.S. EPA, 1980b |
| Copepod, *Pseudodiaptomus coronatus* | S, U | Potassium dichromate | - | 3,650 | 3,650 | U.S. EPA, 1980b |
| Copepod, *Tigropus japonicus* | S, U | Potassium dichromate | - | 17,200 | 17,200 | U.S. EPA, 1980b |
| Mysid shrimp, *Mysidopsis bahia* | S, M | Potassium dichromate | - | 2,000 | 2,000 | U.S. EPA, 1980b |
| Mysid shrimp, *Mysidopsis bigelowi* | S, M | Potassium dichromate | - | 4,400 | 4,400 | U.S. EPA, 1980b |
| Blue crab, *Callinectes sapidus* | S, U | Potassium dichromate | - | 89,000 | - | Frank & Robertson, 1979 |
| Blue crab, *Callinectes sapidus* | S, U | Potassium dichromate | - | 98,000 | 93,000 | Frank & Robertson, 1979 |
| Hermit crab, *Pagurus longicarpus* | S, U | Potassium chromate | - | 10,000 | 10,000 | Eisler & Hennekey, 1977 |

Table 1. (Continued)

| Species | Method* | Chemical | Hardness (mg/l as $CaCO_3$) | LC50/EC50** (µg/l) | Species Mean Acute Value** (µg/l) | Reference |
|---|---|---|---|---|---|---|
| Starfish, *Asterias forbesi* | S, U | Potassium chromate | - | 32,000 | 32,000 | Eisler & Hennekey, 1977 |
| Mummichog, *Fundulus heteroclitus* | S, U | Potassium chromate | - | 91,000 | 91,000 | Eisler & Hennekey, 1977 |
| Atlantic silverside (larva), *Menidia menidia* | S, U | Potassium dichromate | - | 12,400 | - | U.S. EPA, 1980b |
| Atlantic silverside (larva), *Menidia menidia* | S, U | Potassium dichromate | - | 14,300 | - | U.S. EPA, 1980b |
| Atlantic silverside (juvenile), *Menidia menidia* | S, U | Potassium dichromate | - | 20,100 | 15,000 | U.S. EPA, 1980b |
| Speckled sanddab, *Citharichthys stigmaeus* | S, U | Potassium dichromate | - | 31,000 | - | Sherwood, 1975 |
| Speckled sanddab, *Citharichthys stigmaeus* | S, U | Potassium dichromate | - | 30,000 | 30,500 | Mearns, et al. 1976 |
| | | | Trivalent Chromium | | | |
| | | | FRESHWATER SPECIES | | | |
| Annelid, *Nais* sp. | S, M | - | 50 | 9,300 | - | Rehwoldt, et al. 1973 |
| Snail, *Amnicola* sp. | S, M | - | 50 | 8,400 | - | Rehwoldt, et al. 1973 |
| Cladoceran, *Daphnia magna* | S, U | Chromic nitrate | 48 | 2,000 | - | Biesinger & Christensen, 1972 |
| Cladoceran, *Daphnia magna* | S, M | Chromic nitrate | 52 | 16,800 | - | Chapman, et al. Manuscript |

Table 1. (Continued)

| Species | Method* | Chemical | Hardness (mg/l as $CaCO_3$) | LC50/EC50** (µg/l) | Species Mean Acute Value** (µg/l) | Reference |
|---|---|---|---|---|---|---|
| Cladoceran, *Daphnia magna* | S, M | Chromic nitrate | 99 | 27,400 | - | Chapman, et al. Manuscript |
| Cladoceran, *Daphnia magna* | S, M | Chromic nitrate | 110 | 26,300 | - | Chapman, et al. Manuscript |
| Cladoceran, *Daphnia magna* | S, M | Chromic nitrate | 195 | 51,400 | - | Chapman, et al. Manuscript |
| Cladoceran, *Daphnia magna* | S, M | Chromic nitrate | 215 | 58,700 | - | Chapman, et al. Manuscript |
| Scud, *Gammarus* sp. | S, M | - | 50 | 3,200 | - | Rehwoldt, et al. 1973 |
| Mayfly, *Ephemerella subvaria* | S, U | Chromic chloride | 44 | 2,000 | - | Warnick & Bell, 1969 |
| Damselfly, Unidentified | S, M | - | 50 | 43,100 | - | Rehwoldt, et al. 1973 |
| Caddisfly, *Hydropsyche betteni* | S, U | Chromic chloride | 44 | 64,000 | - | Warnick & Bell, 1969 |
| Caddisfly, Unidentified | S, M | - | 50 | 50,000 | - | Rehwoldt, et al. 1973 |
| American eel, *Anguilla rostrata* | S, M | - | 55 | 16,900 | - | Rehwoldt, et al. 1972 |
| Rainbow trout, *Salmo gairdneri* | S, U | - | - | 11,200 | - | Bills, et al. 1977 |
| Rainbow trout, *Salmo gairdneri* | FT, M | Chromic nitrate | - | 24,100 | - | Hale, 1977 |
| Goldfish, *Carassius auratus* | S, U | Chromium potassium sulfate | 20 | 4,100 | - | Pickering & Henderson, 1966 |
| Carp, *Cyprinus carpio* | S, M | - | 55 | 14,300 | - | Rehwoldt, et al. 1972 |

Table 1. (Continued)

| Species | Method* | Chemical | Hardness (mg/l as $CaCO_3$) | LC50/EC50** (µg/l) | Species Mean Acute Value** (µg/l) | Reference |
|---|---|---|---|---|---|---|
| Fathead minnow, *Pimephales promelas* | FT, M | Chromium potassium sulfate | 203 | 29,000 | - | Pickering, Manuscript |
| Fathead minnow, *Pimephales promelas* | FT, M | Chromium potassium sulfate | 203 | 27,000 | - | Pickering, Manuscript |
| Fathead minnow, *Pimephales promelas* | S, U | Chromium potassium sulfate | 20 | 5,070 | - | Pickering & Henderson, 1966 |
| Fathead minnow, *Pimephales promelas* | S, U | Chromium potassium sulfate | 360 | 67,400 | - | Pickering & Henderson, 1966 |
| Banded killifish, *Fundulus diaphanus* | S, M | - | 55 | 16,900 | - | Rehwoldt, et al. 1972 |
| Guppy, *Poecilia reticulata* | S, U | Chromium potassium sulfate | 20 | 3,330 | - | Pickering & Henderson, 1966 |
| White perch, *Morone americana* | S, M | - | 55 | 14,400 | - | Rehwoldt, et al. 1972 |
| Striped bass, *Morone saxatilis* | S, M | - | 55 | 17,700 | - | Rehwoldt, et al. 1972 |
| Pumpkinseed, *Lepomis gibbosus* | S, M | - | 55 | 17,000 | - | Rehwoldt, et al. 1972 |
| Bluegill, *Lepomis macrochirus* | S, U | Chromium potassium sulfate | 20 | 7,460 | - | Pickering & Henderson, 1966 |
| Bluegill, *Lepomis macrochirus* | S, U | Chromium potassium sulfate | 360 | 71,900 | - | Pickering & Henderson, 1966 |
| | | SALTWATER SPECIES | | | | |
| American oyster, *Crassostrea virginica* | S, U | Chromium chloride | - | 10,300 | 10,300 | Calabrese, 1973 |
| Crab (zoea), *Sesarma haematocheir* | S, U | Chromium chloride | - | 56,000 | 56,000 | Okubo & Okubo, 1962 |

**Table 1. (Continued)**

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* S = static, FT = flow-through, U = unmeasured, M = measured

** Results are expressed as chromium, not as the compound.

Trivalent chromium - freshwater

Acute toxicity vs. hardness

Daphnia magna: slope = 1.64, intercept = 2.36, r = 0.84, p = 0.05, N = 6

Fathead minnow: slope = 0.83, intercept = 5.98, r = 0.98, p = 0.05, N = 4

Bluegill: slope = 0.78, intercept = 6.57, r = 1.0, N = 2

Arithmetic mean acute slope = 1.08

Table 2. Chronic values for chromium

| Species | Test* | Chemical | Hardness (mg/l as $CaCO_3$) | Limits (µg/l)** | Chronic Value (µg/l)** | Reference |
|---|---|---|---|---|---|---|
| | | Hexavalent Chromium | | | | |
| | | FRESHWATER SPECIES | | | | |
| Rainbow trout, *Salmo gairdneri* | ELS | Chromium trioxide | 34 | 51-105 | 73 | Sauter, et al. 1976 |
| Rainbow trout, *Salmo gairdneri* | ELS | Sodium dichromate | 45 | 200-350 | 265 | Benoit, 1976 |
| Brook trout, *Salvelinus fontinalis* | LC | Sodium dichromate | 45 | 200-350 | 265 | Benoit, 1976 |
| Fathead minnow, *Pimephales promelas* | LC | Potassium dichromate | 209 | 1,000-3,950 | 1,990 | Pickering, 1980 |
| | | SALTWATER SPECIES | | | | |
| Polychaete worm, *Neanthes arenaceodentata* | LC | Potassium dichromate | - | 17-38 | 25 | Oshida, 1978 |
| Mysid shrimp, *Mysidopsis bahia* | LC | Potassium dichromate | - | 88-198 | 132 | U.S. EPA, 1980b |
| | | Trivalent Chromium | | | | |
| | | FRESHWATER SPECIES | | | | |
| Cladoceran, *Daphnia magna* | LC | Chromic nitrate | 52 | 47-93 | 66 | Chapman, Manuscript |
| Fathead minnow, *Pimephales promelas* | LC | Chromium potassium sulfate | 203 | 750-1,400 | 1,020 | Pickering, Manuscript |

* LC = life cycle or partial life cycle, ELS = early life stage

**Results are expressed as chromium, not as the compound

**Table 2. (Continued)**

| Species | Acute-Chronic Ratio: Acute Value (µg/l) | Acute-Chronic Ratio: Chronic Value (µg/l) | Acute-Chronic Ratio: Ratio |
|---|---|---|---|
| | Hexavalent Chromium | | |
| Rainbow trout, *Salmo gairdneri* | 69,000 | 265 | 260 |
| Brook trout, *Salvelinus fontinalis* | 59,000 | 265 | 220 |
| Fathead minnow, *Pimephales promelas* | 37,000 | 1,990 | 19 |
| Polychaete worm, *Neanthes arenaceodentata* | 3,100 | 25 | 120 |
| Mysid shrimp, *Mysidopsis bahia* | 2,033 | 132 | 15 |
| | Trivalent Chromium | | |
| Cladoceran, *Daphnia magna* | 16,800 | 66 | 250 |
| Fathead minnow, *Pimephales promelas* | 28,000 | 1,020 | 27 |

Table 3. Species mean acute values and intercepts and acute-chronic ratios for chromium

| Rank* | Species | Species Mean Acute Value (µg/l) | Species Mean Acute-Chronic Ratio |
|---|---|---|---|
| | Hexavalent Chromium | | |
| | FRESHWATER SPECIES | | |
| 14 | Largemouth bass, *Micropterus salmoides* | 195,000 | - |
| 13 | Bluegill, *Lepomis macrochirus* | 134,000 | - |
| 12 | Goldfish, *Carassius auratus* | 120,000 | - |
| 11 | Rainbow trout, *Salmo gairdneri* | 69,000 | 260 |
| 10 | Midge, *Tanytarsus dissimilis* | 59,900 | - |
| 9 | Brook trout, *Salvelinus fontinalis* | 59,000 | 220 |
| 8 | Fathead minnow, *Pimephales promelas* | 43,100 | 19 |
| 7 | Striped bass, *Morone saxatilis* | 30,400 | - |
| 6 | Guppy, *Poecilia reticulata* | 30,000 | - |
| 5 | Snail, *Physa heterostropha* | 25,000 | - |
| 4 | Rotifer, *Philodina roseola* | 6,800 | - |
| 3 | Cladoceran, *Daphnia magna* | 6,400 | - |

Table 3. (Continued)

| Rank* | Species | Species Mean Acute Value (µg/l) | Species Mean Acute-Chronic Ratio |
|---|---|---|---|
| 2 | Rotifer, *Philodina acuticornis* | 3,100 | - |
| 1 | Scud, *Gammarus pseudolimnaeus* | 67 | - |
| | SALTWATER SPECIES | | |
| 19 | Mud snail, *Nassarius obsoletus* | 105,000 | - |
| 18 | Blue crab, *Callinectes sapidus* | 93,000 | - |
| 17 | Mummichog, *Fundulus heteroclitus* | 91,000 | - |
| 16 | Soft shell clam, *Mya arenaria* | 57,000 | - |
| 15 | Starfish, *Asterias forbesi* | 32,000 | - |
| 14 | Speckled sanddab, *Citharichthys stigmaeus* | 30,500 | - |
| 13 | Brackish water clam, *Rangia cuneata* | 22,000 | - |
| 12 | Copepod, *Tigriopus japonicus* | 17,200 | - |
| 11 | Atlantic silverside, *Menidia menidia* | 15,000 | - |
| 10 | Hermit crab, *Pagurus longicarpus* | 10,000 | - |
| 9 | Polychaete worm, *Ophryotrocha diadema* | 7,500 | - |
| 8 | Copepod, *Acartia clausi* | 6,600 | - |

Table 3. (Continued)

| Rank* | Species | Species Mean Acute Value (µg/l) | Species Mean Acute-Chronic Ratio |
|---|---|---|---|
| 7 | Polychaete worm, *Capitella capitata* | 6,300 | - |
| 6 | Mysid shrimp, *Mysidopsis bigelowi* | 4,400 | - |
| 5 | Polychaete worm, *Ctenodrilus serratus* | 4,300 | - |
| 4 | Copepod, *Pseudodiaptomus coranatus* | 3,650 | - |
| 3 | Polychaete worm, *Neanthes arenaceodentata* | 3,100 | 120 |
| 2 | Mysid shrimp, *Mysidopsis bahia* | 2,000 | 15 |
| 1 | Polychaete worm, *Nereis virens* | 2,000 | - |

| Rank* | Species | Species Mean Acute Intercept (µg/l) | Species Mean Acute-Chronic Ratio |
|---|---|---|---|
| | Trivalent Chromium | | |
| | FRESHWATER SPECIES | | |
| 18 | Caddisfly, *Hydropsyche betteni* | 1,075 | - |
| 17 | Caddisfly, Unidentified | 728 | - |
| 16 | Damselfly, Unidentified | 633 | - |
| 15 | Striped bass, *Morone saxatilis* | 233 | - |

Table 3. (Continued)

| Rank* | Species | Species Mean Acute Intercept (µg/l) | Species Mean Acute-Chronic Ratio |
|---|---|---|---|
| 14 | Pumpkinseed, *Lepomis gibbosus* | 224 | - |
| 13 | American eel, *Anguilla rostrata* | 224 | - |
| 12 | Banded killifish, *Fundulus diaphanus* | 224 | - |
| 11 | Bluegill, *Lepomis macrochirus* | 191 | - |
| 10 | White perch, *Morone americana* | 191 | - |
| 9 | Carp, *Cyprinus carpio* | 189 | - |
| 8 | Goldfish, *Carrasius auratus* | 161 | - |
| 7 | Cladoceran, *Daphnia magna* | 138 | - |
| 6 | Annelid, *Nais* sp. | 136 | - |
| 5 | Guppy, *Poecilia reticulata* | 132 | - |
| 4 | Snail, *Amnicola* sp. | 123 | - |
| 3 | Fathead minnow, *Pimephales promelas* | 118 | - |
| 2 | Scud, *Gammarus* sp. | 47 | - |

Table 3. (Continued)

| Rank* | Species | Species Mean Acute Intercept (µg/l) | Species Mean Acute-Chronic Ratio |
|---|---|---|---|
| 1 | Mayfly, *Ephemerela subvaria* | 33.4 | - |

* Ranked from least sensitive to most sensitive based on species mean acute value or intercept.

Hexavalent Chromium

Freshwater Final Acute Value = 21.2 µg/l

Saltwater Final Acute Value = 1,260 µg/l

Final Acute-Chronic Ratio = 72 (see text)

Freshwater Final Chronic Value = (21.2 µg/l)/72 = 0.29 µg/l

Saltwater Final Chronic Value = (1,260 µg/l)/72 = 17.5 µg/l

Trivalent Chromium - Freshwater

Final Acute Intercept = 32.3 µg/l

Natural logarithm of 32.3 = 3.48

Acute slope = 1.08 (see Table 1)

Final Acute Equation = $e^{(1.08[\ln(\text{hardness})]+3.48)}$

Table 4. Plant values for chromium

| Species | Chemical | Hardness (mg/l as $CaCO_3$) | Effect | Result* (µg/l) | Reference |
|---|---|---|---|---|---|
| | | Hexavalent Chromium | | | |
| | | FRESHWATER SPECIES | | | |
| Green alga, Chlamydomonas reinhardi | Potassium dichromate | - | Reduction in growth | 10 | Zarafonetis & Hampton, 1974 |
| Green alga, Selenastrum capricornutum | Sodium chromate | - | Inhibition in growth | 45 | Garton, 1972 |
| Green alga, Scenedesmus sp. | Potassium dichromate | - | Inhibition in growth | 500 | Staub, et al. 1973 |
| Diatom, Navicula seminulum | Potassium dichromate | 45 | 50% growth reduction | 187 | Academy of Sciences, 1960 |
| Diatom, Navicula seminulum | Potassium dichromate | 45 | 50% growth reduction | 230 | Academy of Sciences, 1960 |
| Diatom, Navicula seminulum | Potassium dichromate | 45 | 50% growth reduction | 251 | Academy of Sciences, 1960 |
| Diatom, Navicula seminulum | Potassium dichromate | 45 | 50% growth reduction | 272 | Academy of Sciences, 1960 |
| Diatom, Navicula seminulum | Potassium dichromate | 45 | 50% growth reduction | 308 | Academy of Sciences, 1960 |
| Diatom, Navicula seminulum | Potassium dichromate | 45 | 50% growth reduction | 237 | Academy of Sciences, 1960 |
| Diatom, Navicula seminulum | Potassium dichromate | 171 | 50% growth reduction | 254 | Academy of Sciences, 1960 |
| Diatom, Navicula seminulum | Potassium dichromate | 171 | 50% growth reduction | 254 | Academy of Sciences, 1960 |
| Diatom, Navicula seminulum | Potassium dichromate | 171 | 50% growth reduction | 343 | Academy of Sciences, 1960 |
| Diatom, Navicula seminulum | Potassium dichromate | 171 | 50% growth reduction | 343 | Academy of Sciences, 1960 |

Table 4. (Continued)

| Species | Chemical | Hardness (mg/l as $CaCO_3$) | Effect | Result* (µg/l) | Reference |
|---|---|---|---|---|---|
| Diatom, Navicula seminulum | Potassium dichromate | 171 | 50% growth reduction | 424 | Academy of Sciences, 1960 |
| Diatom, Navicula seminulum | Potassium dichromate | 171 | 50% growth reduction | 442 | Academy of Sciences, 1960 |
| Eurasian watermilfoil, Myriophyllum spicatum | Dichromate** | - | 50% root weight inhibition | 1,900 | Stanley, 1974 |
| | | SALTWATER SPECIES | | | |
| Alga, Macrocystis pyrifera | - | - | 50% inhibition of photosynthesis in 4 days | 5,000 | Clendenning & North, 1959 |
| Alga, Macrocystis pyrifera | Potassium dichromate | - | 10 - 20% inhibition of photosynthesis in 5 days | 1,000 | Bernhard & Zattera, 1975 |
| | | Trivalent Chromium | | | |
| | | FRESHWATER SPECIES | | | |
| Eurasian watermilfoil, Myriophyllum spicatum | - | - | 50% root weight inhibition | 9,900 | Stanley, 1974 |

* Results are expressed as chromium, not as the compound.

**Salt not given.

Table 5. Residues for chromium

| Species | Tissue | Chemical | Bioconcentration Factor | Duration (days) | Reference |
|---|---|---|---|---|---|
| | | Hexavalent Chromium | | | |
| | | FRESHWATER SPECIES | | | |
| Rainbow trout, *Salmo gairdneri* | Muscle | Sodium dichromate | <1 | 22 | Buhler, et al. 1977 |
| Rainbow trout, *Salmo gairdneri* | Whole body | Potassium chromate | 1 | 30 | Fromm & Stokes, 1962 |
| | | SALTWATER SPECIES | | | |
| Polychaete worm, *Neanthes arenaceodentata* | - | - | 200* | 150 | Mearns & Young, 1977 |
| Oyster, *Crassostrea virginica* | Soft parts | Sodium dichromate | 125* | 84 | U.S. EPA, 1980b |
| Blue mussel, *Mytilus edulis* | Soft parts | Sodium dichromate | 192* | 84 | U.S. EPA, 1980b |
| | | Trivalent Chromium | | | |
| | | SALTWATER SPECIES | | | |
| American oyster, *Crassostrea virginica* | Soft parts | Chromic nitrate | 116 | 140 | Shuster & Pringle, 1969 |
| Soft shell clam, *Mya arenaria* | Soft parts | Chromic chloride | 153* | 168 | Capuzzo & Sasner, 1977 |
| Blue mussel, *Mytilus edulis* | Soft parts | Chromic chloride | 86* | 168 | Capuzzo & Sasner, 1977 |

* Dry weight to wet weight conversion.

Table 6. Other data for chromium

| Species | Chemical | Hardness (mg/l as $CaCO_3$) | Duration | Effect | Result* (µg/l) | Reference |
|---|---|---|---|---|---|---|
| | | | Hexavalent Chromium | | | |
| | | | FRESHWATER SPECIES | | | |
| Algal community | Potassium dichromate | - | 1 mo | Diatoms reduced blue green algae dominant | 400 | Patrick, et al. 1975 |
| Algal community | Potassium dichromate | - | 1 mo | Diversity of diatoms reduced | 100 | Patrick, et al. 1975 |
| Algal community | Potassium dichromate | - | 1 mo | Bioconcentration of chromium: 8,500 | 400 | Patrick, et al. 1975 |
| Algal community | Potassium dichromate | - | 25 hrs | 32% inhibition of photo-synthesis | 20 | Zarafonetis & Hampton, 1974 |
| Protozoa, *Blepharisma* sp. | Potassium dichromate | - | 3 hrs | Some living | 32,000 | Ruthven & Cairns, 1973 |
| Snail, *Goniobasis livescens* | Potassium dichromate | 154 | 48 hrs | LC50 | 2,400 | Cairns, et al. 1976 |
| Snail, *Lymnaea emarginata* | Potassium dichromate | 154 | 48 hrs | LC50 | 34,800 | Cairns, et al. 1976 |
| Snail, *Physa integra* | Potassium dichromate | 154 | 48 hrs | LC50 | 660 | Cairns, et al. 1976 |
| Cladoceran, *Daphnia magna* | Potassium dichromate | 163 | 72 hrs | LC50 | 64** | Debelak, 1975 |
| Cladoceran, *Daphnia magna* | Potassium dichromate | 163 | 72 hrs | LC50 | 72** | Debelak, 1975 |
| Cladoceran, *Daphnia magna* | Potassium dichromate | 163 | 72 hrs | LC50 | 73** | Debelak, 1975 |
| Cladoceran, *Daphnia magna* | Potassium dichromate | 163 | 72 hrs | LC50 | 74** | Debelak, 1975 |

Table 6. (Continued)

| Species | Chemical | Hardness (mg/l as $CaCO_3$) | Duration | Effect | Result* (µg/l) | Reference |
|---|---|---|---|---|---|---|
| Cladoceran, Daphnia magna | Potassium dichromate | 163 | 72 hrs | LC50 | 81** | Debelak, 1975 |
| Cladoceran, Daphnia magna | Potassium dichromate | 86 | 72 hrs | LC50 | 31** | Debelak, 1975 |
| Cladoceran, Daphnia magna | Potassium dichromate | 86 | 72 hrs | LC50 | 38** | Debelak, 1975 |
| Cladoceran, Daphnia magna | Potassium dichromate | 86 | 72 hrs | LC50 | 39** | Debelak, 1975 |
| Cladoceran, Daphnia magna | Potassium dichromate | 86 | 72 hrs | LC50 | 42** | Debelak, 1975 |
| Cladoceran, Daphnia magna | Potassium dichromate | 86 | 72 hrs | LC50 | 44** | Debelak, 1975 |
| Cladoceran, Daphnia magna | Potassium dichromate | 100 | 100 hrs | LC50 | 140 | Dowden & Bennett, 1965 |
| Cladoceran, Daphnia magna | Sodium chromate | 100 | 100 hrs | LC50 | 130 | Freeman & Fowler, 1953 |
| Cladoceran, Daphnia magna | Potassium dichromate | - | 96 hrs | LC50 | 50 | Trabalka & Gehrs, 1977 |
| Cladoceran, Daphnia magna | Potassium dichromate | - | Life span <32 days | Life span and fecundity reduced | 10 | Trabalka & Gehrs, 1977 |
| Cladoceran, Daphnia magna | Potassium chromate | 44 | 2 hrs | Lethal | 100 | Lee & Bulkema, 1979 |
| Coho salmon, Oncorhynchus kisutch | Potassium chromate | - | 13 days | LC50 | 25,000 | Holland, et al. 1960 |
| Coho salmon, Oncorhynchus kisutch | Sodium dichromate | 60 | 14 days | Mortality after transfer to 30 g/kg seawater | 520 | Sugatt, 1980 |

Table 6. (Continued)

| Species | Chemical | Hardness (mg/l as $CaCO_3$) | Duration | Effect | Result* (µg/l) | Reference |
|---|---|---|---|---|---|---|
| Coho salmon, *Oncorhynchus kisutch* | Sodium dichromate | 60 | 14 days | Mortality after transfer to 20 g/kg seawater | 480 | Sugatt, 1980 |
| Coho salmon, *Oncorhynchus kisutch* | Sodium dichromate | 60 | 28 days | Mortality after transfer to 20 g/kg seawater | 230 | Sugatt, 1980 |
| Chinook salmon, *Oncorhynchus tshawytscha* | Sodium dichromate | 70 | 4 mos | Growth | 16 | Olson & Foster, 1956 |
| Chinook salmon, *Oncorhynchus tshawytscha* | Potassium dichromate | 70 | 12 wks | Mortality and growth | 200 | Olson, 1958 |
| Rainbow trout (embryo), *Salmo gairdneri* | Chromic oxide | 99 | 28 days | LC50 | 180 | Birge, et al. 1978 |
| Rainbow trout, *Salmo gairdneri* | Potassium dichromate | 70 | 7 days | Plasma "cortisol" | 20 | Hill & Fromm, 1968 |
| Rainbow trout, *Salmo gairdneri* | Hexavalent chromium | - | 2 days | Inhibition Na/K-ATPase | 2,500 | Kuhnert, et al. 1976 |
| Rainbow trout, *Salmo gairdneri* | Sodium dichromate | 70 | 14 wks | Growth | 21 | Olson & Foster, 1956 |
| Rainbow trout, *Salmo gairdneri* | Potassium chromate | 334 | 24 hrs | Hematocrits | 2,000 | Schiffman & Fromm, 1959 |
| Rainbow trout, *Salmo gairdneri* | Potassium chromate | 334 | 24 hrs | LC50 | 100,000 | Schiffman & Fromm, 1959 |
| Rainbow trout, *Salmo gairdneri* | Potassium dichromate | - | 15 days | Lethal | 10,000 | Strik, et al. 1975 |
| Goldfish, *Carassius auratus* | Potassium dichromate | 220 | 11 days | LC50 | 30,400 | Adelman & Smith, 1976 |
| Fathead minnow, *Pimephales promelas* | Potassium dichromate | 220 | 11 days | LC50 | 17,300 | Adelman & Smith, 1976 |
| Mosquitofish, *Gambusia affinis* | Potassium chromate | - | 96 hrs | LC50 | 107,000 | Wallen, et al. 1957 |

Table 6. (Continued)

| Species | Chemical | Hardness (mg/l as $CaCO_3$) | Duration | Effect | Result* (µg/l) | Reference |
|---|---|---|---|---|---|---|
| Mosquitofish, *Gambusia affinis* | Potassium dichromate | - | 96 hrs | LC50 | 99,000 | Wallen, et al. 1957 |
| Mosquitofish, *Gambusia affinis* | Sodium chromate | - | 96 hrs | LC50 | 135,000 | Wallen, et al. 1957 |
| Mosquitofish, *Gambusia affinis* | Sodium dichromate | - | 96 hrs | LC50 | 92,000 | Wallen, et al. 1957 |
| Bluegill, *Lepomis macrochirus* | Potassium dichromate | 105 | 2 wks | Increased locomotor activity | 50 | Ellgaard, et al. 1978 |
| Largemouth bass (embryo), *Micropterus salmoides* | Chromic oxide | 99 | 8 days | LC50 | 1,170 | Birge, et al. 1978 |
| Largemouth bass, *Micropterus salmoides* | Potassium chromate | 334 | 36 hrs | Pathology of intestine | 94,000 | Fromm & Schiffman, 1958 |
| Largemouth bass, *Micropterus salmoides* | Potassium chromate | 334 | 48 hrs | LC50 | 195,000 | Fromm & Schiffman, 1958 |
| Salamander (embryo), *Ambystoma opacum* | Chromic oxide | 99 | 8 days | LC50 | 2,130 | Birge, et al. 1978 |
| | | SALTWATER SPECIES | | | | |
| Polychaete worm, *Ctenodrilus serratus* | Chromium trioxide | - | 21 days | 100% mortality | 50,000 | Reish & Carr, 1978 |
| Polychaete worm, *Ophryotrocha diadema* | Chromium trioxide | - | 21 days | 100% mortality | 50,000 | Reish & Carr, 1978 |
| Polychaete worm, *Ophryotrocha diadema* | Chromium trioxide | - | 28 days | Brood size decrease | 500-1,000 | Reish & Carr, 1978 |
| Polychaete worm (juvenile), *Neanthes arenaceodentata* | Chromium trioxide | - | 28 days | 50% mortality | 700 | Reish, et al. 1976 |
| Polychaete worm (adult), *Neanthes arenaceodentata* | Chromium trioxide | - | 28 days | 50% mortality | 550 | Reish, et al. 1976 |

Table 6. (Continued)

| Species | Chemical | Hardness (mg/l as $CaCO_3$) | Duration | Effect | Result* (µg/l) | Reference |
|---|---|---|---|---|---|---|
| Polychaete worm, Neanthes arenaceodentata | Potassium dichromate | - | 7 days | 50% mortality | 1,440-1,890 | Oshida, et al. 1976 |
| Polychaete worm, Neanthes arenaceodentata | Potassium dichromate | - | 56 days | 50% mortality | 200 | Oshida & Reish, 1975 |
| Polychaete worm, Neanthes arenaceodentata | Potassium dichromate | - | 14 days | Inhibition-tube building | 79 | Oshida & Reish, 1975 |
| Polychaete worm, Neanthes arenaceodentata | Potassium dichromate | - | 59 days | 50% mortality | 200 | Mearns, et al. 1976 |
| Polychaete worm, Neanthes arenaceodentata | Potassium dichromate | - | 7 days | 50% mortality | 1,630 | Mearns, et al. 1976 |
| Polychaete worm, Neanthes arenaceodentata | Potassium dichromate | - | 350 days | Brood size decrease | 12.5 | Mearns, et al. 1976 |
| Polychaete worm (adult), Capitella capitata | Chromium trioxide | - | 28 days | 50% mortality | 280 | Reish, et al. 1976 |
| Polychaete worm (adult), Capitella capitata | Potassium dichromate | - | 5 mos | Brood size decrease | 50-100 | Reish, 1977 |
| Polychaete worm, Nereis virens | Sodium chromate | - | 21 days | 50% mortality | 1,000 | Raymont & Shields, 1963 |
| Polychaete worm, Nereis virens | Potassium chromate | - | 7 days | 50% mortality | 700 | Eisler & Hennekey, 1977 |
| Soft shell clam, Mya arenaria | Potassium chromate | - | 7 days | 50% mortality | 8,000 | Eisler & Hennekey, 1977 |
| Mudsnail, Nassarius obsoletus | Potassium chromate | - | 7 days | 50% mortality | 10,000 | Eisler & Hennekey, 1977 |
| Hermit crab, Pagurus longicarpus | Potassium chromate | - | 7 days | 50% mortality | 2,700 | Eisler & Hennekey, 1977 |
| Shore crab, Carcinus maenas | Sodium chromate | - | 12 days | 50% mortality | 60,000 | Raymont & Shields, 1963 |

Table 6. (Continued)

| Species | Chemical | Hardness (mg/l as $CaCO_3$) | Duration | Effect | Result* (µg/l) | Reference |
|---|---|---|---|---|---|---|
| Grass shrimp, Palaemonetes pugio | Potassium chromate | - | 48 hrs 10 C, 10 g/kg salinity | 50% mortality | 81,000 | Fales, 1978 |
| Grass shrimp, Palaemonetes pugio | Potassium chromate | - | 48 hrs 15 C, 10 g/kg salinity | 50% mortality | 39,000 | Fales, 1978 |
| Grass shrimp, Palaemonetes pugio | Potassium chromate | - | 48 hrs 20 C, 10 g/kg salinity | 50% mortality | 37,000 | Fales, 1978 |
| Grass shrimp, Palaemonetes pugio | Potassium chromate | - | 48 hrs 25 C, 10 g/kg salinity | 50% mortality | 21,000 | Fales, 1978 |
| Grass shrimp, Palaemonetes pugio | Potassium chromate | - | 48 hrs 10 C, 20 g/kg salinity | 50% mortality | 147,000 | Fales, 1978 |
| Grass shrimp, Palaemonetes pugio | Potassium chromate | - | 48 hrs 15 C, 20 g/kg salinity | 50% mortality | 107,000 | Fales, 1978 |
| Grass shrimp, Palaemonetes pugio | Potassium chromate | - | 48 hrs 20 C, 20 g/kg salinity | 50% mortality | 78,000 | Fales, 1978 |
| Grass shrimp, Palaemonetes pugio | Potassium chromate | - | 48 hrs 25 C, 20 g/kg salinity | 50% mortality | 77,000 | Fales, 1978 |
| Prawn (juvenile), Leander squilla | Sodium chromate | - | 7 days | Toxic threshold | 5,000 | Raymont & Shields, 1963 |
| Prawn (adult), Leander squilla | Sodium chromate | - | 7 days | Toxic threshold | 10,000 | Raymont & Shields, 1963 |
| Brittle star, Ophiothrix spiculata | - | - | 7 days | 50% mortality | 1,700 | Oshida & Wright, 1978 |
| Starfish, Asterias forbesi | Potassium chromate | - | 7 days | 50% mortality | 10,000 | Elsler & Hennekey, 1977 |

Table 6. (Continued)

| Species | Chemical | Hardness (mg/l as $CaCO_3$) | Duration | Effect | Result* (µg/l) | Reference |
|---|---|---|---|---|---|---|
| Mummichog, *Fundulus heteroclitus* | Potassium chromate | - | 7 days | 50% mortality | 44,000 | Eisler & Hennekey, 1977 |
| Speckled sanddab, *Citharichthys stigmaeus* | Potassium dichromate | - | 21 days | 50% mortality | 5,400 | Sherwood, 1975 |
| Speckled sanddab, *Citharichthys stigmaeus* | Potassium dichromate | - | 21 days | EC50-feeding response | 2,200 | Sherwood, 1975 |
| Speckled sanddab, *Citharichthys stigmaeus* | Potassium dichromate | - | 21 days | 50% mortality | 5,000 | Mearns, et al. 1976 |
| Silver salmon, *Oncorhynchus kisutch* | Potassium chromate | - | 5 days | 33% mortality | 31,800 | Holland, et al. 1960 |
| Silver salmon, *Oncorhynchus kisutch* | Potassium chromate | - | 11 days | 100% mortality | 31,800 | Holland, et al. 1960 |
| | | Trivalent Chromium | | | | |
| | | FRESHWATER SPECIES | | | | |
| Snail (embryo), *Amnicola* sp. | - | 50 | 96 hrs | LC50 | 12,400 | Rehwoldt, et al. 1973 |
| Cladoceran, *Daphnia magna* | Chromic chloride | - | 64 hrs | LC50 | 1,200 | Anderson, 1948 |
| Cladoceran, *Daphnia magna* | Chromic nitrate | 206 | 21 days | Reproduction inhibited | 44 | Chapman, et al. Manuscript |
| Cladoceran, *Daphnia magna* | Chromic sulfate | - | 48 hrs | LC50 | 4-8 | Dowden & Bennet, 1965 |
| Cladoceran, *Daphnia magna* | Chromium chloride | 45 | 3 wks | LC50 | 2,000 | Biesinger & Christensen, 1972 |
| Cladoceran, *Daphnia magna* | Chromium chloride | 45 | 3 wks | Chronic value | 445 | Biesinger & Christensen, 1972 |

Table 6. (Continued)

| Species | Chemical | Hardness (mg/l as $CaCO_3$) | Duration | Effect | Result* (µg/l) | Reference |
|---|---|---|---|---|---|---|
| | | | SALTWATER SPECIES | | | |
| Polychaete worm, Neanthes arenaceodentata | Chromium chloride | - | <24 hrs | 100% mortality | 50,400 (pH=4.5) | Mearns, et al. 1976 |
| Polychaete worm, Neanthes arenaceodentata | Chromium chloride | - | 160 days | Reproduction occurred | 50,400 (pH=7.9) | Mearns, et al. 1976 |

* Results are expressed as chromium, not as the compound.

**Animals were fed during test

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## Mammalian Toxicology and Human Health Effects

### INTRODUCTION

Chromium (Cr) is a common element, present in low concentrations throughout nature. Its toxicity has long been recognized, but detailed analysis of toxic effects is complicated by the occurrence of many different compounds of the metal; these may contain Cr in different valence states and are distinguished by their chemical, physical, and toxicological properties.

This document considers relevant chemical and physical properties of Cr compounds to which man may be exposed, and attempts to evaluate possible health hazards associated with such exposures. The general area of environmental effects of chromium compounds was recently reviewed by the U.S. Environmental Protection Agency (U.S. EPA, 1978); a valuable discussion of the medical and biological effects of Cr in the environment is found also in a volume published by the National Academy of Sciences (NAS, 1974). Occupational hazards of chromium were assessed in a Criteria Document prepared in 1975 [National Institute for Occupational Safety and Health (NIOSH), 1975]. Mertz (1969) provided a valuable survey of the biochemical properties of Cr compounds. A general review of the occurrence, metabolism, and effects of chromium has been presented by the NAS (1977).

To avoid unnecessary duplication, previously reviewed material will not be considered at great length except when it impinges directly on present critical considerations. Detailed documentation for most of the available information can be found in the earlier reviews.

There is little need to discuss here the detailed chemistry of chromium, as this subject has been adequately reviewed in the recent past (U.S. EPA, 1978). However, an evaluation of the significance of various routes of exposure to compounds containing Cr, and of the factors determining rates of uptake and toxicity of such compounds, requires an understanding of their physical properties and of their chemical and biochemical reactions.

The metallic element Cr belongs to the first series of transition elements, and occurs in nature primarily as compounds of its trivalent [Cr (III)] form. Generally speaking, the hexavalent compounds are relatively water-soluble and readily reduced to the more insoluble and stable forms of Cr (III) by reaction with organic reducing matter. Because large amounts of Cr (VI) are produced and utilized in industry (primarily as chromates and dichromates), and because of their ready solubility, traces of such compounds are frequently found in natural waters.

As pointed out, Cr (VI) is rapidly reduced when in contact with biological material. The reverse reaction is not known to occur in the human body. Trivalent Cr forms stable hexacoordinate complexes with many molecules of biochemical interest. Interaction of Cr (III) with such compounds may involve binding to carboxy- groups of proteins or smaller metabolites, coordination with certain amino acids, and binding to nucleic acids and nucleoproteins. This last reaction is of special significance in the consideration of the carcinogenic potential of Cr compounds. The field was reviewed by Mertz (1969) and it suffices here to emphasize the stability of these Cr complexes, and the fact that the element is found combined

with both RNA and DNA. An effect of Cr on the tertiary structure of nucleic acids is clearly indicated. In general, it may be concluded that reduction of Cr (VI) to Cr (III) and its subsequent coordination to organic molecules of biochemical interest explain in large measure the biological reactivity of Cr compounds. Thus, the well-known reaction of Cr with skin proteins (i.e., the tanning process) involves coordination sites of Cr (III). For reasons of solubility, however, uptake of compounds of Cr (VI) by the living organism generally exceeds that of Cr (III) compounds (see Acute, Subacute, and Chronic Toxicity section).

A good illustration of the behavior of Cr compounds in biological systems is furnished by the reaction of Cr with erythrocytes (Gray and Sterling, 1950). These cells do not react to any significant extent with Cr (III); in contrast, they rapidly take up anions of hexavalent Cr compounds, presumably utilizing the broadly specific anion transport facilitation in erythrocytes reviewed by Fortes (1977). Thus, we may invoke as a likely explanation for the greater toxicity of Cr (VI) than of Cr (III) compounds their more rapid uptake by tissues due to their solubility and to the facilitation of their translocation across biological membranes. Once within cells, the Cr (VI) is likely to be reduced to the trivalent state before reacting with cell constituents such as proteins and nucleic acids. In the case of red cells, it is such an intracellular reaction of Cr (III) with hemoglobin which explains the essentially irreversible uptake of the metal and permits use of chromium-51 as red cell marker.

Stable and soluble compounds of Cr (III) are found in many biological systems. Among these is the so-called glucose tolerance factor (GTF) (Mertz, 1969), a compound of unknown structure whose absence is believed responsible for symptoms of chromium deficiency. In the form of GTF and perhaps of other similar complexes Cr (III) can also cross biological membranes with relative ease; thus it is readily absorbed from the intestine in this form (Doisy, et al. 1971). One may recall in this connection the general importance of metal ligands in determining movement of heavy metals within the body (Collins, et al. 1961; Foulkes, 1974). It is not surprising therefore that distribution of Cr in the body also critically depends on the presence of specific ligands (Mertz, 1969).

Chromium plays a role in human nutrition. Because of this fact, lowering of ambient Cr levels to a value where total uptake might lead to overt Cr deficiency must be avoided. Indeed, effects of Cr deficiency in man and experimental animals have been described (Mertz, 1969). Levels of Cr compounds required for optimal nutrition fall greatly below those which have been reported to cause toxic effects (see Acute, Subacute, and Chronic Toxicity section); therefore normal nutritional levels need not be considered further here. It must be pointed out, however, that the American diet may be potentially deficient in Cr so that some increased Cr uptake might be beneficial.

Sources of chromium in the environment have been recently reviewed (U.S. EPA, 1978). Although Cr is widely distributed, with an average concentration in the continental crust of 125 mg/kg, it is rarely found in significant concentrations in natural waters.

Air levels in nonurban areas usually fall below detection limits and may be as low as 5 pg/$m^3$. Much of the detectable Cr in air and water is presumably derived from industrial processes, which in 1972 utilized 320,000 metric tons of the metal in the United States alone. A significant fraction of this amount entered the environment; additional amounts are contributed by combustion of coal and other industrial processes (U.S. EPA, 1974). As a result, levels of Cr in air exceeding 0.010 µg/$m^3$ have been reported from 59 of 186 urban areas examined (U.S. EPA, 1973). Mean concentrations of Cr in 1,577 samples of surface water were reported as 9.7 µg/l (Kopp, 1969). The significance of 9.7 µg/l as a mean value is questionable because only 25 percent of the samples tested contained any detectable Cr. Occasional values of total Cr [Cr (III) and Cr (VI)] exceeded 50 µg/l, a fact which must be noted in relation to the recommended standard for domestic water supplies (see Existing Guidelines and Standards section).

It is important to reemphasize at this time the analytical difficulties attending estimation of low concentrations of Cr, especially in biological materials. Additionally, the different chemical species of Cr which may be present often cannot be clearly separated. Considerable uncertainty attaches to the significance of some results, particularly those obtained with some of the older techniques. This topic was considered in detail recently (U.S. EPA, 1978).

## EXPOSURE

### Ingestion from Water and Food

At an average concentration of approximately 10 ug Cr/l drinking water (Kopp, 1969), and a daily water consumption of 2 l, about 20 ug Cr would be ingested in water per day compared to about 50 to 100 ug/day in the American diet (Tipton, 1960). Dietary Cr intake on a hospital diet averaged about 100 ug/day, while an estimate for self-selected diets is 280 ug/day (NAS, 1974). Fractional absorption of such an oral load from the intestine depends on the chemical form in which the element is presented (see Introduction section). In addition, even though mechanisms involved in the movement of Cr compounds across intestinal epithelial barriers are not understood, it is likely that the extent of this absorption will be greatly influenced by the presence of other dietary constituents in the intestinal lumen (MacKenzie, et al. 1958), as has frequently been observed in the case of other ingested metals.

For a variety of reasons, therefore, net fractional absorption of Cr from the intestine is low and may amount to only a few percent or even less (Mertz, 1969), depending especially on the chemical form in which the element is ingested. Intake of Cr from the air normally amounts to less than 1 ug/day (see Inhalation section), and thus does not contribute significantly to normal Cr balance. Average urinary excretion of Cr has been reported as 5 to 10 ug per day (Volkl, 1971); recent work suggests that because of analytical difficulties, actual values may be somewhat lower (Guthrie, et al. 1979). In any case, it follows that the American diet may become marginally deficient in this element, unable to provide the optimum

level required for normal function (see Introduction section). This conclusion is supported by the finding that Cr levels in tissues generally decrease with age (Mertz, 1969). The situation is not greatly altered by application of Cr-containing fertilizers or sewage sludges to agricultural land. Indeed, uptake of Cr by plants from soil is generally low.

A bioconcentration factor (BCF) relates the concentration of a chemical in aquatic animals to the concentration in the water in which they live. An appropriate BCF can be used with data concerning food intake to calculate the amount of chromium which might be ingested from the consumption of fish and shellfish. Residue data for a variety of inorganic compounds indicate that bioconcentration factors for the edible portion of most aquatic animals is similar, except that for some compounds bivalve molluscs (clams, oysters, scallops, and mussels) should be considered a separate group. An analysis (U.S. EPA, 1980a) of data from a food survey was used to estimate that the per capita consumption of freshwater and estuarine fish and shellfish is 6.5 g/day (Stephan, 1980). The per capita consumption of bivalve molluscs is 0.8 g/day and that of all other freshwater and estuarine fish and shellfish is 5.7 g/day.

The BCF for hexavalent chromium in fish muscle appears to be less than 1.0 (Buhler, et al. 1977; Fromm and Stokes, 1962) but values of 125 and 192 were obtained for oyster and blue mussel, (U.S. EPA, 1980b), respectively. For trivalent chromium BCF values of 116, 153, and 86 were obtained with the American oyster (Shuster and Pringle, 1969) and soft shell clam and blue mussel (Cappuzzo and Sasner, 1977), respectively. It appears that the two valence

states of chromium have about the same BCF values and that the geometric mean of 130 can be used for bivalve molluscs. If the values of 0.5 and 130 are used with the consumption data, the weighted average bioconcentration factor for chromium and the edible portion of all freshwater and estuarine aquatic organisms consumed by Americans is calculated to be 16.

Inhalation

Levels of Cr in air have been carefully monitored. In the United States in 1964, an average value of 0.015 $\mu g/m^3$ was reported, with a maximum of 0.35 $\mu g/m^3$. More recent values show levels below detection limits in most nonurban and some urban areas (U.S. EPA, 1973); yearly averages exceeded 0.01 $\mu g/m^3$ in only 59 of 186 urban areas.

The chemical form of Cr in air will vary, depending primarily on its source. There is little information on the size distribution of the particles, but it is safe to assume that a significant portion will be in the respirable range. Uptake, of course, depends on the aerodynamic diameter of the particles. Assuming both an average alveolar ventilation of 20 $m^3$/day, and an alveolar retention of 50 percent of Cr present at a level of 0.015 $\mu g/m^3$, alveolar uptake would only amount to approximately 0.2 $\mu g$/day. Additional Cr could also be deposited in the upper respiratory passages and contribute ultimately to the intestinal load of Cr. In any case, inhalation under normal conditions does not contribute significantly to total Cr uptake.

Even in the nonoccupational environment the concentration of Cr in air may rise significantly above normal background levels.

Thus, increased ambient concentrations of Cr have been reported in the vicinity of industrial sites (U.S. EPA, 1978). In the proximity of water cooling towers, for instance, where Cr was employed as a corrosion inhibitor, air levels of Cr as high as 0.05 $ug/m^3$ have been reported. However, even such a relatively high level is not likely to greatly alter total Cr uptake. The possibility that smoking might contribute to the pulmonary load of Cr has not been fully evaluated.

Of course, if the lungs represent a target organ for Cr, additional pulmonary loads may assume significance even though total body Cr may not have been materially increased by the inhalation exposure. Although such exposure can lead to a significant increase in urinary excretion of Cr, it is not clear to what extent the Cr added to systemic pools originated in the lungs or was alternatively absorbed from the intestines following pulmonary clearance of the Cr-containing particles. In any case, pulmonary Cr does not appear to be in full equilibrium with other Cr pools in the body. This conclusion is based on the fact that the Cr content of the lungs, unlike that of the rest of the body, may actually increase with age (Mertz, 1969). Prolonged pulmonary retention of inhaled Cr is also reflected by the fact that the pulmonary concentration of the element usually exceeds that of other organs. The relatively slow clearance of Cr from the lungs was also noted by Baetjer, et al. (1959a), who found that 60 days after intratracheal instillation into guinea pigs, 20 percent of a dose of $CrCl_3$ remained in this tissue.

Dermal

Compounds of Cr permeate the skin fairly readily when applied in the hexavalent form; trivalent Cr compounds react directly with epithelial and dermal tissue. Cutaneous exposure is primarily a problem of the workplace; many lesions have been described under these conditions, including ulceration and sensitization. There is little evidence, however, to suggest that cutaneous absorption significantly contributes to the total body load of Cr in the normal environment.

Evaluation of Relative Contribution of Different Exposure Routes to Body Burden

The three previous sections review briefly the uptake of Cr by ingestion, inhalation, and cutaneous absorption. None of the three routes of entry will lead to harmful levels of Cr in the body when exposure involves only the low levels of the element normally found in food, water, and air. Indeed, it may be recalled (see Ingestion section) that the average American may actually suffer from mild Cr deficiency. The major fraction of body Cr originating in the general environment is contributed by ingestion. In industrial surroundings, by contrast, other routes of exposure may become more significant. Uptake of Cr by inhalation may pose special risks here. This conclusion follows from the fact that the lungs tend to retain Cr more than do other tissues (see Inhalation section). The Carcinogenicity section deals further with pulmonary effects of exposure to Cr in air.

Under normal conditions of exposure, considerable variability has been observed in the Cr concentrations of different tissues.

It is difficult to assess, however, to what extent the wide range of values reported reflects analytical problems rather than true individual variations. As a first approximation, an average level of around 2 µg Cr/g ash may be derived from the work of Tipton and Cook (1963) and of Imbus, et al. (1963) for most soft tissues and for whole blood of nonexposed humans. Levels of Cr in the lungs may be ten times higher; there is no evidence to suggest that Cr is a bone-seeking element. If we further assume that the average ash content of soft tissues approximates 1 percent of fresh weight, a total body burden in the adult of the order of 2 mg may be calculated. Results of Schroeder, et al. (1962) showed values of Cr in human tissues of the order of 0.05 µg/g fresh weight, which would correspond to a total adult body burden of around 3 to 4 mg; Schroeder (1965) suggested an upper limit of 6 mg Cr in a 70 kg man. These values are presented here to indicate the net result of Cr uptake by ingestion, inhalation, and cutaneous absorption under normal conditions. As pointed out, this body burden may actually represent a marginally deficient state.

## PHARMACOKINETICS

### Absorption, Distribution, Metabolism, and Excretion

Analysis of the movement of Cr through various body pools, and determination of the size and turnover rates of these pools, are complicated by several facts. In the first place it is likely that different Cr compounds will exhibit different kinetic characteristics in the body; this is well illustrated by the wider body distribution of Cr injected in the form of the glucose tolerance factor than when administered as $CrCl_3$ (Mertz, 1969). Second, the

chemical methods employed for the estimation of biological Cr concentrations do not adequately distinguish between different forms of Cr present in the original sample. For instance, the results of Schroeder, et al. (1962) suggest that both hexavalent and trivalent Cr may occur in the ash of biological materials. However, precise conclusions on this point are difficult because the chemical forms of Cr may be changed during the ashing. Third, difficulties of interpretation arise from the fact that one chemical species of Cr may be transformed into another in the body, for instance as by reduction of Cr (VI) to Cr (III).

The complexity of the pharmacokinetics of Cr to be predicted from such considerations is observed both in man and in experimental animals. This situation may be illustrated by reference to the urinary excretion of Cr under normal conditions. In man, the kidneys account for 80 percent or more of Cr excretion by nonexposed individuals (NAS, 1974); urinary excretion amounts on the average to 5 to 10 μg/day or less (see Ingestion from Water and Food section). Such a value corresponds to less than 1 percent of the total body burden as estimated in the Evaluation of Relative Contribution of Different Exposure Routes to Body Burden section; it also approximately equals the average daily dietary retention of Cr (see Ingestion from Water and Food section). The body thus appears roughly to be in steady state with regard to Cr. It would not be correct to infer, however, that the turnover rates of the various Cr pools in the body all fall below 1 percent/day; this would be true only if Cr taken in by one of the routes of entry discussed in the Exposure section always equilibrated evenly with different body pools.

Although little information is available on changes in specific radioactivities of Cr in different body compartments following administration of $^{51}Cr$, there is strong evidence to show that different compartments exhibit distinctly different turnover kinetics. Lim (1978) reports the kinetics of radiochromium (III) distribution in humans. Three major accumulation and clearance components were found for liver, spleen, and thigh; liver and spleen contained the higher concentrations. Normally in man, the highest concentration of Cr is found in the lungs, and pulmonary levels tend to rise with age while the Cr content of other tissues falls. Apparently the lung obtains most of its Cr from the air, not from oral loads, and pulmonary Cr does not come into equilibrium with other body pools of Cr (see Inhalation section).

Similar conclusions on nonequilibration of body pools can be drawn from measurements on the excretion kinetics of $^{51}Cr$ (III) injected into rats. At least three kinetic compartments were observed in this case (Mertz, et al. 1965), with half-lives respectively of 0.5, 5.9, and 83.4 days. The Cr in a slowly equilibrating compartment in man was estimated to possess a half-life of 616 days (U.S. EPA, 1978). Injection of 1 mg of unlabeled Cr into rats, a very large dose compared to the presumptive body burden as calculated in the Evaluation of Relative Contributions of Different Exposure Routes to Body Burdens section, exerted little effect on the rate of tracer excretion from the slow compartment. The finding that even a very large excess of Cr does not affect this compartment further indicates that ingested or injected Cr does not

necessarily pass through every body compartment on its way to excretion. Finally, this conclusion is supported by the observation that the pool from which Cr (at least in some systems) enters plasma following administration of glucose is not readily labeled by injected $^{51}Cr$ (administered as $CrCl_3$) (Mertz, 1969).

As is the case with other metals, chromium normally circulates in plasma primarily in a bound, nondiffusible form (Mertz, 1969). At low levels of Cr (III) the iron-binding protein siderophilin complexes most of the Cr present, but at higher levels of Cr other plasma proteins also become involved. The high affinity of Cr (III) for siderophilin presumably reflects the fact that this protein provides the normal mechanism of transport for Cr to the tissues. A small fraction of plasma Cr is also present in a more diffusible form, complexed to various small organic molecules which are filtered at the glomerulus and partially reabsorbed in the renal tubule. The suggestion that this reabsorption may involve an active transport process (Davidson, et al. 1974) is not supported by the evidence presented. Chromium very tightly bound in low-molecular weight complexes such as Cr-EDTA may serve as a glomerular indicator, being freely filtered but not reabsorbed (Stacy and Thorburn, 1966).

The half-life of plasma Cr is relatively short, and cells tend to accumulate the element to levels higher than that present in plasma. Presumably this accumulation results from intracellular trapping of Cr compounds which penetrate cells in the hexavalent form and then react with cell constituents, such as hemoglobin in the case of the erythrocyte. Within the cells, Cr (VI) will be re-

duced to Cr (III) and remain trapped in this form. In any case, the lack of equilibration of Cr between plasma and cells renders invalid the use of plasma levels as indicators of total exposure.

Another reason for the limited usefulness of plasma Cr levels as a measure of body burden is the likelihood that plasma Cr can be identified with one of the rapidly excreted Cr compartments discussed above. This is suggested by the finding that even though the rise in plasma Cr reported by some authors to occur after administration of a glucose load is not derived from a rapidly labeled pool, it is followed by increased urinary excretion of Cr (Mertz, 1969). In summary, little can be concluded at this time about the nature, size, or location of the various body pools of Cr whose existence was inferred from tracer equilibration and excretion studies.

The importance of the chemical form of Cr in determining distribution of various compounds between pools is further illustrated by the observation that while inorganic Cr (III) does not appreciably cross the placental barrier, Cr (III) injected into pregnant rats in the form of natural complexes obtained from yeast can readily be recovered from the fetuses (see Mutagenicity section).

As further considered in the Effects section, compounds of Cr (VI) may act as acute irritants whereas those of Cr (III) exert little acute toxic action. Presumably, this fact reflects primarily the poor intestinal absorption of the trivalent compounds, and the strong oxidizing power of Cr (VI). The lungs, however, may accumulate and retain relatively insoluble Cr (III) from respired air, although even in this case Cr (VI) appears to be much more

toxic than Cr (III). Here again toxicity is determined as much by the chemical form of Cr as by its concentration. The additional factor of length of exposure to Cr is apparent in the need to implant the test compound or to inject it intramuscularly before sarcomas are produced at those sites (see Carcinogenicity section). In terms of human exposure, such routes of administration possess little relevance except to emphasize the importance of long-term Cr concentrations in specific body compartments as major determinants of toxicity.

## EFFECTS

### Acute, Subacute, and Chronic Toxicity

Because Cr is generally accepted to be an essential element, the effects of exposure to low levels may be beneficial in deficiency states; such an action of Cr would of course have to be separated from the harmful consequences of exposure to higher concentrations. This can be readily achieved because the amounts of Cr required to produce toxic effects are very much higher than those involved in the correction of possible deficiencies. Thus, the $LD_{50}$ for Cr (III) following its intravenous administration (10 mg/kg weight) exceeds by at least four orders of magnitude the dose needed to relieve impairment of glucose tolerance in Cr-deficient rats (U.S. EPA, 1978). Still higher levels of Cr (III) must be fed by mouth before toxic symptoms appear, a fact related to the relative insolubility and poor intestinal absorption of most compounds of trivalent chromium.

Unlike compounds of Cr (III), those of Cr (VI) tend to cross biological membranes fairly easily and are somewhat more readily ab-

sorbed from the gut or through the skin. The strong oxidizing power of hexavalent Cr explains much of its irritating and toxic properties.

That the concentrations of chromium normally encountered in nature barely meet the requirements for this element in the American diet underlines the fact that natural levels do not constitute a human health hazard. However, acute and chronic toxicity problems associated with exposure to Cr are of concern in the industrial environment or in areas potentially polluted by industrial sources. Such toxic effects are reviewed in detail by NIOSH (1975); they include systemic actions of Cr compounds, in addition to primary lesions at the level of the skin, the respiratory passages, and the lungs. It must be emphasized again that the findings of lesions following exposure to high concentrations of Cr compounds under experimental conditions, or as a result of accidental or deliberate human exposure, may bear little relevance to the probability of Cr exerting similar actions at more normally encountered levels.

Exposure to relatively high levels of Cr has been studied in some detail. Thus, when Cr in the form of $K_2CrO_4$ was administered to dogs over a period of four years at a level of 0.45 mg/l in drinking water, increases in the Cr concentration of liver and spleen were reported; at exposure levels 25 times higher, accumulation in the kidneys also became apparent (Anwar, et al. 1961). However, there were no significant pathological changes associated with such exposures. Similarly, a concentration of 0.45 mg Cr/l did not lead to any overt effects in four cases of prolonged human

exposure (Davids, et al. 1951). Rats tolerated 25 mg/l of Cr (III) or several concentrations of Cr (VI), the highest of which was also 25 mg/l, in the drinking water for one year (MacKenzie, et al. 1958). Exposure to the highest concentration of Cr (VI), however, led to a nine times higher amount of Cr in tissues than the same concentration of Cr (III), a fact reflecting that intestinal absorption of the hexavalent form occurs more readily. An early study by Gross and Heller (1946) mentioned specific symptoms such as rough and dirty coat and tail, sterility, and general sub-normal conditions in young albino rats fed 1,250 pm of $ZnCrO_4$ in the diet for approximately two months. Higher concentrations yielded more severe symptoms. Similar concentrations of Cr given as $K_2CrO_4$, however, induced less severe symptoms. Either $K_2CrO_4$ or $ZnCrO_4$ in drinking water or feed for an unspecified time had no observable adverse effects in mature white mice or albino rats in concentrations of the diet of up to 10,000 ppm (1 percent). Ivankovic and Preussmann (1975) fed $Cr_2O_3$ at 0, 2, or 5 percent of the diet to BD rats of both sexes for 90 days. Dose dependent reductions in organ weights of the liver and spleen were observed, but pathological changes, either macroscopic or histological, were not found in these or other organs. No other effects were noted.

These findings support the conclusion that few systemic changes would be expected to result from even moderately elevated oral exposure to Cr. On that basis the standard of Cr established for drinking water (see Existing Guidelines and Standards section) should provide adequate protection against general systemic effects. The question of the safety of such a level in terms of pos-

sible carcinogenic effects is considered in the Carcinogenicity section.

On the other hand, evidence for systemic lesions following more massive exposure, is well documented (U.S. EPA, 1978; NAS, 1974).

High concentrations of Cr causes renal damage. Thus, intra-arterial injection of dichromate has been used for the experimental production of lesions restricted to the first portion of the proximal tubule (Nicholson and Shepherd, 1959). Similarly, tubular necrosis has repeatedly been observed following massive accidental or deliberate exposure (suicide attempts) to Cr (NAS, 1974). These cases, however, represent acute effects of very high doses and their significance to environmental considerations is small.

In only one instance was an association between occupational chromium exposure and hepatic lesions reported. A small number of workers were excreting large amounts of Cr in their urine. Hepatic changes were observed in biopsies although no overt clinical symptoms were seen. Among other systems shown to respond to high doses of Cr is the dog intestine (U.S. EPA, 1976). Although the possibility of more subtle and long range systemic effects of high Cr exposure cannot be excluded, there is no evidence to support its likelihood.

The effects of Cr compounds on the skin were recognized over 150 years ago. Since that time they have been studied in depth by many investigators, and reviewed in considerable detail (NAS, 1974). Earlier cases described in this review were ulcerative changes developing from contact with various compounds of Cr (VI).

Later studies emphasized that workers exposed to Cr (VI) could develop allergic contact dermatitis; sensitivity also appeared to develop to higher levels of Cr (III). No evidence could be found for an association between chromium exposure and skin cancers.

In general, these reports concern relatively massive exposures, unlikely to occur outside the occupational environment, and made even less likely at the present time because of generally improved industrial hygiene practices (NIOSH, 1975). It is worth noting that the standard set for permissible levels of Cr in drinking water (see Existing Guidelines and Standards section) is much lower than those reported to affect the skin. No evidence was found to suggest that presently permissible concentrations of Cr in domestic water supplies possess much significance in terms of skin disease.

Subtle changes in pulmonary dynamics have been observed among workers employed in the chromium electroplating industry (Bovett, et al. 1977). The major effect of Cr on respiratory passages consists of ulceration of the nasal septum, with subsequent perforation, and of chronic rhinitis and pharyngitis. The incidence of such effects may become remarkably high at elevated Cr levels in air. Thus, Mancuso (1951) observed nasal septal perforations in 43 to 85 percent of workers exposed in a chromate plant to both tri- and hexavalent Cr in concentrations as high as 1 $mg/m^3$. The reported incidence of rhinitis and pharingitis was even higher. In another survey [U.S. Public Health Service (PHS), 1953], 509 of 897 chromate workers were found with nasal septal perforations. Bloomfield and Blum (1928) had concluded that daily exposure to chromic

acid concentrations exceeding 0.1 $mg/m^3$ causes injury to nasal tissue. Effects of lower concentrations have not been carefully studied, so no accurate conclusions on dose-effect relationships can be drawn.

An additional difficulty in interpreting these results arises from the fact that the exposure of the workers discussed here may not have been associated primarily with airborne Cr: poor work practices leading to local contact almost certainly caused a high proportion of the nasal lesions (NIOSH, 1975). All nasal effects, however, presumably reflect the irritating action of soluble compounds of Cr (VI). There is no evidence to suggest that the ulcerative lesions can give rise to cancers.

In an average concentration of 68 $\mu g/m^3$, Cr (VI) caused some irritation to eyes and throat in a chromate-producing plant (U.S. PHS, 1953). Information available does not permit derivation of meaningful dose-effect relationships. Nevertheless, current evidence indicates that the limit recommended by NIOSH (1975) for the concentration of noncarcinogenic compounds of Cr (VI) in air will protect most workers against irritation of the respiratory passages (see Table 2). This recommended standard permits a time-weighted average exposure to 25 $\mu g$ $Cr/m^3$ of ambient air for a 40-hour week, with a ceiling exposure to 50 $\mu g/m^3$ of air for any 15-minute period.

Teratogenicity

Although the mutagenic properties of certain compounds of Cr are well established, little evidence could be found for fetal damage directly attributable to such compounds. This is somewhat

unexpected in light of placental permeability to at least some forms of Cr (Mertz, 1969). Embryonic abnormalities were produced in the chick when $Na_2Cr_2O_7$ or $Cr(NO_3)_3$ were injected into the yolk sac or onto the chorioallantoic membrane (Ridgway and Karnofsky, 1952). The significance of these data in relation to ingestion of chromium compounds is questionable.

Mutagenicity

Because of the close correlation emerging between carcinogenicity of chemicals and their mutagenic properties in suitable test systems, it is of interest to refer to the work of Venitt and Levy (1974), who reported that soluble chromates of Na, K, and Ca stimulated mutagenesis in E. coli. Negative results were obtained with soluble salts of the two metals below Cr in the periodic table (tungsten and molybdenum), as well as with a soluble compound of Cr (III). Earlier reports (Hueper, 1971) classifying Cr salts under the heading of carcinogenic chemicals without mutagenic properties appear to have been in error.

In recent years much evidence has accumulated to show that compounds of Cr possess the ability to cause transformation and mutation. Both Cr (III) (as $CrCl_3$) and Cr (VI) (as $K_2Cr_2O_7$) in concentrations equitoxic to mice produced similar morphologic changes in tertiary cultures of mouse fetal cells (Rafetto, et al. 1977); it is interesting to note that Cr (VI) caused more extensive chromosomal aberrations than did Cr (III). Wild (1978) reported that potassium chromate produces a dose-dependent cytogenetic effect on bone marrow cells in mice. Bigalief, et al. (1976) observed a significant increase in the frequency of bone marrow cells with chro-

mosome aberrations in rats acutely or chronically poisoned with potassium dichromate. In concentrations as low as $10^{-5}$ M, potassium dichromate also significantly increased gene conversion in a strain of yeast (Bonatti, et al. 1976). The transformation frequency of simian adenovirus in Syrian hamster cells was raised by calcium chromate (Casto, et al. 1977). Hexavalent Cr has been suspected of being responsible for the mutagenic effects of welding fumes (Hedenstedt, et al. 1977). Further, aerosols of Cr (VI) have been held responsible for mutagenic effects found in a group of workers engaged in the production of chromium (Bigalief, et al. 1977). The full significance of these results, however, could not be evaluated in the absence of the detailed publication.

There is little question about the mutagenic and cell transforming capability of chromates. However, in the presence of liver enzymes or gastric juice but not lung enzymes, chromates lose this mutagenic activity (Petrilli and DeFlora, 1977, 1978). These observations were later confirmed by Gruber and Jinnette (1978) who clearly demonstrated that Cr (VI) is reduced to Cr (III).

Carcinogenicity

In addition to the many acute and chronic effects discussed in preceeding sections, carcinogenicity of various Cr compounds has been well documented, at least in man. A series of Cr compounds was listed by the National Institute for Occupational Safety and Health (1977) under the heading of suspected or identified carcinogens in humans. Inclusion in this list was largely based on results of animal experimentation. If, however, one excludes sarcoma production at the site of implantation or injection of the suspected car-

cinogen, the evidence for cancer production in experimental animals is not convincing.

In spite of the demonstration that Cr compounds can cause tumors at various sites in experimental animals, the only well-documented evidence for cancers associated with Cr exposure of humans involves the lungs. The relatively high incidence of lung cancer in the chromate industry has been well documented (NAS, 1974). Industrial exposure, as discussed below, greatly exceeds that attributable to food, water, and air under normal conditions. In considering the risks of pulmonary carcinogenesis in man, the low systemic levels of Cr originating from the diet or from drinking water can be ignored; unlike the pulmonary load of Cr, which does not appear to be in equilibrium with other body stores of the element (see Pharmacokinetic section), ingested Cr is poorly absorbed and presents no risks at normal ambient levels.

The primary emphasis in this field must be placed on the problems associated with pulmonary exposure; no evidence has been adduced for an association in humans between Cr and initiation of cancer at sites other than the lungs. The literature on respiratory cancer in humans up to 1950 has been reviewed by Baetjer (1950): 109 cases had been reported up to that date in the chromate-producing industry, and an additional 11 cases were reported from chromate pigment plants. It seems likely that in all instances Cr (VI) was involved in the effect. In any case, the incidence of respiratory cancer among these work populations significantly exceeded expected values.

Further work on this subject after 1950 is considered in the review prepared by the National Academy of Sciences (1974). Of particular interest is the study of Taylor (1966) on a large group of chromate workers who were followed over a period of 24 years on the basis of records from the U.S. Social Security Administration. Death rate from lung cancer in this group exceeded expected values by a factor of 8.5. Excess incidence of all other cancers amounted only to a factor of 1.3, in agreement with the conclusion stated above that respiratory cancers constitute the major cancer risk associated with Cr exposure in humans. Taylor further reported that the age-adjusted death rate from respiratory cancer increased with the period of exposure, a finding suggesting the existence of a definite dose-response relationship. Little predictive use can be made of this fact as no information on the concentration of potential carcinogens in these studies was available.

An additional difficulty arises in attempts to interpret this information because the specific carcinogen (or carcinogens) responsible for the increased incidence of cancer found in the chromate industry has not been fully identified. Several compounds of Cr are likely to be present in industrial surroundings. Further, a significant portion of workers investigated must have been exposed to other potential or actual carcinogens used in the chemical industry. Finally, the lung cancers observed in industry generally resulted from prolonged exposure. Initial exposure levels are often not known and the only information available refers to Cr levels in air at the time of the final survey. All these factors make it difficult to extract, from data on human subjects, conclu-

sions concerning any significant relationship between degree of Cr exposure and the incidence of lung cancer.

This problem may be illustrated in the work of Mancuso and Hueper (1951). In this study an incidence of cancer of the respiratory system of 66.7 percent of all cancers was observed, compared with a figure of 11.4 percent in a control group. Details of the six Cr workers concerned, with the addition of one worker who died of respiratory cancer outside of the county and who was not included in the above calculation, are shown in Table 1. As clearly emerges from these data, lung cancer arises only after a prolonged exposure and latency period (Bidstrup and Case, 1956). A second point apparent from the table is that the reported levels of Cr in air (average 0.74 mg $CrO_3/m^3$) were very high. These exposure levels were calculated for each individual with adjustments for the occupational history, and show that in each case the major exposure involved water-insoluble Cr. However, the failure to separate hexavalent and trivalent chromium leads to potentially serious underestimations of the actual exposure values. The suggestion that carcinogenicity in these cases could be attributed to Cr (III) is probably not justified (U.S. PHS, 1953); this is further borne out by more recent work with Cr (VI).

Thus, Davies (1978) reported that among workers exposed to Zn chromate in three British factories, an increased mortality due to lung cancer was seen after an induction time as short as one year. Concentrations of Cr, however, were not given. Similarly, Langard and Norseth (1975) observed an increased cancer rate among workers in a Zn chromate plant where no trivalent Cr was utilized. Pulmo-

TABLE 1

Deaths Due to Lung Cancer in Chromate Workers*

| Subject | Years of Exposure | Latent Period (years) | Exposure Levels (mg $CrO_3/m^3$) | | |
|---|---|---|---|---|---|
| | | | Water Insoluble | Water Soluble | Total |
| CB | 9.0 | 10.0 | 0.37 | 0.17 | 0.54 |
| TG | 14.5 | 14.3 | 0.37 | 0.08 | 0.45 |
| FJ | 12.5 | 12.5 | 0.19 | 0.02 | 0.21 |
| JK | 7.5 | 9.0 | 0.92 | 0.29 | 1.21 |
| EL | 9.2 | 14.0 | 1.12 | 0.15 | 1.27 |
| ESM | 2.0 | 7.2 | 0.19 | 0.02 | 0.21 |
| WDS | 7.2 | 7.2 | 1.12 | 0.15 | 1.27 |
| Mean | 8.8 | 10.6 | 0.61 | 0.13 | 0.74 |

The exposure levels were calculated for each individual on the basis of his occupational history, and are expressed in terms of $CrO_3$.

*Source: Mancuso and Hueper, 1951

nary cancer was identified in three workers who had been exposed to levels of 0.5 to 0.9 mg $Cr/m^3$ for 6 to 9 years. In addition, a single case of adenocystic carcinoma of the nasal cavity was also reported. Attention must again be drawn to the fact that such exposures involve Cr concentrations which are relatively massive when compared to recommended standards (see Existing Guidelines and Standards section). The standard for occupational exposure in air mandates levels of poorly soluble mono- or dichromates not exceeding 1 $\mu g/m^3$.

Attempts to produce lung cancers in experimental animals by feeding or inhalation exposure to Cr compounds have not been successful. For example, Ivankovic and Preussman (1975) fed $Cr_2O_3$ at 0, 1, 2, and 5 percent of the diet to BD rats of both sexes for two years. No carcinogenic effects were noted at any dose. Inhalation did cause, however, a variety of pulmonary symptoms (Steffee and Baetjer, 1965). Permitting animals to breathe air from a chromate factory, 1 to 3 mg $Cr/m^3$, produced no bronchogenic carcinomas (Baetjer, et al. 1959b). Nettesheim, et al. (1970) exposed mice to $Cr_2O_3$ dust (25 $mg/m^3$) for 5.5 hours per day, five times each week, for as long as 18 months with similarly negative results. Distribution and elimination of Cr from the lungs were affected by simultaneous infection of the animals with influenza virus. This underscores the importance of factors other than Cr itself in determining possible effects. In any case, not even the relatively prolonged retention of inhaled Cr in the lungs (see Inhalation section) suffices to assure an inhalation exposure adequate for the production of lung cancer under experimental conditions. Experi-

mental lung tumors could only be observed following implantation of pellets prepared from Cr (VI) compounds dispersed in an equal quantity of cholesterol carrier (Laskin, et al. 1970). As was already stated above in reference to the data gathered in epidemiological surveys of lung cancer in humans, such results do not lend themselves to the derivation of dose-effect relationships, nor to extrapolation down to acceptable levels by a linear or any other model.

In the very high concentrations employed for the experimental production of cancer, compounds of Cr may also possess some cocarcinogenic properties. As illustrated by the observation of Lane and Mass (1977), 2.5 mg of chromium carbonyl acted mildly synergistically with 2.5 mg benzo(a)pyrene to produce carcinomas in tracheal grafts in rats. No further reports on the possible cocarcinogenicity of Cr compounds were found. It is conceivable, however, that in the very high concentrations employed experimentally, other Cr compounds might also possess cocarcinogenic properties. Especially likely in view of the recognized risks associated with smoking is the probability that smoking increases the incidence of lung cancer following pulmonary exposure to Cr.

## CRITERION FORMULATION

### Existing Guidelines and Standards

A variety of standards have been recommended for permissible Cr (VI) levels in water and air. Table 2 provides information on standards presently established in the United States, as formulated by various agencies. The high acceptable level of Cr in livestock water is based on the poor absorption of Cr compounds in general from the gut (see Ingestion section). Because of this low fractional absorption, and in view of the fact that the sensitivity of the lungs to Cr appears to exceed that of other tissues, as discussed in the Carcinogenicity section, standards for Cr in air are much lower than those for water.

### Current Levels of Exposure

Although lower Cr limits have been prescribed for air than for water, the standard for noncarcinogenic Cr (VI) in air permits significantly greater uptake of Cr than does that for Cr (VI) in drinking water designed for human consumption. Thus, if we assume both a daily consumption of 2 liters and a fractional gastrointestinal absorption of 5 percent, total uptake from that source would amount to 5 μg/day. In contrast, the criteria discussed in the Inhalation section, i.e., an alveolar ventilation of 20 $m^3$/24 hours with 50 percent alveolar retention of inhaled Cr, would lead to Cr uptake through the lungs of around 80 μg during an 8-hour exposure to levels of 25 μg/$m^3$. The upper limit for carcinogenic Cr (VI) would similarly cause retention of 3 to 4 μg Cr under these conditions.

TABLE 2

Recommended or Established Standards for Cr in the United States

| Medium | Chemical Species | Reference | Standard |
|---|---|---|---|
| Drinking Water Total | Cr (VI) | U.S. Pub. Health Serv. (1962) | 50 µg/l |
| Domestic Water Supply | total chromium | U.S. EPA (1976) | 50 µg/l |
| Fresh Water (aquatic life) | total chromium | U.S. EPA (1976) | 100 µg/l |
| Livestock Water | Cr (VI) | Natl. Acad. Sci./ Natl. Acad. Eng. (1972) | 1 mg/l |
| Work Place Air | carcinogenic Cr (VI)[a] | Natl. Inst. Occup. Safety and Health (1975)[c] | 1 $\mu g/m^3$ |
| | noncarcinogenic Cr (VI)[a] | Natl. Inst. Occup. Safety and Health (1975)[c] | 25 $\mu g/m^3$ TWA[b] 50 $\mu g/m^3$ ceiling |
| | chromic and chromous salts | 40 CFR 1910.1000 | 0.5 $mg/cu^3$ |
| | metal and insoluble salts | 40 CFR 1910.000 | 1.0 $mg/cu^3$ |

[a]Carcinogenic compounds are here taken to include all forms of Cr (VI) other than $CrO_3$ and mono- or dichromates of H, Li, Na, K, Rb, Cs, and $NH_4$

[b]Time-weighted average

[c]NIOSH has recommended these criteria for Cr

No minimum daily requirement for Cr has so far been agreed upon. It is clear, however, that diet rather than water provides the major fraction of daily Cr intake. As a consequence, small absolute changes in water Cr levels should have little bearing on Cr deficiency states.

For criterion derivation purposes, the distinction between Cr (III) and Cr (VI) is justifiable. However, it should be understood that analytical methods are not currently available to distinguish between Cr (III) and Cr (VI) in dilute solutions in natural matrices.

Special Groups at Risk

No such groups have been identified outside the occupational environment.

Basis and Derivation of Criterion

Evidence suggests that inhaled hexavalent chromium [Cr (VI)] is a human lung carcinogen. However, evidence for cancer production in experimental animals from Cr (VI) is not convincing if one excludes sarcoma production at the site of implantation or injection. Furthermore, the oral carcinogenicity of Cr (VI) or Cr (III) has never been demonstrated. For example, two intermediate-length oral feeding studies of Cr (VI), one in dogs for four years (Anwar, et al. 1961), the other in rats for one year (MacKenzie, et al. 1958) gave no evidence of carcinogenicity. Although these latter studies did not specifically test for carcinogenicity and their sample sizes were small, they are consistent with recent human epidemiological data that show no evidence of intestinal cancer after inhalation exposure of Cr (VI) (Hayes, et al. 1980). Furthermore,

existing oral carcinogenicity data for Cr (III) is negative (Ivankovic and Preussmann, 1975), and recent data indicate that in the presence of gastric juice, Cr (VI) is reduced to Cr (III) (Petrilli and DeFlora, 1977, 1978; Gruber and Jinnette, 1978). Accordingly, a protective limit for chromium based on carcinogenesis via inhalation data is difficult to justify (see Appendix).

An alternative approach in establishing protective levels for Cr (VI) and Cr (III) would be the derivation of criteria from toxicity data. A review of the toxicity data in the Effects section of this document indicates that the MacKenzie, et al. (1958) study, in which rats were fed various concentrations of Cr (VI) in their drinking water for one year, is the most suitable study for this calculation for Cr (VI). Although Gross and Heller (1946) utilized higher levels of Cr (VI) in drinking water for several experimental groups, the length of exposure was inadequate (i.e., less than 90 days) at levels producing no toxic effects. The Acceptable Daily Intake (ADI) for rats in the MacKenzie, et al. (1958) study can be found by:

(25.0 mg/l x 0.035 l/d)/0.350 kg/rat = 2.50 mg/d/kg/rat,

where 25 mg/l is a well defined no-observable-adverse-effect level (NOAEL), 0.035 l represents the assumed average daily water intake per rat, and 0.350 kg is the assumed average rat weight.

Dividing this ADI for rats by a safety factor of 1,000 according to the methods previously described in the Federal Register (Vol. 44, No. 52, March 15, 1979, P. 15980)* and then multiplying

*Note: This safety factor of 1,000 was chosen considering only oral exposure data. Thus, evidence for carcinogenicity of Cr (VI) or Cr (III) does not exist after oral exposure. Likewise, the animal toxicity data after oral exposure to these valence states are scanty.

this by 70 kg (the average body weight of man) yields the "safe" ADI for man:

(2.50 mg/d/kg/rat/1,000) x 70 kg/man = 0.175 mg/d/man.

The ambient water concentration of Cr (VI) cann be calculated from this ADI for man by the following equation:

$$C = \frac{\text{ADI mg/d/man}}{\text{2 l/d/man + (0.0065 kg/d/man x BCF)}}$$

where BCF is the average bioconcentration factor for total chromium of 16.0 in units of liters per kilograms, 2 l represents the average daily water intake, and 0.0065 kg is the average amount of fish consumed per day. Thus,

$$C = \frac{\text{0.175 mg/d/man}}{\text{2 l/d/man + (0.0065 kg/d/man x 16.0 l/kg)}}$$

$$= 0.083 \text{ mg/l, or } 83 \text{ µg/l.}$$

A similar toxicity approach can be used to establish a protective level for Cr (III). Several studies are available that give dose levels with no evidence of toxicity (NOAELs): cats fed Cr (III) at 50 to 1,000 mg/day (approximately 10 to 200 mg/kg/day) for 80 days (Akatsuka and Fairhall, 1934); rats fed 5 ppm of Cr (III) in the diet for a lifetime (NAS, 1974); rats fed Cr (III) at 25 ppm via drinking water for one year (MacKenzie, et al. 1958); or rats fed Cr (III) five days a week at 2 or 5 percent of the diet for 90 days or 1, 2, or 5 percent of the diet for two years (Ivankovic and Preussmann, 1975). The latter study seems the most reasonable of the four in terms of establishing a criterion; the Akatsuka and Fairhall (1934) study is too short, while the dose levels of the other two studies (NAS, 1974; MacKenzie, et al. 1958) are too low.

The highest NOAEL in the Ivankovic and Preussmann (1975) study is 5 percent of the diet or 50,000 ppm for Cr (III). The ADI for rats in this study can be found by:

$$\frac{50{,}000 \text{ ppm} \times 0.05 \times (5/7)}{0.350 \text{ kg}} = 5{,}102 \text{ mg/d/kg},$$

where 0.05 is assumed to be the daily feed consumption as a fraction of body weight for a rat, 5/7 is an adjustment factor to derive the average daily Cr (III) intake for a 7 rather than a 5-day week, and 0.350 is the assumed average body weight of the rats.

Dividing this ADI for rats by a safety factor of 1,000 and multiplying by 70 kg (the averge body weight of man) yields the ADI for man:

$$(5{,}102 \text{ mg/d/kg}/1{,}000) \times 70 \text{ kg/man} = 357 \text{ mg/d/man}.$$

The ambient water concentration of Cr (III) that results in this ADI for man can be found by:

$$C = \frac{357 \text{ mg/d/man}}{2 \text{ l/d/man} + (0.0065 \text{ kg/d/man} \times 16.0 \text{ l/kg})}$$

$$= 170 \text{ mg/l}.$$

The protective level based on animal toxicity data for Cr (VI) agrees well with the present standard for total chromium permitted in the domestic water supply: 50 µg/l (U.S. EPA, 1976). This standard appears, through past experience, to be satisfactorily protective against Cr (VI) toxicity in humans, and has been approved by several expert committees. Furthermore, a review of present ambient water chromium concentrations indicates that most waterways contain this metal at concentrations below the present standard. Therefore, the recommended ambient water quality criterion for Cr (VI) is 50 µg/l.

The protective level based on animal toxicity data for Cr (III) and an uncertainty factor of 1,000, corresponding to an ADI of 357 mg/d, is recommended as the ambient water quality criterion for Cr (III): 170 mg/l. Drinking water contributes 95 percent of the assumed exposure while eating contaminated fish products accounts for 5 percent. This criterion can similarly be expressed as 3,433 mg/l if exposure is assumed to be from the consumption of fish and shellfish products alone. The amount of trivalent chromium that can be expected to be present in ambient waters is extremely low because 1) Cr (III) is rapidly hydrolyzed and precipitated as $Cr(OH)_3$, and 2) sorption processes remove the remaining Cr (III) from solution. It should be noted that the criterion value of 170 mg/l far exceeds the Cr (III) concentration that can be expected in ambient waters based upon known solubilities for Cr (III) and its salts.

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## APPENDIX

### Relationship Between Carcinogenicity Information and Water Criterion for Chromium (VI)

Six epidemiological studies, five of which were at different locations (Taylor, 1966; Enterline, 1974; Davies, 1978; Langord and Horseth, 1975; Mancuso and Hueper, 1951; Baetjer, 1950), of up to 1,200 chromate workers strongly indicate that inhalation of Cr (VI) produces lung cancer. These studies, supported by the production of local carcinogenic responses in rats and hamsters at the site of implantation or injection (Laskin, 1970) and the positive mutagenicity of Cr (VI) leave little doubt that Cr (VI) is a human carcinogen. The extent to which ingested Cr (VI) induces cancer is not clear, since it has not been well tested experimentally by the oral route and since there is evidence, albeit uncertain, that Cr (VI) is reduced to Cr (III) in the stomach. Because of these uncertainties, no dose data for Cr (VI) exist on which to base a quantitative risk estimmate of oral carcinogenicity. Therefore, the criterion concentration for Cr (VI) of 50 µg/l, based on its toxicity, should be regarded as a strict upper limit; it does not include any consideration of the carcinogenicity of Cr (VI).

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