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EVALUATION OF TOXIC METALS IN NATURAL FISH POPULATIONS IN OKLAHOMA

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EVALUATION OF TOXIC METALS IN

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IN OKLAHOMA

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ABSTRACT

A study was conducted to determine effects of arsenic, cadmium, chromium, copper, lead, and mercury on the natural fish populations in Oklahoma. Fish samples were collected annually at 23 monitoring stations for herbivore and carnivore trophic levels during Water Years (October 1 to September 30) 1977, 1978, 1979, and 1980.

Samples of the water column and sediment were also collected and analyzed for the same toxic metals as the fish samples. Additionally, pH, total hardness, and flow were analyzed at the monitoring stations. These measurements were taken to determine if they had an effect on the toxicity of the toxic metals analyzed to the natural fish populations.

The concentrations of toxic metals studied in the natural fish populations in Oklahoma were generally low and no observed patterns of elevated toxic metals in the fish samples could be determined. There were no direct correlations between the toxic metal levels in the water and sediment samples and the levels measured in the fish samples. No correlations were noted in the pH, total hardness, and flow measurements to the toxic metal levels in the fish samples. Since no patterns were observed in the fish data, it was not possible to predict the sources of these toxic metals.

The level of toxic metals was low in the water samples but were much higher in the sediment samples. There were no significant correlations between the levels of toxic metals in the water to the levels in the sediment.

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CHAPTER I

INTRODUCTION

During the past few years there has been considerable emphasis placed on evaluating and abating the many pollution problems that have plagued the United States (1). Recently, there has been increasing concern about the ultimate impact on the aquatic environment of hazardous, toxic materials. Thomas (2) in the symposium, "Proceedings on the Biological Monitoring for Environmental Effects," stated "there are indications of a growing age of skepticism with regard to monitoring the effects of hazardous pollutants in our environment." He also stressed the need to guard against skepticism developing as the result of misunderstandings or because of inaccurate or misleading information on the effects of these environmental contaminants.

Currently there is considerable interest in the concept of monitoring for the presence of toxic materials in natural fish populations. This thinking has resulted from the fact that fish are an excellent choice for the determination of toxic substances in the aquatic ecosystem since they are always in contact with the ultimate transporter of toxics, the water and sediment. The term toxics is

taken to mean several different things. Organic toxics refer to materials such as pesticides, herbicides, and hydrocarbons. Inorganic toxics refer to materials such as the heavy metals and the substances which cause eutrophication problems. For the purpose of this paper toxics are defined as the toxic metals arsenic, cadmium, chromium, copper, lead and mercury.

The primary goals of the Federal Water Pollution Control Act (Clean Water Act) of 1972 as amended in 1977 (3) were to:

1. "Eliminate the discharge of pollutants into navigable waters by 1985;

2. Provide for the protection and propagation of fish, shellfish, and wildlife and provide for recreation in waters of the U.S. by 1983; and

3. Prohibit the discharge of toxic pollutants in toxic amounts into these waters."

Most of the biological monitoring done to date has been accomplished with the aid of grants authorized by section 106 of the Clean Water Act (3). Specifically these monies have been used to implement what is called the Basic Water Monitoring Program (4). This program was set up as a national technique to address the problem of the accumulation of toxics in fish tissue. EPA Deputy Administrator John P. Quarles established a Standing Work Group on Water Monitoring on December 24, 1975. This Standing Work Group was charged with the task of reviewing ongoing monitoring activities and developing cost-effective water monitoring programs in the EPA regions and the states. The document this group produced was not intended to be a "regulation or set of strict guidelines and should not be implemented blindly" (4). Instead, the document was to be considered a "basic structure" which could be

used to begin a process of consistent nationwide monitoring programs so data could be collected which would be a contribution to more effective use of our monitoring resources.

This "Basic Monitoring Program" was used by the state of Oklahoma to design monitoring programs which would address the data needs for a total water quality management effort. As a practical necessity, the program has undergone a series of minor changes as it evolved to the present effort. It should be noted, however, that consistency in a long-term program was a major consideration in any changes proposed in the total program effort. The program proposal for Water Year 1981 represented the final program which is recognized by water quality management agencies in Oklahoma as the maximum effort which can be done with the resources available (5).

One additional point should be made in the way of introduction to this project. The Oklahoma Water Resources Board (6) in their Water Quality Standards state that water resources development in the last century involved water quality development to a minor degree. During recent years, however, an increase in population resulting in community, industrial and agricultural development, has caused the scientific and engineering communities to reassess the role of water quality and elevate it to a position of comparable importance with water quantity. Section 4.3 of these standards say there are no generalized water quality standards applicable for all kinds of fish and wildlife. Generally, unpolluted waters support a more diverse aquatic community while only tolerant species can survive in comparatively polluted waters. The impact of a given chemical or physical

constituent on a biological community is not mutually exclusive of other constituents since synergistic antagonistic, and other types of interactions are common. The Water Board further states that the narrative and numerical standards are designed to promote fish and wildlife propagation.

Section 6.6 of the water quality standards address the problem of toxic substances. This section states the waters of the state shall be maintained so they will not be toxic to humans, fish and wildlife, and other terrestrial and aquatic life, nor detrimental to any beneficial use including continued ingestion by livestock or for irrigation use. Toxic substances in Oklahoma waters shall not be present in quantities which allow significant bioaccumulation and/or biomagnification in the food chain.

The specific information needed to adquately address these issues is not currently available. Much more work must be done in order to completely define the toxic and bioaccumulation potential of toxics in natural fish populations. This research should help to address a part of the total issue of toxic metals in the natural fish populations in Oklahoma.

CHAPTER II

LITERATURE REVIEW

Introduction

During the past few years much work has been done to document the impact of toxic metals within the aquatic ecosystems of the United States. It should be noted that this literature review is not intended to cover the entire scope of toxic metals in the aquatic ecosystem. Such reviews have been done by other authors and the update on the subject alone is sufficiently large to warrant an entire research project. This literature review is limited in scope to briefly summarize the completed work which relates to the evaluation and interpretation of the data generated in this study.

It is interesting to note at the beginning of this literature review that very few papers were available from the literature addressing the effects of toxic metals on natural fish populations. Of the articles reviewed, only a few were from Oklahoma, and these did not deal with the effects of toxic metals on natural fish populations. Therefore, this project should add to the scientific literature with respect to additional evaluation of toxic metal data for the natural fish populations in Oklahoma.

This literature review consists of a general discussion of the concept of toxic metals impact on the aquatic ecosystem. A brief discussion of the six toxic metals being addressed by this project will follow.

In their introduction to a study on metal accumulation in "Fishes and Aquatic Invertebrates," Phillips and Russo (15) stated that during recent years considerable attention has focused on the fate of metals and their derivatives in the aquatic environment. Although some metals are essential to aquatic organisms in trace amounts, others offer no known direct benefits. Concern for other aspects of environmental health have prompted researchers to explore the extent to which these other metals are concentrated in living tissue, particularly in aquatic organisms.

Metals accumulation studies which focus on the aquatic environment are important for various reasons. The extent to which metals are accumulated by aquatic animals can be related to metals toxicity.

Two types of toxicity are discussed in the literature. Acute toxicity refers to a situation which causes death or severe damage to an organism during a brief exposure period, normally ninety-six hours, or less. Chronic toxicity refers to a situation which causes death or damage to an organism during prolonged exposue, which, depending on the organism tested and the test conditions and purposes, may range from several days, to weeks, months, or years (16). It should be noted that there is no clear line of demarcation between acute and chronic toxicity.

The measure of the toxicity of any material to fish is usually expressed in terms of the LC50, TL50, or TLm. These expressions all refer to the concentration of a toxicant that is lethal (fatal) to fifty percent of the organisms tested under specified test conditions in a specified time.

The effects of acute toxicity is obvious; the fish die or become ill. The effects of chronic toxicity, however can be much more subtle. These effects may be related to changes in appetite, metabolism, disorders of the nervous system or reproduction. Some chronic effects may be reversible but most are not. Chronic effects can occur in the species population rather than the individual. If, for example, eggs fail to develop or the sperm does not remain viable, the species may be eliminated from an ecosystem because of reproductive failure. Additionally, the phenomenon of bioaccumulation of certain materials may result in chronic toxicity to the ultimate consumer in a food chain. Thus, fish may slowly release lethal toxicants from their fatty tissues during periods of physiological stress.

McKee and Wolf (17) say the toxicity of many potential pollutants in water toward plant and animal life is a time-concentration phenomenon; i.e., for a given concentration, toxicity increases with continued exposure. For other sustances, however, toxicity is relatively independent of time; i.e., if a given concentration is not toxic in one or two hours, it will not be acutely or directly toxic.

The relationship between acute toxicity and the concentration of metals in various tissues is useful. Knowledge of relationships between chronic toxicity and tissue levels of metals can aid regulatory agencies in adopting and monitoring compliance with water quality standards. Survey and monitoring programs aimed at pinpointing contamination problems of metals would help regulatory agencies in adopting the necessary restrictions. An understanding of the processes governing the fates, pathways and distribution of metals in natural waters is necessary for assessing the current status of metals

in the environment and for avoiding potential problems due to toxic metals.

Like mercury, other metals concentrated by commercially or recreationally valuable aquatic organisms pose a threat to human consumers and could thereby render these resources less valuable. The United States Food and Drug Administration (FDA) currently lists mercury, lead, cadmium, arsenic, selenium and zinc at the top of its priority list in its program concerning toxic elements in food (18). Of these, only mercury has an FDA-specified regulatory limit for fish and shellfish; FDA guidelines for other metals in foods have not been established.

The fact that living systems, i.e., individuals, populations, species and ecosystems can take up, accumulate and bioconcentrate man-made and natural toxicants is well documented. In aquatic systems biota are exposed directly to pollutant toxicants through submersion in a relatively efficient solvent (water) and are exposed indirectly through food webs and other biological, chemical, and physical interactions. Initial toxicant levels, if not immediately toxic and damaging, may accumulate in the biota or sediment over time and increase to levels that are lethal or sublethally damaging to aquatic organisms or the consumers of these organisms.

Ions of toxic materials frequently cause adverse effects because they pass through the semipermeable membranes of an organism. Some materials may not pass through membranes in their natural or waste-discharged state, but, in water they may be converted to states having increased ability to affect organisms. For example, certain

microorganisms can methylate mercury; thus producing a material that more readily enters physiological systems. Some materials may have multiple effects: for example, an iron salt may not be toxic while an iron floc or gel may be an irritant or clog fish gills resulting in asphyxiation. Iron, at low concentrations, can be a trace nutrient, but, at high concentrations, can be a toxicant . Materials also can affect organisms if their metabolic byproducts cannot be excreted.

Interrelationships and interactions among organisms and their environment as well as the interrelationship between sediment and the water column has been documented. Antagonistic and synergistic reactions among many constituents in water has also been established.

The universe of organisms composing life in water is great in both kinds and numbers. As in the human population, physiological variability exists among individuals of the same species in response to a given stimulus. A much greater response variation exists among species of aquatic organisms. Thus, aquatic organisms do not exhibit the same degree of harm, individually or by species, from a given concentration of a toxicant or potential toxicant within the environment. It is necessary to ensure a reasonable degree of safety for those more sensitive species that are important to the functioning of the aquatic ecosystem even though data on the response of such species to the quality constituent under consideration may not be available. The aquatic food web is an intricate relationship of predator and prey organisms. A water constituent destroying or eliminating an important segment of this web would, in all likelihood, destroy or seriously impair other organisms associated with it.

The ideal data base for criteria development would consist of information on a large percentage of aquatic species and would show the community response to a range of concentrations for a tested constituent during a long time period. This information is not available but investigators are beginning to derive such information for a few water constituents.

Brown and Chow (19) indicated in their study of heavy metal concentrations in Ontario fish that the complex role of trace heavy metals in the biosphere is little understood. However, it is well known that large concentrations of trace heavy metals are toxic to the ecosystem as a whole and to man in particular, since he is at the end of a variety of food chains by virtue of his variegated diet. These authors studied alewife, brown bullhead, carp, freshwater drum, gizzard shad, golden shiner, lake whitefish, largemouth bass, longnose sucker, pumpkinseed, rainbow smelt, rock bass, white bass, white sucker and yellow perch. It was found that the concentrations of metals were similar in the various species studied. The higher levels of metals at one location were attributed to higher concentrations of metals in sediments.

Vinikour, Goldstein, and Anderson (20) were of the opinion that determinations of whole body heavy metal contents are critical to the study of biomagnification, because predators consume entire prey, not selected organs. Consequently whole body metal concentrations from both contaminated and uncontaminated sites are of increasing importance to investigators. Patterns of heavy metal bioconcentration with age or size, can influence, to the extent of masking, observed trends in biomagnification.

These authors noted with few exceptions, whole body metal concentrations showed no change as fish weight increased. Discrepancies in bioconcentration patterns observed by researchers may be due to inconsistencies in analyzing either whole body or various tissues. Furthermore, the susceptibility of individual tissues to metal uptake varies considerably due to new tissues being incorporated at a greater rate than metals being actively transported into the tissues to establish a steady-state concentration.

Rehwoldt et al. (21) did a study of current and historical heavy metal residues in Hudson River fish. Their data indicated that although metal residues may be an indicator of industrial activity and contamination in certain water systems in a relatively clean system such as the Mid Hudson area, the residues are independent of time. While several pathways could be proposed if the data indicated a relationship between residues and industrial development, they are not appropriate in this case. The most likely source of the residue is the absorption of the metals from the waters which leached them from the river banks and bottom. These data do not seem to follow any chronological relationship; in fact, they seem to be independent of time.

Enk and Mathis (22) stated that aquatic insects exhibited higher concentrations of cadmium than did sediments. Although it is known that many metals are toxic to aquatic organisms, the exact actions of these metals and the levels at which they become harmful are still being investigated. In general, metals and other stream contaminants, whether in suspensions or solution, do not simply flow downstream.

Phillips and Russo (15) stated the following conclusions and recommendations in their extensive literature review of the bioaccumulation tendencies of twenty-one metals:

1. Unlike mercury, most metals are not accumulated in the edible portions of fishes and do not represent a threat to human consumers of fish unless the fish are eaten in their entirety. Metals deserving further attention with respect to their propensity for accumulation in edible fish tissues include mercury, arsenic and radioactive cesium.
2. Shellfishes, particularly oysters, passively accumulate many metals much more readily than fishes; this suggests a priority for monitoring in metal-contaminated areas. Potentially dangerous metals in shellfishes include cadmium, arsenic, mercury, lead and silver.
3. Most fishes are capable of accumulating most metals both from their diet via the gastrointestinal system and from water via various membrane surfaces, particularly the gills. With some exceptions, the relative contributions of these two sources of metals to fishes are poorly understood. Considering that food may be an important route of exposure to toxic chemicals of fish, criteria derived from laboratory toxicity experiments during which fish received exposure to chemicals only through the water could be misleading. Further research is needed in this area.
4. Although the distribution of some metals in the tissues of a variety of aquatic organisms has been extensively studied, more information is needed about the actual mechanisms of toxic action, particularly mechanisms of chronic toxicity. Because some metals continue to be accumulated by fishes at the same rate even under conditions which significantly reduce toxicity, and also because some species or individuals are more or less susceptible than others to bioaccumulation of a particular metal, it follows that toxic response is internally determined and that adaptive factors are involved.
5. Sediments are an important sink for most metals in aquatic environments. Further information concerning the biological and physio-chemical factors affecting metals mobilization from and deposition in sediments would be valuable.
6. Although some instances have been reported where high levels of metals in natural waters have been attributed to natural sources, the largest share of contamination is due to man. Waters receiving metal inputs resulting from man's activities should receive the highest monitoring priority.
7. The relationships between chronic toxicity thresholds and metal concentrations in tissues have been determined for a few metals with a few fish species. Studies should be undertaken to determine if these relationships are valid in natural environments; if this concept proves useful, then relationships should be

established for other metals and with other aquatic species.
8. Some chemical forms of metals, such as methylmercury, are far more toxic and more readily accumulated by aquatic organisms than are others. The most bioaccumulative and toxic forms of other hazardous metals should also be determined.

Table 1 shows the summary data of the LC50 values examined in the literature review. Where a range of values was given, the maximum and minimum numbers were selected for inclusion in the table.

Arsenic

Physical State

Arsenic is a naturally occurring element often referred to as a metal, although chemically classified as a metalloid. Arsenic is described as a gray, lustrous, crystalline mass, also black amorphous powder and yellow crystals. Arsenic has a molecular weight of 299.6, specific gravity of 5.73, sublimes without melting at 610^{o}C, and has a vapor pressure of 1 mm of mercury (23, 24). Arsenic and its compounds are used in the manufacturing of glass, cloth, and electrical semiconductors, fungicides and wood preservatives, as growth stimulators for plants and animals, as well as in veterinary applications, textile printing, and to control sludge formation in lubricating oils (23, 25).

The United States consumes half of the world production of arsenic or about 37,500 tons per year, and produces about 18,000 tons per year itself. The principal emission source for arsenic in the United States is thought to be coal-fuel power plants which emit approximately 3,000 tons of arsenic per year (26).

Arsenic as a free element is rarely encountered in natural waters. Soluble inorganic arsenate (+5) predominates under normal conditions

Table 1. Summary of Ranges of LC50 Values (Expressed in Mg/L) From Selected References.

Reference	Year	Arsenic	Cadmium	Chromium	Copper	Lead	Mercury
McKee and Wolf	1963	1.000	0.010-10.000	0.050	0.020	0.100	0.004- 0.200
EPA	1976		0.001- 0.012	0.100			*0.001
Sorensen	1976	150.000					
Cardwell et al.	1976	18.100					
Hughes and Davis	1967	0.290					
Inglis and Davis	1972	16.240					
Clemens and Sneed	1959	15.022					0.035- 2.180
Sorensen	1976	150.000					
Pickering and Gast	1972		2.000-12.000				
Chapman			0.001- 0.004				
Kumada	1973		0.006- 0.007				
Hale	1977		0.006			8.000	
Davies	1977		0.002				
Spehar	1976		2.500				
Pickering and Henderson	1966			4.100-133.000		5.580-482.000	
Wallen	1957			92.000-135.000		630.000	
Trama and Benoit	1960			110.000-170.000			
Adelman and Smith	1976			37.000-133.000			
Benoit	1976			69.000			
Holland et al.	1960				0.190-0.780		
Lorz and McPherson	1976				0.060-0.074		
Chakoumakos					0.044-0.367		
Hawarth and Sprague					0.019-0.298		
Cairns et al.	1978				0.150-2.700		
Geckler et al.	1976				0.340-1.100		
Mount and Stephen	1969				0.075-0.430		
Rehwoldt et al.	1971				4.300-6.200		
Brown et al.	1974				0.580		

* = Less Than Detection Limit

Table 1. (cont.)

Reference	Year	Arsenic	Cadmium	Chromium	Copper	Lead	Mercury
Brown and Dalton	1970				0.750		
Cope	1966				0.150		
Patrick et al.	1968				1.250		
Davies et al.	1976					1.170-507.000	
Tarzwell and Henderson	1960					2.400	
MacLeod and Pessah	1973						0.220 - 0.400
McKim et al.	1976						0.065 - 0.084
Reinert et al.	1974						0.010 - 2.100

since it is thermodynamically more stable in water than arsenite (+3) (29). Analysis of 1,577 U. S. surface waters showed arsenic to be present in 87 samples, with concentrations ranging from .005 mg/l to .336 mg/l and a mean level of .064 mg/l (27). Bowen (28) reported .003 mg/l in sea water. Although arsenic is also found in air and in all living organisms, the low toxicity of elemental arsenic is attributed to its virtual insolubility in water or in the body fluids (25).

Conditions of low pH and low dissolved oxygen in water favor the formation of lower oxidation state arsenicals such as arsenite (+3) and arsine (-3) whereas more basic, oxygenated waters result in an increase in the percentage of arsenic present in the pentavalent state. The reducing action of certain organisms may also cause arsenite to be the predominant form. In waters of high organic content, a considerable amount of arsenic may be bound to colloidal humic matter (29).

Aquatic Organism Toxicity

Complete knowledge of the toxicity pathways of arsenic is less than complete since much of the work has been devoted to monitoring or field assessment studies. It is thought that arsenic forms kinetically stable bonds to sulfur and carbon in organic compounds. Like mercury, arsenic (+3) reacts with sulfhydryl groups of proteins; and, enzyme inactivation by this mechanism appears to be the primary mode of arsenic toxicity.

Arsenic toxicity varied greatly with different species and within the same species of fish. One of the lower LC50 values reported (30)

was approximately 500 times lower than the higher values (31). The LC50 values for bluegills alone differed by as much as 300 times. These large differences were due mainly to the low acute value given for bluegills (30) in which a specialized granular formulation of sodium arsenite was used. This was found to be more toxic than other formulations previously tested. Comparisons of the highest and lowest acute values without this value showed differences of approximately eight times between species and five times for tests with bluegills. Although tests were conducted in both hard and soft water, hardness as indicated by Inglis and Davis (33) did not appear to affect the toxicity of arsenic. No significant difference in toxicity was found by these authors for comparative tests with bluegills in water of hardness ranging from 53 to 368 mg/l as $CaCo_3$. Comparison of LC50 values for fingerling and juvenile channel catfish (32, 34) and for bluegills (30, 32) show early life stages of fish may be more sensitive to arsenic than later stages. This is in agreement with work reported by Sorensen (31) for green sunfish and Gilderhus (35) for bluegills studied in outdoor ponds.

Bioconcentration factors were calculated by Spehar et al. (36) for five invertebrate species and two species of fish. A bioconcentration factor of less than one was obtained for amphipods since residues were less than the detection limit. In the same study a bioconcentration factor of zero was reported for rainbow trout because concentrations in the exposed fish were the same as in the controls after a 28-day exposure period. Daphnia magna, snails, and stoneflies had slightly higher residues than trout tested under the same conditions indicating that lower aquatic forms may accumulate some arsenic.

A bioconcentration factor of four was obtained for bluegills in another study (15). The half-life of this element in bluegill tissues was one day. The low bioconcentration and short biological half-life of arsenic in fish tissue suggest no residue problem will occur at exposure concentrations not directly toxic.

Data on other toxicological effects show there is a wide range of sensitivity of invertebrate and fish species to arsenic. In almost all cases arsenic toxicity was increased with increased length of exposure. Water temperature also appeared to influence arsenic toxicity. Sorensen (37) found higher water temperature decreased the median lethal time of green sunfish after exposure to two concentrations of arsenic.

Although arsenic is concentrated in aquatic organisms, it is evidently not progressively concentrated along a food chain. In addition, arsenic consumed as an organically-bound species in flesh appears to have low toxicity (29).

These data indicate freshwater fish-food organisms are adversely affected by concentrations of arsenic as low as 1.3 mg/l. The data, however, are not considered to be sufficient to recommend any numerical criterion for freshwater aquatic life. Existing data indicate the 50 ug/l criterion established for domestic water supplies should be protective of aquatic life (16). McKee and Wolf (17) concluded that 1.0 mg/l of arsenic was recommended for the adequate protection of fish and other aquatic life.

Human Toxicity

Arsenic exposure occurs within the human population in a number of ways. Arsenic is still used to treat leukemia, anemia, and certain skin diseases. In the diet, vegetables and grain contain an average of 0.44 ppm and meats an average of 0.5 ppm of arsenic. Organic arsenicals are deliberately introduced into the diet of poultry and pigs as growth stimulators and pesticides. Compounds of arsenic may be absorbed industrially by inhalation, ingestion, and through the skin. Additionally, the arsenic content of drinking water supplies in the United States ranges from a trace to approximately 0.1 mg/l . No adverse health effects have been reported from the ingestion of water containing 0.1 mg/l of arsenic (23, 38, 39, 40).

Arsenic is distributed primarily to the liver, kidneys, intestinal wall, spleen, lungs, and to a lesser extent to the body tissues and fluids. The extent to which arsenic is taken up by these tissues depends on the rate of exposure and the chemical form (38). Arsenic is immobilized by binding to sulfhydryl groups in the keratin of hair and nails. Deposition begins within two weeks after the dose. Excretion is slow, requiring up to ten days after acute absorption. It is excreted via the urine, feces, sweat, and the epithelium of the skin (23, 41, 42). After cessation of continuous exposure, arsenic excretion may continue for as long as 70 days (39, 43).

There is a great deal of confusion in the literature regarding the accumulation of arsenic and little is known about the biotransformation of arsenic in man in spite of the long use of arsenicals as pharmaceuticals and pesticides (38). The toxicity of various arsenic

compounds is extremely variable and depends on the species exposed, the formulation of the arsenical, the route of exposure, and the rate and duration of exposure (38, 44). An assumption that all arsenic compounds are equally toxic is wrong and dangerous.

The signs of severe toxicosis in humans include abdominal pain, forceful vomiting, cramps in the legs, restlessness, and spasms. Other signs include collapse, livid and anxious face, sunken eyes, cold and clammy skin, prostration, stupor, convulsions, paralysis, collapse, coma, irritation of the nose and conjunctiva, bronchitis, perforation of the nasal septum and loss of nails and hair (23, 38, 39, 45, 46, 47).

Data on exposure of humans points to a causal relationship between skin cancer and high level exposures to inorganic arsenic compounds (48). Evidence of the carcinogenicity of arsenic in man is based almost entirely on descriptive, retrospective, epidemiologic studies. Thus, a change in the rate of cancer in various population groups has been identified, suggesting the influence of carcinogens in the environment of the groups (38).

Cadmium

Physical State

Cadmium is a soft, ductile, silver-white, electropositive metal which dissolves readily in mineral acids. Spontaneous annealing and recrystallization of chill-cast cadmium occurs at room temperature. Cadmium has an atomic weight of 112.41, a specific gravity of 8.642, a melting point of 320.9^{o}C, and a boiling point of 767^{o}C (23).

The principal uses of cadmium include electroplating, pigment

manufacture, alloys, nuclear reactors, and plastics (17, 38, 49). The solubility of cadmium compounds in water depends on the nature of the compounds and on the water quality. However, in most situations sufficient cadmium can be dissolved to cause toxic effects to aquatic organisms (50). In streams and rivers, the concentration of cadmium tends to be higher in sediment than in filtered running water. Pollution sources and rainfall may be the major contributors of cadmium in river water since cadmium is strongly absorbed to clays, muds, humic and organic materials and some hydrous oxides, all of which tend to remove it from the water column by precipitation (51). Most fresh waters in the United States contain less than .001 mg/l of cadmium although levels as high as .120 mg/l have been reported (52).

Aquatic Organism Toxicity

In the aquatic environment, cadmium is acutely toxic to fish at concentrations as low as .001 mg/l. Chronic toxicity to fish has been reported at approximately the same levels. Tabata (53) and Carroll et al. (54) have shown in acute tests the calcium ion protects fish against cadmium toxicity. Cadmium has been reported to bioconcentrate in fish tissues to levels 2,000 times as great as those of ambient waters (55). Since cadmium is an element, it will not be destroyed and may be expected to persist indefinitely in the environment in some form. There is a tendency for cadmium to accumulate in the liver and kidney of exposed organisms. It also acts synergistically with other substances to increase toxicity. Cadmium concentrations of 0.03 mg/l in combination with 0.15 mg/l of zinc from galvanized screens can cause mortality in salmon fry (17).

McKee and Wolf (17) also state the lethal concentration for fish varies from about 0.01 mg/l to about 10 mg/l depending on the test animal, water type, temperature, and time of exposure. Examination of other LC50 values for fish show concentrations, adjusted only for differences in toxicity test methods, ranging from a low of .006 mg/l to a high of 40.18 mg/l, with several intermediate values showing intraspecific variability, possibly due to water quality effects (56). Although some of the adjusted LC50 values appear relatively low, there is credence in the fact that four independent studies present values below .01 mg/l, which is the maximum allowable cadmium concentration for potable water in the United States (16, 57).

Toxicity data indicate water hardness significantly influences the acute toxicity of cadmium to fish. The only chronic test data relating fish chronic toxicity to hardness is unexplainably contradictory (55). Brook trout were found to be several times more sensitive in soft water than hard water, while channel catfish were equally sensitive in both.

Pickering and Gast (56) conducted two separate flow-through tests on the chronic toxicity of cadmium to the fathead minnow using water of 202 mg/l hardness, 157 mg/l alkalinity, and 7.7 pH. Five cadmium concentrations from .004 mg/l to .35 mg/l were delivered to the exposure chambers in each test over the life history of the fish. A concentration of .06 mg/l cadmium decreased survival of developing embryos. At levels from .005 mg/l to .04 mg/l no adverse effect on survival, growth, or reproduction was found. Eaton (58) exposed bluegill sunfish to five cadmium concentrations ranging from .031 mg/l

to 2.14 mg/l for 11 months in a flow-through system using water of the same hardness as above. Nine of the 18 adult bluegill sunfish exposed to .08 mg/l died by the end of the test, while all of those exposed to .031 mg/l, as well as the control, survived. At .08 mg/l cadmium the hatchability of eggs was not measurably affected, but the survival and growth of the resulting larvae were severely reduced after 60 days. Larvae exposed to .031 mg/l cadmium survived and grew about as well as the control fish. Sixty days after hatching in hard water, growth and survival of channel catfish fry was reduced significantly at a cadmium concentration of .017 mg/l but not at .012 mg/l (59). Thus, in hard water, a criterion of .012 mg/l cadmium represents a demonstrated no-effect level for catfish and therefore was chosen to protect non-salmonid freshwater fish species.

Spehar (60) reported on chronic toxicity tests with cadmium using a topminnow native to Florida in water with a hardness of 41 mg/l to 45 mg/l as $CaCO_3$, alkalinity of 38 mg/l to 43 mg/l, and a pH of 7.4. There was a significant reduction in the number of eggs produced per female at .008 mg/l cadmium, but fish in .004 mg/l were unaffected. A criterion of .004 mg/l cadmium was selected as offering protection to warm water fish species in soft water.

Three consecutive generations of brook trout were exposed to cadmium concentrations ranging from .006 mg/l to .0005 mg/l in a test water of similar hardness (61). Second generation fish exposed to .006 mg/l and .003 mg/l cadmium were smaller at three months than fish exposed to lower concentrations. Both first and second generation fish suffered extensive mortalities during spawning in .003 mg/l cadmium. Egg hatchability, survival, growth, and reproduction of fish exposed

to .002 mg/l were equal to those of control fish.

Eaton et al. (62) reported the effect of cadmium on juveniles of brook trout and eyed embryos of brown trout was greater after exposure for 60 days than after exposure for 30 days. In Coho salmon, lake trout, and the younger embryos of brown trout the longer exposure did not evoke greater sensitivity, nor did a 120-day exposure to cadmium increase the sensitivity of brook trout compared to that at 60 days. Therefore, based on the limited amount of information provided by this study and a few life-cycle chronic exposures, 60 days seems to be an appropriate duration of larval or juvenile exposure to estimate cadmium chronic toxicity.

Biesinger and Christensen (63) measured the toxicity of cadmium to Daphnia magna during an entire life cycle in test water with a hardness of 45 mg/l, alkalinity of 42 mg/l, and a pH of 7.7. It was found that 50 percent of the daphnids exposed to cadmium concentrations of .005 mg/l were killed in three weeks. The production of young was reduced by 50 percent compared to the controls in a cadmium concentration of .0007 mg/l. Several invertebrate species have been found much less sensitive to cadmium in acute tests than in the midge and cladoceran exposures (64, 65, 66).

Bioconcentration factors ranged from 151 for brook trout (61) to 1,988 for flagfish (67). By comparison, brook trout were observed to approach steady-state bioconcentration much more slowly (61) than Daphnia magna (68).

One noteworthy characteristic of cadmium bioconcentration is the possible long half-life of residues. Benoit et al. (61) found

certain organs did not lose significant amounts of cadmium when exposed trout were placed in clean water for several weeks.

Fish and certain other invertebrates have been found to be sensitive to low levels of cadmium in water. Salmonids and cladocerans appear to be the most sensitive among organisms tested. Increased hardness and/or alkalinity have been demonstrated to decrease the toxicity of cadmium in acute freshwater mortality tests, but may have less of an effect at low cadmium levels. Lowman et al. (69) reported a concentration factor of 1,000 for cadmium in fish muscle.

Murphy et al. (70) state that cadmium concentrations in fish do not seem to be correlated to length, weight, condition, or age. The actual incorporation of trace metals into fish can take place by absorption across the gill surface or through the gut tract wall.

Human Toxicity

Evidence for the serious toxic potential of cadmium is provided by: (a) poisoning from cadmium-contaminated food and beverages (b) epidemiologic evidence that cadmium may be associated with renal arterial hypertension under certain conditions (c) epidemiologic association of cadmium with "Itai-itai" disease in Japan and (d) long-term oral toxicity studies in animals (39). Elinder et al. (71) also state that cadmium is a toxic heavy metal which is being dispersed in our environment because of increasing industrial use. The elimination of cadmium takes place extremely slowly, which means cadmium accumulates in the body, especially in the liver and kidneys. There is wide consensus that the cadmium content of food is the major source of

cadmium for the general population. The average daily intake for adults is approximately .05 mg/l (38).

Exposure to cadmium results in symptoms such as nausea, vomiting, salivation, choking attacks, abdominal pains, tenesmus and diarrhea (23, 38). The distribution of cadmium within the body appears to be primarily within the kidney and liver. Recent experimental study indicates cadmium at high doses can interfere with the activation of vitamin D in both liver and kidneys (38).

Chromium

Physical State

Chromium is a metallic element which can exist in several valence states. It has an atomic weight of 52.01, a specific gravity of 7.20, a melting point of 1890^{0}C, and a boiling point of 2480^{0}C (23). Chromium exhibits valence states of +3 or +6 in the aquatic environment. Hexavalent chromium is a strong oxidizing agent which reacts readily with reducing agents such as sulfur dioxide to give trivalent chromium. The valence states are such that most accumulation of chromium in the aquatic environment would occur in the sediments.

Chromium salts are used extensively in the metal finishing industry as electroplating, cleaning, and passivating agents, and as mordants in the textile industry. They are also used in cooling waters, in the leather tanning industry, in catalytic manufacture, in pigments and primer paints, and in fungicides and wood preservatives (17, 38).

Concentrations of chromium in rivers of the United States have been reported to be from .0007 mg/l to .084 mg/l. Kopp (72) reported a mean surface water concentration of .0097 mg/l, based on 1,577 samples.

Aquatic Organism Toxicity

McKee and Wolf (17) report the toxicity of chromium salts toward aquatic life varies widely with the species, temperature, pH, valence and synergistic or antagonistic effects, especially that of hardness. Fish are relatively tolerant of chromium salts, but lower forms of aquatic life are extremely sensitive. There appears to be no evidence leading one to conclude that hexavalent chromium is more toxic toward fish than the trivalent form. They further state the toxicity of chromium toward bacteria is controlled by the valence, the type of organism, the amount of organic matter present, and the presence or absence of dissolved oxygen. The chromate is much more toxic under anaerobic conditions than the chromic ion, whereas, the chromic ion is more toxic under aerobic conditions than the chromate ion.

In the freshwater environment, hexavalent chromium has been shown acutely toxic to invertebrates at concentrations as low as .022 mg/l (73) and 17.6 mg/l for vertebrates (74). For trivalent chromium the toxic value is 2.0 mg/l in freshwater (63). Hexavalent chromium has been shown chronically toxic to freshwater organisms at .105 mg/l (55); for trivalent chromium in freshwater the value is .445 mg/l (63).

Trivalent chromium is substantially more toxic to aquatic life in soft than in hard water. The effect of water hardness on the toxicity of hexavalent chromium is insignificant.

The 96-hour LC50 values for hexavalent chromium for nine species ranged from 9.62 mg/l for the fathead minnow tested in soft water to a high of 138.5 mg/l for the largemouth bass in hard water. Wallen et al. (75) studied the toxicity of hexavalent chromium to mosquito

fish using potassium and sodium salts of both dichromate and chromate. Based on chromium, both dichromate salts were about half as toxic as either chromate salt. Trama and Benoit (76) also studied the toxicity of hexavalent chromium using potassium dichromate and potassium chromate. The adjusted 96-hour LC50 values are 110.0 mg/l for the dichromate salt and 170.0 mg/l for the chromate salt. They attributed the lower LC50 value of the dichromate salt to its acidity being greater than that of the chromate salt because chromium is slightly more toxic at lower pH values.

The variation in toxicity of hexavalent chromium due to water hardness was less than the variation between the dichromate and chromate salts of hexavalent chromium in soft water (74). The fathead minnow LC50 values for both salts in soft water were 17.6 mg/l for dichromate and 45.6 mg/l for chromate. The 96-hour LC50 for bluegill to chromium was 118 mg/l and 133 mg/l for soft and hard water respectively. The difference in LC50 values due to hardness is less than a factor of 2.

Adelman and Smith (77) indicate the LC50 values for hexavalent chromium does not occur within 96 hours. For the mean of 16 LC50 values, the ratio of 11-day to 96-hour values is 0.37 for the fathead minnow and 0.27 for the goldfish. The 96-hour LC50 values for trivalent chromium for 11 species for fish ranged from 1.82 mg/l for the guppy in soft water to 39.3 mg/l for the bluegill tested in hard water.

The LC50 values varied from .019 mg/l as hexavalent chromium for Daphnia hyalina to a high of 55.0 mg/l as trivalent chromium for a caddisfly. The data indicate that cladocerans are more sensitive to

the lethal effects of chromium than are the aquatic insects.

Debelak (78) studied the acute toxicity of hexavalent chromium to Daphnia magna in both a reconstituted water with a hardness of 163 mg/l (as $CaCO_3$) and pH value of 8.3 and pond water with a hardness of 86 mg/l (as $CaCO_3$) and pH value of 8.4. The mean of five 72-hour LC50 values was .039 mg/l in the pond water and .073 mg/l in the reconstituted water. Thus, hexavalent chromium was slightly more toxic in the softer dilution water.

Benoit (79) reported on the long-term effects of hexavalent chromium to brook trout and rainbow trout. The maximum acceptable toxicant concentration (MATC) of .2 mg/l to .35 mg/l was established on the basis of survival. Growth in weight was retarded at all test concentrations during the first eight months of the exposure. However, this was a temporary effect on growth and was not used by the author to establish the MATC.

Sauter et al. (55) studied the toxicity of hexavalent chromium (sodium dichromate) to eggs and fry of six fish species: rainbow and lake trout, northern pike, white sucker, channel catfish, and bluegill. The eggs and fry were continuously exposed in soft water for a maximum of 60 days after hatching. Observations were made of the hatchability of eggs, and the survival, length, and weight of the fry after 30 and 60 days. The majority of the data generated from these chromium exposures indicates a very significant cumulative effect of fry. This was especially true for the rainbow and lake trout since significant mortality occurred between 30 and 60 days.

This cumulative effect is consistent with the observed life cycle tests with the rainbow and brook trout (79).

All of the life cycle and embryo-larval tests were conducted with hexavalent chromium in soft water with a hardness range of 34 mg/l to 45 mg/l (as $CaCO_3$). The effect of hardness on the acute toxicity of hexavalent chromium was insignificant. Olson and Foster (80) reported a statistically significant effect on growth of chinook salmon at .016 mg/l and on rainbow trout at .021 mg/l. At these concentrations, growth was reduced about ten percent.

Olson (81) studied the comparative toxicity of hexavalent and trivalent chromium to chinook salmon. Hexavalent chromium at a concentration of .20 mg/l was more toxic in Columbia River water (hardness, 70 mg/l as $CaCO_3$) than a similar concentration of trivalent chromium. Survival and growth in the trivalent chromium exposure was similar to controls; however, survival and growth in the hexavalent chromium exposure was only about 50 percent of the control.

The lowest concentration to produce an adverse effect on invertebrates was reported by Dowden and Bennett (82) and Trabalka and Gehrs (83). They reported a 48-hour LC50 for Daphnia magna of .03 mg/l of chromic sulfate. This value for trivalent chromium is so much lower than the value of 2.0 mg/l reported by Beisinger and Christensen (63) that .008 mg/l is considered to be a doubtful value. Using the data of Trabalka and Gehrs (83) and comparing the results with other chronic tests with hexavalent chromium, it is estimated that a concentration of .005 mg/l would not produce any deleterious effects.

The data available indicate hexavalent chromium to be somewhat more toxic than trivalent chromium in the case of chinook salmon, and since significant effects were seen on fish at 0.2 mg/l of hexavalent chromium, 0.10 mg/l up to values as high as 1.0 mg/l should provide adequate protection for both freshwater invertebrates and fish (16, 17).

Human Toxicity

The average daily intake of chromium in the United States varies widely due to diet and geography. Estimates range from .005 mg/day to .115 mg/day with an average of .06 mg/day to .065 mg/day all the way to an average of .28 mg/day (38, 84). Comparatively little data are available on the incidence and frequency of chromium distribution in foods (39).

Symptoms of chromium intoxication include damage to the kidneys, irritation at the site of exposure, and action as protein precipitants (23, 38). Organ distribution studies have been inconclusive. It does appear the hexavalent form of chromium is more toxic than the other forms.

The interim primary drinking water standard has been set at 0.05 mg/l (39). However, a family of four individuals is known to have drunk water for a period of three years at a chromium level of 0.45 mg/l without known effects on their health as determined by a single medical examination (85). A study was designed by MacKenzie et al. (86) to determine the toxicity of hexavalent and trivalent chromium ions to rats at various drinking water levels. After one year at levels of 0.45 to 25 mg/l, this study showed no evidence of toxic response in

body weight, food consumption, blood changes, or mortality. Significant accumulations of chromium occurred in the tissues at concentrations greater than 5 mg/l.

Copper

Physical State

Copper is a soft heavy metal with atomic number 29, an atomic weight of 63.54, a melting point of 1083°C, and a boiling point of 2595°C (23, 87). Elemental copper is readily attacked by organic and mineral acids containing an oxidizing agent and is slowly soluble in ammonia water. The halogens attack copper slowly at room temperature to yield the corresponding copper halide. Oxides and sulfides are also reactive with copper. Based on equilibrium constants, Stumm and Morgan (88) calculated copper solubility in a carbonate bearing water. They found the cupric ion to be the dominant copper species up to pH 6 and from pH 6 to 9.3 the aqueous copper carbonate complex would dominate. The presence of organic ligands such as humic acids, fulvic acids, amino acids, cyanide, and detergents would alter this equilibrium (89).

The major industrial uses for copper include electrical products, coins, and metal plating. It is used as castings, sheets, rods,tubing, and wire. Alloyed with other metals, it is used to form various brasses and bronzes (17, 23).

Usual concentrations of .001 mg/l to .01 mg/l are reported for a majority of surface waters in the United States. Fortunately, the various copper complexes and precipitates appear to be largely nontoxic and tend to mask or remove toxicity attributable to copper (90).

This fact greatly complicates the interpretation and application of available toxicity data, since the proportion of free copper ion present is highly variable and is difficult to measure except under ideal laboratory conditions. Seasonally and locally, toxicity may be mitigated by the presence of naturally occurring chelating, complexing, and precipitating agents.

Aquatic Organism Toxicity

Toxicity tests with copper have been conducted on a total of 29 fish species with nearly 250 values available for comparison. Most of these tests have been conducted with four salmonid species, fathead and bluntnose minnows, and bluegills. Toxic values range from a low of .01 mg/l for chinook salmon in soft water to 10.0 mg/l for bluegills tested in hard water (57, 90, 91, 92, 93, 94, 95, 96, 97, 98, 99, 100, 101, 102, 103, 104, 105, 106, 107, 108, 109, 110).

Chinook salmon was the most sensitive fish species. Rainbow trout and the other salmonids are somewhat less sensitive. Fathead minnows and several other cyprinids were approximately three to eleven times more resistant to copper than the salmonids. Bluntnose minnows, however, are nearly as sensitive as the salmonids. Bluegills and other centrarchids are approximately 20 to 110 times more resistant than salmonids.

Additionally, McKim et al. (111) reported on copper toxicity to embryos and larvae of fresh water fish. They stated larvae and early juvenile stages of all species tested were more sensitive to copper than the embryos. Embryo survival was affected only at the higher

concentrations of copper tested in all species except rainbow trout. Embryo mortality was almost complete at the following concentrations: Northern pike, .5 mg/l; rainbow trout, .037 mg/l; white sucker, .333 mg/l; brook trout, .555 mg/l; and herring, .555 mg/l. Copper had no effect on smallmouth bass embryos at any concentration tested.

Doudoroff and Katz (112), in reviewing literature on the toxicity of copper, concluded most natural fresh waters in the United States containing copper concentrations below .025 mg/l evidently are not rapidly fatal for most common fish species. Additionally, McKee and Wolf (17) state toxicity of copper to aquatic organisms varies significantly not only with the species, but, also, with the physical and chemical characteristics of the water. Synergism also exists between copper and mercury. On the other hand, sodium nitrite and sodium nitrate have been reported to decrease the toxicity of copper to fish, and copper has shown evidence of decreasing the toxicity of cyanide. Therefore, they recommend a water concentration of 0.02 mg/l to protect fish in general.

The overall variation observed in acute toxicity values for invertebrate species was nearly the same as for fish. The values ranged from .0042 mg/l for Daphnia hyalina to 10.2 mg/l for snail eggs, and 9.1 mg/l for adult stoneflies.

Human Toxicity

Copper is not considered to be a cumulative systemic poison like lead and mercury. Most of the copper ingested is excreted by the body and little is retained (17, 38). Copper concentrations high

enough to be dangerous to human beings renders water disagreeable to taste. However, ingestion of milligram quantities can cause symptoms of nausea, vomiting, diarrhea, congestion of mucus membranes, ulceration of the nasal septum, salivation, metallic taste, cramps in the calves and prostration (23, 38).

Lead

Physical State

Lead is a soft gray, acid soluble metal. It is used in electroplating, metallurgy, and the manufacture of construction materials, radiation protective devices, plastics and electronics equipment. The solubility of lead compounds in water depends heavily on pH and ranges from about 10.0 mg/l of lead at pH 5.5 to .001 mg/l at pH 9.0. Lead has a melting point of 327.4^{o}C and a boiling point of 1525^{o}C (23).

Lead enters the aquatic environment through precipitation, lead dust fallout, erosion and leaching of soil, municipal and industrial waste discharges, and the runoff of fallout deposits from streets and other surfaces. Extrapolations from recent studies indicate that as much as 5,000 tons of lead per year may be added to the nation's aquatic environment as a result of urban runoff (39, 113).

Some natural waters contain lead in solution as much as 0.4 mg/l to 8.0 mg/l, where mountain limestone is found. In the United States lead concentrations in surface and ground waters used for domestic supplies range from traces to 0.04 mg/l (17). Since lead is an element, it will not be destroyed and may be expected to persist indefinitely in the environment in some form.

Aquatic Organism Toxicity

McKee and Wolf (17) report the following mechanism for lead toxicity to fish in water containing lead salts: a film of coagulated mucus first forms over the gills, and then over the whole body of the fish, probably as a result of a reaction between lead and an organic constituent of mucus. The death of the fish is caused by suffocation due to this obstructive layer.

The toxic effects of lead have been tested on a wide variety of freshwater organisms. Test animals used to determine these effects included fish from six different families. Consequently, the available data base is quite large and clearly demonstrates the relative sensitivity of freshwater organisms to lead (16, 17, 74, 115).

Fifteen LC50 values were available for eight species of fish. Three soft water fathead minnow acute tests were conducted with lead chloride, and these values ranged from 2.4 mg/l to 7.33 mg/l. The close agreement between these tests demonstrates that lead LC50 values for fish can be reproduced with reasonable accuracy. The fourth soft water fathead minnow test was conducted with lead acetate and the calculated LC50 value agreed closely with the lead chloride exposures (17, 99, 114).

Acute tests have been conducted with lead in both hard and soft water with rainbow trout, fathead minnows and bluegills (74, 114). Results from these tests showed the LC50 values for lead differed in hard and soft water and varied by a factor of 237 times for rainbow trout, 65 times for fathead minnows and 19 times for bluegills. Another example of hardness-related lead toxicity to fish was reported by Tarzwell and Henderson (115). These authors conducted 96-hour

exposures of fathead minnows to lead in hard (400 mg/l) and soft (20 mg/l) water. The hard water exposure was not included because an LC50 value was not obtained within 96-hours; however, this test did show the hard water LC50 value was greater than 75.0 mg/l which meant the difference between hard and soft water exposures varied by a factor greater than 31 times. Hale (57) conducted an acute exposure test of rainbow trout to lead and obtained an LC50 value of 6.16 mg/l. This value is almost six times greater than the LC50 value obtained for rainbow trout in soft water by Davies et al. (114). Hale (57) did not report water hardness; however, alkalinity and pH were reported to be 105 mg/l and 7.3 mg/l respectively, which suggests this water was probably harder than the test water used by Davies et al. Acute values obtained by Wallen et al. (75) for the red shiner and mosquito fish were also quite high; however, the authors did not report hardness and both tests were conducted in turbid water containing suspended clay particles at approximately 300.0 mg/l.

Chronic tests have been conducted with lead and six species of fish. All chronic tests were conducted in soft water (33 mg/l).

No acceptable hard water chronic tests were found in the literature to compare with the soft water data. Davies et al. (114) reported the long-term effects of lead on rainbow trout in hard and soft water. Although these tests were neither life cycle, partial life cycle, nor embryo-larval tests, they do provide useful information. During these 19-month exposures a significant number of trout developed spinal deformities, eroded fins and blacktails in both hard (353 mg/l) and soft (28 mg/l) water at measured lead concentrations of .38 mg/l and

.013 mg/l, respectively. These results, therefore, established a definite relationship between water hardness and chronic lead toxicity to fish in which rainbow trout sensitivity varied by a factor of 29 times.

The bioconcentration factor for brook trout was calculated to be 42 from a laboratory exposure by Holcombe et al. (116) which included 20 measurements of lead concentrations in the water during the 140-day test. Lead residues reported by Atchison et al. (117) were obtained from a mixed population of bluegills collected from a small 300 acre lake. The average bioconcentration factor for lead in water for this contaminated lake was determined to be 45 from 36 separate measurements. Since no maximum permissible tissue concentration is available for lead, no residue limited toxicant concentration can be calculated.

Although a wide variety of invertebrate species have been tested, no reports were found in the literature which tested lead toxicity on the same species in both hard and soft water. However, it seems logical to assume that a similar relationship exists between acute lead toxicity and water hardness for invertebrate species as was demonstrated for exposures to fish.

In summary, lead in the aquatic environment has been reported to be acutely toxic to invertebrates at concentrations as low as .45 mg/l and chronically toxic at less than .10 mg/l (63). The comparable figures for vertebrates are .90 mg/l for acute toxicity (99) and .0076 mg/l for chronic toxicity (114). Toxicity is also affected by water hardness (74, 115).

Human Toxicity

As far as is known, lead has no beneficial or desirable nutritional effects. Lead is a toxic metal tending to accumulate in the tissues of man and animals. Although seldom seen in the adult population, irreversible damage to the brain is a frequent result of lead intoxication in children. Such lead intoxication most commonly results from ingestion of lead-containing paint still found in older homes. The major toxic effects of lead include anemia, neurological dysfunction, and renal impairment. The most common symptoms of lead poisoning are anemia, severe intestinal cramps, paralysis of nerves (particularly of the arms and legs), loss of appetite, and fatigue. These symptoms usually develop slowly (38, 39). High levels of exposure produce severe neurological damage, often manifested by encephalopathy and convulsions; such cases frequently are fatal. Lead is strongly suspected of producing subtile effects (effects due to low level or long term exposures insufficient to produce overt symptoms) such as impaired neurological and motor development and renal damage in children (118). Subclinical lead effects are distinct from those of residual damage following lead intoxication.

Mercury

Physical State

Mercury is a silver-white metal, is liquid at room temperature, and can exist in three oxidation states. Mercury has an atomic weight of 200.59, a melting point of -38.87^{o}C and a boiling point varying from 356^{o}C to 358^{o}C, The specific gravity is 13.546 and the vapor pressure is 0.0012 mm Hg (23, 87).

The largest present use of mercury is in electrical apparatus; other uses include industrial control instruments and agricultural and industrial poisons, insecticides, fungicides, bacteriocides, electrolytic cells, pharmaceutical and dental preparations, anti-fouling paint, and catalysts (23).

The Department of the Interior carried out a nationwide reconnaissance of mercury in U.S. water in the summer and fall of 1970 (119). Of those samples from the industrial wastewater category, 30 percent contained mercury at greater than .01 mg/l: nearly 0.5 percent of the samples in this group contained more than 1.0 mg/l. Only 4 percent of surface water samples contained more than 1.0 mg/l. The higher mercury concentrations were generally found in small streams. About half of the 43 samples from the Mississippi River contained less than .0001 mg/l.

Finding certain microorganisms with the ability to convert inorganic and organic forms of mercury to highly toxic methyl or dimethyl mercury has made any form of mercury potentially hazardous to the environment (120). In water, under naturally occurring conditions of pH and temperature, inorganic mercury can be converted readily to methyl mercury (121).

Mercury has long been recognized as one of the more toxic metals but was only recently identified as a serious pollutant in the aquatic environment. Initially, elemental mercury which is a liquid at room temperature, was considered a relatively inert heavy metal. The assumption was made that the mercury would quickly settle to the bottom of a body of water and remain there in an innocuous state.

However, since both aerobic and anaerobic bacteria in the sediments are capable of methylating mercury, elemental mercury is a serious threat to the aquatic environment since this process occurs maximally at a pH of 6.0.

Mercury is also one of the few major pollutants adversely affecting the aquatic environment through both direct toxicity and bioaccumulation. Bioaccumulation has been more thoroughly studied and has raised much concern (122). Methyl mercuric compounds are more toxic than inorganic mercury to mammals as well as aquatic life and most of the tissue residue data reported are for the organic form. There is no known physiological function of mercury and any mercury added to the aquatic environment may increase tissue residues.

Regardless of the mercury form present, a major portion of the mercury will ultimately reside in the bottom sediments. It appears the methylation process takes place at the water/sediment interface, particularly in the sediment area in which the benthic organisms are most active. The movement of benthos within the sediments contributes to the methylation process by physically expanding the area of water/sediment interface. Through ingestion of the detritus in the sediments, benthos acquire a body burden of mercury that will in turn be transported to fish upon ingestion. These forms of mercury are bioconcentrated many-fold in fish and other aquatic organisms because of the very rapid uptake and the relative inability of the fish to excrete mercury from their tissues. As a result, mercury in fish tissues may exceed the 0.5 mg/kg FDA guideline (16).

Aquatic Organism Toxicity

Mercurials will damage the bronchial epithelium and interrupt respiratory function in freshwater invertebrates. Rainbow trout suffered loss of equilibrium, and trout fry were more susceptible to poisoning than fingerlings. Mercurial compounds may interfere with receptor membranes in fish (123).

MacLeod and Pessah (124) reported temperature effects of mercuric chloride toxicity to rainbow trout. At 5, 10, and 15^{o}C, the LC50 values were .4 mg/l, .28 mg/l, and .22 mg/l, respectively. Clemens and Sneed (125) found that at temperatures of 10, 16.5, and 24^{o}C, the LC50 values for channel catfish and phenylmercuric acetate were 1.154 mg/l, .863 mg/l, and .223 mg/l, respectively. They also investigated the influence of life stage of channel catfish on its sensitivity to pyridylmercuric acetate. At 23 to 24^{o}C, they found about the same influence of age between yolk sac fry (48-hour LC50 value of .374 mg/l) and 3-inch juveniles (24-hour LC50 value of 3.75 mg/l) as was found for temperatures between 10 and 24^{o}C.

Cox et al. (126) studied the source of mercury in a new impoundment. During the course of this investigation about 200 fish samples were analyzed for mercury. As expected, species which were high in the food chain contacted more mercury. Their results suggested high mercury levels found in bass and crappie were a result of biomagnification through the food chain.

McKim et al. (127) observed adverse effects of methylmercuric chloride on brook trout at .0009 mg/l but not at .0003 mg/l. Brook

trout were approximately three to four times more resistant than rainbow trout.

The estimate for chronic toxicity for mercury is .0002 mg/l although no equilibrium of mercury in the fish tissues could be demonstrated by Reinert et al. (128) after an 84-day exposure of juvenile rainbow trout. The uptake of methyl mercuric chloride by brook trout had not reached equilibrium after 273 days (127). In the latter study, there was no detectable loss of mercury from various tissues after a 16-week exposure in control water. Since whole fathead minnows were only analyzed once at the end of a life-cycle exposure (129) no comment could be made with regard to equilibrium in this species. Data (128) indicated an influence of temperature on rate of uptake but was not considered for bioconcentration factor calculations since a steady state was not achieved even at the highest temperature studied.

The contrast between fathead minnows (129) and brook trout (127) is one of considerable interest and potential importance. Of the factors differing between these tests, the species and feeding habits, the latter was the most intriguing to consider. Since most of the trout were fed on pelleted trout feed, there was little opportunity for food chain imput to the trout. In contrast, the fathead minnow, a browser, had the opportunity not only to feed on the introduced food but also on the Aufwuchs growing within the mercury-enriched environment of the exposure chamber. The higher bioconcentration factor for the fathead minnows, 62,898, may be more representative of field data.

Since the lowest maximum permissible tissue concentration (1.0 mg/kg) is based on the marketability of fish and shellfish, only data on the edible portion of these organisms may be used to calculate a bioconcentration factor. McKim et al. (127) concluded there was no difference in bioconcentration factors between residues in muscle and total body for brook trout. This bioconcentration factor is 62,898.

Human Toxicity

Mercury is considered to be highly toxic to humans. It is readily absorbed by way of the gastro-intestinal tract, and fatal doses for man vary from 3 to 30 grams (17). In humans, mercurials have been associated with neurological disorders, sensory impairment, tremors, buccal ulceration, gastro-intestinal complaints, and multisystem involvement due to general encephalopathy (16, 23, 38, 122, 130, 131, 132, 133, 134).

CHAPTER III

METHODS AND MATERIALS

Program Description

The data which are presented in this research were collected as a part of the Basic Water Monitoring Program for the state of Oklahoma for water years (October 1 to September 30) 1977 to present. As was mentioned earlier, the EPA Basic Water Monitoring Program (4) was not designed to be a mandatory program. However, in order for continued EPA funding to the state on all water pollution programs, a biological monitoring program was required.

The total Basic Water Monitoring Program in Oklahoma for the last several years included 100 ambient trend monitoring stations where water samples were collected monthly for organic and inorganic content. Also included were approximately 23 biological stations sampled annually or bi-annually for toxic analysis of fish, sediment, and water. It should be noted that this monitoring effort was not all-inclusive for the total water quality monitoring for the state of Oklahoma. Other programs were conducted by various agencies for other purposes.

The fish samples for toxic metal analyses were collected from 23 different stations on 15 different streams in Oklahoma. Appendix A lists these stations and describes the stream, habitat, physical location and legal description. Figure 1 shows a map of the state including all of the monitoring stations. The biological stations

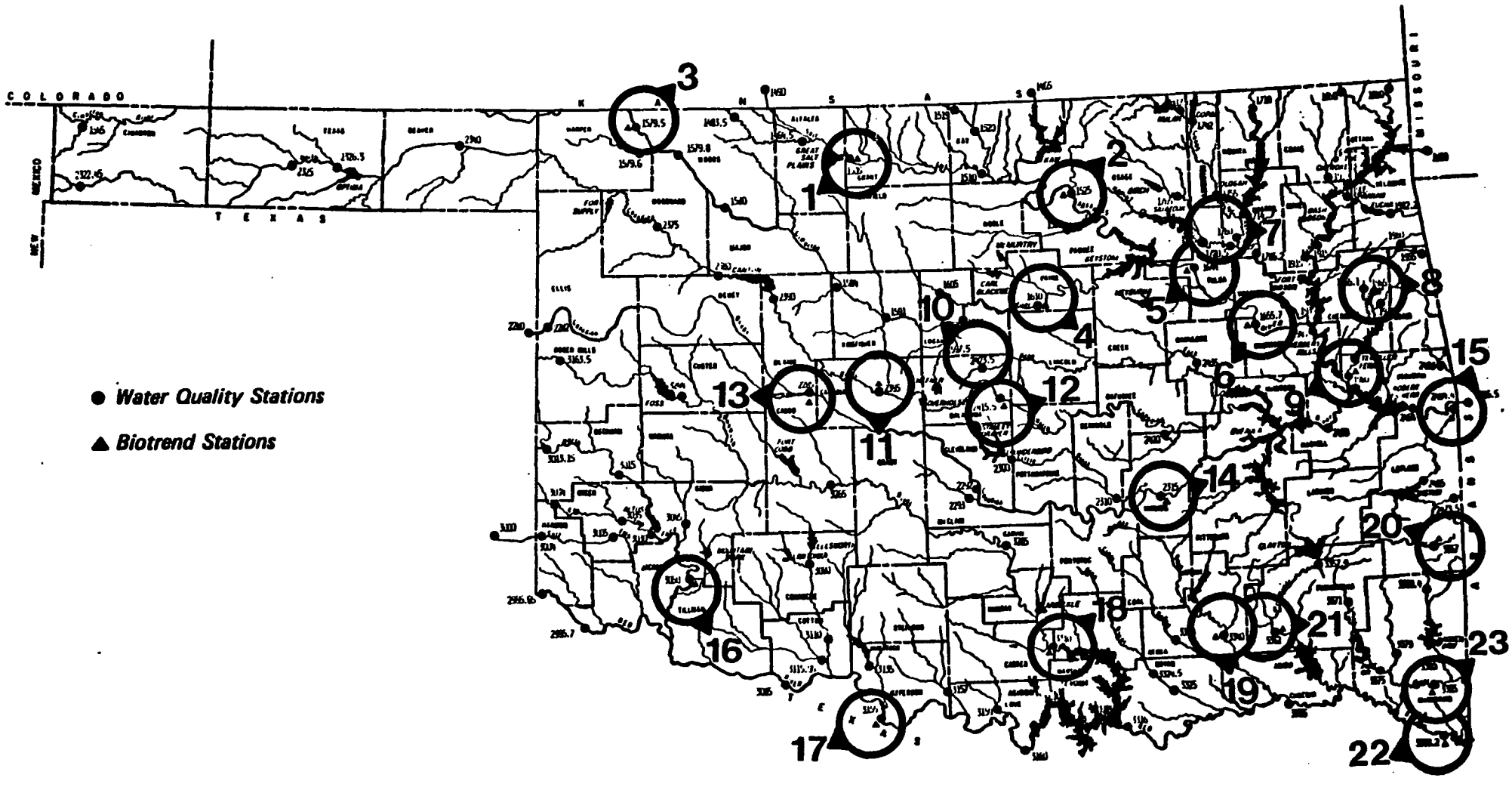

Figure 1. Map of Oklahoma Showing the Location of the Monitoring Stations.

shown were selected so the state needs as well as the national goals of EPA could be met for Oklahoma. These stations represent the various aquatic habitats within the state of Oklahoma. They also represent both high water quality streams as well as streams of poorer quality.

Field Sample Collections

The fish samples for this research effort were collected during June and July at the 23 biotrend sites. This time period was selected to correspond to the most critical low flow periods of the year. The fish populations were sampled over an area of approximately 210 yards at each sampling site. The specific collection techniques utilized included seining, electrofishing, and where habitat dictated, gill-netting (7, 8, 9, 10). Representative samples of the fish community were collected as follows: 1) one sample of the predominate herbivore trophic level, consisting of at least four fish of the same species, and 2) one sample of the predator (carnivore) trophic level, consisting of at least four fish of the same species, were collected. These samples were wrapped in plastic bags, immediately iced, and were frozen immediately upon return to the lab until lab analysis preparation could be accomplished.

Detailed field notes were maintained at each site where fish samples were collected. These notes consisted of habitat type, flow condition, any unusual disturbances to the site (such as construction activities), and any other observations that would aid the interpretation of laboratory data.

Fish Tissue Preparation and Toxic Metal Analysis

Fish preparation and analytical techniques were taken from the EPA Methods for Chemical Analysis of Water and Wastes (11) and Standard Methods for the Examination of Water and Wastewater (12). The heavy metal analyses on the prepared fish tissue samples were analyzed on an Instrumentation Laboratory Model 951 Atomic Absorption Spectrophotometer using the following procedures:

Copper, Cadmium, Chromium, Lead.

1. Take 5 grams of blended (or small) fish.
2. Place in crucible, add 1:1 Nitric Acid.
3. Place on a hot plate and take almost to dryness 3 times. (On the 3rd time, take the solution to complete dryness).
4. Place the crucible in a cold muffle furnace and set the temperature at 525 degrees C.
5. Turn on the muffle furnace and leave the crucible until a white ash is produced (usually 36-48 hours).
6. Cool.
7. Bring the ash to 25 ml final volume with 5% Nitric Acid.
8. Aspirate directly into Atomic Absorption Spectrophotometer.

Mercury.

1. Take 1 gram of blended (or small) fish.
2. Place in a BOD bottle, add 5 ml concentrated Sulfuric Acid and 2.5 ml Nitric Acid.
3. Leave this solution overnight or longer.
4. Add 100 ml deionized water.
5. Add solid Potassium Permanganate and keep the solution purple for 15 minutes, and add additional Potassium Permanganate if necessary during the 2 hours in the 95 degree hot water bath.
6. Add 8 ml of Potassium Persulfate solution to each sample.
7. Heat for 2 hours in a water bath at 95 degrees C.
8. Cool and add 8 ml of Sodium Chloride-Hydroxylamine Hydrochloride solution to each flask.
9. After the sample is totally decolorized and no Permanganate is left in the bottle, the bottle is connected to the aerator and purged until the recorder pen returns to the baseline. Then add 5.0 ml of Stannous Chloride solution and purge until the pen reaches a maximum, then rinse. Rinse the aerator with 1 + 1 Nitric Acid into the sample bottle being removed from the aerator, then start on the next sample.

Analytical Quality Control

The State Water Quality Laboratory is commited to the conduct of a program which will insure accurate and valid data. Therefore, the fundamental agreement for analytical quality control of the data generated under the Basic Water Monitoring Program is approximately 20 percent of the laboratory effort. The total program includes both intra-laboratory procedures such as spiked sample recovery, replicate sample analyses, and reference sample analyses. The laboratory also conducts inter-laboratory analytical quality control procedures such as sample splitting between state and EPA laboratories (13, 14).

The analytical quality control methods on the fish tissue analyses included approximately one in ten samples being split and analyzed as duplicate samples. Thus, this particular sample had two spike analyses of known values run on it. Precision of the analyses is determined by the difference in the two different spike sample readings. The accuracy of the procedures is determined by the following equation:

$$\frac{\text{Spike analytical value - sample value}}{\text{Known spike value}}(100) = \text{Accuracy (or \% recovery)}$$

These data are then used to plot the appropriate quality assurance charts to insure the tests were in control. This type of quality assurance effort was reasonable and, at the same time, gave the laboratory staff the information they needed for insuring sample validity.

CHAPTER IV

RESULTS AND DISCUSSION

Toxic Metals in Fish

General

The data generated by this project were separated into the state of Oklahoma's two major drainage basins for subsequent evaluation. These basins consist of the Arkansas River which drains roughly the northern half of Oklahoma from west to east and the Red River which serves as the southern boundary of Oklahoma and drains roughly the southern half of the state from west to east. There were fifteen sampling sites on the Arkansas River and its tributaries and eight sampling sites on the Red River and its tributaries in Oklahoma. These sites are listed in Appendix A. It is also important to note that there are natural changes in the water quality of streams in Oklahoma as they flow from west to east. In general, the water quality of eastern Oklahoma streams is better than the water quality of western Oklahoma streams. For example, it is known that streams in western Oklahoma tend to have higher turbidity, lower flow, and more minerals than streams from eastern Oklahoma.

Raw Data Discussion

Table 2 presents the raw data from the fish samples which were analyzed in this study. These data are listed by sample site, water year, and the toxic metal content of the fish sample by herbivore and carnivore trophic level. Stations 1 through 15 represent sampling sites within the Arkansas River Basin while Stations 16 through 23 list the data from the Red River Basin. Appendix E shows the species of fish collected, and weights of the samples in Table 2.

These raw data are presented graphically in Appendix B, Figures B-1 through B-24. These graphs show the individual toxic metal within the individual river basin for Water Years 1977, 1978, 1979, and 1980. These figures show the data of the toxic metal (in Mg/Kg of body weight) in a temporal sequence by sample station location for the entire basin. The concentration ranges may be different on each figure due to both analytical detection limits and the level of the toxic metal detected in the fish. Any value which was less than the detection limit was recorded as a zero for the purpose of the graphical presentation of the data. Water years for which no data were available were also recorded as zero values. The data will be discussed from the graphs, which are a presentation of the information in Table 2. Lastly, reference to elevated levels of the individual toxic metals in the fish samples should be interpreted only as a technique to compare the data points within a river basin. These references to elevated levels refer only to a comparison with the mean levels for the individual basin under consideration and do not necessarily refer to any concentrations which would be detrimental to the fish or to persons consuming these fish.

Table 2. Toxic Metal Levels in Herbivore and Carnivore Fish by Station Location and Water Year (WY).

STATION	WY	Arsenic		Cadmium		Chromium		Copper		Lead		Mercury	
		HERB	CARN	HERB	CARN	HERB	CARN	HERB	CARN	HERB	CARN	HERB	CARN
1	1977			0.20	*0.10	*0.50	*0.50	6.20	22.00	0.70	*1.00	0.13	0.49
	1978	*0.10	3.00	0.20	*0.10	0.80	1.50	1.80	2.10	1.70	*1.00	*0.05	0.14
	1979			*0.10	*0.10	2.00	0.60	3.20	2.80	*1.00	2.00	0.24	*0.05
	1980		3.00	0.10	0.20	1.60	1.40	*0.40	0.20	*1.00	*1.00	0.15	0.18
2	1977			0.23	0.05	6.09	0.32	1.80		1.40	0.40	0.70	0.20
	1978	0.05	0.05	0.20	0.05	0.25	1.90	1.20	1.70	1.40	0.50	0.03	0.20
	1979	0.05	0.05	0.05	0.05	0.50	0.25	1.50	1.20	1.50	1.60	0.03	0.09
3	1977			0.10	*0.10	*0.50	*0.50	3.00	4.39	*1.00	*1.00	0.10	0.60
	1978	*0.10	*0.10	0.20	*0.10	*0.10	2.40	1.60	5.50	1.10	*1.00	0.06	0.22
	1979	3.70		0.40		2.50		1.30		*1.00		0.13	
	1980			0.20		1.00		*0.40		*1.00		*0.05	
4	1977			*0.10	0.10	*0.50	2.90	1.96	3.70	0.70	4.70	0.17	*0.05
	1978	0.20	2.00	*0.10	*0.10	*0.50	15.00	1.50	2.00	1.30	9.00	*0.05	*0.05
	1979		5.00	0.20	*0.10	1.30	0.90	0.30	*0.40	*1.00	*1.00	*0.05	0.20
	1980			0.10	*0.10	1.50	*0.50	*0.40	*0.40	*1.00	*1.00	0.27	*0.05
5	1977			0.05	0.05	0.25	1.30	11.50	5.80	4.00	1.80	0.13	0.17
	1978	1.70	0.05	0.10	0.30	0.25	1.60	1.50	2.00	1.90	1.70	0.27	0.09
	1979			0.05	0.05	0.25	0.25	1.20	0.60	2.80	0.50	0.09	0.03
	1980			0.20		1.40		0.20		0.50		0.09	
6	1977			0.05	0.13	0.25	1.30	0.20	1.21	0.50	1.10	0.28	0.30
	1978	0.05	0.05	0.10	0.20	0.60	0.25	2.20	1.50	1.30	1.30	0.03	0.03
	1979			0.20	0.05	0.09	0.25	1.50	0.20	3.50	0.05	0.16	0.05
	1980			0.10		0.75		0.80		0.50		0.19	

* = Less than Detection limit

Table 2. (cont.)

STATION	WY	Arsenic		Cadmium		Chromium		Copper		Lead		Mercury	
		HERB	CARN	HERB	CARN	HERB	CARN	HERB	CARN	HERB	CARN	HERB	CARN
7	1978	*0.10	*0.10	0.20	0.20	1.00	*0.50	1.70	1.50	1.50	1.20	*0.05	0.95
	1979			0.10	0.20	2.00	1.00	0.90	*0.40	*1.00	*1.00	0.05	0.17
	1980			0.20	0.10	*0.50	*0.50	*0.40	*0.40	*1.00	*1.00	0.12	0.19
8	1978	*0.10	*0.10	0.10	*0.10	4.10	1.60	2.20	2.50	1.90	*1.00	*0.05	0.09
	1979			*0.10	0..20	1.60	2.20	0.40	0.90	*1.00	*1.00	*0.05	4.80
9	1977			0.20	0.20	2.80	4.20	1.40	3.60	*1.00	*1.00	*0.05	*0.05
	1978	0.10	*0.10	0.10	*0.10	1.90	*0.50	2.10	0.70	2.90	*1.00	*0.05	*0.05
	1979				*0.10		*0.50		0.70		1.40		0.13
10	1979			*0.10	*0.10	0.50	2.00	1.00	1.70	*1.00	1.10	*0.05	0.12
	1980			*0.10		1.30		*0.40		*1.00		*0.05	
11	1977			*0.10	0.20	*0.50	2.60	18.00	3.90	3.10	2.20	0.06	*0.05
	1978	*0.10		0.20	*0.10	0.60	0.50	2.10	1.10	2.30	*1.00	0.06	0.16
	1979			0.20	0.40	0.50	1.50	*0.40	2.60	*1.00	*1.00	0.10	
	1980			*0.10		*0.50		*0.40		*1.00		*0.05	
12	1977			0.18		0.60		2.80		1.40		0.11	
	1978			*0.10	*0.10	1.50	1.00	2.90	0.70	1.30	1.10	0.12	0.10
	1980			0.20		1.00		*0.40		*1.00		*0.05	
13	1977			0.20	0.20	2.50	3.30	3.10	4.60	0.50	1.50	0.03	0.06
	1978	2.10		0.10		0.25		3.10		1.70		0.03	
	1979			0.05	0.20	1.00	1.40	1.90	2.60	0.50	0.50	0.11	0.26
	1980			0.50	0.15	5.80	0.25	0.26	2.50	1.20	0.50	0.03	0.40

* = Less Than Detection Limit

Table 2. (cont.)

STATION	WY	Arsenic		Cadmium		Chromium		Copper		Lead		Mercury	
		HERB	CARN	HERB	CARN	HERB	CARN	HERB	CARN	HERB	CARN	HERB	CARN
14	1977			0.12	0.20	0.25	2.60	2.90	1.90	1.50	1.50	0.08	0.06
	1978	0.70		0.10	0.05	0.80	1.70	42.00	3.50	1.30	0.50	0.09	0.09
	1979		1.00	0.05	1.00	2.10	1.00	7.00	1.00	2.00	1.00	0.03	1.00
	1980	3.00		0.10	0.10	0.25	0.15	0.20	0.20	0.50	0.50	0.07	0.05
15	1977			0.80	0.20	4.00	1.70	12.00	8.80	3.30	1.40	*0.05	*0.05
	1978	0.20	*0.10	*0.10	*0.10	*0.50	*0.50	1.30	1.30	*1.00	*1.00	*0.05	0.60
	1979			*0.10	0.20	2.10	*0.50	2.10	0.30	1.80	1.80	0.11	0.06
16	1977			*0.10	*0.10	*0.50	*0.50	11.00	4.30	*1.00	1.70	*0.05	*0.05
	1978	0.10	*0.10	0.20	0.10	*0.50	0.60	1.50	1.90	1.30	1.20	*0.05	*0.05
	1979			0.20	*0.10	1.40	1.50	1.60	0.40	*1.00	*1.00	0.06	*0.05
	1980			*0.10		3.10		0.40		1.60		0.10	
17	1977			*0.10	*0.10	8.44	*0.50	3.20	1.93	1.00	*1.00	0.30	0.20
	1978	*0.10		0.10		1.05		2.20		1.50		0.11	
	1979				0.30		1.50		*0.40		*1.00		0.14
18	1977			0.20		1.80		1.20		*1.00		*0.05	
	1978	1.40		*0.10	*0.10	0.80	0.90	2.30	2.30	0.90		*0.05	1.20
	1979			*0.10	*0.10	*0.50	*0.50	0.90	1.00	*1.00	*1.00	*0.05	*0.05
	1980			*0.10		0.50		0.40		*1.00		0.20	
19	1977			*0.10	0.30	*0.50	*0.50	0.90	1.40	*1.00	*1.00	*0.05	0.06
	1978	*0.10	1.80	*0.10	*0.10	0.80	1.00	1.30	3.00	1.30	1.30	0.70	2.10
	1979			*0.10		1.00		0.90		*1.00		*0.05	*0.05
	1980	2.00		0.20		1.30		*0.40		*1.00		*0.05	

* = Less Than Detection Limit

Table 2. (cont.)

STATION	WY	Arsenic		Cadmium		Chromium		Copper		Lead		Mercury	
		HERB	CARN	HERB	CARN	HERB	CARN	HERB	CARN	HERB	CARN	HERB	CARN
20	1977			0.10	0.20	3.70	3.20	3.50	7.,40	*1.00	1.80	*0.05	0.05
	1978	*0.10	*0.10	*0.10	*0.10	1.70	0.80	2.60	0.90	*1.00	*1.00	0.09	0.06
	1979				*0.10		0.50		1.10		*1.00		*0.05
21	1977			0.10	0.30	3.70	3.30	5.00	0.40	1.70	1.50	*0.05	*0.05
	1978			*0.10	*0.10	*0.50	0.80	1.50	2.00	0.70	1.50	0.49	0.62
	1979		2.40	0.20	0.20	1.30	1.10	0.40	1.30	*1.00	*1.00	0.14	0.12
22	1977			0.20	0.30	0.60	2.10	3.00	3.70	*1.00	*1.00	*0.05	*0.05
	1978	1.40	*0.10	*0.10	*0.10	1.40	1.80	3.20	3.00			*0.05	*0.05
	1979			*0.10	0.20	0.50	1.10	0.40	2.20	*1.00	*1.00	*0.05	0.20
23	1977			0.20	0.20	4.60	3.00	3.60	0.60	1.20	2.30	*0.05	*0.05
	1978	*0.10		0.10	*0.10	2.50	0.80	3.80	3.00	1.80	1.50	0.19	0.11
	1979			*0.10	*0.10	0.50	*0.50	0.40	0.50	*1.00	1.10	*0.05	*0.05
	1980			*0.10	*0.10	*0.50	*0.50	*0.40	*0.40	*1.00	*1.00	*0.05	0.16

* = Less Than Detection Limit

Arsenic Levels in Fish

Figure B-1 shows that arsenic levels in herbivores in the Arkansas River Basin are elevated at site 3 (Cimarron River near Buffalo) in 1979, site 5 (Arkansas River near Sand Springs) in 1978, site 13 (Canadian River near Bridgeport) in 1977, and site 14 (Canadian River near Calvin) in 1980. Figure B-2 shows that arsenic levels in the carnivores in the Arkansas River Basin were elevated at site 1 (Salt Fork of the Arkansas River near Jet) in 1978 and 1980, site 4 (Cimarron River near Perkins) in 1978 and 1979, and at site 14 (Canadian River Near Calvin in 1978. No obvious patterns of elevated arsenic were observed from these data.

Figure B-13 shows arsenic levels in herbivores in the Red River Basin to be elevated at site 18 (Washita River near Durwood) in 1978, site 19 (Muddy Boggy near Farris) in 1980, and site 22 (Red River near DeKalb) in1978. Figure B-14 shows the arsenic levels in carnivores to be elevated at site 19 (Muddy Boggy near Farris) in 1978, and site 21 (Kiamichi River near Antlers) in 1979. As was the case with the Arkansas River Basin, no patterns of elevated levels of arsenic were noted.

Cadmium Levels in Fish

Figure B-3 shows the cadmium levels in the herbivores in the Arkansas River Basin. There were elevated levels at site 3 (Cimarron River near Buffalo) in 1979, site 13 (Canadian River near Bridgeport) in 1980, and site 15 (Poteau River near Ft. Smith) in 1977. Figure B-4 shows the cadmium levels in the carnivores in the Arkansas River Basin.

Elevated levels were observed at site 5 (Arkansas River near Sand Springs) in 1978, site 11 (North Canadian River near El Reno) in 1979, and site 14 (Canadian River near Calvin) in 1979. No obvious pattern to these data were observed.

Figure B-15 shows the cadmium levels in the herbivores in the Red River Basin. No elevated levels of cadmium were noted in this figure. Figure B-16 shows the cadmium levels in the carnivores in the Red River Basin. Elevated cadmium levels were noted at site 17 (Red River near Terral) in 1979, site 19 (Muddy Boggy near Farris) in 1977, site 21 (Kiamichi River near Antlers) in 1977, and site 22 (Red River near DeKalb) in 1977. No obvious patterns of elevated cadmium were observed from these data.

Chromium Levels in Fish

Figure B-5 shows the chromium levels in herbivores in the Arkansas River Basin. Elevated levels were noted at site 2 (Arkansas River near Ralston) in 1977, site 8 (Illinois River near Tahlequah) in 1978, site 9 (Illinois River near Gore) in 1977, site 13 (Canadian River near Bridgeport) in 1977, and site 15 (Poteau River near Ft. Smith) in 1977 and 1979. Figure B-6 shows the chromium levels in the carnivores in the Arkansas River Basin. Samples were elevated at site 4 (Cimarron River near Perkins) in 1977 and 1978, site 9 (Illinois River near Gore) in 1977, site 11 (North Canadian River near El Reno) in 1977, and site 13 (Canadian River near Bridgeport) in 1977. The only observed pattern to chromium levels in fish in the Arkansas River Basin were elevated levels at site 4 for consecutive Water Years 1977 and 1978. These

levels were lower in 1977 and 1980.

Figure B-17 shows the chromium levels in herbivores in the Red River Basin. Elevated levels were observed at site 16 (North Fork of the Red near Headrick) in 1980, site 17 (Red River near Terral) in 1977, site 20 (Kiamichi River near Big Cedar) in 1977, site 21 (Kiamichi River near Antlers) in 1977, and site 23 (Little River near Idabel) in 1977 and 1978. No obvious patterns to the data were observed.

Figure B-18 shows the chromium levels in the carnivores in the Red River Basin. Elevated levels were observed at site 20 (Kiamichi River near Big Cedar) in 1977, site 21 (Kiamichi River near Antlers) in 1977, site 22 (Red River near DeKalb) in 1977, and site 23 (Little River near Idabel) in 1977. Both figures B-17 and B-18 indicated higher levels of chromium in fish in the Red River Basin during Water Year 1977.

Copper Levels in Fish

Figure B-7 shows the copper levels for the herbivores in the Arkansas River Basin. Elevated levels were noted at site 1 (Salt Fork of the Arkansas near Jet) in 1977 and 1979, site 5 (Arkansas River near Sand Springs) in 1977, site 11 (North Canadian River near El Reno) in 1977 and 1978, site 12 (North Canadian River near Harrah) in 1977 and 1978, site 13 (Canadian River near Bridgeport) in 1977 and 1978, site 14 (Canadian River near Calvin) in 1977, 1978, and 1979, and site 15 (Poteau River near Ft. Smith) in 1977 and 1979. These data indicate that copper was present in a large number of herbivore fish samples analyzed in the Arkansas River Basin.

Figure B-8 shows the copper levels in the carnivores in the Arkansas River Basin. Elevated levels were noted at site 1 (Salt Fork of the Arkansas near Jet) in 1977, site 5 (Arkansas River near Sand Springs) in 1977, site 9 (Illinois River near Gore) in 1977, site 11 (North Canadian River near El Reno) in 1977 and 1979, site 13 (Canadian River near Bridgeport) in 1977, site 14 (Canadian River near Calvin) in 1978, and site 15 (Poteau River near Ft. Smith) in 1977. These data indicate the presence of copper in carnivores at several sampling sites but no observable trends were noted.

Figure B-19 shows the raw data for the copper levels in the herbivores in the Red River Basin. Elevated levels of copper were noted at site 16 (North Fork of the Red near Terral) in 1977 and 1978, site 20 (Kiamichi River near Big Cedar) in 1977 and 1978, site 21 (Kiamichi River near Antlers) in 1977, site 22 (Red River near DeKalb) in 1977 and 1978, and site 23 (Little River near Idabel) in 1977 and 1978.

Figure B-20 shows the raw data for copper levels in; the carnivores in the Red River Basin. Elevated levels were noted at site 16 (North Fork of the Red near Headrick) in 1977, site 19 (Muddy Boggy near Farris) in 1978, site 20 (Kiamichi River near Big Cedar) in 1977, Site 21 (Kiamichi River near Antlers) in 1978, site 22 (Red River near DeKalb) in 1977 and 1978, and site 23 (Little River near Idabel) in 1978. These data point out the presence of copper in a number of the carnivore fish sampled in the Red River Basin.

Lead Levels in Fish

Figure B-9 shows the lead content of the herbivores collected

from the Arkansas River Basin. These data show the presence of lead in herbivores at most of the monitoring stations in the Arkansas River Basin. Elevated lead levels were noted at site 5 (Arkansas River near Sand Springs) in 1977, 1978, and 1979, site 6 (Arkansas River near Haskell) in 1979, site 9 (Illinois River near Gore) in 1978, site 11 (North Canadian River near El Reno) in 1977 and 1978, and site 15 (Poteau River near Ft. Smith) in 1977.

Figure B-10 shows the lead content of the carnivores in the Arkansas River Basin. Elevated levels were noted at site 1 (Salt Fork of the Arkansas near Jet) in 1979, site 4 (Cimarron River near Perkins) in 1977 and 1978, and site 11 (North Canadian River near El Reno) in 1977. The carnivores, like the herbivores in the Arkansas River Basin have measurable levels of lead in their tissue.

Figure B-21 shows the lead content of the herbivores in the Red River Basin. Elevated levels were noted at site 16 (North Fork of the Red near Headrick) in 1978 and 1980, site 17 (Red River near Terral) in 1978, site 19 (Muddy Boggy near Farris) in 1977 and 1978, site 21 (Kiamichi River near Antlers) in 1977, and site 23 (Little River near Idabel) in 1977 and 1978. The herbivores in the Red River Basin had measureable levels of lead but were lower than the fish sampled in the Arkansas River Basin.

Figure B-22 shows the lead content of the carnivores in the Red River Basin. Elevated levels were noted at site 16 (North Fork of the Red near Headrick) in 1977, site 20 (Kiamichi River near Big Cedar) in 1977, and site 23 (Little River near Idabel) in 1977 and 1978. The carnivores, also had lower levels of lead than the samples from the

Arkansas River Basin.

Mercury Levels in Fish

Figure B-11 shows the mercury content of the Herbivores in the Arkansas River Basin. Elevated levels were noted at site 2 (Arkansas River near Ralston) in 1977, site 4 (Cimarron River near Perkins) in 1980, site 5 (Arkansas River near Sand Springs) in 1978, and site 6 (Arkansas River near Haskell) in 1977. No pattern of elevated mercury levels were noted in the herbivores in the Arkansas River Basin.

Figure B-12 shows the mercury content of the carnivores in the Arkansas River Basin. Elevated levels were noted at site 1 (Salt Fork of the Arkansas near Jet) in 1977, site 3 (Cimarron River near Buffalo) in 1977, site 6 (Arkansas River near Haskell) in 1977, site 7 (Bird Creek near Catoosa) in 1978, site 8 (Illinois River near Tahlequah) in 1979, site 13 (Canadian River near Bridgeport) in 1979 and 1980, site 14 (Canadian River near Calvin) in 1978, and site 15 (Poteau River near Ft. Smith) in 1978. Several samples of carnivores were observed to have elevated mercury levels in the Arkansas River Basin. As with other metals, no observed patterns were obvious from the data base available from this study. Additional monitoring should be done to verify these results before undue concern is expressed.

Figure B-23 shows the mercury content of the herbivores from the Red River Basin. Elevated levels were observed at site 17 (Red River near Terral) in 1977, site 19 (Muddy Boggy near Farris) in 1978, and site 21 (Kiamichi River near Antlers) in 1978. No obvious patterns to the data were noted.

Figure B-24 shows the mercury content of the carnivores from the Red River Basin. Elevated levels were noted at site 19 (Muddy Boggy near Farris) in 1978 and site 21 (Kiamichi River near Antlers) in 1978. Although the mercury level in the carnivores at site 18 and 19 is considered to be very high, the values were low at the same location the preceeding or following year.

Relationship of Toxic Metal Content to Trophic Level

Arkansas River Basin. Figures B-1 through B-12 show the toxic metals data for the Arkansas River Basin. These data do not indicate a general trend of one trophic level having higher metal levels than the other. The one exception in the Arkansas River Basin is the mercury levels. There are several instances where the mercury values are higher in the carnivores than in the herbivores. The levels in the carnivores, however, cannot be directly related to the levels in the herbivores.

Appendix C, Table C-1 shows the mean, standard deviation, minimum, and maximum values for the toxic metals in the Arkansas River Basin. These basic statistics indicate no further relationships between the herbivores and carnivores than the raw data analyses presented in the graphs. The mean values for the toxic metals are very similiar for all the metals except mercury. The mean mercury level for herbivores is 0.098 Mg/Kg, while the mean value for the carnivores is 0.305 Mg/Kg. These values show a close relationship with the data from the graphs.

Red River Basin. Figures B-12 through B-24 show the toxic metal data for the Red River Basin. These data do not indicate a general trend of one trophic level having higher toxic metals than the

other. The one exception in the Red River is chromium. This metal is more elevated in the herbivores than in the carnivores. The levels in the herbivores, however do not appear to be directly related to the levels in the carnivores.

Appendix C, Table C-2 shows the mean, standard deviation, minimum, and maximum values for the toxic metals in the Red River Basin. These basic statistics show no more information about the relationships between the herbivores and carnivores than the raw data analyses presented in the graphs. The mean levels for the toxic metals are very similar for all metals including chromium values. These date do not show any significant relationship to the toxic metals in the herbivores as compared to the levels in the carnivores.

Toxic Metals in Water

Table 3 shows the raw data for the toxic metal analyses of water samples which were collected at the same locations as the fish samples. These data represent an average concentration of four values for each Water Year. A preliminary look at these data indicate low concentrations of toxic metals in the water column. Additionally, Appendix C shows the results of the calculations of the mean, standard deviation, minimum, and maximum values for the individual toxic metals in fish, water, and sediment. Table C-1 lists the basic statistics for the Arkansas River Basin while Table C-2 shows the same data for the Red River Basin. These statistics also point out the low toxic metal content of the water samples collected and analyzed from the monitoring stations.

Table 3. Toxic Metal Data in Water (Reported in Mg/L) by Station Location and Water Year.

Site	Year	Arsenic	Cadmium	Chromium	Copper	Lead	Mercury
1	1977	* 0.001	0.010	0.045	0.026	0.039	* 0.001
	1978	0.003	0.015	0.013	0.019	0.035	* 0.001
	1979	* 0.001	0.008	0.010	0.015	0.040	* 0.001
	1980		0.011	0.011	0.015	0.020	* 0.001
2	1977	0.008	0.003	0.029	0.013	0.013	* 0.001
	1978	0.003	0.008	0.010	0.011	0.062	* 0.001
	1979	* 0.001	0.003	0.010	0.008	0.024	* 0.001
3	1977	0.014	0.008	0.018	0.030	* 0.001	* 0.001
	1978	0.003	0.002	0.021	0.024	0.029	* 0.001
	1979	* 0.001	0.020	0.310	0.033	* 0.001	0.165
	1980	0.039	0.002	0.013	0.007	0.020	* 0.001
4	1977	* 0.001			0.015		0.100
	1978	0.009	0.003	0.025	0.019	0.053	* 0.001
	1979	* 0.001	0.002	0.030	0.009	* 0.001	0.020
	1980	0.001	0.002	0.010	0.013	0.020	* 0.001
5	1977	* 0.001	0.001		0.010	0.001	0.100
	1978	0.003	0.008	0.017	0.012	0.055	* 0.001
	1979	* 0.001	* 0.001	0.020	0.010	* 0.001	0.160
	1980	0.005	0.002	0.018	0.006	0.047	* 0.001
6	1977	* 0.001	0.002	0.032	0.007	0.012	* 0.001
	1978	0.004	0.001	0.027	0.010	0.051	* 0.001
	1979	* 0.001	0.002	0.016	0.009	0.022	* 0.001
	1980	* 0.001	0.002	0.010	0.006	0.036	* 0.001
7	1978	0.001	0.003	0.017	0.012	0.023	0.001
	1979	* 0.001	0.005	0.024	0.008	0.029	* 0.001
	1980	* 0.001	0.002	0.012	0.007	0.030	* 0.001
8	1978	* 0.001	0.001	0.016	0.006	0.015	* 0.001
	1979	* 0.001	0.002	0.010	0.003	0.015	* 0.001
9	1977	0.001	0.001	0.015	0.005	0.009	* 0.001
	1978	0.001	0.001	0.008	0.003	0.011	* 0.001
	1979	* 0.001	0.002	0.015	0.003	0.009	* 0.001
10	1979						
	1980		0.002	0.024	0.012	0.020	* 0.001

* = Less Than Detection Limit

Table 3. (cont.)

Site	Year	Arsenic	Cadmium	Chromium	Copper	Lead	Mercury
11	1977	* 0.001	0.002	0.022	0.005	0.017	* 0.001
	1978	0.004	0.001	0.014	0.004	0.026	* 0.001
	1979	* 0.001	0.002	0.012	0.015	0.012	* 0.001
	1980	* 0.001	0.005	0.010	0.004	0.020	* 0.001
12	1977	* 0.001	0.003	0.032	0.011	0.022	* 0.001
	1978	0.007	0.001	0.010	0.009	0.070	* 0.001
	1980	* 0.001	0.002	0.012	0.009	0.020	* 0.001
13	1977	* 0.001	0.002	0.034	0.021	0.028	* 0.001
	1978	0.002	0.002	0.011	0.005	0.025	* 0.001
	1979	* 0.001	0.002	0.011	0.010	0.035	* 0.001
	1980	0.008	0.005	0.014	0.011	0.020	* 0.001
14	1977	0.006	0.008	0.010	0.011	0.063	* 0.001
	1978	0.005	0.002	0.018	0.016	0.025	* 0.001
	1979	* 0.001	* 0.001	0.040	0.011	* 0.001	0.054
	1980	* 0.001	0.005	0.018	0.010	0.051	* 0.001
15	1977	* 0.001	0.003	0.028	0.004	0.008	* 0.001
	1978	0.004	0.001	0.025	0.013	0.031	* 0.001
	1979	* 0.001	0.002	0.036	0.012	0.030	* 0.001
16	1977	* 0.001		0.013	0.018	* 0.001	0.100
	1978	0.003	0.009	0.010	0.010	0.010	* 0.001
	1979	* 0.001	0.003	0.030	0.006	* 0.001	* 0.001
	1980	0.015	0.003	0.013	0.017	0.020	* 0.001
17	1977	* 0.001	0.004	0.063	0.034	0.038	* 0.001
	1978						
	1979		0.002	0.014	0.017	0.020	* 0.001
18	1977	* 0.001			0.010	* 0.001	0.100
	1978	0.004	* 0.001	0.053	0.012	0.021	* 0.001
	1979	* 0.001	* 0.001	0.030	0.016	0.046	* 0.001
	1980	* 0.001	0.002	0.014	0.007	0.020	* 0.001
19	1977	* 0.001	0.001	0.035	0.008	0.008	* 0.001
	1978	0.001	0.001	0.021	0.008	0.009	* 0.001
	1979	* 0.001	0.002	0.014	0.005	0.009	* 0.001
	1980	0.023	0.003	0.024	0.021	0.021	* 0.001
20	1977	0.001	0.001	0.012	0.001	0.002	* 0.001
	1978	* 0.001	0.002	0.006	0.002	0.030	0.001
	1979	* 0.001	0.002	0.013	0.004	0.020	* 0.001

* = Less Than Detection Limit

Table 3. (cont.)

Site	Year	Arsenic	Cadmium	Chromium	Copper	Lead	Mercury
21	1977	* 0.001	0.001	0.016	0.004	0.025	0.001
	1978	0.001	0.001	0.014	0.004	0.007	0.001
	1979	0.033	0.002	0.012	0.003	0.008	0.001
22	1977	* 0.001	* 0.001	0.016	0.009	* 0.001	0.020
	1978						
	1979	* 0.001	* 0.001	* 0.010	* 0.001	* 0.001	* 0.001
23	1977	* 0.001	* 0.001	0.011	0.006	* 0.001	* 0.001
	1978	0.001	0.008	0.016	0.006	0.115	* 0.001
	1979	* 0.001	0.004	0.022	0.004	* 0.001	* 0.001
	1980	* 0.001	* 0.001	0.010	0.004	* 0.001	* 0.001

* = Less Than Detection Limit

Toxic Metals in Sediment

Table 4 shows the raw data for the toxic metal analyses of the sediment samples which were collected and analyzed at the same time and locations as the fish samples. A preliminary review of these data indicate high toxic levels are present at several monitoring sites. Additionally, Appendix C shows the results of calculations of the mean, standard deviation, minimum, and maximum values for the individual toxic metals in fish, water, and sediment for both the Arkansas and Red River Basins. Table C-1 shows the basic statistics for the Arkansas River Basin. All of the sediment samples had elevated mean toxic metal levels.

Table C-2 shows the basic statistics for the Red River Basin. As was noted in the Arkansas River Basin data, the sediment samples have elevated mean values. It should also be noted that the standard deviations on some of the parameters, especially the metals data were very high. This situation is not considered that unusual for the sediment grab samples collected in this project.

Total Hardness, pH, and Flow

Table 5 presents the raw data for total hardness, pH, and flow by station location and Water Year. The samples for these analyses were collected at several different times of the year. The average values for total hardness and pH are summarized in the table. Additional basic statistics are presented in Appendix C, Table C-3.

Table 4. Toxic Metal Data in Sediment (Reported in Mg/Kg) by Station Location and Water Year.

Site	Year	Arsenic	Cadmium	Chromium	Copper	Lead	Mercury
1	1977	4.00	* 1.00	12.00	8.00	3.00	13.00
	1978	3.00	* 1.00	19.00	6.00	* 1.00	17.00
	1979	* 2.00	* 1.00	5.00	* 2.00	* 1.00	
	1980	3.30	2.00	19.00	7.00	* 1.00	12.00
2	1977						
	1978	* 2.00	1.00	15.00	* 2.00	* 1.00	* 5.00
	1979	* 2.00	* 1.00	* 5.00	* 2.00	* 1.00	
3	1977	128.00	* 1.00	13.00	2.00	* 1.00	
	1978	* 2.00	1.00	11.00	5.00	* 1.00	
	1979						
	1980	* 2.00	1.00	* 5.00	* 2.00		* 5.00
4	1977	3.00	* 1.00	24.00	9.00	* 1.00	11.00
	1978	2.00	* 1.00	15.00	2.00	* 1.00	9.00
	1979	* 2.00	* 1.00	13.00	3.00	1.27	
	1980	* 2.00	* 1.00	* 5.00	* 2.00		* 5.00
5	1977	2.60	* 1.00	17.00	4.00	* 1.00	* 5.00
	1978	* 2.00	1.00	18.00	* 2.00	* 1.00	* 5.00
	1979	2.00	1.00	* 5.00	* 2.00	* 1.00	
	1980	* 2.00	* 1.00	6.00	* 2.00	13.00	* 5.00
6	1977	* 2.00	* 1.00	13.00	3.00	* 1.00	* 5.00
	1978	* 2.00	2.00	32.00	4.00	* 1.00	* 5.00
	1979	* 2.00	1.00	* 5.00	* 2.00	* 1.00	
	1980	* 2.00	* 1.00	* 5.00	* 2.00	* 1.00	
7	1978	* 2.00	2.00	27.00	19.00	34.00	8.00
	1979	* 2.00	* 1.00	5.00	3.00	* 1.00	
	1980						
8	1978	2.20	2.00	45.00	* 2.00	* 1.00	* 5.00
	1979						
9	1977	285.00	* 1.00	21.00	2.00	* 1.00	* 5.00
	1978	2.10	* 1.00	80.00	2.00	* 1.00	* 5.00
	1979	4.00	1.00	22.00	6.00	16.00	11.00
10	1979						
	1980	* 2.00	* 1.00	6.00	* 2.00	* 1.00	
11	1977	* 2.00	* 1.00	5.00	* 2.00	7.00	* 5.00
	1978	* 2.00	* 1.00	15.00	* 2.00	* 1.00	* 5.00
	1979	* 2.00	* 1.00	* 5.00	* 2.00	* 1.00	
	1980	3.60	2.00	* 5.00	* 2.00	* 1.00	

* = Less Than Detection Limit

Table 4. (cont.)

Site	Year	Arsenic	Cadmium	Chromium	Copper	Lead	Mercury
12	1977	13.00	* 1.00	9.00	9.00	19.00	15.00
	1978	* 2.00	1.00	19.00	* 2.00	* 1.00	9.00
	1980	* 2.00	3.00	* 5.00	* 2.00	* 1.00	
13	1977	6.00	* 1.00	* 5.00	* 2.00	* 1.00	* 5.00
	1978	* 2.00	1.50	16.00	* 2.00	* 1.00	* 5.00
	1979	* 2.00	* 1.00	5.00	* 2.00	* 1.00	
	1980		* 1.00	* 5.00	* 2.00	* 1.00	
14	1977	3.60	* 1.00	28.00	20.00	* 1.00	50.00
	1978	* 2.00	* 1.00	8.00	* 2.00	* 1.00	* 5.00
	1979	* 2.00	1.00	6.00	* 2.00	* 1.00	
	1980	32.00	1.00	10.00	7.00	12.00	* 5.00
15	1977	524.00	* 1.00	46.00	14.00	* 1.00	25.00
	1978	2.40	* 1.00	56.00	6.00	* 1.00	16.00
	1979	24.00	* 1.00	11.00	6.00	* 1.00	29.00
16	1977	13.00	* 1.00	9.00	9.00	19.00	15.00
	1978	* 2.00	1.00	19.00	* 2.00	* 1.00	9.00
	1980	* 2.00	3.00	* 5.00	* 2.00	* 1.00	
17	1977	12.00	* 1.00	10.00	3.00	13.00	5.00
	1978	* 2.00	* 1.00	20.00	8.00	* 1.00	13.00
	1979	* 2.00	2.00	9.00	* 2.00	* 1.00	
18	1977	21.00	* 1.00	11.00	3.00	* 1.00	6.00
	1978	4.80	1.00	6.00	2.00	* 1.00	5.00
	1979	2.00	1.00	8.00	3.00	* 1.00	
	1980	2.30	* 1.00	* 5.00	2.00	* 1.00	
19	1977	12.00	* 1.00	7.00	* 2.00	* 1.00	* 5.00
	1978	* 2.00	1.00	42.00	* 2.00	* 1.00	* 5.00
	1979	2.20	* 1.00	5.00	* 2.00	* 1.00	
	1980	* 2.00	* 1.00	* 5.00	* 2.00	* 1.00	
20	1977	9.00	* 1.00	36.00	9.00	3.60	25.00
	1978	* 2.00	* 1.00	28.00	* 2.00	* 1.00	* 5.00
	1979	35.00	* 1.00	18.00	10.00	* 1.00	16.00
21	1977	230.00	* 1.00	30.00	7.00	* 1.00	15.00
	1978	* 2.00	* 1.00	59.00	4.00	* 1.00	12.00
	1979	9.00	1.00	18.00	9.00	* 1.00	52.00
22	1977	105.00	* 1.00	23.00	5.00	* 1.00	10.00
	1978	* 2.00	1.00	90.00	* 2.00	* 1.00	* 5.00
	1979	* 2.00	1.00	10.00	4.00	* 1.00	

* = Less Than Detection Limit

Table 4. (cont.)

Site	Year	Arsenic	Cadmium	Chromium	Copper	Lead	Mercury
23	1977	154.00	* 1.00	14.00	3.00		5.00
	1978	* 2.00	2.00	67.00	* 2.00	* 1.00	* 5.00
	1979	3.00	* 1.00	* 5.00	* 2.00	* 1.00	
	1980	22.50	* 1.00	23.00	* 2.00	* 1.00	34.00

* = Less Than Detection Limit

Table 5. Total Hardness, pH, and Flow by Station Location and Water Year.

Site	Water Year	pH	Total Hardness	Flow (CFS)
1	1977	8.4	681	67
	1978	8.4	532	63
	1979	8.1	772	298
	1980	8.1	604	182
2	1977	8.3	249	11310
	1978	8.4	264	6724
	1979	7.5	326	4037
3	1977	8.2	517	142
	1978	8.1	545	703
	1979	6.9		423
	1980	8.0	1472	
4	1977	8.4	665	1119
	1978	8.2	693	1781
	1979			833
	1980	7.6	613	
5	1977	8.1		
	1978	7.7	230	13230
	1979			6822
	1980	7.5	570	7728
6	1977	8.2	244	9362
	1978	8.1	226	4942
	1979	8.1	272	6561
	1980	7.7	246	7080
7	1978	7.4	164	
	1979	7.3	140	
	1980	7.1	233	
8	1978	7.1	100	422
	1979	7.2	110	333
9	1977	7.6	15	324
	1978	7.8	127	1590
	1979	7.6	93	545
10	1979			110
	1980	7.6	333	43
11	1977	8.3	455	35
	1978	8.2	366	30
	1979	8.0	413	270
	1980	7.2	427	145

Table 5. (cont.)

Site	Water Year	pH	Total Hardness	Flow (CFS)
12	1977	7.9	306	157
	1978	8.1	289	141
	1980	7.5	361	451
13	1977	8.3	573	77
	1978	8.3	344	8
	1979	8.2		500
	1980	7.9	683	7
14	1977	8.2	319	1262
	1978	8.2	330	2071
	1979			1348
	1980	7.8	134	57
15	1977	7.1	38	
	1978	7.1	47	
	1979	7.5	28	
16	1977	8.0	1118	113
	1978	7.9	1192	80
	1979			120
	1980	7.9	860	27
17	1977	8.3	870	977
	1978	7.7	934	487
	1979			996
18	1977	8.1	274	431
	1978	8.1	500	392
	1979			761
	1980	7.7	605	911
19	1977	7.6	59	60
	1978	7.5	86	13
	1979	7.3	78	89
	1980	7.2	102	2
20	1977	7.1		1
	1978	7.3	8	13
	1979	7.2		43
21	1977	7.0	28	48
	1978	7.3	20	182
	1979	7.1	19	223
22	1977	7.8	255	3358
	1978			3101
	1979			4551

Table 5. (cont.)

Site	Water Year	pH	Total Hardness	Flow (CFS)
23	1977	6.7	25	94
	1978	7.4	30	333
	1979	7.0	32	407
	1980	7.3	32	26

Total Hardness

The concentrations for total hardness represent the mean values for all of the data for the entire year. The data from the Arkansas River Basin and the Red River Basin can be discussed together since the basic statistics presented in Table C-3 are very similar for both basins. The range of hardness is more a function of eastern and western Oklahoma than of the Arkansas or Red River Basins. The most important item to be noted from these data is that in most of the sampling locations, sufficient hardness is present to allow for adequate buffering capacity with respect to the toxic properties of the toxic metals being addressed in this paper.

pH

As with the hardness data, the pH values presented in Table 5 represent mean values of all the data for a water year that were available. The data from the Arkansas and Red River Basins are similar enough to warrant discussing them together. The major function of pH differences relates mostly to western or eastern Oklahoma rather than the Arkansas or Red River Basin. The statistics are presented in Table C-3 and indicate the minimum value reported was 7.5 in the Arkansas River Basin and and 6.7 in the Red River basin. These data point out that the pH of the water should not increase the toxicity of the metals to the fish.

Flow

The flow data included herein were obtained from the United States Geological Survey Surface Water Records for Oklahoma for Water

Year 1977 and 1978 (135,136). The flow data for Water Year 1979 and 1980 were not published at the time of this writing. Since complete flow records were not available, and since partial flow data could be obtained verbally, the decision was made to use only the flow data which corresponded to the most critical flow periods at the time the fish samples were collected. Therefore, only the flow data for the month of July were used in Table 5. The flow data for Water Year 1979 and 1980 are subject to change but will be published at a later data by the United States Geological Survey (137).

Table C-3 shows the basic statistics for the flow data. As would be expected, the flow data for all of the streams represented in this study are extremely variable. This variation is also pointed out by the high standard deviation of the data. Additional study needs to be done to address the effects of flow on the transport and deposition of sediments as well as the relationship of flow to the toxic metals in the water column and the sediments and the ultimate impact on the fish populations.

Data Correlation

One concern of this study of the toxic metals in natural fish populations in Oklahoma is the presence or absence of any cause and effect relationships between any of the data that were generated. In order to determine the effect one variable had on another, a series of Pearson moment correlation coefficients were run between paired data sets. Appendix D presents the results of selected correlations. For the purpose of this discussion, a correlation of 0.50 will be taken as

an indication that these relationships might be correlated.

Metals in Water to Metals in Fish

Table D-1, Appendix D shows the correlation coefficients of the toxic metals in water to the toxic metals in fish in the Arkansas River Basin. The relationships with correlations greater than 0.50 were: cadmium to arsenic in herbivores, 0.53; chromium to arsenic in herbivores, 0.65; mercury to arsenic in herbivores, 1.00; and mercury to mercury in herbivores, 0.70. A negative correlation of -0.66 for mercury to mercury in carnivores. The exact reasons for the pattern of these correlations is not known. The perfect correlation of 1.00 indicates that this interrelationship should be explored further.

Table D-2 shows the correlation coefficients of the toxic metals in the water to toxic metals in the fish in the Red River Basin. The relationships with correlations greater thkan 0.50 included: arsenic to arsenic in herbivores, 0.84; arsenic to arsenic in carnivores, 0.65; arsenic to cadmium in herbivores, 0.59; arsenic to cadmium in carnivores, 0.60; chromium to arsenic in herbivores, 0.63; chromium to arsenic in carnivores, 0.64; copper to arsenic in herbivores, 0.88; and, mercury to copper in carnivores, 0.79. Negative correlations greater than the 0.50 level included: arsenic to copper in herbivores, -0.70; arsenic to lead in carnivores, -0.90; cadmium to arsenic in carnivores, -0.58; and, lead to arsenic in carnivores, -0.62. As was stated earlier, these results may be somewhat misleading; but, it appears that the levels of toxic metals in the water is related to the presence of toxic metals in the fish samples.

Metals in Sediment to Metals in Fish

Table D-1 also shows the correlation coefficients of the toxic metals in the sediment samples to the toxic metals in the fish samples from the Arknasas River Basin. The correlations greater than 0.50 were: arsenic to arsenic in herbivores, 0.70; arsenic to cadmium in herbivores, 0.77; arsenic to chromium in herbivores, 0.51; mercury to copper in herbivores, 0.63; and, mercury to lead in herbivores, 0.74. These data indicate that there seems to be less correlation of metals in sediment to the metals in fish than metals in the water samples to the metals in the fish in the Arkansas River Basin.

Table D-2 also shows the correlation coefficients of the toxic metals in the sediment samples to the toxic metals in fish in the Red River Basin. Those relationships with correlations greater than 0.5 included: arsenic to arsenic in carnivores, 0.74; arsenic to cadmium in carnivores, 0.52; arsenic to chromium in carnivores, 0.67; copper to arsenic in carnivores, 0.74; and, lead to chromium in herbivores, 0.85. It should also be noted that a negative correlation of -1.00 was observed between mercury in the sediment to arsenic in the herbivores. The reason for this is not known. Generally, there appears to be less correlation of toxic metals in sediment to toxic metals in fish in the Red River Basin.

Metals in Water to Metals in Sediment

Table D-3, Appendix D, shows the correlation coefficients which relate toxic metals in the water to the toxic metals in the sediment. Correlations in the Arkansas River Basin above 0.50 included: mercury

in water to arsenic in sediment, 0.70 and mercury in water to mercury in sediment, 0,50. Negative correlations greater than 0.50 were: mercury in water to copper in sediment, -0.53; and mercury in water to lead in sediment, -0.60. These data point out the fact that the presence of mercury in water may have a relationship on the concentration of other toxic metals in the sediment.

Table D-3 also shows the correlation coefficients for the toxic metals in water to toxic metals in sediment in the Red River Basin. Correlations greater than 0.50 included: arsenic in water to mercury in sediment, 0.88; chromium in water to lead in sediment, 0.62; copper in water to lead in sediment, 0.64; and, lead in water to cadmium in sediment, 0.56. Negative correlations greater than 0.50 included: arsenic in water to chromium in sediment, -0.50; lead in water to mercury in sediment, -0.57; mercury in water to chromium in sediment, -0.69; and, mercury in water to copper in sediment, -0.52. These relationships are not high enough to indicate direct correlations. They should be looked at more closely in future toxics work.

Total Hardness, pH, and Flow to Toxic Metals in Fish

Tables D-1 and D-2 show the correlation coefficients for pH, total hardness, and flow to the toxic metal levels in the fish samples from the Arkansas and Red River Basins. Coefficients greater than 0.50 in the Arkansas River Basin included: pH to arsenic in carnivores, 0.52 and total hardness to arsenic in carnivores, 0.75. Negative correlations calculated for the Red River Basin were: pH to arsenic in herbivores, -0.59, and total hardness to arsenic in herbivores, -0.54.

The reason for the positive correlations in the Arkansas River Basin and the negative correlations in the Red River Basin is not known. The main point of these data is that there does not appear to be any defensible correlation of total hardness, pH, and flow to the levels of toxic metals in natural fish populations in Oklahoma.

CHAPTER V

CONCLUSIONS AND RECOMMENDATIONS

This research project was designed to investigate the toxic metal levels in the natural fish populations in Oklahoma. Many questions arose throughout the course of the project that need to be examined in more depth and detail. It is hoped that this preliminary work will result in water quality management agencies in Oklahoma implementing a more comprehensive and detailed research program to adequately address the question of the levels and effects of toxic metals on the natural fish populations in Oklahoma.

Conclusions

1. This project should be viewed as a base line study of the toxic metals in natural fish populations in Oklahoma. The concentrations of toxic metals in the fish samples analyzed were low. A few cases of elevated toxic metals in the fish samples were observed but no consistent patterns were detected. For the purpose of this project the term elevated referred only to a comparison of the mean values for the individual basin under consideration and do not necessarily refer to concentrations which are detrimental to the fish.

Due to the scope of this project, it was not possible to determine the sources causing these slightly elevated levels.

2. The data generated by this project were difficult to interpret due to the low concentrations of toxic metals observed in the fish and water samples. These low levels of toxic metals in the fish and water samples indicated that toxic metal pollution did not appear to be a significant problem over a large portion of Oklahoma. The acute, toxic effects of metals was not a problem. It is very difficult to evaluate the chronic effects of the low levels of toxic metals. The chronic effects of sub-lethal doses of toxic metals on fish is not well doccumented in the literature.

3. The toxic metals measured in the sediment samples collected at the same time the fish samples were collected showed elevated levels at many of the sites studied.

4. The pH and total hardness of the water samples were observed to be within levels which generally do not affect the toxicity of metals to the natural fish populations.

5. No significant correlation coefficients were observed between the flow measurements and the levels of toxic metals observed in the fish samples. This is to be expected because of the low levels of toxic metals observed in the fish samples and the low flow conditions.

6. There were virtually no significant correlations observed between the toxic metal levels in the water samples and the levels observed in the sediment samples due to the absorbtive

capacity of the sediments. The release of the metals from the sediments into the water would be very slow.

Recommendations

1. Additional work should be undertaken to determine the correlations of toxic metals content between all of the components of the aquatic ecosystem (fish, water, and sediment). In order to accomplish this objective, analytical methods need to be developed which will increase the speed and decrease the cost of fish and sediment analysis. More sensitive analytical procedures for toxic metal analysis also need to be developed. Lower detection limits would allow for better use of correlation statistics.
2. Research programs which will allow for more effort in the collection and analysis of toxic metals in fish, sediment, and water should be developed. Samples of whole fish and different organs as well as edible portions of the fish should be analyzed. A comparison should be made of older, larger fish and younger, smaller fish relative to toxic metal levels. The same species of fish should be examined at different locations across the state. More fish samples from each site would allow for better data correlation. It is also important to note that this work should be done under critical, low flow conditions of the stream.
3. Future studies of the relationships of toxic metals in water and sediment should include a measure of the organic content

of the water and sediments. The effect of the organics on the retention of toxic metals in the sediment should be investigated.

4. Studies of the toxic metal levels in samples of natural fish populations should include a measure of the biomass of the fish population at the sample site.

5. More consecutive years of monitoring data should be obtained so reliable trends can be evaluated at each station. This would then allow for the evaluation of sources (point and non-point) of toxic metals in the fish tissues.

6. Additional study should be done to evaluate the relationships of flow patterns to the sediment deposition and the levels of toxic metals observed in natural fish populations.

7. Due to the elevated levels of metals in the sediment with respect to the levels in the fish and water samples, studies should be conducted to investigate the release potential of these metals from the sediments.

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APPENDIX A

SAMPLE STATION DESCRIPTIONS

TABLE A-1. SAMPLE STATION DESCRIPTIONS, NAMES AND LEGAL LOCATIONS.

STATION NUMBER	USGS NUMBER	NAME	LEGAL DESCRIPTION	LOCATION
1	1505	Salt Fork of the Arkansas River near Jet	NE/4 NE/4 Sec11 T26N R9W	0.6 mile downstream from Great Salt Plains Dam, 4 miles upstream from Wagon Creek, 6 miles northeast of Jet, and at Mile 102.7
2	1525	Arkansas River at Ralston	NW/4 Sec 1 T23N R5E	2 miles downstream from Salt Creek, 2 miles upstream from Grayhorse Creek, and at Mile 594.0
3	1579.5	Cimarron River near Buffalo	NW/4 SW/4 Sec 7 T28N R20W	6 miles upstream from Keno Creek, 7 miles upstream from Bridge on US Highway 64, 14 miles northeast of Buffalo, and at Mile 296.0
4	1610	Cimarron River at Perkins	SW/4 SW/4 Sec 7 T17N R3E	1 mile south of Perkins, 1.5 miles upstream from Dugout Creek, 4.0 miles downstream from Wildhorse Creek, and at Mile 87.3
5	1644	Arkansas River near Sand Springs	NW/4 SW/4 Sec 14 T19N R11E	5.1 miles downstream from Keystone Dam, and 10 miles upstream from Gaging Station at Tulsa
6	1655.7	Arkansas River at Haskell	NE/4 Sec 31 T16N R16E	2 miles east of Haskell, 23.5 miles upstream from Verdigris River, and at Mile 483.7

TABLE A-1. (cont.)

STATION NUMBER	USGS NUMBER	NAME	LEGAL DESCRIPTION	LOCATION
7	1780.5	Bird Creek near Catoosa Highway 167	NW/4 SE/4 NW/4 Sec 9 T20N R14E	At bridge on US Highway 75, approximately 5.5 miles northwest of Catoosa
8	1965	Illinois River near Tahlequah Highway 62	SE/4 Sec 26 T17N R22E	0.2 mile downstream from US Highway 62, 2.2 miles northeast of Tahlequah, 6.5 miles upstream from Baron Fork, and at Mile 55.8
9	1980	Illinois River near Gore	NE/4 SW/4 Sec 27 T13N R21E	4.3 miles downstream from Tenkiller Ferry Dam, 4.5 miles northeast of Gore, and at Mile 8.5
10	2424	Deep Fork River near Wellston		
11	2395	North Canadian at El Reno	Sec 32 T13N R7W	2.0 miles north of Courthouse in El Reno, 2.2 miles downstream from Target Creek, and at Mile 307.4
12	2415.5	North Canadian River at Harrah	SW/4 NW/4 Sec 22 T12N R1E	2.2 miles northwest of Harrah and at Mile 230
13	2285	South Canadian River at Bridgeport	SE/4 SW/4 Sec 28 T13N R11W	1.0 mile north of Bridgeport, 2.8 miles upstream from Lumpmouth Creek, and at Mile 267.1

TABLE A-1. (cont.)

STATION NUMBER	USGS NUMBER	NAME	LEGAL DESCRIPTION	LOCATION
14	2315	Canadian River at Calvin	NE/4 SW/4 Sec 22 T6N R10E	0.5 mile northeast of Calvin, 2.4 miles upstream from Shawnee Creek, 8.5 miles downstream from Little River, and at Mile 93.9
15	2494.4	Poteau River near Ft. Smith, Arkansas	SE/4 SW/4 Sec 9 T10N R27E	1.2 miles west of State Line, and 2.0 miles southwest of Ft. Smith
16	3050	North Fork of the Red River near Headrick	NW/4 NE/4 Sec 21 T2N R18W	2.5 miles east of Headrick, 12.9 miles upstream from Otter Creek, and at Mile 33.0
17	3155	Red River near Terral		1.2 miles south of Terral, 3.6 miles downstream from Little Wichita River, and at Mile 872
18	3310	Washita River near Durwood	NW/4 SW/4 Sec 3 T4S R3E	1.3 miles downstream from Caddo Creek, 4 miles north of Durwood, and at Mile 63.4
19	3340	Muddy Boggy Creek near Farris	NE/4 NW/4 Sec 26 T3S R13E	1.3 miles downstream from McGee Creek, 2.8 miles northwest of Farris, and at Mile 57.7
20	3357	Kiamichi River near Big Cedar	SW/4 SE/4 Sec 18 T2N R26E	0.2 mile upstream from Rattlesnake Creek, 1.1 miles upstream from Big Branch, 2.1 miles east of Big Cedar, and at Mile 157.6

TABLE A-1. (cont.)

STATION NUMBER	USGS NUMBER	NAME	LEGAL DESCRIPTION	LOCATION
21	3362	Kiamichi River near Antlers	SW/4 Sec 35 T3S R16E	2.0 miles northeast of Antlers, 7.7 miles downstream from Tenmile Creek, 5.4 miles upstream from Cedar Creek and at Mile 59.6
22	3368.2	Red River near DeKalb, Texas		4.8 miles upstream from North Mill Creek, 13 miles north of DeKalb, and at Mile 556.9
23	3385	Little River near Idabel	SE/4 SE/4 Sec 14 T7S R24E	5.0 miles northeast of Idabel, and at Mile 103.4

TABLE A-2. DESCRIPTION OF THE AQUATIC HABITATS AT THE SAMPLE STATIONS.

STATION NUMBER	DESCRIPTION OF AQUATIC HABITAT
1	The Salt Fork of the Arkansas River near Jet has a wide channel (approximately 200 feet) with moderately deep banks and having usually less than four feet of water. There are shallow, still backwater pools along the south bank. The water is clear with sluggish flow with flow occurring only when the water in Great Salt Plains Reservoir is higher than the dam. The environment at this site is highly modified by the Reservoir 0.6 miles upstream from this site due to this flow dependence, and the substrate has been modified by channelization during construction of the dam and the highway bridge. The substrate was very stable, consisting of soft shale bedrock and small to medium size rocks with pockets of hard clay. The area is impacted by heavy utilization by fishermen and minnow dealers, resulting in accumulations of solid wastes in the habitat area.
2	The Arkansas River at Ralston is wide (approximately one mile), bordered by high, steep banks on both sides, with a large sandbar in the bend of the river. The water is clear, approximately six feet in depth, with a swift flowing current. The sampling area was in a shallow channel (approximately three feet deep) with several backwater pools of still water. The river at this site is a permanently flowing stream with turbulent water and eddies due to the Kaw Reservoir discharge. The substrate consisted of fine sand to coarse gravel which formed mixed sand/gravel bars with slight accumulations of plant debris. There was no rooted aquatic vegetation in the area due to an unstable substrate.

TABLE A-2. (cont.)

STATION NUMBER	DESCRIPTION OF AQUATIC HABITAT
3	The Cimarron River near Buffalo is a typical, western Oklahoma river with low banks and wide, sandy floodplains. The channel is narrow (approximately 10 feet wide) and meanders across an usually dry, wide, sandy bed. The water is clear with gentle flow and very shallow riffles which are less than six inches in depth. Sparsely located narrow pools, usually less than two feet in depth, were formed along undercut banks and at the end of sandbars where the channel widens. The habitat was very uniform in its features. The substrate was very unstable, consisting of fine to coarse sand which shifts continually. This unstable substrate prevents the establishment of rooted aquatic vegetation.
4	The Cimarron River at Perkins is a wide, medium size river with rapid flow and very turbid water. There were large sandbars and moderatly high banks of red soil surrounded by a wide, rich flood plain. The main channel was undercutting the north bank and forming a long sandbar which sloped into a deep hole. The water at the sandbar was two to three feet deep and then formed a long, deep channel. There were several large, shallow backwater pools. Several deep holes within the channel were filled with a very soft liquid-like mud. The area is used extensively for recreation. The substrate in the main channel was comprised of very fine, hard packed sand. The substrate out of the main channel was very soft and was covered with a layer of thin mud.

TABLE A-2. (cont.)

STATION NUMBER	DESCRIPTION OF AQUATIC HABITAT
5	The Arkansas River near Sand Springs is a deep, wide, swift flowing stream with clear water and bordered by high banks and broad floodplains. The area is highly modified due to sand removal operations on the east bank and bridge due to sand removal operations on the east bank and bridge construction on the west bank. Flow in the river modified by discharges of water from Keystone Reservoir. The area consisted of shallow to deep pools filled with dense growths of willow and cottonwood trees.
6	The Arkansas River at Haskell is a wide (0.5 mile) swift flowing river with low banks, wide sandbars, and a broad, rich flood plain. The water is clear with turbulent flow, eddies, and waves. There was a daily fluctuating water level as water is discharged from Keystone Reservoir. This was evidenced by established willow trees along the west bank which were inundated at the time. The habitat has been highly modified on the east bank by a sand removal operation. The west bank was channelized during the construction of a highway bridge. The sampling areas were in the main channel in water four to five feet deep, in the backwater at the mouth of a small stream, and in backwater pools formed during the highway bridge construction. The unstable substrate consisted of fine to coarse sand.
7	Bird Creek near Catoosa is a fast flowing, narrow stream with many rapids and waterfalls. At this site a deep cut was made during the construction of the highway, leaving the bedrock exposed on the surface. The water is shallow and flows over and around many big rocks in the streambed. At the end of the rapids a long, narrow channel is found through which the water flows very fast and ends at a small waterfall. The entire area is very rocky with most of the substrate of the stream consisting of bedrock made up of layers of limestone. In the long channel there are many large rocks. There is some mud and trash at the foot of the rapids. The water was very muddy and had an odor of sewage at both sampling periods. High steep banks surround both sides of the stream. The area had a lot of solid waste, both in the stream and around it. The stream is used by fishermen and others for recreation.

TABLE A-2. (cont.)

STATION NUMBER	DESCRIPTION OF AQUATIC HABITAT
8	The Illinois River southeast of Tahlequah consists of the junction of the Illinois and Baron Fork. At this point the river is a narrow (150 feet), fast flowing stream with clear water. A long, medium-deep raceway (2-3 feet) gives way to a shallow, fast flowing riffle and then drops off into a deep pool (6 feet). The water flows over a substrate of medium to small sized chert and flint gravel and a few larger rocks. The river shore consisted of large gravel bars on both sides with well established willow growths lining the banks. Heavy growths of filamentous green algae were found throughout the area. Willow growths furnish shading areas along both shores throughout the day next to the banks. This area has very heavy human usage as a camping and outing area. There is a solid waste disposal problem at this site due to this usage. This area in the past has been used for gravel operations, but it is not now.
9	The Illinois River at Gore is a clear, cool river bordered by woods with low banks and a deep, wide channel with sluggish flow. The channel of the river at this site becomes deeper and the flow more sluggish due to the influence of the Robert S. Kerr Reservoir on the Arkansas River. The area consists of a series of small, segregated ponds through which the river has very dense vegetative growths of Ceratophyllum (coontail) and Potamogeton (pond weed) usually 6-10 feet wide along the margins of the river. Habitats consisted of small. heavily vegetated pools, deep, clear pools, shallow shorelines, and a deep, clear channel. The substrate was stable and consisted of gravel to cobble-sized rocks. In some areas, black mud was mixed with heavy accumulations of detritus. There was dense, woody plant depris in the main channel.
10	The Deep Fork River near Wellston flows through the southwest edge of Wellston. A small stream enters the river at this site from the town. This stream was pea green in color. The river at this point has a high, steep sandbar on the west and a steep bank on the east. The water flows gently over shallow riffles formed by sandbars into shallow pools (less than one foot deep). The water spreads out over the sandbars and slows down except along the edge of the banks where a raceway

TABLE A-2. (cont.)

STATION NUMBER	DESCRIPTION OF AQUATIC HABITAT
	picks up the water and moves faster. The banks are lined by overhanding trees and brush which help to shade the water most of the day. The water was clear except in one small backwater pool where there was a thick growth of green algae. The area surrounding the river consisted of rich floodplains made up of a sandy soil. The area has some solid waste problems. The substrate consisted of coarse to fine sand which was very unstable. There was some woody material piled along the banks by flood waters.
11	The North Canadian River is an intermittent stream whose flow is regulated by releases from Canton Reservoir. The stream was very narrow with high banks and a wide floodplain. The site is composed of a long, moderately deep channel near the south bank. The stream becomes shallow on the north bank because of a small sandbar. This forms a backwater area along the north bank. There is a dense plant cover which hangs over the bank on the south side of the river. The substrate was composed of somewhat stable hard-packed fine grained sand in the main channel.
12	The North Canadian River at Harrah is narrow, shallow, and swift flowing with high banks and wide flood plains. The water is clear and usually bright green in color due to rich algal growths. The river generally has a uniform velocity due to the continuous discharge of treated effluent from the Oklahoma City Southside Wastewater Treatment Facility. The river channel is approximately 5 feet in depth with long, shallow riffles ending in sandbars and pools. There are several deep backwaters along the north bank of the stream and a large amount of wood material both in the water and suspended above the water. The substrate consisted of fine, hard packed sand in the main channel. In pools and backwaters out of the main channel, this compacted sand had a 6-12 inch cover of soft mud.

TABLE A-2 (cont.)

STATION NUMBER	DESCRIPTION OF AQUATIC HABITAT
13	The South Canadian River is a small river with intermittent flow. It has clear water which meanders across a wide, sandy river bed. The river forms long shallow riffles with shallow, deep pools at the edge of sandbars. A few pools with water two to three feet deep are located along the north bank near areas where woody debris has been deposited as a result of historical floods. There was a long, wide sandbar on the south side of the river. This site is used extensively by commercial minnow dealers. The substrate was composed of very fine unstable sand which was continuously shifting.
14	The Canadian River at Calvin has a very wide river bed with high, steep banks and a small, narrow channel which meanders across the sandy riverbed. The water is clear with gentle flow. The aquatic habitat is very uniform in character, consisting of a moderately deep channel with a few shallow pools along its margin. The channel has formed a long sandbar which drops off to form a deep pool along the north bank. The river is undercutting the north bank and filling the pool with dense, woody debris. The substrate consisted of fine to coarse sand with a few large rocks.
15	The Poteau River near Ft. Smith is a typical lowland river with a deep channel, turbid water, sluggish flow, and very steep, muddy banks lined with large hardwood trees and bordered by broad, rich floodplains. The habitat sampled consisted of a deep shoreline mixed with backwater of a small creek that was backed up by the river. Turbid waters have prevented the establishment of rooted aquatic vegetation. The substrate consisted of soft mud, small rocks and gravel.

TABLE A-2. (cont.)

STATION NUMBER	DESCRIPTION OF AQUATIC HABITAT
16	The North Fork of the Red River is a medium sized river with clear water and gentle flow in a wide shallow channel. The west bank is high and slopes to high granite ridges. The east bank is also high and consists of red soil bordered by wide, rich floodplains. The channel consisted of shallow riffles and backwater pools. Deep pools were sparsely spaced at the end of sandbars and along undercut banks. Several deep pools were formed behind logjams in the main channel. The substrate consisted of fine to very coarse sand mixed with large amounts of igneous gravel. The substrate was very unstable and continually shifting. A high sand and gravel bar was formed on the west bank with a dense growth of willow trees. This area was noted to be used extensively for recreational purposes.
17	The Red River near Terral is wide with high, steep, red clay banks on the north and a wide, shallow sandbar and floodplains on the south. The channel is wide (approximately 200 feet) with clear water, gentle flow, and a median depth of five feet. The sampling area consisted of shallow channels (3 feet) and very shallow backwater pools of standing water. One deep pool (approximately 6 feet) with a large driftwood accumulation was located beneath the highway bridge. The substrate consisted of fine to coarse sand with some large rocks and sandstone bedrock along the deep banks. The substrate was covered in the deep pools with a very fine, muddy sediment 6-10 inches deep. The shifting substrate prevents the establishment of rooted aquatic vegetation.
18	The Washita River near Durwood is a moderately sized lowland river with very steep banks and a narrow, deep channel. The water is very turbid with very swift flow. The river consisted of a long, deep raceway along the north bank which shallows out into a long, narrow sandbar on the south side. Water passing over the sandbar divides into several small riffles and pools. The substrate consisted of hard packed fine sand which was very stable. Heavy erosion of the north bank has resulted in very soft and unstable sediments along this area. There was some woody debris lodged along the north bank.

TABLE A-2. (cont.)

STATION NUMBER	DESCRIPTION OF AQUATIC HABITAT
19	Muddy Boggy Creek near Farris is a medium size lowland river with very steep, muddy banks and a narrow, shallow channel filled with woody debris. The water is very turbid and has a sluggish flow. The banks had heavy plant cover with the area showing heavy use by livestock. Small, shallow pools and a sandbar line the west bank of the river. The substrate consisted of hard, black clay and some sandy sediments mixed with small rocks. Within the main channel were large numbers of dead trees and stumps.
20	The Kiamichi River near Big Cedar is a typical upland stream with intermittent flow, clear water, and a gently sloping gradient. The stream is bordered by low banks and a dense pine and hardwood forest. The river channel consisted of clear, moderately deep pools connected by shallow, narrow riffles with dense growth of Potamogeton (pond weed) in the shallow areas. The substrate consisted of tilted sandstone bedrock covered by large boulders to cobble and was very unstable. Small amounts of detritus constitute sedimentation in pockets between the boulders. The area is usually shaded by an overhanging tree canopy.
21	The Kiamichi River near Antlers is formed by a wide, long, moderately deep pool with sluggish flow and slightly turbid water. The river is now influenced by backwaters from Hugo Reservoir. This area of the river was, in the past, one of riffles and swift flowing water, but is now inundated. The river is bordered by very steep banks, dense trees, and black gumbo soil. Along the edges of the river were dense growths of Potamogeton (pond weed) and willow trees in backwater pools and gravel bars. A small, clear, flowing spring entered the river at this station. The substrate consisted of large rocks and boulders mixed with muddy, yellow clay sediments.

TABLE A-2. (cont.)

STATION NUMBER	DESCRIPTION OF AQUATIC HABITAT
22	The Red River is wide (approximately one mile), and swift flowing at this station, with a moderately deep channel and slightly turbid water. The stream is bordered by moderately high banks, broad, flat sandbars, and a wide, rich floodplain. In the south bank sampling area, the river divides and forms an island with a deep water channel during periods of high flow. During periods of low flow, the channel forms isolated pools with minimal current. The substrate consisted of very fine sand which shifts continually and is very unstable. A few willow trees were growing in the river channel. The north bank of the river was high and was in the process of being undercut by a deep, swift flowing channel along this bank.
23	The Little River near Idabel is a lowland stream with steep banks of black gumbo soil surrounded by heavily wooded lowlands. The stream is moderately large with slightly turbid water and a permanent, gentle flow. The river has many diverse habitats ranging from deep sluggish backwaters to shallow, broad, gravel bottom riffles, deep, rocky channels, small, shallow backwater pools with dense vegetation, and areas of woody debris lodged against banks. The substrate consisted of soft mud mixed with leaves and wood debris in a gentle current along the south bank. The substrate along the north bank consisted of fine to large gravel with small amounts of woody material buried in the channel. The stream along the north bank had a swift current and the shoreline had profuse, dense growths of Potamogeton (pond weed). This station is below the mouth of Lukfata Creek and above an industrial point-source discharge.

APPENDIX B

GRAPHS OF TOXIC METAL CONTENT OF FISH SAMPLES

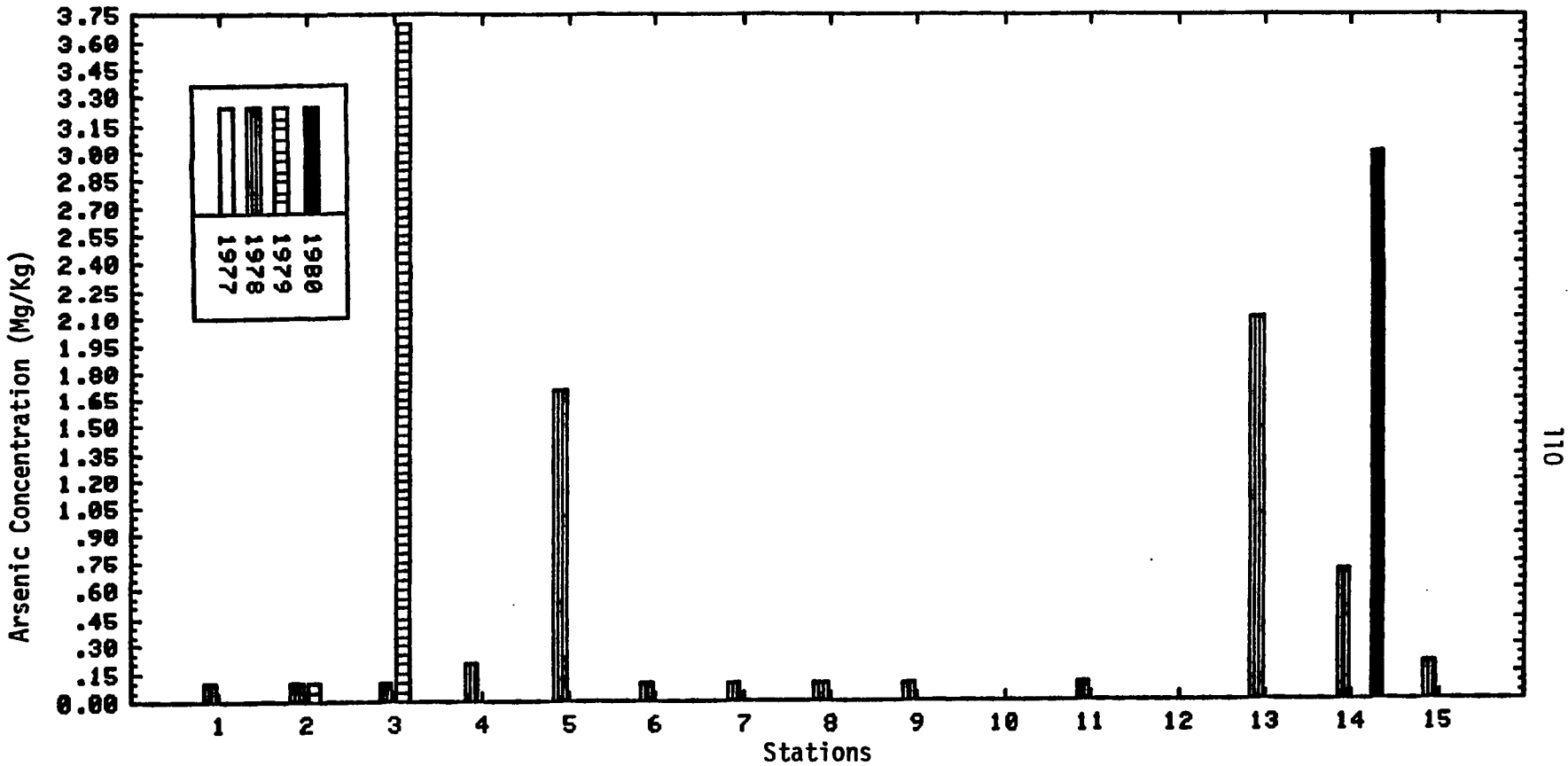

Figure B-1. Arsenic Content (Mg/Kg Body Weight) in the Fish Samples of the Herbivore Trophic Level from the Arkansas River Basin.

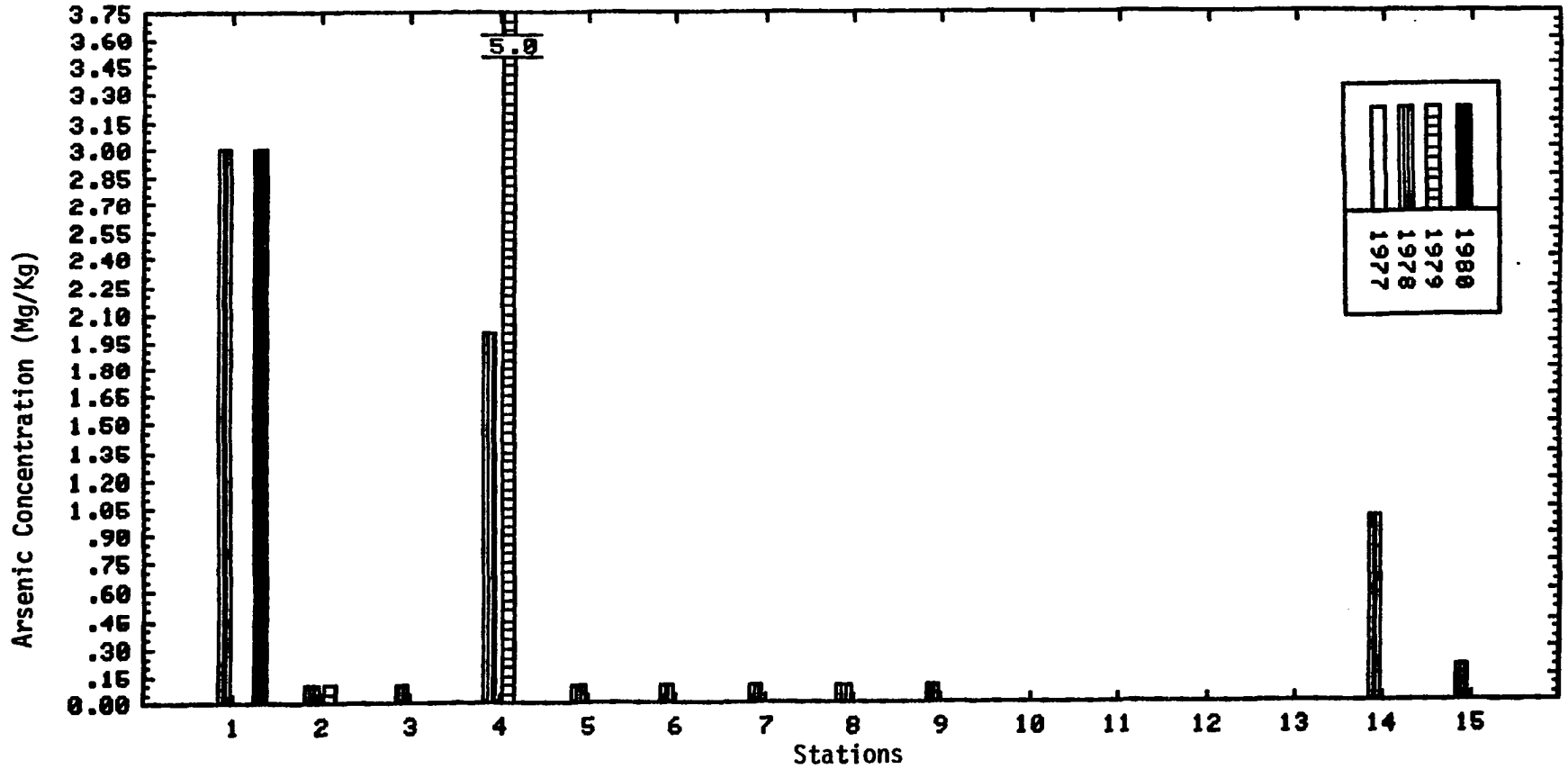

Figure B-2. Arsenic Content (Mg/Kg Body Weight) in the Fish Samples of the Carnivore Trophic Level from the Arkansas River Basin.

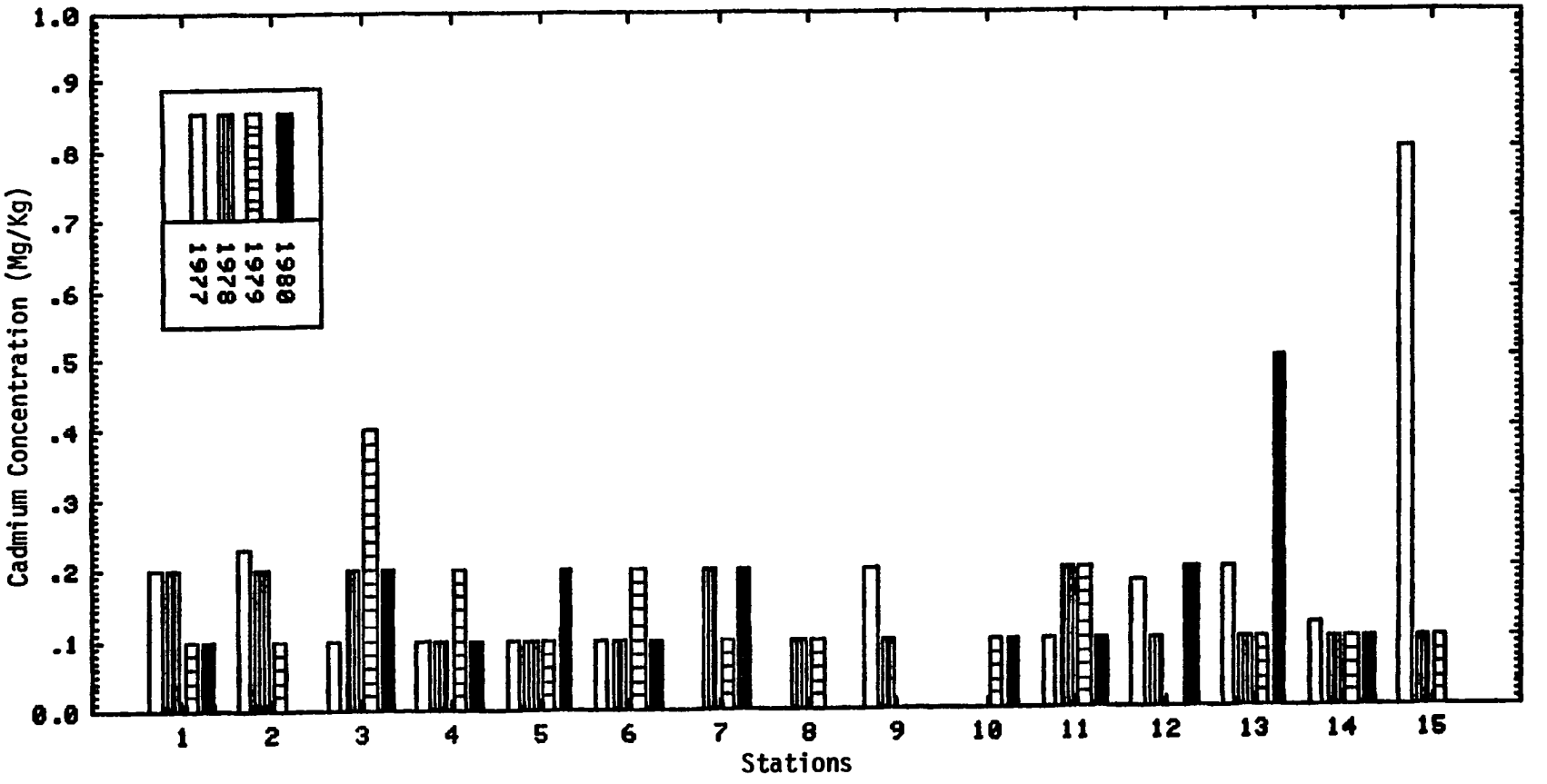

Figure B-3. Cadmium Content (Mg/Kg Body Weight) in the Fish Samples of the Herbivore Trophic Level from the Arkansas River Basin.

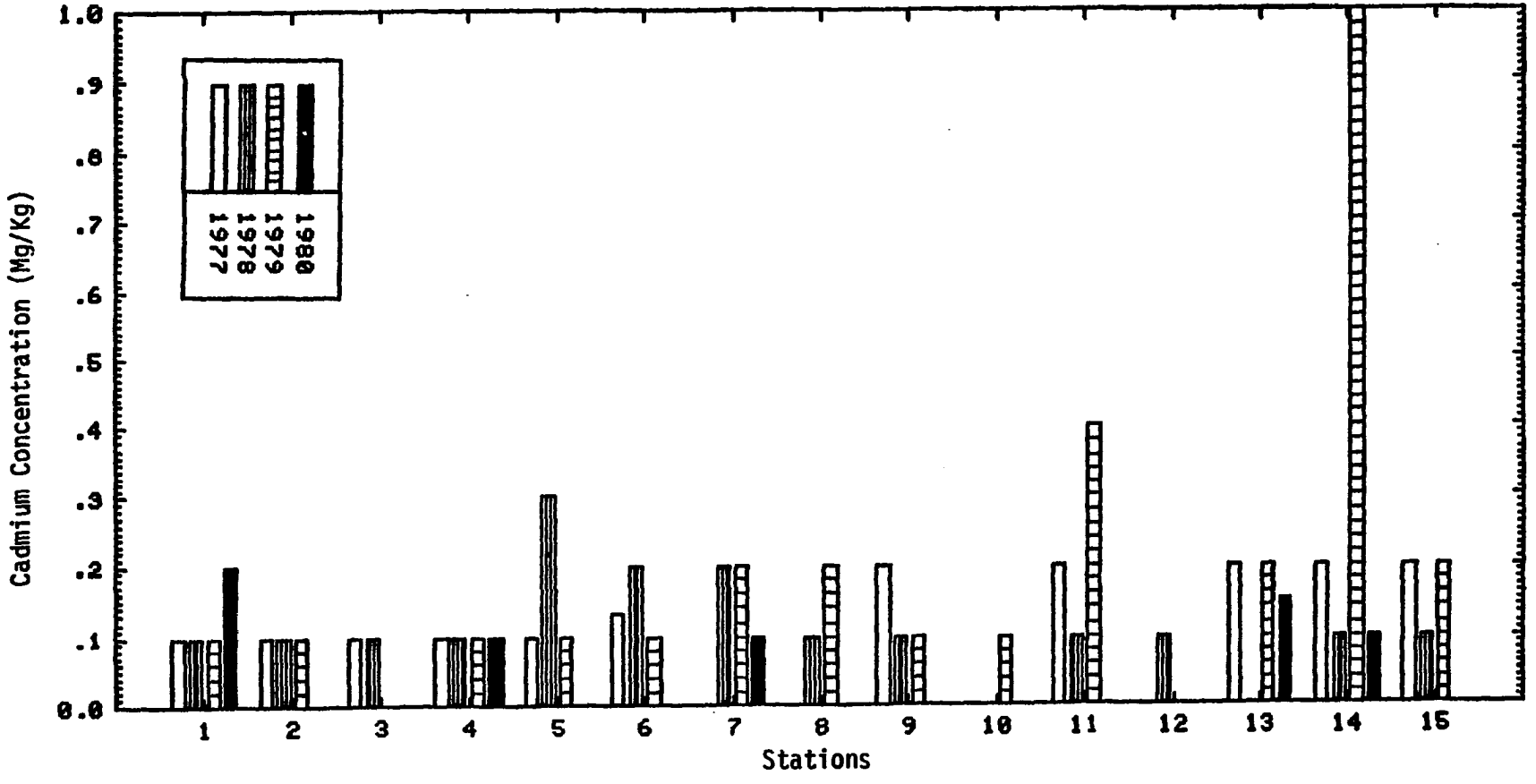

Figure B-4. Cadmium Content (Mg/Kg Body Weight)in the Fish Samples of the Carnivore Trophic Level from the Arkansas River Basin.

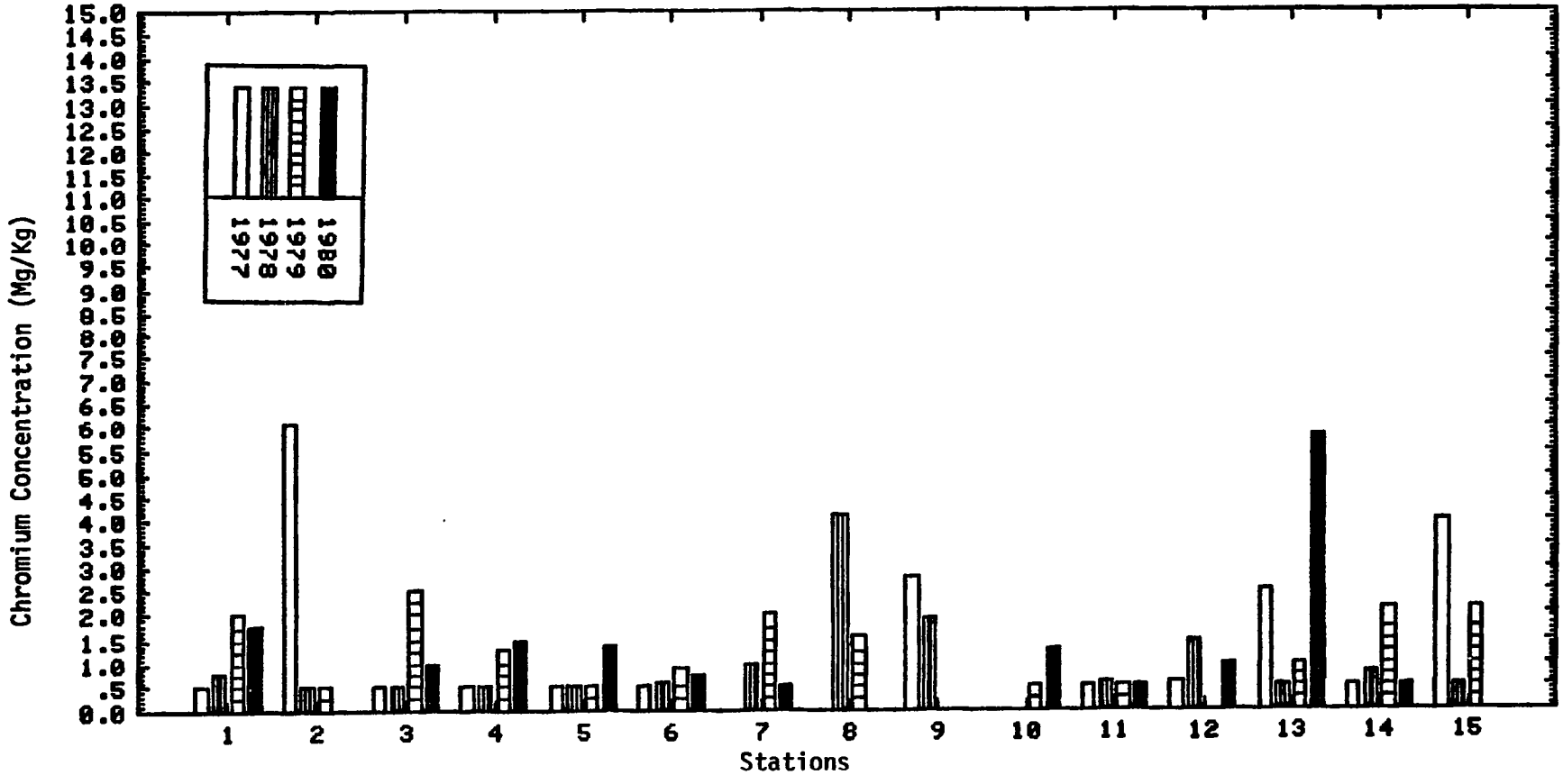

Figure B-5. Chromium Content (Mg/Kg Body Weight) in the Fish Samples of the Herbivore Trophic Level from the Arkansas River Basin.

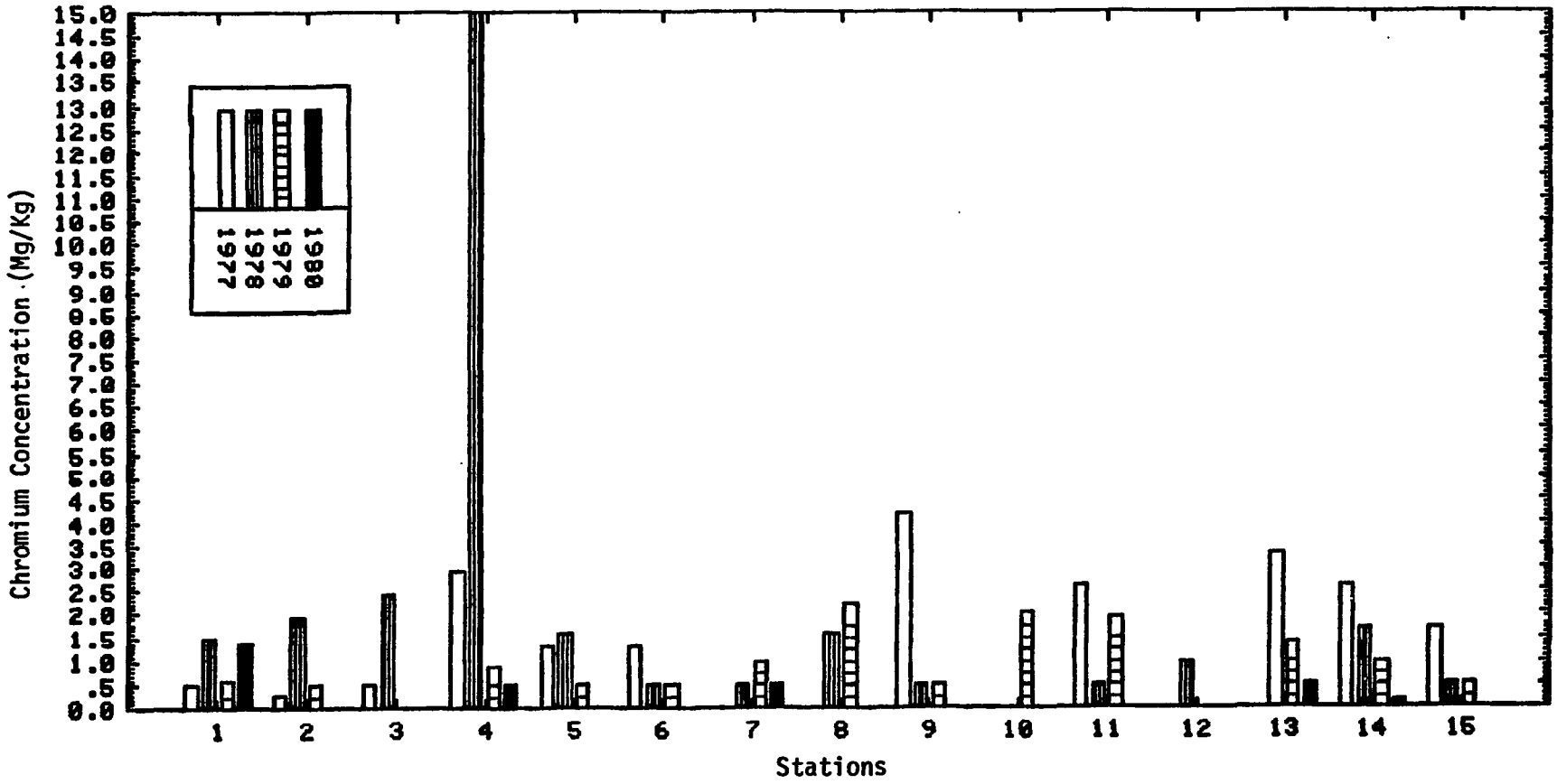

Figure B-6. Chromium Content (Mg/Kg Body Weight) in the Fish Samples of the Carnivore Trophic Level from the Arkansas River Basin.

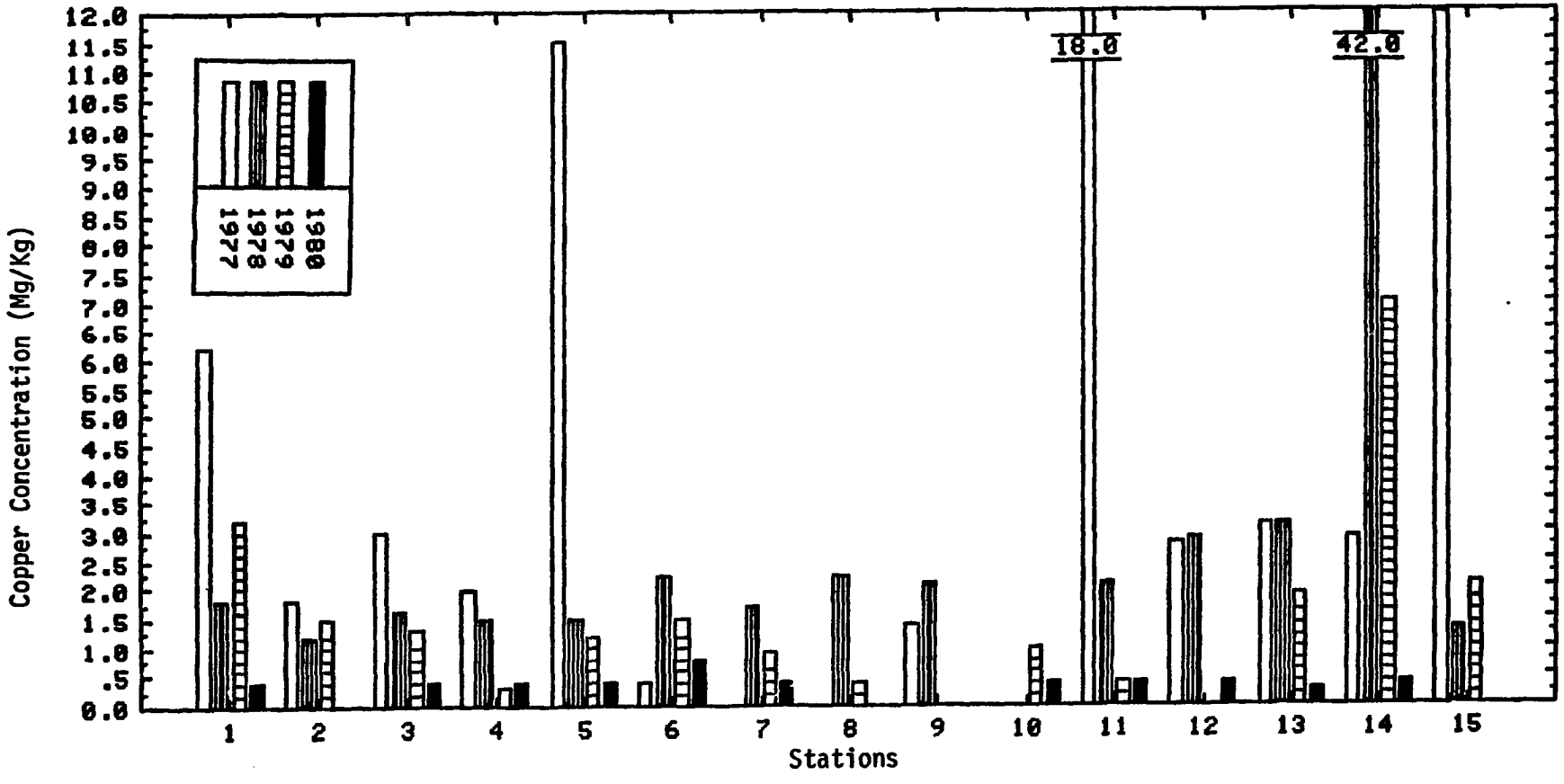

Figure B-7. Copper Content (Mg/Kg Body Weight) in the Fish Samples of the Herbivore Trophic Level from the Arkansas River Basin.

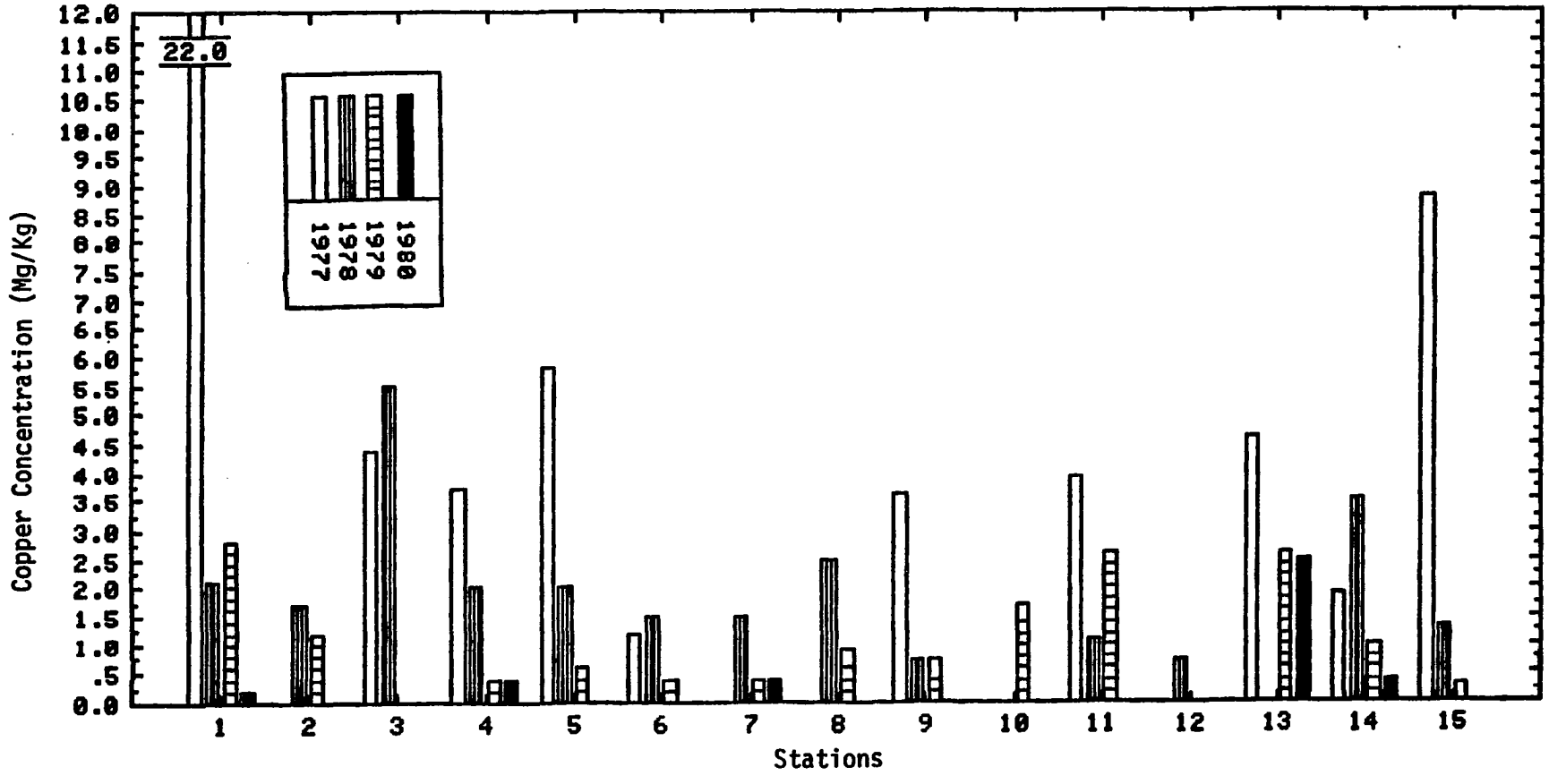

Figure B-8. Copper Content (Mg/Kg Body Weight) in the Fish Samples of the Carnivore Trophic Level from the Arkansas River Basin.

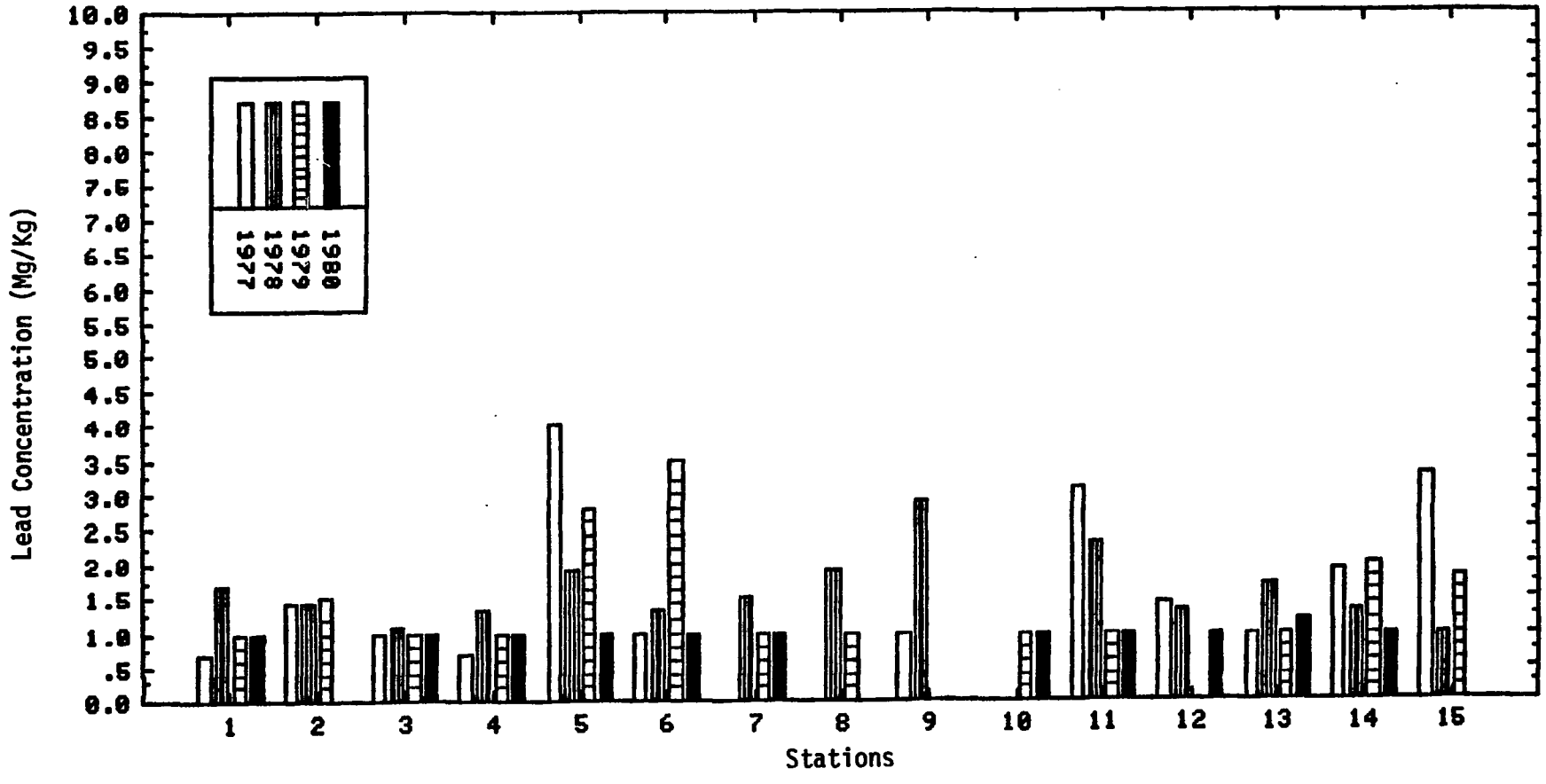

Figure B-9. Lead Content (Mg/Kg Body Weight) in the Fish Samples of the Herbivore Trophic Level from the Arkansas River Basin.

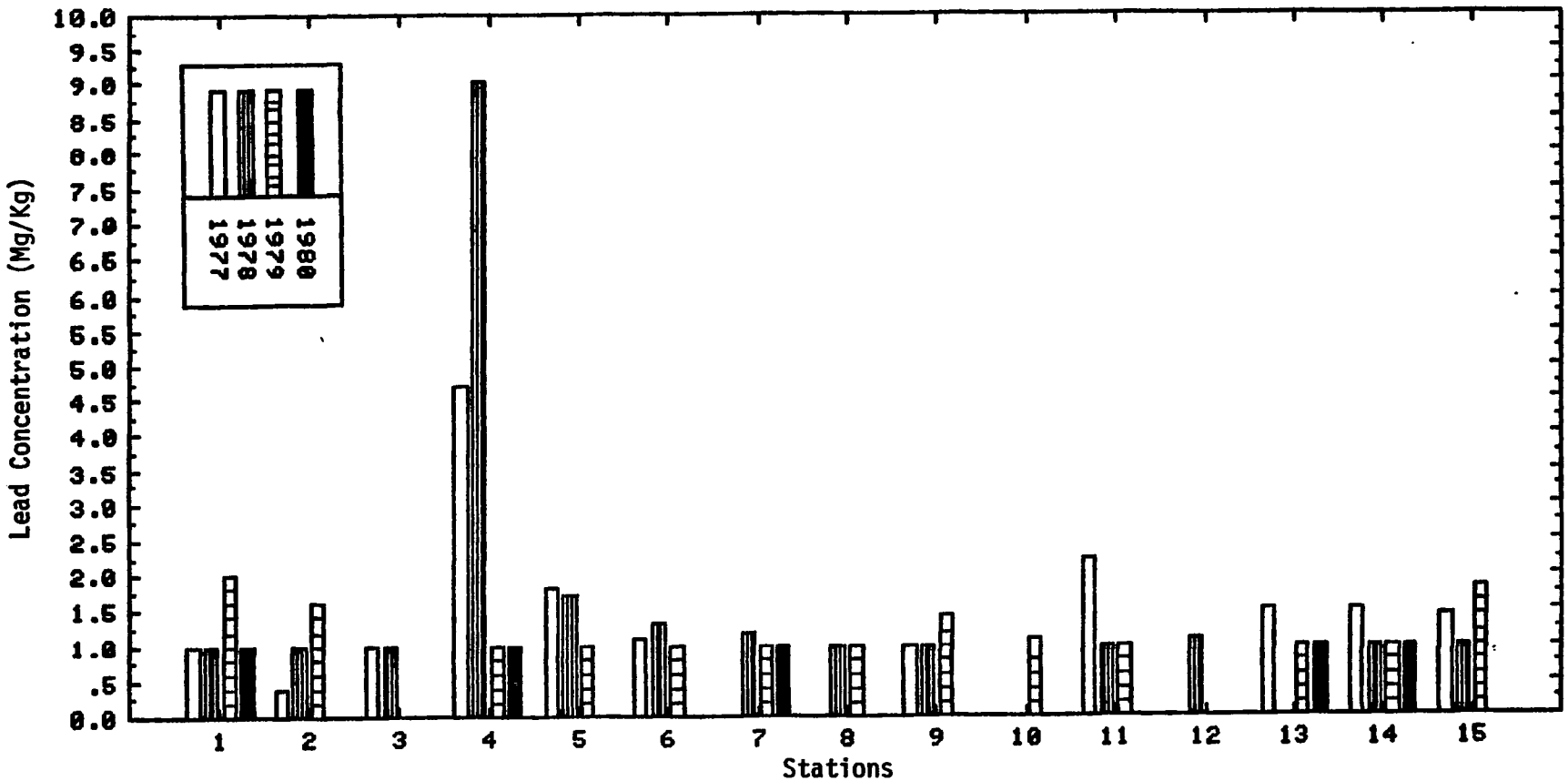

Figure B-10. Lead Content (Mg/Kg Body Weight) in the Fish Samples of the Carnivore Trophic Level from the Arkansas River Basin.

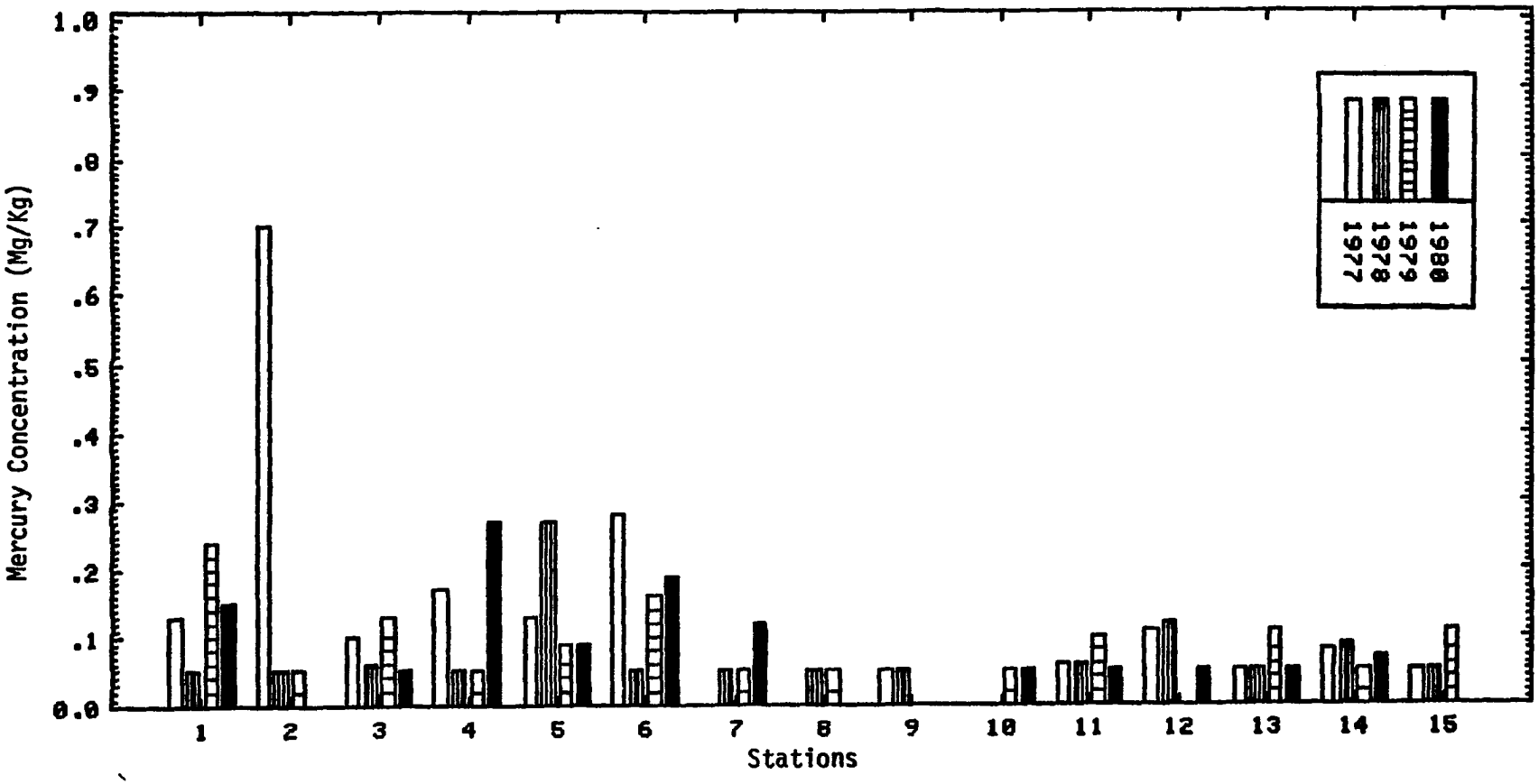

Figure B-11. Mercury Content (Mg/Kg Body Weight) in the Fish Samples of the Herbivore Trophic Level from the Arkansas River Basin.

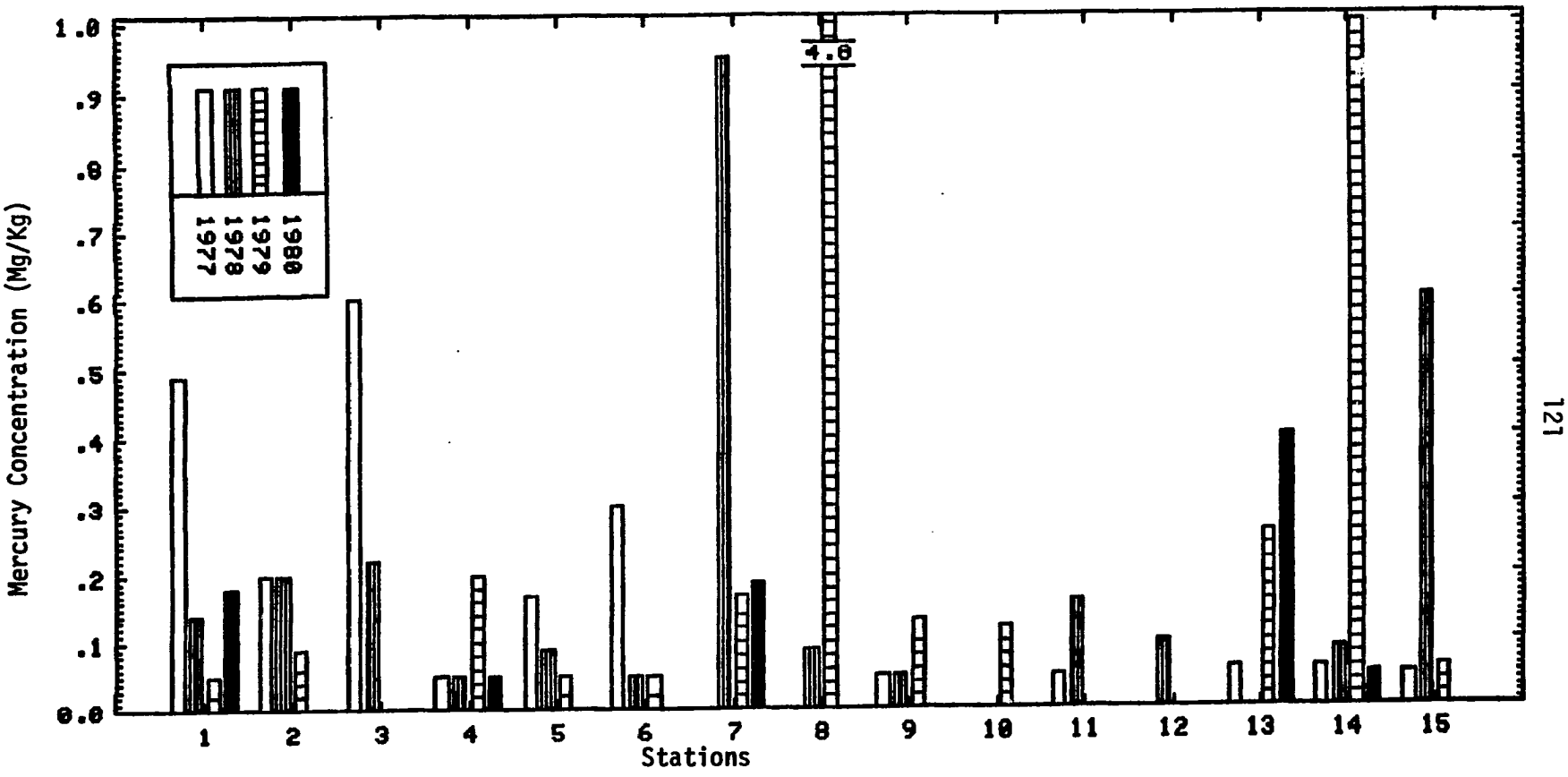

Figure B-12. Mercury Content (Mg/Kg Body Weight) in the Fish Samples of the Carnivore Trophic Level from the Arkansas River Basin.

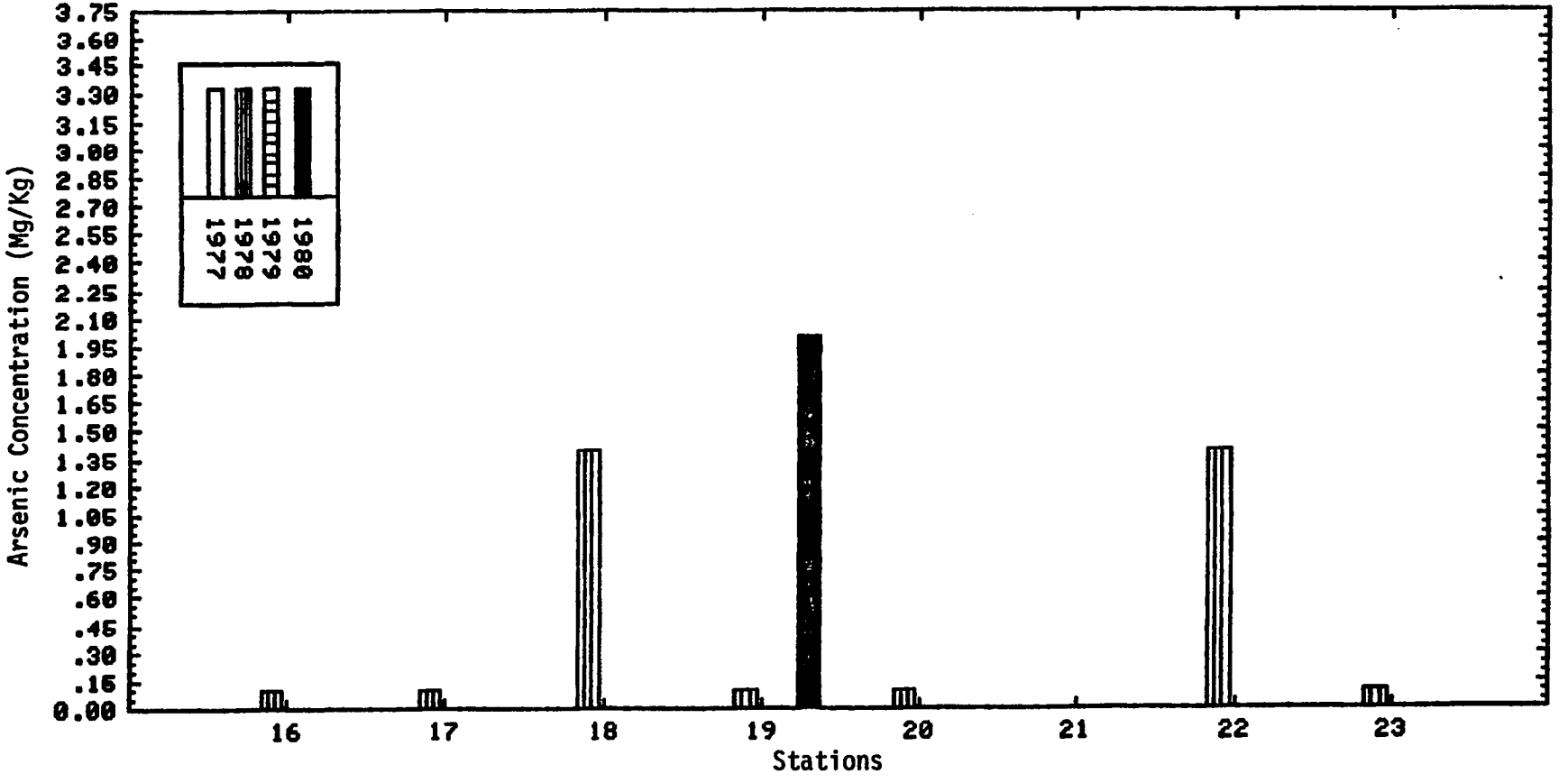

Figure B-13. Arsenic Content (Mg/Kg Body Weight)in the Fish Samples of the Herbivore Trophic Level from the Red River Basin.

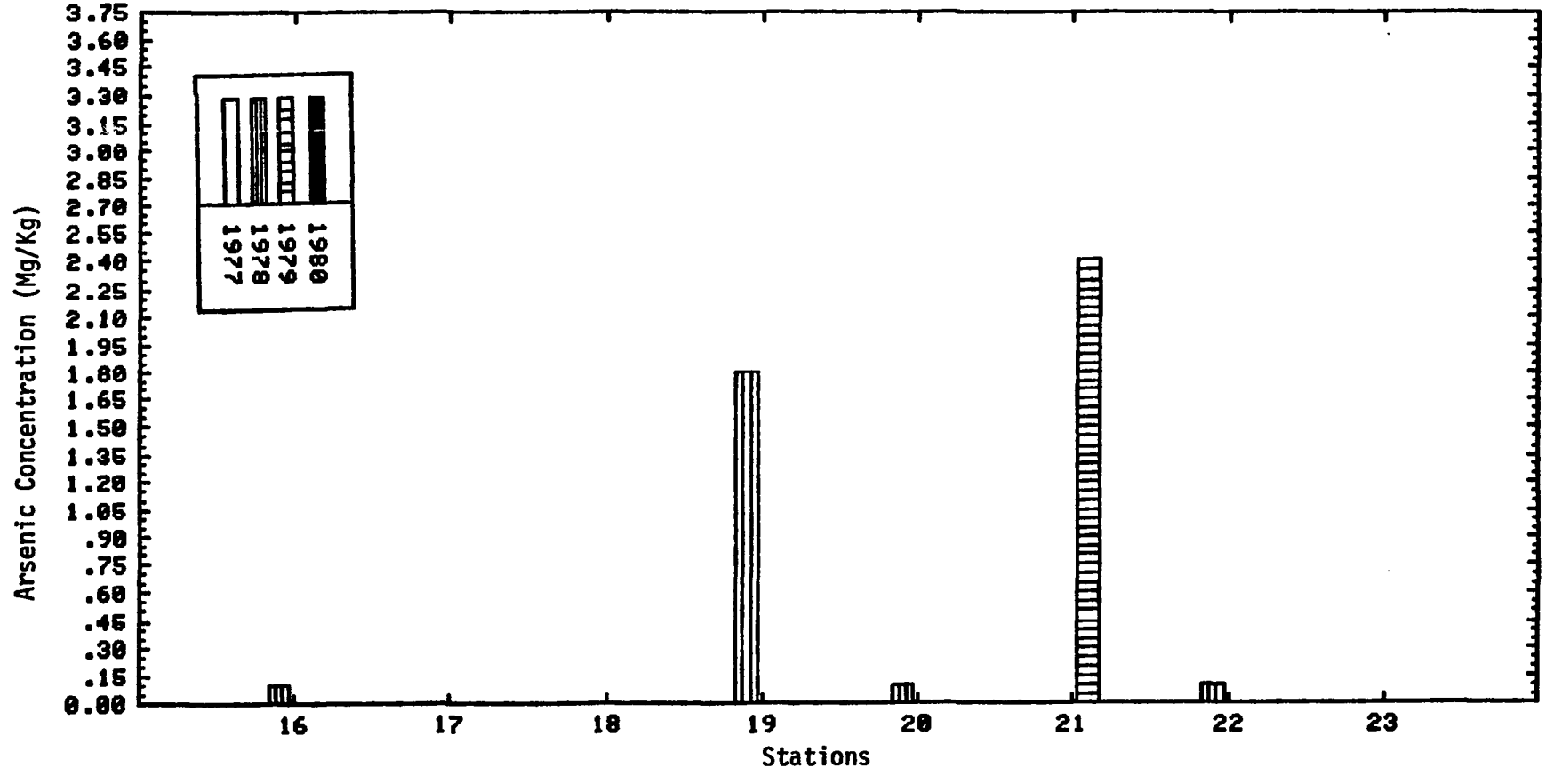

Figure B-14. Arsenic Content (Mg/Kg Body Weight) in the Fish Samples of the Carnivore Trophic Level from the Red River Basin.

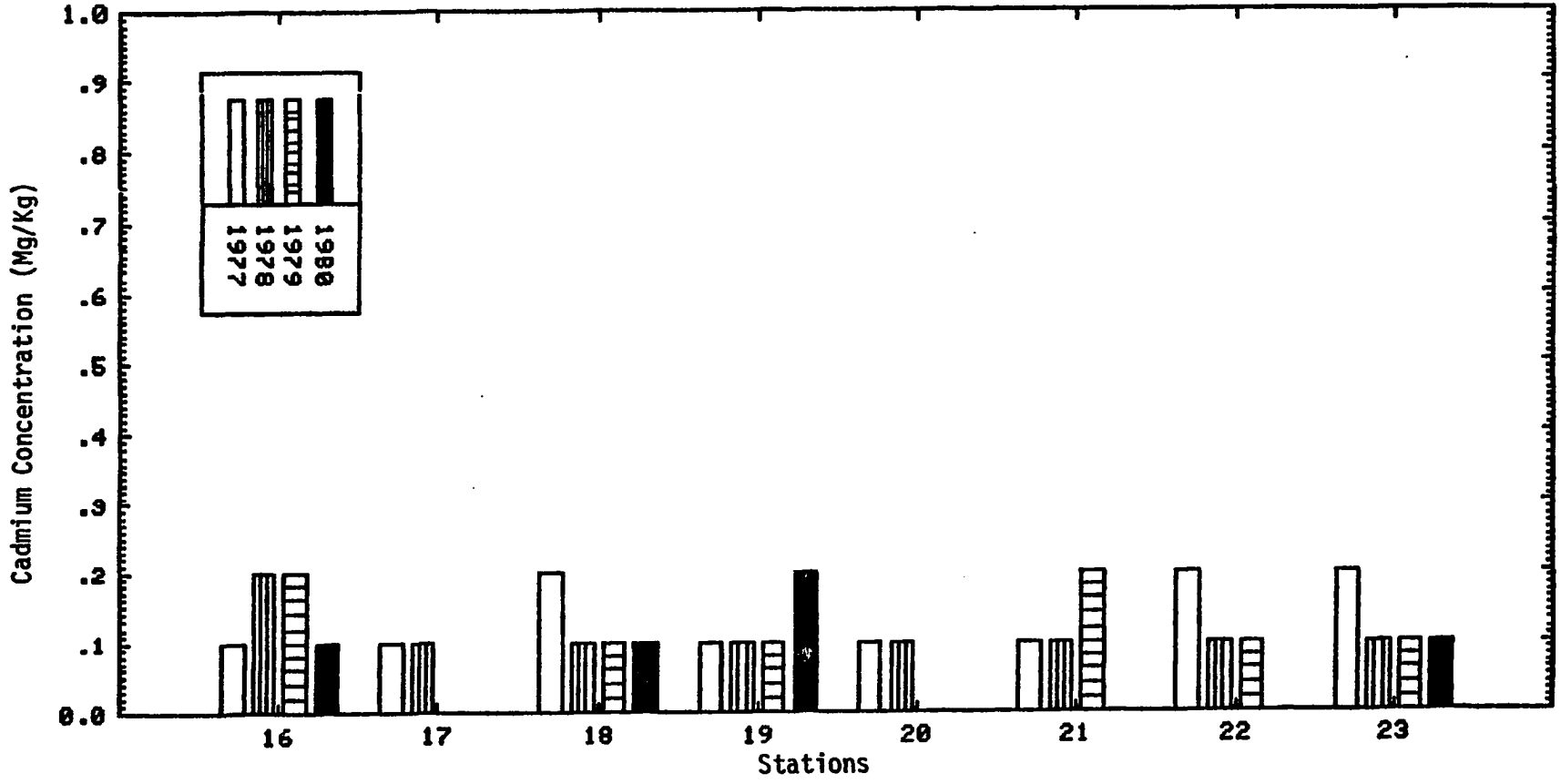

Figure B-15. Cadmium Content (Mg/Kg Body Weight) in the Fish Samples of the Herbivore Trophic Level from the Red River Basin.

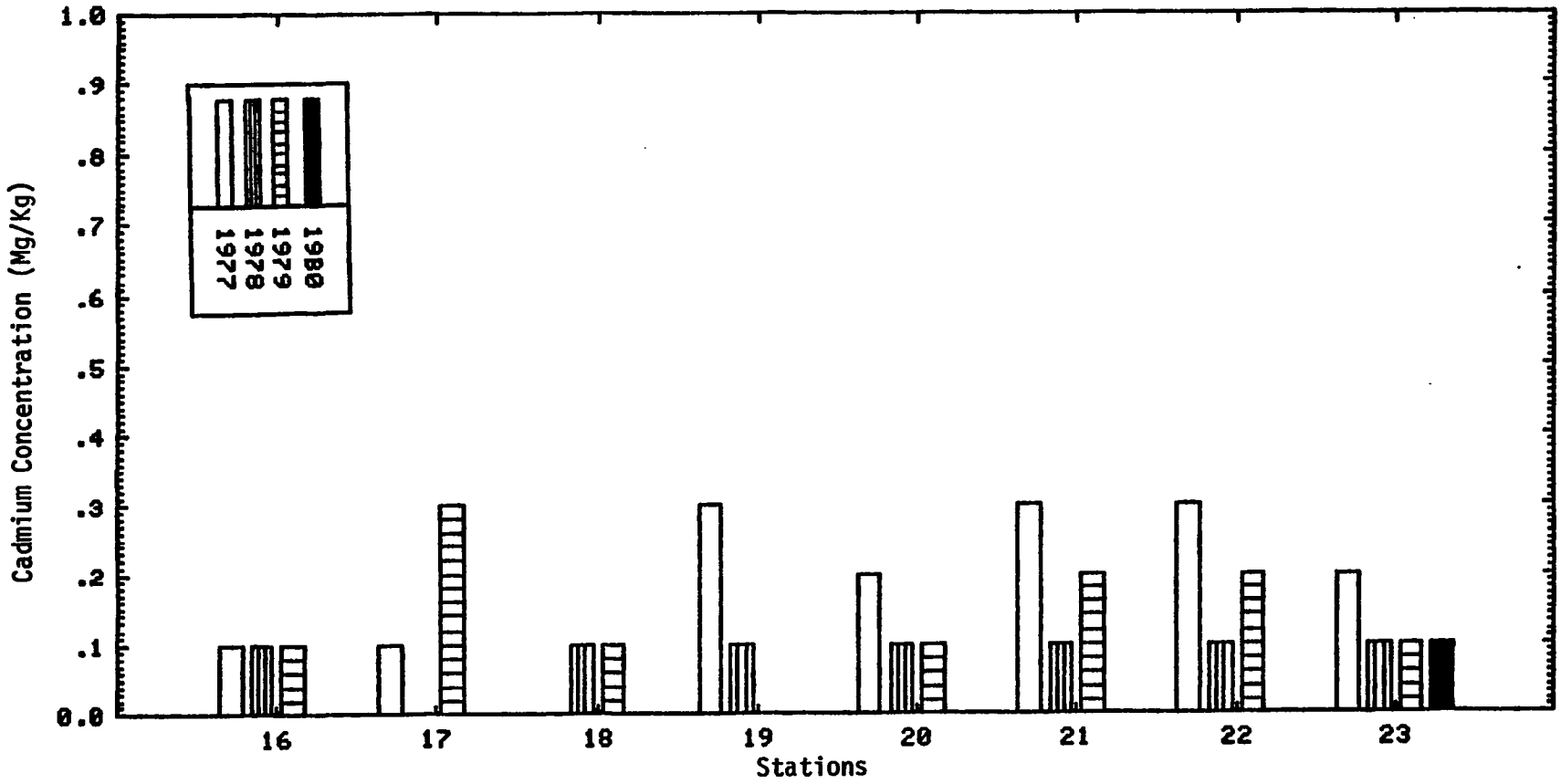

Figure B-16. Cadmium Content (Mg/Kg Body Weight) in the Fish Samples of the Carnivore Trophic Level from the Red River Basin.

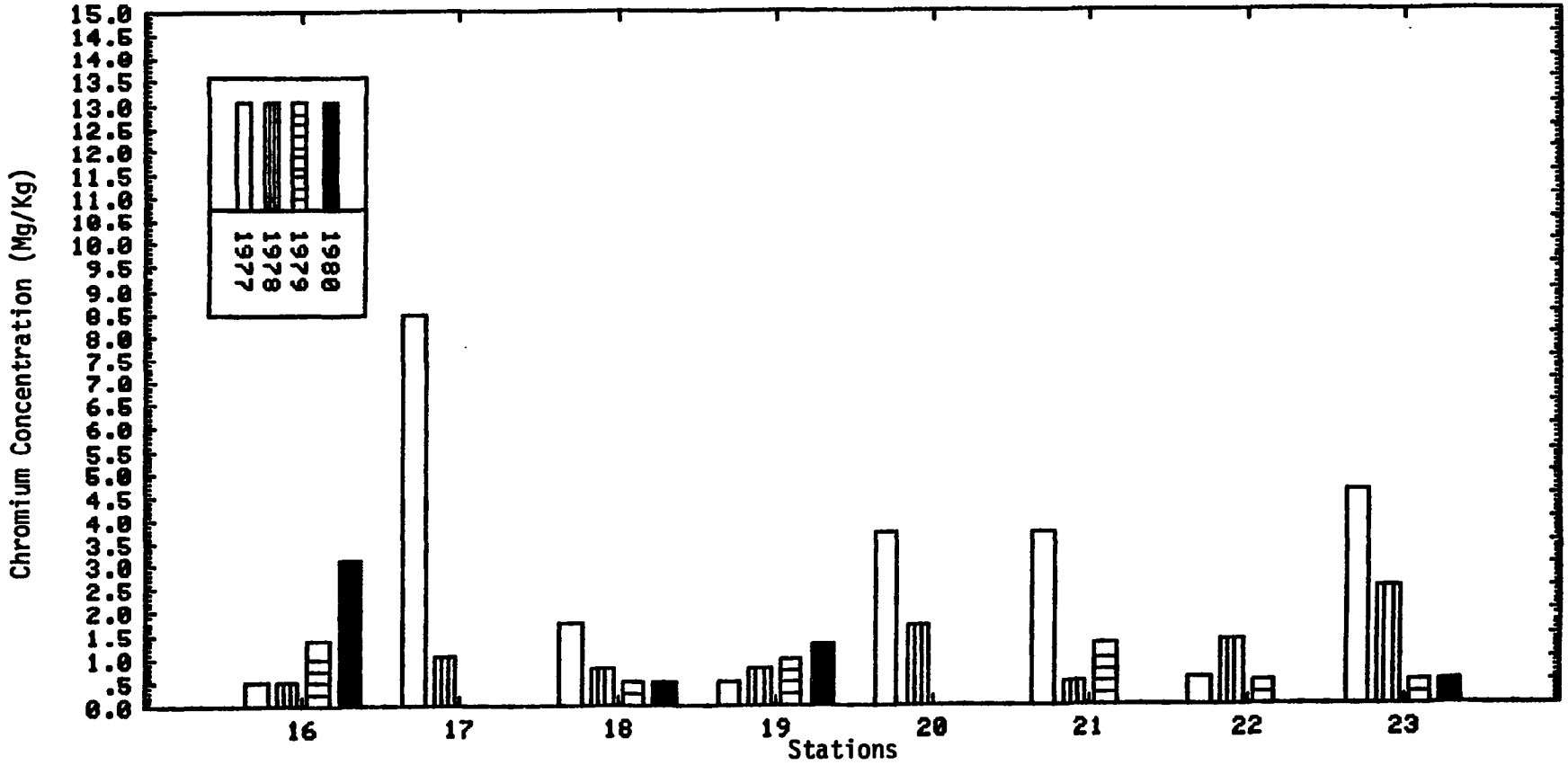

Figure B-17. Chromium Content (Mg/Kg Body Weight) in the Fish Samples of the Herbivore Trophic Level from the Red River Basin.

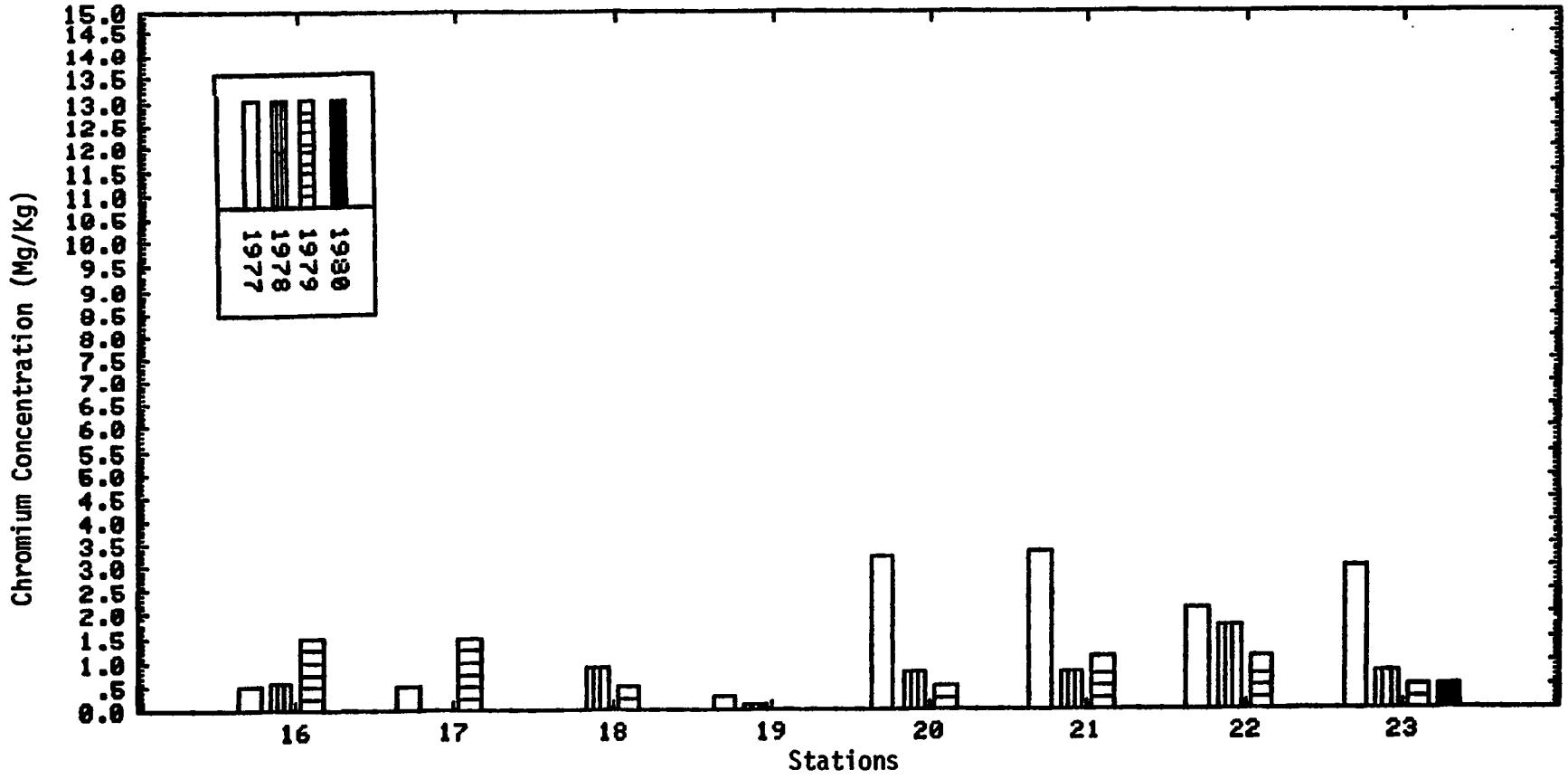

Figure B-18. Chromium Content (Mg/Kg Body Weight) in the Fish Samples of the Carnivore Trophic Level from the Red River Basin.

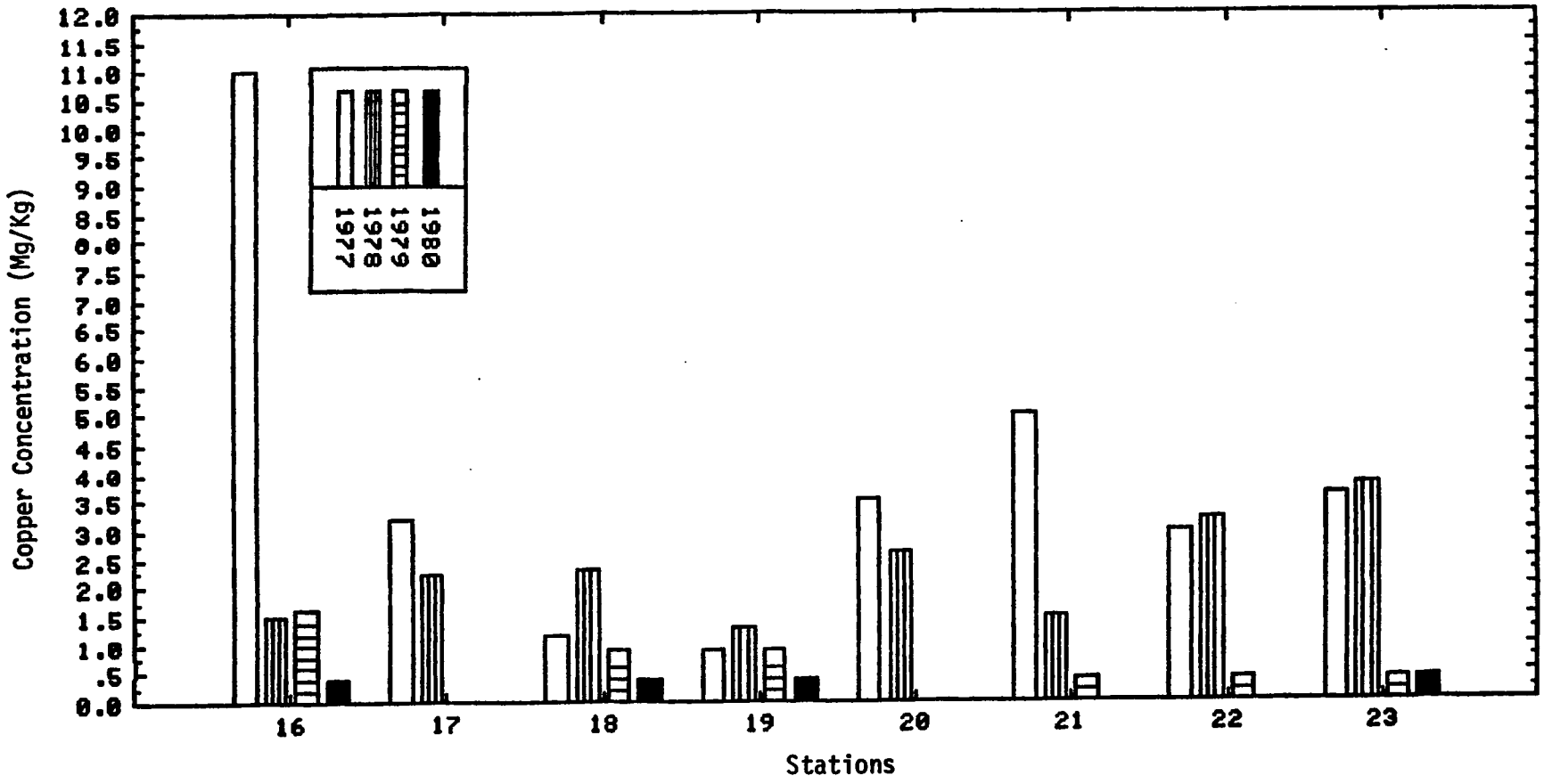

Figure B-19. Copper Content (Mg/Kg Body Weight) in the Fish Samples of the Herbivore Trophic Level from the Red River Basin.

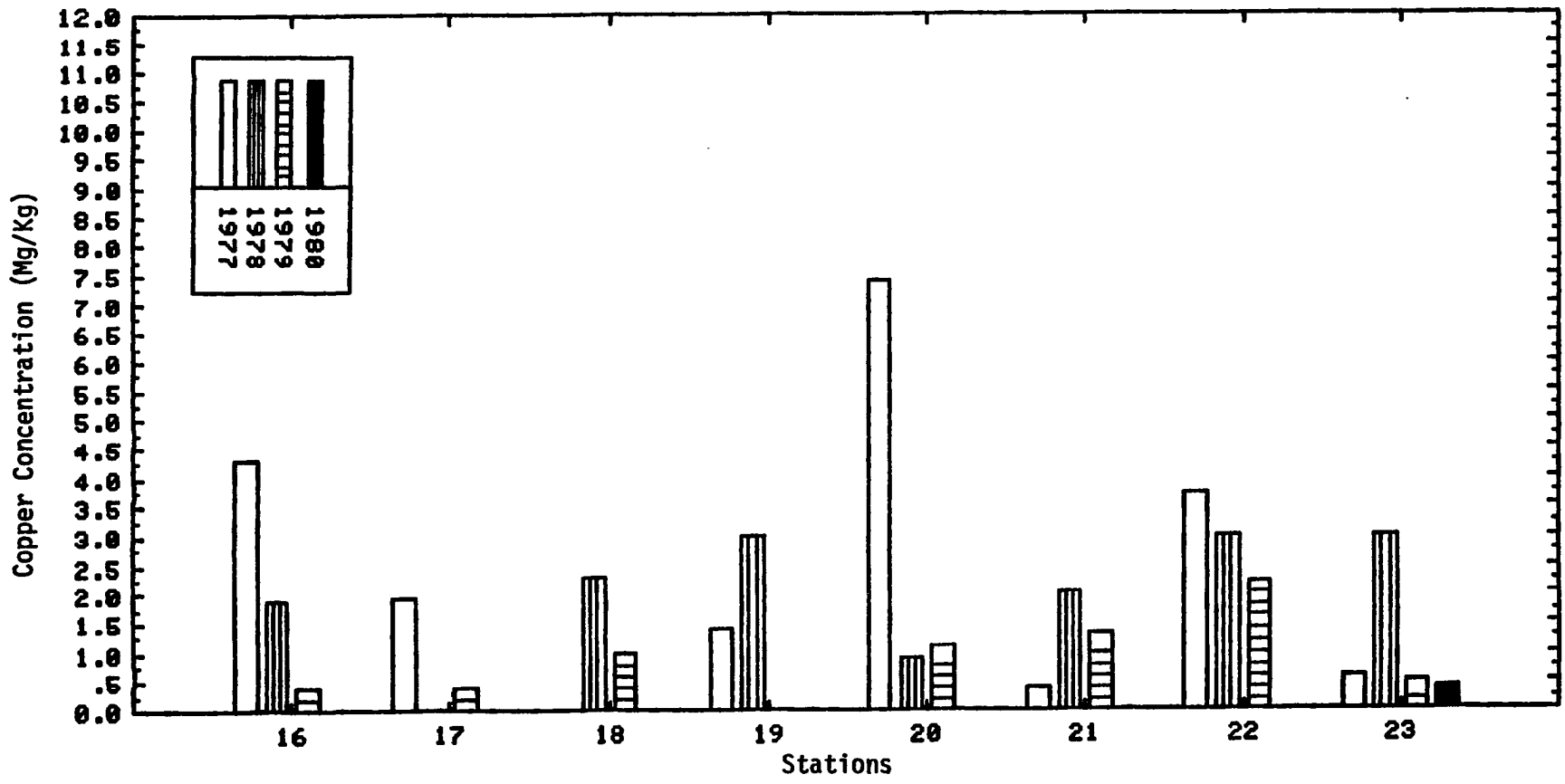

Figure B-20. Copper Content (Mg/Kg Body Weight) in the Fish Samples of the Carnivore Trophic Level from the Red River Basin.

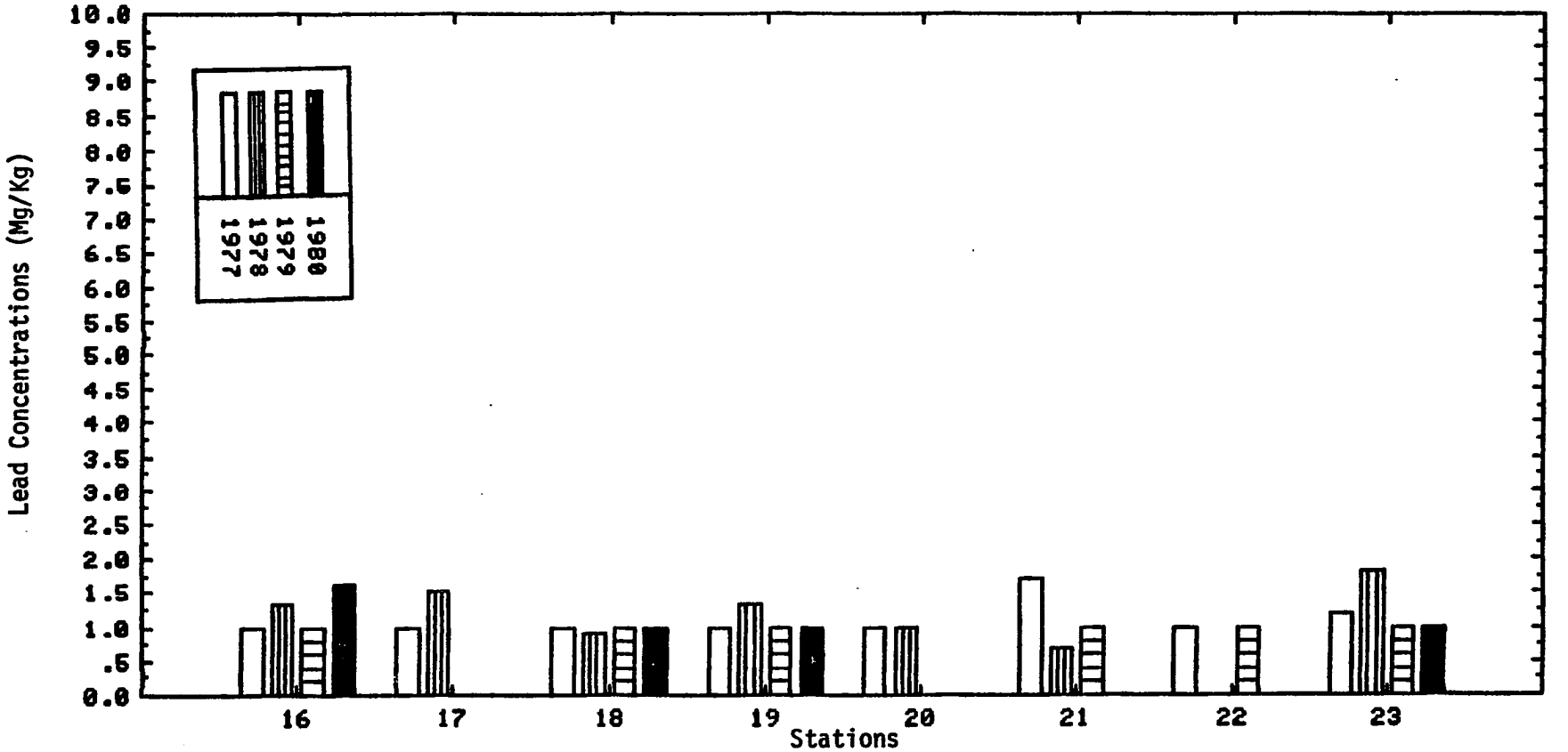

Figure B-21. Lead Content (Mg/Kg Body Weight) in the Fish Samples of the Herbivore Trophic Level from the Red River Basin.

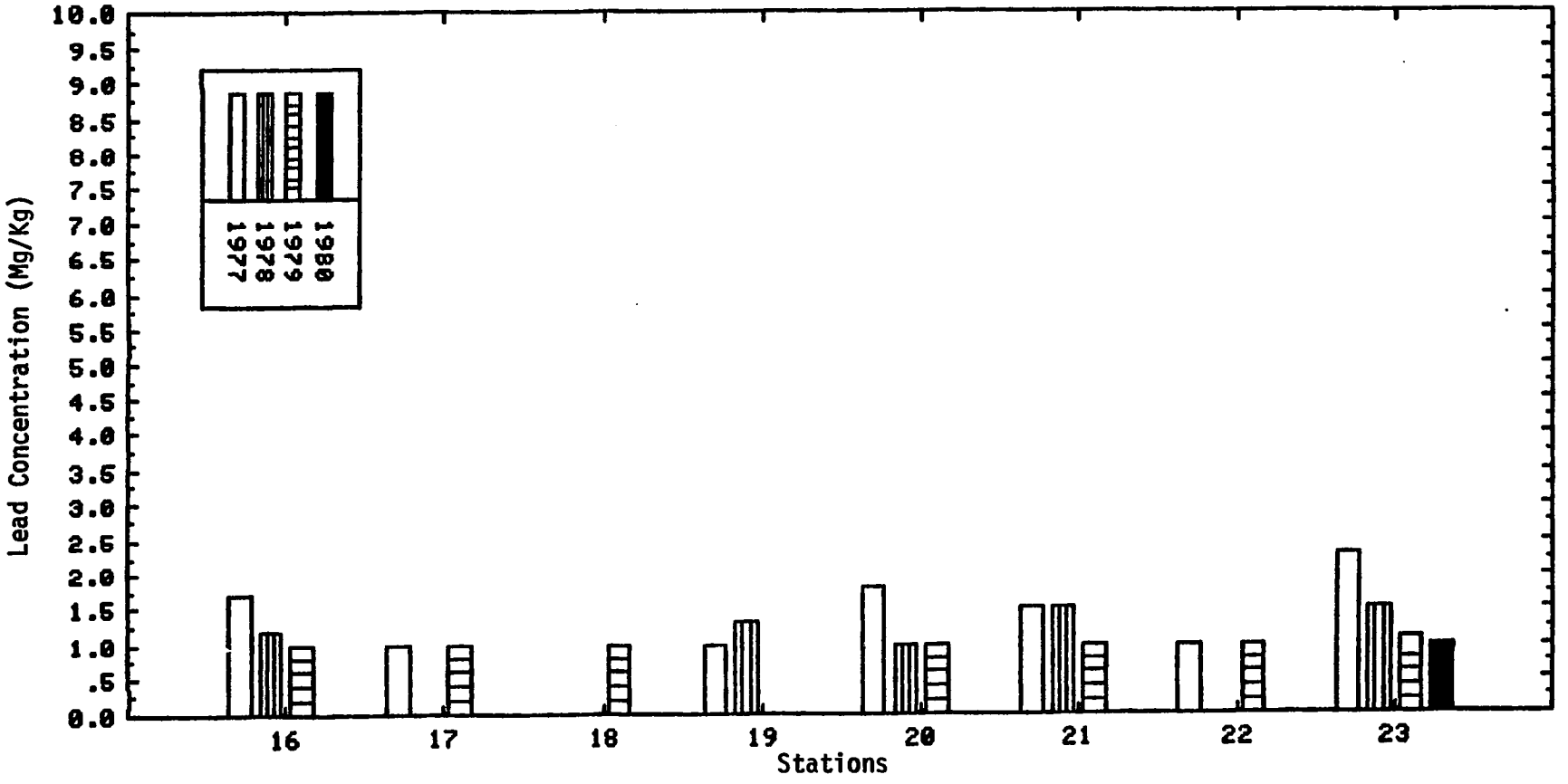

Figure B-22. Lead Content (Mg/Kg Body Weight) in the Fish Samples of the Carnivore Trophic Level from the Red River Basin.

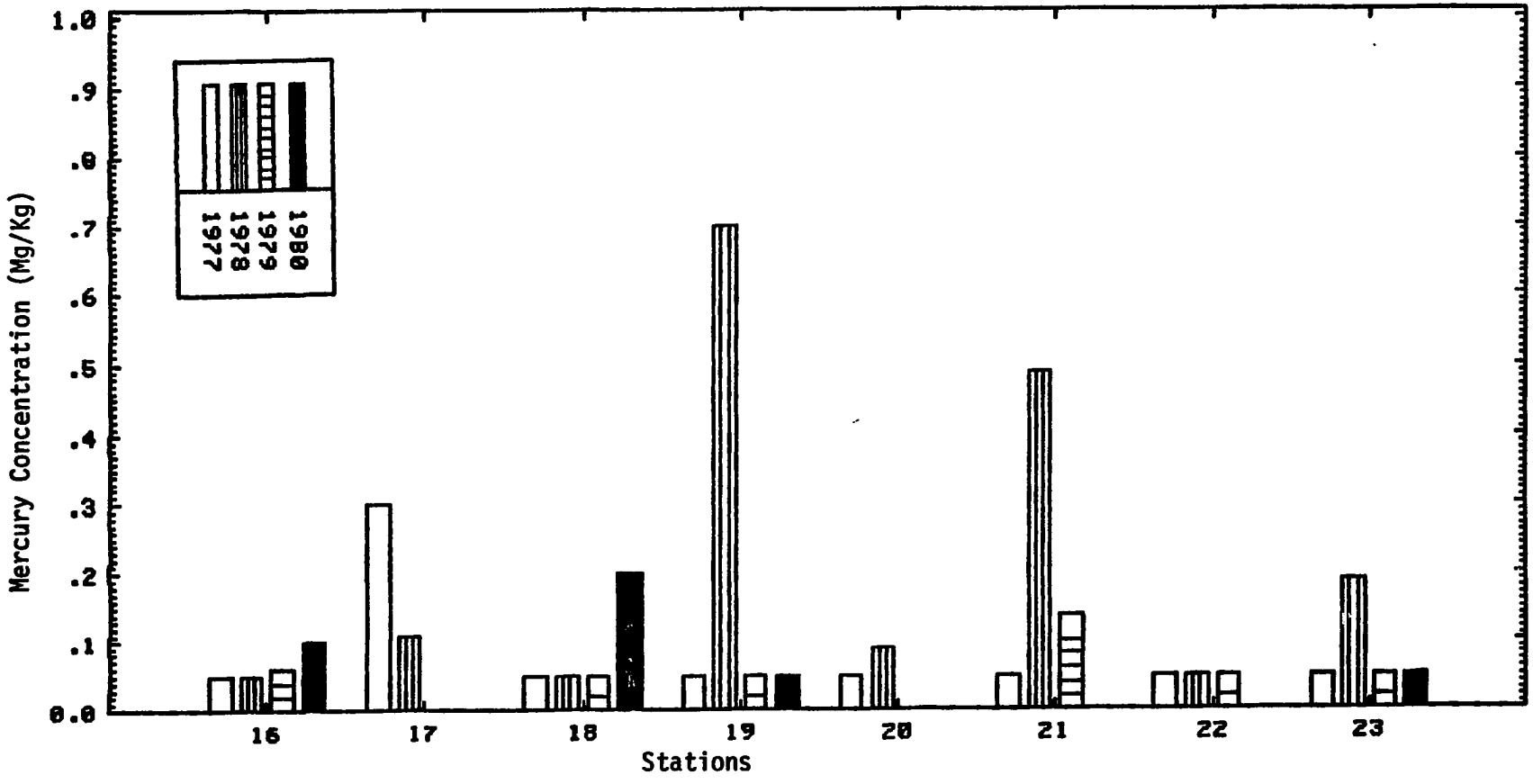

Figure B-23. Mercury Content (Mg/Kg Body Weight) in the Fish Samples of the Herbivore Trophic Level from the Red River Basin.

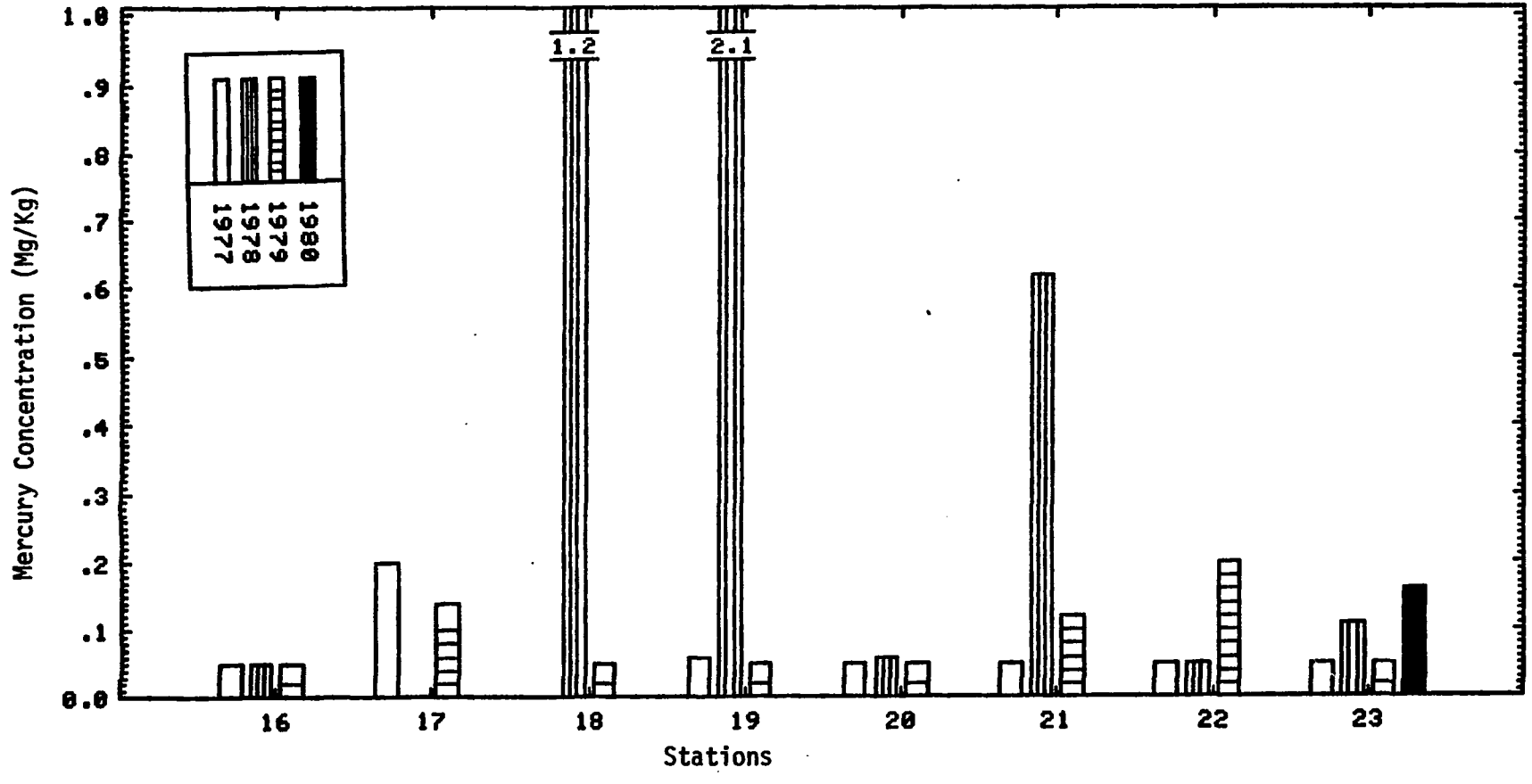

Figure B-24. Mercury Content (Mg/Kg Body Weight) in the Fish Samples of the Carnivore Trophic Level from the Red River Basin.

APPENDIX C

SAMPLE MEANS, STANDARD DEVIATION, MINIMUM, AND MAXIMUM

Table C-1. Mean, Standard Deviation, Minimum, and Maximum Values for Selected Parameters from the Arkansas River Basin.

Metal	Mean	Standard Deviation	Minimum	Maximum
Arsenic				
Herbivores	0.756	1.196	* 0.100	3.700
Carnivores	1.032	1.596	* 0.100	5.000
Cadmium				
Herbivores	0.146	0.132	* 0.100	0.800
Carnivores	0.138	0.161	* 0.100	1.000
Chromium				
Herbivores	1.502	1.367	* 0.500	6.090
Carnivores	3.148	2.353	* 0.500	15.000
Copper				
Herbivores	3.148	6.518	* 0.400	42.000
Carnivores	2.544	3.621	* 0.400	22.000
Lead				
Herbivores	1.240	0.930	* 1.000	4.000
Carnivores	1.174	1.466	* 1.000	9.000
Mercury				
Herbivores	0.098	0.112	* 0.050	0.700
Carnivores	0.305	0.756	* 0.050	4.800
Arsenic (water)	0.006	0.008	* 0.001	0.039
Cadmium (water)	0.004	0.004	* 0.001	0.020
Chromium (water)	0.025	0.043	0.008	0.310
Copper (water)	0.011	0.007	0.003	0.033
Lead (water)	0.028	0.016	* 0.001	0.070
Mercury (water)	0.086	0.064	* 0.001	0.165
Arsenic (sediment)	23.884	88.992	* 2.000	524.000
Cadmium (sediment)	0.848	0.586	* 1.000	3.000
Chromium (sediment)	15.207	15.720	* 5.000	80.000
Copper (sediment)	3.717	4.505	* 2.000	20.000
Lead (sediment)	2.813	6.483	* 1.000	34.000
Mercury (sediment)	17.300	11.629	* 5.000	29.000

* = Less Than Detection Limit

Table C-2. Mean, Standard Deviation, Minimum, and Maximum Values for Selected Parameters from the Red River Basin.

Metal	Mean	Standard Deviation	Minimum	Maximum
Arsenic				
Herbivores	0.638	0.818	* 0.100	2.000
Carnivores	0.870	1.143	* 0.100	2.400
Cadmium				
Herbivore	0.098	0.066	* 0.100	0.200
Carnivores	0.125	0.102	* 0.100	0.300
Chromium				
Herbivores	1.623	1.849	* 0.500	8.400
Carnivores	1.159	0.972	* 0.500	3.300
Copper				
Herbivores	2.138	2.251	* 0.400	11.000
Carnivores	1.942	1.692	* 0.400	7.400
Lead				
Herbivores	0.820	0.456	* 1.000	1.800
Carnivores	0.970	0.582	* 1.000	2.300
Mercury				
Herbivores	0.110	0.161	* 0.050	0.700
Carnivores	0.233	0.484	* 0.050	2.100
Arsenic (water)	0.009	0.012	* 0.001	0.033
Cadmium (water)	0.003	0.002	* 0.001	0.009
Chromium (water)	0.020	0.014	0.006	0.063
Copper (water)	0.009	0.008	* 0.001	0.034
Lead (water)	0.024	0.025	0.002	0.115
Mercury (water)	0.032	0.047	* 0.001	0.100
Arsenic (sediment)	23.840	53.727	* 2.000	230.000
Cadmium (sediment)	0.741	0.424	* 1.000	2.000
Chromium (sediment)	19.786	21.767	* 5.000	90.000
Copper (sediment)	3.070	2.892	* 2.000	10.000
Lead (sediment)	1.100	2.502	* 1.000	13.000
Mercury (sediment)	16.500	14.215	* 5.000	52.000

* = Less Than Detection Limit

Table C-3. Mean, Standard Deviation, Minimum, and Maximum Values for pH, Hardness, and Flow, from the Arkansas and Red River Basins.

Basin	Mean	Standard Deviation	Minimum	Maximum
Arkansas River Basin				
pH	7.8	0.43	6.9	8.4
Total Hardness	367.0	267.50	15.0	1472.0
Flow	2222.0	3443.00	7.0	13230.0
Red River Basin				
pH	7.5	0.42	6.7	8.3
Total Hardness	339.0	412.90	8.0	1192.0
Flow	637.0	1132.00	1.0	4551.0

APPENDIX D

CORRELATION COEFFICIENTS

Table D-1. Correlation Coefficients for Toxic Metals in Water, Sediment, pH, Total Hardness, and Flow to the Toxic Metal Levels in the Fish Samples From the Arkansas River Basin.

	Arsenic		Cadmium		Chromium		Copper		Lead		Mercury	
Water	Herb	Carn	Herb	Carn	Herb	Carn	Herb	Carn	Herb	Carn	Herb	Carn
Arsenic	-.1303	.4565	.1096	-.2387	.0279	.2106	-.0600	.3221	-.3587	.2871	-.0225	.1618
Cadmium	.5364	.3813	.2133	-.0004	.0055	-.0350	-.0921	.2493	-.1664	-.0527	.0828	-.0525
Chromium	.6515	.2141	.2910	.3153	.1535	.0715	-.0085	.4741	-.1064	.1461	.0635	-.0628
Copper	.3554	.2038	.0789	-.0795	-.0597	.1782	.0545	.4512	-.2702	.1515	.1305	-.1288
Lead	.2827	-.3224	-.2255	-.0183	-.3251	.2081	-.1142	-.0238	-.1790	.2617	-.0347	-.1472
Mercury	1.0000	.0199	.1008	-.2429	-.0358	.1866	-.0008	.2767	.1794	.1966	.7053	-.6640
Sediment												
Arsenic	.7028	.3336	.7731	.0584	.5147	.0673	.1373	.2708	.1870	-.0428	-.1606	-.0997
Cadmium	.0672	-.2205	-.0547	.1149	-.0142	-.1495	-.1720	-.2012	-.0476	-.1426	-.2123	.1823
Chromium	-.2803	-.3166	-.1342	-.1509	.1593	-.0344	.0032	.0021	.2965	-.0148	-.2207	.0013
Copper	-.0530	-.0010	.2929	.0066	-.0525	-.0410	.0019	.2002	.0961	.0683	-.0673	.2168
Lead	.0939	-.1609	.0748	-.0021	-.0957	-.1408	-.0321	-.0587	-.0134	-.0135	-.0978	.4036
Mercury	.0000	.2393	-.1355	-.1626	-.1832	-.0887	.6309	.0857	.7404	-.0558	.2196	-.1153
pH	-.2809	.5233	-.2223	-.1557	-.2270	.1898	.1978	.2506	.1485	.1938	.2170	-.2897
Hardness	-.1869	.7536	-.0038	-.1798	-.1021	.2705	-.0461	.3111	-.2785	.3403	.0119	-.1660
Flow	-.0575	-.4442	-.0383	-.0122	.0421	-.1003	-.0826	-.1897	.2241	-.0162	.6128	-.1199

Table D-2. Correlation Coefficients for Toxic Metals in Water, Sediment, pH, Total Hardness, and Flow to the Toxic Metal Levels in the Fish Samples from the Red River Basin.

	Arsenic		Cadmium		Chromium		Copper		Lead		Mercury	
Water	Herb	Carn	Herb	Carn	Herb	Carn	Herb	Carn	Herb	Carn	Herb	Carn
Arsenic	.8402	.6564	.5955	.6071	.0248	-.0832	-.7060	-.4015	-.4000	-.9017	-.3253	-.2660
Cadmium	-.2294	-.5894	.3638	-.3677	.0390	-.3554	.0875	-.0202	-.4312	.1223	-.2015	-.2480
Chromium	.6376	.6401	-.2056	-.1791	.3691	-.2842	-.0544	-.0444	-.0182	-.3327	.0889	.2651
Copper	.8821	-.1803	-.0804	-.1109	.4352	-.3398	.1660	.0048	.0942	-.2114	.0582	.0457
Lead	-.2589	-.6252	-.0682	-.3248	.2248	-.2045	.3993	-.0628	.4651	.0699	-.0317	-.1739
Mercury	.0000	.0000	.1032	-.3267	.2435	-.4504	.4897	.7966	-.3443	.4792	-.4109	-.3017
Sediment												
Arsenic	.3764	.7484	.2880	.5295	.3566	.6764	.3053	-.1880	.3898	.3660	-.1946	-.1793
Cadmium	-.2399	.4011	-.0445	.0642	-.0897	-.0764	-.0176	-.0294	-.4649	-.0641	.1633	.1504
Chromium	-.0919	.2109	-.1704	-.1849	.0493	.2548	.1630	.3171	.3619	.3579	.3588	.1649
Copper	-.2387	.7484	.2541	.3202	.2154	.4110	.0677	.2397	.0660	.0266	-.0368	-.1606
Lead	.0000	.0000	-.1459	-.1248	.8599	-.0880	.3009	.1951	.0478	-.0621	.2033	-.0455
Mercury	-1.0000	.0000	.1173	.1059	-.2977	-.1243	-.5138	-.0222	-.3788	-.3439	-.0710	-.2972
pH	-.0260	-.5934	-.1448	-.2479	.0398	-.4882	.1460	.2599	.0381	-.3427	.0660	.2280
Hardness	-.1817	-.5449	-.0482	-.2582	.1051	-.3251	.3389	.4797	.2336	.0608	-.1028	-.0712
Flow	.3542	-.3664	-.0616	.2440	-.1056	.1178	-.0504	.1663	-.2165	-.3508	-.1508	-.1172

Table D-3. Correlation Coefficients Relating Toxic Metals in the Water to the Levels in Sediment in the Arkansas and Red River Basins.

River Basin	Arsenic Sediment	Cadmium Sediment	Chromium Sediment	Copper Sediment	Lead Sediment	Mercury Sediment
Arkansas River Basin						
Arsenic (water)	-.0406	-.0378	-.3130	-.1579	-.2510	.0945
Cadmium (water)	-.0342	-.0070	-.0962	.2580	-.0741	-.2259
Chromium (water)	.1133	-.2137	-.0387	.1663	.0608	-.0553
Copper (water)	-.1264	-.1695	-.1873	.1025	-.1089	-.1644
Lead (water)	-.2541	.0034	-.0721	.0815	-.0849	-.3906
Mercury (water)	.7044	-.4222	-.4820	-.5325	-.6068	.5088
Red River Basin						
Arsenic (water)	.3811	-.1693	-.5027	.2983	-.2565	.8849
Cadmium (water)	-.2287	.4377	-.0105	-.3445	.0767	-.2184
Chromium (water)	-.1592	-.0302	-.2470	-.1497	.6213	-.4802
Copper (water)	-.1758	.1155	-.4030	-.3146	.6403	-.4640
Lead (water)	-.0114	.5608	.4133	-.2257	.0845	-.5799
Mercury (water)	-.2762	-.2910	-.6986	-.5220	.0000	-.4530

APPENDIX E

DESCRIPTION OF FISH SAMPLES

Table E-1. Species, Common Name, Number Collected, and Total Weight of Herbivore Fish Samples by Site Number and Water Year.

Site	Water Year	Scientific Name	Common Name	Number Collected	Weight (grams)
1	1977	Notropis lutrensis	Red Shiner	5	33
	1978	Notropis lutrensis	Red Shiner	30	109
	1979	Dorsoma cepedanum	Gizzard Shad	7	214
	1980	Ictiobus bubalus	Smallmouth Buffalo	2	1125
2	1977	Dorsoma cepedanum	Gizzard Shad	8	201
	1978	Notropis lutrensis	Red Shiner	30	160
	1979	Notropis lutrensis	Red Shiner	32	102
3	1977	Notropis lutrensis	Red Shiner	5	40
	1978	Notropis lutrensis	Red Shiner	36	71
	1979	Hybognathus placitus	Plains Minnow	24	8
	1980	Hybognathus placitus	Plains Minnow	14	16
4	1977	Notropis lutrensis	Red Shiner	4	4
	1978	Notropis lutrensis	Red Shiner	36	31
	1979	Dorsoma cepedanum	Gizzard Shad	1	36
	1980	Cyprinus carpio	Carp	5	1000
5	1977	Dorsoma cepedanum	Gizaard Shad	10	120
	1978	Dorsoma cepedanum	Gizzard Shad	24	200
	1979	Carpio carpio	River Carpsucker	2	66
	1980	Cyprinus carpio	Carp	8	5902
6	1977	Dorsoma cepedanum	Gizzard Shad	4	84
	1978	Dorsoma cepedanum	Gizzard Shad	14	260
	1979	Cyprinus carpio	Carp	1	1135
	1980	Ictiobus bubalus	Smallmouth Buffalo	1	1576
7	1978	Dorsoma cepedanum	Gizzard Shad	1	10
	1979	Dorsoma cepedanum	Gizzard Shad	1	54
	1980	Cyprinus carpio	Carp		
8	1978	Notropis pilsbryi	Dusky-striped Shiner	24	15
	1979	Notropis pilsbryi	Dusky-striped Shiner	14	4
9	1977	Dorsoma cepedanum	Gizzard Shad	1	13
	1978	Compostoma anomalum	Stoneroller	13	9
	1979	Dorsoma cepedanum	Gizzard Shad	2	34
10	1979				
	1980	Cyprinus carpio	Carp	3	300

Table E-1. (cont.)

Site	Water Year	Scientific Name	Common Name	Number Collected	Weight (grams)
11	1977	Notropis lutrensis	Red Shiner	6	10
	1978	Notropis lutrensis	Red Shiner	86	71
	1979	Notropis lutrensis	Red Shiner	48	23
	1980	Notropis lutrensis	Red Shiner	24	13
12	1977	Notropis lutrensis	Red Shiner	5	13
	1978	Notropis lutrensis	Red Shiner	25	23
	1980	Notropis lutrensis	Red Shiner	29	32
13	1977	Notropis lutrensis	Red Shiner	10	12
	1978	Notropis lutrensis	Red Shiner	36	33
	1979	Notropis lutrensis	Red Shiner	48	47
	1980	Hybognathus placitus	Plains Minnow	37	42
14	1977	Notropis lutrensis	Red Shiner	5	10
	1978	Notropis lutrensis	Red Shiner	36	42
	1979	Hybognathus placitus	Plains Minnow	12	14
	1980	Notropis lutrensis	Red Shiner	80	60
15	1977	Dorsoma cepedanum	Gizzard Shad	10	31
	1978	Dorsoma cepedanum	Gizzard Shad	8	27
	1979	Dorsoma cepedanum	Gizzard Shad	3	6
16	1977	Notropis lutrensis	Red Shiner	10	13
	1978	Notropis lutrensis	Red Shiner	25	23
	1979	Notropis lutrensis	Red Shiner	5	4
	1980	Cyprinus carpio	Carp	1	681
17	1977	Hybognathus placitus	Plains Minnow	36	32
	1978	Notropis lutrensis	Red Shiner	80	84
	1979				
18	1977	Notropis lutrensis	Red Shiner	10	13
	1978	Notropis lutrensis	Red Shiner	24	23
	1979	Notropis lutrensis	Red Shiner	12	11
	1980	Dorsoma cepedanum	Gizzard Shad	50	127
19	1977	Notropis lutrensis	Red Shiner	10	11
	1978	Notropis lutrensis	Red Shiner	25	20
	1979	Notropis lutrensis	Red Shiner	16	13
	1980	Notropis lutrensis	Red Shiner	30	20
20	1977	Notropis umbralitus	Redfin Shiner	10	16
	1978	Notropis boops	Bigeye Shiner	25	26
	1979				

Table E-1. (cont.)

Site	Water Year	Scientific Name	Common Name	Number Collected	Weight (grams)
21	1977	Notropis umbralitus	Redfin Shiner	10	13
	1978	Notropis boops	Bigeye Shiner	8	16
	1979	Notropis boops	Bigeye Shiner	14	25
22	1977	Notropis lutrensis	Red Shiner	10	12
	1978	Notropis lutrensis	Red Shiner	48	53
	1979	Notropis lutrensis	Red Shiner	16	21
23	1977	Notropis whipplei	Steelcolor Shiner	10	11
	1978	Notropis venustus	Blacktail Shiner	12	10
	1979	Notropis umbralitus	Redfin Shiner	13	21
	1980	Dorsoma cepedanum	Gizzard Shad	1	272

Table E-2. Species, Common Name, Number Collected, and Total Weight of Carnivore Fish Samples by Site Number and Water Year.

Site	Water Year	Scientific Name	Common Name	Number Collected	Weight (grams)
1	1977	Gambusia affinis	Mosquitofish	5	6
	1978	Aplodinotus grunniens	Freshwater Drum	4	10
	1979	Lepomis megalotis	Longear Sunfish	8	50
	1980	Lepisosteus osseus	Longnose Gar	2	2724
2	1977	Pomoxis annularis	White Crappie	3	170
	1978	Pomoxis annularis	White Crappie	1	454
	1979	Pomixis annularis	White Crappie	1	525
3	1977	Lepomis cyanellus	Green Sunfish	1	10
	1978	Fundulus kansae	Plains killfish	22	21
	1979				
	1980	Fundulus kansae	Plains killfish	30	25
4	1977	Pomoxis annularis	White Crappie	2	120
	1978	Lepomis megalotis	Longear Sunfish	5	60
	1979	Lepomis megalotis	Longear Sunfish	1	40
	1980	Pomixis annularis	White Crappie	2	65
5	1977	Morone chrysops	White Bass	5	110
	1978	Morone chrysops	White Bass	24	48
	1979	Micropterus salmoides	Largemouth Bass	3	1200
	1980				
6	1977	Morone chrysops	White Bass	2	72
	1978	Morone chrysops	White Bass	18	621
	1979	Ictalurus melas	Black Bullhead	1	561
	1980				
7	1978	Lepomis megalotis	Longear Sunfish	1	41
	1979	Pomoxis annularis	White Crappie	4	140
	1980	Ictalurus punctates	Channel Catfish		
8	1978	Lepomis megalotis	Longear Sunfish	1	22
	1979	Pomixis annularis	White Crappie	3	114
9	1977	Micropterus salmoides	Largemouth Bass	10	23
	1978	Micropterus salmoides	Largemouth Bass	2	8
	1979	Aplodinotus grunniens	Freshwater Drum	2	681
10	1979	Lepomis macrochirus	Bluegill Sunfish	1	1
	1980	Pomoxis annularis	White Crappie	3	117

Table E-2. (cont.)

Site	Water Year	Scientific Name	Common Name	Number Collected	Weight (grams)
11	1977	Lepomis megalotis	Longear Sunfish	1	42
	1978	Lepomis megalotis	Longear Sunfish		
	1979	Pomoxis annularis	White Crappie	1	39
	1980				
12	1977				
	1978	Pomoxis annularis	White Crappie	1	20
	1980				
13	1977	Lepomis cyanellus	Green Sunfish	2	28
	1978	Lepomis megalotis	Longear Sunfish	2	26
	1979	Lepomis cyanellus	Green Sunfish	59	119
	1980	Pomoxis annularis	White Crappie	1	50
14	1977	Pomoxis annularis	White Crappie	2	30
	1978	Lepomis megalotis	Longear Sunfish	4	16
	1979	Pomoxis annularis	White Crappie	7	2450
	1980	Ictalurus punctatus	Channel Catfish	1	744
15	1977	Pomoxis annularis	White Crappie	1	13
	1978	Lepomis macrochirus	Bluegill Sunfish	3	6
	1979	Lepomis macrochirus	Bluegill Sunfish	1	81
16	1977	Gambusia affinis	Mosquitofish	10	8
	1978	Micropterus salmoides	Largemouth Bass	3	27
	1979	Lepomis megalotis	Longear Sunfish	5	220
	1980				
17	1977	Lepomis megalotis	Longear Sunfish	2	13
	1978				
	1979	Lepisosteus osseus	Longnose Gar	3	681
18	1977				
	1978	Ictalurus punctatus	Channel Catfish	1	25
	1979	Ictalurus punctatus	Channel Catfish	1	60
	1980				
19	1977	Micropterus salmoides	Largemouth Bass	1	13
	1978	Micropterus salmoides	Largemouth Bass	4	30
	1979	Micropterus punctulatus	Spotted Bass	9	802
	1980				
20	1977	Lepomis cyanellus	Green Sunfish	2	24
	1978	Lepomis megalotis	Longear Sunfish	5	51
	1979	Lepomis megalotis	Longear Sunfish	3	507

Table E-2. (cont.)

Site	Water Year	Scientific Name	Common Name	Number Collected	Weight (grams)
21	1977	Lepomis megalotis	Longear Sunfish	2	31
	1978	Lepomis megalotis	Longear Sunfish	8	60
	1979	Lepomis cyanellus	Green Sunfish	1	10
22	1977	Lepomis megalotis	Longear Sunfish	1	11
	1978	Lepomis megalotis	Longear Sunfish	7	68
	1979	Lepomis humilis	Orangespotted Sunfish	2	86
23	1977	Lepomis megalotis	Longear Sunfish	2	18
	1978	Lepomis megalotis	Longear Sunfish	3	24
	1979	Lepomis megalotis	Longear Sunfish	2	21
	1980	Lepomis megalotis	Longear Sunfish	5	232