KWAME NKRUMAH UNIVERSITY OF SCIENCE TECHNOLOGY, KUMASI, GHANA

DISTRIBUTION OF TOTAL-MERCURY IN DIFFERENT TISSUES OF FISH EATING BIRDS ALONG THE VOLTA LAKE, GHANA.

BY

DOUGLAS AGYEMANG (B.Sc. Applied Chemistry)

THESIS SUBMITTED TO THE DEPARTMENT OF CHEMISTRY, COLLEGE OF SCIENCE IN PARTIAL FULFILMENT OF THE REQUIREMENTS FOR THE AWARD OF MASTER OF PHILOSOPHY IN ANALYTICAL CHEMISTRY

JUNE, 2013

DECLARATION

I hereby declare that, this thesis is the outcome of my own work towards MPhil and that to the best of my knowledge, it contains no material previously published by another person nor material which has been accepted for the award of any other degree of the University, except where due acknowledgement has been made in the text.

…… Date : ……

Agyemang Douglas

(PG 5033110)

(Student)

Certified by

…… Date:…….

Edward Ebow Kwaansa-Ansah

(Supervisor)

Certified by

…… Date:……..

Dr. R. B. Voegborlo

(H. O. D. Chemistry)

ABSTRACT

The distribution of Total mercury (THg) concentrations was determined in ten (10) different tissues of four different species of fish-eating birds from the Volta Lake, Ghana. The four species of fish-eating birds analysed in this study were Yellow billed kite (*Milvus migrans parasitus*), Squacco heron (*Ardeola rolloides)*, Little egret (*Egretta garzetta*) and Grey heron (*Ardea Cinerea*). Elevated levels of total mercury were found in all the tissues studied and were found to increase with body weight/age of the birds. Correlation analysis showed a very high positive relationship between concentrations of total mercury found in the tissues and weight/age of the birds. Highest total mercury concentrations were found in the liver, kidney and feather tissues of all the birds and they appeared to be the most preferred organs for mercury accumulation in the birds analyzed in this study. The order of mercury accumulation in the tissues obtained was as follows: lungs < heart <brain < gizzard < intestines < flesh < blood < kidney < liver < feather. Total mercury concentrations in the tissues of Little Egret, Yellow Billed Kite, Squacco Heron, and Grey Heron ranged from 0.91-1.78 µg/g; 0.43-2.14 µg/g; 0.85-1.92 µg/g and 0.77-2.86 µg/g respectively. The highest mean concentration of total mercury (1.84 ± 0.40 µg/g, range 1.40-2.86 µg/g, n=10) was found in *Ardea cinerea,* followed by *Ardeola ralloides* (1.40 ± 0.15 µg/g, range 1.19-1.65 µg/g n=10) , *Egretta garzetta* (1.38 ± 0.10 µg/g, range 1.24-1.58 µg/g, n=10) and *milvus migrans parasitus* (1.32 ± 0.19 µg/g, range 1.00-1.78 µg/g, n= 10). The highest mean concentration of mercury found in the grey herons (*Ardea cinerea*) suggests that they feed at a higher trophic level and prey on bigger fishes. Although the study reported elevated levels of total mercury concentrations in the tissues of the birds, they were however far lower than the threshold level (25-60 µg/g

w.w.) known to be detrimental to the health of the birds and humans and as such the Volta Lake has not been impacted so heavily as far as mercury pollution is concerned.

DEDICATION

This research is dedicated to my lovely daughter Princess Brianna Adusei Agyemang and my dearest wife Mrs. Vivian Mwin-Ira Agyemang whose support and prayers have made this work a success.

ACKNOWLEDGEMENT

I would like to express my sincere thanks and profound gratitude to the Almighty God for His guidance and protection throughout the period of the research.

I thank my mother for encouraging me and for teaching me patience and endurance, and for never allowing me to think for one moment that anything I wanted was beyond my reach.

I also thank my wife (Vivian), my sister (Cynthia) and Her Husband, Mr Elvis Boafo and my in laws Mr and Mrs Abdorn for their prayers and support.

I extend special thanks to Elder Abednego (boat owner), Ofotwo (boat operator) and Emmanuel Bobae (Hunter) all of Jatakpo-Yeji for their invaluable field assistance, insight, inspiration, and daily motivation.

Special thanks also go to Mr Elikim Otuachere Dept. of Wildlife, Faculty of Renewable Natural Resources who helped me with the identification of the birds. I also wish to acknowledge the effort of Mr. David Azanu Dept. of Chemistry, KNUST. who out of busy schedules took time to supervise the analysis.

I am also indebted to Mr. Osei Akoto, Dr. Darko, Dr. Osafo Acquah and Dr. Twumasi of Chemistry Dept. KNUST, for their immense contributions and suggestions.

Special thanks also go to all my course mates for their help, words of encouragement and their companionship.

Finally, I would like to give special thanks to my mentor and Supervisor Edward Ebow Kwaansa-Ansah for his invaluable support, patience, guidance, comments, criticisms and his fatherly pieces of advice. I especially thank him as he is the brain behind the success of this work and also for keeping such an unfading faith in me. Sir, may God richly bless you.

TABLE OF CONTENT

LIST OF TABLES

LIST OF FIGURES

LIST OF ABBREVIATIONS/ACRONYMS

AVS	**:**	**Acid-volatile sulfides**
AWQC	**:**	**Ambient Water Quality Criteria**
BAFs	**:**	**Bioaccumulation factors**
BCFs	**:**	**Bioconcentration factors**
CLP	**:**	**Contract Lab Program**
DOC	**:**	**Dissolved organic carbon**
DOLT-4	**:**	**Dogfish liver**
Eh	**:**	**Oxidation-reduction potential**
ERL	**:**	**Effects range-low**
ERM	**:**	**Effects range-median**
USFDA	**:**	**U.S. Food and Drug Administration**
Hg	**:**	**Mercury**
MT	**:**	**Metallothioneins**
µeq/l	**:**	**Micro equivalent per liter**
Me-Hg	**:**	**Methyl mercury**
NRCC	**:**	**National Research Council of Canada**
SRB	**:**	**Sulfate-reducing bacteria**
SPSS	**:**	**Statistical Package for the Social Sciences**
SRMs	**:**	**Standard Reference Materials**
T-Hg	**:**	**Total mercury**
TOC	**:**	**Total organic carbon**
TORT-2	**:**	**Lobster hepatopancreas**
w.w.	**:**	**Wet weight**

CHAPTER ONE

1.0 INTRODUCTION

1.1 BACKGROUND

Heavy metals are potentially toxic substances and their stability may cause certain problems in the environment (Spalding *et al*., 1994). Because of biomagnification of heavy metals, the higher members of the food chain may contain higher amounts of the metals several times more than the amount found in water or in air. It will consequently endanger the plants and the animals that consume the heavy metal contaminated food (Caldwell *et al*., 1999). Mercury (Hg) is one of the most toxic elements in the environment, considered as the most significant among other heavy metal pollutants. The increase in the global mercury levels in the last few decades is of concern because mercury is a persistent toxic heavy metal that bio-accumulates and biomagnifies in the environment. Its terrible damages to human health and that of other creatures have already been investigated in detailed. Mercury directly or indirectly has the potential to cause many diseases and side effects in human beings. It can also have toxic effects and damage on wild life and may cause miscellaneous side effects (Boening, 2000). It has been revealed that birds are quite sensitive to all pollutants and other damaging changes in the environment. To evaluate mercury accumulation, birds are often used as bioindicators of mercury in both the marine and freshwater environments (Fimreite, 1974; Barr, 1986; Scheuhammer, 1987; Wolf *et al*., 1998; Cohen *et al.*, 2000; Rumbold *et al.*, 2001; Henny *et al.*, 2002; Evers *et al.*, 2003; and Evers *et al.*, 2005). The position of the carnivorous birds at the top of the food chain and their long lifespan indicate that they are more affected by the pollutants and by the changes in the various parts of the ecosystem with time (Furness and Greenwood, 1993). Among birds, fish eating birds will suffer more damages due to Hg pollution, since the toxic Hg compounds specially

methyl mercury is formed in water and taken up into the food chain by planktons. Sundlof *et al*. (1994) revealed that the concentration of Hg in the liver of some wading birds collected from south Florida was so much that it caused some apparent nervous symptoms and some damages to their reproduction system and hence concluded that the reduction of some Ciconiiforms in Florida might be partially due to the Hg pollution of their food resources. In another study it was reported that some of the great white herons which suffered from chronic liver and kidney diseases as a result of Hg pollution were dead and it was concluded that the Hg pollution is harmful for the wading birds' health and reproduction (Spalding *et al*., 1994).

Anthropogenic activities that contribute significantly to the global input of Hg include: combustion of fossil fuels, mining and reprocessing of gold, copper, and lead, operation of chloralkali plants; and disposal of batteries and fluorescent lamps (NAS, 1978; Das *et al*., 1982).

Mercury amalgamation for gold extraction has been widely used in the mining communities in Ghana. Amalgamation of gold is still the preferred method employed by artisanal gold miners today because Hg is an effective, simple, and inexpensive reagent with which to extract gold (Veiga *et al,*. 1999). For this reason, mercury is widely used by small-scale, transient mining operations, which are numerous along the Black and the White Volta Basins (Boubacar, *et al*., 2005), rivers and creeks in the tropical forests and other locations in Ghana where there are abundant gold deposits.

In the Volta River Basin, the riverine ecosystems provide favorable conditions, such as high temperatures, high concentrations of organic matter and low pH, for the methylation of mercury and the subsequence exposure of organisms to methylmercury (MeHg) (Boubacar, *et al*., 2005). Methylmercury is readily taken up into the biosphere,

where it bioconcentrates in organisms and biomagnifies through food chains (Eisler, 1987).

The Volta Lake is a unique and an extraordinary natural resource that provide drinking water, food, recreation, employment, and transportation to millions of Ghanaians including tourist from abroad. As the world's second largest and Africa's largest artificial inland water body occupying about 3.6% of the total land area of Ghana and supplying about 90% of the nations inland fish supply, the impact of pollution in the Volta Lake has significant consequences for recreational, anglers, commercial fishermen, and subsistence fishers, as well as for the economic status of Ghana's valuable fisheries and tourism and the health of people and wildlife that depend on this ecosystem. Mercury is attracting a renewed global attention as there is evidence to show that environmental Hg levels have increased considerably since the onset of the industrial age and it is now present in various components of the environment especially fish across the globe at potentially toxic levels (Morel *et al*., 1998).

In recent years, growing attention has been paid to the environmental and health effects of Hg contamination and this is illustrated by the increasing number of international conferences devoted exclusively to mercury cycling in the environment. However, information on mercury contamination of fish eating birds in Ghana particularly the resident birds on the Volta Lake is very scanty and this research therefore seeks to provide data on the extent of mercury contamination in these birds**.**

1.2 PROBLEM STATEMENT

Mercury is a naturally occurring toxic metal that has become an important environmental and human health concern. Once Hg enters the aquatic environment it is transformed into MeHg which is the most toxic and the most bioavailable form of

mercury for living organisms. Because MeHg is formed in aquatic systems and not readily eliminated from organisms, it biomagnifies in aquatic food chains from bacteria, to plankton, through macro invertebrates, to herbivorous fish, to piscivorous fish and to piscivorous birds. At each step in the food chain, the concentration of MeHg in the organism increases. The concentration of MeHg in the top level aquatic predators can reach a level a million times higher than the level in the water. This is because MeHg has a half-life of about seventy two (72) days in aquatic organisms resulting in its bioaccumulation within the food chains. Organisms, including fish-eating birds and humans that consume fish from the top of the aquatic food chain may receive the MeHg that has accumulated through this process. As a result it has become very necessary to determine the levels of Hg in the tissues of these fish eating birds to ascertain the extent of Hg contamination in the Upper Volta Basin in Ghana.

1.3 AIMS AND OBJECTIVES

The main objective of this research is to conduct studies into the distribution of mercury in the different tissues of fish-eating birds' resident on the Volta Lake in Ghana.

1.4 SPECIFIC OBJECTIVES

The specific objectives include:

- Determination of the total mercury levels in the blood, brain, flesh, gizzard, heart, lungs, feathers, kidney, and liver of fish- eating birds.
- Determination of the correlation between the levels of mercury in the tissues of the birds and the body weight.
- Comparison of the results obtained with those from other water bodies worldwide.

- To make recommendations based on the outcome of the study.

1.5 JUSTIFICATION

The construction of the Akosombo dam which resulted in flooding of large forest areas of the Volta River Basin and its upstream fields, coupled with the perennial flooding of the River basin has led to accumulation of pollutants in the Volta Lake that needs to be closely monitored to ascertain whether they are reaching the threshold levels set out by World Health Organization. Increasingly it is becoming very necessary to understand the fate and effect of chemicals to assess the health of ecosystems and to provide early warning of changes in the environment that might indicate adverse effects. Mercury is one of the heavy metals which is especially toxic to humans and wildlife (Ohlendorf *et al*., 1978); thus its levels must be appropriately monitored in food chains and in different species (Thompson, 1990). To evaluate mercury accumulation in ecosystems, birds are often used as a bio-indicator of mercury in both marine and freshwater environments. Birds are particularly at risk from mercury poisoning because many species exclusively eat mercury laden fish. Birds also have a relatively high tolerance to mercury contamination in comparison to mammals, allowing them to live with much greater body burdens of mercury. Therefore, predatory birds are useful for representing the contamination of the ecosystem at levels higher than mammalian bioindicators (Shrum, 2009). In addition, they are also long-lived animals which accumulate mercury in their bodies over a long period of time (Evers *et al*, 2005). Fish-eating birds with high mercury contents might be a threat to people's health because most of these birds are delicacies for a lot of people particularly those who live along the Volta Lake. In recent years, most research on total and methyl mercury pollution in the Volta Lake has focused on sediments and different species of fish in the Volta Lake. In all these studies significant amount of mercury was reported (Kwaansa-Ansah *et al*., 2012).

However no research has been done on the resident birds on the Volta Lake which feed on fish in the Volta Lake. This research is therefore aimed at determining the extent of mercury pollution in these birds as a pointer to assess the health threats of people who eat these birds.

CHAPTER TWO

2.0 LITERATURE REVIEW

2.1 SOURCES OF ENVIRONMENTAL MERCURY

As a direct result of human activities, mercury levels in river sediments have increased fourfold since precultural times, and twofold to fivefold in sediment cores from lakes and estuaries (Das *et al*. 1982). During the past 100 years, it has been estimated that more than 500,000 metric tons of Hg entered the atmosphere, hydrosphere, and surface soils, with eventual deposition in subsurface soils and sediments (Das *et al*., 1982). Several activities that contribute significantly to the global input of Hg include the combustion of fossil fuels; mining and reprocessing of gold, copper, and lead; operation of chloralkali plants; and disposal of batteries and fluorescent lamps (NAS 1978; Das *et al*., 1982). The atmosphere plays an important role in the mobilization of Hg; 25% to 30% of the total atmospheric Hg burden is of anthropogenic origin (NAS, 1978).

In the United States, mercury consumption rose from 1,305 metric tons in 1959 to 2,359 tons in 1969 (Montague and Montague, 1971). The major use of mercury has been as a cathode in the electrolytic preparation of chlorine and caustic (Nriagu, 1979). In 1968 this use accounted for about 33% of the total U.S. demand for Hg (EPA, 1980). Of recent U.S. mercury consumption in electrical apparatus have accounted for about 27%; industrial and control instruments, such as switches, thermometers, and barometers, and general laboratory appliances, 14%; antifouling and mildew-proofing paints, 12%; Hg formulations to control fungal diseases of seeds, bulbs, and vegetables, 5%; and dental amalgams, pulp and paper manufacturers, pharmaceuticals, metallurgy and mining, and catalysts, 9% (EPA, 1980). Mercury, however, is no longer registered for use in antifouling paints, or for the control of fungal diseases of bulbs (EPA, 1980).

Mercury from natural sources enters the biosphere directly as a gas, in lava (from terrestrial and oceanic volcanic activity), in solution, or in particulate form; cinnabar (HgS), for example, is a common mineral in hot spring deposits and a major natural source of mercury (Das *et al*. 1982). The global cycle of Hg involves degassing of the element from the Earth's crust and evaporation from natural bodies of water, atmospheric transport (mainly in the form of Hg vapor), and deposition of Hg back onto land and water. Oceanic effluxes of Hg are tied to equatorial upwelling and phytoplankton activity and may significantly affect the global cycling of this metal. If volatilization of Hg is proportional to primary production in the world's oceans, oceanic phytoplankton activity represents about 36% of the yearly Hg flow to the atmosphere, or about 2,400 tons per year (Kim and Fitzgerald, 1986). Mercury finds its way into sediments, particularly oceanic sediments, where the retention time can be lengthy and where it may continue to contaminate aquatic organisms (Lindsay and Dimmick, 1983). Estimates of the quantities of Hg entering the atmosphere from degassing of the surface of the planet vary widely, but a commonly quoted figure is 30,000 tons annually (Clarkson *et al*., 1984). In aquatic ecosystems, removal of the source of anthropogenic Hg results in a slow decrease in the Hg content of sediments and biota (NAS, 1978). The rate of loss depends, in part, on the initial degree of contamination, the chemical form of Hg, physical and chemical conditions of the system, and the hydraulic turnover time (NAS, 1978).

2.2 CHEMICAL PROPERTIES OF MERCURY

Mercury, a silver-white metal which is liquid at room temperature and is highly volatile, can exist in three oxidation states: elemental mercury (Hg^0), mercurous ion (Hg_2^{2+}), and mercuric ion (Hg^{2+}). It can be part of both inorganic and organic compounds (EPA 1980; Clarkson *et al*., 1984). All mercury compounds interfere with

thiol metabolism, causing inhibition or inactivation of proteins containing thiol ligands and ultimately leading to mitotic disturbances (Das et al. 1982; Elhassani, 1983). The mercuric species is the most toxic inorganic chemical form, but all three forms of inorganic Hg may have a common molecular mechanism of damage in which Hg^{2+} is the toxic species (Clarkson and Marsh, 1982).

Chemical speciation is probably the most important variable influencing ecotoxicology of Hg, but Hg speciation is difficult, especially in natural environments (Boudou and Ribeyre, 1983). Mercury compounds in an aqueous solution are chemically complex. Depending on pH, alkalinity, redox, and other variables, a wide variety of chemical species are liable to be formed, having different electrical charges and solubilities. For example, HgCl2 in solution can speciate into $Hg(OH)_2$, Hg^{2+}, $HgCl^{+}$, $Hg(OH)^{-}$, $HgCl_3^{-}$, and $HgCl_4^{2-}$; anionic forms predominate in saline environments (Boudou and Ribeyre, 1983). In the aquatic environment, under naturally occurring conditions of pH and temperature, Hg may also become methylated by biological or chemical processes or both (Beijer and Jernelov, 1979; EPA 1980; Ramamoorthy and Blumhagen, 1984) although abiological methylation is limited (Callister and Winfrey, 1986). Methylmercury is the most hazardous mercury specie due to its high stability, its lipid solubility and possession of ionic properties that lead to its high ability to penetrate membranes in living organisms (Beijer and Jernelov, 1979).

All mercury discharged into rivers, bays, or estuaries as elemental (metallic), inorganic divalent, phenyl, or alkoxyalkyl can be converted into methylmercury compounds by natural processes (Jernelov, 1969). The mercury methylation in ecosystems depends on mercury loadings, microbial activity, nutrient content, pH and redox condition, suspended sediment load, sedimentation rates, and other variables (NAS, 1978; Compeau and Bartha, 1984; Berman and Bartha, 1986; Callister and Winfrey, 1986;

Jackson, 1986). The ascertion that certain microorganisms are able to convert inorganic and organic forms of Hg into the highly toxic methylmercury or dimethylmercury has made it clear that any form of Hg is highly hazardous to the environment (EPA 1980, 1985). The synthesis of methylmercury by bacteria from inorganic Hg compounds present in the water or in the sediments is the major source of this molecule in aquatic environments (Boudou and Ribeyre, 1983). This process can occur under both aerobic and anaerobic conditions (Beijer and Jernelov, 1979; Clarkson et al. 1984), but seems to favor more for anaerobic conditions (Olson and Cooper, 1976; Callister and Winfrey, 1986). Transformation of inorganic mercury to an organic form by bacteria alters its biochemical reactivity and hence its fate (Windom and Kendall, 1979). Methylmercury is decomposed by bacteria in two phases. First, hydrolytic enzymes cleave the C-Hg bond, releasing the methyl group. Second, a reductase enzyme converts the ionic Hg to the elemental form, which is then free to diffuse from the aquatic environment into the vapor phase. These demethylating microbes appear to be widespread in the environment; they have been isolated from water, sediments, and soils and from the gastrointestinal tract of mammals including humans (Clarkson *et. al*., 1984).

Methylmercury is produced by methylation of inorganic mercury present in both freshwater and saltwater sediments, and accumulates in aquatic food chains in which the top-level predators usually contain the highest concentrations (Clarkson and Marsh, 1982). Organomercury compounds other than methylmercury decompose rapidly in the environment and behave much like inorganic Hg compounds (Beijer and Jernelov, 1979). In organisms near the top of the food chain, such as carnivorous fishes, almost all Hg accumulated is in the methylated form, primarily as a result of the consumption of prey containing methylmercury. Methylation also occurs at the organism level by

way of mucous, intestinal bacteria, and enzymatic processes, but these pathways are not as important as diet (Huckabee *et al*. 1979; Boudou and Ribeyre, 1983).

The biological cycle of Hg is delicately balanced, and small changes in input rates and the chemical form of Hg may result in increased methylation rates in sensitive systems (NAS, 1978). For example, the acidification of natural bodies of freshwater is statistically associated with elevated concentrations of methylmercury in the edible tissues of predatory fishes (Clarkson *et al*., 1984). In chemically sensitive waterways, such as poorly buffered lakes, the combined effects of acid precipitation and increased emissions of Hg to the atmosphere (with subsequent deposition) pose a serious threat to the biota if optimal biomethylation conditions are met (NAS, 1978).

Mercury binds strongly with sulfhydryl groups and has many potential target sites during embryogenesis. Phenylmercury and methylmercury compounds are among the strongest known inhibitors of cell division (Birge *et al*., 1979). Organomercury compounds, especially methylmercury, cross placental barriers and can enter mammals by way of the respiratory tract, gastrointestinal tract, skin, or mucous membranes (Elhassani, 1983). When compared with inorganic mercury compounds, organomercurials are more completely absorbed, are more soluble in organic solvents and lipids, pass more readily through biological membranes, and are slower to be excreted (Clarkson and Marsh, 1982; Elhassani 1983; Greener and Kochen, 1983). Biological membranes, including those at the blood-brain interface and placenta, tend to discriminate against ionic and inorganic Hg, but allow relatively easy passage of methylmercury and dissolved Hg vapor (Greener and Kochen, 1983). As judged by membrane model studies, it appears that electrically neutral mercurials are responsible for most of the diffusion transport of Hg, although this movement is modified significantly by pH and Hg speciation. It seems, however, that the liposolubility of

methylmercury is not the entire reason for its toxicity and does not play a major role in its transport. This hypothesis needs to be examined further in studies with living membranes (Boudou *et al*., 1983).

Mercury-antagonistic drugs include 2,3-dimercaptopropanol, polythiol resins, selenium salts, vitamin E, and sulfhydral agents (Nriagu, 1979). Thiols (R-SH), which compete with Hg for protein binding sites, are the most important antagonists of inorganic mercury salts, and have been used extensively in attempts to counteract Hg poisoning in humans (Das *et al*., 1982). The protective action of selenium (Se) against adverse or lethal effects induced by inorganic or organic mercury salts has been reported for algae, aquatic invertebrates, fish, and mammals (Magos and Webb 1979; Heisinger 1979; Chang *et al*., 1981; Lawrence and Holoka 1981; Das *et al*., 1982; Gotsis, 1982; Eisler, 1985; Satoh *et al*., 1985). Selenite salts can release methylmercury from its linkage to proteins, and there is general agreement that a true antagonism exists between Se and Hg, although the exact mechanism is not fully established (Das *et al*., 1982). In marine mammals and humans, for example, Se and Hg concentrations are closely related, almost linearly in a 1:1 molar ratio, but this relation blurs in teleosts (in which Se is abundant) and fails in birds (Eisler, 1985).

2.3 ENVIRONMENTAL CHEMISTRY OF MERCURY

Mercury is among the most toxic of the heavy metals, has complex behavior in the environment, and may persist for decades following abatement of the source. Mercury's environmental persistence is due in part to its high affinity for particulates and organic matter. Even if mercury concentrations in sediment and water decrease over time, concentrations in organisms may not decrease due to the slow rate of elimination of the highly bioavailable methylmercury form. The physical properties, bioavailability, and toxicity of mercury are governed by speciation into both organic and inorganic forms.

Elemental mercury, bivalent inorganic mercury, and monomethylmercury are the three most important forms of mercury occurring in natural aquatic environments (Battelle, 1987). Elemental mercury in aquatic environments has a high vapor pressure, a low solubility in water, and an octanol-water partition coefficient (Kow) = 4.15 (Shoichi and Sokichi, 1985 as cited in Major *et al*., 1991). Elemental and dimethylmercury can occur as dissolved gaseous mercury. Mercury can also occur as particulate and dissolved ionic and monomethylmercury species. In natural water, ionic mercury is consumed by methylation, reduction, and particulate scavenging (Mason *et al*,. 1995a). Bivalent inorganic mercury binds to inorganic and organic ligands, especially sulfur-containing ligands, and forms both inorganic and organic complexes. Figure 1 shows a schematic of some common pathways of mercury speciation in the environment. Although most mercury occurs in the inorganic form, methylmercury, an organic form, is the most toxic and readily bioaccumulated form of mercury. Methylmercury normally occurs in the environment at extremely low concentrations; however, it is taken up easily by aquatic organisms and bioaccumulated. Consequently, methylmercury may comprise more than 95% of the mercury in fish tissue while only 5-15% of the total mercury burden in sediments and water of contaminated lakes is methylmercury (Saroff, 1990).

2.4 CHEMICAL SPECIATION OF MERCURY

Chemical speciation forms commonly used include total, inorganic, organic, and methylmercury and are based on the oxidation state and associated compounds. Mercury has three stable oxidation states: the native element (Hg[0]), mercurous (Hg[I]), and mercuric (Hg[II]). Inorganic mercury includes elemental Hg and some complexes of the mercurous and mercuric oxidation states. Hg [I] forms inorganic compounds only and, like Hg[0], cannot be methylated. Hg [I] compounds include

mercurous salts (halides) and mercurous chlorides such as calomel. Both Hg[0] and Hg[I] can be oxidized to form Hg[II]. H g[II] (bivalent mercury), the form that can be methylated, forms both organic and inorganic compounds. Mercury [II] can combine with inorganic ligands including chloride, hydroxide, nitrate, and sulfate anions (Benes and Havlik, 1979) to form inorganic mercury compounds that include mercuric halides, mercuric chloride (cinnabar), and mercuric sulfides. Chloride concentration and pH affect the proportions of the uncharged inorganic species in solution. For example, at low chloride concentrations most of the inorganic mercury occurs in the form of mercuric hydroxide ($Hg(OH)_2$), with mercuric chloride ($HgCl_2$) and mercury hydroxide chloride (HgOHCl) also important (Mason et al. 1996). As chloride concentration increases (e.g., high-chloride lake water), the proportion of $HgCl_2$ increases and the other two species constitute only a few percent of the total inorganic mercury. As pH increases, more Cl is needed than at lower pHs to increase the percent of $HgCl_2$. At even higher chloride concentrations, $HgCl_4^{2-}$ becomes the dominant species. This speciation chemistry affects the accumulation and toxicity as described later in this report. The term *organic mercury* can include different types of organically bound mercury. Hg [II] combines with organic compounds (humic/fulvic acids, amino acids) via an organic ligand bond to form organomercury salts. The Hg [II]-organic ligand bond is relatively weak compared to a C-Hg bond (Beckvar *et al*., 1996). The organomercury salts resemble their corresponding inorganic mercuric salts in their properties and reactions. The organomercury compounds methylmercury, dimethylmercury, and phenyl mercury have a C-Hg bond; methyl (CH_3) and phenyl groups (C_6H_5) link to a mercury atom via a carbon atom (Beckvar *et al*., 1996). Some authors group the organically bound Hg [II] complexes (without a C-Hg bond) with inorganic mercury compounds, while others group all organically bound mercury

together as organic mercury. Mercury compounds may be grouped according to their form based on chemical speciation discussed above, or based on the analytical technique used to measure the mercury. The analytical technique determines which forms of mercury are detected. Terminologies based on analytical procedures include as acid-soluble, reactive or acid labile and calcium chloride-extractable. Measurements of reactive mercury include Hg [II] bound to inorganic substances and weakly bound to organic matter (however, methylmercury is not included due to the strong C-Hg bond). To estimate methylmercury concentrations, some authors measure total mercury and reactive mercury, then subtract the reactive from the total measurement. Total mercury measurements include all the various inorganic and organic forms of mercury, including dissolved, colloidal, and/or particulate states. Speciation of mercury is affected by environmental conditions such as pH, oxidation-reduction potential (Eh), oxygen content, sulfide content, chloride concentration, organic matter content, and microbial activity. Similarly, biological and chemical processes control the conversion of inorganic mercury to methylmercury.

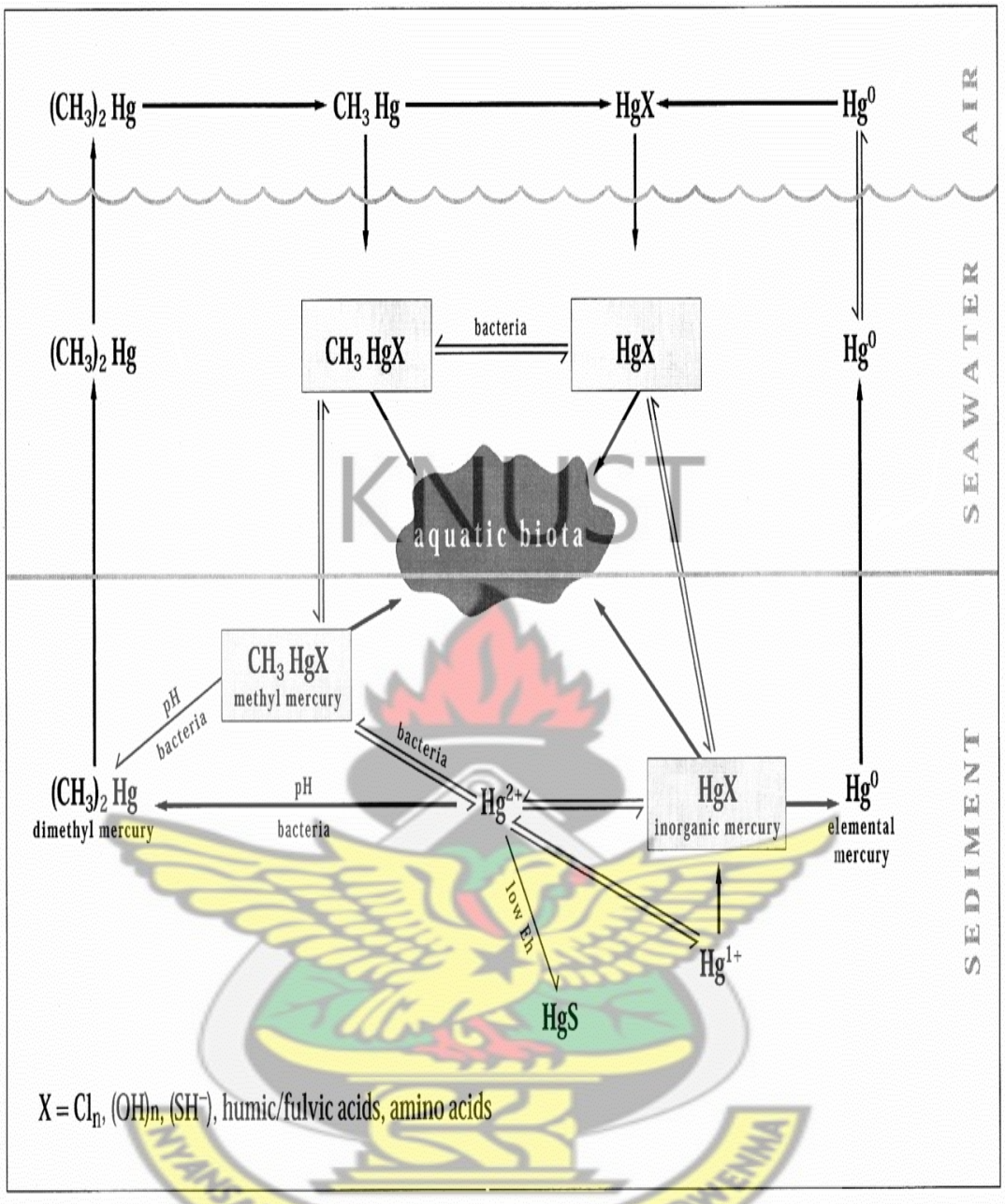


Figure 1. Important pathways of mercury speciation in the aquatic environment (Source: Beckvar *et al*, 1996)

2.5 METHYLATION OF MERCURY

In both freshwater and saltwater environments, mercury is converted from inorganic bivalent mercury (Hg[II]) to methylmercury primarily by microorganisms (Berman and Bartha, 1986), although chemical methylation also occurs (Craig and Moreton, 1985; Weber, 1993). Two forms of monomethylmercury, methylmercuric hydroxide

(CH_3HgOH) and methylmercuric chloride (CH_3HgCl) occur in both fresh and saltwater, with the former dominant at low chloride concentrations (low chloride freshwater) and the latter dominant at high chloride concentrations (high chloride lakes, seawater). As with inorganic mercury, the organic chloride species (Kow = 1.7) is more hydrophobic than the hydroxide species (Kow = 0.07) (Major *et al*. 1991; Faust 1992; Mason *et al*. 1996). Dimethylmercury (Kow = 182) readily volatilizes from surface water and is generally not persistent in aquatic environments at concentrations of concern; therefore, discussions of methylmercury in this review refer to the monomethylmercury species, unless otherwise stated. Methylmercury production depends on both the availability of Hg[II] for methylation and microbial activity. Methylation is usually greatest at the sediment water interface, but also occurs in the water column. Net methylmercury production is a function of both the rate of methylation and the rate of demethylation (Korthals and Winfrey, 1987). Methylmercury is not readily decomposed so the methylation rate is usually higher than the demethylation rate. Degradation of methylmercury is also primarily a microbial process. Methylation is influenced by the availability of Hg[II], oxygen concentration, pH, redox potential (Eh), presence of sulfate and sulfide, type and concentrations of complexing inorganic and organic agents (Parks *et al*., 1989), salinity (Blum and Bartha, 1980), and organic carbon (Jackson 1989; Winfrey and Rudd, 1990). Strongly bound Hg[II] is not available for methylation. For example, insoluble mercuric sulfide (HgS) will be methylated in aerobic sediments at rates 100 to 1,000 times slower than for the less strongly bound HgCl2 (Olson and Cooper, 1976).

Anaerobic, sulfate-reducing bacteria (SRB) are the primary methylators of mercury in both lacustrine and estuarine sediments (Compeau and Bartha 1985; Gilmour and Henry 1991). The primary methylators of mercury in the water column have not been

identified. SRB are common in sulfate-rich estuarine sediments (Hines *et al*., 1989) but are more limited in freshwater sediment with lower sulfate concentrations. A sulfate concentration of 200-500 μM in the water column is optimal for mercury methylation by SRB in sediment (Gilmour and Henry, 1991). The activity of the methylating microbes is affected by environmental conditions (Jackson, 1986) with nutrient availability and seasonality particularly important. The concentration of inorganic mercury in environmental media may not be a good indication of the concentration of methylmercury present due to the influence of environmental variables and biological activities. The importance of environmental factors in the production of methylmercury is as follows:

pH: Neutral or low pH conditions favor the production of monomethylmercury over dimethylmercury (Beijer and Jernelov, 1979). An alkaline (high) pH favors the formation of dimethylmercury, which tends to escape into the atmosphere. Elevated tissue concentrations of methylmercury have been noted in numerous pristine lakes of the northern United States and Canada that receive acid rain and no point sources of mercury (Xun *et al.*, 1987; Bloom *et al*., 1991). The mechanism(s) causing increased bioaccumulation in low pH lakes are not understood (Ramlal *et al*., 1985; Winfrey and Rudd 1990; Richardson and Currie, 1996). The factors primarily responsible for net methylmercury production in lakes are, in decreasing order of importance, pH, dissolved organic carbon (DOC) concentration, and microbial respiration (Miskimmin *et al*., 1992). The importance of pH and sediment properties (Fe and Mn content) on methylation rates in saltwater environments has not been well studied.

Sulfide, Sulfate, and Other Ions: In the presence of sulfides, the mercuric ion (Hg [II]) becomes tightly bound to sulfide as insoluble mercuric sulfide and is not available for methylation. Sulfide activity may be the main factor influencing the availability of

Hg [II] (Bjornberg *et al*., 1988) and the concentration of methylmercury in sediment (Craig and Moreton, 1983). If pH is high or Eh is low, sulfide activity will be high and mercury will be precipitated as insoluble mercuric sulfide. If the sulfide is oxidized to sulfate, the mercuric ion will become available for methylation. Both free sulfides and acid-volatile sulfides (AVS) appear to inhibit methylation (Gilmour and Capone, 1987). The presence of other minerals may affect this relationship. Excess ferrous iron has been found to bind the sulfide and limit its Hg-binding effectiveness such that no differences in methylation rates are noted between sulfide-rich and sulfide-poor sediments (Rudd *et al*., 1983). Selenium similarly binds the Hg [II] ions and reduces their availability for methylation. The redox cycling of manganese in lakes may be more important than iron-scavenging of mercury (Bonzongo *et al*., 1996). Addition of sulfate to anoxic lake sediment slurries or the overlying water column can increase methylmercury production by stimulating the SRB population (Gilmour *et al*., 1992). SRB can both methylate mercury and produce sulfide, which inhibits methylation: the kinetics of this are not understood. Mercury methylation was once viewed as a detoxification process in SRB, but it may ultimately serve some other function (Gilmour and Henry, 1991).

Oxygen Conditions/Eh*:* Although methylation occurs under both aerobic (oxidizing) and anaerobic (reducing) conditions, methylation is greater under anaerobic conditions (Callister and Winfrey 1986; Weis *et al*. 1986; Regnell 1994). In addition, demethylation rates are lower under anaerobic conditions, so the net methylmercury production is higher in oxygen-depleted environments (Jackson, 1987). Over 90 percent of methylmercury is formed biochemically in anaerobic sediment (Berman and Bartha, 1986). In anoxic lake bottoms containing hydrogen sulfide, mercury is bound to sediment as insoluble mercuric sulfide. If conditions become aerobic due to a decrease

in the organic load or seasonal turnover, sulfide can be oxidized to sulfate, releasing the mercury in the ionic form Hg[II], which is available for methylation (Jernelov, 1968).

Nutrients /Organic content: /DOC: Nutrients can enhance the rate of methylmercury production by stimulating the methylating bacteria. Decaying organic matter can enhance microbial activity and create low oxygen conditions, both of which cause higher methylation rates (Olson and Cooper, 1974; Gilmour and Henry, 1991). In freshwater areas with a high organic input, methylation rates can become locally elevated if other environmental conditions do not inhibit methylation (i.e., high sulfide levels; Jackson, 1986). When sulfide and sulfate concentrations are not limiting, organic matter may be the major factor controlling mercury methylation rates in estuarine sediments (Choi and Bartha, 1994). Increased DOC levels may inhibit methylation due to the binding of free mercury ions (Jackson, 1989; Winfrey and Rudd, 1990) even though supplemental DOC increases microbial respiration (Miskimmin *et al*., 1992). In clear freshwater lakes, DOC and pH may interact such that less of the Hg [II] is bound by DOC at low pH, resulting in higher methylation rates. Acid rain may also limit the amount of DOC transported into a system because at lower pH, DOC solubility and mobility is reduced (De Haan 1992; Schindler *et al*., 1992).

Humic/Fulvic material: The geochemistry of Hg in lake and stream water may be dominated by humic material interactions (Mierle and Ingram, 1991). Hg complexes with humic and fulvic substances and Hg retention and export from watersheds in Canada have been correlated with the export of humic substances. Hintelmann *et al*. (1995) assumed that the methylmercuric ion is bound to sulfidic binding sites of humic acid. At lower pHs, the amount of free unbound methylmercury ion was higher in their laboratory study of humic and fulvic acids. Acidification could potentially release

bound methylmercury from humic acids into the aqueous phase where it would be readily bioavailable.

Salinity: There appears to be a negative correlation between the rate of methylmercury formation and salinity in estuarine sediments (Blum and Bartha, 1980). The rate is lower in more saline environments because the bicarbonate component of seawater slows methylation of Hg [II] under both aerobic and anaerobic conditions (Compeau and Bartha, 1983). The release of reactive Hg [II] and Hg [0] is slowed when chloride ions bind to mercury, thereby inhibiting methylmercury formation (Craig and Moreton, 1985). Salinity also affects methylation due to the high pore-water sulfide concentrations as a result of rapid sulfate reduction in saline water compared to sulfate-limited freshwater environments (Gilmour *et al*., 1992). Along a salinity gradient in the lower Hudson River, methylation rates decreased downriver with increasing salinity and sediment sulfide concentrations (Gilmour and Capone, 1987).

The percentage of total mercury that is methylmercury is higher in freshwater sediments (up to 37%) and water (up to 25% in aerobic water and 58% in anoxic bottom water) than in estuarine and marine water (<5%) and associated sediments (<5%) (Gilmour *et al*., 1991). Dissolved reactive mercury (inorganic species) forms the majority of the total mercury in open oceans (Bloom and Crecilius, 1983; Gill and Fitzgerald, 1987).

Season: Biological productivity of methylating microbes is affected by seasonal changes in temperature, nutrient supply, oxygen supply, and hydrodynamics (changes in suspended sediment concentrations and flow rates). Methylmercury concentrations varied seasonally by an order of magnitude at most sites studied (Parks *et al*., 1989). Methylation may tend to increase during the summer months when biological

productivity and temperature are high and decrease during winter months when biological productivity and temperature are low (Callister and Winfrey, 1986; Jackson, 1986; Weis *et al*. 1986; Korthals and Winfrey, 1987; Parks *et al*., 1989; Kelly *et al*., 1995; Leermakers *et al*., 1995). Although the potential methylmercury production is greatest during the summer, actual production may not peak during this time (Kelly *et al*., 1995). In Onondaga Lake, New York, the mercury species in the water column varied temporally (Battelle, 1987; Bloom and Effler, 1990). Total mercury concentrations may also vary seasonally due to physical factors such as winter storms resuspending mercury-contaminated sediments (Gill and Bruland, 1990).

2.6 DISTRIBUTION OF MERCURY IN THE ENVIRONMENT

The distribution and abundance of inorganic mercury and methylmercury in the environment may vary independently as they are controlled by different physicochemical processes. The concentration of total mercury (which is mainly inorganic) in the environment is generally not a good predictor of methylmercury concentration (Gilmour and Henry 1991; Kelly *et al*. 1995). Inorganic mercury has a high affinity for sediments; a significant portion of the total mercury in fresh water is in particulate form (Gill and Bruland, 1990). Most of the mercury in estuaries was associated with particulate matter (Cossa and Noel, 1987). The environmental distribution of inorganic mercury appears to be controlled by processes such as transport, sorting, and sedimentation as related to the hydrologic regime. Resuspension and resettling of sediments caused persistently high concentrations of mercury in the surface sediments of Lavaca Bay (Reigel, 1990).

Total mercury concentrations in surface water may decrease as mercury bound to particulate matter settles or is transported downstream (Bonzongo *et al*., 1996). The distribution of biotically produced methylmercury initially depends on the microbial

populations that methylate the mercury. Although more abundant in the sediment where it is formed, methylmercury forms a greater percentage of the total mercury in the overlying water column (Gilmour *et al*., 1992). The distribution of both inorganic and methylmercury is also affected by the large-scale physical characteristics of the environment such as type of system (river, lake, estuary, ocean) and its physical configuration, water circulation patterns, catchment type, sediment characteristics, rainfall, and the introduction of terrestrial sediments. The physical characteristics of the system influence the mechanisms of mercury distribution, availability, and cycling in varying degrees. Water flow regimes in particular subenvironments (mainstream versus backwater), characteristics such as the stratification cycle and amount of nutrients in lakes, or the configuration of the estuary (open circulation versus restricted), may have very different, distinct features that control both the persistence of mercury in the environment and how the different species of mercury behave and are distributed among sediment, water, and organisms.

In freshwater systems, Kelly *et al*. (1995) found a predictive, linear relationship between total and methylmercury concentrations in unfiltered water samples from some specific lake systems, but not from stream systems. Runoff from wetland catchments contributed more methylmercury to lake systems than did runoff from upland catchments (St. Louis *et al*., 1994). In estuarine systems, total dissolved mercury concentrations were found to be enhanced where salinity was less than 10 ppt, coinciding with the maximum turbidity zone (Cossa and Noel 1987; Cossa *et al*. 1988). Terrestrial sediment influxes can also affect mercury availability. In the estuarine Ala Wai Canal in Hawaii, total mercury increased over two orders of magnitude in polychaetes and shrimp during the rainy season (Luoma, 1977). Mercury bound to freshwater sediments and introduced into the estuary from urban runoff during rainfall

was desorbed upon contact with saline water. The increased concentration of dissolved mercury temporarily increased total mercury in filter-feeding worms and shrimp. Total mercury concentrations in the water column and biota decreased after the runoff stopped. In contrast, lower mercury concentrations were found in plankton in a freshwater lake after input of high concentrations of clean, fine-grained sediment. Sediments washed into the lake during rainfall bound the mercury, inhibiting uptake by plankton (Jackson, 1988).

Sediment composition can also affect the way that mercury is distributed in the environment. Mercury concentrations in freshwater benthic organisms appeared to be determined by the sediment composition, such as the concentration of hydrated Fe and Mn oxides and carbon-rich humic matter in bottom sediment. The mercury appeared to be less available when it was bound by iron hydroxide (FeOOH), manganese hydroxide (MnOOH), and possibly by higher-molecular-weight humic substances (Jackson, 1988).

In a freshwater river-lake system in Canada (Parks *et al.,* 1989) methylmercury concentrations in surface water were highest 80 kilometers downstream from the most contaminated sediments (contaminated with inorganic mercury). Fish were contaminated as far as 270 km downstream from the inorganic mercury source, with the most contaminated fish found more than 100 km downstream of this source. Methylmercury concentrations in the water increased as inorganic mercury concentrations in the sediment decreased. The most highly contaminated sediments were located near a sewage outfall. The researchers surmised that the mercury in these sediments was bound to sulfide and thus not available for methylation. Further downstream the mercury became available for methylation probably due to a decrease in sediment sulfide levels. Even though concentrations of inorganic mercury in the sediment here were much lower than upstream concentrations, methylmercury

production was much higher and biotas were more contaminated. There may be a similar situation in low-salinity water of Berrys Creek, New Jersey, where high inorganic sediment mercury concentrations were also found next to a sewage outfall (Weis *et al*., 1986). However, concentrations of mercury in fish inhabiting the area were not as high as expected. This was attributed to the presence of sulfide, which binds mercury and limits methylation. No downstream studies have been conducted to determine whether the mercury is more bioavailable further from the source of high sulfide concentrations.

An eight-ton cargo of elemental mercury located within the hold of the sunken Empire Knight in offshore marine waters did not contaminate invertebrates living outside the hold of the ship (Hoff *et al.,* 1994). Only a small percentage of invertebrates sampled from within the hold had elevated concentrations of total mercury. However, it is not know whether the mercury was incorporated into the tissue of the organisms. The large source of elemental mercury in this environment was not bioavailable to organisms located away from the source.

2.7 BIOACCUMULATION OF MERCURY

Mercury bioaccumulates in aquatic plants, invertebrates, fish and mammals. Concentrations increase (biomagnify) in higher-trophic-level organisms. Even though the different types of mercury have relatively low Kow values (compared to organic compounds such as PCBs), they are readily accumulated. Inorganic mercury (excluding elemental) and methylmercury's strong reactivity with intracellular ligands is thought to be responsible for their high degree of accumulation. Uptake and accumulation of mercury are affected by the type of mercury present, with neutral mercury species (e.g., $HgCl_2^0$ and CH_3HgCl^0) absorbed more efficiently than charged mercury species (e.g., $HgCl^{-3}$ CH_3Hg+; Mason *et al*., 1996**)**.

Despite the fact that the neutral inorganic and organic complexes have similar lipid solubilities, methylmercury is selectively accumulated (due to a higher transfer efficiency and lower rate of elimination), resulting in biomagnification in higher trophic levels (Mason *et al*., 1995b). Inorganic mercury species are not biomagnified (Surma-Aho and Paasivirta 1986; Riisgård and Hansen 1990; Hill *et al*., 1996). Environmental factors that enhance mercury methylation result in greater bioavailability and accumulation of methylmercury. Environmental variables also influence the bioavailability and accumulation of inorganic mercury. Although concentrations of mercury in the environment may correlate with concentrations in resident plants and biota, correlation is often difficult. Correlating total mercury in sediment with total mercury in upper-trophic-level organisms is complicated by high methylmercury concentrations in high-trophic-level organisms relative to low methylmercury concentrations in the environment.

Tissue concentrations of mercury are often positively correlated with organism length, weight, and/or age. Diet has a significant role in the overall body burden of mercury, both between and within species. Differences in total mercury concentrations between species reflect diet differences due to trophic position; within-species differences are related to dietary requirements of various developmental stages.

2.8 THE EFFECT OF THE FORM OF MERCURY ON BIOACCUMULATION

Both inorganic and methylmercury are taken up directly from water and food (or ingested sediment). However, methylmercury is more efficiently accumulated than inorganic mercury for most aquatic organisms (Fowler et al. 1978; Julshamn *et al*., 1982; Riisgård and Hansen, 1990; Mason *et al*., 1995b). The uptake and depuration of mercury depends on the form of mercury, source of mercury (water or food), and the type of receptor tissue, resulting in different patterns of accumulation. Methylmercury

is readily transferred across biological membranes. Within the organism, methylmercury is strongly bound to sulfhydryl groups in proteins of tissues such as muscle, and is much slower to depurate than inorganic mercury. Thus, methylmercury has a much greater potential for bioaccumulation and a longer half-life in organisms than inorganic mercury.

The accumulation of mercury from water occurs via the gill membranes. Gills take up aqueous methylmercury more readily than inorganic mercury (Huckabee *et al*. 1979; Boudou *et al*. 1991). Methylmercury is eventually transferred from the gills to muscle and other tissues where it is retained for long periods of time (Julshamn *et al*. 1982; Riisgård and Hansen, 1990). Inorganic mercury taken up with food initially accumulates in the tissues of the posterior intestine of fish (Boudou *et al*. 1991). Inorganic mercury is not easily transferred through this organ to other parts of the body. After 15 days, 80% had depurated from the fish intestine. Liver and kidney in fish tend to have higher percentages of inorganic mercury than muscle tissue, although percentages vary by organ and species (Windom and Kendall, 1979; Riisgård and Hansen, 1990).

Methylmercury ingested in food is efficiently transferred from the intestine to other organs (Boudou *et al*., 1991). Methylmercury has been reported to constitute from 70 to 95% of the total mercury in skeletal muscle in fish (Huckabee *et al*. 1979; EPA 1985; Riisgård and Famme 1988; Greib *et al*. 1990; Spry and Wiener, 1991). Methylmercury accounted for almost all (»99%) of the mercury in muscle tissue in a wide variety of both freshwater and saltwater fish found in waters not highly contaminated by other organomercurial species (Bloom, 1992). The ratio of liver to muscle total mercury concentration usually fluctuates around one and can reflect the exposure history of the organisms. For example, the liver : muscle ratio may be less than one in chronically

exposed fish, while a recent exposure to mercury may result in a ratio greater than one (Riisgård and Hansen, 1990).

McKim *et al.* (1976) reported that mercury could be transferred from adult to offspring in brook trout. Exposure of the parent population to aqueous methylmercury concentrations of 0.03 to 2.93 μg/l in the laboratory resulted in mercury concentrations as high as 2 mg/kg in their embryos. Total mercury concentrations in eggs of several species of adult fish from Swedish lakes were much lower than concentrations in other tissues; therefore, spawning did not lower their total mercury body burden (Lindqvist, 1991). The main depuration pathway is through the kidney and liver in fish. Half-lives for methylmercury in fish range from one to three or more years (McKim et al. 1976; Pentreath 1976a, b; Riisgård and Famme 1986; Riisgård and Hansen 1990), while estimates of half-lives for inorganic mercury are much lower, ranging from approximately five days to five months (Pentreath 1976a, b; Huckabee *et al.*, 1979).

Invertebrates accumulate and partition inorganic and methylmercury in tissues similar to the trends exhibited by fish (Fowler 1978; Riisgård and Famme 1986; Saouter et al. 1991; Saouter *et al.*, 1993). However, invertebrates generally contain a lower percentage of methylmercury than fish or mammals (Lasorsa and Allen-Gil, 1995), with highly variable concentrations. This wide variation of mercury content in invertebrates is most likely a function of different feeding strategies (and trophic levels) and different environmental exposures. Reported percentages of methylmercury compared to total mercury concentrations are less than 1% for the polychaetes *Nereis succinea* (Luoma, 1977); 10% in copepods, mussels and shrimp (Horva,t 1991); 10-100% in the cockle (Møhlenberg and Riisgård, 1988); 16% in urchin gonads (Eganhouse and Young, 1978); 30-90% in lake zooplankton (Lindqvist 1991); 87% in crab muscle (Eganhouse and Young, 1978); and 100% in red rock crab, Dungeness crab, and spot shrimp

(Bloom, 1992). Becker and Bigham (1995) found an increasing percentage of methylmercury compared to total mercury in higher trophic levels in the Onondaga lake food web. Lake water contained 5% of total mercury as methylmercury; phytoplankton 24%; benthic macro invertebrates 26%; zooplankton 40%; and fish fillets 96%. Viscera in mussels contained the highest tissue concentration of total mercury (Fowler *et al.*, 1978). The total mercury concentration was highest in the midgut and muscle tissue in crab (Bjerregaard and Christensen, 1993) and in the viscera in shrimp (Fowler *et al.*, 1978). The shrimp molts had the lowest mercury content; therefore, molting is not considered an important depuration pathway in crustaceans (Fowler *et al.*, 1978).

Half-lives for total mercury in salt-water mussels ranged from two months to one year (Riisgård *et al.*, 1985). Inorganic mercury was eliminated more rapidly than methylmercury in mussels and shrimp (Fowler *et al.*, 1978).

Marine mammals have some of the highest tissue mercury concentrations of all marine organisms investigated (Andre *et al.*, 1991a); however, concentrations are highly variable both within and among species. These variations have been attributed to collection locations (Wren, 1986), concentrations in prey items (Szefer *et al.*, 1993), and organism age (Julshamn *et al.*, 1987; Thompson, 1990). For example, species that feed primarily on benthic invertebrates, such as walruses and baleen whales, tend to have relatively low mercury concentrations. In contrast, fish eating species, such as porpoises and seals, exhibit relatively high mercury concentrations (Born *et al.*, 1981).

In contrast to fish, adult marine mammals have a much higher percentage of total mercury as inorganic mercury, although the concentration of methylmercury may also be elevated. Less than 10% of the total mercury content is methylmercury (Eisler, 1987). Juveniles tend to have higher percentages of methylmercury. The liver generally

exhibits the highest total and methylmercury concentration (Holden, 1978; Wagemann *et al*. 1983; Julshamn *et al*. 1987; Thompson 1990; Andre *et al*. 1991a), followed by kidney and muscle tissues (Szefer *et al*., 1993). Julshamn *et al*. (1987) measured the highest concentrations of methyl- and total mercury (13 and 150 mg/kg) in pilot whale livers (*Globicephalus meleanus*) compared to total and methylmercury concentrations in muscle (2.8 mg/kg total mercury; 1.7 mg/kg methylmercury) and kidney (15.3 mg/kg total mercury; 5.1 mg/kg methylmercury). Andersen *et al*. (1987) also measured the highest methylmercury concentrations in pilot whale liver (20 mg/kg; 14% of total mercury). The fraction that is methylated, however, is usually lower in the liver compared to muscle and kidney. The methylated fraction of total mercury ranged from 1% to 36% in seal liver (Holden,

1978); 30% in older specimens to 100% in young specimens in the muscle of harbor porpoise (Joiris *et al*., 1991); and 24% to 86% in the muscle of pilot whales (*Globicephalus meleanus*; Julshamn *et al*., 1987). The liver: muscle ratio for methylmercury concentration in harbor porpoises was approximately one, while the ratio for liver: muscle for total mercury concentration was two. In some of the harbor porpoises and some other species (sperm whale, common dolphin, and adult bottle-nose dolphin) the liver: muscle ratio for total mercury ranged up to 20 (Joiris *et al*., 1991) while the liver: muscle ratio for methylmercury was still one. Schintu *et al*. (1992) observed an age-related change in the percentage of methylmercury compared to total mercury in pilot whale livers. The liver of three to seven-year old pilot whales (with a relatively low total mercury body burden) contained 30% to 60% organic mercury, compared to 3% to 17% organic mercury in livers of 30- to 40-year old pilot whales (with a relatively high total mercury load). Porpoises exhibited a similar trend. Juveniles had a higher percentage of methylmercury in liver (100%), while the

percentage of methylmercury in adult specimens decreased to 2 to 3% of the total mercury (Joiris *et al*., 1991). Although mercury in the diet of many marine mammal species is predominantly methylmercury, it has been proposed that the mammals are able to mineralize methylmercury into the more harmless inorganic form, which then accumulates in the liver of adult specimens (Holden 1978; Joiris *et al*. 1991). The estimated half-life of total mercury in pinnipeds and dolphins is about 1.4 and 2.7 years, respectively (Eisler 1987; Andre *et al*. 1991b).

2.9 EXPOSURE PATHWAYS OF MERCURY

Aquatic organisms can accumulate mercury from water (including pore water) and food sources (including sediment). Quantity accumulated is a function of the exposure pathway and the physical and environmental factors such as temperature, pH, salinity, total organic carbon, and sulfides. If conditions are favorable for methylation, organisms can accumulate high concentrations of mercury even with low concentrations in the water and sediment (Beckvar et al., 1996). Phytoplankton, invertebrates, fish (including eggs and larvae), and mammals take up inorganic and organic mercury from the water column (McKim et al., 1976; Pentreath 1976a; 1976b). In phytoplankton, algae, and microorganisms, mercury uptake is primarily a passive process that occurs by adsorption to the cell surface either through interaction with functional groups in the cell wall or through sorptive properties associated with the extracellular matrices (Darnell et al. 1986; Gadd 1988). Passive diffusion of lipid-soluble species (uncharged chloride complexes) is responsible for mercury uptake in a marine diatom (Mason et al. 1996). Uptake in phytoplankton and aquatic plants has been correlated with the concentration of mercury in the water (Windom and Kendall 1979; Lenka et al. 1990). Water is an important exposure pathway for mercury uptake by lower organisms and thus into the food web (Francesconi and Lenanton, 1992).

Dissolved mercury concentrations in water are typically very low; the major increase in mercury concentrations occurs between water and phytoplankton of about a factor of 105 to 106 (Mason et al., 1995b). In contrast to microorganisms, uptake is primarily an active process for fish and invertebrates, and is related to respiration rate and metabolic rate (Rodgers and Beamish, 1981). Uptake of methylmercuric chloride in water by different tissues of brook trout was found to be directly related to the water concentration of the mercury (McKim et al., 1976). BCFs are the concentration of mercury in tissue divided by the concentration in the exposure water. They have been calculated from laboratory experiments for many species of aquatic organisms to estimate uptake from water. However, BCFs have limited use for several reasons. First, BCFs reported in the literature most likely underestimate actual values because laboratory studies were done before the use of trace-metal free protocols and used higher water concentrations than found in the field (Zillioux *et al*., 1993). More recent BCF calculations for mercury have yielded values one to two orders of magnitude higher than previous estimates. Second, BCFs only reflect uptake of a contaminant from the water. Higher trophic species accumulate mercury primarily through the food web. Reported BCFs for mercury vary considerably due to differences between species, exposure concentration, and duration. Further, BCFs for the same species may be several orders of magnitude higher for methylmercury than for inorganic mercury. Brook trout exposed to varying concentrations of methylmercury for 28 to 38 weeks had bioconcentration factors ranging from 69,000 to 630,000 (McKim *et al*., 1976). The wide range of BCFs reported for brook trout are related to the tissue analyzed. Bioconcentration factors for muscle tissue in brook trout were higher at lower water concentrations of mercury. BCFs for other tissues remained the same when water methylmercury concentrations were varied.

Bioconcentration factors for inorganic mercury (mercuric chloride) in saltwater species were 129 for adult lobster (*Homarus americanus*), 1,000 for mussels, and 10,000 for oysters (*Crassostrea virginica*; Kopfler, 1974; Roesijadi *et al*. 1981). Bioaccumulation factors (BAFs) were calculated from field studies for yearling yellow perch from five freshwater lakes. These factors ranged from 106-107 for methylmercury and more than 104 for other mercury species (Bloom, 1992).

Sediment is an important exposure pathway for all forms of mercury to aquatic organisms. High concentrations of organic substances and reduced sulfur that complex free Hg [II] ions in sediment can reduce the availability of mercury to biota (Luoma, 1977; Rubinstein *et al*., 1983). Correlating mercury concentrations in sediment with concentrations in biota may be difficult, particularly for higher trophic- level species.

The bioavailability of total mercury to benthic invertebrates was reported to be inversely correlated to the organic content of the sediment (Langston 1982, 1986). Normalizing sediment mercury concentrations to percent organic matter improved the correlation between total mercury concentrations in sediment and invertebrate species (including gastropods, polychaetes, and deposit- and suspension-feeding bivalves) in a marine environment (Bryan and Langston, 1992). Good sediment tissue correlations for mercury have been found in amphipods from a freshwater lake (Becker *et al*., 1993). Breteler *et al*., (1981) studied mercury uptake by plants and invertebrates from several types of sediments in salt marsh environments. Concentrations of total mercury in mussels, fiddler crabs, and *Spartina alterniflora* increased as organic matter in sediments decreased.

Many investigators report no correlation between sediment and tissue concentrations of mercury for higher-trophic-level species (Nishimura and Kumagai, 1983; Jackson 1988;

Rada *et al*., 1989b; Lindqvist, 1991; Duckerschein *et al*., 1992). Organic carbon normalization of sediment concentrations did not improve the correlations for pike, a high trophic level species (Lindqvist, 1991). The difficulty in correlating mercury in sediment with mercury in organisms reflects the complexity of variables that affect both the methylation of mercury in surface sediments and the transfer of mercury between trophic levels. Since methylation occurs primarily in surface sediments, the physical factors that affect the rate of methylation (and demethylation) also affect the availability of mercury for uptake by organisms. Sediment total-mercury concentrations alone may not provide information on the exposure potential of resident organisms. Though sediment may be the ultimate source of mercury for many higher trophic species, the food web is the primary pathway to most organisms (Lindqvist, 1991; Bryan and Langston, 1992). Most of the differentiation between inorganic and methylmercury accumulation occurs during trophic transfer (Mason *et al*., 1995b) because of the differences in assimilation of the different mercury forms and how efficiently the different forms are transferred to predators. Mason *et al*., (1995b) detected assimilation efficiency four times greater for methylmercury compared to inorganic mercury from phytoplankton to zooplankton, and ten times greater between phytoplankton and planktivorous fish. The transfer efficiency of methylmercury over inorganic mercury in zooplankton was attributed to mercury partitioning in the algal cell. Methylmercury accumulated in the algal cytoplasm, which zooplankton digest, with 62% of the methylmercury transferred, while inorganic mercury was primarily bound to thiols in the algal cell membrane. Therefore, a smaller percentage (15%) of inorganic mercury was transferred to zooplankton. As methylmercury increases in prey items, the transfer efficiency also increases (Windom and Kendall, 1979). Since methylmercury concentrations are highest in fish, piscivorous fish will be exposed to higher

concentrations of methylmercury than fish that feed on invertebrates. For example, walleye accumulated mercury at a faster rate and at higher concentrations than pike from the same freshwater lake (Mathers and Johansen, 1985). A high proportion of the diet of walleye was smelt, the most contaminated prey item, whereas pike ate only a small proportion of this prey item. Dietary changes during life history development or due to season or habitat differences can change exposure. Dietary shifts in prey items of similar trophic levels but from different habitats, or dietary shifts due to a different size structure of prey, can also affect the mercury concentrations in top-level predators (Lindqvist, 1991).

The relative importance of dietary versus aqueous mercury uptake pathways is unclear. Probably less than 10% of the mercury in fish tissue residues is obtained by direct (gill) uptake from water (Francesconi and Lenanton, 1992; Spry and Wiener, 1991). Methylmercury concentrations used in laboratory studies of aqueous uptake are 1,000 to 10,000 times the ambient concentration of methylmercury in natural water (Spry and Wiener, 1991), thereby overestimating the significance of direct aqueous uptake. The proportion of mercury taken up from dietary sources versus water in invertebrates has not been estimated. Suspension-feeding bivalves may principally accumulate mercury by consuming algal cells (Riisgård and Hansen, 1990).

Although mercury correlations are complicated by the importance of the food chain exposure pathway, mercury concentrations in predators and prey have been correlated (e.g., Allard and Stokes, 1989; Lindqvist, 1991; Spry and Wiener, 1991). For example, mercury concentrations in small mouth bass from Ontario lakes were directly correlated with mercury in crayfish, which comprised 60% of their diet. Detritus can be a very important source of mercury, particularly in estuarine habitats. Organic detritus from *Spartina alterniflora* may contain 30 times more mercury than plankton. Organisms in

detritus-based food webs are thus exposed to higher mercury concentrations than are animals feeding on plankton (Lindberg and Harriss, 1974). Mercury associated with humic matter in lakes is fed upon by bacteria and zooplankton, which incorporate mercury into the detrital food web (Lindqvist, 1991). Mercury in the fecal matter of marine mammals can also be a significant source to other aquatic organisms near breeding colonies or haul-out areas (Eisler, 1987).

2.10 BIOLOGICAL FACTORS AFFECTING ACCUMULATION OF MERCURY

The primary biological factors governing the accumulation of mercury include age, weight, and diet. Differences in accumulation between the sexes have been attributed to differences in diet.

Numerous field studies have shown that the concentration of total mercury in fish positively correlates with length, age, and weight (Hall *et al*., 1976a, b; Huckabee *et al*., 1979; Rada *et al*., 1986; Møhlenberg and Riisgård 1988; Greib *et al*. 1990; Leah *et al*., 1992). However, total mercury concentrations may not always correlate with size due to differences associated with diet, residence time in a contaminated habitat, and type of mercury (Francesconi and Lenanton, 1992). The percentage of methylmercury increases with age in both fish and invertebrates (Møhlenberg and Riisgård, 1988; Riisgård and Hansen, 1990). In some species of fish and invertebrates, sex differences in mercury tissue concentrations have been reported. For example, total mercury concentrations in the muscle tissue of freshwater sunfish were greater in females than males at ages 2 to 3 (Nicoletto and Hendricks, 1987). This may be due to increased food demands for females related to reproduction. In contrast, there was little relationship between sex and bioaccumulation of mercury in three species of fish (roach, perch, and pike) collected from Swedish lakes (Lindqvist, 1991). Bloom (1992) did not find a

relationship between lipid content and methylmercury concentrations in a variety of fresh- and saltwater fish.

Cockles (*Cardium edule*) from a polluted estuary were found to have a positive linear correlation between their age and the percentage of organic mercury in their tissues (Møhlenberg and Riisgård, 1988). Organic mercury comprised 30% of the total mercury in two-year old cockles; 60% in three-year olds; and 90% in four-year olds. This relationship was attributed to the rapid loss of inorganic mercury and continued uptake of organic mercury over time. However, the correlation was not as strong when weight was used instead of age due to variations in growth rate at different locations. Total mercury concentrations in mussels were found to be higher in 27 mm than 31-mm sized individuals (Riisgård and Hansen, 1990). This difference was perhaps due to a decrease in both weight-specific filtration rate and surface area-to-volume ratio in larger mussels (Fowler *et al*., 1978; Riisgård *et al*. 1985).

Total mercury concentrations may (Allard and Stokes, 1989) or may not (Rada *et al*, 1986) correlate with weight or age in crustaceans. Concentrations of mercury in male and female emergent mayflies (*Hexagenia bilineata*) in the upper Mississippi River differed. The authors recommend sampling male and female mayflies separately (Dukerschein *et al*.,1992).

Mercury concentrations (both organic and inorganic) are positively correlated with body length in marine mammals (Arima and Nagakura, 1979; Wagemann *et al*. 1983; Joiris *et al*., 1991). Hansen et al. (1990) found a highly significant correlation between age and tissue content of mercury in whales from West Greenland. This correlation has been used to separate immature specimens from adults. Joiris *et al*. (1991) found that

the concentration of methylmercury in muscle and liver tissue in harbor porpoises did not increase with increasing length as strongly as did total mercury.

Leonzio *et al*. (1992) suggest that the elevated concentrations of inorganic mercury measured in mammals, as compared to fish, may be related to differences in respiratory systems. In contrast to fish, where the gills allow contaminants to be lost to the environment because blood flow has contact with the water, the mammalian respiratory system does not have a similar exchange. Mammals have developed different defense mechanisms. For example, selenium combines with mercury to form the non-toxic compound tiemannite that is stored within cells. The processes of intracellular storage tend to increase concentrations of the metal in certain organs while reducing the toxicity. In marine mammals, intracellular storage of mercury occurs as complexes of both selenium and metallothioneins (MTs).

2.11 OTHER FACTORS AFFECTING ACCUMULATION OF MERCURY

Temperature and season influence the availability and accumulation of mercury in addition to the factors already discussed. Changes in temperature can affect mercury concentrations in organisms either directly by affecting metabolic rate and thereby exposure, or indirectly by influencing the methylation of mercury and therefore enhancing availability. Rates of methyl- or inorganic mercury uptake increases with increase in aqueous concentrations and/or increase in temperature in the water for some species (e.g., phytoplankton, gastropods, fish; Windom and Kendall, 1979; Rodgers and Beamish, 1981; Tessier *et al*., 1994). A rise in temperature (and a corresponding rise in respiratory volume) can increase the rate of uptake via the gills (EPA, 1985). Total concentrations of mercury in killifish from an estuarine wetland were five times higher in spring and summer than in other seasons (Weis *et al*., 1986), presumably due to higher methylation rates in summer. Zooplankton mercury concentrations peaked in

June in Swedish lakes and fish tissue levels varied by a factor of two, reaching a maximum in spring (Lindqvist, 1991). Mercury content of mussels from the Gulf of St. Lawrence estuary varied seasonally by a factor of two (Cossa and Rondeau, 1985).

The relationship of pH, conductivity, and salinity to mercury accumulation is not well understood. Elevated mercury concentrations have frequently been found in piscivorous fish in poorly buffered (alkalinity < 55 μeq/l and calcium < 2 mg/l), low pH lakes (pH 6.0-6.5) in areas removed from industrial inputs of mercury (Rada *et al*,. 1989a; Winfrey and Rudd, 1990; Spry and Wiener, 1991). Total mercury concentrations in yellow perch were inversely correlated with pH in ten (10) Wisconsin Lakes (Cope *et al*., 1990). Mercury concentrations in zooplankton in Swedish lakes were correlated with pH but the relative importance of this correlation changed over time (Lindqvist, 1991).

In freshwater lakes removed from direct sources of mercury, conductivity explained 54% of the variability in mercury concentrations in crayfish (Allard and Stokes, 1989). Conductivity was also highly correlated with calcium, magnesium, alkalinity, pH, and sodium. This correlation suggests that the buffering capacity of the lake was an important influence on crayfish accumulation of mercury. Low calcium ion concentrations enhanced the efficiency of methylmercury uptake across the gills of rainbow trout (Rogers and Beamish, 1983).

2.12 TOXICITY OF MERCURY

The toxicity of mercury to aquatic organisms is affected by both abiotic and biotic factors including the form of mercury (inorganic versus organic), environmental conditions (e.g., temperature, salinity, and pH), the sensitivity of individual species and life history stages, and the tolerance of individual organisms. Toxicological effects

include neurological damage, reproductive impairment, growth inhibition, developmental abnormalities, and altered behavioral responses. Wiener and Spry (1996) concluded that neurotoxicity seems to be the most probable chronic response of wild adult fishes to methylmercury exposure, based on observed effects such as uncoordination, inability to feed, diminished responsiveness, abnormal movements, lethargy, and brain lesions. In laboratory studies, reproductive endpoints are generally more sensitive than growth or survival, with embryos and the early developmental stages being the most sensitive (Hansen, 1989). Impaired reproduction in sensitive aquatic organisms has been shown to occur at aqueous concentrations of mercury between 0.03 and 1.6 µg/l (Eisler, 1987). Long-term mercury exposure to adult fish also has been shown to result in retarded growth of offspring (Snarski and Olson, 1982) and teratogenic effects (Weis *et al.* 1981). Chronic exposure to low concentrations of mercury may result in populations that become tolerant to the toxic effects of mercury contamination (Weis and Weis, 1989).

The toxic concentration of mercury compounds can vary by an order of magnitude or more depending on the exposure condition. For example, toxicity is greater at elevated temperatures (Armstrong, 1979), at lower oxygen content (Sloof *et al.*, 1991), and at reduced salinities in marine environments (McKenney and Costlow 1981).

Site-specific factors (such as TOC) affect the bioavailability and toxicity of mercury contaminated sediment (Langston, 1990). Even though correlations exist between toxicological observations and sediment pollution gradients, Langston (1990) recommends collecting site-specific data because biological responses cannot always be satisfactorily predicted from chemical data or modeling results. The sensitivity of aquatic organisms to either inorganic or methylmercury varies considerably between species — more than the difference in sensitivity of a particular species to various

mercury compounds (EPA, 1985). Methylmercury is more acutely toxic to aquatic organisms than inorganic mercury, but the range among different species in sensitivity to either compound is quite large. For example, the concentration of inorganic mercury inducing acute toxicity was observed to range over almost three orders of magnitude from 0.1 μg/l to more than 200 μg/l when results from tests with different species were compared (Eisler, 1987). Tests on the same freshwater species with both inorganic and methylmercury showed that methylmercury was more than 30 times more acutely toxic than inorganic mercury (EPA, 1985).

The general mechanism of action for toxic effects for inorganic mercury which has the form Hg(II), the divalent mercury cation, is believed to be the high affinity for thiol or sulfhydryl groups of proteins (Clarkson 1972; Hughes 1957, Passow *et al*., 1961) resulting in altered protein production or synthesis (Syversen, 1977). Methylmercury is lipid soluble, allowing rapid penetration of the blood-brain barrier (Feltier *et al*. 1972, Giblin and Massaro, 1973; McKim *et al*. 1976; Olson *et al*., 1978; Beijer and Jernelov, 1979). Injury to the central nervous system results from accumulation of methylmercury in the cerebellum and cerebral cortex where it binds tightly to sulfhydryl groups resulting in pathological changes (Sastry and Sharma, 1980). Inside the cell, methylmercury inhibits protein synthesis/RNA synthesis (Yoshino *et al*. 1966; Chang *et al*. 1972). Zillioux *et al*. (1993) suggest that, prior to the mid-1980s; few data are available on the biological effects of mercury at environmental concentrations because laboratory studies used exposure concentrations that were much higher than actual concentrations in the field. This was in part due to contamination during sample collection and analysis. Improvements in trace-metal-free clean protocols during sample collection, handling, and processing as well as lower analytical detection limits have resulted in lower environmental concentrations of mercury and lower

concentrations reported to elicit adverse effects. Although pre-1980 data are useful in identifying modes of effect and relative toxicity of the various mercury compounds, these data should be used with caution.

2.13 TOXICITY OF MERCURY IN WATER

Nearly all of the studies evaluating the toxicity of mercury compounds where the route of exposure is through water have been conducted under laboratory conditions. Due to the nature of laboratory studies and differences in experimental design and technique, a wide range of toxic concentrations have been reported for a given species (Table 1). For example, toxicity tests using flow-through systems generally show higher toxicity at lower concentrations than static-renewal systems using the same (nominal) concentrations and the same species. This difference is probably due to loss of mercury from the test container in the static-renewal tests (Birge *et al*. 1979; Biesinger *et al*. 1982; WHO 1989).

Fish tend to be more sensitive to sublethal effects from chronic exposure to both inorganic and organic mercury than invertebrates, but fish are less sensitive to acute effects (EPA 1985; Hansen, 1989). The early life stages of fish are generally the most sensitive to mercury. Birge *et al*. (1979) conducted several tests designed to evaluate embryo survival, hatching success, teratogenic effects, and the effects of mercury on six species of freshwater fish. The sensitivity of the embryo-larval stage for various species was correlated with the length of time for eggs to develop and hatch and the duration of exposure. Trout eggs treated in a flow-through system experienced approximately 40% mortality after a five-day exposure and 100% mortality after an eight-day exposure to an average mercury concentration of 0.12 µg/l. Birge *et al*. (1979) also evaluated the long-term effects of mercury exposure on fish reproduction by conducting chronic bioassays with rainbow trout. Their results suggest that exposure of adult fish to

mercury can have significant adverse effects on their offspring; with the effects enhanced if the embryos are also reared in a mercury-contaminated environment. Their data show a dose-dependent response in both bioaccumulation of mercury in gonadal tissues and toxic effects on embryos. Short-term exposures of embryos to high concentrations of mercury can also elicit significant adverse effects (Sharp and Neff, 1980) and such exposures should be taken into account in the evaluation of potential long-term impacts to receiving environments. Although time-integrated concentrations may be within accepted guidelines, a short-term exposure to an elevated mercury concentration could result in inhibition of hatching, teratogenic development, and possible population effects.

Low concentrations of mercury in freshwater reportedly result in olfactory and chemoreceptor impairment in salmonids and other fish, which may interfere with normal migratory behavior (Hara *et al*., 1976; Rehnberg and Schreck, 1986). For example, Hara *et al*. (1976) reported that rainbow trout exposed to inorganic mercury concentrations as low as 0.1 mg/l for two hours showed reduced olfactory responses. Further physiological and behavioral studies by Rehnberg and Schreck (1986) showed that mercury exposure reduced the ability of coho salmon to detect natural odors and disrupted simple upstream movement in laboratory experiments.

Weis and Weis (1989) suggest that prior exposure to mercury may produce populations that are more tolerant to the toxic effects of mercury contamination. Differences in tolerance to the effects of methylmercury were observed between organisms from mercury-contaminated and clean environments. Eggs collected from killifish in a contaminated area were mostly resistant to the teratogenic effects of methylmercury, while eggs of fish from the clean area showed a range of sensitivity. The susceptible eggs from the clean area also accumulated higher levels of mercury than did the eggs

from the contaminated area (Weis *et al*. 1981; 1982). Offspring from fish previously exposed to mercury contamination were more tolerant to environmental mercury concentrations than offspring from clean environments (Weis and Weis, 1984).

The situation is complicated by the fact that some fish that build up a tolerance to low concentrations of mercury can also detoxify the free metal within cells via the production of metallothioneins (MTs) and other metal-binding proteins. Brown *et al*. (1983) proposed that toxic effects occur as the binding capacity of MT becomes saturated, due to the interaction of excess free metal in the cell with the enzyme pool.

Calabrese *et al*. (1977) suggest that marine bivalves' embryos are more sensitive than the larvae in their susceptibility to mercury. They further indicated that growth of fully-developed larvae may be retarded at concentrations too low to elicit significant mortality, thus prolonging the pelagic stages and increasing the risk of predation, disease, and dispersion. Several endpoints have been used to measure the effect of mercury exposure on bivalves, including biomarkers. The prophyrin precursor α-aminolevulinic acid (ALA) may be useful as a biomarker of mercury exposure in bivalves (Brock, 1993).

The effects of salinity on the toxicity of mercury have been demonstrated in a study conducted with the megalopae of the blue crab, *Callinectes sapidus* (McKenney and Costlow, 1981). Their results indicated that as salinity was reduced below 20 parts per-thousand, less mercury was required to produce equivalent toxicity among megalopae. This is significant for blue crab and other estuarine species which inhabit, migrate through, and use areas of lower salinity for foraging, spawning, and nursery grounds. Their data imply that the impact to a given population of fish or invertebrates is highly dependent on the life stage and surrounding environmental conditions. The significance

of experimental design and exposure period on evaluating the toxicity of mercury was demonstrated in a series of studies conducted by Biesinger *et al*. (1982). In acute flow-through toxicity tests with *Daphnia magna*, methylmercuric chloride was about 10 times more toxic than inorganic mercury, but only about 4 times as toxic under static-renewal conditions. In the static renewal tests with methylmercury, it was discovered that about 90 percent of the mercury had been converted to inorganic mercury during the testing period. In chronic flow-through toxicity tests with *Daphnia magna*, methylmercuric chloride was about 30 times more toxic than inorganic mercury.

Chronic toxicity (as demonstrated by reduced population growth) in a marine diatom (*Thalassiosira weissflogii*), exposed to inorganic mercury, methylmercury, dimethylmercury, and elemental mercury, was related to the aqueous concentration of a single mercury species, (the chloride species $HgCl_2$ or CH_3HgCl), not to the total mercury or free mercury ion concentration (Mason *et al.*, 1996). Approximately the same concentration of CH_3HgCl and $HgCl_2$ reduced growth in the diatom by 50 percent. Mason *et al*. (1996) explain the apparently higher toxicity of methylmercury compared to inorganic mercury (expressed as a total concentration of all inorganic forms) observed by numerous authors as a result of the low percentage of the chloride form ($HgCl_2$) in the inorganic mercury fraction of seawater (3.3 %) compared to the high percentage of the methyl mercuric chloride species (CH_3HgCl), which forms 100% of the methylmercury in seawater. Elemental and dimethylmercury, even though more hydrophobic than $HgCl_2$ and CH_3HgCl respectively, were neither accumulated nor toxic to the diatom. The hypothesis by Fisher *et al.* (1984), that the metals that are most bioaccumulated by phytoplankton are the most toxic, may also be true about individual mercury species.

2.14 TOXICITY OF MERCURY IN SEDIMENT

The complex behavior of mercury in the environment makes it difficult to predict toxic effects based on bulk sediment total mercury concentrations. All the available data for effects of mercury in sediment are based on measurements of either inorganic mercury or total mercury. The concentrations of mercury in sediment associated with toxicity are primarily derived from field studies, in contrast to the large number of laboratory toxicity tests for water exposure. The results from only two spiked sediment bioassays are available. Birge *et al*. (1979) reported reduced survival (70% and 45%) of rainbow trout eggs exposed to sediment contaminated with inorganic mercury (mercuric chloride) for 20 days at concentrations of 1.05 and 0.18 mg/kg respectively. Swartz *et al*. (1988) reported an LC50 of 13.1 mg/kg for the marine amphipod (*Rhepoxynius sp*.).

Considerable data are available, however, from field-collected samples that include both measurements of mercury concentrations in sediment and adverse biological effects. Long and MacDonald (1992) reviewed the concentrations of mercury that were associated with measures of adverse biological effects in 169 studies that included both marine and estuarine systems. Data from those studies were used to calculate Effects Range-Low (ERL) and Effects Range-Median (ERM) concentrations of 0.15 mg/kg and 0.71 mg/kg, respectively. The ERL and ERM concentrations are the lower (10 percentile) and median (50 percentile) of the study concentrations associated with toxic effects. Of the total number of studies in the data set, 8.3% had biological effects below the ERL (Long *et al*., 1994). The incidence of effects between the ERL and ERM concentrations was 23.5%. The incidence of effects above the ERM concentration was 42.3% for mercury, while for other metals (e.g., chromium, copper, lead and silver) the incidence of adverse effects above the ERM was in the range of 75%. The low accuracy of the ERL and ERM mercury guidelines in predicting adverse effects compared to

these other metals highlights the need for site-specific effects-based data for determining sediment mercury concentrations that are a threat to aquatic biota. The Washington State Department of Ecology uses Apparent Effects Threshold (AET) concentrations as the basis for sediment criteria for mercury. The AETs, based on laboratory bioassays and benthic community studies, represent the concentration of a contaminant above which significant adverse effects were always observed for a specific biological indicator (PTI, 1988). The AETs for mercury were empirically derived from studies conducted with contaminated and reference marine sediments collected from Puget Sound. AETs for total mercury are 0.41 mg/kg for the Microtox™ bacterial luminescence bioassay, 0.59 mg/kg for oyster larvae abnormality, 2.1 mg/kg for amphipod (*Rhepoxynius abronius*) lethality, and 2.1 mg/kg for reductions in the abundance of major taxa of benthic macro invertebrates (Beckvar *et al*. 1996).

A laboratory study by McGreer (1979) demonstrated that clams, *Macoma balthica*, avoided burrowing into field-collected sediment containing a suite of metals. The concentrations of both cadmium (1.4 ppm) and mercury (0.46 ppm) best explained the behavioral responses. Avoidance of mercury-contaminated habitats by aquatic species may be important ecologically. Inhibited burrowing response, relocation, and lack of larval settlement can decrease population sizes and reduce overall community composition. Species that avoid contact with contaminated sediment and do not burrow into the sediments are more vulnerable to predators and adverse environmental conditions (e.g., temperature extremes, wave action, and contaminants in the water column) (Beckvar *et al*., 1996).

2.15 TOXICITY ASSOCIATED WITH MERCURY IN TISSUES

It is important to stress that both the tissue concentration and the exposure time and route (i.e. water, food, and maternal transfer) are critical factors in producing toxic symptoms in aquatic receptors (Beckvar *et al*., 1996).

According to Wiener and Spry (1996), mercury transferred from the female to the eggs during oogenesis may pose a greater risk to embryos than exposure to mercury in the water column. For rainbow trout, mercury residues in ovaries of 0.5 mg/kg were associated with a significant reduction in larval survival and abnormal development (Birge *et al*., 1979). Whitney (1991) reported that hatching success and embryonic survival in walleye were inversely correlated with mercury concentrations in the egg (range 0.002 to 0.058 mg/kg). However, only one of 12 samples had hatching success or embryonic survival less than 90%, and there was no apparent dose-response relationship.

Mercury concentrations in brain tissue associated with lethal effects appear to show less variation than that of other tissues (e.g., muscle, whole body). For example, mercury concentrations in most types of tissues of brook trout killed by exposure to 2.9 µg/l of mercury in the water column varied among individuals, whereas concentrations in the brain showed little variation (McKim *et al*., 1976). These results are consistent with the hypothesis that the central nervous system, rather than muscle tissue or other organs, is the site of the most harmful toxic action in fish exposed to mercury (Wiener and Spry, 1996). In their review of the literature, Wiener and Spry (1996) concluded that mercury concentrations of 7 mg/kg or greater in fish brain probably cause severe, potentially lethal effects. In sensitive species such as the walleye, brain tissue concentrations of 3 mg/kg or greater probably indicate significant toxic effects. Based on a review of the literature, Niimi and Kissoon (1994) suggest that a total mercury body burden of 1-5

mg/kg represents a threshold concentration for chronic adverse effects in aquatic organisms. Wiener and Spry (1996) reviewed the literature and provided guidance for interpreting mercury residues in the axial muscle tissue in adult fish associated with toxicity; both field and laboratory studies indicate that residues of 6 to 20 mg/kg are toxic. Whole body mercury concentrations of about 5 mg/kg in brook trout and 10 mg/kg in rainbow trout were associated with sublethal and lethal effects. Both of these papers are recent examples of attempts to identify a threshold of mercury in tissue that is associated with adverse effects. The "thresholds" presented in these papers are based on effects in adult fish and probably do not represent a truly protective level for all species and life stages, including maternal transfer. We begin to become concerned about reproductive or early life stage effects when total Hg in whole bodies of fish are between 0.5 and 1.0 ppm.

2.16 TOXICITY OF MERCURY TO BIRDS

Mercury toxicity to birds varies with the form of the element, dose, and route of administration, species, sex, age, and physiological condition (Fimreite, 1979). For example, in northern bobwhite chicks fed diets containing methylmercury chloride, mortality was significantly lower when the solvent was acetone than when it was another carrier such as propylene glycol or corn oil (Spann *et al*., 1986). In addition, organomercury compounds interact with elevated temperatures and pesticides, such as DDE and parathion, to produce additive or more-than-additive toxicity, and with selenium to produce less-than-additive toxicity (*Fimreite,* 1979). Acute oral toxicities of various mercury formulations ranged between 2.2 and about 31.0 mg/kg body weight for most avian species tested. Similar data for other routes of administration were 4.0 to 40.0 mg/kg for diet and 8.0 to 15.0 mg/kg body weight for intramuscular injection.

Residues of mercury in experimentally poisoned passerine birds usually exceeded 20 mg/kg fresh weight, and were similar to concentrations reported in wild birds that died of mercury poisoning (Finley *et al*., 1979). Mercury levels in tissues of poisoned wild birds were highest (45 to 126 mg/kg fresh weight) in red-winged blackbirds (*Agelaius phoeniceus*), intermediate in starlings (*Sturnus vulgaris*) and cowbirds (*Molothrus ater*), and lowest (21 to 54) in grackles (*Quiscalus quiscula*). In general, Hg residues were highest in the brain, followed by the liver, kidney, muscle, and carcass. Some avian species are more sensitive than passerines (Solonen and Lodenius, 1984): liver residues (in mg Hg/kg dry weight) in birds experimentally killed by methylmercury ranged from 17 in red-tailed hawks (*Buto jamaicensis*) to 70 in jackdaws (*Corvus monedula*); and values were intermediate in ring-necked pheasants, kestrels (*Falco tinnunculus*), and magpies (*Pica pica*). Experimentally poisoned grey herons (*Ardea cinerea*) seemed to be unusually resistant to Hg; lethal doses produced residues of 415 to 752 mg Hg/kg dry weight of liver (Van der Molen *et al*., 1982). However, levels of this magnitude were frequently encountered in livers from grey herons collected during a massive die-off in the Netherlands during a cold spell in 1976; the interaction effects of cold stress, mercury loading and poor physical condition of the herons are unknown (*Ardea cinerea*) in the Netherlands (Van der Molen *et al*., 1982).

Mercury is a known mutagen, teratogen, and carcinogen. At comparatively low concentrations in birds and mammals, it adversely affects reproduction, growth and development, behavior, blood and serum chemistry, motor coordination, vision, hearing, histology, and metabolism. It has a high potential for bioaccumulation and biomagnification and is slow to depurate. Organomercury compounds were more effective in producing adverse effects than were inorganic Hg compounds; however, effects were significantly enhanced or ameliorated by numerous biotic and

nonbiological modifiers. For sensitive aquatic species, adverse effects were observed at water concentrations of 0.03 to 0.1 µg Hg/l. For sensitive species of birds, harmful levels were 640 µg Hg/kg body weight daily, or 50 to 500 µg Hg/kg in the diet; for sensitive mammals, these levels were 250 µg Hg/kg body weight daily, or 1,100 µg Hg/kg diet.

Sublethal effects of mercury on birds, administered by a variety of routes, included adverse effects on growth, development, reproduction, blood and tissue chemistry, metabolism, and behavior; histopathology and bioaccumulation were also noted (Eisler, 1987).

The dietary route of administration is the most extensively studied pathway of avian Hg intake. Domestic chickens fed diets containing as little as 50 µg/kg of mercury, as methylmercury, contained elevated total Hg (2.0 mg/kg fresh weight) residues in liver and kidney after 28 weeks; at 150 µg/kg, residues ranged from 1.3 to 3.7 mg/kg in heart, muscle, brain, kidney, and liver, in that general order; at 450 ug/kg in diets, residues in edible chicken tissues (3.3 to 8.2 mg/kg) were considered hazardous to human consumers, although no overt signs of mercury toxicosis were observed in the chickens (March *et al*., 1983). High inorganic mercury levels (500 mg/l) in drinking water of chickens decreased growth rate and food and water consumption, and elevated hemoglobin, hematocrit, and erythrocyte content within 3 days (Grissom and Thaxton, 1985). The dietary concentration of 0.5 mg Hg/kg dry weight (equivalent to about 0.1 mg/kg fresh weight) in the form of methylmercury was fed to three generations of mallards (Heinz, 1979). Females laid a greater percentage of their eggs outside nest boxes than did controls, and also laid fewer eggs and produced fewer ducklings. Ducklings from parents fed methylmercury were less responsive than controls to tape-recorded maternal calls, but were hyper responsive to a fright stimulus in avoidance

tests. The tissues and eggs of ducks and other species of birds collected in the wild have sometimes contained levels of mercury equal to, or far exceeding, those associated with reproductive and behavioral deficiencies in domestic mallards (e.g., 9 to 11 mg/kg in feathers; >2.0 mg/kg in other tissues); therefore, it is possible that reproduction and behavior of wild birds have been modified by methylmercury contamination (Heinz, 1979). Tissue mercury residues of wild-strain mallards and game-farm mallards were not significantly different after the birds were fed diets containing 0.5 mg Hg/kg as methylmercury for extended periods--indicating that game-farm mallards are suitable substitutes for wild mallards in toxicological evaluations (Heinz, 1980). Dietary concentrations of 1.1 mg total Hg/kg have been associated with kidney lesions in juvenile starlings (*Sturnus vulgaris*) and with elevated residues in the liver (6.5 mg/kg dry weight and kidney (36.3 mg/kg), after exposure for 8 weeks (Nicholson and Osborn, 1984). In American black ducks (*Anas rubripes*) fed diets containing 3.0 mg Hg/kg as methylmercury for 28 weeks, reproduction was significantly inhibited; tissue residues were elevated in kidney (16.0 mg/kg fresh weight) and liver (23.0 mg/kg); and brain lesions characteristic of mercury poisoning were present (Finley and Stendell, 1978). Japanese quail (*Coturnix japonica*) fed diets containing 8 mg Hg/kg of inorganic mercury for 3 weeks had depressed gonad weights; those fed 3 mg/kg inorganic mercury or 1 mg/kg methylmercury for 9 weeks showed alterations in brain and plasma enzyme activities (Hill and Soares, 1984). Grossly elevated tissue residues of 400 mg/kg in feathers and 17 to 130 mg/kg in other tissues were measured in gray partridge (*Perdix perdix*) after dietary exposure of 20 to 25 mg total Hg/kg for 4 weeks (McEwen *et al*., 1973).

Mercury exposure by immersion and oral administration have caused reproductive and behavorial modifications. Brief immersion of mallard eggs in solutions of

methylmercury resulted in a significant incidence of skeletal embryonic aberrations at dosages of 1.0 µg Hg/egg, and higher; no increases in embryonic malformations were noted at 0.3 µg Hg/egg (Hoffman and Moore, 1979). Reduced reproductive ability was noted in grey pheasants ingesting 640 µg Hg (as organomercury)/kg body weight daily for 30 days (McEwen *et al*., 1973); similar results were observed in ring-necked pheasants (Spann et al. 1972; Mullins *et al*., 1977). Behavioral alterations were noted in pigeons (*Columba livia*) given 3,000 µg inorganic Hg/kg body daily for 17 days (Leander *et al*., 1977) or 1,000 µg/kg body weight of methylmercury for 5 weeks (Evans *et al*., 1982). Observed behavioral changes in posture and motor coordination of pigeons were permanent after the brain accumulated >12,000 µg Hg/kg fresh weight, and were similar to the "spastic paralysis" observed in wild crows during the Minamata, Japan, outbreak of the 1950's, although both species survived for years with these signs (Evans *et al*., 1982).

Mercury residues of 790 to 2000 µg/kg in egg and 5000 to 40000 µg/kg in feathers are linked to impaired reproduction in various bird species (Spann *et al*., 1972; NAS, 1978; Heinz, 1979; Fimreite, 1979; Solonen and Lodenius, 1984). Residues in eggs of 1,300 to 2,000 µg Hg/kg fresh weight were associated with reduced hatching success in white-tailed sea-eagles (*Haliaeetus albicilla*), the common loon (*Gavia immer*), and in several seed-eating species (Fimreite,1979); this range was 900 to 3,100 µg/kg for ring-necked pheasant (Spann *et al*., 1972), and 790 to 860 ug/kg for mallards (Heinz, 1979). Residues of 5,000 to 11,000 µg Hg/kg in feathers of various species of birds have been associated with reduced hatch of eggs and with sterility (NAS, 1978). Sterility was observed in the Finnish sparrow hawk (*Accipiter nisus*) at mercury concentrations of 40000 µg/kg in feathers (Solonen and Lodenius, 1984). Chicks of the common tern (*Sterna hirundo*) from a colony in Long Island, New York, with abnormal feather loss,

had significantly elevated mercury levels in blood and liver (Gochfeld, 1980); however, the linkage of feather loss to mercury contamination requires further examination.

Interaction effects of mercury with other contaminants, such as herbicides and pesticides, could intensify hazards to avian populations (Mullins *et al*., 1977). For example, a striking parallel exists between levels of Hg and of DDT and its metabolites in birds of prey, suggesting the existence of common ecotoxicological mechanisms (Delbeke *et al*., 1984; Wiemeyer *et al*., 1984).

2.17 TOXICITY OF MERCURY TO MAMMALS

Methylmercury affects the central nervous system in man especially the sensory, visual, and auditory areas concerned with coordination; the most severe effects lead to widespread brain damage, resulting in mental derangement, coma, and death (Clarkson and Marsh, 1982). In mule deer (*Odocoileus hemionus hemionus*), after acute oral Hg poisoning was induced experimentally, additional signs included belching, bloody diarrhea, piloerection (hair more erect than usual), and loss of appetite (Hudson *et al*., 1984). The kidney is the probable critical organ in adult mammals due to the rapid degradation of phenylmercurials and methoxyethylmercurials to inorganic Hg compounds and subsequent translocation to the kidney (Suzuki, 1979), whereas in the fetus the brain is the principal target (Khera, 1979). Most human poisonings were associated with organomercury compounds used in agriculture as fungicides to protect cereal seed grain (Elhassani, 1983); judging from anecdotal evidence, many wildlife species may have been similarly afflicted. Organomercury compounds, especially methylmercury, were the most toxic mercury species tested. Among sensitive species of mammals, death occurred at daily organomercury concentrations of 0.1 to 0.5 mg/kg body weight, or 1.0 to 5.0 mg/kg in the diet (Eisler, 1987). Larger animals such as mule deer and harp seals appear to be more resistant to Hg than smaller mammals such as

mink, cats, dogs, pigs, monkeys, and river otters; the reasons for this difference are unknown, but may be related to differences in metabolism and detoxication rates. Tissue residues in fatally poisoned mammals (in mg Hg/kg fresh weight) were 6.0 in brain, 10.0 to 55.6 in liver, 17.0 in whole body, about 30.0 in blood, and 37.7 in kidney (Eisler, 1987).

Mercury has no known physiological function (EPA 1985). In humans and other mammals, it causes teratogenic, mutagenic, and carcinogenic effects; the fetus is the most sensitive life stage (NAS, 1978; Chang, 1979; Khera, 1979; EPA, 1980, 1985; Elhassani, 1983; Greener and Kochen, 1983; Clarkson *et al*., 1984). Methylmercury irreversibly destroys the neurons of the central nervous system. Frequently, a substantial latent period intervenes between the cessation of exposure to Hg and the onset of signs and symptoms; this interval is usually measured in weeks or months, but sometimes in years (Clarkson *et al*., 1984). At high sublethal doses in man, mercury causes cerebral palsy, gross motor and mental impairment, speech disturbances, blindness, deafness, microcephaly, intestinal disturbances, tremors, and tissue pathology (Chang, 1979; EPA, 1980, 1985; Elhassani, 1983; Clarkson *et al*., 1984). Pathological and other effects of Hg may vary from organ to organ, depending on factors such as the effective toxic dose in the organ, the compound involved and its metabolism within the organ, the duration of exposure, and the other contaminants to which the animal is concurrently exposed (Chang, 1979). Many compounds especially salts of selenium protect humans and other animals against mercury toxicity, although their mode of action is not clear (NAS, 1978; Chang 1979; EPA, 1980, 1985; Eisler, 1985).

Adverse effects of organomercury compounds to selected species of mammals have been recorded at administered doses of 0.25 mg Hg/kg body weight daily, dietary levels of 1.1 mg/kg, and blood Hg levels of 1.2 mg/l (Eisler, 1987).

Mercury transfer and biomagnification through mammalian food chains is well documented (Galster, 1976; NAS, 1978; Eaton *et al*., 1980; Eisler, 1981; Huckabee *et al*., 1981; Sheffy and St. Amant, 1982; Kucera, 1983; Clarkson *et al*., 1984; Wren 1986), but considerable variation exists. Among terrestrial mammals, for example, herbivores such as mule deer, moose (*Alces alces*), caribou (*Rangifer tarandus*), and various species of rabbits usually contained less than 1.0 mg Hg/kg fresh weight in liver and kidney, but carnivores such as the marten (*Martes martes*), polecat (*Mustela putorius*), and red fox (*Vulpes vulpes*) frequently contained more than 30 mg/kg (NAS, 1978). The usually higher mercury concentrations in fish-eating furbearers than in herbivorous species seemed to reflect the amounts of fish and other aquatic organisms in the diet. In river otter and mink from the Wisconsin River drainage system, Hg levels paralleled those recorded in fish, crayfish, and bottom sediments at that location. Highest Hg levels in all samples were recorded about 30 km downstream from an area that supported 16 pulp and paper mills and a chloralkali plant; residues were highest in the fur, followed by the liver, kidney, muscle, and brain (Sheffy and St. Amant, 1982).

In marine mammals, more than 90 % of the mercury content is inorganic; however, enough methylmercury occurs in selected tissues to result in the accumulation of high tissue concentrations of methylmercury in humans and wildlife consuming such meat (Clarkson *et al*., 1984). The liver of the ringed seal (*Phoca hispida*) normally contains 27,000 to 187,000 µg Hg/kg fresh weight, and is a traditional and common food of the coastal Inuit people (Eaton *et al*., 1980). Although levels of Hg in hair (10900 µg/kg) and blood (3µg/l) of Inuits were grossly elevated, no symptoms of Hg poisoning were

evident in the coastal Inuits. Similar high concentrations have been reported for Alaskan Eskimo mothers who, during pregnancy, ate seal oil twice a day, and seal-meat or fish from the Yukon-Kuskokwim Coast every day (Galster, 1976). Despite the extremely high total Hg content of seal liver, only the small organomercury component was absorbed and appeared in the tissues. Cats fed a diet of seal liver (26000 µg Hg/kg fresh weight) for 90 days showed no neurologic or histopathologic signs (Eaton *et al*., 1980). It seems that the toxic potential of seal liver in terms of accumulated tissue levels in cats (up to 862 µg total Hg/l blood, and 7,600 µg total Hg/kg hairs) is better indicated by the organomercury fraction in seal liver than by the concentration of total Hg (Eaton *et al*., 1980).

2.18 MERCURY TOXICITY TO AQUATIC ORGANISMS

Mercury at comparatively low concentrations adversely affects the reproduction, growth, behavior, metabolism, blood chemistry, osmoregulation and oxygen exchange of marine and freshwater organisms. In general, the accumulation of mercury by aquatic biota is rapid, and depuration is slow. It is emphasized that organomercury compounds, especially methylmercury, were significantly more effective than inorganic mercury compounds in producing adverse effects and accumulations.

Reproduction was inhibited among sensitive species of aquatic organisms at water concentrations of 0.03 to 1.6 µg Hg/l. In the planarian (Best *et al*., 1981); in the slipper limpet (*Crepidula fornicata*), spawning was delayed and fecundity was decreased at 0.25 µg Hg^{2+}/l (Thain, 1984); in the zebrafish (*Brachydanio rerio*), hatching success was reduced at 0.1 ug Hg^{2+}/l and egg deposition was reduced at 0.8 µg/l (Armstrong, 1979); fathead minnows (*Pimephales promelas*) exposed to 0.12 ug methylmercury/l for 3 months failed to reproduce (Birge et al. 1979); the leopard frog (*Rana pipiens*) did not metamorphose during exposure to 1.0 ug methylmercury/l for 4 months (EPA,

1980); and in the mysid shrimp (*Mysidopsis bahia*), the abortion rate increased and population size decreased after lifetime (i.e. 28 days) exposure to 1.6 µg/l of mercury as mercuric chloride (Gentile *et al*., 1983). For sensitive marine invertebrates such as hydroids, protozoans, and mysid shrimp, reproduction was inhibited at concentrations between 1.1 and 2.5 µg Hg^{2+}/l; this range was 5 to 71µg/l for more resistant species of marine invertebrates (Gentile *et al*., 1983).

Reduced growth of sensitive species of aquatic organisms has been recorded at water concentrations of 0.04 to 1.0 µg Hg/l. The rainbow trout (*Salmo gairdneri*) was the most sensitive species tested; growth reduction was observed after 64 days in 0.04 µg Hg/l as methylmercury, or 0.11 µg Hg/l as phenylmercury (EPA, 1980). In adults of the marine mollusc *Crepidula fornicata*, growth was reduced after 16 weeks in 0.25 µg Hg^{2+}/1 (Thain, 1984). Growth inhibition was recorded in freshwater algae after exposure of 24 hours to 10 days to 0.3 to 0.6 µg organomercury/l, in brook trout (*Salvelinus fontinalis*) alevins after exposure for 21 days to 0.79 µg organomercury/l (EPA, 1980), and in the marine alga *Scripsiella faeroense* exposed to 1.0 µg Hg^{2+}/l for 24 hours (Kayser, 1976).

Adverse effects of mercury to aquatic organisms, in addition to those listed on reproduction and growth, have been documented at water concentrations of 0.88 to 5.0 µg/l: enzyme disruption in brook trout (*Salvelinus fontinalis*) embryos immersed for 17 days in solutions containing 0.88 ug/l, as methylmercury (EPA, 1980); an increased incidence of frustule abnormalities and burst thecae in two species of marine algae during exposure to 1.0 µg Hg^{2+}/l for 24 hours (Kayser, 1976; Saboski, 1977); arrested development of sea urchin larvae at 3.0 µg Hg^{2+}/l for 40 hours (EPA, 1980); decreased rate of intestinal transport of glucose, fructose, glycine, and tryptophan in the murrel (*Channa punctatus*) at 3.0 µg Hg^{2+}/l for 30 days (Sastry *et al*., 1982); altered blood

chemistry in striped bass (*Morone saxatilis*) at 5.0 ug Hg^{2+}/l in 60 days (Dawson, 1982); and decreased respiration in striped bass 30 days postexposure after immersion for 30 to 120 days in 5.0 µg Hg^{2+}/l (Armstrong, 1979; EPA, 1980). In marine molluscs exposed to water concentrations of 6 to 10 ug Hg^{2+}/l for 96 hours, the feeding of adults ceased and the swimming rate of larval stages declined (Thain, 1984). At 44 µg Hg^{2+}/l for 30 days, the freshwater fish *Notopterus notopterus* showed generalized metabolic derangement (Verma and Tonk, 1983). In freshwater planarians exposed to 80 to 100 µg/l as methylmercury, behavior was modified and regeneration retarded (Best et al., 1981). And at high sublethal concentrations of methylmercury, rainbow trout were listless and darkly pigmented; appetite was reduced, and digestion was poor (Rodgers and Beamish, 1982).

At lower trophic levels, the efficiency of mercury transfer was low through natural aquatic food chains; however, in animals of higher trophic levels, such as predatory teleosts and fish-eating birds and mammals, the transfer was markedly amplified (Eisler 1978, 1981).

High uptake and accumulation of mercury from the medium by representative species of marine and freshwater teleosts and invertebrates have been documented (Kopfler 1974; Eisler 1978, 1981; Birge *et al*. 1979; Huckabee *et al*. 1979; EPA 1980, EPA. 1985; Stokes *et al*. 1981; Rodgers and Beamish 1982; Hirota *et al*., 1983; Clarkson *et al*., 1984; McClurg, 1984; Niimi and Lowe-Jinde, 1984; Ramamoorthy and Blumhagen, 1984; Ribeyre and Boudou, 1984; Thain, 1984). Accumulation patterns were enhanced or significantly modified by numerous biological and abiotic factors (NAS 1978; Eisler 1978, 1981, 1984, 1985; EPA 1980, 1985; Stokes *et al*. 1981; Rodgers and Beamish, 1982; Clarkson *et al*., 1984; Ramamoorthy and Blumhagen, 1984; Ribeyre and Boudou, 1984). In general, the accumulation of mercury was markedly enhanced at elevated

water temperatures, reduced water salinity or hardness, reduced water pH, increased age of the organism, and reduced organic matter content of the medium; in the presence of zinc, cadmium, or selenium in solution; after increased duration of exposure; and in the presence of increased nominal concentrations of protein-bound mercury. Uptake patterns were significantly modified by sex, sexual condition, prior history of exposure to Hg salts, the presence of complexing and chelating agents in solution, dietary composition, feeding niche, tissue specificity, and metabolism; however, trends were not consistent between species and it is difficult to generalize. In one example, Ribeyre and Boudou (1984) immersed rainbow trout in solutions containing 0.1 µg Hg/l, as methylmercury: after 30 days, bioconcentration factors (BCF) ranged from 28,300 for brain to 238,000 for spleen; values were intermediate for muscle (30,000), whole fish (36,000), blood (102,000), liver (110,000), kidney (137,000), and gill (163,000). The values may have been higher if exposure had extended beyond 30 days; Rodgers and Beamish (1982) showed that whole body Hg residues in rainbow trout subjected to mercury insult continued to increase for the first 66 days before stabilizing. When mercury was presented as inorganic mercuric ion at 0.1µg/l for 30 days, BCF values were usually lower than in trout exposed to methylmercury: 2,300 for muscle; 6,800 for brain; 7,000 for whole trout; 14,300 for blood; 25,000 for liver; 53,000 for kidney; 68,600 for gill; and 521,000 for spleen (Ribeyre and Boudou, 1984). The high BCF values recorded for rainbow trout were probably due to efficient uptake from water, coupled with slow depuration (Rodgers and Beamish, 1982). Whole body levels up to 100 mg Hg/kg were reportedly not lethal to rainbow trout, although 20 to 30 mg/kg were associated with reduced appetite, loss of equilibrium, and hyperplasia of gill epithelium (Niimi and Lowe-Jinde, 1984). However, brook trout showed toxic signs and death at whole body residues of only 5 to 7 mg/kg (Armstrong, 1979). In another

example, the marine copepod *Acartia clausi*, subjected to 0.05µg/l of mercury and higher, reached equilibrium with the medium in only 24 hours. In that study (Hirota et al., 1983), BCF values for whole *Acartia* after 24-hour exposures were 14,360 for inorganic mercuric ion (0.05 µg/l) and, for methylmercury, 179,200 (0.05 µg/l) and 181,000 (0.1 µg/l).

Elimination of accumulated mercury, both organic and inorganic, from aquatic organisms is a complex multicompartmental process, but appears to be largely dependent on its rate of biological assimilation. This rate, in turn, varies widely (20% to 90%) between species, for reasons as yet unexplained (NAS, 1978). For example, mercury associated with dietary components that are not assimilated is eliminated rapidly with feces. The rest is absorbed across the gut and incorporated into tissues. This assimilated fraction of mercury is depurated much more slowly, at a rate positively correlated with the organism's metabolism (NAS 1978; Rodgers and Beamish, 1982). Time to eliminate 50% of biologically assimilated mercury and its compounds (Tb 1/2) is variable. Among various species of freshwater teleosts, Tb 1/2 values (in days) were 20 for guppies *Poecilia reticulatus*, 23 for goldfish *Carassius auratus*, 100 for northern pike, and 1,000 each for mosquitofish *Gambusia affinis*, brook trout, and rainbow trout (Huckabee *et al*., 1979) Similar range in Tb 1/2 values was recorded for invertebrates and marine fishes: 297 days for the crayfish *Astacus fluviatilis*, 435 days for mussel, 481 days for the clam *Tapes decussatus* 1,030 days for the eel *Anguilla vulgaris*, and 1,200 days for the flounder *Pleuronectes flesus* (NAS, 1978).

Mercury-tolerant strains of bacteria (Colwell *et al*., 1976), protozoa (Berk *et al*. 1978), crustaceans (Green *et al*., 1976; Weis 1976), and fish (Weis, 1984) have been reported. It has been suggested that the mercury-resistant strains of bacteria that have been cultured or discovered may have application in mobilization or fixation of mercury

from contaminated aquatic environments to the extent that polluted areas may become innocuous (Colwell *et al*., 1976). The marine protozoan *Uronemia nigricans*, after feeding on Hg-laden bacteria, acquired mercury tolerance within a single generation (Berk *et al*., 1978). The white shrimp (*Penaeus setiferus*), preexposed for 57 days to 1 µg Hg/l, did not differ from controls during either exposure or subsequent Hg stress experiments (Green *et al*., 1976); this observation suggested that nonsensitization or adaptation mechanisms are involved. The fiddler crab (*Uca pugilator*) seemed unusually resistant and showed negligible uptake or effects during exposure to 100 µg Hg/l for 2 weeks (Weis, 1976). Reasons to account for Hg adaptation of the estuarine cyprinodontiform teleost *Fundulus hete roclitus* to both methylmercury and inorganic mercury are under investigation (Weis, 1984).

2.19 INTERACTIONS OF MERCURY WITH OTHER METALS

The effects on aquatic organisms due to interactions of mercury with cadmium, copper, selenium, and zinc were found to be dependent on exposure concentrations (Birge *et al*., 1979). In general, effects were less than additive at lower exposure concentrations and greater than additive (synergistic) at higher concentrations. Zinc and cadmium were reported to reduce the teratogenic effects of methylmercury to killifish (Weis *et al*. 1981). Cadmium added to methylmercury reduced the retardation effect on fin regeneration in mullet (Weis and Weis 1978). The percentage of embryos affected and degree of malformation observed due to exposure of killifish eggs to 20-50 µg/l methylmercury was reduced when cadmium or zinc was added. Selenium was reported to reduce the developmental effects of inorganic mercury to embryos of the medaka (Japanese ricefish), but only after the formation of the embryonic liver (Bowers *et al*. 1980). Interactions between inorganic mercury and zinc, PCBs, and a PAH

(fluoranthene) were observed to be generally additive in sediment exposure to a marine amphipod (Swartz *et al*., 1988).

A mixture of an inorganic form of mercury (mercuric chloride) and the chlorides of zinc and lead had a synergistic toxic effect on the water exposure of a marine ciliate *Uronema marinum* (Parker, 1979).

CHAPTER THREE

3.0 MATERIALS AND METHODS

3.1 STUDY AREA

The Volta Lake is Africa's largest artificial inland fresh water body. It was formed in 1965 as a result of the construction of the Akosombo dam which generates hydroelectric power for the Ghana. It has a surface area of 8500 km^2 and covers about 4% of Ghana's total land area. It has shoreline of 4880 km. The Volta Lake stretches from the Akosombo dam in the south-eastern to the town of Yapei, 520 km to the north of Ghana (Fig 2). It covers 1,232 communities with a population of over 300,000 people living along the shoreline whose livelihood depend on the Volta Lake (Braimah, 2001). The Lake has 32 landing sites which also serve as market centres. It is estimated that about 80,000 of the people living along the Lake shores are fishers, 20,000 are fish processors and the others are traders involved in the Volta Lake fishing industry (NAFAG, 2004 – 2005). Fishing in the Volta Lake contributes about 90% of the total inland fish (NAFAG, 2004-2005). There are about 140 different fish species contributing approximately 20% to the total fish catch in the country (Braimah, 2001). The most landed species belongs to the tilapia species such as *Chrysichtys sp*. (34.3%), *Synodontis sp*. (11.4%), *Mormyrids* (2.0%), *Heterotis* (1.5%), *Clarias sp*. (1.5%), *Schilbeide* (1.4%), Odaxothrissamento (1.4%), *Bagrus sp*. (1.35%), and *Citharinus sp*. (1.2%). The different species of fishes in the lake makes it a hot spot for a lot of fish-eating birds. The high population of these fish-eating birds on the lake makes them more accessible to the residents that live along the shores of the lake. As a result of the feeding habits of the people that live along the lake most of them have incorporated a lot of these birds into their diets as an alternate source of meat.

3.2 SAMPLING

The distribution of mercury in ten different tissues of fish-eating birds was studied using forty birds on the Volta Lake. The birds were sampled from eight landing sites which serve as marketing centers along the shores of the Volta Lake. The sampling sites included: Yeji, Makango, Kete-Krachie, Kpando Torkpo, Kwame Krom, Dambai, Akateng and Tepa Abotoase. The sampling sites were chosen due to their accessibility and the population of birds.

The birds were killed on site with a garden gun and placed on ice in clean polyethene bags and stored in an ice chest. The samples were transported to the Department Of Wildlife, Faculty of Renewable Natural Resources, KNUST for identification. The birds were identified by using their external features and comparing the features with those found in literature the ‘‘Birds in Ghana’’. After identification the birds were weighed and stored in a deep freezer prior to analysis.

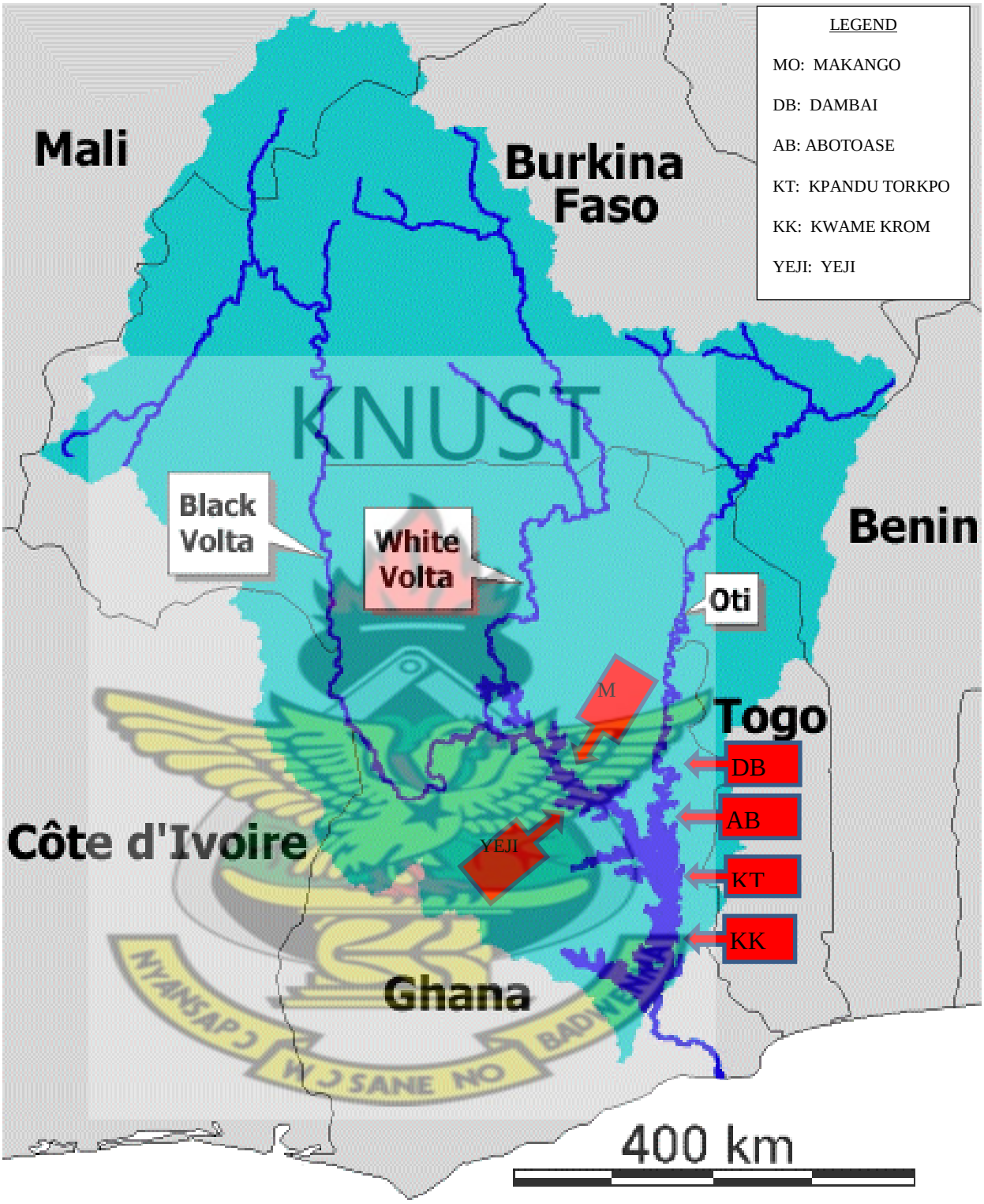


Fig 2: Map of Volta Lake with Sampling Sites Inset

(Source: VRA)

3.3 SAMPLE PREPARATION

The birds were dissected with dissection apparatus (made of stainless steel) and the various tissues separated into kidney, intestines, lungs, heart, gizzard, blood, brain, liver, flesh and feathers. Each tissue was washed with deionized water minced into smaller pieces and homogenized.

3.4 ANALYSIS OF SAMPLES

The samples were digested for total mercury determination by an open flask procedure developed by Akagi and Nishimura (1991). The accuracy of this method has been verified at NIMD through interlaboratory comparison exercise and by participation in the analyses of Certified Reference Materials (CRM) supplied by the International Atomic Energy Agency (IAEA). In the procedure, approximately 0.5 g of each tissue was weighed into a 50 ml digestion tube and 1 ml deionized water, 2ml of a mixture of HNO_3 and $HClO_4$ (1:1) and 5 ml H_2SO_4 was added. The samples were digested at 200^{o}C ±5 for 30 minutes, cooled to room temperature and topped to the 50 ml mark with deionized distilled water. A blank and standard solution digest using 25, 50, and 100 L of l g/mL standard Hg solution were subjected to the same treatment. The total mercury concentrations were determined in all the digests by cold vapour atomic absorption spectrophotometry using an automatic Mercury Analyzer Model HG-5000 (Sanso Seisakusho Co., Ltd., Japan) developed at NIMD. The reducing reagent used for the analysis was 0.5 mL of 10% (w/v) $SnCl_2$ in1 M HCl. Quality assurance samples analyzed included procedural blanks, replicate samples and post-digestion spikes. The accuracy of the procedure was determined by analysis of standard reference materials (SRMs). The SRMs included National Research Council of Canada (NRCC) DOLT-4 (dogfish liver) and TORT-2 (lobster hepatopancreas). All CRMs were recovered at ±10% of the certified value; 0.27±0.06 for Tort-2 and 2.58±0.22 for Dolt-4

3.5 STATISTICAL ANALYSIS

Statistical analyses were conducted using Statistical Package for the Social Sciences (SPSS 16.0 Inc, 2007). Mean Hg concentrations for each individual sample, for each species, and then for the entire sample pool were analyzed. Simple Linear Regression was used to analyze relationships between Hg concentrations in the tissues and weight of the birds. Microsoft excel was also used for plotting graphs. Differences in mean values were accepted as being statistically significant if $p < 0.05$ and $p < 0.01$.

CHAPTER FOUR

4.0 RESULTS AND DISCUSSION

4.1 SPECIES OF BIRDS ANALYSED IN THE STUDY

A total of 40 birds representing four (4) different species were shot and used for the study. The species captured in the study included; ten (10) Yellow billed kite (*Milvus migrans parasitus*), ten (10) Squacco heron (*Ardeola rolloides)*, ten (10) Little egret (*Egretta garzetta*) and ten (10) Grey heron (*Ardea Cinerea*). The range and mean weight of the birds analyzed in the study are presented in Table 1. The results of the T-Hg analysis are presented in Tables 1-7 and Figure 3. Individual results and results by species are presented in Tables 8-11 and Figures 4-7 in appendix.

Table 1: Range and mean weight of the birds analyzed in the study

Species Of Bird	Weight (mean +SD) /g	Range / g
Egretta garzetta	430.86 ± 24.85	388.00– 460.00
Milvus migrans parasitus	818.34 ± 56.99	720.78 – 880.00
Ardeola ralloides	224.03 ± 16.77	198.02 – 245.12
Ardea cinerea	1378.27 ± 144.84	1187.45 – 1556.00

4.2 TOTAL MERCURY CONCENTRATIONS IN THE BIRDS ANALYSED IN THE STUDY

The concentrations of mercury in the tissues of all the birds sampled for the study ranged between 0.43 – 2.86 µg/g. Mercury concentrations in the tissues of *Egretta garzetta*, *Milvus migrans parasitus*, *Ardeola rolloides* and *Ardea Cinerea* ranged from 0.91-1.78 µg/g; 0.43-2.14 µg/g; 0.85-1.92 µg/g and 0.77-2.86 µg/g respectively. The

highest mean concentration of total mercury (THg) (1.84 ± 0.40 µg/g, range 1.40-2.86 µg/g, n=10) was found in *Ardea cinerea,* followed by *Ardeola ralloides* (1.40 ± 0.15 µg/g, range 1.19-1.65 µg/g n=10) , *Egretta garzetta* (1.38 ± 0.10 µg/g, range 1.24-1.58 µg/g, n=10) and *milvus migrans parasitus* (1.32 ± 0.19 µg/g, range 1.00-1.78 µg/g, n= 10) (Table 2).

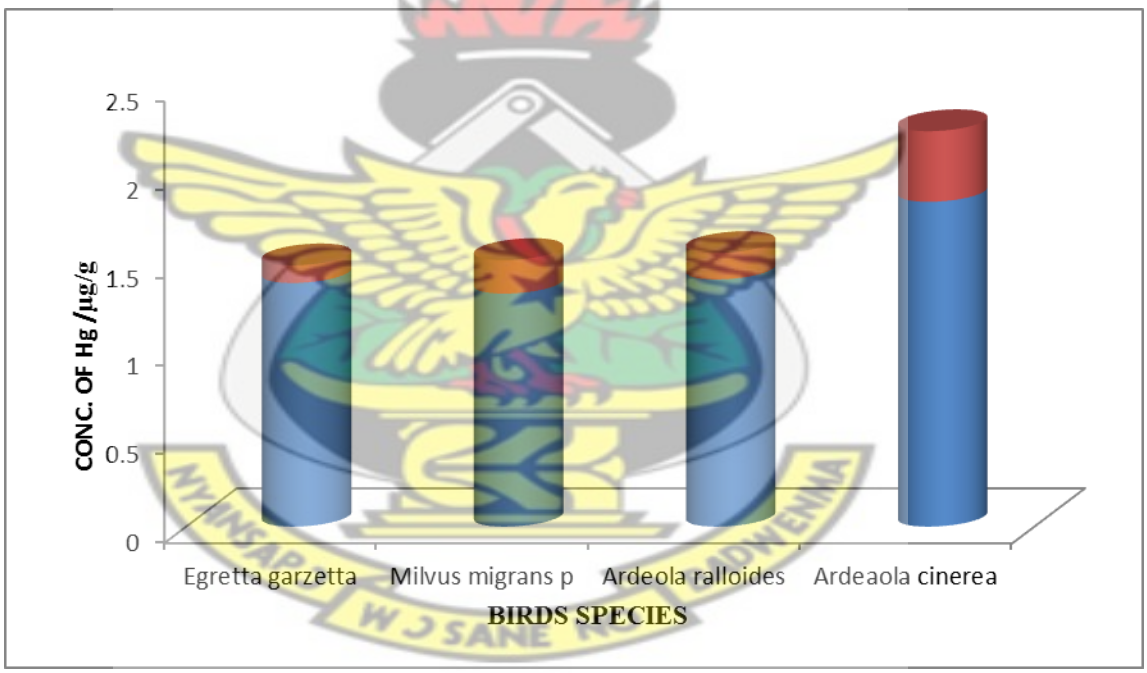
Almost all the birds analyzed in this study had mean total mercury concentration exceeding 1.00 µg/g. About 63% of the birds had mean THg concentration between 1.00µg/g and 1.50µ/g and 37% had mean THg concentrations between 1.50 µg/g and 2.80µg/g. About 70% of the *Ardea cinerea* population fell within the 1.5-2.80 µg/g range (Tables 8-11 in Appendix).

In this study, significant variations were found between the average THg concentrations in all the four species of birds with *Ardea cinerea* having the highest mean mercury concentration. The variations in the THg concentrations recorded in the birds may be due to their eating habits since they prey on different types of fish. *Ardea cinerea* usually capture fishes of 10–40 cm long, (Del Hoyo *et al*., 1992). According to other published works, size can play an important role in the choice of prey and *Ardea cinerea* generally show preference for larger prey (Britton & Moser, 1982; Feunteun & Marion, 1994). Gwiazda and Amirowicz (2006) concluded that larger prey were more profitable for *Ardea cinerea* to forage. Storelli *et al*. (2005) reported that mercury concentrations in fish increase with body size. Mercury concentrations are highest in large, long-lived predatory fish. The highest mercury concentration found in the *Ardea cinerea* in this study suggests that they feed at a higher trophic level in the aquatic food chain and therefore more susceptible to mercury pollution.

Table 2: Mean and range of THg concentrations of the birds analysed in the study

Species Of Bird	Conc. of THg(mean +SD)/ µg/g	Range / µg/g
Egretta garzetta	1.38 ± 0.10	1.24-1.58
Milvus migrans parasitus	1.32 ± 0.19	1.00-1.78
Ardeola ralloides	1.40 ± 0.15	1.19-1.65
Ardea cinerea	1.84 ± 0.40	1.40-2.86

Fig 3 : Levels of mercury (mean + SD) in the birds analysed in the study in µg/g w.w.



4.3 COMPARISON OF THg LEVELS IN THE TISSUES STUDIED

Among the tissues studied the lung tissues had the least mean THg concentration and the highest was found in the kidney, liver and feather tissues of all the birds. The least mean lung THg concentration (1.08±0.18 µg/g, range 0.85-1.44 µg/g) was found in *Ardeola ralloides* followed by *Milvus migrans parasitus* (1.09±0.32 µg/g, range 0.43-1.60 µg/g), *Egretta garzetta* (1.09±0.32 µg/g, range 0.91-1.36 µg/g) and *Ardea cenerea* (1.23±0.57 µg/g, range 0.77-2.70 µg/g). These mean concentrations accounted for 7.66%, 8.33%, 8.03% and 6.84% of the tissues THg of *Ardeola ralloides, Milvus migrans parasitus; Egretta garzetta* and *Ardea cenerea* respectively (Table 3). Total mercury concentrations in kidney, liver and feather tissues of *Egretta garzetta* ranged from 1.34-1.73, (mean 1.51±0.14); 1.40-1.75, (mean 1.54±0.13) and 1.43-1.78, (mean 1.59±0.12) respectively (Table 3). *Milvus migrans parasitus* had kidney, liver and feather tissues THg concentration ranging from 1.25-1.94, mean1.39±0.27; 1.35-1.97, mean 1.54±0.21 and 1.33-2.14, mean 1.33±0.28 respectively. The concentration of THg in the Kidney, liver and feather tissues of *Ardeola ralloides* ranged from 1.38-1.84, mean 1.56±0.24; 1.39-1.83, mean 1.61±0.22 and 1.41-1.92, mean 1.68±0.19 and was about 30% higher than the concentrations found in the kidney, liver and feather tissues of both *Egretta garzetta* and *Milvus migrans parasitus.* The highest THg concentration in kidney, liver and feather tissues were found in *Ardea cinerea* and ranged from 1.97-2.85, mean 2.26±0.31; 2.02-2.86, mean 2.32±0.30 and 2.05-2.84, mean 2.45±0.29 respectively. THg concentration in kidney, liver and feather tissues accounted for between 35 – 45% of the total tissue mercury in all the four species of birds. Among the tissues studied THg concentrations in the birds were found to increase in the order of lungs < heart <brain < gizzard < intestines < flesh < blood < kidney < liver < feather. Highest values of THg concentrations found in the liver, kidney and feather tissues, is

accounted for in terms of their role in demethylation, storage and excretion of mercury from the birds.

Table 3: Mean and range of THg concentrations in the tissues of the birds in µg/g

Tissue	BIRDS			
	Egretta garzetta	***Milvus migrans parasitus***	***Ardeola ralloides***	***Ardea cinerea***
lung	**1.09±0.32** (0.91-1.36)	**1.09±0.32** (0.43-1.60)	**1.08±0.18** (0.85-1.44)	**1.23±0.57** (0.77-2.70)
heart	**1.19±0.11** (1.0-1.39)	**1.17±0.27** (0.79-1.68)	**1.28±0.16** (1.19-1.55)	**1.30±0.58** (0.79-2.76)
brain	**1.32±0.07** (1.23-1.43)	**1.17±0.20** (0.71-1.48)	**1.28±0.26** (0.99-1.36)	**1.45±0.46** (1.18-2.57)
gizzard	**1.23±0.07** (1.14-1.36)	**1.22±0.24** (1.00-1.69)	**1.31±0.18** (1.00-1.60)	**1.49±0.62** (0.96-2.79)
intestines	**1.33±0.14** (1.22-1.65)	**1.23±0.22** (1.01-1.67)	**1.36±0.15** (1,21-1.61)	**1.68±0.56** (1.15-2.79)
flesh	**1.32±0.07** (1.35-1.69)	**1.33±0.28** (1.02-1.89)	**1.51±0.25** (1.29-1.73)	**1.94±0.55** (1.91-2.80)
blood	**1.46±0.10** (1.34-1.67)	**1.32±0.19** (1.13-1.73)	**1.43±0.18** (1.23-1.66)	**1.86±0.58** (1.20-2.85)
kidney	**1.51±0.14** (1.34-1.73)	**1.39±0.27** (1.25-1.94)	**1.56±0.24** (1.38-1.84)	**2.26±0.31** (1.97-2.85)
liver	**1.54±0.13** (1.40-1.75)	**1.54±0.21** (1.35-1.97)	**1.61±0.22** (1.39-1.83)	**2.32±0.30** (2.02-2.86)
feather	**1.59±0.12** (1.43-1.78)	**1.62±0.24** (1.33-2.14)	**1.68±0.19** (1.41-1.92)	**2.45±0.29** (2.05-2.84)

4.4 CORRELATIONS BETWEEN THg IN TISSUES AND BODY WEIGHT OF BIRDS

Within a particular species, concentrations of THg in the tissues were found to increase with body weight. About 18.6% increase in weight of *Egretta garzetta* corresponded to about 34% increase in THg concentration. Also about 23% increase in weight corresponded to about 38% increase in THg concentration in the tissues of *Ardeola ralloides*. Furthermore, about 22% and 31% increase in weight of *Milvus migrans parasitus* and *Ardea cinerea* respectively, corresponded to almost 100% increase in THg concentration in the two species. A very good correlation was found between weight and all the tissues studied. Pearson correlation coefficient between weight and THg concentration in all the tissues studied showed a linear relationship. THg concentrations in the lungs, heart, gizzard, intestines, blood, brain, flesh, kidney, liver, and feather tissues of *Egretta garztta* were positively correlated with weight with correlation coefficients of 0.94, 0.97, 0.99, 0.93, 0.97, 0.98, 0.99, 0.99, 0.99 and 0.99 respectively (Table 4). Concentration of THg in almost all the tissues of *Egretta garzetta* correlated positively with each other. Similarly, almost perfect correlations were found between weight and the THg concentrations in the tissues of both *Milvus migrans parasitus* and *Ardeola ralloides (*Tables 5 and 6). These relationships suggest that as the birds mature, mercury is accumulated in all the tissues studied.

Total mercury concentrations in the lungs, heart, gizzard, intestines, blood, brain, flesh, kidney, liver, and feather tissues of *Ardea cinerea* were positively correlated with weight with correlation coefficients of 0 .56, 0.76, 0.81, 0.91, 0.96, 0.66, 0.73, 0.95, 0.89 and 0.96 respectively (Table 7). The correlation coefficients between the concentrations of THg in the tissues and weight of *Ardea cinerea* ranged from good (0.56) to almost perfect (0.96). This suggests that total mercury is diversely

distributed in the tissues of *Ardea cinerea* as they grow.

Table 4: Correlation of THg between the tissues of *Egretta garzetta*

r	Lung	Heart	Gizz.	Intest.	Blood	Brain	Flesh	Kid.	Liver	Feath.
Wt	.943**	.973**	.992**	.932**	.971**	.981**	.989**	.989**	.997**	.994**
Lung	1.000	.953**	.937**	.829**	.916**	.952**	.903**	.931**	.938**	.970**
Heart		1.000	.971**	.920**	.963**	.959**	.955**	.972**	.964**	.979**
Gizz.			1.000	.932**	.961**	.974*	.986*	.991**	.996**	.987**
Intest.				1.000	.931**	.869**	.921**	.917**	.924**	.915**
Blood					1.000	.961**	.959**	.971**	.964**	.967**
Brain						1.000	.980**	.985**	.982**	.974**
Flesh							1.000	.994**	.992**	.978**
Kid								1.000	.993**	.987**
Liver									1.000	.991**
Feather										1.000

***P < 0.05, **P < 0.01**

Table 5: Correlation coefficients (r values) of THg between the tissues of *Milvus migrans parasitus*

r	Lung	Heart	Gizz.	Intest.	Blood	Brain	Flesh	Kid.	Liver	Feath.
Wt	.866**	.869**	.836**	.892**	.907**	.855**	.858**	.833**	.849**	.894**
Lung	1.000	.954**	.879**	.896**	.896**	.989**	.911**	.807**	.861**	.900**
Heart		1.000	.981**	.974**	.972**	.909**	.992**	.942**	.963**	.959**
Gizz.			1.000	.981**	.975**	.813**	.995**	.985**	.984**	.949**
Intest.				1.000	.988**	.841**	.986**	.971**	.990**	.979**
Blood					1.000	.851**	.986**	.973**	.970**	.971**
Brain						1.000	.854**	.734*	.799**	.866**
Flesh							1.000	.976**	.978**	.958**
Kid.								1.000	.973**	.929**
Liver									1.000	.978**
Feath.										1.000

*P < 0.05, **P < 0.01

Table 6: Correlation coefficients (r values) of THg between the tissues of *Ardeola ralloides*

r	Lung	Heart	Gizz.	Intest.	Blood	Brain	Flesh	Kid.	Liver	Feath
Wt	.930**	.760*	.905**	.754*	.915**	.833**	.876**	.892**	.908**	.929**
Lung	1.000	.786**	.874**	.844**	.867**	.626**	.763*	.796**	.789**	.846**
Heart		1.000	.867**	.686*	.855**	.650*	.797**	.828**	.808**	.823**
Gizz.			1.000	.813**	.931**	.746*	.869**	.879**	.883**	.933**
Intest.				1.000	.835**	.466*	.733**	.772**	.688**	.716*
Blood					1.000	.776**	.948**	.955**	.930**	.932**
Brain						1.000	.885**	.860**	919**	.866**
Flesh							1.000	.992**	.988**	.950**
Kid.								1.000	.981**	.945**
Liver									1.000	.976**
Feath										1.000

*P < 0.05, **P < 0.01

Table 7: Correlation coefficients (r values) of THg between the tissues of *Ardea cinerea*

r	Lung	Heart	Gizz.	Intest.	Blood	Brain	Flesh	Kid.	Liver	Feath.
Wt	.863*	.964**	.813**	.911**	.960**	.661**	.736**	.951**	.892**	.962**
Lung	1.000	.995**	.893**	.860**	.840**	.978**	.753*	.820**	.819**	.719*
Heart		1.000	.874**	.834**	.831**	.978**	.758*	.817**	.818**	.712*
Gizz.			1.000	.855**	.882**	880**	.807**	.830**	.800**	.772**
Intest.				1.000	971**	.861**	.693*	.856**	.877**	.896**
Blood					1.000	.850**	.737*	.873**	.888**	.936**
Brain						1.000	.736*	.813**	822**	.739*
Flesh							1.000	.729*	.734*	.554*
Kid.								1.000	.987**	.879**
Liver									1.000	.886**
Feath.										1.000

*P < 0.05, **P < 0.01

4.5 COMPARISON OF LEVELS OF T-Hg IN THE BIRDS STUDIED WITH THOSE OF OTHER BIRDS WORLDWIDE

In this study, mercury was not uniformly distributed among the tissues studied and this finding is generally similar to other studies on fish eating birds. In all the tissues analysed, liver, kidney and feathers appeared to be the most preferred organs for mercury accretion in the birds. Frodello *et al*., (2000) and Storelli *et al*., (2005) reported similar results of which the liver appeared to be the preferential organ for mercury accumulation, followed by the kidney. High THg concentration in the liver is accounted for possibly by the role played by the liver in terms of biotransformation, of which the organ demethylates organic mercury into less toxic form (Kenabo *et al.,* 2009). The liver is the primary filtering and detoxifying organ in birds and it is thought that methylated mercury is demethylated in the liver, resulting in the production of inorganic mercury. In a similar study, estimation of mercury and cadmium concentration in the common cormorants (*Phalacrocorax carbo*), gathered from the Biwa and Tokyo lakes in Japan, revealed the maximum mercury concentration in the liver and the kidney (Saeki *et al.*, 2000). Also the findings of this study are consistent

with the results reported by Kim *et al.*, (1996), who revealed that the highest concentration of methyl mercury was in the liver of sea bird species.

The high concentration of mercury found in the kidneys in this study is explained on the basis that the kidney is the major reservoir of inorganic mercury in birds as well as mammals (USDI, 1998). The kidney is equipped with an efficient, energy-dependant mechanism for disposing of metals such as mercury. Kidney tissue contains a thiol-rich protein called metallothionein. Exposure to toxic metals triggers the production of this protein which binds tightly to the metal, retaining it in the kidney tissue in a relatively harmless form. As long as the kidney's capacity for production of metallothionein is not overwhelmed, mercury excretion can eventually balance intake, thereby limiting worsening of symptoms. However, acute high doses of mercury can readily precipitate renal failure, one of the classic symptoms of mercury poisoning. It is therefore not surprising that the major toxic effect of inorganic mercury is kidney damage specifically; necrosis of the proximal tubular cells (Ware *et al*., 1975). Spalding *et al*., (1994), found that kidney disease and gout were present in great white herons that had greater than 25µg/g w.w. mercury concentrations in the liver and the kidneys. Though elevated mercury levels were found in the kidney tissues of the birds in this study, the highest concentration of THg (2.85µg/g w.w.) found in the kidney tissues of *Ardea cinerea* was about nine times less than the concentration reported to cause diseases and adverse effects in birds.

High concentration of mercury found in the feathers is accounted for probably due to the fact that mercury is excreted mainly via the feathers and also via the eggs (Parslow, 1973). Feathers represent the major pathway for elimination of mercury in birds and body feathers are useful indicators for assessment of whole bird mercury burdens (Furness *et al*., 1986; Liu *et al*., 2006; Thompson *et al*., 1996). Almost all mercury in

feathers is present as methylmercury (Thompson and Furness, 1989). The keratin in bird's feathers is not easily degradable and mercury is probably associated with the disulphide bonds of the keratin. Laboratory studies with the great skua (*Catharcta skua*) fed with mercury contaminated prey excreted mercury via feathers (Bearhop *et al*., 2000).

Mercury in the food eaten during feather growth considerably affects the mercury levels of the feathers. From the result of this study, it can be confirmed that feathers can be used as indicators of environmental mercury exposure. Since feather mercury concentration analysis is a useful, non-invasive method for monitoring mercury exposure in wild populations without sacrificing the individual (Mautino and Bell, 1987). Total feathers should be analyzed rather than only parts of them (Solonen and Lodenius, 1990). Metal pollutants can be incorporated into birds' feathers along three routes: from the blood during feather growth, from the excretion of salt or the secretion of preen glands, and through contact with the habitat (Goede and de Bruin 1984). Feathers may serve as a useful indicator of inorganic pollutants pollution because concentrations of metals correlate well with their internal levels during the time of feather formation.

Moreover, mercury levels in feathers are stable and the metal may bind to the sulphydryl groups of the keratin as the feathers grow. Boening (2000) and Ochoa-Acuna *et al*. (2002) found some differences in Hg concentrations in feathers across taxonomic bird groups. They assumed that Hg contents in feathers depended on feeding strategies and to a lesser extent on differences in the metabolism and excretion of this metal. Mercury concentrations in birds also depend on body size, moult strategy, migration patterns and physiology (Stewart *et al*. 1997). Fish-eating birds are at risk of higher contents of Hg because its circulation is associated mainly with water basins.

The most important pathway of mercury elimination in birds is its ‘‘excretion’’ when the feathers are moulted (Ochoa-acuna *et al*., 2002; Dauwe *et al*., 2003).

In this study, a very high significant positive relationships between T-Hg levels in the tissues and body weight (age) were observed for all the tissue samples analysed (Tables 4-7), suggesting that T-Hg increases in all the tissues as the birds increase in size. For instance, as the weight of *Ardea cinerea* increases from 1187.45g to 1556.00 (about 31% increase in weight), T-Hg mercury concentration doubles in the matured bird. Similarly, as the weight of *Milvus migrans parasitus* increase from 720.78 to 880.00g (about 22% increase in weight), the concentration of T-Hg in the matured bird almost increased by two folds. Similar observations were made for the other species.

In another study, determination of the mercury concentration in the liver of seven species of the wading birds in Southern Florida, it was revealed that mercury concentration in the adult livers was three times more than that of the chicks (Sundlof *et al.*, 1994). This obviously indicates that mercury is accumulated in the body as the birds grow up and feed. These findings are also consistent with the results of other researchers who reported that mercury concentrations in adult birds are higher than juvenile birds. In fact, nutritionally, mercury is a non-essential element in the body and has such a long biological half-life that it accumulates in the body with age and with increasing levels of exposure to the environment (Saeki *et al.*, 2000).

In further comparison, the mean THg concentration, 1.84±0.40 µg/g w.w (Table 2) found in the grey herons in this study was about seven times less than 12 µg/g w.w reported in grey herons from Netherlands (van der Molen *et al*., 1981). Also the mean THg concentrations (8.32 and 9.25 µg/g w.w.) in the liver and kidney tissues of ducks reported by Kinabo *et al*., (2009) in Tanzania were about four times higher than the

highest mean concentration of THg in the liver (2.32±30) and in the kidney (2.26±0.31) tissues of *Ardea cinerea* reported in this study. Burger and Gochfeld (1993), found among heron and egret species in China (Hong Kong and Szechuan), mean chick-feather mercury values that ranged from 0.27 to 2.4 µg/g which are similar to what was found in the little egrets in this study. Houserova *et al*., (2005) reported a mean Hg concentration of 2.3 µg/g in the kidney of 20 cormorants *Phalacrocorax carbo* in the Czech Republic. This value was almost similar to what was found in the kidney tissues of grey heron *Ardea cinerea* analysed in this study. The mercury levels found in feather tissues of little egret in this study (mean 1.58 µg/g w.w., range 1.15-1.7 µg/g w.w. n=10) were far lower than those found (5µg/g w.w) in the same species at Camargue region of the Mediterranean (Cosson *et al*,. 1988). However they were similar to levels found (1–2 µg/g w.w.) in the feathers of flamingo *Phoenicopterus ruber* from the same site (Cosson *et al*. 1988). Goutner and Furness (1993), reported a mean mercury concentration (1.69 µg/g w.w.) in little egret chicks from the Axios Delta Greece that was identical to the results found in the same species in this study. The mean Hg concentration found in the little egrets was higher than the mean concentration (0.37±0.051 µg/g range 0.083-0.246 µg/g) reported by James *et al* in little egret in Hong Kong, China. Furness, (1993) reported that the mean values of Hg in Squacco Heron from the Axios Delta Greece varied from 2.79 to 3.56 these values are however higher than the mean value (1.40 ± 0.15) found in the squacco herons in this study.

The range of mercury concentrations in the tissues of *milvus migrans parasitus* resulting from this study (0.43 - 2.14 µg/g) were generally higher than the concentrations found in outer tail feathers of Finnish Sparrow hawks (*Accipiter nisus*, 0.35 - 0.58 µg/g, Dauwe et al. 2003) and was similar to the concentrations in feathers from various raptor species in southwest Iran (1.25 - 1.87 µg/g, Zolfaghari *et al*. 2007).

Concentrations found in Peregrine Falcons (*Falco peregrinus*) in Texas (2.50 µg/g, Mora *et al*, 2002), Lagger Falcons (*Falco biarmicus jugger*, 3.34 µg/g) in Pakistan (Movalli 2000), Bald Eagles (*Haliaeetus leucocephalus*) in Florida (3.28 µg/g, Wood *et al*. 1996) were higher than the mean concentration found in *milvus migrans parasitus* species in this study. Also the Concentrations found in White-tailed Eagles (*Haliaeetus albicilla*, 16 - 37 µg/g Altmeyer et al, 1991), Peregrine Falcons in Sweden (17.6 µg/g, Lindberg 1984), Osprey (*Pandion haliaetus*, 2 - 23 mg/kg, Anderson *et al*. 2008) and Bald Eagles from the Great Lakes Region (13 - 21 mg/kg, Bowerman *et al*. 1994) were extremely higher than the concentrations determined in the hawk samples in this study.

CHAPTER FIVE

5.0 CONCLUSION AND RECOMMENDATION

5.1 Conclusion

From the study, it can be concluded that;

1. Elevated levels of T-Hg mercury were found in the tissues of all the birds studied however, they were far lower than the levels that have been reported to have adverse effects on both birds and humans.
2. Very high significant positive relationship between T-Hg levels in the tissues and body weight/age were observed for all the tissues analysed in the study and suggest that mercury may accumulate in the tissues as the birds grow since concentrations of THg in the tissues increased with weight/age.
3. The results of the study were generally similar to the results of other studies on fish eating birds. However, in most cases the levels of THg concentrations reported for the birds in this study were far lower than those reported for similar birds in other related studies.

Finally, from the low concentrations of T-Hg found in the birds studied, it can also be concluded that the Volta Lake has not been impacted anthropogenically as far as Hg pollution is concerned and the fish eating birds studied can be considered safe for consumption.

5.2 RECOMMENDATION

From the outcome of this study, it is recommended that;

1. Periodic monitoring should be done to determine whether mercury concentration in the Volta Lake is getting to levels that will pose health threat to the birds and humans.
2. Grey herons should be used as indicator organism for monitoring Hg pollution in the Volta Lake since they accumulate Hg more than the other birds.
3. The liver, kidney and feather tissues should be the target organs for monitoring mercury pollution in birds since they are the preferred organs for mercury accretion in birds.
4. Mining activities along the Black and the White Volta river basins should be banned
5. Human samples such as blood, urine and hair of people living along the Volta Lake who feed on these birds must be analyzed to ascertain the extent of mercury exposure to the populace.

REFERENCES

Akagi, H. and Nishimura, H. (1991). 'Speciation of Mercury in the Environment', in T. Suzuki, N.Imura, and T. W. Clarkson (eds), *Advances in Mercury Toxicology*, Plenum Press, New York,pp. 5-76.

Alberts, J.J., M.T. Price and M. Kania. (1990). Metal concentrations in tissues of *Spartina alterniflora* (Loisel.) and sediments of Georgia salt marshes. *Estuarine ,Coastal and Shelf Science 30*: 47-58.

Allard, M. and P. Stokes. (1989). Mercury in crayfish species from thirteen Ontario Lakes in relation to water chemistry and smallmouth bass (*Micropterus dolomieui)* mercury. *Canadian Journal of Fisheries and Aquatic Sciences 46:*pp 31-34**.**

Andersen, A., K. Julshamn, O. Ringdal, and J. Morkore. (1987). Trace Elements Intakein the Faroe Islands, II: Intake of Mercury and Other Elements by Consumption of Pilot Whales (Globicephalus meleanus). *Science of the Total Environment 65*:*63-68.*

Anderson D.W, Suchanek T.H, Eagles-Smith C.A, Cahill T.M. (2008). Mercury residues and productivity in Osprey and Grebes from a mine dominated ecosystem. *Ecological Applications 18(8) Supp. A227-A238.*

Andre, J. M., A. Boudou, F. Ribeyre, and M. Bernhard. 1991a. Comparative study of mercury accumulation in dolphins (*Stenella coeruleoalba*) from French Atlantic and Mediterranean coasts. *Sci. Total Environ. 104:191-209.*

Andre, J. M., A. Boudou, and F. Ribeyre. (1991b). Mercury accumulation in Delphinidae. *Wat. Air Soil Pollut. 56:187-201.*

Arima, S., and K. Nagakura. (1979). Mercury and selenium content of *Odontoceti*. *Bull. Japanese Soc. Scientific Fish. 45(5):623-626.*

Armstrong, F. A. J. (1979). Effects of mercury compounds on fish. In: J. O. Nriagu (Ed.), *The Biogeochemistry of Mercury in the Environment*. *Elsevier/North-Holland Biomedical Press New York.* pp. *657-670.*

Barr J.F. (1986) Population dynamics of the Common Loon (*Gavia immer*) associated with mercury-contaminated waters in northwestern Ontario. *Canadian Wildlife Service Occasional* 34-67**.**

Battelle. (1987). Measurement of bioavailable mercury species in fresh water and sediments, Palo Alto, California: Electric Power Research Institute.

Bearhop S., Waldron S., Thompson D., Furness R. (2000) Bioamplification of mercury in great skua Catharacta skua chicks: the influence of trophic status as determined by stable isotope signatures in blood and feathers. *Marine Pollution Bulletin 40:181-185*

Becker, D. S., G. N. Bigham, and M. H. Murphy. (1993). Distribution of mercury in a lake food web. Poster presented at 14th annual meeting of the Society of Environmental Toxicology and Chemistry (SETAC), Houston, Texas, *November 14- 18, 1993.*

Becker, D.S. and G.N. Bigham. (1995). Distribution of mercury in the aquatic food web of Onondaga Lake, New York. *Water, Air, and Soil Pollution 80*:563-571.

Beckvar Nancy, Jay Field, Sandra Salazar and Rebecca Hoff (1996). Contaminants in aquatic habitats at hazardous waste sites; mercury. NOAA Technical memorandum NOS Orca 100 seattle: *Harzadous materials Response and Assessment Division, National Oceanic and Atmospheric Administration* p *74*

Beijer, K. and A. Jernelov. (1979). Methylation of mercury in aquatic environments. In: J. O. Nriagu (Ed.), *The Biogeochemistry of Mercury in the Environment*. pp. 203- 210.

Benes, P. and B. Havlik. (1979). Speciation of mercury in natural waters. In: J. O. Nriagu (Ed.), *The Biogeochemistry of Mercury in the Environment*. pp. *175-202.*

Berman, M. and R. Bartha. (1986). Levels of chemical versus biological methylation of mercury in sediments. *Bulletin of Environmental Contamination and Toxicology 36:401-404.*

Biesinger, K.E., L.E. Anderson, and J.G. Eaton. (1982). Chronic effects of inorganic and organic mercury on Daphnia magna: toxicity, accumulation, and loss. *Arch. Environ. Contam. Toxicol. 11*: 769-774.

Birge, W. J., J. A. Black, A. G. Westerman, and J. E. Hudson. (1979). The effects of mercury on reproduction of fish and amphibians. In: J. O. Nriagu (ed.). *The Biogeochemistry of Mercury in the Environment*. pp. 629-655.

Bjerregaard, P. and L. Christensen. (1993). Accumulation of organic and inorganic mercury from food in the tissues of Carcinus maenas: effect of waterborne selenium. *Marine Ecol. Prog. Ser. 99*: 271-281.

Bjornberg, A., L. Håkanson, and L. Lundbergh. (1988). A theory on the mechanisms regulating the bioavailability of mercury in natural waters. *Environmental Pollution 49*:53-61. 46

Bloom, N. S. 1992. On the chemical form of mercury in edible fish and marine invertebrate tissue. *Can. J. Fish. Aquat. Sci. 49*:1010-1017.

Bloom, N.S. 1989. Determination of picogram levels of methylmercury by aqueous phase ethylation, followed by cryogenic gas chromatography with cold vapor atomic fluorescence detection. *Canadian Journal of Fisheries and Aquatic Sciences 46*:1131-1140.

Bloom, N.S. and E.A. Crecelius. (1983). Determination of mercury in seawater at subnanogram per litre levels. *Marine Chemistry 14*:49-59.

Bloom, N.S. and S.W. Effler. (1990). Seasonal variability in the mercury speciation of Onondaga Lake (New York). *Water, Air, Soil Pollut. 53*:251-265.

Bloom, N.S., C.J. Watras, and J.P. Hurley. (1991). Impact of acidification on the methylmercury cycle of remote seepage lakes. *Water, Air, Soil Pollut. 56*:477-491.

Blum, J. M. and R. Bartha. (1980). Effect of salinity on methylation of mercury. *Bulletin of Environmental Contamination and Toxicology 25*:404-408.

Boening D.W. (2000). Ecological effects transport and fate of mercury; a general review. *Chemosphere 40: 1335-1351*.

Bonzongo, J.J., Heim, K.J., Y. Chen, W.B. Lyons, J.J. Warwick, G.C. Miller and P.J. Lechler. (1996). Mercury pathways in the Carson River-Lahontan Reservoir System,Nevada, USA. *Environ. Toxicol. and Chem. 15*(5):677-683.

Born, E. W., I. Kraul, and T. Kristensen. (1981). Mercury, DDT, and PCB in the Atlantic walrus (*Odobenus rosmarus rosmarus*) from the Thule District, North Greenland. *Arctic 34*:255.

Boubacar Barry, Emmanuel Obuobie ,Marc Andreini ,Winston Andah , Mathilde Pluquet, (2005). Comprehensive Assessment of Water Management in Agriculture. A Comparative study on the Volta river basin development and management p 42.

Boudou, A., M. Delnomdedieu, D. Georgescauld, F. Ribeyre, and E. Saouter. (1991). Fundamental roles of biological barriers in mercury accumulation and transfer in freshwater ecosystems (analysis at organism, organ, cell and molecular levels). *Water, Air, and Soil Pollution 56*:807-822.

Bowerman W. W., Evans E.D., Giesy J.P. and Postupalsky S. (1994). Using feathers to assess risk of mercury and selenium to bald eagle reproduction in the Great Lakes Region. Arch. Environ. Contam. Toxicol 27: 294-298.

Bowerman WW, Evans ED, Giesy JP, Postupalsky S (1994) Using Feathers to Assess Risk of Mercury and Selenium to Bald Eagle Reproduction in the Great Lakes Region. *Arch Environ Contam Toxicol 27: 294-298*

Bowers, M. A., D. Dostal, and J. F. Heisinger. (1980). Failure of selenite to protect against mercuric chloride in early developmental stages of the Japanese ricefish (*Oryzias latipes*). *Comparative Biochemistry and Physiology 66C*:175-178.

Braimah, L. I. (2001): Volta lake fisheries management plan: Fisheries sub-sector capacity building project pp 18 - 32.

Breteler, R.J., I. Valiela, and J.M. Teal. (1981). Bioavailability of mercury in several North-eastern U.S. Spartina ecosystems. *Estuarine, Coastal and Shelf Science 12*: 155-166.

Brock, V. (1993). Effects of mercury on the physiological condition and content of the biomarker ALA in the oyster *Ostrea edulis*. *Mar. Ecol. Prog. Series. 96*:169-175.

Brown, D. A., R. W. Gosset, P. Hershelman, H. A. Schaefer, K. D. Jenkins, and E. M. Perkins. (1983). Bioaccumulation and detoxification of contaminants in marine organisms from Southern California coastal waters. In: D. F. Soule and D. Walsh (Eds.), *Waste Disposal in the Oceans*. p. 171.

Bryan, G. W. and W. J. Langston. (1992). Bioavailability, accumulation and effects of heavy metals in sediments with special reference to United Kingdom estuaries: a review. *Environ. Pollut. 76*: 89-131.

Burger J., Nisbet I.C.T and Gochfeld M. (1992a). Metal levels in regrown feathers: Assessment of contamination on the wintering and breeding grounds in the same individuals. *Toxicol. Environ. Heailth 36: 327-338.*

Burger J, Gochfeld M. (2000) Metal levels in feathers of 12 species of seabirds from Midway Atoll in the northern Pacific Ocean. *Sci Total Environ 257:37–52*

Burger J., Gochfeld M. (2001) Metal levels in feathers of cormorants, flamingos and gulls from the coast of Namibia in Southern Africa. Environ Monit Assess 69:195–203

Burger J, Parsons K, Benson T, Shukla T, Rothstein D. and Gochfeld M. (1992). Heavy metal and selenium levels in young cattle egrets from nesting colonies in the northeastern United States, Puerto Rico and Egypt. *Arch Environ Contam Toxicol 23:435–439*

Calabrese, A., J. R. MacInnes, D. A. Nelson, and J. E. Miller. (1977). Survival and growth of bivalve larvae under heavy metal stress. *Mar. Biol. 41*:179-184.

Caldwell C.A., Arnold M.A., and Gould W.R. (1999). Mercury distribution in blood, tissues, and feathers of double-crested cormorant nestlings from arid-lands reservoirs in south central New Mexico pp 36-47

Caldwell, C.A., M.A. Arnold and W.R. Gould, (1999). Mercury distribution in blood, tissues and feathers of double-crested cormorant nestling from arid-lands reservoirs in south central New Mexico. Arch. Environ. Contam. Toxicol., 36: 456-461.

Caldwell, C.A., M.A. Arnold and W.R. Gould, (1999). Mercury distribution in blood, tissues and feathers of double crested cormorant nestling from arid-lands reservoirs in south central New Mexico. *Arch. Environ. Contam. Toxicol., 36: 456-461*.

Callister, S. M. and Winfrey, M. R. 1986. Microbial methylation of mercury in upper Wisconsin River sediments. *Water, Air and Soil Pollution 29*: 453-465.

Choi, S.-C., and R. Bartha. (1994). Environmental factors affecting mercury methylation in estuarine sediments. *Bull. Environ. Contam. Toxicol. 53*:805-812.

Clarkson, T. W. (1972). Recent advances in toxicology of mercury with emphasis on the alkyl mercurials. *Crit. Rev. Toxicol.* 203-234:.

Cohen, J.B., A.R. Barclay, A.R. Major and J.P. Fisher, (2000). Wintering greater scaup as biomonitors of metal contamination in federal wildlife refuges in the Long Island region. *Arch. Environ. Contam. Toxicol., 38: 83-92.*

Cohen, J.B., A.R. Barclay, A.R. Major and J.P. Fisher, (2000). Wintering greater scaup as biomonitors of metal contamination in federal wildlife refuges in the Long Island region. Arch. Environ. Contam. Toxicol., 38: 83-92.

Compeau, G. and R. Bartha. (1983). Effects of sea salt anions on the formation and stability of methylmercury. *Bulletin of Environmental Contamination and Toxicology 31*:486-493.

Compeau, G. and R. Bartha. (1985). Sulfate-reducing bacteria: principal methylators of mercury in anoxic estuarine sediment. *Appl. Environ. Microbiol. 50*: 498-502.

contaminated harbor sediments. *Aquatic Toxicology 3*:249-260.

Cope, W. G., J. G. Wiener, and R. G. Rada. (1990). Mercury accumulation in yellow perch in Wisconsin seepage lakes: relation to lake characteristics. *Environmental Toxicology and Chemistry* 9:931-940.

Cossa, D. and J. G. Rondeau. (1985). Seasonal, geographical and size-induced variability in mercury content of *Mytilus edulis* in an estuarine environment: a reassessment of mercury pollution level in the Estuary and Gulf of St. Lawrence. *Marine Biology 88*:43-49.

Cosson RP, Amiard J-C, Amisard-Triquet C (1988a) Trace elements in little egrets and flamingos of Camargue, France. *Ecotoxicol and Environ Safety 15:107–116*

Cosson RP, Amiard-Triquet C, Amiard J-C (1988b) Utilisation des plumes dans le recherche des sources de contamination des oiseaux par les elements traces: Cd, Cu, Hg, Pb, Se, et Zn, chez les

Craig, P.J., and P.A. Moreton. (1983). Total mercury, methyl mercury and sulphide in River Carron sediments. *Marine Pollution Bulletin 14*(11):408-411.

Craig, P.J., and P.A. Moreton. (1985). The role of speciation in mercury methylation in sediments and water. *Environmental Pollution Series B* 10:141-158.

Alexander. D. (1986). Selective recovery of gold and other metal ions from an algal biomass. *Environ. Sci. Technol.* 20: 206-208.

Darnell, D. W., B. Greene, M. T. Henzl, J. M. Hosea, R. A. McPherson, J. Sneddon, M.

Dauwe T, Bervoets L, Pinxten R, Blust R, Eens M (2003) Variation of heavy metals within and among feathers of birds of prey: effects of molt and external contamination. *Environ Pollut 124:429–436*

Dauwe, T., Bervoets, L., Pinxten, R., Blust, R., Eens, M., (2003). Variation of heavy metals within and among feathers of birds of prey: effects of molt and external contamination. Environmental Pollution 124: 429-436.

De Haan, H. (1992). Impacts of environmental changes on the biogeochemistry of aquatic humic substances. *Hydrobiologia* 229:59-71.

Denton, G.R.W., H. Marsh and G.E. Heinsohn (1980). The unusual metal status of the dugong Duging dugon. *Mar. Biol., 57: 201-219.*

DeSorbo, C. R., P. E. Nye, J. J. Loukmas and D. C. Evers. (2008). Assessing mercury exposure and spatial patterns in adult and nestling bald eagles in New York State, with an emphasis on the Catskill Region. Report BRI 2008-06 Submitted to The Nature Conservancy, Albany, New York. *Bio Diversity Research Institute, Gorham, Maine. 34 **xx-xx.***

Dukerschein, J. T., R. G. Rada, and M. T. Steingraeber. (1992). Cadmium and mercury in emergent mayflies (Hexagenia bilineata) from the upper Mississippi River. *Arch. Environ. Contam. Toxicol.* 23: 109-116.

Edward Ebow Kwaansa-Ansah, Anthony Apeke Adimado and James Hawkins Ephraim (2012). Distribution of mercury in water, sediment and fish from the Volta Lake and its major tributaries**.** *J.Environ. Occup. Sci. 1(1): 27-36*

Eganhouse, R.P. and D.R. Young. (1978). Total and organic mercury in benthic organisms near a major submarine wastewater outfall system. *Bulletin of Environmental Contamination and Toxicology* 19:758-766.

Eisler, R. (1987). *Mercury hazards to fish, wildlife, and invertebrates: a synoptic review*. U.S. Fish and Wildlife Service Biology Report 85(1.10). Washington, D.C.: U.S. Department of the Interior. p. 90.

Eisler, Ronald. (1987). Mercury Hazards to Fish, Wildlife, and Invertebrates: A Synoptic Review. Biological Report 85 (1.10). U.S. Fish and Wildlife Service.

EPA (environmental Protection Agency) (1997a) Water quality standards; Establishment of numeric criteria for priority toxic pollutants for the state of California; proposed rule,Fedral register August 5 1997, 62 (150): 42159-42208

EPA (environmental Protection Agency) (1997b). An ecological assessment of anthropogenic mercury emission in the United Statees Vol. 6 of mercury study report tto congressbU.S environmental protection agency Washington *EPA-452/r-97-103*

Evans, D.W. and D.W. Engel. (1994). *Mercury bioaccumulation in finfish and shellfish from Lavaca Bay, Texas: descriptive models and annotated bibliography*. NOAA Technical Memorandum NMFS-SEFSC-348.

Evers D.C., Wiener J.G., Driscoll C.T., Gay D.A., Basu N., Monson B.A.,Lambert K.F., Morrison H.A., Morgan J.J., Williams K.A., Soehl A.G. (2011). Great Lakes mercury connections; the extent and effects of mercury pollution in the Great Lakes Region. *Biodiversity Research Institute. Gorham, Maine Report BRI2011:18-44*

Evers DC, Burgess NM, Champoux L, Hoskins B, Major A, Goodale W, et al. (2005) Patterns and interpretation of mercury exposure in freshwater avian communities in northeastern North America. *Ecotoxicology 14:193-221*

Evers DC, Taylor KM, Major A, Taylor RJ, Poppenga RH, Scheuhammer AM (2003) Common loon eggs as indicators of methylmercury availability in North America. *Ecotoxicology 12:69-81*

Faust, B.C. (1992). The octanol/water distribution coefficients of methylmercuric species: the role of aqueous-phase chemical speciation. *Environ. Toxicol. Chem.* 11:1373-1376.

Feltier, J. S., E. Kahn, B. Salick, F. C. Van Natta, and M. W. Whitehouse. (1972). *Ann. Intern. Med.* 76:779-792.

Fimreite N. and L. Karstad (1971). Effects of dietary methylmercury on Red-tailed Hawks. *Journal of Wildilfe Management 35:293-300.*

Fimreite, N. (1974). Mercury contamination of aquatic birds in northwestern Ontario. J. Wildl. Manage. 38: 120-131.

Fisher, N.S., M. Bohé, and J.-L. Teyssié. (1984). Accumulation and toxicity of Cd, Zn, Ag, and Hg in four marine phytoplankters. *Mar. Ecol. Prog. Ser. 18*:201-213.

Fitzgerald, W.F. (1990). Mercury in seawater. In: *Mercury in the Marine Environment*. Workshop proceedings. U.S. Dept. Interior, Mineral Management Service, Anchorage,

Alaska. OCS Study MMS 89-0049. *flamants de Camargue, France. Water, Air Soil Pollut* 42:103-115

Fowler, S. W., M. Heyraud, and J. La Rosa, (1978). Factors affecting methyl and inorganic mercury dynamics in mussels and shrimp. *Marine Biology* 46: 267-276.

Francesconi, K. and R. C. J. Lenanton (1992). Mercury contamination in a semienclosed marine embayment: organic and inorganic mercury content of biota, and factors influencing mercury levels in fish. *Marine Environmental Research* 33: 189-212.

Frodello JP, Romeo M. and Viale D. (2000) Distribution of mercury in the organs and tissues of five toothed-whale species of the Mediterranean. *Environ. Pollut.* 108(3):447-452.

Furness, R. W. (1993). Birds as monitors of pollutants. In: Furness R.W. Greenwood J.J.D. (eds), Birda as monitors of environmental Change. *Chapman and Hall, London UK*, pp 86-143.

Furness, R.W. and J.J.D. Greenwood (1993). Birds as Monitors of Environmental Changes British Trust for Ornithology. 1st Edn. Thetford Norfolk, UK, p. 342.

Furness, R.W., Muirhead, S.J., Woodburn, M. (1986). Using bird feathers to measure mercury in the environment: relationships between mercury content and moult. *Marine Pollution Bulletin 17:27-30.*

Gadd, G. M. (1988). Accumulation of metals by microorganisms and algae. In: Rehm H-J and G. Reed (eds) *Biotechnology*. Vol 6b, 401-433.

Gentile, J. H., S. M. Gentile, G. Hoffman, J. F. Heltshe, and N. Hairston, Jr. (1983). The effects of a chronic mercury exposure on survival, reproduction and population dynamics of *Mysidopsis bahia*. *Environ. Toxicol. Chem. 2*:61-68.

Giblin, F. J. and E. J. Massaro. (1973). Pharmacodynamics of methyl mercury in rainbow trout (*Salmo gairdneri*): tissue uptake, distribution and excretion. *Toxicol. Appl. Pharmacol. 24*:81-91.

Gill, G. A. and K. W. Bruland. (1990). Mercury speciation in surface freshwater systems in California and other areas. *Environmental Science and Technology 24*(9): 1392-1400.

Gill, G.A. and W.F. Fitzgerald. (1987). Picomolar mercury measurements in seawater and other materials using stannous chloride reduction and two-stage gold amalgamation with gas phase detection. *Marine Chemistry 20*: 227-243.

Gilmour, C. C. and Capone, D. G. (1987). Relationship between Hg methylation and the sulfur cycle in estuarine sediments. *EOS 68*:1718.

Gilmour, C. C. and E. A. Henry. (1991). Mercury methylation in aquatic systems affected by acid deposition. E*nvironmental Pollution 71*(2-4): 131-169.

Gilmour, C.C., E.A. Henry, and R. Mitchell. (1992). Sulfate stimulation of mercury methylation in freshwater sediments. *Environ. Sci. Technol. 26* (11): 2281-2287.

Goede A.A. and De Bruin M. (1984). The use of bird feather parts as a monitor for metal pollution. *Environ. Poll. (B) 8: 281-298*

Goede AA, de Bruin M (1984). The use of bird feather parts as a monitor for metal pollution. *Environ Pollut 8:281–298*

Goutner, V., Furness, R.W., Papakostas, G., (2001). Mercury in feathers of Squacco Heron *(Ardeola ralloides)* chicks in relation to age, hatching order, growth, and sampling dates. *Environmental Pollution 111: 107-115.*

Greib, T.M., C.T. Driscoll, S. P. Gloss, C.L. Schofield, G.L. Bowie, and D.B. Porcella. (1990). Factors affecting mercury accumulation in fish in the upper Michigan peninsula. *Environmental Toxicology and Chemistry 9*:919-930.

Hall, A.S., F.M. Teeny, and E.J. Gauglitz. (1976a). Mercury in fish and shellfish of the northeast Pacific. II. Sablefish, *Anoplopoma fimbria*. *Fishery Bulletin 74*:791-797.

Hall, A.S., F.M. Teeny, L.G. Lewis, W.H. Hardman, and E.J. Gauglitz, Jr. (1976b). Mercury in fish and shellfish of the northeast Pacific. I. Pacific Halibut, *Hippoglossus stenolepis*. *Fishery Bulletin 74*:783-789.

Hall, B.D., R.A. Bodaly, R.J.P. Fudge, J.W.M. Rudd and D.M. Rosenberg, (1997). Food as the dominant pathway of methyl mercury uptake by fish. *Water Air. Soil. Pollut., 100: 13-24.*

Hansen, C. T., C. O. Nielsen, R. Dietz, and M. M. Hansen. (1990). Zinc, cadmium, mercury and selenium in Minke Whales, Belugas and Narwhals from West Greenland. *Polar Biology 10*:529-539.

Hansen, D. J. (1989). U.S. Environmental Protection Agency regulations and criteria for mercury in water. Summary presentation to the coordination team. In: *Mercury in the Marine Environment*. Workshop proceedings. U.S. Dept. Interior, Mineral Management Service, Anchorage, Alaska. OCS Study MMS 89-0049.

Hara, T.J., Y.M.C. Law, and S. McDonald. (1976). Effects of mercury and copper on the olfactory response in rainbow trout (*Oncorhynchus mykiss*). *J Fish Res Bd Can 33*:1568-1573.

Heisinger, F. J. and W. Green. (1975). Mercuric chloride uptake by eggs of the rice fish and resulting teratogenic effects. *Bull. Environ. Contam. Toxicol. 14*:665-673.

Henny CJ, Hill EF, Hoffman DJ, Spalding MG, Grove RA (2002) Nineteenth century mercury: hazard to wading birds and cormorants of the Carson River, Nevada. *Ecotoxicology 11:213-231*

Hill, W.R., A.J. Stewart, and G.E. Napolitano. (1996). Mercury speciation and bioaccumulation in lotic primary producers and primary consumers. *Can. J. Fish. Aquat. Sci. 53*:812-819.

Hines, M. E., S. L. Knollmeyer, and J.B. Tugel. (1989). Sulfate reduction and other sedimentary biogeochemistry in a northern New England salt marsh. *Limnol. Oceanogr. 34*(3): 578-590.

Hintelmann, H., P. M. Welbourn, and R. D. Evans. (1995). Binding of methylmercury compounds by humic and fulvic acids. *Water, Air, and Soil Pollution 80*:1031-1034.

Hoff, R., H. Curl, Jr., J. Farr, and N. Beckvar. (1994). *Empire Knight: Assessing environmental risk*. NOAA Technical Memorandum NOAA ORCA 81. Seattle: Hazardous Materials Response and Assessment Division, National Oceanic and Atmospheric Administration, p.3.

Hoffman, D.J., and J.M. Moore. (1979). Teratogenic effects of external egg applications of methyl mercury in the mallard, *Anas platyrhynchos*. *Teratology 20:453-462*.

Holden, A. V. (1978). Pollutants and seals-A review. *Mammal Rev. 8*(1-2):53-66.

Honda K, Nasu T, Tatsukawa R (1986) Seasonal changes in mercury accumulation in the black-eared kite, (*Milvus migrans lineatus*) *Environ Pollut 42: 325-334*

Honda, K., Nasu, T., Tatsukawa, R., (1986). Seasonal changes in mercury accumulation in the black-eared kite, Milvus migrans lineatus. *Environmental Pollution (A) 42, 325334.*

Horvat, M. (1991). Determination of methylmercury in biological certified reference materials. *Water, Air, and Soil Pollution 56*:95-102.

Huckabee, J., J. Elwood, and S. Hildebrand. (1979). Accumulation of mercury in freshwater biota. In: Nriagu (ed.) *The Biogeochemistry of Mercury in the Environment*. pp 277-302.

Hughes, W. L. (1957). A physicochemical rationale for the biological activity of mercury and its compounds. *Ann. New York Acad. Sci. 65*: 454-460.

Jackson, T. A. (1986). Methyl mercury levels in a polluted prairie river-lake system: seasonal and site specific variations, and the dominant influence of trophic conditions. *Canadian Journal of Fisheries and Aquatic Sciences 43*:1873-1877.

Jackson, T. A. (1987). Methylation, demethylation, and bio-accumulation of mercury in lakes and reservoirs of northern Manitoba, with particular reference to effects of environmental changes caused by the Churchill-Nelson River diversion. In: *Summary Report, Canada-Manitoba Agreement on the Study and Monitoring of Mercury in the Churchill River Diversion*. Ottawa: Governments of Canada and Manitoba pp 340-405.

Jackson, T. A. (1988). Accumulation of mercury by plankton and benthic invertebrates in riverine lakes of northern Manitoba (Canada): Importance of regionally and seasonally varying environmental factors. *Canadian Journal of Fisheries and Aquatic Sciences 45*:1744-1757.

Jackson, T. A. (1989). The influence of clay minerals, oxides, and humic matter on the methylation and demethylation of mercury by micro-organisms in freshwater environments. *Applied Organometallic Chemistry 3*:1-30.

Jackson, T.A. (1991). Biological and environmental control of mercury accumulation by fish in lakes and reservoirs of northern Manitoba, Canada. *Can. J. Fish. Aquat. Sci. 48*:2449-2470.

Jernelov, A. (1968). Laboratory Experiments regarding the conversion of mercury into its different forms of occurrence 1. *Vatten 24*(1):53-56.

Joiris, C. R., L. Holsbeek, J. M. Bouquegneau, and M. Bossicart. (1991). Mercury contamination of the harbour porpoise *Phocoena phocoena* and other cetaceans from the North Sea and the Kattegat. *Wat. Air Soil Pollut. 56*: 283-293.

Julshamn, K. O. Ringdal, and O. R. Braekkan. (1982). Mercury concentration in liver and muscle of Cod (*Gadus morhua*) as an evidence of migration between waters with different levels of mercury. *Bull. Environm. Contam. Toxicol. 29*:544-549.

Julshamn, K., A. Andersen, O. Ringdal, and J. Morkore. (1987). Trace elements intake in the Faroe Islands, I. Element levels in edible parts of Pilot Whales (*Globicephalus meleanus*). *Sci. Total Environ. 65*:53-62.

Kehrig H. (1998). Methylmercury in fish and hair samples from Balbina Reservoir, Brazilian amazon. environmental research, *v.77, p.84-90*.

Kehrig HA, Seixas TG, Palermo EA, Di Beneditto A.P.M., Souza CMM and Malm O. (2008). Different Species of Mercury in the Livers of Tropical Dolphins. Anal. Lett. 41(9): 1691-1699.

Kelly, C.A., J.W.M. Rudd, V.L. St. Louis, and A. Heyes. (1995). Is total mercury concentration a good predictor of methyl mercury concentration in aquatic systems? *Water, Air, and Soil Pollution 80*:715-724.

Khaniki, G.R.J., A. Inteaz, E. Nowroozi and R. Nabizadeh, (2005). Mercury contamination in fish and public health aspects: A review. *Pak. J. Nutr., 4:276-281.*

Kihlstrom, J. E. and L. Hulth. (1972). The effect of phenylmercuric acetate upon the frequency of hatching in the zebrafish. *Bull. Environ. Contam. Toxicol. 7*:111.

Kim E.Y, Ichihashi H., Saeki K., Atrashkevich G., Tanabe S., Tatsukawa R. (1996b). Metal accumulation in tissues of seabirds from Chaun, northeast Siberia. Russia. *Environ Pollut 92:247-252*

Kim E.Y., Murakami T., Saeki K., Tatsukawa R. (1996a) Mercury levels and its chemical form in tissues and organs of seabirds. *Arch Environ Contam Toxicol 30:259-266*

Kim, E.Y., T. Murakami, K. Sacki and R. Tatsukawa, 1996. Mercury levels and its chemical form in tissues and organs of seabirds. *Environ. Contam. Toxicol., 30: 256-266.*

Kinabo C. and Lyatuu H. (2009). Mercury contamination in domestic ducks in Geita Northwest Tanzania. *Tanz. J. Vol 35: 36-45*

Kobayashi, N. (1984). Marine ecotoxicological testing with echinoderms. In: G. Persoone, E. Jaspers, and C. Claus (Eds.), *Ecotoxicological Testing for the marine Environment*. Bredene, Belgium: State University Ghent and Institute Mar. Scient. Res. pp. 341-381.

Koeman J. H., Vink J.A.J.and De Goeij (1969). Causes of mortality in birds of prey and owls in the Netherlands in the winter of 1968-1969. *Ardea 57: 67-76*

Konieczka P, Misztal-Szkudlin´ska M, Namies´nik J, Szefer P (2010). Determination of total mercury in fish and cormorants using cold vapour atomic absorption spectrometry. *Polish J Environ Stud 19:931-936*

Kopfler, F. C. (1974). The accumulation of organic and inorganic mercury compounds by the eastern oyster (*Crassostrea virginica*). *Bull. Environ. Contam. Toxicol. 11*:275-280.

Korthals, E. T and M. R. Winfrey. (1987). Seasonal and spatial variations in mercury methylation and demethylation in an oligotrophic lake. *Applied and Environmental Microbiology 53*:2397- 2404.

Langston, W. J. 1982. The distribution of mercury in British estuarine sediments and its availability to deposit-feeding bivalves. *J. Mar. Biol. Ass. U.K. 62*: 667-684.

Langston, W. J. (1986). Metals in sediments and benthic organisms in the Mersey Estuary. *Estuarine, Coastal and Shelf Science 23*:239-261.

Langston, W. J. (1990). Chapter 7: Toxic effects of metals and the incidence of metal pollution in marine environments. In: R. W. Furness and P. S. Rainbow (Eds.), *Heavy Metals in the Marine Environment.* pp. 101-122. Boca Raton, Florida: CRC Press, Inc.

Lasorsa, B. and S. Allen-Gil. (1995). The methylmercury to total mercury ratio in selected marine, freshwater, and terrestrial organisms. *Water, Air, and Soil Pollution 80*:905-913.

Leah, R.T., S.J. Evans, and M.S. Johnson. (1992). Mercury in flounder (Platichthys flesus L.) from estuaries and coastal waters of the north-east Irish Sea. *Environmental Pollution 75*:317-322.

Lenka, M., K.K. Panda, and B. B. Panda. (1990). Studies on the ability of water hyacinth (Eichlornia crassipes) to bioconcentrate and biomonitor aquatic mercury. *Environ. Pollut. 66*: 89-99.

Leonzio, C., S. Focardi, and C. Fossi. (1992). Heavy metals and selenium in stranded dolphins of the Northern Tyrrhenian (NW Mediterranean). *Sci. Total Environ. 119*:77-84.

Lindberg P. (1984). Mercury in feathers of Swedish gyrfalcons in relation to diet. *Bull. Environ. Contam. Toxicology 32:453-459.*

Lindberg, S. E. and R.C. Harriss. (1974). Mercury-organic matter associations in estuarine sediments and interstitial water. *Envir. Sci. Tech. 8*(5):459-462.

Lindqvist, O. (1991). Mercury in the Swedish environment: Recent research on causes, consequences and corrective methods. *Water, Air, and Soil Pollution 55*(1- 2). Special Issue. p.261.

Long, E. R. and D. D. MacDonald. (1992). National Status and Trends Program Approach. In: *Sediment Classification Methods Compendium*. EPA 823-R-92-006. EPA Office of Water (WH-556). Washington, DC.: U.S. Environmental Protection Agency.

Long, E. R., D. D. MacDonald, S. L. Smith, and F. D. Calder. (1995). Incidence of adverse biological effects within ranges of chemical concentrations in marine and estuarine sediments. *Environmental Management 19*(1):81-97.

Luoma, S.N. (1977). The dynamics of biologically available mercury in a small estuary. *Estuarine Coast. Mar. Sci. 5*:643-652.

Major, M.A., D.H. Rosenblatt and K.A. Bostian. (1991). The octanol/water partition coefficient of methylmercuric chloride and methylmercuric hydroxide in pure water and salt solutions. *Environ. Toxicol. Chem. 1*0:5-8.

Mason, R.P., J.R. Reinfelder, and F.M.M. Morel. (1995b). Bioaccumulation of mercury and methylmercury. *Water, Air, and Soil Pollution 80*:915-921.

Mason, R.P., J.R. Reinfelder, and F.M.M. Morel. (1996). Uptake, toxicity, and trophic transfer of mercury in a coastal diatom. *Environ. Sci. Technol. 30*:1835-1845.

Mason, R.P., K.R. Rolfhus and W.F. Fitzgerald. (1995a). Methylated and elemental mercury cycling in surface and deep ocean waters of the North Atlantic. *Water, Air, and Soil Pollution 80*: 665-677.

Mathers, R. and P. Johansen. (1985). The effects of feeding ecology on mercury accumulation in walleye (*Stizostedion vitreum*) and pike (*Esox lucius*) in Lake Simcoe. *Canadian Journal of Zoology 63*: 2006-2012.

Matida, Y., H. Kumada, S. Kimura, Y. Saiga, T. Nose, M. Yokote, and H. Kawatsu. (1971). Toxicity of mercury compounds to aquatic organisms and accumulation of the compounds by the organisms. *Bull. Freshwater Fish. Res. Lab. (Tokyo) 21*:197-227.

Mautino M. and Bell J.U. (1987). Experimental Lead Toxicity in the Ring-necked Duck.

Mazloomi S, Esmaeili A, Ghasempoori SM, Omidi A (2008) Mercury distribution in liver, kidney, muscle and feathers of Caspian Sea common cormorant (*Phalacrocorax carbo). Res J Environ Sci. 2:433-437*

McGreer, E. R. (1979). Sublethal effects of heavy metal contaminated sediments on the bivalve *Macoma balthica* (L). *Mar. Pollut. Bull. 10*:259.

McKenney, C. L., Jr. and J. D. Costlow, Jr. (1981). The effects of salinity and mercury on developing megalopae and early crab stages of the blue crab *Callinectes sapidus* Rathbun. In. F. J. Vernberg, A. Calabrese, F. P. Thurberg, and W. B. Vernberg (Eds.), *Biological Monitoring of Marine Pollutants*. pp. 241-262. New York: Academic Press.

McKim, J.M., G. F. Olson, G. W. Holcombe, and E.P. Hunt. (1976). Long-term effects of methylmercuric chloride on three generations of brook trout (*Salvelinus fontinalis*): Toxicity, accumulation, distribution, and elimination. *Journal Fisheries Research Board of Canada 33*:2726-2739.

Meili, M.. (1995). Liming effects on mercury concentrations in fish. In: L. Henrikson and Y. W. Brodin (eds). *Liming Acidified Sur. Waters*. pp. 383-398. Berlin: Springer.

Mierle, G., and R. Ingram. (1991). The role of humic substances in the mobilization of mercury from watersheds. *Water, Air, and Soil Pollution 56*:349-357.

Miskimmin, B. M., J. W. M. Rudd, and C. Kelly. (1992). Influence of dissolved organic carbon, pH, and microbial respiration rates on mercury methylation and demethylation in lake water. *Can. J. Fish. Aquat. Sci. 49*:17-22.

Møhlenberg, F. and H.U. Riisgård. (1988). Partitioning of inorganic and organic mercury in cockles Cardium edule (L.) and C. Glaucum (Bruguiere) from a chronically polluted area: influence of size and age. *Environ. Pollut. 55*:137-148.

Monitoring of Marine Pollutants. pp. 357-376. New York: Academy Press.

Mora M, Skiles R, McKinney B, Paredes M, Buckler D, Papoulias D, and Kleine D. (2002) Environmental contaminants in prey and tissues of the peregrine falcon in the Big Bend Region, Texas, USA. *Environmental Pollution 116: 169-176.*

Morel, F. M. M., A. M. L. Kraepiel, and M. Amyot. (1998). The chemical cycle and bioaccumulation of mercury. Annual Review of Ecology and Systematics 29:543–566.

Movalli P.A. (2000). Heavy metal and other residues in feathers of lagger falcons from six districts of Pakistan. *Environmental Pollution 109:267-275*.

Nam D.H., Anan Y., Ikemoto T., Okabe Y, Kim E. Y., Subramanian A. Saeki K and Tanabe S (2005). Specific accumulation of 20 trace elements in great cormorants *(phalacrocorax carbo)* from Japan. *Environ. Pollut. 134: 503-514.*

NAS. (1978). An assessment of mercury in the environment. Natl. *Acad. Sci., Washington, DC.p.185* .

National Fisheries Association of Ghana (NAFAG). (2004-2005), also *available at: http://www.nafagfish.org/aboutus.htm*

Nelson BJ (2005) Pelicans, cormorants and their relatives: *Pelecanidae, Anhingidae, Fregatidae, Phaethontidae.* Oxford University Press, Oxford

Nicoletto, P. and A. Hendricks. (1987). Sexual differences in accumulation of mercury in four species of centrarchid fishes. *Canadian Journal of Zoology 66*:944- 949.

Niimi, A.J. and G.P. Kissoon. (1994). Evaluation of the critical body burden concept based on inorganic and organic mercury toxicity to rainbow trout (*Oncorhynchus mykiss*). *Arch. Environ. Contam. Toxicol. 26*:169-178.

Nishimura H., and M. Kumagai. (1983). Mercury pollution of fishes in Minamata Bay and surrounding water: analysis of pathway of mercury. *Water, Air, and SoilPollution 20*:401-411.

Ochoa-acuna H, Sepu´lveda MS, Gross TS (2002) Mercury in feathers from Chilean birds: influence of location, feeding strategy, and taxonomic affiliation. *Mar Pollut Bull 44:340-349*

Ohlendorf H.M. Risebrough R.W. and Vermeer K. (1978). Exposure of marine birds to environmental pollutants (Wildl Rep. 9) US Dept. Interior Fish and Wildlife Service Washington D.C p. 15.

Olson, B.H. and R.C. Cooper. (1976). Comparison of aerobic and anaerobic methylation of mercuric chloride by San Francisco Bay sediments. *Water Res. 10*: 113-116.

Olson, K R., K. S. Squibb, and R. J. Cousins. (1978). Tissue uptake, subcellular distribution, and metabolism of 14CH_3HgCl and CH3203 HgCl by rainbow trout, *Salmo gairdneri*. *J. Fish. Res. Board Can. 35*:381-390.

Paper 56:1-25

Parks, J. W., A. Lutz, and J. A. Sutton. (1989). Water column methylmercury in the Wabigoon/English River-Lake System: Factors controlling concentration, speciation, and net production. *Canadian Journal of Fisheries and Aquatic Sciences 46*:2184-2202.

Parslow J.L.F., Jeffries D. J. and Hason H.M. (1973) Gannet mortality incidents in 1972. *Marine Poll. Bull. 4(1):85-94.*

Passow, H., A. Rothstein, and T. Clarkson. (1961). The general pharmacology of the heavy metals. *Pharmacol. Rev. 13*:185-224.

Shrum P. H. (2009) analysis Of Mercury And Lead In Birds Of Prey From Goldmining Areas Of The Peruvian Amazon. A Thesis Presented to the Graduate School of Clemson University pg 12-13

Pentreath, R.J. (1976a). The accumulation of organic mercury from sea water by the plaice, *Pleuronectes platessa L. Journal of Experimental Marine Biology and Ecology 24*:121-132.

Pentreath, R.J. (1976b). The accumulation of inorganic mercury from sea water by the plaice, *Pleuronectes platessa L. Journal of Experimental Marine Biology and Ecology 24*:103-119.

PTI. (1991). Onondaga Lake RI/FS Work Plan. PTI Environmental Services, Bellevue, Washington. PTI. 1988. *Briefing report to the EPA Science Advisory Board: The Apparent Effects Threshold approach*. Seattle: Environmental Protection Agency, Region 10, Office of Puget Sound. P.57.

Rada, R. G., J. E. Findley, and J. G. Wiener. (1986). Environmental fate of mercury discharged into the upper Wisconsin River. *Water Air Soil Pollut. 29*:57-76.

Rada, R. G., J. E. Findley, and J. G. Wiener. (1989b). Environmental fate of mercury discharged into the upper Wisconsin River. *Water, Air, and Soil Pollution 29*:57-76.

Rada, R. G., J. G. Wiener, M. R. Winfrey, D. E. Powell. (1989a). Recent increases in atmospheric deposition of mercury to north-central Wisconsin lakes inferred from sediment analyses. *Arch. Environ. Contam. Toxicol. 18*:175-181.

Rajaei F, Esmaili Sari A, Savabieasfahani M, Bahramifar N, Ghasempouri M (2011) Liver and breast feathers mercury in piscivorous birds of the Caspian Sea: monitoring changes. *Bull Environ Contam Toxicol 86:521-524*

Ramlal, P. S., J.W.M. Rudd, A. Furutani, L. Xun. (1985). The effect of pH on methyl mercury production and decomposition in lake sediments. *Canadian Journal of Fisheries and Aquatic Sciences 4*2: 685-692.

Regnell, O. (1994). The effect of pH and dissolved oxygen levels on methylation and partitioning of mercury in freshwater model systems. *Environmental Pollution 84*:7- 13.

Rehnberg, B.C. and C.B. Schreck. (1986). Acute metal toxicology of olfaction in coho salmon: behavior, receptors, and odor-metal complexation. *Bull. Environ. Contam. Toxicol. 36*:579-586.

Reigel, D. V. (1990). *The distribution and behavior of mercury in sediments and marine organisms of Lavaca Bay, Texas*. College Station, Texas: The Texas A&M University. Masters Thesis.

Richardson, G.M. and D.J. Currie. (1996). Does acid precipitation exacerbate the problem of fish mercury contamination? *SETAC News 16*(2):14.

Riisgård , H. U. and P. Famme. (1986). Accumulation of inorganic and organic mercury in shrimp, *Crangon crangon*. *Marine Pollution Bulletin 17*:255-257.

Riisgård , H. U. and S. Hansen. (1990). Biomagnification of mercury in a marine grazing food-chain: algal cells *Phaeodactylum tricornutum*, mussels *Mytilus edulis* and flounders *Platichthys flesus* studied by means of a stepwise-reduction-CVAA method. *Marine Ecology Progress Series 62*:259-270.

Riisgård , H.U. and P. Famme. (1988). Distribution and mobility of organic and inorganic mercury in flounder, Platichthys flesus, from a chronically polluted area. *Toxicol. Environ. Chem. 16*:219-228.

Riisgård, H.U., T. Kiorboe, F. Møhlenberg, I. Drabk, and P. Pheiffer Madsen. (1985). Accumulation, elimination and chemical speciation of mercury in the bivalves Mytilus edulis and Macoma balthica. *Mar. Biol. 86*:55-62.

Rodgers, D.W. and F. W. H. Beamish. (1981). Uptake of waterborne methylmercury by rainbow trout (*Salmo gairdneri*) in relation to oxygen consumption and methylmercury concentration. *Canadian Journal of Fisheries and Aquatic Sciences 38*(11): 1309-1315.

Rodgers, D.W. and F.W.H. Beamish. (1982). Dynamics of dietary methylmercury in rainbow trout, Salmo gairdneri. *Aquatic Toxicology 2*: 271-290.

Roesijadi, G., A.S. Drum, and J.R. Bridge. (1981). Mercury in mussels of Bellingham Bay, Washington (U.S.A.): the occurrence of mercury-binding proteins. In: Vernberg, F.J., Calabrese, A. Thurberg, F.P. and W.B. Vernberg (Eds.), *Biological*

Rubinstein, N.I., E. Lores, and N.R. Gregory. (1983). Accumulation of PCBs, mercury

Rudd, J. W. M. and M. A. Turner. (1983). The English-Wabigoon River System: V. Mercury and selenium bioaccumulation as a function of aquatic primary productivity. *Canadian Journal of Fisheries and Aquatic Sciences 40*:2251-2259.

Rudd, J. W. M., M. A. Turner, A. Furutani, A. L. Swick, and B. E. Townsend. (1983). The English-Wabigoon River System: I. A synthesis of recent research with a view towards mercury amelioration. *Canadian Journal of Fisheries and Aquatic Sciences 40*:2206-2217.

Rumbold, D.G., S.L. Niemczyk, L.E. Fink, T. Chandraesekhax, B. Harkanson and K.A. Lane, (2001). Mercury in eggs and feathers of great egrets (Ardea a1hus) from Florida Everglades. Arch. Environ. Contam. Toxicol., 41:501-507.

Rumbold, D.G., S.L. Niemczyk, L.E. Fink, T. Chandraesekhax, B. Harkanson and K.A. Lane, (2001). Mercury in eggs and feathers of great egrets *(Ardea a1hus)* from Florida Everglades. *Arch. Environ. Contam. Toxicol., 41:501-507.*

Saeki K, Okabe Y, Kim EY, Tanabe S, Fukuda M, Tatsukawa R (2000) Mercury and cadmium in common cormorants *(Phalacrocorax carbo). Environ Pollut 108:249-255*

Saeki K, Tanabe S (2005) Specific accumulation of 20 trace elements in great cormorants *(Phalacrocorax carbo)* from Japan. *Environ Pollut 134:503-514*

Saeki, K., Okabe, Y., Kim, E.-Y., Tanabe, S., Fukuda, M. and Tatsukawa, R., (2000). Mercury and cadmium in common cormorants *(Phalacrocorax carbo). Environmental Pollution 108: 249-255.*

Saeki, K., Y. Okabe, E.Y. Kim, S. Tanabe, M. Fukuda and R. Tatsukawa, (2000). Mercury and cadmium in common cormorants *(Phalacrocorax carbo). Environ. Pollut., 108: 249-255*.

Saouter, E. F. Ribeyre, A. Boudou, and R. Maury-Brachet. (1991). *Hexagenia rigida* (Ephemeroptera) as a biological model in aquatic ecotoxicology: experimental studies on mercury transfers from sediment. *Environmental Pollution 69*:51-67.

Saouter, E., L. Hare, P. G. C. Campbell, A. Boudou, and F. Ribeyre. (1993). Mercury

Saroff, S. T. (1990). *Proceedings of the Onondaga Lake Remediation Conference*. Bolton Landing, New York: New York State Department of Law and New York State Department of Environmental Conservation. p.193.

Sastry, K. V. and K. Sharma. (1980). Effects of mercuric chloride on the activities of brain enzymes in a freshwater teleost, *Ophiocephalus (Channa) punctatus*. *Arch. Environ. Contam. Toxicol. 9*:425-430.

Scherer, E., F. A. J. Armstrong, and S. H. Nowak. (1975). *Effects of mercurycontaminated diet upon walleyes Stizostedion vitreum vitreum (Mitchell)*. Fish. Mar. Serv. Tech. Rep. No. 597. Winnipeg, Manitoba: Fisheries Marine Service. p.21.

Scheuhammer A. M.(1991) Effeects of acidification on the availability of toxic metals and calcium to wild birds and mammals. *Environ. Poll. 71:329-375*

Schindler, D.W., S.E. Bayley, P.J. Curtis, B.R. Parker, M.P. Stanton and C.A. Kelly. (1992). Natural and man-caused factors affecting the abundance and cycling of dissolved organic substances in precambrian shield lakes. *Hydrobiologia 229*:1-21.

Schintu, M., F. Jean-Caurant, and J. C. Amiard. (1992). Organomercury determination in biological reference material: application to a study on mercury speciation in marine mammals off the Faröe Islands. *Ecotoxicology and Environmental Safety 24*:95-101.

Sharp, J. R. and J. M. Neff. (1980). Effects of the duration of exposure to mercuric chloride on the embryogenesis of the estuarine teleost, *Fundulus heteroclitus*, *Mar. Environ. Res. 3*: 195-213.

Shoichi, O. and S. Sokichi. (1985). The 1-octanol/water partition coefficient of mercury. *Bull. Chem. Soc. Japan. 58*:3401-3402.

Slooff, W., P.F.H. Bont, M. van Ewijk, and J.A. Janus. (1991). *Exploratory report mercury*. Report no. 710401006. Bilthoven, The Netherlands: National Institute of Public Health and Environmental Protection .

Snarski, V. M. and G. F. Olson. (1982). Chronic toxicity and bioaccumulation of mercuric chloride in the fathead minnow (*Pimephales promelas*). *Aquatic Toxicology* 2:143-156.

Solonen, T. and M. Lodenius. (1990). Feathers of birds of prey as indicators of mercury contamination in southern Finland. *Holarctic. Ecol., 13: 229-237*

Sorensen N.,Murata K., Budtz-Jorjensson E.,Weihe P. and Grandjean P. (1999). Prenatal methylmercury exposure as a cardiovascular risk factor at seven years of age. *Epidemiol 10: 370-375*

Spalding M.G., R.B. Bjork, G. Powel and S.F. Sundlof (1994). Mercury and causes of death in great white herons. *J. Wildl. Manag 58(4):735-739*

Spry, D. J. (1991). Metal bioavailability and toxicity to fish in low-alkalinity lakes: a critical review. *Environ. Pollut. 71*:243-304.

Spry, D. J. and J. G. Wiener. (1991). Metal bioavailability and toxicity to fish in lowalkalinity lakes: A critical review. *Environmental Pollution 71*:243-304.

St. Louis, V.L., J.W.M. Rudd, C.A. Kelly, K.G. Beaty, N.S. Bloom and R.J. Flett. 1994.Importance of wetlands as sources of methyl mercury to boreal forest ecosystems. *Can. J. Fish. Aquat. Sci. 51*:1065-1076.

Stewart FM, Phillips RA, Catry P, Furness RW (1997) Influence of species, age and diet on mercury concentrations in Shetland seabirds. *Mar Ecol Prog Ser 151:237–244*

Stewart, F.M., Phillips, R.A., Catry, P., Fumess, R.W., (1997). Influence of species, age and diet on mercury concentrations in Shetland seabirds. *Marine Ecology Progress Series 151,237-244.*

Storelli A. and Marcotrigiano G.O. (2005) Accumulation of mercury, cadmium, lead and arsenic in swordfish and bluefin tuna from the Mediterranean Sea: A comparative study *Mar. Pollut. Bull. 50(9): 1004-1007.*

Storelli M.M., Giacominelli-stuffler R., Storelli A. and Marcotrigiano G. O. (2005). Accumulation of mercury, cadmium, lead and arsenic in swordfish and bluefin tuna from the Mediterranean Sea. A comparative study. *Mar. Pollut. Bull. 50(9): 1004 - 1007*

Stromgren, T. (1982). Effect of heavy metals (Zn, Hg, Cu, Cd, Pb, Ni) on the length growth of *Mytilus edulis*. *Mar. Biol. 72*:69-72.

Sundlof S.F., Spalding M.G., Wentworth J. D. and Steible C.K. (1994). Mercury in livers of wading birds (*ciconiiformes)* in southern Florida. *Arch. Contam.Toxicol. 27:299-305*

Sundlof SF, Spalding MG, Wentworth JD, Steible CK (1994) Mercury in the livers of wading birds (Ciconiiformes) in southern Florida. *Archives of Environmental Contamination and Toxicology 27:299-305*

Sundlof, S. F., Spalding, M. G., Wentworth, J. D., and Steible, C. K. (1994). Mercury in livers of wading birds (Ciconiiformes) in southern Florida. Arch. Environ. Contam. Toxicol. 27: 299-305.

Surma-Aho, K. and J. Paasivirta. (1986). Organic and inorganic mercury in the food chain of some lakes and reservoirs in Finland. *Chemosphere 15*(3):353-372.

Swartz, R. C., P. F. Kemp, D. W. Schults, and J. O. Lamberson. (1988). Effects of mixtures of sediment contaminants on the marine infaunal amphipod, *Rhepoxynius abronius*. *Environmental Toxicology and Chemistry 7*:1013-1020.

Syversen, T. (1977). Effects of methylmercury on in vivo protein synthesis in isolated cerebral and cerebellar neurons. *Neuropathol. Appl. Neurobiol. 3*:225-236.

Szefer. P., W. Czarnowski, J. Pempkowiak, and E. Holm. (1993). Mercury and major essential elements in seals, penguins, and other representative fauna of the Antarctic. *Arch. Environ. Contam. Toxicol. 25*:422-427.

Tessier, L., G. Vaillancourt, and L. Pazdernik. (1994). Temperature effects on cadmium and mercury kinetics in freshwater molluscs under laboratory conditions. *Arch. Environ. Contam. Toxicol. 2*6:179-184.

Tetra Tech, Inc. (1988). *Health risk assessment of chemical contamination in Puget Sound seafood*. Seattle: Environmental Protection Agency, Region 10, Office of Puget Sound pp 20-35.

Thain, J. E. (1984). Effects of mercury on the prosobranch mollusc *Crepidula fornicata*: Acute lethal toxicity and effects on growth and reproduction of chronic exposure. *Mar. Envir. Res. 12*:285-309.

Thompson D. (1996). Mercury in birds and terrestrial mammals in Beyer W.N. Heinz G.H., Redmon A. (eds) interpreting environmental contaminants in animal tissues. *Lewis Boca Raton Fl USA pp.341-356*

Thompson D.R. and Furness R. W. (1989). Comparison of the levels of total and organic mercury in seabird feathers. *Marine poll. Bull 20: 577-579*

Thompson DR, Stewart FM, Furness RW (1990). Using seabirds to monitor mercury in marine environments: the validity of conversion ratios for tissue comparison. *Marine Pollution Bulletin*

Thompson, D. R. (1990). Metal levels in marine vertebrates. In: W. D. Furness and P. S. Rainbow (Eds.), *Heavy Metals in the Marine Environment*. pp. 144-182. Boca Raton, Florida: CRC Press.

Thompson, D.R., Furness, R.W., Barrett, R.T., 1992b. Mercury concentrations in seabirds from colonies in the Northeast Atlantic. *Archives of Environmental Contamination and Toxicology 23:383-389.*

Thompson, D.R., Furness, R.W., Walsh, P.M., 1992a. Historical changes in mercury concentrations in the marine ecosystem of the north and north-east Atlantic Ocean as indicated by seabird feathers. *Journal of Applied Ecology 29:74-84.*

U.S. Environmental Protection Agency (EPA). (1985). *Ambient water quality criteria for mercury - 1984*. U.S. EPA 440/5-84-026.

U.S. Environmental Protection Agency (EPA). (1996). Update: National listing of fish and wildlife consumption advisories. EPA Fact Sheet EPA-823-F-96-006.

U.S. Food and Drug Administration (FDA). (1984). Action level for methyl mercury in fish. *Federal Register 49*:45-663.

USDI, (1998). Guidelines for Interpretation of the Biological Effects of Selected Constituents in Biota, Water, and Sediment. National Irrigation Water Quality Program Information Report 3, 1998.Denver, Bureau of Reclamation.

Van der Molen, E.J., A.A. Blok, and G.J. De Graaf. (1982). Winter starvation and mercury intoxication in grey herons *(Ardea cinerea)* in the Netherlands. *Ardea 70:173-184.*

Veiga, Marcello M.; Hinton, Jennifer; Lilly, Cameron. (1999). Mercury in the Amazon: A Comprehensive Review with Special Emphasis on Bioaccumulation Bioindicators. Proceedings of National Institute for Minamata Disease, Forum 1999. p 19-39.

Wagemann R. Trebacz E., Boila G., Lockhart W.L. (1998) Methylmercury and total mercury in tissues of arctic marine mammals. *Sci. Total Environ. 218:1931.*

Wagemann, R., N. B. Snow, A. Lutz, and D. P. Scott. (1983). Heavy metals in tissues and organs of the narwhal (*Monodon monoceros*). *Can. J. Fish. Aquat. Sci. 40* (Suppl. 2):206-214.

Ware R.A.,Burkholder P.M. and Chang L.W. (1975) Ultrastructural changes in renal proximal tubule after chronic organic and inorganic mercury intoxication. *Environ. Res 10:121-140.*

Weber, J. H. (1993). Review of possible paths for abiotic methylation of mercury (II) in the aquatic environment. *Chemosphere 26*(11):2063-2077.

Weech SA, Scheuhammer AM, Elliott JE (2006) Mercury exposure and reproduction in fish-eating birds breeding in the Pinchi Lake region, British Columbia, Canada. *Environ Toxicol Chem 25:1433-1440.*

Weis, J. S, and P. Weis. (1989). Effects of environmental pollutants on early fish development. *Rev. Aquat. Sci. 1*:45-73.

Weis, J. S., P. Weis, and M. Heber. (1982). Variation in response to methylmercury by killifish (*Fundulus heteroclitus*) embryos. In: J.G. Pearson, R.B. Foster, and W.E. Bishop (eds.), *Aquatic Toxicology and Hazard Assessment: Fifth Conference*. ASTM STP 766. pp. 109-119. Philadelphia: American Society for Testing and Materials.

Weis, J. S. and P. Weis. (1977). Effects of heavy metals on development of the killifish, *Fundulus heteroclitus*. *J. Fish. Biol. 11*:49-54.

Weis, J. S. and P. Weis. (1984). A rapid change in methylmercury tolerance in a population of killifish, *Fundulus heteroclitus*, from a golf course pond. *Marine Environmental Research 13*:231-245.

Weis, J. S., P. Weis, M. Heber, and S. Vaidya. (1981). Methylmercury tolerance of killifish (*Fundulus heteroclitus*) embryos from a polluted vs non-polluted environment. *Marine Biology 65*:283-287.

Weis, P., J. S. Weis, and J. Bogden. (1986). Effects of environmental factors on release of mercury from Berry's Creek (New Jersey) sediments and its uptake by Killifish *Fundulus heteroclitus*. *Environmental Pollution 40*:303-315.

Weis, P. (1984). Metallothionein and mercury tolerance in the killifish, *Fundulus heteroclitus*. *Marine Environmental Research 14*:153-166.

Weis, P., and J.S. Weis. 1978. Methylmercury inhibition of fin renereration in fishes and its interaction with salinity and cadmium. *Estuarine Coastal Mar. Sci. 6*:327- 334.

Whitney, S. D. (1991). *Effects of maternally-transmitted mercury on the hatching success, survival, growth, and behavior of embryo and larval walleye (Stizostedion vitreum vitreum)*. Masters Thesis. La Crosse, Wisconsin: University of Wisconsin. 71 pp.

Wiener, J. G. and D. J. Spry. (1996). Toxicological significance of mercury in freshwater fish. In: Beyer, W.N., G.H. Heinz, and A.W. Redmon-Norwood (eds.).

Environmental Contaminants in Wildlife: Interpreting Tissue Concentrations. Special Publication of the Society of Environmental Toxicology and Chemistry. Boca Raton, Florida: Lewis Publishers. 494 pp.

Wiener, J. G., D. P. Krabbenhoft, G. H. Heinz, and A. M. Scheuhammer. (2003). Ecotoxicology of mercury in D. J. Hoffman, B. A. Rattner, G. A. *Burton, Jr., and J. Cairns, Jr.* Pages 409-463

Windom, H.L. and D. R. Kendall. (1979). Accumulation and biotransformation of mercury in coastal and marine biota. In: J.O. Nriagu (ed.) *The Biogeochemistry of Mercury in the Environment*. pp 277-302.

Winfrey, M. R. and J. W. Rudd. (1990). Environmental factors affecting the formation of methylmercury in low pH lakes. *Environ. Toxicol. Chem. 9:853-869.*

Wobeser, G. (1975). Prolonged oral administration of methyl mercury chloride to rainbow trout (*Salmo gairdneri*) fingerlings. *J. Fish.Res.Board Can. 32:2015-2023*.

Wolfe M.F. Schwarzbach S, Sulaiman R.A. (1998). Effects of mercury on wildlife; a comprehensive review. *Environmental toxicology and Chemistry 17:146-160.*

Wolfe MF, Schwarzbach S, Sulaiman RA (1998) Effects of mercury on wildlife: a comprehensive review. *Environmental Toxicology and Chemistry 17:146-160.*

Wood P.B., White J.H., Steffer A, Wood J.M., Facemire C.F. and Percival H.F. (1996). Mercury concentrations in tissues of Florida Bald eagles. *J Wildl. Manag. 60: 178-185*

Wood PB, White JH, Steffer A, Wood JM, Facemire CF, and Percival HF. (1996). Concentrations in tissues of Florida Bald Eagles. Journal of Wildlife Management 60:178-185.

World Health Organization (WHO). (1989). Mercury: Environmental Aspects. International Programme on Chemical Safety (IPCS). Geneva, Switzerland. 115 pp.

Wren, C. D. (1986). A Review of Metal Accumulation and Toxicity in Wild Mammals, I: Mercury. *Environmental Research 40:210 -244.*

Xun, L., N.E.R. Campbell, and J.W.M. Rudd. (1987). Measurements of specific rates of net methyl mercury production in the water column and surface sediments of acidified and circumneutral lakes. *Can. J. Fish. Aquat. Sci. 44*:750-757.

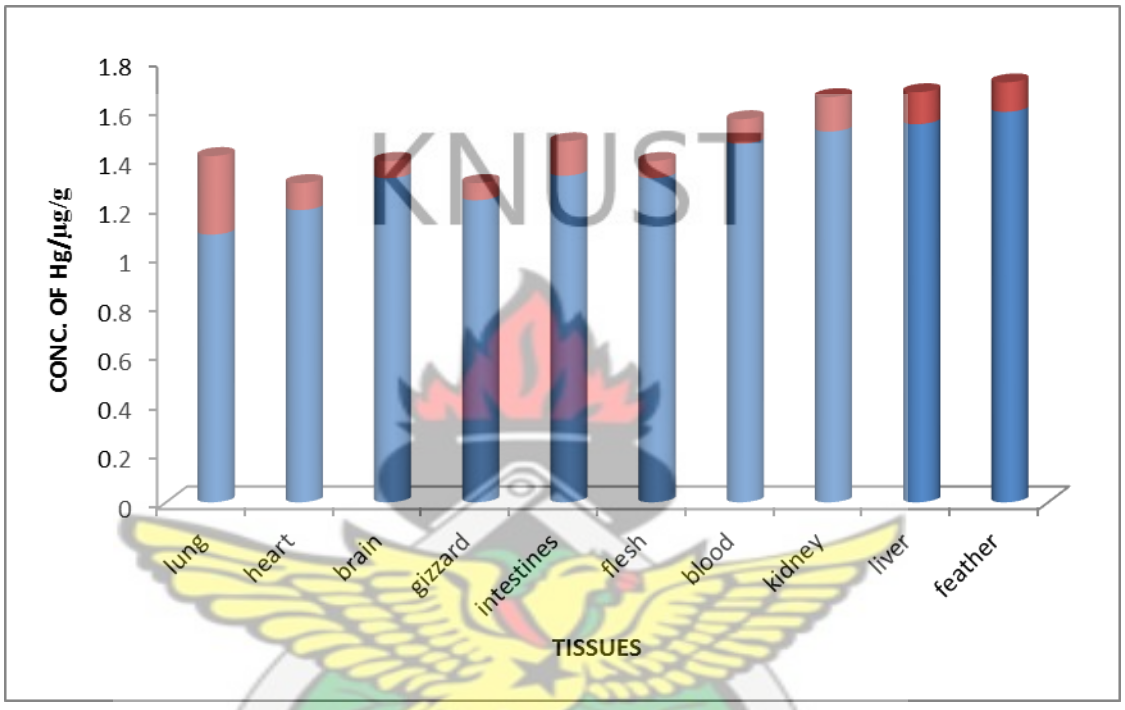
Zillioux, E. J., D. B. Porcella, and J. M. Benoit. (1993). Mercury cycling and effects in freshwater wetland ecosystems. *Environmental Toxicology and Chemistry 12*:1-20.

Zolfaghari G, Esmaili-Sari A, Ghasempouri SM, Kiabi BH. (2007). Environmental Research Vol 104 Issue 2 258-265.

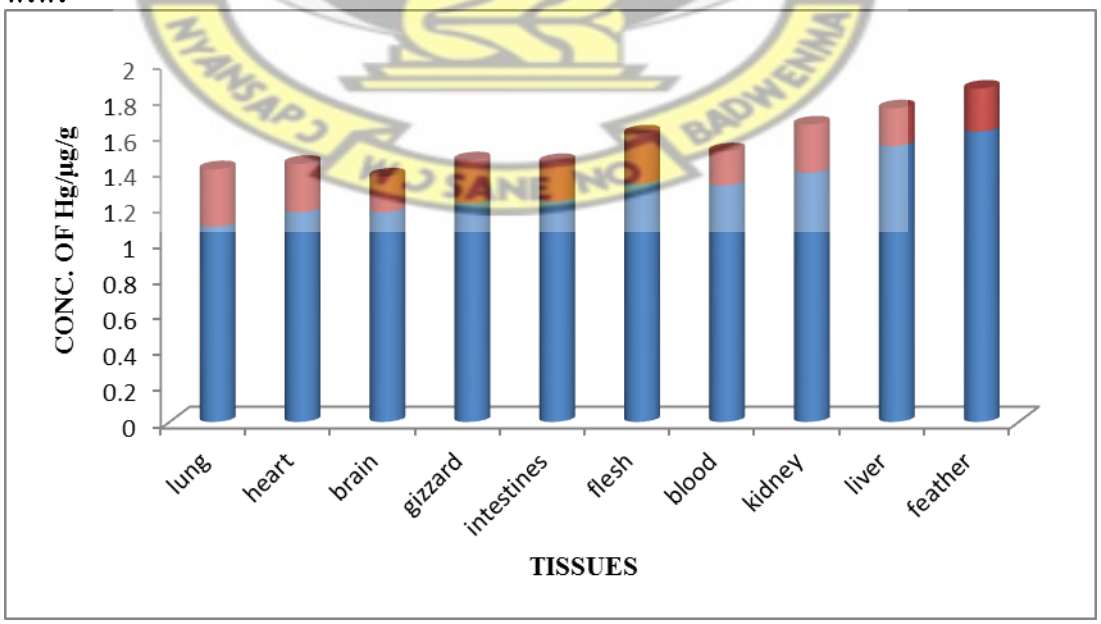
APPENDICES

APPENDIX I: GRAPH SHOWING THE LEVELS OF TOTAL MERCURY IN THE TISSUES OF THE BIRDS

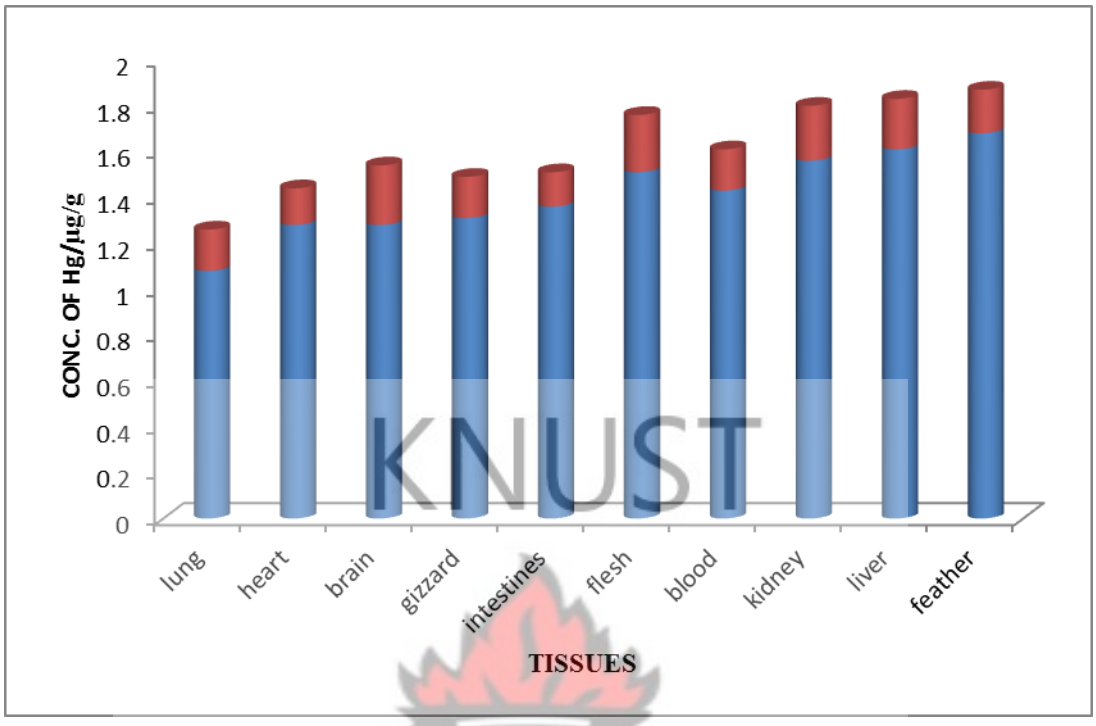
Levels of mercury (mean+SD) in the tissues of *Egretta garzetta* of in µg/g w.w.



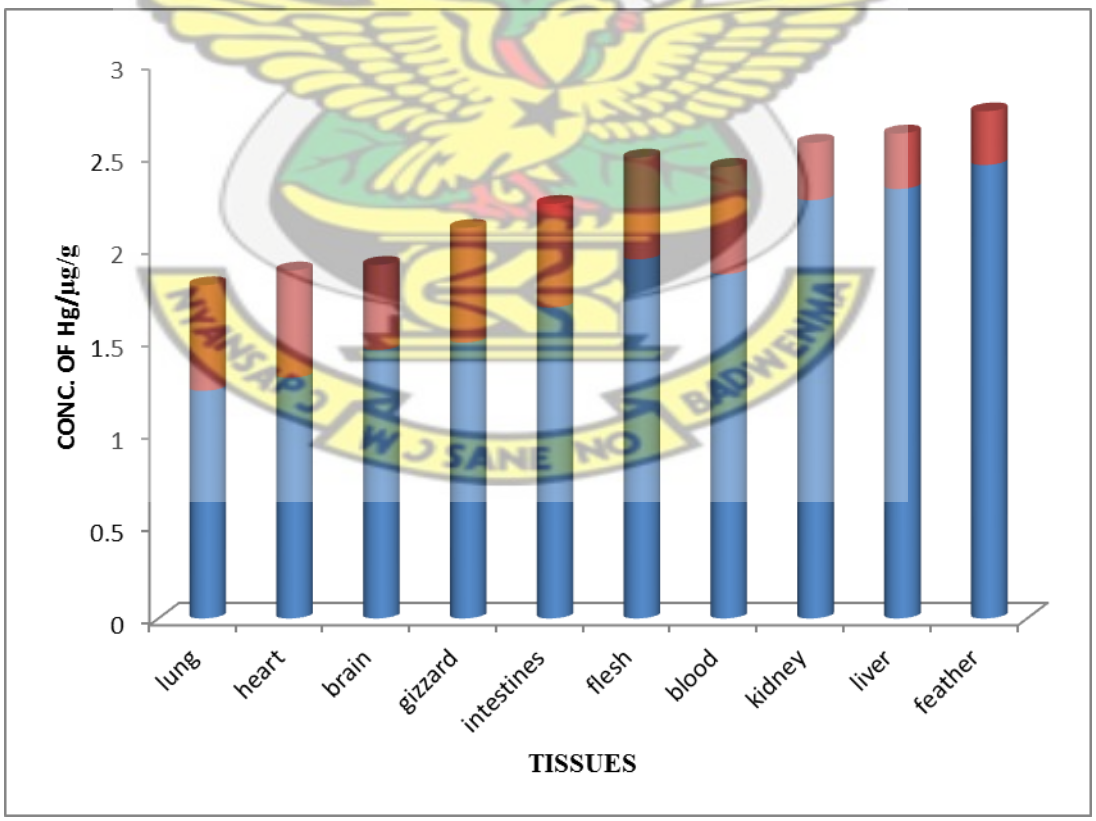
Levels of mercury (mean +SD) in the tissues of *Milvus migrans parasitus* of in µg/g w.w.



Levels of mercury (mean +SD) in the tissues of *Squacco heron* of in µg/g w.w.



Levels of mercury (mean+SD) in the tissues of *Ardea cinerea* of in µg/g w.w.



APPENDIX 2: SPECIES OF BIRDS SAMPLED FOR THE STUDY

Yellow billed kite

(Milvus migrans parasitus)

Little egret

(Egretta garzetta)

Squacco heron

(*Ardeola ralloides*)

Grey heron

(*Ardea cinerea*)