Biological Report 85(1.6)
January 1986

Contaminant Hazard Reviews
Report No. 6

CHROMIUM HAZARDS TO FISH, WILDLIFE, AND INVERTEBRATES: A SYNOPTIC REVIEW

by
Ronald Eisler

Patuxent Wildlife Research Center
U.S. Fish and Wildlife Service
Laurel, MD 20708

SUMMARY

Ecological and toxicological aspects of chromium (Cr) in the environment are reviewed, including its chemistry, background residues in biological and abiotic materials, beneficial and protective properties, and toxic and sublethal effects. Recommendations are presented, including proposed criteria for protection of sensitive species of wildlife and aquatic organisms.

Most authorities agree on eight points: (1) chromium levels are elevated in soil, air, water, and biota in the vicinity of electroplating and metal finishing industries, publicly owned municipal treatment plants, tanneries, oil drilling operations, and cooling towers; (2) hexavalent chromium (Cr+6) is the most biologically active Cr chemical species, although little is known about the properties of organochromium compounds, water soluble species, or their interactions in complex mixtures; (3) chromium chemistry is imperfectly understood, and existing analytical methodologies are inadequate for quantification of Cr species and ionic states; (4) chromium is an essential trace element in humans and some species of laboratory animals, but the data base is incomplete for other groups of organisms; (5) at high environmental concentrations, Cr is a mutagen, teratogen, and carcinogen; (6) no biomagnification of Cr has been observed in food chains, and concentrations are usually highest at the lowest trophic levels; (7) toxic and sublethal properties of Cr are modified by a variety of biological and abiotic factors; and (8) sensitivity to Cr varies widely, even among closely related species. As reported here, adverse effects of Cr to sensitive species have been documented at 10.0 ug/L (ppb) of Cr+6 and 30.0 ug/L of Cr+3 in freshwater and 5.0 ug/L of Cr+6 in saltwater and, to wildlife, 5.1 and 10.0 mg of Cr+6 and Cr+3, respectively, per kilogram of diet (ppm). Tissue levels in excess of 4.0 mg total Cr/kg dry weight should be viewed as presumptive evidence of Cr contamination, although the significance of tissue Cr residues is unclear. Some of these findings are in sharp contrast to Cr criteria proposed by regulatory agencies.

This report should be cited as:

Eisler, R. 1986. Chromium hazards to fish, wildlife, and invertebrates: a synoptic review. U.S. Fish and Wildlife Service Biological Report 85 (1.6). 60 pp).

TABLES

INTRODUCTION

Environmental effects of chromium (Cr) have been extensively reviewed (NAS 1974; Steven et al. 1976; Snyder et al. 1977; Towill et al. 1978; Taylor and Parr 1978Langard and Norseth 1979; Post and Campbell 1980; Hatherill 1981; Ecological Analysts 1981). These authorities agree that Cr is used widely in domestic and industrial products and that some of its chemical forms, primarily hexavalent chromium (Cr+6) and trivalent chromium (Cr+3), are toxic. In North America, thousands of tons of Cr ore and concentrates are imported annually for the production of stainless steels, chrome plated metals, pigments for inks and paints, and a wide variety of chemicals. Reports from Europe, Scandinavia, Asia, and North America all emphasize the high incidence of lung cancer and other respiratory diseases among workers involved in the manufacture of chromates. Others document that land dumping of wastes from chromate production and electroplating operations have been responsible for groundwater contamination; that discharge of Cr wastes into streams and lakes has caused damage to aquatic ecosystems and accidental poisoning of livestock; and that large amounts of Cr+3 and Cr+6 are reintroduced into the environment as sewage and solid wastes by the disposal of consumer products containing Cr.

In this account I briefly review the ecological and toxicological effects of Cr to fish and wildlife, with emphasis on migratory species, their predators, and their prey, and also provide current recommendations for the protection of sensitive species of concern to the U.S. Fish and Wildlife Service. This report is part of a continuing series of synoptic reviews prepared in response to informational requests from FWS environmental specialists.

ENVIRONMENTAL CHEMISTRY

The annual world production of chromium is estimated at 7 million metric tons; most of the ore, in the form of chromite ($FeOCr_2O_3$), is produced by the USSR and the Republic of South Africa. In the United States, chromium is used principally in metallurgy and chemical industries (Langard and Norseth 1979). Although natural mobilization of Cr by weathering processes is estimated at 32 thousand tons/year, the amounts of Cr added to the environment as a result of anthropogenic activities are far greater. New York City alone contributes about 440 tons of Cr annually to the environment (Steven et al. 1976). Major atmospheric emissions of Cr are from the chromium alloy and metal producing industries; lesser amounts come from coal combustion, municipal incinerators, cement production, and cooling towers (Towill et al. 1978). Atmospheric emissions contribute 4 to 6 times more Cr to aquatic ecosystems than do liquid wastes (Ecological Analysts 1981). In aquatic environments, the major sources of chromium are the electroplating and metal finishing industries and publicly owned treatment plants; relatively minor sources (other than localized contamination) are iron and steel foundries, inorganic chemical plants, tanneries, textile manufacturing, and runoff from urban and residential areas (Towill et al. 1978; Ecological Analysts 1981). Chromium in phosphates used as fertilizers may be an important source of Cr in soil, water, and some foods (Langard and Norseth 1979). In general, elevated levels of Cr in biological or other samples have been positively correlated with increased industrial and other uses of the element—especially uses associated with plating and foundry applications, chemical manufacturing, and corrosion inhibition (Taylor and Parr 1978).

Chromium, in the crystalline form, is a steel-gray, lustrous, hard metal characterized by an atomic weight of 51.996, an atomic number of 24, a density of 7.14, a melting point of 1900°C, and a boiling point of 2642°C. Four Cr isotopes occur naturally—Cr-50 (4.3%), -52 (83.8%), -53 (9.6%), and -54 (2.4%)—and seven are man-made. Elemental Cr is very stable, but is not usually found pure in nature. Chromium can exist in oxidation states ranging from -2 to +6, but is most frequently found in the environment in the trivalent (+3) and hexavalent (+6) oxidation states. The +3 and +6 forms are the most important because the +2, +4, and +5 forms are unstable and are rapidly converted to +3, which in turn is oxidized to +6 (Towill et al. 1978; Langard and Norseth 1979; Ecological Analysts 1981).

Most compounds prepared from chromite ore contain Cr in the more stable +3 and +6 states. The Cr in essentially all environmentally important Cr compounds is in one of these two oxidation states. Chromium in biological materials is usually in the +3 form (Langard and Norseth 1979), and is the form that functions as an essential element in mammals by maintaining efficient glucose, lipid, and protein metabolism (Steven et al. 1976). In general, the toxicity of trivalent chromium to mammals is low because its membrane permeability is poor and it is noncorrosive; further, there is little tendency for Cr+3 to biomagnify in food chains in the inorganic form. However, organo-trivalent Cr compounds may have significantly different accumulation tendencies

although little is known about these compounds (Steven et al. 1976). Hexavalent chromium is more toxic than the +3 form because its oxidizing potential is high and it easily penetrates biological membranes (Steven et al. 1976; Taylor and Parr 1978; Langard and Norseth 1979; Ecological Analysts 1981). Most of the Cr+6 found in nature is a result of domestic and industrial emissions (Steven et al. 1976). Interaction of +6 chromic oxide, dichromate, or chromate compounds with organic compounds can result in reduction to the comparatively less toxic trivalent form (Taylor and Parr 1978).

Little is known about the relation between concentrations of total Cr in a given environment and biological effects on the organisms living there. Depending on the physical and chemical state of the Cr, the same element concentration has a wide variety of mobilities and reactivities and thus has different effects (Steven et al. 1976). Chromium toxicity to aquatic biota is significantly influenced by abiotic variables such as hardness, temperature, pH, and salinity of water; and biological factors such as species, life stage, and potential differences in sensitivities of local populations (Ecological Analysts 1981). In both freshwater and marine environments, hydrolysis and precipitation are the most important processes that determine the fate and effects of chromium, whereas adsorption and bioaccumulation are relatively minor (Ecological Analysts 1981). Both Cr+3 and Cr+6 can exist in water with little organic matter; Cr+6 is usually the major species in seawater (Towill et al. 1978). Under oxygenated conditions, Cr+6 is the dominant dissolved stable Cr species in aquatic systems. The hexavalent form exists as a component of a complex anion that varies with pH and may take the form of chromate (CrO_4^{-2}), hydrochromate ($HCrO_4^{-1}$), or dichromate ($Cr_2O_7^{-2}$). These ionic Cr+6 forms are highly soluble in water and thus mobile in the aquatic environment. All stable Cr+6 anionic compounds strongly oxidize organic matter on contact and yield oxidized organic matter and Cr+3 (Ecological Analysts 1981). Trivalent Cr tends to form stable complexes with negatively charged inorganic or organic compounds, and thus is unlikely to be found uncomplexed in aqueous solution if anionic or particulate compounds (such as decaying plant or animal tissues, or silt or clay particles) are present (Steven et al. 1976; Pfeiffer et al. 1980; Ecological Analysts 1981). Precipitated Cr+3 hydroxides remain in the sediments under aerobic conditions; under low pH and anoxic conditions, however, Cr+3 hydroxides may solubilize and remain as ionic Cr+3 unless oxidized to Cr+6 through mixing and aeration (Ecological Analysts 1981). Among estuarine sediments, Cr content tends to be highest in those of small grain size and high organic and iron content; concentrations in European estuaries ranged from 3.9 mg/kg in intertidal sands to 162.0 mg/kg in anaerobic muds (Rehm et al. 1984). Adsorption of Cr by sediments is salinity-dependent; adsorption is greatest at salinities of 0.1 to 1.0 ‰ (Mayer and Schick 1981). Colloidal iron strongly scavenges Cr+3 from river water; flocculation of the colloids when they are mixed with seawater, coupled with lack of removal of the colloids to the sediments by gravitational settling or scavenging by suspended sediments, promotes the flux of Cr+3 through the estuary to the open ocean (Mayer et al. 1981).

The solubility and potential bioavailability of waste Cr added to soils through sewage sludge, animal manures, and industrial wastewater are modified by soil pH and organic complexing substances (James and Bartlett 1983a, 1983b). Although soil pH can affect oxidation rates of Cr+6 to Cr+3, organic complexes appear to play a more significant role. For example, organically complexed Cr+3 added to soils may remain soluble for at least a year, whereas the free Cr+3 metal ion in the absence of soluble complexing ligands quickly becomes adsorbed, or hydrolyzed and precipitated. The biological effects of organochromium compounds, which are not well documented, appear to be high-priority subjects for further research.

All toxic effects of Cr+6 seem to be related to the strong oxidizing action of chromates, and all biological interactions of chromates seem to result in reduction to the Cr+3 form and subsequent coordination to organic molecules (Langard and Norseth 1979).

Data on the environmental cycling of Cr are lacking, and those on the biochemistry of Cr are incomplete and sparse (Towill et al. 1978); it is clear that these two subjects merit additional research. Furthermore, there is increasing concern about the uncertainties in the analysis of some types of biological and environmental samples (Towill et al. 1978; Taylor and Parr 1978). For example, collaborating laboratories have reported order-of-magnitude differences in persistence of Cr in standard bovine liver. Until more is learned about the reasons for these differences, caution should be exercised in interpreting past analytical results.

BACKGROUND CONCENTRATIONS

Chromium concentrations in selected nonbiological materials are elevated in the vicinity of industrial operations and municipal waste treatment facilities where chromium is a significant component of wastes discharged into the environment (Tables 1 and 2). The bioavailability of Cr in these materials was mentioned earlier, but the mechanism still remains largely unknown. It is generally agreed that suspended particulates are a major source of transport in aquatic systems, that most Cr in soil and sediment is unavailable to living organisms, that Cr+6 in air and water is hazardous to fish and wildlife, and that the grossly elevated levels of Cr (especially inorganic fractions) in sludge components may have serious implications to wildlife when the sludge is applied to croplands.

Concentrations of Cr in representative species of plants and animals collected worldwide are shown in Table 3. Additional and more comprehensive data were given by Jenkins (1980) and Eisler (1981). Chromium concentrations in species of individual taxonomic groups tended to be elevated when collection localities were near electroplating plants, tanneries, oil drilling operations, sewage outfalls, drift cooling towers, dump sites, or other sources of Cr-containing wastes that were being discharged into the environment.

Among marine algae and invertebrates, for example, comparatively high concentrations of Cr were recorded in algae, clams, and annelids from the vicinity of electroplating plants; in crabs collected near an ocean dump site receiving large quantities of metals; and in algae and echinoderms near urbanized areas in Puerto Rico. Grossly elevated levels of Cr were also noted (Table 3) in selected plasma fractions of tunicate blood, in scales from a few species of teleosts, and in corals from Cr-rich areas containing high concentrations of scandium and titanium; however, these accumulations were not attributed to anthropogenic activities. Many factors are known to modify Cr levels. In marine molluscs, as one example, Cr concentrations tended to increase with the age of the organism (Eisler et al. 1978), but uptake was significantly inhibited at high salinities (Olson and Harrel 1973). Accidental contamination of field samples by metal particles in the samples, rust from stainless hydrowire, or flaking paint from the hull of the collecting ship may also constitute significant sources of elevated Cr residues (Martin and Knauer 1973). In terrestrial ecosystems, elevated Cr levels were reported in cotton rats and plants collected near drift cooling towers and in earthworms and plants from sludge-amended soils (Table 3). However, the high levels of Cr reported in the hair of pronghorns and elk (Table 3) require verification.

A major source of concern at present is the accuracy and precision of chromium analyses in biological samples. One interlaboratory calibration study, involving 87 laboratories, showed that an oyster homogenate averaged 1.1 ppm dry weight, with a standard deviation of 0.5 ppm (Fukai et al. 1978). This means that about 67% of the laboratories were in the range of 0.6 to 1.6 ppm and about 33% of the laboratories reporting were outside this range. It seems clear that more rigorous and more standardized sample preparation techniques and analyses for chromium are needed.

BENEFICIAL AND PROTECTIVE PROPERTIES

Chromium is essential for normal metabolism of insulin and glucose in humans (Langard and Norseth 1979) and for regulating carbohydrate metabolism in mammals (Preston et al. 1976; Onkelinx 1977; Gale 1978; Towill et al. 1978; Post and Campbell 1980). Chromium deficiency has been described in rats, guinea pigs, and squirrel monkeys; signs include reduced growth, decreased life span, elevated serum cholesterol, increased formation of aortic plaques, and signs resembling those of diabetes mellitus. Subjecting Cr-deficient animals to stress can exacerbate the signs (Preston et al. 1976). In humans, Cr deficiency has been suggested as a possible factor in the incidence of diabetes and atherosclerosis. Autopsy data from 31 areas of the world suggested that many Americans, but few non-Americans, were deficient in Cr. One characteristic feature of Cr levels in human tissues is a decline with increasing age (Onkelinx 1977).

Table 1. Chromium concentration in nonbiological materials collected worldwide.

Sample (units in parentheses)	Concentration	Reference[a]
Terrestrial (mg/kg)		
Earth's crust	100–300	1
"	mean, 125	2
Soils	5–300	3
"	trace to 250	2
Granite and limestones	10	3
Serpentine materials	1,800	3
Marsh sediments		
Receiving fertilizers containing sewage sludge, for		
7 years (total dose of 10,300 mg Cr/m^2)	2,150–4,750	4
Control areas	50–54	4
Aquatic		
Suspended particulates (mg/kg)		
Atlantic coastal streams	≤460	5
United States	37–2,000	3
Brazil, electroplating plant		
Distance from discharge site (meters)		
0	2,210–61,070	6
50	15,260	6
600	18,620	6
Sediments (mg/kg)		
Freshwater		
Maine, receiving tannery wastes	≤25,000	7
California	90–140	3
Wisconsin	1–49	3
Rhine River, Germany	max. 1,240	3
Brazil, electroplating plan, distance from discharge site (meters)		
0	1,420–54,300	6
50	24,820	6
600	1,700	6
Marine		
United Kingdom	30–52	8
Rhode Island, near electroplating plant	60–80	9
Maine, near tanneries	80–3,000	10
Water (µg/L)		
Freshwater		
Rivers and lakes	1–10	2
Streams	0–112, mean 9.7	3
Drinking water	Usually <8; rarely >50	3
Untreated industrial effluents	≤5,000,000	11
Vicinity Brazilian electroplating plant		
Waste stream	1,290,000	6
At discharge	≤80,000	6
50 m downstream	54	6
600 m downstream	0.23	6
Seawater	<1–5	2
Seawater	0–0.5	3

Air (µg/m^3)		
Background level	0.001	11
Urban	0.06	11
Occupational exposure, chromate plants	≤1,000	11
Rural areas	≤0.01	2

[a]References: 1, Ecological Analysts 1981; 2, Langard and Norseth 1979; 3, Towill et al. 1978; 4, Giblin et al. 1980; 5, Turekian and Scott 1967; 6, Pfeiffer et al. 1980; 7, Duval et al. 1980; 8, Bryan et al. 1983; 9, Eisler et al. 1977; 10, Mayer et al. 1981; 11, Steven et al. 1976.

Table 2. Concentrations of total Cr and Cr+6 in air, water, soil, and sludge near industrial sites and sewage outfalls in the United States (modified from Snyder et al. 1977).

	Air (µg/m^3)		Water				Soil	Sludge (mg/L)	
			Filtered (mg/L)		Particulates	Sediments	Total	Inorganic	Organic
Industry	Total	Cr+6	Total	Cr+6	Total (mg/L)	Total (mg/kg)	(mg/kg)	Total	Total
Chromium pigment producer	13.5	2.1	3.3	1.3	59.6	568.0	41.0	-	-
Chromium plating facility	1.1	-	0.6	0.6	0.14	1.2	36.9	-	-
Tanning operation	<0.2	-	2.3	0.1	10.8	14.8	-	-	-
Sewage treatment plant									
Receiving tannery wastes	<0.4	-	0.05	0.001	0.5	23.2	-	13,950.0	101.0
Not receiving tannery wastes	<0.2	-	0.04	<0.001	0.09	-	-	911.0	8.6

Table 3. Chromium concentration in field collections of selected species of marine, freshwater, and terrestrial flora and fauna. Values shown are in mg Cr/kg (ppm) whole organism or designated body part fresh weight (FW), dry weight (DW), or ash weight (AW); ND = nondetectable.

Ecosystem, taxonomic group organism, tissue, location and other variables	Concentration (ppm)	Reference[a]
MARINE		
Algae and macrophytes		
Algae, whole		
Sea of Japan, 12 spp.	1.0-14.0 DW	1
United Kingdom, 11 sp.	2.8-30.0 DW	2
Algae and macrophytes, whole		
Puerto Rico, 18 spp.	0.4-110.0 DW	3
Knotted wrack, *Ascophyllum nodosum*		

Whole		
Norway	4.0 DW	4
Great Britain	1.1-10.0 DW	5,6
Bladder wrack, *Fucus vesiculosus*		
Whole		
United Kingdom	2.6-4.5 DW	4,5,6
Phytoplankton, whole		
Narragansett Bay, RI	4.3-73.3 DW	7
Seaweeds, whole		
Japan, 44 spp.	0.1-2.5 DW	8
Korea, 20 spp.	0.7-7.4 DW	9
Marsh grasses, *Spartina* spp., whole		
Control areas	2.3-3.1 DW	10
From areas treated with sewage-amended sludge		
at 10,300 mg Cr/m^2 over 7-year period	31.0-44.0 DW	10
Coelenterates		
Corals, 34 spp.		
Deep open ocean	0.8-3.0 DW	11
Shallow open ocean	2.0-35.0 DW	11
Shallow coastal zone	0.2-23.0 DW	11
Molluscs		
Red abalone, *Haliotis rufescens*		
Gill	0.6-4.0 DW	12
Mantle	0.0-12.6 DW	12
Digestive gland	2.0-13.2 DW	12
Foot	ND	12
Hardshell clam, *Mercenaria mercenaria*		
Soft parts	3.3-24.7 DW	7
Soft parts	0.2-5.8 FW	13
Soft parts	0.8 DW	14
Shell	0.4 DW	14
Periwinkle, *Littorina littorea*		
Soft parts, United Kingdom	<0.1-1.6 DW	15
Common mussel, *Mytilus edulis*		
Soft parts	0.9-2.7 DW	14,16,17,18,19
Soft parts	0.4-21.0 DW	20
Shell	0.1 DW	18
Shell	1.0-2.0 DW	4
Digestive gland	7.4 FW	21
Hepatopancreas	3.5-15.0 DW	22
Gonad	3.0 FW	21
Muscle	11.0 FW	21
Clam, *Pitar morrhuana*		
Soft parts	14.2 DW	23
Squid, unidentified		
Various tissues	3.1-5.4 DW	24
Crustaceans		
Edible tissues		
7 spp.	0.1-0.2 FW	25
9 spp.	0.2-0.3 FW	25
Crab, *Cancer irroratus*		
Flesh	<0.3-0.6 FW	26
Digestive gland	<0.5-1.2 FW	26
Gills	0.8-2.5 FW	26
Annelids		
Polychaete annelids		

Whole	8.1-14.7 DW	27
Whole	23.8-38.0 DW	7
Annelid worm, *Nereis diversicolor*		
Whole	0.6 DW	19
Echinoderms		
U.K., whole, 5 spp.	DW	28
Puerto Rico, whole, 2 spp.	24.2-43.2 FW	3
Greece, whole, 7 spp.	0.5-13.0 DW	29
Sea cucumber, *Holothuria forskalii*		
Muscle	0.3 DW	16
Tunicates		
Tunicates, whole		
Greece, 2 spp.	5.5-6.6 DW;	30
	0.2-1.1 FW	30
Tunicate, *Podoclavella moluccensis*		
Blood		
Plasma, whole	0.4 DW	31
Plasma fractions 9-12	940.0 DW	31
Cell residues	22.0 DW	31
Elasmobranchs		
Smooth dogfish, *Mustelus canis*		
New York Bight		
Muscle	<0.3 FW	32
Liver	<0.8 FW	32
Fish		
Gills, 7 spp.	<0.1-0.6 FW	33
Gonad, 7 spp.	<0.1-0.3 FW	33
Heart, 7 spp.	<0.1-0.8 FW	33
Kidney, 7 spp.	<0.1-0.3 FW	33
Liver, 86 spp.	<0.1-0.4 FW	25,33
Liver, 4 spp.	0.4-2.0 FW	25
Muscle, 196 spp.	<0.1-1.9 FW	25,33,34,35,36
Muscle, 31 spp.	0.2-7.3 DW	24,35,37
Otoliths, 8 spp.	2.5-6.9 DW	38
Scales, 6 spp.	0.6-97.0 DW	38
Skin, 8 spp.	3.1-8.1 DW	24
Spleen, 7 spp.	<0.1-4.8 FW	34
Vertebrae, 8 spp.	<0.1-1.2 FW	34
Viscera	<0.1-4.5 DW	24,27
Whole, 17 spp.	<0.1-0.8 FW	25
Mammals		
Harbor seal, *Phoca groenlandica*		
Kidney	0.2-0.6 FW	39
Heart	0.7-1.2 FW	39
Spleen	0.8-1.4 FW	39
Brain	1.0-2.8 FW	39
Blubber	<0.5 FW	39
FRESHWATER		
Molluscs		
Snails, 8-9 km below electroplating plant discharge		
Soft parts	450.0 DW	40
Amphibians		
Anurans, Laurel, Maryland		
Tadpoles		

Whole, 2 spp.	1.6-3.8 FW	41
Adults		
Whole, 3 spp.	1.8-5.4 FW	41
Frog, *Rana pipiens*		
Whole	0.5 FW	41
Fish		
Alewife, *Alosa pseudoharengus*		
Whole	1.1 FW	42
Pumpkinseed, *Lepomis gibbosus*		
Whole, Laurel, Maryland	5.7 FW	41
Lake trout, *Salvelinus namaycush*		
Lake Cayuga, New York		
Whole		
Ages 1-10 years	<0.013 FW	43
Age 11	0.032 FW	43
Age 12	0.09 FW	43
Eastern mudminnow, *Umbra pygmaea*		
Whole, Laurel, Maryland	0.9 FW	41
Fish		
Muscle, 12 spp.	0.03-1.1 FW	44
TERRESTRIAL		
Plants		
Big sagebrush, *Artemisia tridentata*		
Whole, Idaho		
Distance from phosphate plant		
Downwind 3 km	270.0-400.0 DW	45
Upwind 3 km	77.0-117.0 DW	45
Fescue, *Festuca arundinacea*		
Distance downwind from drift of cooling towers		
15 meters	342.0 DW	46
130 meters	15.0 DW	46
Control areas	0.6 DW	46
Tobacco, *Nicotiana tabacum*		
Kentucky		
Burley leaf	2.5 DW	47
Cigarette leaf	0.3-6.5 DW	47
Pipe leaf	2.8 DW	47
Cigar leaf	3.1-6.2 DW	47
Rye, *Secale cerealis*		
United States		
Seed	0.05 FW	48
Whole	0.04 FW	48
Ontario, Canada, on sludge-amended soil		
Whole	2.2-3.3 DW	49
Corn, *Zea mays*		
Seed	0.25 DW	44
Kernel	0.02 FW	44
Oil	0.47 FW	44
Meal	0.06-0.13 DW	44
Grain	0.1 DW; 3.4 AW	44
Insects		
Termites, Rhodesia, 2 spp.		
Worker	1,500.0 DW	44
Soldier	300.0 DW	44

Queen	20.0 DW	44
Annelids		
Earthworms		
Whole, 2 spp.	5.0-10.2 DW	44
Earthworm, *Eisenia foetida*		
From sewage treatment plant		
sludge containing 299-650 ppm Cr		
Whole less gut		
2 weeks residence	1.0 DW	50
28 weeks residence	13.0 DW	50
Grain fed worms	0.8 DW	50
Feeding on cattle manures	ND	50
Birds		
American black duck, *Anas rubripes*		
Egg	0.6 FW	51
Canvasback, *Aythya valisineria*		
Liver	0.02 FW	52
Lesser black-backed gull, *Larus fuscus*		
Muscle, liver, kidney, and egg	<1.0 DW	4
Osprey, *Pandion haliaetus*		
Liver	□ 0.2 FW	54
Brown pelican, Pelecanus occidentalis		
Liver	<0.2 FW	54
Common eider, *Somateria mollissima*		
Muscle, liver, kidney, and egg	<1.0 DW	4
Waterfowl		
Feathers, 4 spp.	<0.05 DW	55
Mammals		
Pronghorn, *Antilocapra americana*		
Hair		
Idaho	1.9-640.0 DW	44
Wyoming	0.3-130.0 DW	44
Coyote, *Canis latrans*		
Hair	0.7-12.0 DW	44
Elk, *Cervus canadensis*		
Hair	1.9-570.0 DW	44
Bighorn sheep, *Ovis canadensis*		
Hair	<0.1 DW	44
Cotton rat, *Sigmodon hispidus*		
Controls		
Bone	0.2 DW	46,56
Pelt	0.1 DW	46,56
Hair	0.4 DW	46,56
GI tract	1.1 DW	46,56
Whole	0.06 FW; 0.19 DW	46,56
Collected 100-130 m from cooling tower drift		
Bone	0.5 DW	46,56
Pelt	1.1 DW	46,56
Hair	4.4 DW	46,56
GI tract	1.0 DW	46,56
Whole	0.12 FW; 0.40 DW	46,56
Western jumping mouse, *Zapus princeps*		
Hair	23.0-45.0 DW	44

[a]References: 1, Gryzhankova et al. 1973; 2, Riley and Roth 1971; 3, Bernhard and Zattera 1975; 4, Lande

1977; 5, Foster 1976; 6, Bryan and Uysal 1978; 7, Phelps et al. 1975; 8, Ishibashi and Yamamoto 1960; 9, Pak et al. 1977; 10, Giblin et al. 1980; 11, Livingston and Thompson, 1971; 12, Anderlini 1974; 13, Shuster and Pringle 1968; 14, Segar et al. 1971; 15, Bryan et al. 1983; 16, Fukai 1965; 17, Graham 1972; 18, Bertine and Goldberg 1972; 19, Bryan and Hummerstone 1977; 20, Karbe et al. 1977; 21, Young and McDermott 1975; 22, Young et al. 1979; 23, Eisler et al. 1978; 24, Horowitz and Presley 1977; 25, Hall et al. 1978; 26, Greig et al. 1977; 27, Fukai and Broquet 1965; 28, Riley and Segar 1970; 29, Papadopoulu et al. 1976; 30, Papadopoulu and Kanias 1977; 31, Hawkins et al. 1980; 32, Greig and Wenzloff 1977; 33, Brooks and Rumsey 1974; 34, Van As et al. 1973; 35, Plaskett and Potter 1979; 36, De Clerck et al. 1979; 37, Roth and Hornung 1977; 38, Papadopoulu and Kassimati 1977; 39, Duinker et al. 1979; 40, Duval et al. 1980; 41, Hall and Mulhern 1984; 42, Lucas et al. 1970; 43, Tong et al. 1974; 44, Jenkins 1980; 45, Gough and Severson 1976; 46, Taylor and Parr 1978; 47, Nadkarni and Ehmann 1970; 48, Schroeder et al. 1962; 49, Bates et al. 1975; 50, Hartenstein et al. 1980; 51, Haseltine et al. 1980; 52, White et al. 1980; 53, Wiemeyer et al. 1980; 54, Blus et al. 1977; 55, Kelsall 1970; 56, Taylor et al. 1975.

Chromium is beneficial but not essential to growth in higher plants. Residues in plants seldom exceed a few parts per million, except in plants living on infertile serpentine soils containing high Cr concentrations, or grown on soils amended with sewage sludge. Plants with elevated Cr residues show no toxic effects, although concentrations in excess of 1 ppm in the aqueous medium may inhibit germination of the seed and growth of roots and shoots (Towill et al. 1978).

Chromium has proved effective in counteracting the deleterious effects of cadmium in rats and of vanadium in chickens. High mortality rates and testicular atrophy occurred in rats subjected to an intraperitoneal injection of cadmium salts; however, pretreatment with Cr ameliorated these effects (Stacey et al. 1983). In chickens, 10 ppm of dietary Cr counteracted adverse effects on albumin metabolism and egg shell quality induced by 10 ppm of vanadium salts (Jensen and Maurice 1980).

Additional research on the beneficial aspects of Cr in living resources appears warranted, especially where the organism is subjected to complex mixtures containing Cr and other potentially toxic heavy metals.

TOXICITY

GENERAL

Biocidal properties of chromium salts to aquatic organisms are modified, sometimes by an order of magnitude or more, by a variety of biological and abiotic factors. These include the species, age, and developmental stage of the organism; the temperature, pH, salinity, and alkalinity of the medium; interaction effects of Cr with other contaminants; duration of exposure; and chemical form of Cr tested. For hexavalent chromium, LC-50 (96 h) values for sensitive freshwater and marine species were between 445 and 2,000 ppb. For trivalent chromium, LC-50 (96 h) concentrations were 2,000 to 3,200 ppb for sensitive freshwater organisms and 3,300 to 7,500 ppb for marine biota.

Among warm-blooded organisms, hexavalent chromium was fatal to dogs in 3 months at 100 ppm in their food and killed most mammalian experimental animals at injected doses of 1 to 5 mg Cr/kg body weight, but had no measurable effect on chickens at dietary levels of 100 ppm over a 32-day period. Trivalent chromium compounds were generally less toxic than hexavalent chromium compounds, but significant differences may occur in uptake of anionic and cationic Cr+3 species, and this difference may affect survival.

AQUATIC ORGANISMS

Records of acute toxicities of hexavalent and trivalent chromium salts to representative species of aquatic life (Table 4) make it clear that Cr+6 is the more toxic to freshwater biota in comparatively soft and acidic waters, that younger life stages are more sensitive than older organisms, and that 96 h is insufficient to attain stable mortality patterns. There are at least five ionic species of hexavalent Cr, of which two—the hydrochromate ion and the chromate ion—are the predominant species and probably the agents that are toxic to freshwater life (Van der Putte et al. 1981b). However, water pH dramatically affects the concentration of each: as pH decreased from 7.8 to 6.5 the hydrochromate ion increased by a factor of about 3, and the chromate ion decreased by a factor of about 6.8 (Van der Putte et al. 1981b). More research is needed to fully elucidate chromium's mode of action. The organisms most sensitive to Cr+6, as judged by 96-h LC-50 values, were freshwater crustaceans and rotifers, and marine crustaceans, for which LC-50 values were 445 to 3,100 ppb;

longer exposures of 28 to 84 days produced LC-50 values of 200 to 500 ppb (Table 4). Other investigators had confirmed that Cr+6 is more toxic to freshwater daphnids and teleosts in water of comparatively low alkalinity, low pH, and low total hardness (Muller 1980). In marine teleosts, the toxicity of Cr+6 increased at elevated temperatures; furthermore, Cr was additive in toxicity when present as a component in a complex mixture of cadmium, zinc, and Cr+6 salts, (Negelski 1976).

For trivalent chromium and freshwater biota, toxicity was significantly increased in comparatively soft waters; this pattern was especially pronounced for daphnids (Table 4). Among freshwater teleosts, survival was reduced at comparatively low pH (EPA 1980). Also, organisms exposed previously to Cr+3 salts were not unusually sensitive or resistant when subjected to additional Cr+3—suggesting that they were unable to acclimatize or to become sensitized to Cr+3 (Stevens and Chapman 1984). As judged by 96-h LC-50 values, Cr+3 was toxic to sensitive freshwater organisms at concentrations of 2,000-3,000 ppb, or slightly less toxic than Cr+6 (Table 4). Toxicity of Cr+3, like that of Cr+6 increased with increasing exposure in rainbow trout. However, Cr+3 was significantly less toxic than Cr+6 in freshwater to salmon fingerlings, and was dramatically less toxic than Cr+6 to polychaetes and crustaceans (but not to molluscs or teleosts) in saltwater (Table 4).

Table 4. Acute toxicities of hexavalent and trivalent chromium to aquatic life.

Chemical species, ecosystem, taxonomic group, organism, modifiers, and other information	Acute toxicity: Concentration (µg/L)	Percent dead	Duration of test[a]	Reference[b]
Hexavalent Chromium				
Freshwater				
Plants	2,500-25,000	50	96 h	1
Rotifers				
Philodena acuticornis				
Water hardness, in mg $CaCO_3$/L				
25	3,100	50	96 h	2
81	15,000	50	96 h	2
Molluscs				
Snail, *Physa heterostropha*				
Water hardness, in mg $CaCO_3$/L				
45	17,300	50	96 h	3
171	31,600-40,600	50	96 h	3
Crustaceans				
Amphipod, *Gammarus pseudolimnaeus*	67,000	50	96 h	3
Freshwater prawn, *Machrobrachium lamarrei*	1,840	50	96 h	4
Cladoceran, *Daphnia magna*	435	50	24 h	5
Fish				
Mud skipper, *Bolephthalmus dussumieri*	30,500	50	96 h	6
Rainbow trout, *Salmo gairdneri*				
Weight 0.2 g				
Water pH 7.8	12,200	50	96 h	7
Water pH 7.0	7,600	50	96 h	7
Water pH 6.5	3,400	50	96 h	7
Weight 25 g				
Water pH 7.8	65,500	50	96 h	7
Water pH 7.0	45,000	50	96 h	7

Water pH 6.5	20,200	50	96 h	7
Fish				
2 spp.	17,600-118,000	50	96 h	8
3 spp.				
Softwater	<18,000	50	96 h	9
Hardwater	>133,000	50	96 h	9
Salmon fingerlings, *Oncorhynchus* sp.	200	53	12 w	9
Goldfish, *Carssius auratus*	110,000	50	96 h	10
Water hardness, in mg $CaCO_3$/L				
20	37,500	50	96 h	3
220	>90,000	50	96 h	3
Bluegill, *Lepomis macrochirus*				
Water hardness, in mg $CaCO_3$/L				
20	118,000	50	96 h	3
44	113,000	50	96 h	3
45	110,000-170,000	50	96 h	3
120	213,000	50	96 h	3
171	130,000-135,000	50	96 h	3
360	133,000	50	96 h	3
Striped bass, *Morone saxatilis*	30,400	50	96 h	3
Marine				
Molluscs				
3 spp.	14,000-105,000	50	96 h	3
Annelids				
Polychaetes				
Neanthes arenaceodentata	550	50	28 d	11
Neanthes arenaceodentata	200	50	56 d	11
Nereis virens	1,000	50	21 d	11
Capitella capitita	280	50	28 d	11
Capitella capitita	5,000	50	96 h	11
4 spp.	2,000-7,500	50	96 h	3
Echinoderms				
Starfish, *Asterias forbesi*	32,000	50	96 h	3
Crustaceans				
7 spp.	2,000-98,000	50	96 h	3
Copepod, *Tisbe holothuriae*	8,100	50	48 h	12
Copepod, *Acartia clausi*	8,830-19,270	50	48 h	13
Blue crab, *Callinectes sapidus*				
Early life stages	930	50	96 h	14
Early life stages	320	50	40 d	14
Fish				
Small-mouthed hardy head, *Atherinasoma*	36,000	50	96 h	8
microstoma	19,300	0	168 h	8
Yellow-eye mullet, *Aldrichetta*	24,000	50	96 h	8
forsteri	17,900	0	96 h	8
Atlantic silverside, *Menidia menidia*				
Larva	12,400-14,300	50	96 h	3
Juvenile	20,100	50	96 h	3
Mummichog, *Fundulus heteroclitus*	91,000	50	96 h	3

Speckled sanddab, *Citharichthys stigmaeus*	30,000-31,000	50	96 h	3
Trivalent chromium				
Freshwater				
Molluscs				
Snail, *Amnicola* sp.	8,400	50	96 h	3
Annelids				
Worm, *Nais* sp.	9,300	50	96 h	3
Arthropods				
Cladoceran, *Daphnia magna*				
Water hardness, in mg $CaCO_3$/L				
48	2,000	50	96 h	3
52	16,800	50	96 h	3
99	27,400	50	96 h	3
110	26,300	50	96 h	3
195	51,400	50	96 h	3
215	58,700	50	96 h	3
Amphiod, *Gammarus* sp.	3,200	50	96 h	3
Insects, 4 spp.	2,000-64,000	50	96 h	3
Fish				
9 spp.	3,300-71,900	50	96 h	3
3 spp.				
Soft water	< 3,000	50	96 h	9
Hard water	72,000	50	96 h	9
Salmon fingerlings	200	0	12 w	9
Rainbow trout				
Juveniles	4,400	50	96 h	15
Eggs	495	100	30 d	15
Fathead minnow				
Water hardness, mg $CaCO_3$/L				
20	5,070	50	96 h	3
203	7,000-29,000	50	96 h	3
360	67,400	50	96 h	3
Marine				
Molluscs				
American oyster, *Crassostrea virginica*	10,300	50	96 h	3
Annelids				
Polychaete, *Neanthes arenaceodentata*	12,500	0	21 d	11
Crustaceans				
Crab, *Sesarma haematocheir*, zoea	56,000	50	96 h	3
Copepod, *Acartia clausi*	17,000	0	48 h	13
Fish				
Yellow-eye mullet	53,000	50	96 h	8
2 spp.	3,300-7,500	50	96 h	8

[a]Abbreviations: h = hour; d = day; w = week.

[b]Reference: 1, Mangi et al. 1978; 2, Buikema et al. 1974; 3, EPA 1980; 4, Murti et al. 1983; 5, Jouany et al. 1982; 6, Krishnaja and Rege 1982; 7, Van der Putte 1981b; 8, Negelski 1976; 9, Steven et al. 1976; 10, Riva et al. 1981; 11, Reish 1977; 12, Moraitou-Apostolopoulou and Verriopoulos 1982a; 13, Moraitou-Apostolopoulou and Verriopoulos 1982b; 14, Bookhout et al. 1984; 15, Stevens and Chapman 1984.

Maximum acceptable toxicant concentrations (MATC) of chromium to aquatic life were derived from life cycle or partial life cycle exposures, and expressed as the highest concentration tested having no significant adverse effect on the characteristics measured—usually survival, growth, and reproduction—and the lowest concentration at which these effects were observed. For Cr and freshwater teleosts, MATC values ranged from as low as 51 to 105 ppb in rainbow trout to as high as 1,000 to 31,950 ppb in fathead minnows (Table 5). The most sensitive saltwater organism tested was a polychaete worm with a MATC range of 17 to 38 ppb (Table 5). For Cr+3 the MATC range for freshwater organisms was 47 to 1,400 ppb, which was quite similar to that for Cr+6 for freshwater life. No MATC data were available for Cr+3 and marine biota.

TERRESTRIAL INVERTEBRATES

Data on toxicity of Cr to terrestrial invertebrates are sparse. Studies conducted in India showed that a concentration of 10 to 15 ppm of Cr+6 in irrigation water, when applied to soils for agricultural purposes, was lethal to two species of earthworms in 58 to 60 days (Soni and Abbasi 1981; Abbasi and Soni 1983).

Table 5. Maximum acceptable toxicant concentration (MATC) values for hexavalent and trivalent chromium to aquatic life based on life cycle or partial life cycle exposures.

Chemical species, ecosystem, organism	MATC (µg/L, ppb)	Reference[a]
Hexavalent chromium		
Freshwater		
Rainbow trout, *Salmo gairdneri*		
Water hardness, mg $CaCO_3$/L		
34	51-105	1
45	200-350	2
Brook trout, *Salvelinus fontinalis*	200-350	2
Fathead minnow, *Pimephales promelas*	1,000-3,950	3
Lake trout, *Salvelinus namaycush*	105-194	1
Channel catfish, *Ictalurus punctatus*	150-305	1
Bluegill, *Lepomis macrochirus*	522-1,122	1
White sucker, *Catostomus commersoni*	290-538	1
Northern pike, *Esox lucius*	538-963	1
Walleye, *Stizostedion vitreum*	>2,161	1
Saltwater		
Polychaete worm, *Neanthes arenaceodentata*	17-38	4
Mysid shrimp, *Mysidopsis bahia*	88-198	2
Trivalent chromium		
Freshwater		
Cladoceran, *Daphnia magna*	47-93	2
Fathead minnow	750-1,400	2
Rainbow trout	30-157	5

[a]References; 1, Sauter et al. 1976; 2, EPA 1980; 3, Pickering 1980; 4, Reish 1977; 5, Stevens and Chapman 1984.

MAMMALS AND BIRDS

Acute and chronic adverse effects of chromium to warm-blooded organisms are caused mainly by Cr+6 compounds; there is little conclusive evidence of toxic effects caused by Cr+2 or Cr+3 compounds (Langard and Norseth 1979). Most investigators agree that chromium in biological materials is probably always in the trivalent state, that greatest exposures of Cr+3 in the general human population are through the diet (but no adverse effects have been reported from such exposures), and that no organic trivalent chromium complexes of toxicological importance have been described. Studies with guinea pigs fed Cr+3 for 21 weeks at concentrations up to 50 ppm dietary Cr+3 showed no adverse effects (Preston et al. 1976). Domestic cats were apparently

unaffected after exposure to aerosol levels of 80 to 115 mg Cr+3/m^3 for 1 h daily for 4 months, or after consuming diets with high amounts of chromic (Cr+3) salts over a similar period (Langard and Norseth 1979). When chromium was administered by injection, trivalent salts were substantially less toxic than hexavalent salts in producing effects in embryos of golden hamsters (Gale 1978). A similar pattern was evident in mice and in embryos of chickens. The LD-50's for mice were 260 mg/kg body weight for Cr+3, but only 5 mg/kg body weight for Cr+6 (Steven et al. 1976). For chicken embryos, the LD-50 values (mg/kg body weight) were 22.9 for Cr+3 and 1.7 for Cr+6 (Ridgeway and Karnofsky 1952). However, survival was depressed in young American black ducks fed 10 or 50 ppm dietary Cr+3 for 10 weeks—suggesting that black duck broods along the Atlantic coast (an area of high anthropogenic chromium discharge) may be adversely affected if they remain in contaminated areas for extended periods (Haseltine et al. 1985). In the black duck study, the authors administered chromium in the form of $CrK(SO_4)_2$ 12 H_2O; absorption of this anionic trivalent chromium compound (and an anionic hexavalent Cr compound) through black duck intestine was superior to that of cationic forms (Eastin et al. 1980).

Steven et al. (1976), in studies with Cr+6 and dogs, showed that 100 ppm in food for 3 months was fatal, that 11.2 ppm in drinking water was not lethal over a 4-year period (although significant accumulation was observed), and that 6.0 ppm in drinking water for 4 years had no measurable effects. In rats, 1,000 ppm dietary Cr+6 represented the toxic threshold, but all animals survived 134 ppm Cr+6 in drinking water for 3 months (Steven et al. 1976). For most mammalian experimental animals, including mice, dogs, rabbits, cats, and guinea pigs, the minimum injected fatal dose of Cr+6 ranged from 1 to 5 mg/kg body weight—although doses of 0.2 to 0.5 mg/kg body weight produced marked kidney damage (Steven et al. 1976). Repeated sublethal injections of Cr+6 did not promote tolerance in mice, but rather decreased the minimum lethal dose, suggesting that the animals were unable to develop tolerance to repeated chromium exposures (Steven et al. 1976). Investigators have not yet been able to identify a specific hexavalent Cr compound, or group of compounds, that could account for the most pronounced biological activity (Langard and Norseth 1979). A lethal oral dose of Cr+6 for a 14-year-old boy was estimated to be 10 mg/kg body weight—much lower than that tolerated by test animals on a repeated basis over a period of several months (Steven et al. 1976). Domestic chickens appear to be more resistant than mammals. No adverse effects were observed in chickens exposed to 100 ppm dietary Cr+6 in a 32-day study (Bosomer et al. 1961), although embryolethal and teratogenic effects have been observed in the range of 0.2 mg/kg (Gilani and Marano 1979) to 1.7-22.9 mg/kg (Ridgeway and Karnofsky 1952), depending on the method of administration.

SUBLETHAL EFFECTS

GENERAL

Under laboratory conditions, chromium is mutagenic, carcinogenic, and teratogenic to a wide variety of organisms, and Cr+6 has the greatest biological activity. However, information is lacking on the biological activities of water soluble Cr+3 compounds, organochromium compounds, and their ionic states. Aquatic plants and marine polychaete worms appear to be the most sensitive groups tested. In exposures to Cr+6, growth of algae was inhibited at 10.0 ppb, and reproduction of worms at 12.5 ppb. At higher concentrations, Cr+6 is associated with abnormal enzyme activities, altered blood chemistry, lowered resistance to pathogenic organisms, behavioral modifications, disrupted feeding, histopathology, osmoregulatory upset, alterations in population structure and species diversity indices, and inhibition of photosynthesis. Not all sublethal effects observed were permanent, but the potential for acclimatization of organisms to Cr is not well documented. The great variability among species and tissues in the accumulation or concentration of Cr is attributed partly to the route of administration, partly to the concentration of Cr and its chemical species, and partly to numerous biotic and physicochemical modifiers. High accumulations of Cr have been recorded among organisms from the lower trophic levels, but there is little evidence of biomagnification through food chains. Marine bivalve molluscs, for example, accumulated measurable concentrations at ambient water concentrations of 5.0 ppb of Cr+6, but the significance of Cr residues in molluscs and other organisms is not well understood. Depuration of accumulated Cr among organisms differs markedly, but usually follows a complex multicompartmental excretion pattern.

AQUATIC ORGANISMS: FRESHWATER

BACTERIA

The role of sewage bacteria in Cr kinetics and cycling is unresolved and promises to be a fruitful field of research. Of 362 bacterial isolates from Cr+6 liquid sanitary sewage and chemical waste sludges, only 1—an

isolate of *Arthrobacter* sp.—could tolerate 400 ppm of Cr+6 (Coleman and Paran 1983); however, this isolate could not effectively accumulate Cr at low ambient levels of 5 ppm of Cr+6, whereas *Agrobacter* sp., another isolate, could. Hexavalent Cr in a wide array of forms showed dose dependent responses for mutagenic activity in the bacterium *Salmonella typhimurium* (Del Carratore et al. 1984); moreover, among 56 metal compounds tested, Cr+6 elicited the strongest mutagenic responses in *Bacillus subtilis* (Hatherill 1981). In some tests, Cr+3 was genetically active, but only when present as a stable organic complex (Del Carratore et al. 1984).

ALGAE AND MACROPHYTES

Growth of freshwater algae was reduced at Cr+6 concentrations of 10 ppb for *Chlamydomonas reinhardi* and >45 ppb for other species tested; effects were most pronounced in water of low alkalinity (EPA 1980). Frond growth of the common duckweed, *Lemna minor*, the most sensitive aquatic plant tested, was reduced at 10 ppb Cr+6 in days (Mangi et al. 1978). Jouany et al. (1982) reported that a green alga, *Chlorella vulgaris*, biomagnified Cr+6 from the medium about 1000X in 28 days at ambient concentrations of 300 ppb; growth was inhibited at 445 ppb Cr+6 in 96 hours, and adenosine triphosphate (ATP) production was reduced at 470 ppb in 24 hours. At 10 ppb Cr+6 in the medium, bioconcentration factors for the chlorophytes *Hydrodictyon reticulatum* and *Oedogonium* sp. ranged from 200 to 600X in 14 days (Mangi et al. 1978). Accumulation of Cr by living and dead plant tissue is extensive, uptake linearly approximating concentration on a logarithmic basis (Mangi et al. 1978).

Trivalent chromium is far less effective than Cr+6 in producing root weight inhibition in Eurasian watermilfoil, *Myriophyllum spicatum*: 9,900 ppb Cr+3 vs. 1,900 ppb Cr+6 (EPA 1980).

INVERTEBRATES

Hexavalent chromium was associated with adverse effects in invertebrates of widely separated taxa: reduced survival and fecundity of the cladoceran *Daphnia magna* at a concentration of 10 ppb and exposure for 32 days (EPA 1980); growth inhibition of the protozoan *Chilomonas paramecium* at 1,100-3,000 ppb at temperatures of 10-30 °C during exposures of 19-163 h (Honig et al. 1980); abnormal movement patterns of larvae of the midge *Chironomus tentans* at 100 ppb in 48 h (Catalan 1982); and a temporary decrease in hemolymph glucose levels in the freshwater prawn *Macrobrachium lamarrei* surviving 1,840 ppb Cr+6 for 96 h (Murti et al. 1983).

Trivalent Cr was less effective than Cr+6 in reducing fecundity of *Daphnia magna*: 44 ppb Cr+3 vs. 10 ppb Cr+6 (EPA 1980). Annelid worms (*Tubifex* sp.) accumulated about 1 ppm Cr during exposure for 2 weeks in sediments containing 175 ppm Cr+3, suggesting that benthic invertebrates have only a limited ability to accumulate chromium from sediments or clays (Neff et al. 1978).

FISH

Among sensitive species of freshwater teleosts, Cr+6 concentrations of 16 to 21 ppb in the medium resulted in reduced growth of rainbow trout and chinook salmon fingerlings during exposure of 14 to 16 weeks, and altered plasma cortisol metabolism in rainbow trout after 7 days; locomotor activity in bluegills increased after 2 weeks in 50 ppb Cr+6 (EPA 1980). Long-term exposure of rainbow trout by Calamari et al. (1982) for 180 days to high, but environmentally realistic, concentrations of 0.2 ppm Cr+6 resulted in elevated levels of Cr in kidney (3.5 mg/kg fresh weight), liver (2.0), and muscle (0.6); after 90 days in Cr-free media, Cr levels were 1.6, 1.3, and 0.5, respectively. Time required to reach median asymptotic uptake ranged from 36 to 55 days for various tissues; extrapolated values for almost complete equilibrium were 237 to 365 days (Calamari et al. 1982). The rudd (*Scardinus erythrophthalmus*), exposed to Cr+6 for 24 hours, did not accumulate detectable levels of Cr in tissues during exposure to 16 ppm, but did during exposures to 20 ppm; the kidney contained the highest residues—10.3 mg Cr/kg fresh weight (Van Hoof and Van San 1981).

At high environmental concentrations of Cr+6 (i.e., 2.0 ppm in water) and at alkaline pH, concentrations in rainbow trout tissues were greatest in gill, liver, kidney, and digestive tract; after transfer of the fish to Cr-free media, residues tended to remain high in kidney and liver; concentration in gill tissues tended to be greater at pH 7.8 than at pH 6.5 (Van der Putte et al. 1981a). Studies with perfused gills showed that the transfer of Cr was directly coupled with the transfer of oxygen from the external solution to the internal perfusion medium and that this transfer was significantly more rapid at pH 6.5 than at alkaline pH (Van der Putte and Part 1982). Uptake rate of Cr+6 was rapid, equilibrium usually being reached in 2 to 4 days of exposure for various tissues,

except for gill, which continued to accumulate Cr with increasing exposure at acidic pH. In rainbow trout, the excretion pattern was biphasic. The biological half-life of the short-lived component (34% of the total Cr) was 1.0 day, and that of the long-lived component was 25.6 days (Van der Putte et al. 1981a). Other sublethal effects were observed in freshwater teleosts following Cr+6 insult. In the snakehead (*Channa punctatus*), enzyme activities were altered in a wide variety of organs and tissues after exposure for 30 days to 2.6 ppm (Sastry and Sunita 1984); the effects became life threatening after exposure for 120 days (Sastry and Tyagi 1982; Sastry and Sunita 1982,1983). In the mud skipper (*Boleophthalmus dussumieri*), chromosomal aberrations in the gill increased after injection of 1.0 mg/kg body weight, or exposure to 24 ppm in the medium for 24 h (Krishnaja and Rege 1982). In juvenile coho salmon (*Oncorhynchus kisutch*), disease resistance and serum agglutinin production both decreased after 2 weeks in water containing 0.5 ppm (Sugatt 1980b). In seaward migrating coho salmon, salinity tolerance and serum osmolality were impaired during exposure to 0.23 ppm Cr+6 for 4 weeks (Sugatt 1980a).

Chromium uptakes and effects in teleosts were modified significantly by many biological and abiotic variables, including water temperature and pH, the presence of other contaminants or compounds, and sex and tissue specificity. In rainbow trout, only males showed significant changes in liver enzyme activity during exposure to 0.2 ppm Cr+6 for 6 months; the effects were intensified by the presence of nickel and cadmium salts in solution (Arillo et al. 1982). Rainbow trout are able to regulate Cr somewhat, either actively, by reduced absorption or increased excretion, or passively, by the limitation of binding sites for Cr *in vivo* (Buhler et al. 1977). Tests with goldfish and high Cr+6 concentrations indicated that toxic and sublethal effects were more pronounced at comparatively high water temperatures and reduced pH; further, Cr residue levels were abnormally high in dead or moribund fish, suggesting that residue values from dead or dying fish should be interpreted with extreme caution (Riva et al. 1981). In rainbow trout, acute Cr poisoning caused morphological changes in gills, kidney, and stomach tissues at pH 7.8, but only in the gills at pH 6.5 (Van der Putte et al. 1981b). Chromium uptake in trout increased when 10 ppb of ionic cadmium was present in solution (Calamari et al. 1982)—again demonstrating that uptake patterns are not necessarily predictable for single components in complex mixtures.

AQUATIC ORGANISMS: MARINE

ALGAE AND MACROPHYTES

Algae and higher plants accumulated chromium from seawater by factors up to 8,600 (Van As et al. 1973), and from solutions containing 50 ppm Cr by a factor of 18 in 48 h (Sivalingam 1978). Algae also accumulated Cr from sewage sludge, showing increases in Cr of 25 to 60 mg/kg dry weight (Montgomery et al. 1978). The unusually high Cr concentrations observed in some species of algae and macrophytes from Narragansett Bay, Rhode Island (Phelps et al. 1975) and from Puerto Rico (Bernhard and Zattera 1975) almost certainly came from chromium wastes discharged from electroplaters (in Narragansett Bay) and from other anthropogenic sources (in Puerto Rico). A similar situation probably exists wherever grossly elevated Cr levels are observed.

Although chromium is abundant in primary producers, there is little evidence of biomagnification through marine food chains consisting of herbivores and carnivores (Osterberg et al. 1964). Baptist and Lewis (1969) followed the transfer of assimilated and unassimilated radiochromium through an experimental food chain that included phytoplankton, brine shrimp, postlarval fish, and adult fish. When chromium was successively transferred through each of the four trophic levels, concentrations declined after each transfer. Comparisons of the results from the food chain with laboratory studies on chromium uptake from seawater suggest that the food chain, despite the successive declines, was generally the more efficient pathway for uptake of chromium by all trophic levels.

Among sensitive species of marine algae, concentrations of 10 ppb of Cr+6 partly inhibited growth of *Olisthodiscus lutens*. All cultures, including those in which growth was inhibited, contained viable, active (75%) cells at the end of 10 days. Inhibitory effects were reversed by chelators such as EDTA (Mahoney 1982), suggesting that naturally occurring ligands and sequestering agents in seawater may alleviate the toxicity of Cr+6, and perhaps other metals. In the giant kelp (*Macrocystis pyrifera*), photosynthesis was inhibited 20% in 5 days at 1,000 ppb of Cr+6, and 50% in 4 days at 5,000 ppb (EPA 1980); this kelp appears to be one of the more resistant aquatic plants.

MOLLUSCS

Edible tissues of commercially important North American molluscs contained 0.1 to 0.6 mg Cr/kg fresh weight (Hall et al. 1978). Although this concentration is in general agreement with molluscan data from other geographic areas (Eisler 1981), Shuster and Pringle (1968) reported values (mg Cr/kg fresh weight) in edible portions as high as 3.4 in oysters (*Crassostrea virginica*), 5.8 in hardshell clams (*Mercenaria mercenaria*), and 5.0 in softshell clams (*Mya arenaria*). The ability of marine molluscs to accumulate Cr far in excess of that in ambient seawater was documented by Papadopoulu (1973), who found that the Cr concentration in 5 species of bivalves from Greek waters exceeded that in seawater by 16,000 times (*Pinna nobilis*) to 260,000 times (*Astralium rogosum*). No deleterious health effects have been reported among consumers of molluscs that contained occasional high Cr residues. Anthropogenic and natural chromium gradients in sediments or the water column were reflected in the wide range of values reported for this element in field collections of clams (Phelps et al. 1975; Eisler et al. 1978) and mussels (Alexander and Young 1976; Fowler and Oregioni 1976; Lande 1977; Karbe et al. 1977).

Two factors known to modify Cr accumulations in molluscs are the weight of the organism and the salinity of the medium. Concentrations of Cr in clams were reported to decrease with increasing body weight (Eisler et al. 1978) and increasing salinity (Olson and Harrel 1973). Accumulation of Cr by oysters (*Crassostrea gigas*) was independent of sediment Cr levels and dependent on organism size—suggesting some homeostatic regulation of this metal (Ayling 1974). In a 20-week laboratory study of chromium accumulation rates by oysters (Shuster and Pringle (1969) continuously subjected the animals to seawater solutions containing 50 or 100 ppb Cr+6. After 5, 10, or 20 weeks in 50 ppb, maximum whole body concentrations (mg Cr/kg fresh weight) were 2.4, 3.7, and 6.3, respectively (up from control values of <0.12); in 100 ppb, the values were 4.4 (5 weeks), 6.4 (10 weeks), and 11.5 (20 weeks). Preston (1971) concluded that *C. virginica*, under laboratory conditions, accumulated Cr more readily by direct absorption from the medium than from ingestion of radiochromium-labeled algae (*Chlamydomonas* spp.). In natural environments, however, Cr concentration is likely to be greater in the food supply than in the water. As a consequence, food might be the primary source of Cr to oysters, even though accumulation occurs more readily by direct absorption (Preston 1971).

Clams, oysters, and mussels accumulate Cr from the medium or from contaminated sediments at comparatively low concentrations. For example, oysters subjected to 5.0 ppb of Cr+6 for 12 weeks contained 3.1 mg Cr/kg dry weight in soft parts and retained 52% of the accumulated Cr after they were transferred to Cr-free seawater for 28 weeks (Zaroogian and Johnson 1983). Mussels (*Mytilus edulis*) subjected to the same dose-time regimen contained 4.8 mg/kg, but retained only 39% after 28 weeks of depuration. Both oysters and mussels contained higher residues after exposure to 10.0 ppb Cr+6 for 12 weeks—5.6 and 9.4 mg Cr/kg dry weight in soft parts, respectively—and both contained substantial (30-58%) residues after 28 weeks in a Cr-free environment (Zaroogian and Johnson 1983). In studies with mussels and softshell clams (*Mya arenaria*), Capuzzo and Sasner (1977) demonstrated that Cr in New Hampshire sediments (contaminated with Cr+3 from tannery wastes) was bioavailable to clams by diffusion from seawater, and that both diffusion and particulate uptake were important pathways for mussels. Accumulation was observed at sediment Cr concentrations as low as 150 ppm. Kaolinite sediments containing up to 1,200 ppm of Cr+3 produced the most pronounced adverse effects on filtration rates and ciliary activity of bivalve molluscs, leading the authors to conclude that Cr that has accumulated in areas affected by industrial wastes might have serious consequences to filter feeding bivalves.

It is emphasized that Cr+3, probably because of its very low solubility in seawater, appears to have a much lower bioavailability to most groups of marine animals than Cr+6, which is more water soluble (Carr et al. 1982). The clam *Rangia cuneata* appears to be an exception: it accumulated up to 19 mg Cr/kg in soft parts, on a dry weight basis, during exposure for 16 days to chromium-contaminated muds, and retained most of it for an extended period; the estimated biological half-time was 11 days (Carr et al. 1982). In general, benthic invertebrates rarely accumulate Cr from contaminated sediments (82-188 ppm Cr+3); only a few examples have been recorded (Neff et al. 1978).

NEMATODES

Representatives of this phylum have been used extensively as indicators of stressed environments. Population structure and species diversity of free-living nematodes inhabiting sediments in the New York Bight were moderately influenced by the heavy metal content of sands. In medium-grained sands, species diversity was inversely correlated with increased concentrations of Cr and other metals. Sands containing 3.0 to 21.5 mg

Cr/kg were also marked by high relative abundances of one or two nematode species; the tolerance of these species to Cr stress probably exceeded that of the normal nematode inhabitants of such sediments (Tietjen 1980).

CRUSTACEANS

In general, chromium seldom exceeds 0.3 mg/kg fresh weight in edible crustacean tissues (Eisler 1981). The highest value (0.6 mg Cr/kg fresh weight) reported in muscle of rock crab (*Cancer irroratus*) was from specimens collected near an ocean dump site receiving large quantities of metals. Digestive glands and gills from these crabs also contained the highest Cr residues for these tissues in crustaceans (Greig et al. 1977).

Sather (1967) observed that uptake and loss of radiochromium by the crab *Podophthalmus vigil* was independent of sex and eyestalk hormone influences. Most of the radiochromium accumulated in gills. Equilibrium was reached in gill and muscle in 2-3 days, but in midgut and hemolymph in 4-5 days. Iron interfered with chromium uptake and retention. Tennant and Forster (1969) demonstrated that Cr concentrated in setae, gills, and hepatopancreas of Dungeness crab (*Cancer magister*) and suggested that surface adsorption and physiological processes were both instrumental in Cr accumulation. Barnacles (*Balanus* sp.) incorporated Cr+6 in soft tissues up to 1,000X over ambient concentrations, reaching equilibrium in 7 days (biological half-life for some components was 120 days); however, Cr+3, which precipitates in seawater, was quickly removed by filtering activity, was not concentrated in soft tissues, and was rapidly excreted by way of the digestive system (^{51}Cr (VI) and ^{51}Cr (III) by barnacles (van Weerelt et al. 1984).

Sediment Cr concentrations of 3,200 ppm in the New Bedford (Massachusetts) Acushnet estuary, and 100 ppm in the New York Bight have been recorded (Doughtie et al. 1983). Massive cuticular lesions suggestive of shell disease characterized up to 30% of the lobsters, crabs, and shrimp collected from the New York Bight, and these lesions could also be induced in crustaceans exposed to New York Bight sediments in the laboratory. This shell disease syndrome has been induced in 41% of grass shrimp (*Palaemonetes pugio*) during exposure to 0.5 ppm Cr+6 for 28 days (Doughtie et al. 1983). It is proposed that Cr interferes with the normal functions of subcuticular epithelium, particularly cuticle formation, and subsequently causes structural weaknesses or perforations to develop in the cuticle of newly molted shrimp. Because of these Cr-induced exoskeletal deficiencies, a viaduct for pathogenic bacteria and direct Cr influx is formed that perpetuates the development of the lesion.

Of the 65,000 tons of Cr compounds used annually in exploratory oil drilling, a significant portion enters the marine environment through the discharge of used drilling muds. It has been estimated that more than 225 tons of drilling mud may be used in a single 3,000-m well (Carr et al. 1982). One of the most frequently used muds in offshore drilling operations is a chrome lignosulphonate mud containing barium sulphonate, bentonite clay, and ferrochrome or chrome lignosulphonates (Carr et al. 1982). The bioavailability of Cr to grass shrimp from used chrome lignosulphonate drilling muds is most pronounced at the mud aqueous layers. At Cr concentrations of 248 ppb in the mud aqueous fraction, grass shrimp accumulated 23.7 mg Cr/kg dry weight whole body after 7 days (Carr et al. 1982). Concentrations of drilling mud of 1% or greater in seawater were toxic to sensitive species of crustaceans (Neff et al. 1981); uptake of 4 to 5 mg/kg was reported in grass shrimp exposed to sediments containing 188 ppm Cr (Neff et al. 1978). The toxicity of Cr-contaminated drilling muds to grass shrimp may sometimes be attributable to large residuals of petroleum hydrocarbons in the sediments (Conklin et al. 1983).

ANNELIDS

Uptake and excretion studies of Cr+3 by *Hermione hystrix* (Chipman 1967) showed that Cr+3 was not readily accumulated from seawater, owing to the formation of particles and surface adsorption phenomena; furthermore, little accumulation was evident on contact with contaminated sediments. Hexavalent chromium in the medium was readily accumulated by *Hermione*; the process was slow and only small amounts were taken up in 19 days—i.e., 0.03 to 0.10 mg/kg fresh weight from media containing 3 to 10 ppb Cr+6. Higher body burdens of 0.5 to 1.8 mg Cr/kg fresh body weight were reported at 100 to 500 ppb of Cr+6, but some deaths were noted at these concentrations. Chromium accumulation by *Hermione* is a passive process and directly related to Cr+6 concentration in the medium. At least two rates of biological loss are involved—one of 8 days and another of 123 days. Chipman (1967) concluded that most of the Cr accumulated by *Hermione* from long exposure is bound in a body component having a slow turnover rate and an estimated biological half-life of

about 123 days.

Uptake of Cr+6 from seawater has been reported for *Neanthes arenaceodentata*. Whole *Neanthes* contained 30.0 mg Cr/kg dry weight after exposure for 150 days in ppb of Cr+6 (Mearns and Young 1977) and 0.5 to 1.6 mg Cr/kg fresh weight after exposure for 440 days (Oshida et al. 1976); both of these observations were similar to those of Chipman (1967), after adjustment for wet and dry weights. Concentrations as low as 12.5 ppb of Cr+6 decreased brood size in *Neanthes* (Mearns et al. 1976; Oshida et al. 1976), although no significant body residues were evident. Uptake of Cr+6 by *Neanthes* was related to dose at low ambient Cr concentrations. Worms subjected to 2.6, 4.5, 9.8, or 16.6 ppb Cr+6 for 309 days contained 0.5, 0.7, 2.2, and 2.5 mg Cr/kg whole fresh organism, respectively (Oshida and Word 1982). There was no direct relationship between tissue concentration and brood size, suggesting that Cr in *Neanthes* attaches to proteins in the body wall, gut, and parapodial regions (Oshida and Word 1982).

Neanthes arenaceodentata is the most sensitive marine organism yet tested. In worms exposed to sublethal concentrations of Cr+6, feeding was disrupted after 14 days at 79 ppb (EPA 1980), reproduction ceased after 440 days (three generations) at 100 ppb (Oshida et al. 1981), brood size was reduced after 309 to 440 days at 12.5 to 16.0 ppb (Oshida et al. 1981; Oshida and Word 1982), and abnormalities in larval development increased after 5 months at 25 ppb (Reish 1977). On the other hand, exposure for 293 days (two generations) in 50,400 ppb Cr+3 caused no adverse effects on survival, maturation time required for spawning, or brood size (Oshida et al. 1981). The polychaete *Capitella capitata* was more resistant than *Neanthes*; a decrease in brood size was noted only after exposure for 5 months to 50 and 100 ppb Cr+6 (EPA 1980).

ECHINODERMS

With the exception of two sea urchin samples collected from Puerto Rico, most Cr residues reported in echinoderms have been less than 1.0 mg/kg dry weight (Eisler 1981). The exceptions—elevated levels of 24 and 43 mg/kg fresh weight of whole organism in Puerto Rican sea urchins—were not reflected in sea cucumber muscle from the same vicinity (Fukai 1965), and thus should be viewed with caution. Echinoderms from the United Kingdom and environs were comparatively low in chromium; concentrations were less than 0.46 mg Cr/kg dry weight whole organism (Riley and Segar 1970). Embryos of a sea urchin (*Anthocidaris* sp.) developed normally in solutions containing 3.2 to 4.2 mg Cr/L, but failed to develop at 8.4-10.0 mg Cr/L (Okubo and Okubo 1962; Kobayashi 1971). Larvae of another species of sea urchin (*Hemicentrotus* sp.) were more sensitive, showing abnormal development or dying within 24 h at concentrations of less than 1.0 mg Cr/L (Okubo and Okubo 1962). Hexavalent chromium at 6.0 mg/L was associated with abnormal development in embryos of *Anthocidaris crassispina* (Kobayashi 1977).

FISH

Individual tissues of most species of finfishes contained between 0.1 and 0.6 mg Cr/kg fresh weight (Hall et al. 1978). For still unexplained reasons, chromium concentrated in the scales of some species collected in Greek waters, values ranging up to 97.0 mg Cr/kg dry weight (Papadopoulu and Kassimati 1977). Chromium concentrations also vary significantly among different species of fish collected from the same geographic area. For example, muscle Cr concentration was 1,430X greater in a porgy (*Pachymetopan qrande*) than in a goosefish (*Lophius piscatorius*) from the same collection (Van As et al. 1973).

Accumulation of chromium under controlled conditions has been documented for speckled sanddab (Mearns and Young 1977) and Atlantic croaker (Baptist et al. 1970). Sanddabs held in seawater solutions containing 3 to 5 ppm Cr+6 contained up to 100 mg Cr/kg intestine (dry weight), 10 in liver, and 3 in muscle (Mearns and Young 1977). Sanddabs accumulated significant concentrations of Cr in various tissues during long-term exposure in seawater concentrations as low as 16 ppb Cr+6 (Mearns and Young 1977). Baptist et al. (1970), who studied the retention of radiochromium-51 in croakers following a single intraperitoneal injection, wrote that retention was expressed as two exponential rate functions: 70 days for the long-lived component and 20 days for the short-lived component.

BIRDS

Male domestic chickens fed diets containing up to 100 ppm of Cr+6 for 32 days showed no adverse effects in survival, growth, or food utilization efficiency (Rosomer et al. 1961). However, teratogenic effects were documented in chicken embryos after eggs had been injected with Cr+6. Deformities included short and twisted

limbs, microphthalmia, exencephaly, everted viscera, growth stunting, and parrot beaks (Ridgeway and Karnofsky 1952; Gilani and Marano 1979). The highest incidence of teratogenic effects was observed at Cr+6 concentrations that caused some deaths, and when the administration route was through the chorioallantoic membrane as opposed to the yolk; no teratogenic effects were observed with Cr+3 salts (Ridgeway and Karnofsky 1952).

Young American black ducks (*Anas rubripes*) absorbed anionic Cr species more readily than cationic forms from the intestines, strongly indicating that ionic Cr state should be considered when avian dietary toxicity studies are being planned (Eastin et al. 1980). Adult black ducks fed diets containing 10 or 50 ppm anionic Cr+3, as Cr $K(SO_4)_2.12\ H_2O$, for 5 months were normal in survival, reproduction, and blood chemistry. However, in ducklings from treated groups that were fed Cr-contaminated diets at original parental dosages, growth patterns were altered and survival was reduced (Haseltine et al. 1985). In another study with black ducks, adults were fed diets containing 0, 20, or 100 ppm anionic Cr+3 and ducklings from these pairs were fed the same diets for 7 days; tests of avoidance responses of the ducklings to a fright stimulus showed that the Cr had no significant effect on their behavior (Heinz and Haseltine 1981).

MAMMALS

Chromium is causally associated with mutations and malignancy (Leonard and Lawerys 1980; Norseth 1981). Under appropriate conditions, Cr is a human and animal carcinogenic agent; its biological effects depend on chemical form, solubility, and valence. In general, Cr+6 compounds are hazardous to animals, whereas metallic Cr and Cr+3 are essentially nontoxic (Gale 1978); however, exposure to water solubilized Cr+3 has caused cancers and dermatitis in workers, and toxicity in rabbits (Hatherill 1981). In the chromate producing industry workers who developed respiratory cancer had been exposed to 30 to 1,100 ug/m^3 Cr in air for periods of 4 to 24 years, and workers producing chromate pigment who developed respiratory cancer had been subjected to an estimated Cr+6 exposure of 500 to 1,500 ug/m^3 for 6 to 9 years. Carcinogens released in the chromate manufacturing process have not yet been identified (Post and Campbell 1980). Levels as low as 10 ug/m^3 of Cr+6 in air produced strong irritation in nasal membranes, even after short exposures. In some persons whose lower respiratory tissues became Cr-sensitized, asthmatic attacks occurred at levels of Cr+6 as low as 2.5 ug/m^3 (Steven et al. 1976). There is no evidence of Cr sensitization in mammals other than humans. In the only animal study demonstrating a carcinogenic effect of an inhaled chromate, adenocarcinomas were reported in the bronchial tree of mice exposed throughout life to $CaCrO_4$ dust at 13 mg/m^3 (4,330 ug Cr+6/m^3) for 35 h weekly (Langard and Norseth 1979). Trivalent Cr compounds did not produce respiratory cancers (Steven et al. 1976). In rabbits, both Cr+3 and Cr+6, given 1.7 mg/kg body weight daily for 6 weeks, adversely affected blood and serum chemistry, and both produced significant morphological changes in liver (Tandon et al. 1978); similar results were observed in rats (Laj et al. 1984). Although damage effects and residue accumulations were greater in rabbits treated with Cr+6, water soluble Cr+3 compounds also may have significant biological activity (Tandon et al. 1978).

Hexavalent Cr compounds may cause skin ulceration, irritative dermatitis, ulcerations in mucous membranes, and perforations of the nasal septum. That inhalation of Cr+6 compounds may cause bronchial carcinomas has been well documented in humans (Langard and Norseth 1979). Skin lesions or ulcers were produced in guinea pigs when solutions containing 30,000 ppm of Cr+6 were applied to abraded skin or if the natural oils were removed from the skin beforehand; Cr+3 in concentrations as high as 100,000 ppm had no ulcerogenic effects (Steven et al. 1976). Allergic guinea pigs developed dermatitis when exposed to solutions of either Cr+6 or Cr+3 at concentrations as low as 10 ppb (Steven et al. 1976). In nonallergic animals, these effects were observed after repeated exposures to solutions containing 1,000 to 3,000 ppm of Cr+3 or Cr+6 salts. Local sarcomas in muscle and local carcinomas of the skin have also been demonstrated in small laboratory animals exposed to Cr+6 (Langard and Norseth 1979). Kidney and liver lesions in rats were observed when the drinking water contained 134 ppm of Cr+6 for 2 to 3 months (Steven et al. 1976).

Hexavalent Cr has established its mutagenic activity in a wide array of screening tests, whereas insoluble Cr+3 forms appear to be inactive, in analogous evaluations—perhaps because Cr+3 absorption is poor in the systems analyzed (Hatherill 1981). Studies with tissue cultures of ovary cells of the Chinese hamster showed that the addition of 52 ppb of Cr+6 not only induced sister chromatid exchanges but also inhibited cell

proliferation; there was no measurable effect at 0.52 ppb Cr+6. Trivalent Cr at 520 ppb did not measurably affect cell proliferation or chromatid exchanges (Uyeki and Nishio 1983). Genotoxic effects of Cr+6 are reversed by the addition of reducing agents or ascorbic acid (Hatherill 1981; Uyeki and Nishio 1983). Chromosomal rearrangements and aberrations were recorded in rabbit cells after exposure to Cr+6 (Hatherill 1981). Teratogenic effects induced by intravenous administration of 5 mg Cr+6/kg body weight to pregnant golden hamsters included cleft palates and defects in the ossification of the skeletal system (Gale 1978).

Accumulations of Cr in tissues and organism depend heavily on its chemical form, route of entry, and amount administered (Yamaguchi et al. 1983). Tissue accumulations were significant in dogs exposed to drinking water concentrations of 11.2 ppm Cr; but were nil at 6 ppm (Steven et al. 1976). Although both Cr+3 and +6 accumulated in brain, kidney, and myocardium of rabbits, the accumulation of Cr+6 was highest in brain and that of Cr+3 in kidney; for both valence states there was no correlation between dose and concentration of stored Cr, or extent of tissue damage (Hatherill 1981). Tissue residues in mice given 0.1 ppm Cr+6 in food and water during lifetime exposure ranged from 0.1 mg Cr/kg fresh weight in liver to 0.7 in heart; mice given 5.1 ppm for a similar period contained 0.5 to 1.8 mg Cr/kg fresh weight in tissues, the residues being highest in the heart and spleen (Schroeder et al. 1964). Trivalent Cr was poorly absorbed from the intestinal tract of rats (<1% of an oral dose), whereas absorption of Cr+6 ranged from 3 to 6% (Langard and Norseth 1979). However, both Cr+3 and Cr+6 traverse placental barriers in mice when administered intravenously (Steven et al. 1976; Langard and Norseth 1979). All chemical forms of chromium, except chromates, cleared rapidly from the blood of rats. At dose levels of 60 to 250 ug/kg body weight, Cr+6 tended to accumulate in the reticuloendothelial system, liver, spleen, and bone marrow; at the much lower doses of 10 and 1 ug/kg body weight, major accumulation sites were the bone, marrow, spleen, testes, and epididymis (Langard and Norseth 1979). Female rats given a single i.v. injection of radiochromium-51 depurated the isotope primarily by urinary clearance, and secondarily by fecal and residual clearances over an 11-day period. Retained radiochromium-51 accumulated over time in bone, kidney, spleen, and liver (Onkelinx 1977). For multicompartmental excretion patterns recorded in rats, biological half-lives of the three components were estimated to be 0.5, 5.9, and 83.4 days; in mammals, Cr is excreted primarily in urine (Langard and Norseth 1979). At least three distinct Cr+6 excretion patterns exist in rats: blood has a single component, with a biological half-life of 13.9 days; testes, brain, kidney and lung have two components; and liver has three components with half-lives of 2.4 hours, 52.8 hours, and 15.7 days (Yamaguchi et al. 1983). Excretion patterns for Cr+3 in rats were unpredictable and impossible to calculate (Yamaguchi et al. 1983). The excretion patterns for fecal Cr among 40 grazing Angus cows given 20 g dietary Cr_2O_3 (13.6 g Cr+3) daily for 72 days was diurnal; excretion was lowest at 8 p.m. and highest at 9 a.m. (Hopper et al. 1978).

FIELD INVESTIGATIONS

There is a wealth of data concerning the effects of chromium on living organisms under laboratory conditions simulating those encountered in the vicinity of high Cr discharges and accumulations typical of electroplating plants, tanneries, ocean dumping sites, and municipal waste outfalls. However, little research has been conducted under actual field conditions, except in three general fields: occupational exposures of humans in the chromate industry (discussed earlier), accidental poisoning of livestock resulting from oil-field activities, and Cr accumulations in ecosystems impacted by discharges associated with cooling waters or cooling towers.

All cases of accidental chromate poisoning in cattle have resulted from the exposure of animals to chromate compounds associated with oil field activities. Chromates are used as a corrosion inhibitor between the pipe and casing and are often added to drilling fluids (in the form of chromelignosulfonate) to improve thermal stability. One recorded case involved 20 mature cows and their 8-month-old calves, grazing in a native pasture where an oil well had just been completed. One cow and calf died and another cow and calf became uncoordinated and thin, and the feces contained bloody mucous. The calf soon died. The cow aborted, but appeared to recover completely. Liver from the dead calf had 14.8 mg Cr/kg fresh weight vs 1.8 in controls; levels of arsenic and lead were not elevated (Reagor and McDonald 1980). The cause of death was the consumption by the animals of concentrated sodium chromate found near the well site. In other cases, 2 of 80 heifers died after consuming concentrated zinc chromate, and 10 cows and one calf died after they had ingested ammonium chromate. In poisoned cows, Cr concentrations were 500 mg/kg in stomach contents, 15.8 ppm fresh weight in kidney vs. 3.0 ppm in controls, and 1.1 ppm in blood vs. 0.02 ppm for controls (Kerr and Edwards 1981).

Chromium is widely used as a corrosion inhibitor in cooling waters by the electric power industry. Its use in this capacity involves addition of a Cr+6 salt, typically sodium dichromate, which forms an oxide on metal

surfaces. Chromates are subsequently released to surface waters in high concentrations, compared with background levels of Cr in most freshwaters. In White Oak Lake (Eastern Tennessee), which received chronic inputs of chromates from cooling towers located on two tributary streams, typical Cr+6 concentrations of 3 to 10 ppm in waste effluents produced 100 to 300 ppb of Cr+6 in White Oak Lake vs. 5 ppb in a control area (Elwood et al. 1980). Concentrations of Cr in muscle of bluegills and largemouth bass from White Oak Lake did not differ significantly from those in fish from a control site—suggesting that these species either effectively regulated Cr concentration or that the elevated Cr levels in White Oak Lake (where 20 to 73% of the total chromium was Cr+6) were in a form that was unavailable for absorption into tissues. Elwood et al. (1980) suggested that Cr is an element with a determinant concentration in fish, and that accumulation is independent of environmental concentration. This concept requires validation. Noteworthy is the observation that Cr concentrations were lower in muscle and body of older freshwater teleosts—an observation consistent with the findings of Eisler (1984), who noted that liver Cr decreased with increasing age in marine teleosts.

Cooling towers of uranium enrichment facilities and gaseous diffusion plants, similar to those of 1,000-MW conventional steam electric stations, contain a chromate zinc-phosphate compound to inhibit corrosion and fouling within the cooling system. A small fraction of the cooling water, containing about 20 ppm of Cr+6, becomes entrained within the exit air flow and is deposited as drift on the landscape, together with other salts found in the recirculating water system, such as sodium pentachlorophenate, chromated copper arsenate, and acid copper chromate. Effects of the Cr component on biological systems have been under investigation in Kentucky and Tennessee for many years (Taylor and Parr 1978; Taylor 1980;Taylor et al. 1975, 1979, 1983). Analysis of vegetation along distance gradients from the cooling towers identified areas of significant drift deposition, accumulation, and magnitude of atmospheric transport over the landscape. At 13 m downwind from the point source, plant foliar concentrations of Cr were highest in winter at 1,390 ppm dry weight, decreasing to 190 in spring, and to 173 in summer as the demand for cooling—and hence the operation time of the facility—decreased. Decreased accumulations on foliage probably reflected high mobility due to leaching, and the short life span of individual leaves. In contrast, Cr concentrations in plant litter at 13 m increased from 894 ppm dry weight during winter to 1,890 ppm in summer and 2,140 ppm in autumn. Accumulation of Cr in the litter was probably related to the higher surface-to-volume ratio in the litter biomass resulting from seasonal senescence of foliage. Taylor and his coworkers emphasized that no adverse biological effects were observed in native vegetation bearing high Cr residues. Concentrations in plants and litter decreased with increasing distance from the cooling towers: concentrations in foliage at 168, 530, and 923 m downwind were 157, 10, and 1.3 ppm (dry weight), respectively; for litter, these values were 421, 24, and 5.8. Potted tobacco plants (*Nicotiana tabacum*) proved to be sensitive indicators of Cr contamination. Tobacco plants placed 15 m from the towers contained 30X background levels after 1 week and up to 237 ppm dry weight in 5 weeks; in plants placed 200 m downwind, leaf growth was reduced 75% after 7 weeks. Beetles and crickets collected near the towers contained 9 to 37 ppm Cr in gut contents (vs. 0.5 to 0.8 ppm for controls); however, assimilation rates were not measured. Cotton rats trapped in a fescue field adjacent to a large mechanical draft cooling tower contained up to 10X more Cr in hair, pelt, and bone than controls, but accumulations were negligible in viscera and other internal organs. Licking of the coat by rats appeared to be a primary route of Cr uptake—a likelihood confirmed experimentally by Langard and Nordhagen (1980). Feeding of radiochromium-51 to cotton rats demonstrated low assimilation (0.8%), and rapid initial loss of Cr+6 (99% in 1 day)—suggesting that Cr is neither essential to cotton rats nor accumulated to any great extent through ingestion of drift-contaminated vegetation or inhalation of drift-contaminated air (Taylor 1980). Biological half times of Cr assimilated by man and cotton rats were similar: 616 and 693 days, respectively. The magnitude of the half times suggests that Cr derived from a chromate has a high potential for biological interaction, but that fractional assimilation is very low—thus reducing the likelihood of toxic effects.

CURRENT RECOMMENDATIONS

As reported here, sensitive species of freshwater aquatic organisms showed reduced growth, inhibited reproduction, and increased bioaccumulation at 10.0 ug/L of Cr+6, and other adverse effects at 30.0 ug/L of Cr+3. Among marine organisms, measurable accumulations were recorded in oysters and worms at 5.0 ug/L of Cr+6, algal growth was reduced at 10.0 ug/L, and reproduction of polychaete annelid worms was inhibited at 12.5 ug/L; in all situations, Cr+3 was less damaging than Cr+6. For birds and mammals, dietary levels of 10.0 mg Cr+3/kg adversely affected young black ducks, and 5.1 mg Cr+6/kg in food and water of mice was associated with elevated tissue residues. The significance of Cr residues is unclear, but available evidence suggests that organs and tissues of fish and wildlife that contain 4.0 mg total Cr/kg dry weight should be viewed

as presumptive evidence of Cr contamination. Aerosol concentrations in excess of 10.0 ug Cr+6/m^3 are potentially harmful to human health; in the absence of supporting data, this value is recommended for protection of sensitive species of wildlife, especially migratory waterfowl.

Proposed criteria for the protection of various environmental compartments against chromium are numerous, disparate, and often contradictory (Table 6). Some of this confusion may be attributable to the general lack of confidence in analyses of chromium residues conducted some years ago, and some to the continued inability to quantify chemical species and ionic states of Cr. Uncertainties about the metabolic role of organochromium compounds, water soluble Cr+3 species, and their interactions with other components in complex and potentially toxic mixtures, further confound the issue. The essentiality of Cr to some, but not all, species of mammals is recognized, but comparable data for other groups of organisms are missing. Finally, the wide range in sensitivities and accumulation rates documented between different taxonomic groups, and even among closely related species, to Cr+3 and Cr+6 salts merits elucidation. Until these questions are resolved, the acceptance of uniform Cr criteria is debatable, and their passage to administratively legislated standards is contraindicated.

Table 6. Proposed chromium criteria for protection of selected resources.

Category and criterion (units in parentheses)	Chromium concentration	Reference[a]
Freshwater aquatic life protection (µg/L)		
USA	<0.29 Cr+6 as 24 h average; not to exceed 21 Cr+6 at any time	1
Water hardness, in mg $CaCO_3$/L		
50	<2,200 Cr+3 at any time	1
100	<4,700 Cr+3 at any time	1
200	<9,900 Cr+3 at any time	1
Colorado	<25 Cr+6; <100 Cr+3	2
Florida		
Effluent discharges	<500 Cr+6; <1,000 total Cr	2
Recovery waters	<50 total Cr	
Indiana		
Most waters	Not to exceed 0.1X 96-h LC-50 of aquatic species	2
Lake Michigan	<50 total Cr	2
Marine aquatic life protection (µg/L)		
USA	<18 Cr+6 as 24 h average; not to exceed 1,260 Cr+6 at any time	1
USA	Insufficient data base for Cr+3 at this time, but presumably less stringent than Cr+6	1
California	<2 total Cr, 6 month median; <8 total Cr, daily maximum; <20 total Cr, instantaneous mix	2
California		
Waste discharges into marine waters	<5 total Cr for 50% of measurements; <10 for 10% of measurements	3
Drinking water (µg/L)		
USA	<50 Cr+6; <170,000 Cr+3	1
California	<50 total Cr	2
Colorado	<50 Cr+6; <50 Cr+3	2
Florida	<50 total Cr	2
Brazil	<50 total Cr	4
USSR	<600 total Cr	4
Agricultural water (µg/L)		
Irrigation Colorado	<100 of Cr+6; <100 of Cr+3	2
Groundwater Florida	<50 total Cr	2
Diet		
Human health protection		
Normal dietary intake	50 to 70 □g Cr+3 daily	2
"	30 to 100 µg total Cr daily	5
Food stuffs	100 to 300 □g total Cr/kg fresh weight	2
Tissue residues (µg/kg fresh weight)		
Human soft tissues	<30 total Cr	2
Animal tissues	<200 total Cr	2
Air (µg/m^3)		
USA	<50 total Cr	6

[a]References: 1, EPA 1980; 2, Ecological Analysts 1981; 3, Reish 1977; 4, Pfeiffer et al. 1980; 5, Langard and Norseth 1979; 6, Steven et al. 1976.

ACKNOWLEDGMENTS

I thank I. Moore for secretarial help; L. J. Garrett, M. Shawe, and B. Graff for literature retrieval; B. A. Rattner, J. B. Hunn, and J. F. Moore for reviewing the manuscript; and P. H. Eschmeyer and J. R. Zuboy for editorial services.

LITERATURE CITED

Abbasi, S. A., and R. Soni. 1983. Stress-induced enhancement of reproduction in earthworm *Octochaetus pattoni* exposed to chromium (VI) and mercury (II)—implications in environmental management. Int. J. Environ. Stud. 22:43-47.

Alexander, G. V., and D. R. Young. 1976. Trace metals in southern California mussels. Mar. Pollut. Bull. 7:7-9.

Anderlini, V. 1974. The distribution of heavy metals in the red abalone, *Haliotis rufescens*, on the California coast. Arch. Environ. Contam. Toxicol. 2:253-265.

Arillo, A., C. Margiocco, F. Melodia, and P. Mensi. 1982. Biochemical effects of long term exposure to Cr, Cd, Ni on rainbow trout (*Salmo gairdneri* Rich.): influence of sex and season. Chemosphere 11:47-57.

Ayling, G. M. 1974. Uptake of cadmium, zinc, copper, lead and chromium in the Pacific oyster, *Crassostrea gigas*, grown in the Tamar River, Tasmania. Water Res. 8:729-738.

Baptist, J. P., D. E. Hoss, and C. W. Lewis. 1970. Retention of chromium-51, iron-59, cobalt-60, zinc-65, strontium-85, niobium-95, indium-114m, and iodine-131 by the Atlantic croaker (*Micropogon undulatus*). Health Phys. 18:141-148.

Baptist, J. P., and C. W. Lewis. 1969. Transfer of Zn and Cr through an estuarine food chain. Proc. 2nd Nat. Symp. Radioecology. U.S. Atom. Ener. Comm. Conf. 670503:420-430.

Bates, T. E., A. Haq, Y. K. Soon, and J. R. Moyer. 1975. Uptake of metals from sewage sludge amended soils. Int. Conf. Heavy Metals in the Environment. Symp. Proc. (Toronto) 2(1):403-416.

Bernhard, M., and A. Zattera. 1975. Major pollutants in the marine environment. Pages 195-300 *in* E. A. Pearson and E. D. Frangipane (eds.). Marine pollution and marine waste disposal. Pergamon Press, New York.

Bertine, K. K., and E. D. Goldberg. 1972. Trace elements in clams, mussels, and shrimp. Limnol. Ocean. 17:877-884.

Blus, L. J., B. S. Neely, Jr., T. G. Lamont, and B. M. Mulhern. 1977. Residues of organochlorines and heavy metals in tissues and eggs of brown pelicans, 1969-1973. Pestic. Monitor. J. 11:40-53.

Bookhout, C. G., R. J. Monroe, R. B. Forward, Jr., and J. D. Costlow, Jr. 1984. Effects of hexavalent chromium on development of crabs, *Rithropanopeus harrisii* and *Callinectes sapidus*. Water, Air, Soil Pollut. 21:199-216.

Brooks, R. R., and D. Rumsey. 1974. Heavy metals in some New Zealand commercial sea fishes. N.Z. J. Mar. Freshwater Res. 8:155-166.

Bryan, G. W., and L. G. Hummerstone. 1977. Indicators of heavy metal contamination in the Looe estuary (Cornwall) with particular regard to silver and lead. J. Mar. Biol. Assoc. U.K. 57:75-92.

Bryan, G. W., W. J. Langston, L. G. Hummerstone, G. R. Burt, and Y. B. Ho. 1983. An assessment of the gastropod, *Littorina littorea*, as an indicator of heavy-metal contamination in United Kingdom estuaries. J. Mar. Biol. Assoc. U.K. 63:327-345.

Bryan, G. W., and H. Uysal. 1978. Heavy metals in the burrowing bivalve *Scrobicularia plana* from the Tamar estuary in relation to environmental levels. J. Mar. Biol. Assoc. U.K. 58:89-108.

Buhler, D. R., R. M. Stokes, and R. S. Caldwell. 1977. Tissue accumulation and enzymatic effects of hexavalent chromium in rainbow trout (*Salmo gairdneri*). J. Fish. Res. Board Canada 34:9-18.

Buikema, A. L., Jr., J. Cairns, Jr., and, G. W. Sullivan. 1974. Evaluation of *Philodena acuticornis* (Rotifera) as a bioassay organism for heavy metals. Water Resour. Bull. 10:648-661.

Calamari, D., G. F. Gaggino, and G. Pacchetti. 1982. Toxicokinetics of low levels of Cd, Cr, Ni and their mixture in long-term treatment on *Salmo gairdneri* Rich. Chemosphere 11:59-70.

Capuzzo, J. M., and J. J. Sasner, Jr. 1977. The effect of chromium on filtration rates and metabolic activity of *Mytilus edulis* L. and *Mya arenaria* L. Pages 225-237 *in* F. J. Vernberg, A. Calabrese, F. P. Thurberg, and W. B. Vernberg (eds.). Physiological responses of marine biota to pollutants. Academic Press, New York.

Carr, R. S., W. L. McCullough, and J. M. Neff. 1982. Bioavailability of chromium from a used chrome lignosulphonate drilling mud to five species of marine invertebrates. Mar. Environ. Res. 6:189-203.

Catalan, Z. B. 1982. Behavioral responses of *Chironomus tentans* to stress as determined by an optical fiber monitoring system. Ph.D. thesis, Univ. Michigan, Ann Arbor. 94 pp.

Chipman, W. A. 1967. Some aspects of the accumulation of chromium-51 by marine organisms. Proc. Int. Symp. Radioecol. Conc. Proc., 1966 (Stockholm):931-941.

Coleman, R. N., and J. H. Paran. 1983. Accumulation of hexavalent chromium by selected bacteria. Environ. Technol. Lett. 4:149-156.

Conklin, P. J., D. Drysdale, D. G. Doughtie, K. R. Rao, J. P. Kakareka, T. R. Gilbert, and R. F. Shokes. 1983. Comparative toxicity of drilling muds: role of chromium and petroleum hydrocarbons. Mar. Environ. Res. 10:105-125.

DeClerck, R., R. Vanderstappen, W. Vyncke, and P. Van Hoeyweghen. 1979. La teneur en metaux lourdes dans les organismes marins provenant de la capture accessoire de la peche coteire belg. Rev. Agric. (Brussels) 3(32):793-801.

Del Carratore, R., E. Cundari, C. Morganti, J. Moretton, C. Bauer, C. Corsil, R. Nieri, M. Paolini, and G. Bronzetti. 1984. Detection of mutagens in leather and tannery industry assay of 28 commercial mixtures. Bull. Environ. Contam. Toxicol. 32:400-404.

Doughtie, D. G., P. J. Conklin, and K. R. Rao. 1983. Cuticular lesions induced in grass shrimp exposed to hexavalent chromium. J. Invert. Pathol. 42:249-258.

Duinker, J. C., M. T. J. Hillebrand, and R. F. Nolting. 1979. Organochlorines and metals in harbour seals (Dutch Wadden Sea). Mar. Pollut. Bull. 10:360-364.

Duval, R. L., C. R. Ferris, R. B. Owen, Jr., K. E. Gibbs, L. K. Fink, Jr., and F. E. Woodward. 1980. Effects of a cattle skin tannery on stream quality and benthic macroinvertebrates in central Maine. Bull. Environ. Contam. Toxicol. 25:269-276.

Eastin, W. C., Jr., S. D. Haseltine, and H. C. Murray. 1980. Intestinal absorption of 5 chromium compounds in young black ducks (*Anas rubripes*). Toxicol. Lett. 6:193-197.

Ecological Analysts, Inc. 1981. The sources, chemistry, fate, and effects of chromium in aquatic environments. Avail. from American Petroleum Institute, 2101 L St., N.W., Washington, DC 20037. 207 pp.

Eisler, R. 1981. Trace metal concentrations in marine organisms. Pergamon Press, New York. 687 pp.

Eisler, R. 1984. Trace metal changes associated with age of marine vertebrates. Biol. Trace Elem. Res. 6:165-180.

Eisler, R., M. M. Barry, R. L. Lapan, Jr., G. Telek, E. W. Davey, and A. E. Soper. 1978. Metal survey of the marine clam *Pitar morrhuana* collected near a Rhode Island (USA) electroplating plant. Mar. Bio. 45:311-317.

Eisler, R., R. L. Lapan, Jr., G. Telek, E. W. Davey, A. E. Soper, and M. Barry. 1977. Survey of metals in sediments near Quonset Point, Rhode Island. Mar. Pollut. Bull. 8:260-264.

Elwood, J. W., J. J. Beauchamp, and C. P. Allen. 1980. Chromium levels in fish from a lake chronically contaminated with chromates from cooling towers. Int. J. Environ. Stud. 14:289-298.

EPA. 1980. Ambient water quality criteria for chromium. U.S. Environ. Protection Agency Rep. 440/5-80-035.

Foster, P. 1976. Concentrations and concentration factors of heavy metals in brown algae. Environ. Pollut. 10:45-53.

Fowler, S. W., and B. Oregioni. 1976. Trace metals in mussels from the N.W. Mediterranean. Mar. Pollut. Bull. 7:26-29.

Fukai, R. 1965. Analysis of trace amounts of chromium in marine organisms by the isotope dilution of Cr-51. Pages 335-351 *in* Radiochemical methods of analysis. Int. Atom. Ener. Agen. (Vienna).

Fukai, R., and D. Broquet. 1965. Distribution of chromium in marine organisms. Bull. Inst. Ocean. (Monaco) 65(1336):1-19.

Fukai, R., B. Oregioni, and D. Vas. 1978. Interlaboratory comparability of measurements of trace elements in marine organisms: results of intercalibration exercise on oyster homogenate. Ocean. Acta 1:391-396.

Gale, T. F. 1978. Embryotoxic effects of chromium trioxide in hamsters. Environ. Res. 16:101-109.

Giblin, A. E., A. Bourg, I. Valiela, and J. M. Teal. 1980. Uptake and losses of metals in sewage sludge by a New England salt marsh. Am. J. Bot. 67:1059-1068.

Gilani, S. H., and M. Marano. 1979. Chromium poisoning and chick embryogenesis. Environ. Res. 19:427-431.

Gough, L. P., and R. C. Severson. 1976. Impact of point source emissions from phosphate processing on the element content of plants and soils, Soda Springs, Idaho. Pages 225-233 *in* D. D. Hemphill (ed.). Trace substances in environmental health. Vol. X, Univ. Missouri, Columbia.

Graham, D. L. 1972. Trace metal levels in intertidal mollusks of California. Veliger 14:365-372.

Greig, R., and D. Wenzloff. 1977. Final report on heavy metals in small pelagic finfish, euphausid crustaceans and apex predators, including sharks, as well as on heavy metals and hydrocarbons (C 15+) in sediments collected at stations in and near deepwater dumpsite 106. Vol. III. Contaminant inputs and chemical characteristics. Pages 547-564 *in* U.S. Dep. Com. NOAA, Rockville, MD, Baseline report of the environmental conditions on deepwater dumpsite 106.

Greig, R. A., D. R. Wenzloff, A. Adams, B. Nelson, and C. Shelpuk. 1977. Trace metals in organisms from ocean disposal sites of the middle eastern United States. Arch. Environ. Contam. Toxicol. 6:395-409.

Gryzhankova, L. N., G. N. Sayenko, A. V. Karyakin, and N. V. Laktionova. 1973. Concentrations of some metals in the algae of the Sea of Japan. Oceanology 13:206-210.

Hall, R. A., E. G. Zook, and G. M. Meaburn. 1978. National Marine Fisheries Service survey of trace elements in the fishery resources. U.S. Dep. Comm. NOAA Tech. Rep. NMFS SSRF-721. 313 pp.

Hall, R. J., and B. M. Mulhern. 1984. Are anuran amphibians heavy metal accumulators? Pages 123-133 *in* R. A. Seigel, L. E. Hunt, J. L. Knight, L. Malaret, and N. L. Zuschlag (eds.). Vertebrate ecology and systematics—a tribute to Henry S. Fitch. Mus. Nat. Hist., Univ. Kansas, Lawrence.

Hartenstein, R., E. F. Neuhauser, and J. Collier. 1980. Accumulation of heavy metals in the earthworm *Eisenia foetida*. J. Environ. Qual. 9:23-26.

Haseltine, S. D., B. M. Mulhern, and C. Stafford. 1980. Organochlorine and heavy metal residues in black duck eggs from the Atlantic flyway, 1978. Pestic. Monitor. J. 14:53-57.

Haseltine, S. D., L. Sileo, D. J. Hoffman, and B. M. Mulhern. 1985. Effects of chromium on reproduction and growth of black ducks. Manuscript in preparation.

Hatherill, J. R. 1981. A review of the mutagenicity of chromium. Drug Chem. Toxicol. 4:185-195.

Hawkins, C. J., D. L. Parry, and C. Pierce. 1980. Chemistry of the blood of the ascidian *Podoclavella moluccensis*. Biol. Bull. 159:669-680.

Heinz, G. H., and S. D. Haseltine. 1981. Avoidance behavior of young black ducks treated with chromium. Toxicol. Lett. 8:307-310.

Honig, R. A., M. J. McGinniss, A. L. Buikema, Jr., and J. Cairns, Jr. 1980. Toxicity tests of aquatic pollutants using *Chilomonas paramecium* Ehrenberg (Flagellata) populations. Bull. Environ. Contam. Toxicol. 25:169-175.

Hopper, J. T., J. W. Holloway, and W. T. Butts, Jr. 1978. Animal variation in chromium sesquioxide excretion patterns of grazing cows. J. Anim. Sci. 46:1096-1102.

Horowitz, A., and B. J. Presley. 1977. Trace metal concentrations and partitioning in zooplankton, neuston, and benthos from the South Texas Outer Continental Shelf. Arch. Environ. Contam. Toxicol. 5:241-255.

Ishibashi, M., and T. Yamamoto. 1960. Inorganic constituents in seaweeds. Rec. Ocean. Wks. Japan 5:55-62.

James, B. R., and R. J. Bartlett. 1983a. Behavior of chromium in soils: V. Fate of organically complexed Cr (III) added to soil. J. Environ. Qual. 12:169-172.

James, B. R., and R. J. Bartlett. 1983b. Behavior of chromium in soils. VI. Interactions between oxidation-reduction and organic complexation. J. Environ. Qual. 12:173-176.

Jenkins, D. W. 1980. Biological monitoring of toxic trace metals. Vol. 2. Toxic trace metals in plants and animals of the world. Part I. U.S. Environ. Protection Agency Rep. 600/3-80-090:364-443.

Jensen, L. S., and D. Maurice. 1980. Dietary chromium and interior egg quality. Poult. Sci. 59:341-346.

Jouany, J. M., P. Vasseur, and J. F. Ferard. 1982. Ecotoxicite directe et integree du chrome hexavalent sur deux niveaux trophiques associes: *Chlorella vulgaris* et *Daphnia magna*. Environ. Pollut. 27A:207-221.

Karbe, L., C. Schnier, and H. O. Siewers. 1977. Trace elements in mussels (*Mytilus edulis*) from coastal areas of the North Sea and the Baltic. Multielement analysis using instrumental neutron activation analysis. J. Radioanal. Chem. 37:927-943.

Kelsall, J. P. 1970. Chemical elements in waterfowl flight feathers. Prog. Notes, Canad. Wildl. Serv. 17:1-11.

Kerr, L. A., and W. C. Edwards. 1981. Chromate poisoning in livestock from oil field wastes. Veterin. Human Toxicol. 23:401-402.

Kobayashi, N. 1971. Fertilized sea urchin eggs as an indicatory material for marine pollution bioassays, preliminary experiments. Publ. Seto Mar. Biol. Lab. 18:379-406.

Kobayashi, N. 1977. Preliminary experiments with sea urchin pluteus and metamorphosis in marine pollution bioassay. Publ. Seto Mar. Biol. Lab. 24:9-21.

Krishnaja, A. P., and M. S. Rege. 1982. Induction of chromosomal aberrations in fish *Boleophthalmus dussumieri* after exposure in vivo to mitomycin C and heavy metals mercury, selenium and chromium.

Mutat. Res. 102:71-82.

Laj, S., V. K. Jain, and S. K. Tandon. 1984. Comparative toxicity of trivalent and hexavalent chromium IV: biochemical changes in blood and liver of rat. J. Environ. Biol. 5:29-35.

Lande, E. 1977. Heavy metal pollution in Trondheimsfjorden, Norway, and the recorded effects on the fauna and flora. Environ. Pollut. 12:187-198.

Langard, S., and A.-L. Nordhagen. 1980. Small animal inhalation chambers and the significance of dust ingestion from the contaminated coat when exposing rats to zinc chromate. Acta Pharmacol. Toxicol. 46:43-46.

Langard, S., and T. Norseth. 1979. Chromium. Pages 383-397 *in* L. Friberg, G. F. Nordberg, and V. B. Vouk (eds.). Handbook on the toxicology of metals. Elsevier/North Holland Biomedical Press.

Leonard, A., and R. R. Lauwerys. 1980. Carcinogenicity and mutagenicity of chromium. Mutat. Res. 76:227-239.

Livingston, H. D., and G. Thompson. 1971. Trace element concentrations in some modern corals. Limnol. Ocean. 16:786-795.

Lucas, H. F., D. N. Edgington, and P. J. Colby. 1970. Concentrations of trace elements in Great Lakes fishes. J. Fish. Res. Board Canada 27:677-684.

Mahoney, J. B. 1982. The effects of trace metals on growth of a phytoflagellate *Olisthodiscus luteus*, which blooms in lower New York Bay. Bull. N.J. Acad. Sci. 27:53-57.

Mangi, J., K. Schmidt, J. Pankow, L. Gaines, and P. Turner. 1978. Effects of chromium on some aquatic plants. Environ. Pollut. 16:285-291.

Martin, J. H., and G. A. Knauer. 1973. The elemental composition of plankton. Geochim. Cosmochim. Acta 37:1639-1653.

Mayer, L. M., and L. L. Schick. 1981. Removal of hexavalent chromium from estuarine waters by model substrates and natural sediments. Environ. Sci. Technol. 15:1482-1484.

Mayer, L. M., L. L. Schick, P. M. Rossi, H. H. Patterson, C. A. Chang, D. E. Bause, and L. K. Fink, Jr. 1981. Analysis, distribution, and interactions of chromium in the aquatic environment. Univ. Maine at Orono, Land and Water Res. Cent., Compl. Rep. Proj. B-016-ME. 58 pp.

Mearns, A. J., P. S. Oshida, M. J. Sherwood, D. R. Young, and D. J. Reish. 1976. Chromium effects on coastal organisms. J. Water Pollut. Control Feder. 48:1929-1939.

Mearns, A. J., and D. R. Young. 1977. Chromium in the southern California marine environment. Pages 125-142 *in* C. S. Giam (ed.). Pollutant effects on marine organisms. D.C. Heath, Lexington, Massachusetts.

Montgomery, J. R., M. Price, J. Thurston, G. L. de Castro, L. L. Cruz, and D. D. Zimmerman. 1978. Biological availability of pollutants to marine organisms. U.S. Environ. Protection Agency Rep. 600/3-78-035. 134 pp.

Moraitou-Apostolopoulou, M., and G. Verriopoulos. 1982a. Individual and combined toxicity of three heavy metals, Cu, Cd and Cr for the marine copepod *Tisbe holothuriae*. Hydrobiologia 87:83-87.

Moraitou-Apostolopoulou, M., and G. Verriopoulos. 1982b. Toxicity of chromium to the marine planktonic copepod *Acartia clausi*, Giesbrecht. Hydrobiologia 96:121-127.

Muller, H. G. 1980. Acute toxicity of potassium dichromate to *Daphnia magna* as a function of water quality. Bull. Environ. Contam. Toxicol. 25:113-117.

Murti, R., O. Omkar, and G. S. Shukla. 1983. Chromium toxicity to a freshwater prawn *Macrobrachium lamarrei* (H. M. Edwards). Toxicol. Lett. 18:257-261.

Nadkarni, R. A., and W. D. Ehmann. 1970. Trace element determination in biological materials by neutron activation analysis. Pages 407-419 *in* D. D. Hemphill (ed.). Trace substances in environmental health IV. Univ. Missouri, Columbia.

NAS. 1974. Medical and biologic effects of environmental pollutants. Chromium. Nat. Acad. Sci., Div. Med. Sci., Nat. Res. Coun., Washington, DC. 155 pp.

Neff, J. M., R. S. Carr, and W. L. McCullough. 1981. Acute toxicity of a used chrome lignosulfonate drilling mud to several species of marine invertebrates. Mar. Environ. Res. 4:251-266.

Neff, J. M., R. S. Foster, and J. F. Slowey. 1978. Availability of sediment-absorbed heavy metals to benthos with particular emphasis on deposit-feeding infauna. Tech. Rep. D-78-42, U.S. Army Waterways Exp. Sta., Vicksburg, Mississippi. 286 pp.

Negilski, D. S. 1976. Acute toxicity of zinc, cadmium and chromium to the marine fishes, yellow-eye mullet (*Aldrichetta forsteri* C. & V.) and small-mouthed hardyhead *Atherinasoma microstoma* Whitley). Aust. J. Mar. Freshwater Res. 27:137-149.

Norseth, T. 1981. The carcinogenicity of chromium. Environ. Health Perspect. 40:121-130.

Okubo, K., and T. Okubo. 1962. Study on the bioassay method for the evaluation of water pollution—II. Use of the fertilized eggs of sea urchin and bivalves. Bull. Tokai Reg. Fish. Res. Lab. 32:131-140.

Olson, K. R., and R. C. Harrel. 1973. Effect of salinity on acute toxicity of mercury, copper, and chromium for *Rangia cuneata* (Pelecypoda, Mactridae). Contrib. Mar. Sci. 17:9-13.

Onkelinx, C. 1977. Compartment analysis of metabolism of chromium (III) in rats of various ages. Am. J. Physiol. 232:E478-E484.

Oshida, P. S., A. J. Mearns, D. J. Reish, and L. S. Word. 1976. The effects of hexavalent and trivalent chromium on *Neanthes arenaceodentata* (Polychaeta: Annelida). S. Calif. Coastal Water Res. Proj., 1500 E. Imperial Hwy., El Segundo, Calif., TM 225. 58 pp.

Oshida, P. S., and L. S. Word. 1982. Bioaccumulation of chromium and its effects on reproduction in *Neanthes arenaceodentata* (Polychaeta). Mar. Environ. Res. 7:167-174.

Oshida, P. S., L. S. Word, and A. J. Mearns. 1981. Effects of hexavalent and trivalent chromium on the reproduction of *Neanthes arenaceodentata* (Polychaeta). Mar. Environ. Res. 5:41-49.

Osterberg, C., W. G. Pearcy, and H. J. Curl. 1964. Radioactivity and its relation to oceanic food chains. J. Mar. Res. 22:2-12.

Pak, C. K., K. R. Yang, and I. K. Lee. 1977. Trace metals in several edible marine algae of Korea. J. Oceanol. Soc. Korea 12:41-47.

Papadopoulu, C. 1973. The elementary composition of marine invertebrates as a contribution to the sea pollution investigation. Proc. MAMBO meeting, Castellabate, Italy, June 18-22, 1973. 18 pp.

Papadopoulu, C., and G. D. Kanias. 1977. Tunicate species as marine pollution indicators. Mar. Pollut. Bull. 8:229-231.

Papadopoulu, C., G. D. Kanias, and E. M. Kassimati. 1976. Stable elements of radioecological importance in certain echinoderm species. Mar. Pollut. Bull. 7:143-144.

Papadopoulu, C., and E. M. Kassimati. 1977. Stable elements in skeletal formations of fish species from Greek waters. Thal. Jugoslavica 13:187-192.

Pfeiffer, W. C., M. Fiszman, and N. Carbonell. 1980. Fate of chromium in a tributary of the Iraja River, Rio de Janeiro. Environ. Pollut. 1B:117-126.

Phelps, D. K., G. Telek, and R. L. Lapan, Jr. 1975. Assessment of heavy metal distribution within the food web. Pages 341-348 *in* E. A. Pearson, and E. D. Frangipane (eds.). Marine pollution and marine waste disposal. Pergamon Press, New York.

Pickering, Q. H. 1980. Chronic toxicity of hexavalent chromium to the fathead minnow. Arch. Environ. Contam. Toxicol. 9:405-413.

Plaskett, D., and I. C. Potter. 1979. Heavy metal concentrations in the muscle tissue of 12 species of teleost from Cockburn Sound, Western Australia. Aust. J. Mar. Freshwater Res. 30:607-616.

Post, M. A., and P. G. Campbell. 1980. Lead chromate pigments—a literature survey on environmental and toxic effects. U.S. Dep. Comm. Nat. Bur. Stand. Rep. NBSIR 80-1974. 38 pp.

Preston, A. M., R. P. Dowdy, M. A. Preston, and J. N. Freeman. 1976. Effect of dietary chromium on glucose tolerance and serum cholesterol in guinea pigs. J. Nutr. 106:1391-1397.

Preston, E. M. 1971. The importance of ingestion in chromium-51 accumulation by *Crassostrea virginica* (Gmelin). J. Exp. Mar. Biol. Ecol. 6:47-54.

Reagor, J. C., and D. McDonald. 1980. Chromate poisoning in cattle—a case report. Southwest. Veterin. 33:10-11.

Rehm, E., M. Schulz-Baldes, and B. Rehm. 1984. Geochemical factors controlling the distribution of Fe, Mn, Pb, Cd, Cu and Cr in Wadden areas of the Weser estuary (German Bight). Veroff. Inst. Meeresforsch. Bremerh. 20:75-102.

Reish, D. J. 1977. Effects of chromium on the, life history of *Capitella capitata* (Annelida: Polychaeta). Pages 199-207 *in* F. J. Vernberg, A. Calabrese, F. P. Thurberg, and W. B. Vernberg (eds.). Physiological responses of marine biota to pollutants. Academic Press, New York.

Ridgeway, L. P., and D. A. Karnofsky. 1952. The effects of metals on the chick embryo: toxicity and production of abnormalities in development. Ann. N.Y. Acad. Sci. 55:203-215.

Riley, J. P., and I. Roth. 1971. The distribution of trace elements in some species of phytoplankton grown in culture. J. Mar. Biol. Assoc. UK 51:63-72.

Riley, J. P., and D. A. Segar. 1970. The distribution of the major and some minor elements in marine animals. I. Echinoderms and coelenterates. J. Mar. Biol. Assoc. UK 50:721-730.

Riva, M. C., R. Flos, M. Crespi, and J. Balasch. 1981. Lethal potassium dichromate and whitening (blankophor) exposure of goldfish (*Carassius auratus*): chromium levels in gills. Comp. Biochem. Physiol. 68C:161-165.

Rosomer, G. L., W. A. Dudley, L. J. Machlin, and L. Loveless. 1961. Toxicity of vanadium and chromium for the growing chick. Poult. Sci. 40:1171-1173.

Roth, I., and H. Hornung. 1977. Heavy metal concentrations in water, sediments, and fish from Mediterranean coastal area, Israel. Environ. Sci. Tech. 11:265-269.

Sastry, K. V., and K. Sunita. 1982. Effect of cadmium and chromium on the intestinal absorption of glucose in the snakehead fish, *Channa punctatus*. Toxicol. Lett. 10:293-296.

Sastry, K. V., and K. Sunita. 1983. Enzymological and biochemical changes produced by chronic chromium exposure in a teleost fish, *Channa punctatus*. Toxicol. Lett. 16:9-15.

Sastry, K. V., and K. Sunita. 1984. Chronic toxic effects of chromium in *Channa punctatus*. J. Environ. Biol. 5:47-52.

Sastry, K. V., and S. Tyagi. 1982. Toxic effects of chromium in a freshwater teleost fish, *Channa punctatus*. Toxicol. Lett. 11:17-21.

Sather, B. T. 1967. Chromium absorption and metabolism by the crab *Podophthalmus vigil*. Proc. Int. Symp. Radioecol. Conc. Proc., 1966, Stockholm, Sweden: 943-976.

Sauter, S., K. S. Buxton, K. J. Macek, and S. R. Petrocelli. 1976. Effects of exposure to heavy metals on selected freshwater fish. Toxicity of copper, cadmium, chromium and lead to eggs and fry of seven fish species. U.S. Environ. Protection Agency Rep. 600/3-76-105. 75 pp.

Schroeder, H. A., J. J. Balassa, and I. H. Tipton. 1962. Abnormal trace metals in man—chromium. J. Chron. Dis. 15:941-964.

Schroeder, H. A., J. J. Balassa, and W. H. Vinton, Jr. 1964. Chromium, lead, cadmium, nickel and titanium in mice: effect on mortality, tumors, and tissue levels. J. Nutr. 83:239-250.

Segar, D. A., J. D. Collins, and J. P. Riley. 1971. The distribution of the major and some minor elements in marine animals. Part II. Molluscs. J. Mar. Biol. Assoc. U.K. 51:131-136.

Shuster, C. N., Jr., and B. H. Pringle. 1968. Effects of trace metals on estuarine molluscs. Proc. First Mid-Atl. Indus. Waste Conf. 13-15 Nov. 1967:285-304. Avail. from Dept. Civil Engin., Univ. Delaware, Newark, Del.

Shuster, C. N., Jr. and B. H. Pringle. 1969. Trace metal accumulation by the American oyster, *Crassostrea virginica*. Proc. Nat. Shellfish. Assoc. 59:91-103.

Sivalingam, P. M. 1978. Effects of high concentration stress of trace metals on their biodeposition modes in *Ulva reticulata* Forskal. Jpn. J. Phycol. 26:157-160.

Snyder, A. D., D. G. DeAngelis, E. C. Eimutus, D. M. Haile, J. C. Ochsner, R. B. Reznik, and H. D. Troy. 1977. Environmental monitoring near industrial sites: chromium. U.S. Environ. Protection Agency Rep. 560/6-77-016. 56 pp. plus Appendices.

Soni, R., and S. A. Abbasi. 1981. Mortality and reproduction in earthworms *Pheritima posthuma* exposed to chromium (VI). Int. J. Environ. Stud. 17:147-149.

Stacey, N. H., K-L. Wong, and C. D. Klaassen. 1983. Protective effects of chromium on the toxicity of cadmium in vivo. Toxicology 28:147-153.

Steven, J. D., L. J. Davies, E. K. Stanley, R. A. Abbott, M. Ihnat, L. Bidstrup, and J. F. Jaworski. 1976. Effects of chromium in the Canadian environment. Nat. Res. Coun. Canada, NRCC No. 15017. 168 pp. Avail. from Publications, NRCC/CNRC, Ottawa, Canada, K1A OR6.

Stevens, D. G., and G. A. Chapman. 1984. Toxicity of trivalent chromium to early life stages of steelhead trout. Environ. Toxicol. Chem. 3:125-133.

Sugatt, R. H. 1980a. Effects of sublethal sodium dichromate exposure in freshwater on the salinity tolerance and serum osmolality of juvenile coho salmon *Oncorhynchus kisutch*, in seawater. Arch. Environ. Contam. Toxicol. 9:41-52.

Sugatt, R. H. 1980b. Effects of sodium dichromate exposure on the immune responses of juvenile coho salmon *Oncorhynchus kisutch*, against *Vibrio anguillarum*. Arch. Environ. Contam. Toxicol. 9:207-216.

Tandon, S. K., D. K. Saxena, J. S. Gaur, and S. V. Chandra. 1978. Comparative toxicity of trivalent and hexavalent chromium. Alterations in blood and liver. Environ. Res. 15:90-99.

Taylor, F. G., Jr. 1980. Chromated cooling tower drift and the terrestrial environment: a review. Nuclear Safety 21:495-508.

Taylor, F. G., S. R. Hanna, and P. D. Parr. 1979. Cooling tower drift studies at the Paducah, Kentucky gaseous diffusion plant. U.S. Dep. Ener. Conf. 790109-1, Oak Ridge National Lab. 31 pp.

Taylor, F. G., L. K. Mann, R. C. Dahlman, and F. L. Miller. 1975. Environmental effects of chromium and zinc in cooling-water drift. Pages 408-425 *in* Cooling Tower Environment. 1974. U.S. Ener. Res. Serv. Admin.,

Conf. Rept. 740302, Washington, DC.

Taylor, F. G., Jr., and P. D. Parr. 1978. Distribution of chromium in vegetation and small mammals adjacent to cooling towers. J. Tenn. Acad. Sci. 53:87-91.

Taylor, F. G., J. A. Watts, and P. D. Parr. 1983. Seasonality of cooling tower drift in vegetation at ORGDP. Environ. Int. 9:149-152.

Tennant, D. A., and W. D. Forster. 1969. Seasonal variation and distribution of 65-Zn, 54-Mn, and 51-Cr in tissues of the crab *Cancer magister* Dana. Health Phys. 18:649-659.

Tietjen, J. H. 1980. Population structure and species composition of the free-living nematodes inhabiting sands of the New York Bight Apex. Estuar. Coast. Mar. Sci. 10:61-73.

Tong, S. S. C., W. D. Youngs, W. H. Gutenmann, and D. J. Lisk. 1974. Trace metals in Lake Cayuga lake trout (*Salvelinus namaycush*) in relation to age. J. Fish. Res. Board Canada 31:238-239.

Towill, L. E., C. R. Shriner, J. S. Drury, A. S. Hammons, and J. W. Holleman. 1978. Reviews of the environmental effects of pollutants: III chromium. U.S. Environ. Protection Agency Rep. 600/1-78-023. 287 pp.

Turekian, K. K., and M. R. Scott. 1967. Concentrations of Cr, Hg, Mo, Ni, Co, Mn in suspended material of streams. Environ. Sci. Technol. 1:940-942.

Uyeki, E. M., and A. Nishio. 1983. Antiproliferative and genotoxic effects of chromium on cultured mammalian cells. J. Toxicol. Environ. Health 11:227-235.

Van As, D., H. O. Fourie, and C. M. Vlegaar. 1973. Accumulation of certain trace elements in the marine organisms from the sea around the Cape of Good Hope. Pages 615-624 *in* Radioactive contamination of the marine environment. Int. Atom. Ener. Agen. (Vienna).

Van der Putte, I., J. Lubbers, and Z. Kolar. 1981a. Effect of pH on uptake, tissue distribution and retention of hexavalent chromium in rainbow trout (*Salmo gairdneri*). Aquatic Toxicol. 1:3-18.

Van der Putte, I., M. A. Brinkhorst, and J. H. Koeman. 1981b. Effect of pH on the acute toxicity of hexavalent chromium to rainbow trout (*Salmo gairdneri*). Aquatic Toxicol. 1:129-142.

Van der Putte, I., and P. Part. 1982. Oxygen and chromium transfer in perfused gills of rainbow trout (*Salmo gairdneri*) exposed to hexavalent chromium at two different pH levels. Aquatic Toxicol. 2:31-45.

Van Hoof, F., and M. Van San. 1981. Analysis of copper, zinc, cadmium and chromium in fish tissues. A tool for detecting metal caused fish kills. Chemosphere 10:1127-1135.

van Weerelt, M., W. C. Preiffer, and M. Fiszman. 1984. Uptake and release of ^{51}Cr (VI) and ^{51}Cr (III) by barnacles (*Balanus* sp). Mar. Environ. Res. 11:201-211.

White, D. H., K. A. King, and R. M. Prouty. 1980. Significance of organochlorine and heavy metal residues in wintering shorebirds at Corpus Christi, Texas, 1976-77. Pestic. Monitor. J. 14:58-63.

Wiemeyer, S. N., T. G. Lamont, and L. N. Locke. 1980. Residues of environmental pollutants and necropsy data for eastern United States ospreys, 1964-1973. Estuaries 3:155-167.

Yamaguchi, S., K. Sano, and N. Shimojo. 1983. On the biological half-time of hexavalent chromium in rats. Industr. Health 21:25-34.

Young, D. R., G. V. Alexander, D. McDermott-Ehrlich. 1979. Vessel-related contamination of southern California harbours by copper and other metals. Mar. Pollut. Bull. 10:50-56.

Young, D. R., and D. J. McDermott. 1975. Trace metals in harbor mussels. Ann. Rept. S. Calif. Coast. Water Res. Proj., El Segundo, CA:139-142.

Zaroogian, G. E., and M. Johnson. 1983. Chromium uptake and loss in the bivalves *Crassostrea virginica* and *Mytilus edulis*. Mar. Ecol. Prog. Ser. 12:167-173.