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The accumulation of metal contaminants in fishes of the Red River, New Mexico: Task 1 Literature review and Task 2 residue analyses.

Completion Report
for
New Mexico State Office, Bureau
of Land Management

by

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INTRODUCTION

The Red River, one of the larger trout streams in northeastern New Mexico, ranks second to the Pecos River in the number of trout planted annually by state fish hatcheries and ranks as one of the most important trout fisheries within the state. The stream originates in high mountainous terrain on the east slope of Wheeler Peak at about 3350 M elevation. Three small tributaries converge to form the main stream while four additional fish-inhabitated tributaries add water to the Red River prior to its junction with the Rio Grande, 11 kilometers southeast of Questa, New Mexico. The overall length of the Red River is about 51 km and the average gradient 27 m per km. The tributary streams provide good cutthroat fishing while the lower section of the main stream is excellent naturally-reproducing brown trout water. The middle sections receive the greatest fishing pressure and are heavily stocked with rainbow trout. The majority of the Red River Drainage lies in the Carson National Forest and is open to public fishing.

The lower 6.5 km of the Red River and 77 km of the Rio Grande, from the Colorado state line to New Mexico highway 96 southwest of Taos, have been designated Wild and Scenic Rivers under P.L. 90-542. The Bureau of Land Management is charged to administer the parts of these rivers under their control "in such a manner as to protect and enhance the values which caused it to be included in said system" without limiting other uses of the river that do not substantially interfere with public use and enjoyment of these values.

A large open pit molybdenum mine and mill is in operation between the towns of Questa and Red River, New Mexico. A tailings pipeline 13.7 km in

length runs from the mill along the banks of the Red River and terminates in a series of large diked ponds situated on a mesa above the Red River about 1.6 km west of Questa (Fig. 1). The decanted water finally flows through a small holding pond and down a rocky draw into the Red River. The river also receives water from natural springs in the vicinity of the settling ponds. There have been numerous accidental spills of raw tailings directly into the river due to leaks and breaks in the pipeline.

Operation of the mine and discharge of water from the mill effluent into the Red River is thought to have increased the levels of some metals in the river but the effects, if any, on aquatic resources are not known. A cooperative agreement was entered into by the Bureau of Land Management and the U.S. Fish and Wildlife Service to determine the contaminant levels that organisms are being exposed to, if bioconcentration of cadmium, copper, zinc or molybdenum is occurring and if the ambient concentrations present a current or potential hazard to continued fishery production in the Red River.

LITERATURE REVIEW

Potentially every element has a biologic function. As discussed by Vallee and Ulmer (1972), the functional requirements can only be determined by first totally eliminating the element in question from the animals diet and water and then performing controlled experiments to measure the response to increasing levels. Every element is potentially toxic when taken up in a high enough concentration.

A literature search was conducted for information on the uses and effects of cadmium, copper, zinc, molybdenum and cyanide on salmonid fishes and to determine the background levels of these metals in fish tissue. Cyanide is included because it is used in the floculation process in the mill and is released with the tailings water and some samples taken at the Pope Lake outfall have shown elevated cyanide levels.

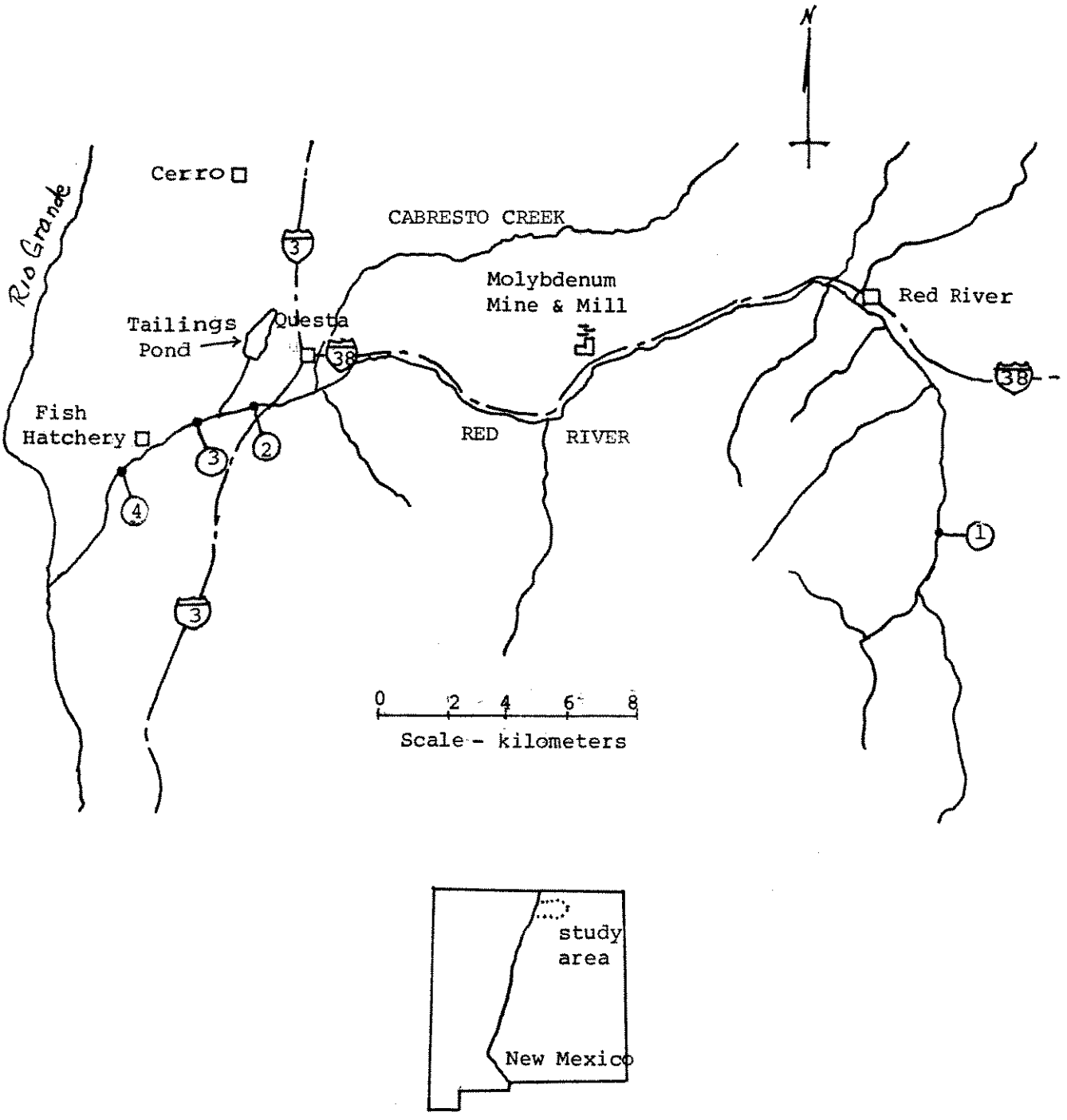

Figure 1. Location of study area showing sampling sites, molybdenum mine and mill and tailings pond.

Cadmium

Cadmium is a relatively rare metal which occurs chiefly in nature as a sulfide salt. Cadmium is obtained as a byproduct of the zinc, copper and lead producing industries. Its primary uses include electroplating, plastic stabilizers, batteries and pigment production. The predominant sources of pollution are smelters, wastes from electroplating plants, pigment works, textile and chemical industries, municipal waste water discharge and sludge disposal. It always occurs with zinc and varies from 0.1 to 5.0 percent of the amount of zinc present (Smith 1975). Boyle and Jonasson (1979) found that the cadmium content of Canadian natural waters normally ranged from <0.01 - 5 µg/l but up to 1140 µg/l near cadmium deposits. Typically, natural waters in the U.S. contain less than 1µg/l (Fleisher, et al. 1974, Hammons, et al. 1978). Cadmium may precipitate or be adsorbed on particulate surfaces as Gardiner (1974) determined it has adsorption concentration factors of 5000-50,000. The major portion of cadmium in water is found on suspended particles and sediments (Smith 1975).

Biologically, cadmium is a nonessential, nonbeneficial element of high toxic potential (EPA 1976). Cadmium readily reacts with sulfhydryl groups and may compete, especially with zinc, for binding sites on proteins, thus inhibiting a variety of enzymatic reactions (Hammons, et al. 1978). It also binds to cell membranes and may cause alterations in membrane integrity with resulting effects on cellular metabolism (Mustafa, Cross and Tyler, 1971 as cited in Hammons, et al. 1978). Cadmium enhances activity of no less than 25 enzymes *in vitro* and/or *in vivo* and inhibits an even greater number (Kennedy 1973). Metallothioneins are produced, especially in the liver and kidney, in response to exposure to several metals, mainly cadmium and zinc (Hammons, et al. 1978). However, metallothionein bound cadmium is more toxic to the

kidneys than cadmium salts because it is readily taken up by the kidneys whereas inorganic cadmium is not (Nordberg, et al., 1975, cited in Hammons, et al. 1978).

Cadmium is absorbed through both the gills and digestive tract. Cadmium obtained from food is rapidly eliminated, but when it is absorbed from water it has a long biological half-life so that its concentration in the body increases with age, therefore acting as a cumulative poison (Hammons, et al. 1978). Williams and Giesy (1978) also found cadmium to be a cumulative poison and determined that most accumulation occurs through the gills as food was a significant source of cadmium only after 8 weeks of treatment. Higher ambient concentrations result in higher tissue concentrations but an equilibrium is reached after 8-20 weeks of exposure (Mount and Stephan 1967, Eaton 1974, Cearley and Coleman 1974, Benoit, et al. 1976). Cadmium accumulates mostly in the gills, liver and kidney (Mount and Stephan 1967, Sangalang and Freeman 1979, Roberts, et al. 1979, Lorz, et al. 1978). Benoit, et al. (1976) found that cadmium loss from gill tissue of trout placed in control water was rapid but no loss was detectable from the liver and kidney. Phillips and Russo (1978) suggest the kidney is the route of elimination. Cadmium concentrations in whole freshwater fish tissue from NPMP collections had a range of 0.01 - 1.04 µg/g and 85% of the fish contained residues of 0.11 (May and McKinney, In press).

Acute toxicity data for salmonids are summarized in Table 1. Mount and Stephan (1967) suggest that death results when a threshold level of cadmium in the gills is reached. McCarty, et al. (1978) noted a biphasic mortality curve which they attributed to two modes of lethality. The first phase is attributed to reduction of gas exchange efficiency while the second phase results from cadmium absorption and subsequent biochemical reactions. Bilinski and Jonas (1973) found that the oxidation of lactate

by gills was reduced over 50% in fish surviving 1.12 mg/l, $CdCl_2$ for 24 hours. At lower concentrations this effect was not detected although mortality did occur. In fish exposed to 50 mg/l cadmium in sea water Gardner and Yevich (1970) found pathological changes in the intestinal tract after one hour, the kidney after 12 hours and in the gill filaments and respiratory lamellae after 20 hours, while the abundance of eosinophils increased steadily after 4 hours to approximately 45% above normal with a reduction in the relative numbers of mature eosinophils. After 24 hours in 0.025 mg/l Cd in softwater Sangalang and O'Halloran (1973) found that the testes of mature brook trout (Salvelinus fontinalis) showed marked discolorations and extensive hemorrhagic necrosis and disintegration of lobule-boundary cells. At 0.010 mg/l these effects were less extensive. Eisler and Gardner (1973) found that male mummichog (Fundulus heteroclitus) exposed to 10 mg/l Cd always contained lesions in the proximal tubules. Sullivan, et al. (1978) exposed fathead minnows (Pimephales promelas) to sublethal levels of cadmium prior to interaction with largemouth bass (Micropterus salmoides). This resulted in increased predation on the exposed minnows. This was attributed to altered behavior, including abnormal schooling behavior. Bengtsson, et al. (1975) reported spinal deformities and vertebral fractures in minnows (Phoxinus phoxinus) exposed to cadmium. Fractures resulted from cadmium ions prolonging the muscle action potential, causing muscles on both sides of the fish to be contracted simultaneously thus overloading the vertebrae. Fish in all concentrations were affected somewhat with those in 0.06 mg/l having a high of 67% injuries, but at higher concentrations the fish may die before the damage becomes apparent. Beattie and Pascoe (1978) also noticed deformed vertebral columns in fish exposed to Cd. Birge, et al. (1977) reported substantial frequencies of mortality and teratogenesis in rainbow trout (Salmo gairdneri) eggs cultured over cadmium enriched sediments.

Cadmium in the sediments was substantially more toxic to eggs and embryos than to free swimming larvae or fry. In hard water, Sauter, et al. (1976) found total length was significantly reduced in brook trout after 30 days at exposure $\geq$ 0.021 mg/l. After 60 days total length and wet weight were reduced by concentrations $\geq$ 0.012 mg/l. Kearns and Atchison (1979), in results from polluted and unpolluted parts of a lake, showed a significant correlation between cadmium concentrations and RNA-DNA ratios, indicating that as the cadmium content in fish increased, growth (RNA-DNA ratio) decreased. Benoit, et al. (1976) and Christensen (1975) also reported reductions in growth from cadmium. Christensen (1975) and Christensen, et al. (1977) noted changes in several biochemical factors of brook trout that related to cadmium exposure. Similarly, Merlini (1978) found that cadmium interferes with the normal physiological process of storing vitamin B_{12} in the liver, causing depletion of reserves and eventually anemia. Roch and Maly (1979) noticed that the rate of decline of plasma calcium concentrations was correlated with the rate of mortality in rainbow trout. They suggest that cadmium causes disturbances in ion regulation resulting in hypocalcemia. The effects of hypocalcemia, ie hypersensitivity, rhythmic muscle contractions and tetany, have been reported by other researchers (Clearly and Coleman 1974, Servizi and Martens 1978, Pickering and Gast 1972, Bengtsson, et al. 1975). Benoit, et al. (1976) noted that male brook trout seemed more susceptible to these effects than females because at 0.034 mg/l they became hyperactive while spawning and died.

Cadmium toxicity might be influenced by a number of factors. Chapman (1978b) working with steelheads (Salmo gairdneri) and chinook salmon (Oncorhyncus tshawytscha) found newly hatched alevins of both species were more resistent to cadmium than later juveniles, but there was a slight increase in tolerance as fish developed through the swim-up, smolt, and parr

stages. Eaton, et al. (1978) found embryos of several salmonid species to be more tolerant than larvae or juveniles. Other researchers finding similar effects include Servizi and Martens (1978), and Lorz, et al. (1978). Christensen (1975) noted a difference in susceptability between embryos and alevins of brook trout and suggested that toxic materials affect their vital processes when the animals are undergoing changes from embryo to alevin to fry. Loss of protective embryonic coat, use of new tissues, and development of internal mechanisms may contribute to the greater sensitivity of the later alevins and juvenile stages.

Roch and Maly (1979) report that temperature has a marked effect on cadmium toxicity as they found that cold adapted rainbow trout exposed to lethal concentrations survived longer and could resist greater concentrations than could warm adapted trout. Lloyd (1962) contends, however, that temperature only effects survival time. Chapman (1973) contends that the effect of temperature, as well as pH, hardness, alkalinity, and dissolved oxygen content, on cadmium toxicity relates to their effects on metabolic rates.

Kinkade and Erdman (1975) showed water hardness to have an effect on both rate of uptake and total tissue residues of cadmium. Fish in soft water had higher total cadmium concentrations than those in hard water. Zitko and Carson (1976) determined that neither Ca or Mg ions affect the lethality of cadmium through competition for active binding sites in fish tissues, apparently because of specific binding sites for cadmium. Carroll, et al. (1979) tested the effects of various hardness components on cadmium toxicity and found that calcium ions were the major source of protection while magnesium and the carbonate system provide little or no protection. These results are not necessarily contradictory to Zitko and Carson's findings as the mechanisms may not be the same. Besides its effect on ventilatory rate, pH also determines the solubility of toxic forms of cadmium (McCarty, et al.

1978).

The effects of cadmium in combinations with other metals varies. Eaton (1973) found that in chronic tests using a mixture of copper, cadmium and zinc, that the effects of cadmium were reduced. However, in acute tests the effects of all three metals were enhanced. Lorz, et al. (1978) reported that an apparent synergistic effect of a copper/cadmium mixture adversely affected downstream migration and gill (NA+K) - ATPase activity. Spehar, et al. (1978) found the chronic toxicity of a zinc/cadmium mixture was little, if any, greater than the toxicity of zinc alone. They determined that cadmium and zinc did not act additively at sublethal concentrations when combined as mixtures; however, a joint action of the toxicants was indicated. Also, the accumulation of one metal was not affected by the presence of the other. Eisler and Gardner (1973) found sublethal levels of Cd/Zn and Cd/Cu mixtures produced lesions in the proximal tubules similar to that documented following acute Cd poisoning. They concluded that fish intoxicated by Cu, Zn, or both, are more sensitive to levels of Cd that were not normally lethal.

Thurston et al. (1979) recommended a sliding scale based on hardness for maximum soluble cadmium concentration in freshwaters. For the Red River in New Mexico the maximum allowable cadmium concentration is from 2.5-6.0 µg/l. This is lower than the acute toxicities for salmonids given in Table 1 for hard waters and therefore direct cadmium toxicity should not result if cadmium concentrations are kept below these levels. It is also slightly below the levels of reported sublethal effects discussed earlier so it should represent "no effect" levels for known adverse effects. All water quality measurements from Oct. 1978 to Sept. 1980 were below these levels.

Table 1 . Acute toxicity of cadmium to salmonids.

Species	Age	Acute Cd Conc. ppb	Test	pH	Hardness mg/l	Temp. c	D.O. mg/l	Reference
Coho salmon (Oncorhynchus kisutch)	juvenile	10.4	96 h LC50	7.59	90	10	9.9	Lorz et al. 1978
Coho salmon	adult	2.6-5.0[b]	215 h LC50	7.3	22	10.0	9.8	Chapman and Stevens 1978
Chinook salmon (Oncorhynchus tshawytscha)	alevin	>26	96 h LC50 & 200 h LC50	7.3-7.5	23	11.6-12.8	10.2	Chapman 1978b
Chinook salmon	swim up fry	1.8 1.6	96 h LC50 200 h LC50	7.3-7.5	23	11.6-12.8	10.2	Chapman 1978b
Chinook salmon	parr	3.5 2.0	96 h LC50 200 h LC50	7.1	23	11.6-12.8	10.2	Chapman 1978b
Chinook salmon	smolt	>2.9 2.3	96 h LC50 200 h LC50	7.1	23	11.6-12.8	10.2	Chapman 1978b
Rainbow trout (Salmo gairdneri)	2 months	6.6	96 h LC50	6.4-8.3	82-132[a]	---	4.8-9.0	Hale 1977
Rainbow trout	38 mm 1.6 g	2.85	96 h LC50	6.65	12	16	8.8	Goettl and Davies 1975
Rainbow trout	---	8-15	14 d LC50	---	290	11-12.5	---	Ball 1967 in Chapman 1973
Steelhead trout (Salmo gairdneri)	---	0.95	96 h LC50	7.4	20-25	12	---	Chapman 1973
Steelhead trout	adults	2.9-4.95	17 d LC50	7.3-7.65	29-90	6.7-11.9	---	Chapman 1973

Table 1 continued.

Species	Age	Acute Cd Conc. ppb	Test	pH	Hardness mg/l	Temp. c	D.O. mg/l	Reference
Steelhead trout	adult	2.8-8.5[b]	408 h LC50	7.5	54	9.6	10.7	Chapman and Stevens 1978
Steelhead trout	alevin	>27	96 h LC50 & 200 h LC50	7.1	23	11.6-12.8	10.2	Chapman 1978b
Steelhead trout	swim up fry	1.3	96 h LC50 & 200 h LC50	7.1	23	11.6-12.8	10.2	Chapman 1978b
Steelhead trout	parr	1.0 0.9	96 h LC50 200 h LC50	7.3-7.5	23	11.6-12.8	10.2	Chapman 1978b
Steelhead trout	smolt	>2.9 1.6	96 h LC50 200 h LC50	7.1	23	11.6-12.8	10.2	Chapman 1978b

[a] alkalinity mg/l

[b] 95% confidence intervals

Copper

Copper is relatively common in natural waters. Kopp and Kroner (1967 in EPA 1976) examined over 1500 surface water samples from the U.S. and found soluble copper in 74% of the samples with an average concentration of 15 mg/l and a maximum concentration of 280 mg/l. High concentration of Cu may occur in water from mines or in water that has leached ore dumps or mill tailings. Cu contents of ground and surface water in areas where ore bodies occur may be rather large. Cu is used extensively in water treatment and in fabrication of pipe, valves and pumping equipment. Copper is an essential element in animal metabolism. It is used in enzymes and is important in the synthesis of hemoglobin. Waiwood and Beamish (1978) found that elevated Cu levels reduced metabolism; possibly by inhibition of oxygen transfer or transport and reduced efficiency of gas exchange. They also found that it increased energy expenditure by osmotic stress and nervous impairment which reduces efficiency.

Copper is known to accumulate in fish. Servizi and Martens (1978) found that Cu was concentrated in eggs, alevins, and fry in proportion to exposure concentrations. Nehring (1976) also found this to be true for mayflies (Ephemerella grandis) and stoneflies (Pteronarcys californica). O'Rear (1971) determined that the Cu concentration in striped bass (Morone saxatilis) reached an equilibrium after the fish reached 15 g. Patrick and Loutit (1978) noticed that young fish seemed to accumulate more copper than older fish. They also showed that fish can concentrate Cu when fed Cu enriched tubifex worms, but the increased levels were not noticed until 4 days after the initial feeding. The gills, kidneys and liver are the predominant sites of Cu accumulation in the body (Benoit 1975, Phillips and Russo 1978). In fish samples collected for the National Pesticide

Monitoring Program, 85% of the residue levels in whole fish were below 1.3 µg/g (May and McKinney, In press).

Copper toxicity is related to the amount of soluble copper in the water (Shaw and Brown 1974, Brungs, et al. 1976) Andrew (1976) found that Cu precipitates were not biologically active, and that soluble Cu complexes of $CuCO_3$ and Cu-NTA were much less toxic than ionic Cu. He hypothesized that the increasing toxicity of cupric ion at high pH is due to interactions with proteins or enzymes containing sulfhydryl groups. Waiwood and Beamish (1978a and 1978b) found Cu^{2+} and $CuOH^{+}$ to be the major toxic species while Chakoumakos, et al. (1979) found that $Cu(OH)_2$ also contributed to toxicity. Table 2 summarizes the toxicity data for copper.

Copper has been shown to affect growth. Pickering, et al. (1977), Hazel and Meith (1970), Sauter, et al. (1976), and Horning and Neiheisel (1979) all reported significantly less growth increases in fish exposed to copper when compared to controls. Waiwood and Beamish (1978b) and Lett, et al. (1976) report Cu initially reduced the growth rate of fish but rates gradually returned to normal. Thus, the initial reduction in growth rate may also be responsible for the inhibited growth in the first examples. In both of these studies as well as Drummond, et al. (1973) and Richey and Roseboom (1978) it was noted that copper caused an initial cessation or reduction of feeding activity but that feeding gradually returned to normal. However, Waiwood and Beamish (1978b) found that decreased growth rates also occurred in copper concentrations that did not affect appetite. They attributed the decrease in growth rate to decreased efficiency of energy utilization and increased maintenence requirements caused by copper.

levels, synergism was indicated. Broderius and Smith (1979) determined a Zn/HCN mixture to be supra-additive at lethal levels but no interaction was noticed at sublethal levels. Herbert and Van Dyke (1964) found a Zn/phenol mixture exhibited additive toxicity. Herbert and Shurben (1964) got the same results in a Zn/ammonia mixture in hardwater, but at low dissolved oxygen concentrations, additivity was not as evident. Brown, et al. (1969) found that the 48h LC50 of Zn, ammonia, and an ammonia/Zn mixture were similar regardless of whether the exposure concentrations were constant or fluctuated within 50% of the 48h LC50. This held as long as the periodicity of the fluctuations did not exceed the resistence time (period of exposure after which irreversible damage occurs) for the toxicants involved. The toxicity of the mixture was additive except that when Zn comprised more than 70% of the mixture, antagonistic effects were apparent. Wedemeyer (1968, cited in Phillips and Russo 1978) showed Cu concentrations below 2 mg/l inhibited Zn uptake by coho salmon eggs but above 2 mg/l, Cu stimulated accumulation.

No effect levels for the Red River are below 50-300 µg/l total zinc based on a sliding scale depending on hardness proposed in Thurston et al. (1979). Brungs (1969) however, found reduced egg production at 180 µg/l in waters of similar hardness. These levels should produce no direct toxicity however. Water quality samples show these levels for total zinc are exceeded periodically, particularly during the spring.

Table 3 . Acute toxicity of Zn to salmonid fish.

Species	Age	Acute Zn Conc. ppb	Test	pH	Hardness mg/l	Temp. c	D.O. mg/l	Reference
Atlantic salmon (Salmo salar)	---	600	7 d LC50	7.1-7.5	20	15	---	Sprague 1964
Atlantic salmon	---	340-1600	21 d LC50	6.1-7.1	12.1-24.4	10	---	Farmer et al. 1979
Brook trout (Salvelinus fontinalis)	juvenile	1550-2420	96 h LC50	7.38-7.63	44.4-47.0	14.9-15.2	---	Holcombe and Andrew 1978
Brook trout	juvenile	4980-6980	96 h LC50	7.17-7.41	169.7-179.0	14.8-15.7	---	Holcombe and Andrew 1978
Brook trout	---	960	14 d TL50	7.3	34-54[a]	8.6-12.0	7.5	Niehring and Goettl 1974
Brown trout (Salmo trutta)	---	640	14 d TL50	7.2	22-55	7.0-7.8	8.5	Niehring and Goettl 1974
Chinook salmon (Oncorhyncus tshawytscha)	alevin	>661	96 h LC50 & 200 h LC50	7.3-7.5	23	11.6-12.8	10.2	Chapman 1978b
Chinook salmon	swim up	97	96 h LC50 200 h LC50	7.3-7.5	23	11.6-12.8	10.2	Chapman 1978b
Chinook salmon	parr	463 395	96 h LC50 200 h LC50	7.1	23	11.6-12.8	10.2	Chapman 1978b
Chinook salmon	smolt	701 364	96 h LC50 200 h LC50	7.1	23	11.6-12.8	10.2	Chapman 1978b
Coho salmon (Oncorhyncus kisutch)	yearling	4600	96 h LC50	6.8-7.9	89-99	10-12	6.0-10.8	Lorz and McPherson 1976

Table 3 continued.

Species	Age	Acute Zn Conc. ppb	Test	pH	Hardness mg/l	Temp. c	D.O. mg/l	Reference
Coho salmon	---	636-1211[b]	96 h LC50	7.4	25	13.7	9.8	Chapman and Stevens 1978
Cutthroat trout (Salmo clarki)	---	670	14 d TL50	7.2	22-58	8.3-8.6	8.5	Niehring and Goettl 1974
Cutthroat trout	---	90	96 h LC50	6.6-7.6	17-32[c]	11.5-16.0	---	Sappington 1969 (in Chapman 1973)
Rainbow trout (Salmo gairdneri)	eyed eggs	2720	96 h TL50	6.8	26	11.0	6.8	Sinley et al. 1974
Rainbow trout	juvenile	7210	96 h TL50	7.81	333	15.0	6.8	Sinley et al. 1974
Rainbow trout	juvenile	430	96 h TL50	6.80	26	15.0	6.8	Sinley et al. 1974
Rainbow trout	---	135	120 h TL50	6.80	26	12.7	6.8	Sinley et al. 1974
Rainbow trout	---	1560-2800	24 h LC50	7.6	36	5-18	---	Cairns et al. 1978
Rainbow trout	1 year	4000	48 h LC50	7.3-7.5	240	15.3-18.4	~90% sat.	Brown and Dalton 1970
Rainbow trout	juvenile	370-756	96 h LC50	7.38-7.63	44.4-47.0	14.9-15.2	---	Holcombe and Andrew 1978
Rainbow trout	juvenile	1910-2960	96 h LC50	7.17-7.41	169.7-179.0	14.8-15.7	---	Holcombe and Andrew 1978

Table 3 continued.

Species	Age	Acute Zn Conc. ppb	Test	pH	Hardness mg/l	Temp. c	D.O. mg/l	Reference
Rainbow trout	---	410	14 d TL50	7.3	20-51	7.7-8.0	7.8	Niehring and Goettl 1974
Rainbow trout	---	240-560	96 h LC50	7.2-7.3	30	14-15	---	Colorado Game Fish & Parks (in Chapman 1973)
Rainbow trout	---	410-830	96 h LC50	6.6-7.1	30	6-10	---	Colorado Game Fish & Parks (in Chapman 1973)
Steelhead trout (Salmo gairdneri)	---	110	96 h LC50	7.4	20-25	12	---	Chapman 1973
Steelhead trout	adult	1499-16731[b]	96 h LC50	7.45	83	10.3	10.4	Chapman and Stevens 1978
Steelhead trout	alevin	815 555	96 h LC50 200 h LC50	7.1	23	11.6-12.8	10.2	Chapman 1978b
Steelhead trout	swim up	93	96 h LC50 & 200 h LC50	7.1	23	11.6-12.8	10.2	Chapman 1978b
Steelhead trout	parr	136 120	96 h LC50 200 h LC50	7.3-7.5	23	11.6-12.8	10.2	Chapman 1978b
Steelhead trout	smolt	>651 278	96 h LC50 200 h LC50	7.1	23	11.6-12.8	10.2	Chapman 1978b
Sockeye salmon (Oncorhyncus nerka)	8 month	749	96 h LC50	7.3	22	12.4	10.7	Chapman 1978a
Sockeye salmon	---	1100	96 h LC50	7.4	20-25	12	---	Chapman 1973

a=alkalinity mg/l b=95% confidence intervals c=methyl orange alkalinity

Cyanide

Free cyanide occurs only rarely in nature because of the high reactivity of the molecule and therefore, cyanide content of natural waters is usually low (Towill, et al 1978). Cyanide occurs in water as free cyanide (HCN or CN^-), simple cyanide salts (NaCN, etc.), and complex cyanides. Major pollution sources include ore extraction and milling processes, coal-coking furnaces, electroplating, petrochemical industries, photographic processing, paint manufacture and the steel industry.

Cyanide is absorbed mainly through the gills and intestinal tract and is a non-cumulative poison that inhibits aerobic respiration and enzymatic activity by binding to the metallic cofactor in metalloenzymes (Towill, et al. 1978). Cytochrome oxidase is particularly sensitive to cyanide, being completely inhibited by 3.3×10^{-8} moles/ml of cyanide. This inhibition prevents further oxidation and reduction reactions and results in histotoxic anoxia (DePalma 1971, cited in Towill, et al. 1978). Death probably results from anoxia of the central nervous system. Cyanide can also bind reversibly with methaemoglobin (Chen and Rose 1952, cited in Towill, et al. 1978). Only undissociated HCN inhibits the consumption of oxygen in the tissues (EPA 1976).

The major detoxification pathway is the reaction of cyanide with thiosulfate in the presence of the enzyme rhodanese to produce thiocyanate (Lang 1933a; 1933b, cited in Towill et al., 1978). Several other minor detoxification pathways exist also.

Some acute toxicity bioassay data are given in Table 4. Leduc (1978) found that exposure of Atlantic salmon to sublethal levels of cyanide delayed hatching, reduced hatching success and the conversion of yolk to body tissue, and increased teratogenesis. Leduc and Chan (1975) found

that exposure of juvenile rainbow trout to sublethal cyanide levels caused 4-8% changes in blood plasma osmolality and chloride ion levels when the fish were transferred to seawater. They considered this evidence of serious physiological impairment that would have costly energetic implications and speculated that a reduction in kidney tubular electrolyte reabsorption induced by cyanide caused the changes. Koenst, et al. (1977) reported that for brook trout, the number of eggs produced per female was significantly reduced after exposure to 0.0112 mg/l HCN. Growth of embryos and juveniles was significantly less in cyanide concentrations of 0.033 mg/l and above compared to controls. However, cyanide did not effect the growth of adults. Ruby, et al. (1979) determined that short-term sublethal exposures of juvenile rainbow trout to cyanide may cause permanent damage to the fixed number of spermatogonia in the testis, reducing the reproductive potential. They found that cyanide blocked the mitotic process resulting in a reduced number of dividing spermatogonia and that formation of the mitotic spindle was also affected resulting in multipolar spindles and multinucleate interphase cells. A high incidence of necrosis in spermatogonia occurred at higher concentrations. Kimball, et al. (1978) found spawning in bluegills (Lepomis macrochirus) was completely inhibited at 0.052 mg/l in hardwater. Other sublethal effects found by Doudoroff (1976) in his literature review include avoidance reactions, impaired swimming ability, acceleration of the loss of energy reserves after periods of starvation, reduced rate of oxygen consumption, reduced food conversion efficiency, increased rate of food consumption and adverse affects on osmoregulation, respiration, heart beat, and embryonic development. He also determined that while growth may be affected for short periods of time, cyanide has no overall effect on growth.

Broderius, et al. (1977) determined that a pH increase from 6.8 to 8.3

had no effect on the toxicity of cyanide. However, from 8.3 to 9.3 the 96h - LC50 for free cyanide ($HCN+CN^-$) increased while that for molecular HCN decreased. The apparent increase in toxicity of molecular HCN was believed to result from the CN^- ions penetrating the gill epithelium, albeit less readily than do molecular forms, and enhancing the toxicity of the solution. Doudoroff (1976) noted a trend of decreasing toxicity with increasing pH and concluded that molecular HCN is more toxic than CN^-. Smith and Heath (1979) showed that temperature had no effect on the actual lethal concentration of cyanide. However, through its effect on metabolic rate, temperature may affect survival time, i.e. increased temperature increases the metabolic rate and decreases survival time (Towill, et al. 1978). This is in contrast to the findings of Smith, et al. (1979) who found that for bluegill and brook trout the 96h LC50 increased with increasing temperature. It is generally agreed that low concentrations of dissolved oxygen can seriously depress the cyanide tolerance of fishes (Smith, et al. 1979, Doudoroff 1976). Doudoroff also reported that previous exposure to low, sublethal levels of cyanide can somewhat increase the resistence to lethal levels by setting in motion some adaptive mechanism. However, the adaptation can be largely or entirely cancelled or counterbalanced by concomitant injury when fish are exposed to higher sublethal levels. Smith, et al. (1979) found that sublethal exposures to HCN caused increased predation of fathead minnows (Pimephales promelas) by green sunfish (Lepomis cyanellus) but they could not determine whether the prey were easier to capture or if the sunfish had greater appetites. They also found that eggs were the most tolerant life history stage while fry and juveniles were least tolerant.

The effects of mixtures of cyanide with other pollutants varies with the pollutant. Broderius and Smith (1979) determined that zinc/cyanide and

ammonia/cyanide mixtures exerted a supra-addition effect while a chromium/cyanide mixture had a less than additive effect on survival. They noticed, however, that the sublethal stress imposed on fish in most binary mixtures studied disrupted normal physiological processes so that the relationships between concentration and reduction in growth were essentially equal to those when the toxicants were present alone. Doudoroff (1976) concluded that cyanide complexed with zinc or cadmium in wastewaters should be about as hazardous as free cyanide to fish due to cooperative joint action, or synergism with the zinc and cadmium ions. This is because there is almost complete dissociation of these complexes at pH levels commonly found in natural waters. He also found that while the acute toxicity of silver-cyanide and cuprocyanide complex anions is much less than that of molecular HCN it is not negligable. These ions can be the principal toxicants even in some very dilute solutions. For example, ferrocyanide and ferricyanide may photodecompose releasing toxic HCN. Nickelocyanide dissociates at the pH found in some processing waters.

The recommended upper limit for free cyanide ($HCN + CN^-$) is 5 µg/l (Thurston et al. 1979). This limit will prevent direct toxicity of aquatic organisms but may reduce production in sensitive organisms. The cyanide concentrations in the Red River were generally within the recommended limit, but occasionally exceeded them.

Table 4. Acute toxicity of HCN.

Species	Age	Acute HCN Conc. ppb	Test	pH	Hardness mg/l	Temp. c	D.O. mg/l	Reference
Brook trout (Salvelinus fontinalis)	egg	>212->242	96 h LC50	7.71-7.88	220	7.1-13.0	3.64-8.13	Smith et al. 1978
Brook trout	sac fry	108-518	96 h LC50	7.68-7.84	220	10.0-13.0	3.50-7.96	Smith et al. 1978
Brook trout	swim up fry	55.8-106	96 h LC50	7.73-7.85	220	7.0-13.0	3.90-8.02	Smith et al. 1978
Brook trout	juvenile	53.0-143	96 h LC50	7.74-8.06	220	4.0-18.0	4.02-9.26	Smith et al. 1978

METHODS

Water quality samples were collected at approximately monthly intervals from eight stations on the Red River and analyzed by the USGS lab in Denver, Colorado. Data were placed in the EPA STORET system and later retrieved electronically from a remote terminal. Fish were collected in the vicinity of four USGS gauging stations on the Red River. (1) Station 08264500. Fish were captured above a well constructed beaver dam which prevented up or downstream movement of fish. Because no fish had been stocked since the preceding summer, the rainbow trout had been exposed to natural levels of metals in the water for at least seven months. (2) Station 08266500, below the towns of Red River and Questa. At this station fish were blocked from downstream movement by a large dam composed of logs and debris. (3) Station 08266790, downstream of discharge from the effluent settling ponds. Movement of fish upstream into the area of this station is blocked, except at high flows, by a dam formed by the large pipes at the fish hatchery intake structure. (4) Station 08266820, approximately 2.5 km below the fish hatchery (Fig. 1). At each station fish were captured using a Smith-Root battery operated, back-pack mounted electrofisher. The experimental design was to collect three pooled samples of five fish each, of uniform size, of rainbow trout, Salmo gairdneri and brown trout, Salmo trutta, at each station. All fish were sorted, wrapped in aluminum foil, placed on ice and later frozen on dry ice and transported to the Victoria Field Station where thry were weighed, measured and further prepared for shipment to the Columbia National Fisheries Research Laboratory, Columbia, Mo., for analysis.

At CNFRL all samples remained frozen until prepared for analysis. Each fish was first sawed into blocks with a band saw and then ground in a Hobart meat grinder. All tools were cleaned with acetone and distilled water before each fish was processed. For composite samples each fish was

ground individually then all fish in that composite were mixed together and then ground again to achieve a homogeneous mixture. All samples were analyzed at the Columbia National Fisheries Research Laboratory using procedures given in May and McKinney (In press).

RESULTS AND DISCUSSION

A total of 171 fish were collected from the four stations. At station 1 three brook trout, Salvelinus fontinalis, and three cutthroat trout, Salmo clarki were collected and at station 2 four specimens of white sucker, Catostomus commersoni, were collected. Only 1 brown trout, Salmo trutta, was captured at station 1 and no rainbow trout, Salmo gairdneri, large enough to be individually analyzed were collected at station 4. So that valid comparisons between species and stations could be made only 41 specimens of rainbow and brown trout were utilized in the metal determinations. Rainbow trout analyzed ranged from 12.5 to 21 cm in length and from 24 to 142 g in weight. The brown trout were 15 to 27 cm long and from 40 to 232 g in weight. The sample size and tissues analyzed from each station are shown in Table 5.

The environmental concentrations of metals to which fish had been exposed were determined by averaging the concentration values as reported by USGA for the period 17 Oct. 1978 through 28 April 1980. Examination of the water quality data show that the level of dissolved molybdenum is elevated below the effluent settling basin discharge (Table 8). The dissolved zinc concentration in the water was highest at station 2, above the discharge point and the concentration in the water declined at stations 3 and 4 (Table 9). The Red River receives a large influx of groundwater (about 0.6 m^3/second) below station 2 and this reduces the concentration of metals in the river water. In addition, metal complexes might be formed due to the action of the settling basin effluent. The fish hatchery

adds organic matter to the river above station 4 and since metals are known to adsorb to particle surfaces (Gardiner 1974, Andrew 1976) they may be removed from solution below the hatchery further lowering the concentration in the water. A recent review of EPA water quality criteria (Thurston et al. 1979) recommends that for salmonid species living in water with the hardness of the Red River (about 50 to 300 mg/l as $CaCO_3$) that total zinc be not more than 0.05 to 0.1 mg/l. Water in the Red River exceeds this value for total zinc (USGS data print out). The reported values for cadmium levels are below the criteria suggested and Thurston et al. (1979) did not make recommendations for molybdenum or copper.

In Tables 6 through 9 comparisons are shown relating metal residue levels in fish tissue to the exposure concentration (amount of metal dissolved in water) for each metal at each station. Differences in residue levels between stations were determined by F tests on brown trout data from stations 2, 3 and 4. After consulting a statistician the data were transformed by converting each value to its square root to stabilize the variance. The results of these analyses are shown in Tables 10 and 11.

Table 5. Sample analysis matrix for data reported in Tables 8 through 11.

Station	1	2	3	4
Composite sample, whole rainbow trout. Number of individuals in composite	4	3	3	-
Individual whole brown trout	-	4	4	-
Individual brown trout carcass	-	4	4	2
Individual brown trout liver	-	4	4	2

Table 6. Cadmium residue levels in fish tissue from the Red River, New Mexico. Values are in µg/g wet weight. The water concentration values are for the dissolved fraction as reported by USGS.

Station	1	2	3	4
rainbow trout, composite	<0.01	0.06	0.16	-
brown trout, individual				
mean (SD)	-	0.13(.08)	0.13(.04)	-
range	-	0.06-0.20	0.10-0.18	-
brown trout carcass				
mean(SD)	-	.04(.02)	0.04(.01)	0.02(.006)
range	-	0.02-0.06	0.03-0.05	0.01-0.02
brown trout liver				
mean(SD)	-	0.91(.35)	0.49(.10)	0.31(.12)
range	-	0.41-1.18	0.38-0.61	0.23-0.40
water (µg/l)				
mean	<0.001	<0.001	<0.001	<0.001
max. reported value	<1	2	1	2

Table 7. Copper residue levels in fish tissue from the Red River, New Mexico. Values are in µg/g wet weight. The water concentration values are for the dissolved fraction as reported by USGS.

Station	1	2	3	4
rainbow trout composite	1.17	3.54	3.66	-
brown trout				
mean(SD)	-	4.20(1.6)	3.13(.66)	-
range	-	2.24-5.57	2.41-3.93	-
brown trout carcass				
mean(SD)	-	0.90(.24)	0.71(.12)	0.58(.11)
range	-	0.71-1.24	0.556-0.831	0.51-0.66
liver				
mean(SD)	-	179.5(55.2)	75.3(26.4)	62.6(48.0)
range	-	138-257	42.9-106.0	28.6-96.5
water (µg/l)				
mean	<0.010	<0.010	<0.010	<0.010
max. reported value	<10	<10	<10	<10

Table 8. Molybdenum residue levels in fish tissue from the Red River, New Mexico. Values are in µg/g wet weight. The water concentration values are for the dissolved fraction as reported by USGS.

Station	1	2	3	4
rainbow trout composite	<0.050(4)	0.09(3)	0.62(3)	-
brown trout				
mean(SD)	-	0.27(.12)	0.26(.07)	-
range	-	0.121-0.423	0.210-0.355	-
brown trout carcass and gills				
mean(SD)	-	0.06(.01)	0.07(.03)	0.10(.03)
range	-	<0.050-0.060	<0.05-0.107	0.084-0.120
liver				
mean(SD)	-	0.35(.10)	0.39(.06)	0.43(.05)
range	-	0.273-0.474	0.316-0.467	0.393-0.461
water (µg/l)				
mean	<7.8	10.3	128.4	132.9
max. reported value	<10	14	340	340

Table 9. Zinc residue levels in fish tissue from the Red River, New Mexico. Values are in µg/1 wet weight. The water concentration values are for the dissolved fraction as reported by USGS.

Station	1	2	3	4
rainbow trout composite	20(4)	23.3	37.1	-
brown trout				
mean(SD)	-	32.3(7.9)	34.4(2.4)	-
range	-	21.2-39.9	31.7-37.6	-
brown trout (4) carcass and gills				
mean(SD)	-	15.6(3.1)	15.2(1.1)	18.4(.1)
range	-	12.8-19.2	13.9-16.4	18.3-18.5
liver				
mean(SD)	-	39.6(6.5)	30.5(.7)	28.9(4.5)
range	-	30.1-44.9	29.6-31.3	25.8-32.1
water (µg/1)				
mean	9.3	28	12.9	11.9
max. reported value	40	50	20	20

Table 10. F values from analysis of variance of metal residues in tissue from brown trout collected at stations 2 and 3. Data were transformed to $\sqrt{x}$.

Metal	Brown Trout Tissue			
	Whole	Carcass	Liver	
Cadmium	0.012	0.14	5.17	*
Copper	1.34	2.15	13.85	**
Molybdenum	0.0001	2.04	0.39	
Zinc	0.32	0.02	5.52	*

** $F_{.05(1,6)} = 5.99$
* $F_{.1(1,6)} = 3.78$

Table 11. F values from analysis of variance of metal residues in tissue from brown trout collected at stations 2, 3 and 4. Data were transformed to $\sqrt{x}$.

Metal	Brown Trout Tissue			
	Carcass		Liver	
Cadmium	3.52	*	5.36	**
Copper	2.62		7.46	**
Molybdenum	4.11	*	0.66	
Zinc	1.55		5.24	**

** $F_{.05(2.7)} = 4.74$
* $F_{.10(2.7)} = 3.26$

Because of the location of samplings station, as previously described, fish were prevented from moving into an area of suspected higher metal concentration, therefore, the metal residues reported should be representative of the amount bioaccumulated from that section of the river.

Residue levels of all four metals in the composite samples of rainbow trout increased in fish from the downstream stations compared to station 1. In the individual whole brown trout samples the levels of cadmium, copper and molybdenum decreased slightly at station 3 compared to station 2 while zinc levels increased slightly. An analyses of variance showed that none of these changes were significant (Table 9). Comparisons of liver residues from stations 2,3 and 4 showed that there were statistically significant differences between stations in copper, cadmium and zinc residues in the liver tissue (Table 11). In each case, residues were highest at station 2 and lowest at station 4 (Tables 6,7,9). Comparison of residue levels from the brown trout carcass data shows that molybdenum and cadmium residue levels were statistically different between stations (Table 11). Cadmium residues were highest at station 2 and lowest at 4 while molybdenum was highest at station 4 and lowest at 2 (Tables 6,8).

A recent review of data on whole fish residue levels from the National Pesticide Monitoring Program (NPMP) uses the 85th percentile to identify stations having fish with "higher than normal" concentrations (May and McKinney, In press). The 85th percentile for cadmium, 0.11 mg/kg, was exceeded by rainbow trout from station 3 and by whole brown trout from stations 2 and 3 (Table 6). All whole fish from stations 2 and 3 exceeded the 85th percentile of 1.3 mg/kg for copper (Table 7). This means that residue levels in these fish exceeded levels found in more than 85% of the fish sampled from U.S. waters. Zinc residues in Red River fish are much lower than the 85th percentile of 76.0 mg/kg and molybdenum

was not measured in the NPMP samples.

Most previous studies of the effects of metals have concentrated on acute toxicity as determined in laboratory tests. There have been few studies of sublethal effects at low exposure levels and even fewer field studies where the effects of actual environmental exposures have been studied. Data from the literature review indicates that the low levels of cadmium dissolved in Red River water should not be directly toxic to salmonid fishes. Biochemical parameters that have been studied in the lab such as respiration, blood chemistry, reproductive functioning, growth and kidney function, were not affected at these low levels, however, a recent study showed that dissolved cadmium concentrations above 1 mg/l inhibited the activity of liver catalase in killifish (Pruell and Engelhardt 1980). Even though copper levels are, on the average, an order of magnitude higher than cadmium they are lower than "no effects" levels that have been reported in the literature and, alone, should not be toxic. Zinc concentrations in the Red River sometimes exceed levels that have been shown to inhibit egg production and spawning in non-salmonid fishes (Brungs 1969, Benoit and Holcombe 1978) but the effect on brown or rainbow trout is unknown. The molybdenum concentration in the Red River is elevated above background level. Not enough is known about the function of this element in fish to predict what effects might occur, but Molybdenum is known to interfere with the development of bones and other hard structures in mammals (Chappill et al., 1979).

Even though the concentrations of cadmium, copper, molybdenum and zinc in the water are low, salmonid fishes in the Red River are bioaccumulating these metals considerably, especially in liver tissue. This in contrast to McKim and Benoit (1974) who found no copper accumulation in brook trout at exposure concentrations up to 9.4 µg/l. A high level of metals in an

organ may not be a sign of biological malfunctioning, but there have been few studies that relate the accumulation of metals by an organ to the functioning of that organ. Pruell and Engelhardt (1980) report that after 96h exposure to 1 mg/l Cd that the liver had a concentration of 4 µg/g, exposure to 10 mg/l Cd gave a liver value of 10 µg/g and after exposure to 25 mg/l there was 42 µg/g Cd in the liver and liver catalase was inhibited at exposures above 1 µg/l. The liver is important to several different body systems and is the primary storage site for glycogen which is the carbohydrate metabolized for energy utilization in fish. Any impairment of the liver or its enzymes would be detrimental to the survival or functioning of the animal.

The results of this study indicate that levels of cadmium, copper, molybdenum and zinc in the Red River did not reach acutely toxic levels during the time period studied. However, the nature and extent of all the interactions of these metals and their toxicity are not known. Direct toxicity is not the only effect that could be acting upon aquatic populations in the Red River. Chronic low levels of these metals could be affecting the physiology of the animals and reducing their ability to function. It is recommended that further studies be undertaken to determine the effects of exposure to chronic low level metal concentrations on cold water fishes.

Definition of Terms used in Report

Acute - involving a stimulus severe enough to bring about a response speedily, usually within 48 hours in toxicity tests.

Additive toxicity - a mixture of two or more toxicants may have one of three effects. If the toxicity of a mixture is equal to the sum of the toxicities of the individual toxicants then the effect is said to be additive.

Alevin - a developmental phase in fish that do not have a larval period which starts with external feeding and lasts until the development of complete scalation or ossified spines. Term is generally restricted to use in describing salmonid life cycles.

Antagonistic toxicity - a mixture of two or more toxicants may have one of three effects. If the toxicity of a mixture is less toxic than any of the toxicants is when tested by itself, then the effect is said to be antagonistic.

ATPase activity - the activity of any enzyme which breaks down ATP (adenosine triphosphate) to ADP, resulting in a release of energy.

Chronic - involving a stimulus which is lingering or continues for a long time. When used in describing toxicity tests implies exposure for greater than 30 days, generally to a level of toxicant below the LC_{50}.

Corticosteroids - steroids formed and secreted by the interrenal tissue (adrenal cortex of the kidney) including cortisol, cortisone and aldosterone; major functions include alteration of lipid, protein and carbohydrate metabolism, osmoregulation and resistence to noxious stimuli as well as other functions.

Parr - see smolt.

Proximal tubules - portion of the nephron between Bowmans capsule and the loop of Henle in the renal cortex of the kidney; functions in urine formation.

Smolt - a phase in salmonid development occurring between the alevin and juvenile phases. Encompasses the interval of downstream migration and physiological adaptation of the fish to survive in seawater.

Sublethal - below the level which directly causes death, but may cause other less noticeable effects such as reduced growth impaired reproduction or stress.

Sulfhydryl - a chemical group containing a sulfur atom and a hydrogen atom.

Synergistic toxicity - a mixture of two or more toxicants may have one of three effects. If the toxicity of a mixture is greater than the sum of the individual toxicities then the effect is said to be synergistic.

Threshold level - minimum toxicant concentrations that will cause an effect.

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