CHAPTER ONE
INTRODUCTION

1.1 Background to the Study

Lagos lagoon is reported to be usually impacted by anthropogenic activities compared to the other seven lagoons in Nigeria(Edokpayi and Olowoporoku, 2010).According to these authorsthe effect of contamination observed in Lagos Lagoon is probably due to population density and incessant deposits of waste in the lagoon. Ajao and Fagade (1990), also reported that Lagos Lagoon serves as a sink for untreated waste while Kamaldeen and Wahab (2011), affirmed the presence of indiscriminate disposal of waste in the lagoon.

Lagos metropolis have been reported to account for larger percentageof industries in Nigeria (Akinsanya, 2003 and Oketola and Osibanjo, 2009a) and most of these industries discharge untreated effluent directly or indirectly through drainages and canals into the Lagos lagoon complex thereby polluting the nursery ground of both fin-fishes and crustaceans (Oyewo, 1998 and Adebayo *et al*., 2007). Documentations from several authors give the daily disposal of effluent into the lagoon to be about 10,000m^3. (Oyewo, 1998 and Oketola, and Osibanjo, 2009b). Other sources of pollution in Lagos lagoon are hydrocarbons from oil pollution (Ekundayo, 1977), untreated faecal waste (Akinsanya, 2003 and Kamaldeen and Wahab,2011), Sewage and disposal of saw dust (wood waste) from sawmill industry situated at the fringes of the lagoon (Ekundayo, 1977; Nwankwo*et al.,* 1994; Nwankwo, 1998. Akpata, 2002; Dosunmu and Ajayi, 2002 andKamaldeen and Wahab,2011), and industrial effluent (Oyewo, 1998 and Oketola, and Osibanjo, 2009b). Furthermore, the pollution status of Lagos lagoon has also been greatly influenced by influx from other surrounding fluvial inputs into the lagoon area(Nkono *et al.,* 1999 and Beattie, 2005).

Fish, shell fish, benthic organisms and other living resources have been affected by the pollution status andused greatly by toxicologist for biomonitoring studies. Majority of the coastal dwellers consumed shell fish including crab for food as a sources of protein, therefore crab state ofhealth is vital to public health.Several studies on pollution status in aquatic environments have been monitored using bottom dwelling organisms (Adebayo *et al.,* 2007).

Benthic fauna are incorporators of pollutants because they are relatively sedentary and are therefore useful in monitoring and assessing the overall health of the aquatic environment. They are also used for long-term monitoring of anthropogenic impacts (Simboura *et al.,* 1995 and Nkwoji, 2017).

Various authors have documentedimpact of effluent discharge on the ecology; population, relative occurence, and distribution of sessile and benthic fauna in Lagos lagoon (Nwankwo and Akinsoji, 1989, Nwankwo *et al.,* 1994; Nwankwo, 1998; Akpata, 2002; Nkwoji *et al.,* 2010 and Nkwoji, 2017). However, there is need for consistent environmental monitoring in the Lagos lagoon for proper time specific pollution assessment and its impact on aquatic living resources as mitigation measures as well as for management purpose.

There has been regular monitoring of physicochemical qualities of surface-waterincluding temperature, salt concentration and amount of dissolved oxygen, nutrients, heavy metals among others, only recently were biological parameter considered to be relevant to ecological studies. Lam and Gray (2003) reported that sediment analysis include quantitative grab samples andthephysico-chemical measurements of sediment including grain size distributions, organic matters and pollutants. However, results of sediment analyses only provide information on the level of contamination but not on the effects on biological systems. This is because the sediment grain size or texture has affinity for heavy metals especially when is less than 0.063mm.

The presence of a particularpollutants in the environment based on the result ofchemical analyses, does not show that the pollutants bio-accumulate in biological system and conclusions on the pollutanteffects on the system cannot be made. In addition, biological systems are often exposed to mixtures of contaminantswhich may be chemicals that exist in different forms with different bioavailability and toxicity while others may interact additively, antagonistically or synergistically.It is however important to investigate different biological responses to ubiquitous but harmful chemicals in the environment in order to conserve diversity and protect natural ecosystems. This realization has resulted in a shift from contaminant monitoring that is chemical based, to effect monitoring which is biological-based (Lam and Gray, 2003).

The biological monitoring is based on different physiological, biochemical, behavioural and morphological features in organisms and ecological community attributes such as abundance and diversity.

Knowledge and understanding of the harmful effects of these chemicals on aquatic living resources are vital. This information will aid to narrowing down point-sources of contaminants, arm regulatory agencies with relevant information for enforcement and management strategies, to safeguard against extereme risks to biodiversity. The pathway of pollutants in the environment seldom involves chemical reaction where the pollutants bind with lipids and protein of most organism in the aquatic environment. Thisgeneration of Reactive Oxygen Species (ROS)can cause damage to cellular molecules which portends negative implications for the affected organism including oxidative stress. The physiological mechanisms of many aquatic organisms, in the breakdown and metabolism of these contaminants into resultant byproducts, show a strong evolutionary overlap with those identified in humans (Carney, 2008). In most aquatic organisms, various endpoints have been utilized to determine the biological responses due to xenobiotics as express in the oxidative stress formed.

The use of biomarkers by ecotoxicologist as biological monitoring of pollution or contaminant in an aquatic environment often reflect effect of these pollutants or contaminants in many aquatic dwellers and has been widely used as a bio-indicator of pollution(Lam and Gray, 2003). However, this study is designed to examine and quantify oxidative stress via antioxidant biomarkers in selected tissues of the blue crab(*Callinectes amnicola*) due to their exposure todomestic, faecal and saw-mill waste pollution in Lagos lagoon.

1.2 Research problem and hypotheses

The Lagos lagoon,while serving as a receptacle for pollution, also serves as an ecosystem for a vast diversity of aquatic organisms. Regular disposal of untreated waste(domestic, sewage, saw-mill, industrial effluent, oil pollution and so on) in the Lagos lagoon has contributed immensely to decline in species richness and distribution of aquatic organism(Akinsanya, 2003 and Kamaldeen and Wahab,2011).

At the cellular level, the effects of pollution may result in detoxification and compensation, at organism level effects of pollution may result in reduced growth and reproduction, in the same vein, at the community level, negative effect of pollution can cause loss of abundance and distribution of a species with evident low diversity and depauperate community.At population level, the effect of pollutionareloss of indicator sentinel species which cause restricted gene pool and loss of genotype (Wright and Welbourne 2002).

Hence hypotheses are highlighted as follow:

- Biochemical indices are better indicators of pollution-related stress in invertebrates than routine monitoring of physical and chemical indices.

- Population of *Callinectes amnicola* at marginal locations of Lagos lagoon are at greater risk as a result of waste disposal into the lagoon than populations from further away into the mid-lagoon regions with less pollution.
- Biomarkers of oxidative stress are effective and reliable indices for heavy metal exposure.

1.3 Justificationfor theStudy

The involvement of fish, shell fish and other sea food in daily intake of organic pollutant, heavy metals and other xenobiotic from aquatic environment in some developing country are enormous, and calls for regular monitoring of contaminant in the living resources ofwater bodies.Most of these pollutants are lipophilic and penetrate biological membranes easily. They accumulate in the organisms and the pathway of pollution in food chain has negative effect on man and animal health through bio-magnification. Biotransformation of some pollutants in animals and humans results in unstable intermediates that affect DNA, RNA and protein, hence, cell toxicity and carcinogenesis which eventually lead to diseases or cell death. (Adeniyi and Yusuf, 2007).

Therefore, reducing pollution in Lagos lagoon would require adequatemethods for monitoring and evaluation of the environmental effect of pollutants. This could also prove to be vital to public health.

The present study has the following significance: First, the study will invariably aid the maintenance of shellfish population of the Lagos lagoon and coastal waters by providing information on the status of heavy metal concentrations in the aquatic water bodies; Second, the findings will also assist regulatory bodies like National Environmental Standard and Regulations Enforcement Agency (NESREA) and National Agency for Food and Drug Administration and Control (NAFDAC) to review the existing effluents standards and surface-water quality guidelines for the protection of resident biota in Lagos Lagoon and other similar bodies of water.

1.4 **Aim and specific objectives of the study**

The aim of this study is to measure heavy metals burden in water and sediment of the Lagos Lagoon and oxidative stress responses in the blue crab,C*allinectes amnicola* fromthe Lagos lagoon, with the following specific objectives:

- To determine the quality parameters of surface water at the sampling stations.
- To investigate the heavy metals concentration in surface water, sediment and selected blue crab organs.
- To determine the oxidative stress parameters; superoxide dismutase(SOD), catalase (CAT), glutathione peroxidase (GPx), reduced glutathione (GSH) and lipid peroxidation (MDA)levels in hepatopancreas, gill, gonad and muscle of *Callinectes amnicola*..
- To examine the histopathology of the blue crab organs.

CHAPTER TWO
LITERATURE REVIEW

2.1 Pollution Status of Lagos Lagoon

Pollution is disposal of untreated waste materials into the natural environment which can cause a negative effect. (*Merriam-webster.com, 2010*). Pollution constituents can be natural or chemical based substances or energy, such as water, air, noise and light.Water pollutants are grouped based on the source, either point or diffuse sources. Point-sources could be direct or distinct, including intentional or accidental spills from industrial sites that channel into aquatic environment e.g marine, brackish and rivers. Non-point sources are indirect, diffused and occasional influenced by factors like climate, vegetations, hydrology and topography. We have urban and rural non-point sources, the examples are run-off from fields or streets which contains different pollutants including heavy metals, chemicals and sediments; other examples are pollutants related to agricultural activities, mining and mineral exploration or forestry respectively.Diffuse sources of pollutants pose serious challenges for monitoring and mitigation (Akinsanya, 2003).

The indirect and direct discharge of anthropogenic waste such as domestic, sewage, saw mill, industrial waste and so on is attributed to Lagos lagoon(Kamaldeen and Wahab,2011). However, fluvial influx empty into the Lagoon from different inland sources and networks.The aquatic organisms and non-living resources are sink of most of these pollutants including house-hold and domestic sewage, industrial wastewater discharges, sawdust and particulate wood and pulp matter, petroleum hydrocarbon, cooling water from a power station and emissions from automobile exhaust. Organic waste, trace heavy metals and chlorinated hydrocarbon are micro pollutants examined in water, sediment and biota.(Akinsanya,2003; Oketola, and Osibanjo, 2009a). The different sections of the Lagos lagoon system are characterized by different types of pollutants ranging from oils and greases, heavy metals, organic biodegradation, cooling water and so on emanating from various sources(Table 1), according to Ajao (1990) and Oyewo, (2009).

Table 1: Sources and Pollutants in Lagos Lagoon

Establishments (Polluter)	Sources of Pollution	Pollutants Types
Atlas Cove	Tank farm for refined petroleum products	Oil and grease
LagosWharf	Shipping lane, regularly dredge	Oil and grease, various spillages and ship garbage
Lever brothers	Detergents(sulphonated) soaps, margarine etc.(partial treatment of wastes in a factory located plant)	Oil and grease, sulphonated detergents, caustic soda from washing. Cooling water
Bordpak	Packaging plant	Mainly organic matter pollutant
Naval Western Command	Navy Oil spillages	Oil and grease
National oil and Agip Depot	Refined petrol /Gasoline diesel oil	Oil and grease
Fisheries Service Company(FISESCO)	Fish trash, diesel oil trawler garbage	Oil and grease, fish trash, organic pollutants, biodegradable.
Good Morning Towel plant (NITOL)	Garment manufacturing plant	Organic pollutants biodegradable
Nig. Breweries Iganmu	Malt fermentation, spillages spent brew Printing, dyeing and gray	Organic pollutants biodegradable
Afriprint Textile mill	Finishing plant	Organic pollutants- Heavy metals Cr, Cu, Zn, Fe, Sn etc. Coloration (Dyes) Organic matter(starch some and grease from diesel plant).

PHCN Egbin	Electric power generating plant	Cooling H_2O (high temp)
Iddo/ijora Municipal Sewage Dump	Major domestic sewage discharges	Biodegradable, organic matter
Iddo market garbage input	Miscellaneous organic wastes etc	Biodegradable organic waste.
Ikoyi Park	Land reclamation or sand filling.	Sedimentation and flocculation
Okobaba sawmill industry	Logging, sawdust input	Biodegradable, organic matter
Eko bridge, Carter bridge and 3rd mainland bridge	Major traffic artery between Lagos island and the mainland	Toxic heavy metal, poisonous fumes from automobile exhausts e.g Lead in gasoline, carbon monoxide and carbon.

Source: (Ajao,1996 ; Oyewo*et al*.,2009).

2.2 Sources of water pollution

Water pollutants originate from two major sources; namely; natural sources, and anthropogenic sources and they may be from a point source or a dispersed source.

Point sources are domestic, municipal and sewer discharge, power generation plants and industrial waste discharge. Some of these, such as breweries, slaughter houses and sanitary operations, paper mills and wastewater treatment plants contribute major quantities of oxygen-demanding substances. These substances can deplete Dissolved Oxygen (DO) and create anaerobic conditions in water bodies. Suspended particulate matter also contributes to oxygen depletion in water bodies by blocking penetration of sunlight and interfering with photosynthesis activities. This results modulates Biochemical Oxygen Demand (BOD) and Chemical Oxygen Demand (COD). Compoundscontainingnitrogenous and phosphorous rich matter(nutrients) can promote accelerated eutrophication of water bodies. Heatis a universal pollutant, as it drastically alters the ecology of water bodies by lowering the amount of DO in water; thus accentuating the oxygen deficiency for aquatic organisms.Non-Point sources are storm drainages, and operations involving agricultural, timber and forest product operations.

2.2.1 *Natural sources of pollution*

These include volcanic activity, earthquakes, landslides, streams runoff, dissolved minerals, aquatic growth and decay, are metrological and geographical.

Radioactive substances and heat are also natural sources of water pollutants.

2.2.2 *Anthropogenic Sources*

Anthropogenic sources are domestic, municipal sewage and other sanitary waste discharges, agricultural and industrial waste (both liquid and gaseous), mining waste and leachates and waste products from other human related activities.

2.2.2.1 *Domestic Waste:* This means any non-perishable waste, consisting of combustible materials, such as paper, cardboard, nylon, wood, or similar materials generated in a living house. It is also called household waste. Large no ofwaste generated in Lagos City, is as a result of many situated households. However, efforts by agencies and inter-ministerial departmentsin Lagos state have achieved about 60% proper management(Kamaldeen and Wahab, 2011).United Nations Educational, Scientific and Cultural Organization(UNESCO), 2000 andAwosika *et al.*, 2000, reported the negative effect of improperly managed wastes which plague the aquatic environment in notable urban environments. Direct disposal of unprocessed domestic-waste including kitchen-waste, and loads of faecal matter into the Lagos lagoon environment could pose a threat to the aquatic ecosystem (Table 2). These threats include increase in the microbialload, concentration of organic material, soil contamination within aquatic ecosystems (Oyelola and Babatunde, 2008). A number of reports (Harold, 1997 and Nubi *et al*., 2008) have attributedloss of diversityclarity of surface water to incidencesoflarge number of undissolved solids and phytoplankton bloom. The high microbial load on aquatic organisms is a consequence that makes them to be susceptible to various diseases at high densities. Incidences of nutrient enrichment and the resultant bloom in plant biota (e.g. water hyacinth plant (*Eichorrnia crassipes*) have been documented for lagoon environments (Kamaldeen and Wahab,2011).

2.2.2.2*Municipal Waste*: This is a combination of household, industrialand commercial waste which include Municipal solid wastes (MSW), municipal effluent or sewage, and bio-solids from wastewater treatment. Landfill gas generated from waste disposed in landfills and biogas from wastewater treatment is also included in municipal waste. These materials include construction and demolition, wood residue, paper and cardboard, grass, landscape tree removals, other green waste, food waste (for example, meat and vegetable scraps) and other organics. Putrescibles isan alternate term commonly used to describe the garbage faction. The uses of landfills are stated below as reported by Kidder, (2002), wastes are disposed in landfills, the mass-burn incineration facilities, landfill gas is used for heat and power generation as well as being upgraded to pipeline quality, it is also being used as transportation fuel. Diverted wastes are used for compost, recycling, and energy.

2.2.2.3*Saw-mill Waste*: Saw-mills generate a lot of saw dust as waste naturally, wood cut-off, wood chippings, and waste wood parts. Often times, the proper disposal methods are not followed and when these wastes are incinerated by open-burning around the lagoons shores. Following the increased demand for wood and wood-products, there is a commensurate spike in the volume of wastes produced and discharged. As such sawmill wastes constitute a major waste management challenge (Dosunmu and Ajayi, 2002).

Table 2: Sources of Domestic waste and its Pollutants Threats posed to organisms

Source	Pollutants	Threats to organism	References
Organised urban settlements, schools, hostel facilities, domestic garbage and medical utility wastes etc	Caustic soda, detergents, kitchen wastes, household rubbish, organic pollutants, biodegradables, solid wastes.	Reduced dissolved oxygen in surface water and, increased Biological Oxygen Demand (BOD).	FEPA, 1992; Ajao, 1996; Nubi *et al.,*2008.
	Domestic sewage (faeces and urine)	Unpleasant odour, bacterial and fungal growth, nutrient enrichment, microbial load contamination	Akpata, 1975; Ajao, 1990 ; Kamaldeen and Wahab,2011

Dosunmu and Ajayi, (2002) documenting anthropogenic effects of a sawmill industries located at Okobaba area adjacent to the Lagos lagoon environment, highlighted it as a major contributor of organic wastes into the lagoon. Further reports (Akpata (1980); Dosunmu and Ajayi, 2002) demonstrated that wood wastes and floated logs stored on surface water for prolonged and sometimes indefinite durations also impact surface water quality. Negative effects include reduced dissolved oxygen content, and deterioration of water odour and clarity.

Sawdust, which is the waste product from sawing wood, is dumped into the Lagos lagoon. The effects of this organic load on the Lagoon system have been documented by Akpata (1980) and Dosunmu and Ajayi (2002).

2.2.2.4 *Faecal/Sewage Waste*: Excreta constitutes largely solid and liquid waste discharge from humans and animals. It is characterized by enteric microbes that could constitute health hazards to humans and livestock. Elsewhere it has been documented that the socio-economic importance and ecological role of the Lagos Lagoon (Lalèyè and Moreau, 2005). Others categorized the fish resources in the lagoons into; (a) littoral euryhaline organisms that isorganism with seasonally or circumstantial occurrence (b) estuarine species that is which occur mostly in mixo-haline environments and (c) inland water species which are thosefound during rainy season when salinity in the lagoon is diluted and accommodating for fresh water species.

Various reports have documented notable cases of direct input of waste water and industrial effluent in to the Lagos lagoon (Ajayi and Akonai, 2005). Damage to human health is highest rated out of all the negative effect of urban environmental degradation (Obire and Aguda, 2002;Adelegan, 2004). Infectious diseases was rated 33% in 1995, which cause world wide deaths of about 17.3 million.

World Health Organisation, (2007) documented a direct relationship between large-scale urban development projects and degeneration of environmental quality which inturn impacts public health. Hygiene related microbes can also be transmitted through waste or poor waste management practices (Kamaldeen and Wahab(2011). According to Akpata (2002) nothing less than 30 million litres of faeces was released into the lagoon environment in 1973.

Others confirmed from recent studies such trend is still in practice with more than 27,000 tons of suspended solids and about 2,000 tons ofdecomposing matter are introduced into coastal lagoons annually (Rufus, 2006). The trend is largely unchanged and unabated with loads of liquid and solid waste intermittently released into fishing grounds and waters proximal to domestic settlements. Baarschers (1996) andNebel and Wright (2002) documented sewage releases into surface water while Miller (2000) documented similar incidences in many developing countries and some developed countries. The unhygienic disposal of faecal matter into the canals and lagoons result in the emergence of enteric organisms into aquatic environment. Numerous appearances of enteric organisms in aquatic environment indicate recent contamination by raw sewage. Reports by Akpata and Ekundayo (1978) confirm deteriorated water quality including decreased concentration of oxygen and poor transparency following discharge of faecal matter into surface water.

Such incidences and practices increase the likelihood for humans living in coastal areas to have direct contact with dangerous microbes via drinking water and contaminated seas food. The fish in the lagoon might have been contaminated because of untreated chemical disposed therein and these made fish consumers prone to risk ofillness (Raufu 2006). Abulude *et al,* (2006) reported that benthic species like crustaceans may habour toxic materials in aquatic environment with imminent risks to end users and consumers.

2.2.2.5 *Industrial waste*: The different waste content from industries and pollutants present is a factor of industry-specific waste types. A number of these various types and likely risks or impact on aquatic species are given in Table3.The negative effect of industrial effluent and the resultant pollutants in aquatic environment reported by UNESCO (2000), are highlighted; The improper handling of both household and industrial waste including toxic ones has resulted into soil and ground water pollution.

2.2.2.6 *Hospital Waste*: These are waste generated and disposed in the hospital. It may be waste generated in diagnosis, treatment, or immunization of human beings or animals or in research studies carried out in hospitals.

Table 3: Industry-specific waste varieties in Lagos State and their potential risks to resident biota

Source	Pollutants	Threats to aquatic organisms	References
INDUSTRIAL-FACILTY WASTE e.g. Dumex Industry (Nig) Ltd, Vitamalt Plc	Decomposable pollutant, alcoholic, Brewery waste, biodegradable, Zn, Pb	Significantly lowered dissolved oxygen, eutrophication, clogging of fish gills by pollutants	Ajao, 1990 Adebayo *et al.,*2007.
PAPER-MAKING INDUSTRIES	Inorganic salts	Moderately toxic. Elevated metal levels in environment and biota matrices.	Ajao, 1996 Adeleye *et al*,2011
SOAP-MAKING FACILITIES	Caustic and organic pollutants. Biodegradable detergents. Thermal pollution from process water.	High turbidity of surface water due to onset of eutrophication.	FEPA, 1992 Adeleye, 2011.
			FEPA, 1992
PLASTIC-MAKING INDUSTRIES	Heavy metals including lead.	Increases the temperature and bioaccumulation of metals often result.	FEPA, 1992
FOOD AND BEVERAGE MAKING FACILITIES	Organic compounds and Decomposable food materials. Rise in surface water temperature may impact oxygen uptake in fish.	Eutrophication, gill clogging, lowered oxygen content of surface water	FEPA, 1992
CHEMICAL INDUSTRIES	Caustic waste water and halogen radicals.	Low dissolved oxygen, lowered surface water temperature	FEPA, 1992
			FEPA, 1992
PHARMACEUTICALS	Caustic waste water,	Direct erosion of exposed tissue	

	halogen radicals and trace metal	e.g. fish gills	
METAL-WORKS FACILITIES	Acids, alkali, fluoride, inorganic salt, cyanide,	Direct physiological toxicity and increase biological oxygen demand.	Akpata, 1980 Dosunmu and Ajayi, 2002
WOOD-WORK AND SAW-MILLING FACILITIES	Decomposable plant parts	Increased BOD due to biodegradable material, fungal and microbe proliferation due to organic substrate.	Ajao 1990
Artisanal sand mining and mechanized dredging	Increased siltation with highly turbid appearance of surface water	Habitat disruption, modification and patch fragmentation	Okoye, 1991,Ajao, 1990,
High alkenes, alkyne content from oily discharge disposal, harbor a ccidental spills)	Oily pollutants and grease	Moldering of aquatic organisms including birds and furred animals	Ladigbolu *et al.,* 2011
Complex mixtures from large scale industrial facilities	Pollutants, hot-water releases into surface water, variety of bioavailable xenobiotics		Adeleye *et al*., 2011,
Thermal/ heat pollution	Waste heat from cooling machinery		Ajao, 1990 Akinsanya, 2003

Classification of hospital wastes according to Salih and Aljabre (2002) are as follow:

- Pathological waste: wastes of hospital origin largely made up of excised tissue, amputated parts and infectious fluid and body parts. It is a highly category of hazardous waste.
- Infectious waste: This is hazardous because these contain disease agents or microbes in tangible quantities that could initiate inferior with a previously uninfected body. These include discarded material from tissue culture and infectious wastes from laboratories, as well as from surgeries of infected patients.
- Sharps: This refers to disposable materials and tools with sharp ends that could accidentally injure the handler and cause accidental infection.
- Pharmaceutical waste: This includes drug-related products and chemicals discarded due to expiry or accidentally spilled.
- Chemical waste: This comprises discarded solid, liquid and gaseous chemicals such as cleaning, house keeping, and disinfecting products.
- Radioactive waste: This is listed as wastes either in solid, liquid, or gaseous state with radioactive properties. Most of them are of hospital origin.

2.2.2.7*Agricultural Sources*: Agricultural wastes generally, consist of organic products. Fertilizers and other chemicals are spread over agricultural lands. These materials and agricultural animal wastes enter water bodies, mainly in run-off from watershed lands, and cause pollution. The inflow of manure from livestock feed lots also adds to organic pollutants. Most pesticides, fungicides, herbicides and other industrial chemicals are highly toxic and are carcinogenic and mutagenic (Adeleye *et al*., 2011).

2.2.*2.8 Mining Wastes*: Mining, milling, dressing and processing of ores give rise to dust, ore and metal discards as well as large quantities of effluents, which are discharged into streams, ponds and lakes. They not only increase sediments but also release toxic metals into the water sources.

Common trace metals found in sediment and mine effluents are Co, Cu, Fe, Hg, Man, Ni, Pb and Zn. Cadmium being chemically similar to Zinc, replaces the latter in enzymes and thus affects enzyme action of Zn containing proteins. Mercury as a heavy metals pollutant has been a source of great concern. Lead occurs in water in Pb (II) state and is highly poisonous causing anemia, central nervous system disorders, kidney and liver dysfunction (Dirk, 1993).

2.2.2.9 *Local and Industrial Dredging:* Dredging entailsexcavation of top soils and particulate matter from benthic parts of aquatic environment. Dredging is also carried out to reduce the exposure of living resources in water bodies and populationsto pollutants and to block the transport of pollutants to other areas of the water body.

2.3 Water quality analysis

The quality of a water sample can be determined on the basis of physical, chemical, biological and aesthetic points of view. Physical parameters are color, odor, turbidity, particulate matter and conductivity. Chemical parameters that affect the quality of water are Hydrogen ion concentration, DO, BOD, COD, dissolved substances, metals, and redo chemical reactants. Industrial operations are the main sources of metallurgical and chemical pollutants. Biological parameters that affect the water quality are the presence of algae, viruses, coliform, pathogens and other vectors that are responsible for health hazards and diseases (Narayanan, 2011).

2.3.1 Physical Methods of Water Analysis

Organolepic properties of water are related to odour, taste and colour. Odour in water is a sign of decaying organic matter. Contaminants that contribute to odour are generally volatile organic compounds (aldehydes, ketones and phenols). Methods of determination of odour and taste are qualitative and subjective.

Transparency of water is measured by the depth of penetration of light. This parameter depends on coloring substances, turbidity due to suspended particles as well as metal ions and their complexes. Turbidity is an optical phenomenon. It is the measure of attenuation of a beam of light that passes through a non-absorbing medium. Turbidity in water is due to suspended particulate matter such as clay, silt, coagulants, organic matter and photoplanktons. Nephelometers and turbidimeters are used to determine turbidity. Generally, turbidimeters are used to determine dense suspensions (Narayanan, 2011).

2.3.2 Chemical Methods of Water Analysis

Chemical parameters for determination of water quality are dissolved solutes, salinity, hardness, Hydrogen ion concentration, DO, BOD, and COD. Water salinity is mainly due to dissolved salts of sodium and potassium (NaCl and KCl). It can be determined by hygrometric and titrimetric methods.

Acidity or alkalinity (pH) of water can be determined by potentiometric method (pH meter). Ion-selective electrodes (ISEs) can be used to determine ions and gases dissolved in water. Most of the inorganic pollutants in water (nitrates, sulfates, chlorides, hydrated metal complexes) can be determined by wet-chemical methods, monitored by spectrometry, or by biological methods. Ion-Chromatography (IC) is a versatile technique that can be automated for environmental pollution analysis.

The determination of organic pollutants in water can be done by monitoring DO, BOD and COD. Respiratory methods can also be used to determine O_2 uptake of organisms.

2.3.3 Biological Methods of Water Analysis

Biological methods for assessing water quality rely on the estimation of biomass, coliform bodies, micro-organisms and nutrients. Estimation of biomass includes determination of total carbon, nitrogen, phosphorus, lipids, carbohydrates and benthal sediments. Sediments are usually the most appropriate candidates for long-term monitoring of contaminants in aquatic systems.

Algal proliferation is a useful indicator of hydrospheric and atmospheric pollution. Measurement of chlorophy II content is a suitable parameter for the evaluation of algal biomass. Chlorophyl II determination is carried out spectrophotometrically. Determination of photosynthesis is carried out by measuring the absorption of Infrared Radiation (IR) of CO_2 in a non-dispersive analyzer. Water pollution from domestic, food, beverage, and sewage, dairy and agricultural wastes contains pathogens (bacteria, protozoan's and viruses and disease carrying vectors).

2.4 Indicator organisms

Pollution occurrences in the aquatic environment have received special attention lately. Biological monitoring tools have been utilizes have significant tools to pollution studies in aquatic ecosystem. Several species of aquatic organisms utilized as biological indicators species in aquatic ecotoxicological studies compared to others due to their merits and demerits in practical biological monitoring of inorganic and organic pollutants. Presently there are general biological monitoring method categorizes as biochemical alterations, bioaccumulation, surface morphological and behavioral observation, population, community and ecosystem generally. Biological monitoring applications have been proposed as an appraisal tools for establishing the extent of contaminant release in surface water and sediment, prediction of toxicological mechanisms. Additional view point on biological monitoring issues of organic and inorganic in aquatic flora and fauna (Qunfang *et al*., 2008).

2.4.1 ***Fish***

The fish biological features which include body architecture, survival cycle, easy of culture and others, has made most ecologist or eco-toxicologist often time use it in the environmental bio-monitoring of aquatic pollution. Furthermore, the position of fish species in the aquatic food chain may have a direct effect on human health and this makes it very vital to bio-monitoring. Odumuyiwa (2010) investigated the levels of Pb, Cod, Cu, Cr and Zn in two common edible fish species *(Solae sole* and *Pseudolithus spp)* from Lagos Lagoon and Cocoa Lagoon of Lagos and Delta States. The obtained results showed that the mean values of Cd and Cr in the fish samples were higher than the tolerable levels. Generally, *Solea solea* showed higher levels of metal concentrations than *Pseudolithussop.*

Metal incidence showed increased levels in samples sourced within the Lagos Lagoon in Lagos state compared to values in samples from the Cocoa Lagoon in Delta state, probably due to inclusion of more effluents from industrial, commercial and municipal discharges in Lagos state and its environs as explained by Lowe*et al.* (2003).

2.4.2*Blue Crab*(*Callinectes amnicola*)

Blue Crab are an in-shore, bottom-dwelling crustacean species, with a habitat range spanning the downstream ebbs of lentil ecosystems, coastal flood plains. Blue crabs are swimming crabs and this character makes it easy for transition across habitat terrains (Micheli and Peterson 1999; Ryer *et al*., 1997). The colour of blue crab is brown or grayish are modified for habitat adaptation to integrate with the scenery of its habitat. Their carapace color often changes in relation to the surroundings of their habitat, in other words they have colormorphotypes (Hovel and Lipcius, 2001).

Shellfish, for example; crabs, are incorporators of pollutants because they are relatively sedentary, thus are important in monitoring and assessing the overall health of the aquatic environment. Shellfish are also used for long-term monitoring of anthropogenic impacts (Simboura *et al*., 1995 and Nkwoji, 2017).

2.4.3*Bivalve mollusks*

They are filter-feeding organisms, they bio-accumulate hazardous contaminants leading to adverse population outcomes. A widespread monitoring programs founded on the concept of indicator organisms has proven effective indiscerning contaminnat patterns. Features of mussels that qualify it as a uninanimous sentinel of environmental health include: broad geographical coverage, relatively higher abundance, sessile, accommodative of wide environmental fluctuations, tolerance to extreme contmainant concentrations in the environment, high bio-concentration-indexusable as a reference of aquatic food chain, tissue contaminants are little metabolized and unchanged by metabolizing enzyme, wide and stable populations, life-span favorable for ecological study, reasonable size, adaptive for laboratory studies and experimental setups.

2.4.4*Gastropod*

Gastropod: a class of organisms which feed as filter feeder by enlargening their buoyancy. These group of organisms have been identified as bio-accumulators of metals. Gul*et al*. (2004) documented simulated experimental exposures of mud-snail (*Cipangopaludina cahayensis*) which demonstrated features suitable as a bioindicator for metal bioavailability and toxicity. Various gastropod species have however, demonstrated varied responses and capabilities for uptake of metal compounds, Li, *et al*.(2009).

2.4.5*Insects*

Variety of insects inhabit the aquatic system and are useful for the biomonitoring of aquatic metal pollution. Bonada *et al* (2006) reported that the recent developments in insect aquatic bio-monitoring have led to the establishment of performance indices. These criteria have been described as necessary for an ideal bio-monitoring tool. Han (2002) documented high, metal uptake by *I. elegans* from surface water. The lack of statistical difference among the contents of heavy metal in male adult *I. elegans*from same sites at the same time, suggested that the organism is an indicator for contamination of Cod in water system. The ability of cadmium exposures to induce tissue concentrations of metallothionein-like protein has been taken as an indication of insects to respond to metal toxicity in a measurable way. Elsewhere, Werner (1999) documented modulated levels of stress proteins that is. hsp70, in species of caddisfly following exposure to heavy metals in sediments.

2.5 Monitoring aquatic pollution

Biomarkers, either physiological modulations or biochemical responses to xenobioticuptake in living tissue within the organisms, indicate habitat health status. Despite the increase cumulative knowledge in this field, many xenobiotic-related physiological outcomesremain largely misunderstood, resulting in uncertainties for regulatory decision makers. Zhou *et al*., (2008) correlated fish health with pollutant concentration in the environment.

2.5.1 *Chemical-based monitoring*

The environmental monitoring of coastal environment in the past often times involve measurement ofphysicochemical parameters and biological variables were rarely incorporated. In the aquatic environment, the examples of routine physicochemical parameters measurement are temperature of surface water, salt concentration, and dissolved oxygen levels, nutrient levels. Sediment monitoring entails sampling sediment to exact and characterize benthic organisms. Direct sediment parameters includecharacterisation of grain particles, organic matter content and contaminants levels including heavy metals. Such analysis gave vital information on concentration of contamination with the exception of the benthic organisms of sediments, which did not give information on effects of the contaminants on biota.

The incidence of pollutants in water bodies has received greater attention. Some heavy metals may transform into the persistent metallic compounds with high toxicity, which can be bioaccumulated in the organisms, and magnified in the food chain, thus threatening human health (Lopez *et al*., 2006). Several toxic effects including tetratogenic effects, procreation failure, and immune modulations have been attributed to incidences of aquatic pollution(Wu *et al*., 2003). According to Finkelman (2005), heavy metals in aquatic ecosystem occur naturally through leaching from soil or rock to water, usually at low concentration, with no harmful consequences for human and ecological health. The emergence of large-scale industrial processes and integrated agricultural activities has significantly contributed to the unnatural incidence of elements in the contemporary environment.

Incidences of heavy metal pollution in aquatic environments has often recorded elevatedlevelsofmetal species in surface water, sediment, and tissues of resident organisms. Anthropogenic effects have been implicated in incidences of aquatic pollution; the source of effluent is mainly from manufacturing and raw material processing industries(Xu and Yang, 1996). Some heavy metals including Hg, Cr, Cd, Ni, Cu, Pb and so on, when introduced into environmental water systems may lead to high toxicities in the aquatic organisms (Wu and Zhao, 2006). Intermittent reports on global pollution is unabated Nigeria inclusive. A typical investigation documented the occurrence of Fe to be the highest, followed by Zn, Pb, then Cu while levels of Cd was generally below 0.002 mg/Kg in all the stations studied. (Adeleye*et al*., 2011). Varied concentrations of various metal contaminates have been documented in areas on the Lagos Lagoon and have been attributed to adjacent land use around the lagoon(Ajao,1996 and Nubi*et al.,* 2008).

2.5.1.1 Contaminant sources for metal-pollutants into coastal areas

Cadmium (Cd)

Occurs in nature as oxides, sulfides, and carbonates in metal ores (Finkelman, 2005). It is very similar to zinc and often associated with mineral deposits of Zn (Callender, 2003). Cadmium is potentially toxic to living species particularly in its ionized state (Denton *et al.,* 2001). Its main sources include effluents from metal-work industries, urban domestic waste water(Denton *et al.,* 2001). In sediments, tendency for adsorption is determined by total organic matter content, including humus and plant debris. Its strength of an adsoroption is strongly dependent on the clay content and increases with pH, while its Sequestration from sediment into surface water is influenced mainly by redox conditions and complexing agents in the water. Cadmium is less mobile Under alkaline and saline conditions(Fergusson, 1990).

Chromium (Cr)

It is one of the most abundant heavy metals in the earth crust having an average concentration of about 69ug/g, and moderate toxic capacities. Major sources within the coastal marine environment include river discharge, point source discharge from industries and their dispersed sources (Jerome *et al.,* 2016). Others include waste water runoff from electroplating, and metal finishing industry (Callender, 2003; Finkelman, 2005). Its concentrations in marine sediments range from 2.4 go/g at unpolluted sites to 749 go/g at grossly contaminated sites(Finkelman, 2005).

Copper (Cu)

Copper is a moderately abundant heavy metal with mean concentration in the lithosphere of about 39µg/g. It is an essential trace element for the growth of most aquatic organisms. However it becomes toxic to aquatic organisms at levels as low as 10µg/g (Callender, 2003). Heavily polluted sediments have been reported to exceed 200 μg/g. Inputs of copper into the natural waters come from various sources including mining, smelting, domestic and industrial wastewaters, steam electrical production, incinerator emissions, and the dumping of sewage sludge (Denton *et al.,* 2001). Algaecides and antifouling paints are identified as major contributors of copper to harbor areas whereas coastal waters generally receiving input from rivers and atmospheric sources (Denton *et al.,* 2001). Copper has a high affinity for clay mineral fractions, especially those rich in coatings containing organic carbon and manganese oxides (Callender, 2003). As a result, residues are often elevated in sediments near localized sources of inputs (Denton *et al.,*1997).

Lead (Pb)

It occurs in various chemical complexes in the environment. Its inorganic forms moderately lethal to invertebrates comparedto other metals. Organo lead complexes, including alkyl-lead mixtures applied as antiknock agents in gasoline, are potent toxic agents (Denton *et al.,* 1997). Anthropogenic activities that contribute to its incidence in the environment include wastes from manufacturing processes including metal production, particulate discharge from combustion engines using leaded fuel; combustion processes using wood and coal; and open burning of municipal refuse. Domestic waste and sewage discharge also contribute to its availability in the environment. Lead species are strongly absorbed to matrices within clays and organic materials (Fergusson, 1990). The absorption process is strongly dependent on the ambient phi. As such it shows limited mobility and accumulative tendencies (Morrison, *pers. comm*.). Lead is reported to be in the 15-50 go/g range for coastal and estuarine sediments around the world (Denton *et al.* 1997) with < 25 go/g in clean coastal sediments.

Mercury (Hg)

This element constitutes high toxic potentials to fish and aquatic invertebrates, especially when it is present in the organic form (Denton *et al.,* 1997). This compound is particularly a concern due to its ability to concentrate in tissue of certain commercially important fresh-water and marine fish in quantities many times higher than the ambient water levels (Finkelman, 2005). Current major sources include power generation facilities including high temperature combustion processes for example, urban waste incineration facilities (Denton *et al.* 1997; Fitzgerald *et al.,* 2003). Occurence of mercury in urban runoff are often very low, with a possibility of higher incidence in petroleum-related waste (Denton *et al.,* 1997).

Mercury has high affinities for organic carbon as well as sulfides. In marine and estuarine environments, where seawater provides sufficient sulfate, rates of sulfate reduction are affected mostly by availability of organic matter and temperature (Fitzgerald *et al.,* 2003). In seawater, mercury is associated with surface active organic materials and mercury ions and the metal are rapidly sorbet by sediments(Fergusson, 1990).

Zinc (Zn)

Zinc is a very common environmental contaminant and usually outranks other `metals like Cu and Pb in terms of abundance. It is commonly found in association with lead and cadmium (Denton *et al.,* 2001; Finkelman, 2005). Although it is not regarded as particularly toxic, it is sometimes released into the sea in substantial quantities (Denton *et a.,*2001). Major sources of Zinc to the aquatic environment include the discharge of domestic wastewaters; coal-burning power plants; manufacturing processes involving metals; and atmospheric fallout (Denton *et al,.* 2001). Approximately one third of all atmospheric zinc emissions are from natural sources, the rest come from nonferrous metals, burning of fossil fuels and municipal wastes, and from fertilizer and cement production (Denton *et al.,* 2001; Callender, 2003). Sediments are known as major receptacles for zinc in the aquatic environment, and residue in excess of 3000 go/g have been reported close to mines and smelters (Denton *et al.,* 2001). The average level of occurrence within the lithosphere is documented to be approximately 75-80 go/g (Callender, 2003) and depending on the local geological profile and terrain, for uncontaminated waters, sediment levels occurs within the range of 5-50 go/g.

2.5.2*Biological-based-monitoring*

Biomonitoring a process of environment assessment, following human or fauna exposure to natural and synthetic chemicals. It entails the use of sampled biological samples from an individual organism's tissues and fluids. It utilizes responses or reactions that serve as imprints or markers after exposure or uptake of a contaminant or xenobiotic. These markers may be a chemical response e.g. presence metabolites, tissue alterations, los of tissue architecture or modulated immune responses(Zhou*et al.*, 2008).

2.5.2.1 *Biomarkers in environmental monitoring*: Biomarker although a largely vague term, could be depicted as a range of biological responses associate with hazards in the immediate or ambient environment of a species (WHO, 2011). A key feature is that these responses are measurable and unmistakable as reactions to toxic conditions (Peakall, 1994). On the basis of NRC (1987) and WHO (2011), biomarkers can be subdivided into three classes:

1) *Biomarkers of exposure*: Subsets of this category entail responses due to the presence of abnormal concentrations of a chemical or mixtures of chemicals within the immediate environment of the organism. It's also usable to confirm and assess the exposure of individuals or populations to a particular substance (group), thus providing a link between external exposure and internal dosimeter (Livingstone, 2001).
2) *Biomarkers of effect*: These highlight quantifiable physiological and biochemical responses usable as indices for poor or deteriorated health of biota. It can also be used to document either preclinical alterations or adverse health effects due to external exposure and absorption of a chemical.
3) *Biomarkers of susceptibility*: indicating the inherent or acquired ability of an organism to respond to the challenge of exposure to a specific xenobiotic substance, including genetic factors and changes in receptors which alter the susceptibility of an organism to that exposure.

The responses following certain toxicant exposure are useful as indicators of both exposure and effects but body burdens of contaminants are not considered to be biomarkers or bio-indicators since they do not provide information on deviations related to health.

Therefore, biochemical responses may be similar in a large variety of organisms. Good biomarkers are sensitive indices of both pollutant bioavailability and early biological responses.

Biomarkers may be used after exposure to dietary, environmental or occupational sources, to elucidate cause/effect and dose/effect relationships in health risk assessment, in clinical diagnoses, and for monitoring purposes. Generally, biomarker responses are considered to be intermediates between pollutant sources and higher-level effects (Suter, 1990; Livingstone, 2001). Beyond certain threshold (in pollutant dose or exposure time) the pollutant-responsive biomarker signals deviate from the normal range in an unstressed situation, finally leading to the manifestation of a multiple effect situation at higher hierarchical levels of biological organization (McCarthy *et al*., 1991; Livingstone, 2001).

2.5.2.1.1Antioxidant Biomarkers (Antioxidant Enzymes, Antioxidant Substances (Vitamin, Carotenoids): Antioxidants are uninanimous depictors of oxidative stress in marine organisms (Verlecar *et al*., 2008). Various aspects of biomedical research havehighlighted that contaminant-mediated biological effect on the organismscould be depicted or understood via variations in the levels or activities of antioxidants (Sole`*et al*., 1996 and Camus *et al.*, 2003). A tangible number of components pertaining to the detoxification and antioxidant system in mollusk species have been shown to be specifically induced by metals or Polycyclic Aromatic Hydrocarbon (PAH) in controlled laboratory conditions (Regoli and Principato, 1995). Environmental factors were found to play a crucial role in regulating the oxidative stress capacity of tissues of *P. verifies*(Verlecar *et al*., 2008). Superoxide dismutase has been reported to mix well with immune competence of mollusks (Liu *et al*., 2004). As a free radical elimination enzyme, SOD is essential to minimize the oxidative damage to host cells in the immune defense (Zikic, 2001). Elsewhere studies have demonstrated that that Cu induces an imbalance in the oxygen metabolism during the first week of exposure due to a decrease in mitochondrial SOD and CAT selenium dependent, and total glutathione peroxidase activities (Geret, Serafim*et al*. (2002). Superoxide dismutase and selenium dependent glutathione peroxidase are two main antioxidants in organisms (Orbea *et al*., 2002and Carney, 2008). Catalase is a commonly studied antioxidant enzyme involved in the initial anti-oxidative mechanism and widely used as a biomarker in mussel (Cajaraville *et al*., 2000; Khessiba *et al*., 2005and Romeo *et al*., 2000). Levels of lipid peroxidation in digestive gland, mantle tissue and gills of`` the mussel obtained from a contaminated site has also been demonstrated as a reliable inside of habiat-quality effects (Almeida, *et al*., (2007).

Alteration in antioxidant system due to copper stress was investigated by Isani *et al*., (2003); Das and Mukherjee (2000); other reports deocumented that significant reductions in the activities of antioxidants along with increased lipid peroxidation in infected shrimp confirmed increased oxidative stress. Increased lipid peroxidation was observed when the clam *R. decussatus* and mussel *M. galloprovincialis* were exposed to Cu (Geret, Jouan*et al*., 2002 and Viarengo *et al*., 1990). Lipid peroxidation is considered a biomarker of damage. Variations in SOD, CAT, GPx and MDA activities suggest their potential use as biomarkers of effects, such as oxidative stress, resulting from Cod contamination in the mollusk*R. decussates* (Geret, Serafim *et al*., 2002).

These antioxidants could either be from exogenous sources e.g. diet or formed endogenously in the body. The mechanisms of selected antioxidant biomarkers are explained below:

2.5.2.1.2 *Superoxide Dismutase*: The SODs are a group of metalloenzymes that catalyse the conversion of reactive superoxide anions (O_2^-) to yield hydrogen peroxide H_2O_2, which in itself is an important ROS. Hydrogen peroxide is subsequently detoxified by two types of enzymes: CATs and Glutathione Dependent Peroxidases (GPOXs). Superoxide dismuthase are considered to play a pivotal antioxidant role; their importance is indicated by their presence in all aerobic organisms examined (Stegeman *et al*., 1992). Additionally, the rate of SOD-catalysed O_2^- dismutation approximates the diffusion limit, making it one of the most active enzymes described (Fridovich, 1986). Most techniques for the measurement of SOD activity are indirect assays in which an indicating scavenger competes with endogenous SOD for O_2^-+. A unit of SOD activity is defined as the amount that causes 50% inhibition of the reduction of the scavenger under specified conditions (Stegeman *et al*., 1992).

2.5.2.1.3 *Catalase*:Catalase is a notable enzymatic antioxidant enzyme which facilitates the initiation of antioxidative mechanism and has also been reported in aquatic invertebrates(Jerome *et al*, 2016)while its role in antioxidant defense in aquatic invertebrates has been demonstrated in early reports (Livingstone *et al.*, 1992). It carries out a reduction reaction on H_2O_2 produced by the SOD enzyme, to form water and oxygen as indicated in the chemical formula $2\ H_2O_2 \rightarrow O_2 + 2H_2O$.

Catalysesare enzymes that contain hematin, the protein whichenhances the removal of hydrogen peroxide, which is eventually metabolized to molecular oxygen (O_2) and water. Unlike some peroxidases that can reduce various lipid peroxides as well as H_2O_2, it can only reduce H_2O_2 (Stegeman *et al*., 1992; Filho, 2001; Asagba *et al*., 2008 and Ruas *et al*., 2008).

Following its functions and occurrence in erythrocytes, it may be adopted as a possible marker or indicatorof oxidant exposure in vertebrates. A phase-by-phaseelevation of CAT activity within erythrocyte was observed in crustacean carp exposed to pesticides (Gabryelak and Klekot, 1985; Thomas and Wofford, 1993;Reméo *et al*., 2000 and Asagba *et al*., 2008). A commonly employed assay for the measurement of CAT activity involves the use of the spectrophotometer to observe the disappearance of exogenous H_2O_2(Stegeman *et al*., 1992 and Lopez *et al*., 2006).

2.5.2.1.4 *Glutathione peroxidase (GPx)*: Peroxidases (PXs) are enzymes that reduce a variety of peroxides to their corresponding alcohols. While CAT employs one molecule of H_2O_2 as donor in the reduction of another H_2O_2 molecule, peroxidases employ other reductants. The principal peroxidase in fish is a selenium-dependent tetrametric cytosolic enzyme that employs GSH as a cofactor. GPx catalyses the metabolism of H_2O_2 to water, involving a concomitant oxidation of reduced GSH to its oxidized form (GSSG). Its criticalimportance in preventing membrane-damage due to LPx(Livingstone, 2001).

Elevated GPx activity following contaminant exposure that is paraquat, PAH, PCBs and HCB-contaminated food has been documented, while lowered activity was only noted after exposure to 3MC.

2.5.2.1.5 *Glutathione-S-Transferase*: This antioxidant detoxifies a range of xenobiotics via conjugation to glutathione, which leads to its final expunge from theorganisms system. Categories of GSTs have been documented with some demonstrating peroxidase activity, using the mechanism of oxygen-detoxification function i.e., reduction of lipid peroxides. This category isnotable for invertebrate class of aquatic organisms compared to vertebrates where it is absent. (Livingstone, 2001; Ruas *et al*., 2008 and Firat *et al.*, 2009).

2.5.2.1.6 *Reduced Glutathione (GSH)*: This enzyme is regarded as critical to cellular antioxidant activities(Vijayavel *et al*., 2004). It is the co-factor of many enzymes catalyzing the detoxification and excretion of several toxic compounds. Its characteristic rich tripeptide feature provides an intial defense mechanisms to protect cells from effects of radicals. Its depletion or low activity clearly indicates stress and an overwhelmed antioxidant capacity (Ringwood *et al*.,2005).

2.5.2.1.7 *Lipid Peroxidation*: This is a notable index of cellular injury and tissue damage in most organisms. Mallondialdehyde correlates strongly with incidences of lipid peroxidation and prostate gland in biosynthesis, thus, its measurement is accepted as a reliable measure for lipid peroxidation. It could also interracts spontaneously with functional protein and other bio-molecules to achieve different types of complexes (Livingstone, 2001; Asagba *et al*., 2008 and Ruas *et al*., 2008).

oxidized polyunsaturated fatty acids otherwise called LPOs, is a notable outcome of oxidative stress (Stegeman *et al*., 1992; Hageman*et al*., 1992 and Livingstone, 2001). Numerous studies have demonstrated enhancements of LPOX in various tissues from fish species exposed in vivo to a variety of chemicals, such as paraquat-exposed carp (Gabryelak and Klekot, 1985 and Livingstone, 2001), channel catfish and brown bull head exposed to t-butyl hydro peroxide sea bass exposed to heavy metals (Romeo *et al*., 2000) and blue gill sunfish exposed to anthracite and UV-light (Choi and Oris, 2000). New trends in the demonstration of LPOX by measurement of degradation products such as aldehydes, acetone and malondialdehyde have been described by De Zwart *et al*. (1997). LPOX appears to have considerable potential as a biomarker for Environmental Risk Assessment (ERA) (Stegeman *et al.*, 1992; Hai *et al*., 1997 and Livingstone, 2001), although it can occur as a consequence of cellular damage due to a variety of factors other than exposure to xenobiotics causing oxidative stress (Kappus, 1987 and Lopez *et al*., 2006).

2.5.3 *Reactive Oxygen Species*

Reactive Oxygen Species (ROS) are molecules derived from oxygen that are produced naturally as by-products of metabolism and aerobic respiration (Kelly *et al*., 1998 and Valavanidis *et al*., 2006) and as cell signaling molecules. However, an electron may be detrimental to proper cell function and viability (Maiti*et al*., 2010). Concentrations of ROS are normally kept in check by antioxidant defense mechanisms comprised of a number of low molecular weight molecules and enzymes. Exposure to certain contaminants can cause elevated concentrations of ROS due to enzymatic and non-enzymatic reactions that either directly generate ROS (e.g. cytochrome P450 reductase activity and redox cycling, respectively) or indirectly increase ROS by interfering with the antioxidant defense system (Kelly *et al*., 1998). When antioxidant defenses can no longer keep ROS concentrations to within a non-toxic range, major biological macromolecules such as DNA, proteins and the phospholipids of membranes can be oxidatively damaged. This situation, where ROS overwhelm antioxidant defenses leading to sub-cellular damage, is called oxidative stress (Kelly *et al*., 1998). Metals have been shown to cause an increase in ROS through a variety of mechanisms including redo cycling and disruptions to antioxidant defenses (Halliwell and Gutteridge, 1999 andLivingstone, 2001).

One of the key low molecular weight molecules that are part of the antioxidant defense system is reduced GSH. This molecule is present in the cytosol, mitochondria and nucleus of virtually all types of cells (Livingstone, 2003). Reduced glutathione can neutralize ROS directly or enzymatically via glutathione peroxidase thereby becoming oxidized in the process. Glutathione reductase uses Nicotinamide Adenine Dinucleotide Phosphate Hydrogen (NADPH) as reducing equivalents to regenerate the reduced form of glutathione (Liviingstone, 2003). The main enzymes directly involved in the antioxidant defense system are glutathione peroxidase, catalase and superoxide dismutase. Glutathione peroxidase has a selenocysteine moiety at its active site that is oxidized upon reacting with inorganic and organic peroxides. Reduced glutathione provides the reducing equivalents necessary for regenerating the active site of the enzyme. There are different forms of glutathione peroxidase and most can be found in the cytosol, mitochondria and nucleus (Halliwell and Gutteridge, 1999). One type of glutathione peroxidase, phospholipid hydroperZoxide glutathione peroxidase, is closely associated with cell membranes and is essential for neutralizing lipid peroxides (Halliwell and Gutteridge, 1999). Measures of reduced, oxidized glutathione and the activity of glutathione peroxidase are common biomarkers of exposure of oxidative stress. Since ROS effectively oxidizes and damages cell membrane phospholipids, lipid peroxidation is a typical biomarker of the effects of oxidative stress (Halliwell and Gutteridge, 1999). As a result of oxidative damage, cell membranes become less fluid and more permeable (Kelly *et al*., 1998) and cell death may occur (Das and Mukherjee2000).

In mammalian studies, ROS and oxidative stress are linked to a number of diseases including cancer (Kelly *et al*., 1998), atherosclerosis, cataracts, rheumatoid arthritis, and neurodegenerative disorders. Studies using fish have demonstrated reduced growth (Fontagné *et al*., 2006), survival and lesions to DNA (Kelly *et al*., 1998) that were associated with oxidative stress. Although oxidative stress may not be the primary mechanism behind some of these effects (Pandey *et al*., 2001), oxidative damage to biological macromolecules signals the importance of ROS and oxidative stress as a mechanism of toxic action of contaminants such as metals and organic pollutants.

2.5.4*Oxidative Imbalance and Antioxidant activity*

The incidence of oxidative imbalances within living tissue is triggered by uptake of xenobiotic substances ranging from metal-ions, pesticide molecules, to contaminants rich inhydrocarbonmolecules (Livingstone, 2003).

2.5.5. ***Environmental Changes, oxidative stress and antioxidant activity in marine biota***

2.5.5.1 *Natural Sources*:Organisms within the marine and coastal habitat range inherently encounter extreme and harsh environmental conditions ranging from frosty and heat stress, dehydration, fluctuating salinity, carbon limitation, and limited access to sunlight in the intertidal regions, and intermittent limited access to O_2. These could act synergistically to induce stress in marine biota (for example UV-B or temperature and salinity fluctuations). The extremes environmental conditionscould also induce a photo-catalysed inhibition of photosynthesis deleterious effects on cellular metabolism in various organisms (Ahmad *etal*., 2006).

2.5.5.1.1 *Temperature fluctuations*: Change in temperature regimes have consequences for that are known to affect anddetermine the survival of all living organisms. Current trends in the concentration of atmospheric CO_2portend that, global temperature will peak by the year 2100 to 2.5-6.4°C in the earths atmosphere (Alexiadis, 2007), and 2-3°C in the ocean surfaces. A rise in average temperature of the environment, could lead to metabolic activation, which in combination with increased in O_2 consumption, triggers oxidative stress (Halliwell and Gutteridge, 2007). Chronic exposure to high temperatures results in crucial modifications of intermediary metabolism and cell membrane properties. Ectoderms from permanently cold waters are less flexible to such variationsin temperature compared to organisms from other regions(Abele and Puntarulo, 2004). Elevated vulnerability of polar animals to metabolic stress via oxygen radicals demands physiological adjustments for antioxidant defense systems to function at low temperatures(Regoli *et al.*, 1997) Moreover, some enzymatic systems including antioxidant enzymes, like SOD, display temperature optimum curves with a maximal activity within the habitat temperature range in temperate ectotherms (Abele *et al.,* 2002).

An essential adaptation to cold temperature involves biochemical compensation to the physiological functioning of cellular membranes. This adaptation is expressed by the production of low molecular weight lipids and the increase in higher levels of unsaturated and branched chain fatty acids in the composition of cellular membranes (Hazel and Williams., 1990). However, achieving membrane functional homeostasis by increasing levels of lipid unsaturation occurs at the expense of enhancing the vulnerability of cellular membranes to oxidative damage. Thus, the biochemical adaptation of cellular membranes to function at low temperature affects a corresponding need for enhanced lipid-phase antioxidant protection, and this is demonstrated by the need for a significant dietary uptake of temperature by coldwater teleost (Woodward.,1994).The biochemical selection for Marine-Derived Tocopherol (MDT) synthesis in cold-water marine producers may thus evolve to provide enhanced antioxidant protection for metabolic adaptation to low temperature (Yamamoto *et al*., 2001).

2.5.5.1.2 *Ultra Violet Rays (UV-R) Solar exposure*: These class of raysattain ecological relevant depths of surface waterwith a wide range of overlapping effects, reaching from producers higher up the food-chain. Organisms which inhabit shallow waters with high water clarity clear, are vulnerable to the deleterious and hazardous wavelengths of solar UVR. Solar UV-B (280-315 nm) radiation at the Earth's surface has been shown to increase due to the ozone depletion and its interplay with climate change (Manney *et al*., 2011). UV-A (315-400 nm) + UV-B is known to inhibit growth and photosynthesis (Helbling *et al*., 1992; Heraud and Beardall., 2000; Gao *et al*., 2007; Jiang and Qiu., 2011) and to damage proteins and DNA (Grzymski.,2001; Xiong., 2001; Gao *et al*., 2008). However, moderate UV-R levels were shown to increase photosynthetic carbon fixation (Nilawati *et al*., 1997; Barbieri *et al*., 2002), with UV-A even driving photosynthetic carbon fixation in the absence of PAR (Gao *et al*., 2007). The absorption of solar radiationby dissolved organic matter within the marine environment, particularly the UV-R wavelengths, triggers the photo catalyzed supply of reactive metabolic intermediates, including ROS (Mopper *et al*., 2000). Hernando *et al. (*2011) demonstrated that reduced vulnerabilityto UV-induced damage is possible via short-term intake of long-term synthesis of mycosporine-like amino acids (MAAs) in some antartic invertebrates. Apart from vertical migration and screening of UVs, some invertebrate crustacean are capable of photorepair of UV-B-induced DNA damage (Grad *et al*., 2003) as shown in species from Patagonia, Argentina (Gonç alves *et al*., 2002).

2.5.5.1.3 *Salinity Variations*: Although marine environments have a stable salinity with values around 35 ups, variations could occur between 10 to 70 ups due to natural processes such as evaporation or precipitation or influx of freshwater from fluvial sources (Graham and Wilcox, 2000). Such sudden fluctuating salinities could result in osmotic stress, which in turn could exert considerable oxidative stress in vulnerable groups of organism i.e. intertidal species. In aquatic organisms, salinity change causes a variety of physiological responses such as plasma enhanced stress-related hormones, altered equilibrium of electrolytes, and growth inhibition (Choi *et al*., 2008). The salinity-related stress has been associated with modulated production of ROS, causing oxidative damage to tissue (Liu *et al*., 2007). Rijstenbil (2003) demonstrated the synergistic potential of UV-B and salt stress (as in immersion) to inhibit growth in diatoms. Elsewhere, studies with microalgae exposed to chronic conditions of hypo-saline and hyper saline conditions also demonstrated high antioxidant activity concurrent with growth inhibitions (Lu *et al*., 2006). Experimental findings from shrimp exposures demonstrated that changes in salinity might be toxic because it can reduce the activities of antioxidant enzymes thereby increasing vulnerabilityto oxidative stress. Vitamin E dietary supplementation can be potentially useful in the prevention of metabolic damage under the tested conditions (Liu *et al*., 2007). Hamer *et al.* (2008) demonstrated a close inverse relationship between salinity and susceptibility of DNA to oxidative damage across seasons. Thus fluctuations in salinity allow for oxidative metabolism, including the lowered antioxidant defense systems and the higher incidence oxidative damage.

2.5.5.1.4 *Oxygen Availability*: Most marine invertebrates exhibit a high-surface to volume ratio, thusdiffusive O_2absoprtion by the teguments critical forfunctional existence within its habitat range. A number of invertebrate species are oxy-conformers where O_2 consumption is varies in response to the prevailing O_2 partial pressure in the environment (Hamer, 1986). Since some of these species are negatively impacted by higher thresholds of O_2, they occupy or resort to environment with lower O_2 (Abele, 2002; Tschischka*et al.*, 2000). Alternatively, adaptiveness in low tide, entails the availability of complex and finely tuned set of mechanisms working in close coordination. In hypoxic conditions (Chandel *et al*., 1998; Chandel and Schumacker, 2000) or following a hypoxic episode (Boveris, 1973; Duranteau*et al*., 1998), ROS are generated in the event of tissue re-oxygenation. Thus, intertidal organisms, which experience intermittent episodes of environmental and physiological hypoxia, are likely to receive modulated levels of ROS production during or, on recovery from physiological stress. In molluscs, oxidative stress during low tides,affects the foot and gills tissues thus explaining the higher activity of antioxidant enzymes compared to other tissues. Intertidal organisms exhibit more rigorous responses to air exposure than sub-littoral limpets, which are able to sustain shell water O_2 pressure at low levels irrespective of being submerged or not. The importance of gills in the antioxidant capability of the organism has been documented. Intertidal limpets for instance, reduce SOD activities, whereasSOD activity in gill of sub-littoral limpets, increases under both forms of stress.

2.5.5.1.5 *Abundance of Chemical Elements in Nature*: The toxic outcomes of heavy metal exposure and uptake has been strongly attributed to the post-exposure generation of ROS and the resultantdisequilibriumin cellular redox status (Pinto *et al*., 2003). Iron is a Fenton (Ft) reactant, which, is not tightly bound to Ft, has the capacity to form •OH (Puntarulo and Cederbaum, 1988). The manner and dimensions in which the oxidative metabolism of an organism responds to the incidence of high chemical elements, depends on certain variables including element type, bioavailable concentration, exposure period, and metabolic/excretion efficiency of these elements by the organism. Reports about King-George Island (Antarctic) documents a 5-7% occurence of Fe within volcanic rocks (Tatur *et al*., 1999). Processes like glacier melting due to sediment ablation of eroding the rock surface beneath the glacier mass, can result inlarge-scale metal enrichment (Ahn *et al*., 1996; Dierssen, 2002). González and Puntarulo (2011) demonstrated that when earth elements Fe is regularly taken up by the aquatic mollusks, it could eventually become incorporated into the foot. Studies in sub-littoral limpet's species revealed high content of heavy metals (Fe, Al, Zn) in digestive glands attributed to ingested sediments, which was also associated with elevated incidence of ROS compared to intertidal animals. In addition, there was a concurrent increase in SOD activity following the modulated generation of ROS formation in the sub-littoral group (Weihe *et al*., 2010). The differential stress responses of both exposure sub-groups indicate physiological tendencies for different metabolic strategies, which most likely could be traced to genetic profile (Aranzamendi, 2008).

2.5.5.1.6 *Pathogen Invasion*: The role of parasitism in ecosystems is well documented as a tool for regulating the dynamics of various benthic invertebrates that inhabit coastal areas. The incidence of antioxidant activity and alongside incidence of oxidative cellular damage in coral larvae under pathogenic and unpathogenic conditions attributes tissue damage to parasite presence (Yakovleva *et al*., 2009). Moreover, Neves *et al.,* (2000)examined a group of crustaceans infected by the isopod *Probopyrus ringueleti*, a gill chamber parasite with the potential for metabolic disruption. Although, activities of CAT or GPx was not significantly different across groups, the activity of SOD activity reduced significantly in the infected shrimp. Similarly, the oyster parasite *Polydora* sp., alters the respiratory physiology of the mollusks, and modulates the incidence of oxidative stress by inhibiting the expression SOD within gill tissue (Chambon*et al*., 2007).

2.5.5.2 Unnatural Sources: Studies have attributed the toxicity of pollutants to be largely dependent on their inherent potential to modulate cellular levels of ROS(Jerome *et al*, 2016). This is plausible because physiological disruptions in organisms can be related to incidences of exposure and uptake of toxic chemicals in surface water. Groups like microalgae are particularly interesting indicator species for habitat quality because they are spans a wide range of environments and a critical place at the base of the food chain (Torres *et al*, 2008). The incidence of oxidative stress in invertebrates and fishes has been studies progressively for decades and is being harnessed as a possible sentinel for monitoring altered habitat quality in coastal and more remote environments (Regoli*et al*., 1997; Ahn *et al.,* 1996; Kirchin *et al*.1992; Regoli, 1992; Palace and Klaverkamp,1993; Pellerin-Massicote,1994; Regoli *et al*., 1998; Angel *et al*., 1999).

2.5.5.2.1Deposition of Heavy Metals from Human Activities: Anthropogenicactivities have been implicated in the increased occurrence of toxic metals in coastal environments. For instance increased mining and industrial activities, disposal of large quantities of waste from chemical industries, large scale agricultural and the increased run off of pesticides due to large quantities applied, indiscriminate domestic garbage dumps, are typical scenarios of anthropogenic impacts. aeolian deposition of atmospheric dust, from polluted areas may also introduce metals to the antioxidants, including diverse enzymes such as SOD, CAT, GPx and Px, and the synthesis of low molecular weight compounds such as carotenoids and GSH (Pinto *et al.,*2003). Cu has been observed to induce metallothionein gene expression in the seagrass *Posidonia oceanic* (Linnaeus) (Giordani *et al*., 2000) and in the brown alga *Fucus vesiculosus* (Linnaeus) (Morris *et al.,* 1999), and malondialdehyde (MDA) generation in the marine diatom *Phaeodactylum tricornutum* (Bohlin) (Liping and Binghui, 2008). In the marine dinoflagellate *polyhedron*(Fasten), heavy metals cause increased oxidation of proteins and lipids, levels of SOD, APx and carotene; and a decrease in GSH content (Okamoto *et al*., 2001).

Lately, studies have demonstrated higher incidence of metals such as Fe, Zn, Cu, Cd and Pb in tissues of organisms sampled from impacted sites (Giarratano *et al*., 2013). Reports of aquatic organism to develop tolerance in degraded environments have also been reported.

2.5.5.2.2 *Presence in Seawater of Hydrocarbons Due to Oil Manufacturing*: Contaminant presence strongly attributable to oil spill and discharge incidences includes Polycyclic Aromatic Hydrocarbons (PAH), alkylphenols, and hydrocarbons (Sturve *et al.,* 2006). These compounds have low vapor pressures (log K> 5); therefore, they are rapidly absorbed by particulate matter and by living organisms (Nielsen *et al* 1997). Exposure to several PAH causes oxidative stress in aquatic organisms (Winston and Di Giulio,1991; Livingstone, 2001). Liping and Binghui (2008) using fluoranthene expose diatoms, demonstrated a time-dependent increased concentration of MDAin algal cells with increasing exposure concentrations. Sureda *et al.* (2011) demonstrated significant increases in activities of Glutathione-S-transferase (GST) and cytochrome P4501A and metallothionein gene expression in the digestive gland of wild mussels *M. galloprovincialis* following a major oil spill accident. Elsewhere, studies with the Atlantic Cod*Gauds morhua* (Linnaeus) demonstrated that exposure to alkylphenols induced elevated levels of GR and total GSH levels, an observation that was attributable to induced oxidative stress (Hasselberg et *al*, 2004).

2.5.5.2.3 *Industrial and Urban Wastes*: Anthropogenic wastes span a wide variety of types ranging from industrialwastewater and domestic and municipal effluents, storm-water runoff, dumpsite and garbage leachates, to agricultural wastes. Key contaminants that originate from municipal effluents include metals, hydrocarbons, and nutrients (Moore *et al*, 2004). The absenceof sewage processing have also been implicated in the large amount of contaminants being released to the environment (Esteves *et al*., 2006). Biomarkers of oxidative stress in gills and digestive gland of mollusks have been successfully appliedas indices of altered habitat quality following the release of heavy metals, inorganic nutrients and particulate organic matter from various anthropogenic activities (Duarte *et al*, 2011).

2.5.5.2.4 *Pesticides*: Pesticides is a broad name for agents i.e. physical, chemical or biological designed to eliminate undesirable occurrence of plant or animals. The syntheticproperties of most of these agents poses relatively unpredictable risks for biological systems particularly aquatic habitats. Intensive agricultural setups, could significantly contribute to the incidence of a variety of contaminantswhich leach from fertilizers and pesticides applied during rains that runoff into adjacent surface water in inland and coastal environments. This could impact resident organisms and biota via toxic effects and induction of oxidative stress (Lushchak, 2011). A recentstudy demonstrated the relationship between pesticide exposure and ROS generation using benthic mollusks. An increase in theROS generation within heamocytes was recorded after paraquat exposure. (Gómez-Mendikute and Cajaraville, 2003)

2.6 Mechanism of metal bioaccumulation in fish

A number of factors such as sex, age, season, spawning period, variability of food habitats and pollutant exposure and phylogenetic differences in regulatory mechanisms, may influence the uptake, retention and bioaccumulation of trace contaminants in fish tissues (Nesto *et al*., 2007). Zhao *et al*., (2012) shown correlation of heavy metals in the tissue of fish to their living environments both qualitatively and quantitatively and there was diverse metal bioaccumulation characteristics which was significantly affected by environment factors and living habits. The bioaccumulation model showed that Uptake Efficiency factor of essential heavy metals such as Cu and Zn decreases as exposure concentration increases, due to homeostasis regulation while for non essential heavy metal Hg, it is increases as the exposure concentration increases and excretion was observed as manifestation of homeostasis regulation (Neogrohati, 2006).

2.7 Mechanism of Histopathological Damage

Histopathological damage in tissues is outcome of various biochemical and physiological interactions within cell owing to exposure to various xenobiotics. Heavy metals generates Reactive Oxygen Species (ROS) which damages protein, lipid and DNA content of exposed animal which on gross level can be visualized through histopathology, Heavy metals grouped as Redo active (Fe, Cu, Cr etc) undergo redox cycling whereas redox inactive metals (such as Pb, Cd and Hg) undergo covalent electron sharing with cells major antioxidant enzymes (Thiols). Both types lead to the production of ROS as hydroxyl radical (OH), Superoxide radical (O_2^{--})or hydrogen peroxide (H_2O_2) which deplete cells intrinsic antioxidant defense. ROS lead to lesions to lipid, protein and DNA which can be visualized through cross index i.e., histopathology of tissues (Ercal *et al.,* 2001). Histopathology is a broader term and mirror of effects of exposure to a variety of anthropogenic pollutants (Hinton *et al.,* 1992). Histological responses are relatively easily recognized provided that proper reference and control data are available (Hinton, 1994). Histopathology thus is a long term and reliable biomarker or toxicant exposure. Heavy metals undergo metabolic activation that induces a cellular change in affected fish. The tissue lesions and apoptosis arises from bioaccumulation stimulate necrotic alterations in the fish with-.an inflammatory defensive reaction (Roganovic-Zafirova *et al..,* 2003). Below are given few mechanistic insight of metal toxicity leading to microscopically visible alterations.

Heavy metal ions can enter blood vessels some of them arc carried by proteins like albumin and can be taken up by endothelial cells lining the vessels. Heavy metal ions induce mechanisms of gene activation in endothelial cells as do pro inflammatory mediators, indicating that corroding metal ion containing biomaterials can provoke, inflammatory reactions by known, as well as by yet unknown, intracellular signaling pathways (Wagner *et al.,* 1998). And thus blood profile, changes with respect to heavy metal exposure and become sensitive bioindicator of heavy metal pollution as also shown by many workers (Kori-Siakpere and Ubegu, 2008; Maheswaran *et al.,* 2008). Teleost liver is major organ for heavy metal metabolism thus frequently studied by many workers (Canli *et al.,* 1998; Javed, 2005: Vinodhini and Narayanan, 2008) to observe different deformities. Fish hepatocyte has relatively more glycogen/lipid content which lead to hepatocytes more vacuolated (Weber and Gingerich, 1982). Macrophage aggregates act as repositories for product of coil membrane and erythrocyte breakdown include lipofuscin, corroid, hemosiderin and melanin (Wolke, 1992). Reason behind hepatocellular enlargement is organelle proliferation (hypertrophy), failed mitotic division of hepatocytes (megalocytosis) and vacuolar swelling of endoplasmic reticulum cisternae that is hydropic degeneration(Hinton *et al.,* 1992). Toxic chemicals lead to increased number of organelles such as myelinated bodies and mitochondria.

CHAPTER THREE

MATERIALS AND METHODS

3.1 Study Area

Lagos lagoon is the largest lagoon system of the Gulf of Guinea coast in West Africa. It stretches from Cotonou in the Republic of Benin and extends to the fringes of Niger Delta in Nigeria along its 257km course (Hill and Webb, 1958; Don-Pedro *et al*., 2004). The Lagoon lies between longitude 3^0 22' E and $3^0$40'E and latitude $6^0$17'N and 6^0 28'N. It is generally shallow with a depth range between 0.3 and 3.2m with the exception of some dredged parts, notably in the Lagos harbor.

3.2 Study Locations:

The crab, water and sediment samples were collected in six stations in the Lagos lagoon namely; Makoko-site, Okobaba-site, Iddo-site, Ajah-site, Ikoyi- site and Mid-lagoon (control) site which are identified by the different types of anthropogenic activities received (Figure 1).

3.2.1 Makoko

Makoko is a coastal community consisting of wooden ` houses built on water (Plate 1). The major means of livelihood for men was fishing, while the women sell fish, food stuffs, cooked food and provisions. Other profession found in this area was carpentry. Domestic waste and sewage were directly release into the water from different houses, located along the length of the Lagoon

3.2.2 Okobaba

Okobaba was dominated by saw-mill industries, most of which are located near the shore. Smoke from saw-dust dump-sites was usually very thick with floated logs of wood on water (Plate 2). Burnt saw-dust dump-site located at the shore were used as walk path by people to access the floated logs of wood used as stand support while defecating directly into the water.

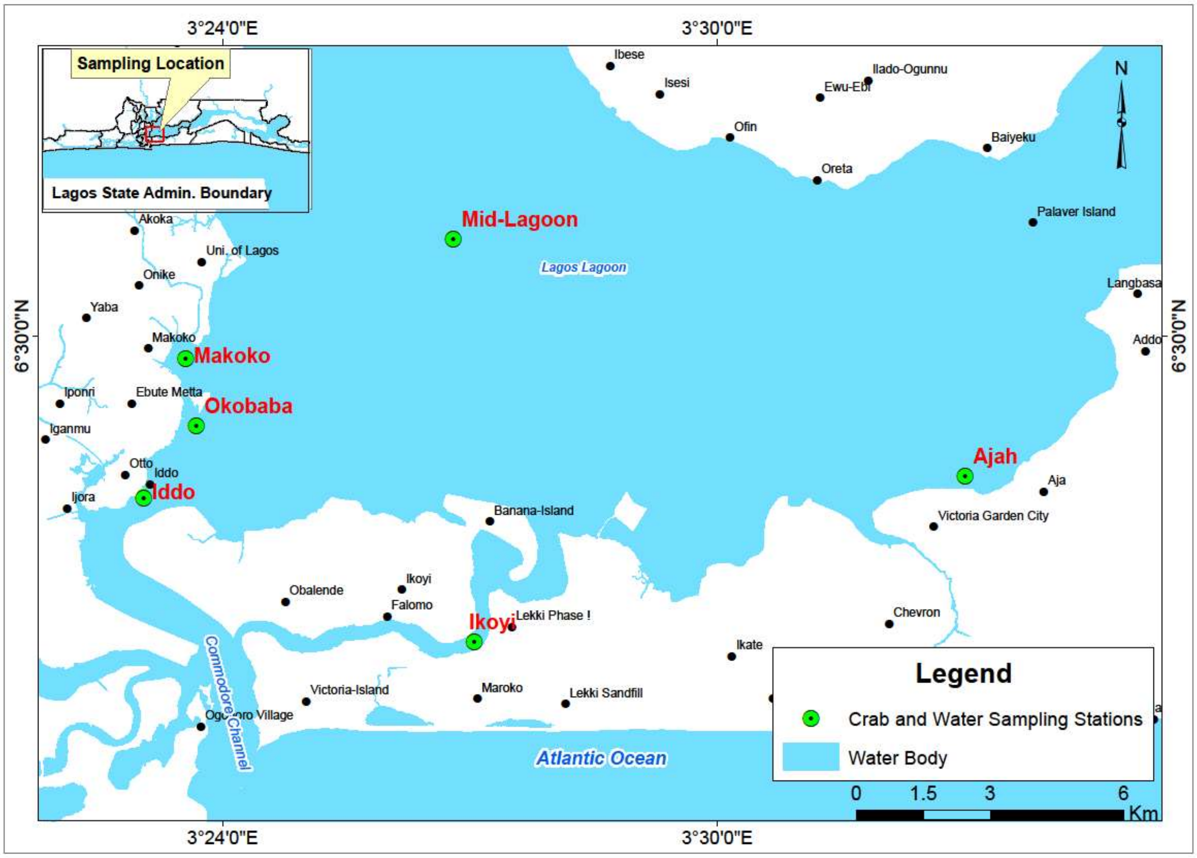


Fig.1. Lagos Lagoon Showing the Sampling Stations


Plate 1: Makoko Settlement with dug-out boat for different activities

Plate 2: Okobaba with Saw-mill Industries and floating logs of wood.

3.2.3 Iddo

Iddo represents a major point-source of sewage disposal into the Lagoon system. Septic tankers discharged untreated sewage daily through pipes into the Lagos Lagoon (Plate 3). There were different sizes of pipes through which the sewage was channeled to the Lagoon. The Lagoon is also a shallow area with a pungent smell.

3.2.4 Ajah

Anthropogenic activities in Ajah included local and industrial dredging. The water was yellowish in colour. Few cases of fishing activities were occured.

3.2.5 Ikoyi

There were clusters of domestic waste such as pure water nylons, plastic bottles, nylon bags, wood, broken plastic plates, and so on at Ikoyi. Solid waste was found lining the shore, while the Lagoon was shallow. There were a few on-going fishing activities sighted.

3.2.6 Mid-Lagoon

There were no human activities within the mid-lagoon sampling station. The water was clear and shallow. Fishing occurred a few kilometers away from the station.

3.3 Sampling and Sample Collection

Sampling was carried out between December 2011 and November 2013 on a monthly basis. The coordinates were determined for each sampling station using the GPS (Garmin etrex legend and magellan exporist 210).

3.3.1 *Crab Sample*: A fiber boat was used for monthly sampling in all the six stations. A crab fisherman was employed for crab fishing. Crabs (n=1750; circular lift nets 50-76mm)were sampled within the ranges of each sampling stations and transported alive to the laboratory (Plate 4)

3.3.2 *Water Sample*: samples (n=432) of surface water were collected from all the stations and fixed with $HN0_3$ for heavy metal analysis. Water temperature was taken in-situ using mercury in a glass thermometer. Water samples for Dissolved Oxygen (DO) and Biochemical Oxygen Demand (BOD) analysis were collected using the DO and BOD bottles. Salinity, conductivity, and alkalinity were taken using Horiba-U-10, while pH was taken using a pH meterH9050.model

Plate 3: Septic tanker packed to release sewage at Iddo

3.3.3 *Sediment Sample*: Clean stainless van-veen grab was used to collect sediment samples in all the stations. The samples were put in polythene nylons properly labeled and transported to the laboratory at the Nigerian Institute for Oceanography and Marine Research (NIOMR) for further studies.

3.4 Laboratory Studies

3.4.1 Morphometric and Meristic Analysis of Blue Crab

The FAO (1995) identification guide was used to identify the crabs. Carapace length and body depth or carapace width (Plate 4) were measured using a vernier caliper. The individual weight was measured using sensitive weighing balance in the Marine Biology Laboratory of Nigeria Institute for Oceanography and Marine Research. The samples were preserved at -21^{0}c in a refrigerator, for further analysis.

3.4.2 Physicochemical Parameters of surface water

Dissolved Oxygen (DO) and BOD were measured using gravimetric method, while salinity, alkalinity, pH, and conductivity levels were taken using the Hanner instrument H9010 model. The colour of the water samples was noted through physical observation. The odours were subjectively perceived.

3.4.3 Dissection and preservation of crab organs

Crab sample were dissected using forcept to remove the selected organs according to the method of Harriet (2001) and preservedat -21^{0}c in a refrigerator, for further analysis.

3.4.4 Heavy metal analysis:

3.4.4.1 *Digestion of Crab Sample*: The crab organs (hepatopancreas, gills, gonad and muscles) were allowed to thaw, after which 5g of hepatopancreas, gills, gonad and muscles of crab were weighed on a sensitive weighing balance into different 100ml beakers and digested with 25ml of freshly prepared concentrated Nitric acid (HNO_3) and Hydrochloric acid (HCl) mixture in ratio 1:1.Digestion was carried out according to standard methods (FAO/SIDA, 2003).

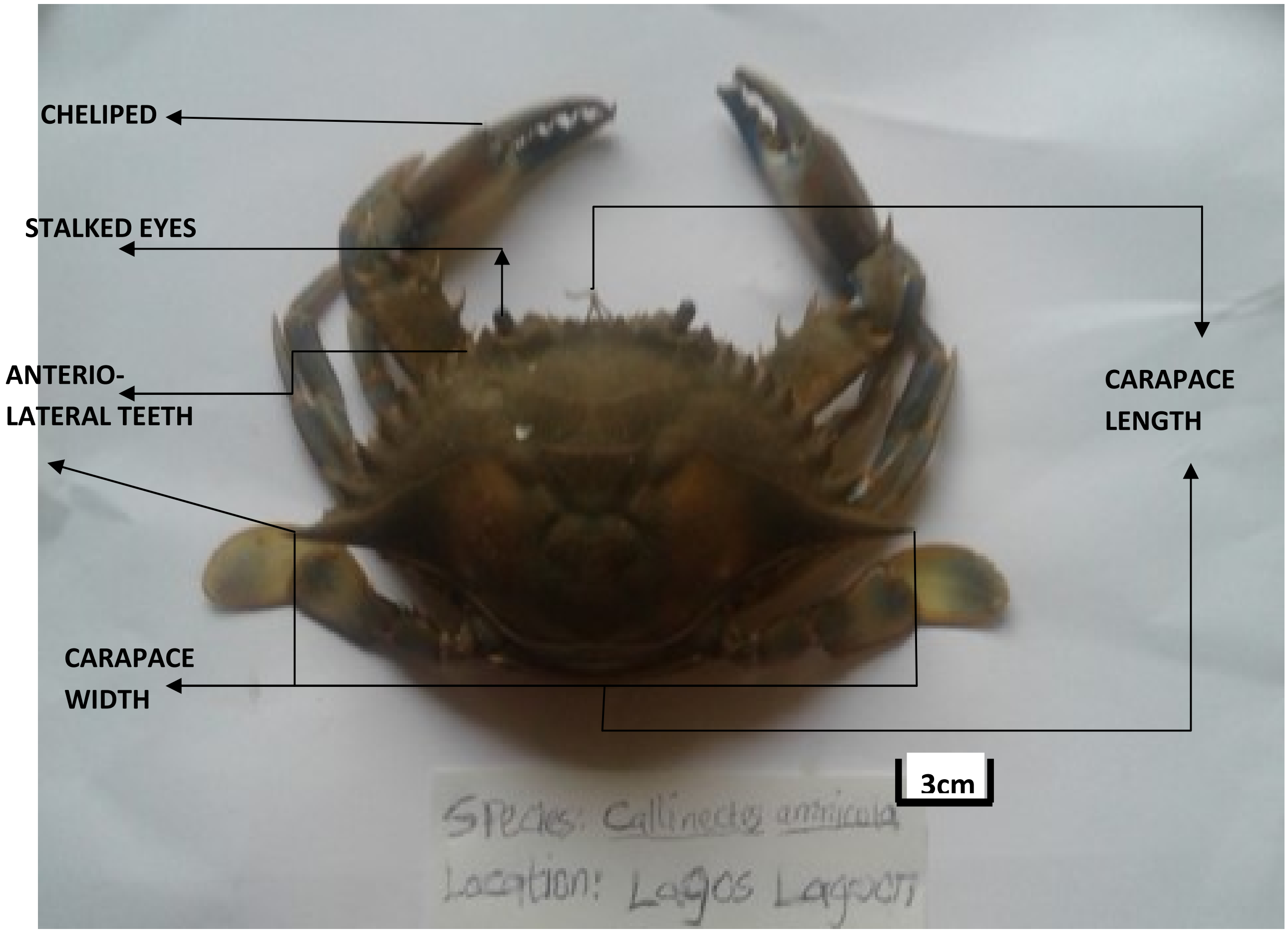


Plate 4: Dorsal view of *Callinectes amnicola*

3.4.4.2 *Digestion of Water sample* : 250ml of water samples for heavy metal analyses was filtered and digested with 25ml of freshly prepared concentrated Nitric acid HNO_3) and Hydrochloric acid (HCl) mixture at a ratio of 1:1. Digestion was carried according to standard methods (FAO/SIDA, 2003).

3.4.4.3 Digestion of Sediment Sample: The bottom sediment samples from each study station weredried in open air and subsequently sieved with a 200mm mesh screen. 5g of the sediment wastaken into 100 mL conical flasks and digested with 25ml of freshly prepared concentrated Nitric acid (HNO_3) and Hydrochloric acid (HCl) mixture at a ratio of 1:1. Digestion wascarried out on electric cooker in a fume cupboard while the temperature gradually roseup to a maximum of 160'C. Heating process was sustained for about 2 hrs, while reducing the volume in the beaker to about 5ml. After the beaker and its contents cooled, the content wastransferred into a 50ml volumetric flask using aWhatman filter paper (filtration) and topped up with distilled water to 50ml mark (FAO/SIDA, 2003)

The digested samples were then analyzed in the Central Laboratory of NIOMR using a Flame Atomic Absorption Spectrophotometer model Varian SpectAA 400 plus AAS with aqueous calibration standard prepared from the stock standard solutions of the respective elements. (APHA-AWWA-WEF, 2005).The results obtained were compared with the NESREA and WHO standard limit.

3.4.5Condition Factor

The Condition Factor (CF) which describes the physiological condition of the crabs (Voight, 2003) was calculated according to the equation given by Busacker *et al*. (1990):

$$CF = W \text{ X } 100 / L^3$$

where, W = the crab weight (g)

L = Carapace length (cm)

Univariate ANOVA analyses were performed to test for differences in Condition Factor index (CF) of the blue crab from the different stations.

3.4.6 Determination of oxidative stress parameters

Biomarker analysis was carried out in the Biochemistry Laboratory of the Nigerian Institute for Medical Research, Yaba, Lagos. The incidence of imbalanced oxidative parameters in the

Hepatopancreas, gill-tissue, gonad and muscle-tissue from *Callinectes amnicola* was analysed by quantifying the activity of the selected antioxidant endpoints; MDA, SOD, CAT,GPx, and GSH. Homogenization: One gram each of the hepatopancreas, gills, gonad and muscles of *Callinectes amnicola* was weighed with OHAUS Sensitive Weighing Balance and homogenized with 9ml of 0.4 M Phosphate buffer using a pestle and mortar. The organism homogenate was centrifuged using Axiom Centrifuge-80 at 3000 r.p.m for 15minutes and the supernatant samples were stored at -20^{0}C for biochemical analysis.

3.4.6.1*Determination of Superoxide Dismutase (SOD)*:The activity of Superoxide Dismutase was also analysed by quantifying the inhibition of auto-oxidation of epinephrine at pH 10.2 at 30^{0}C as described by Magwere *et al.* (1997), and McCord and Fridovish (1989).

Reagent Preparation: Epinephrine, 5.5mg was added to 100mL of 0.05N of HCl. 50mM of Na_2Co_3 buffer was prepared by adding 50g of the salt(Na_2Co_3) which was dissolved in 100mL of distilled water. The chemicals were weighed using sensitive weighing balance.

Procedure: 3.0ml of Na_2CO_3 buffer-solution was added to 0.02ml of homogenated tissue (Tris-Hcl buffer, pH 7.5) and treated with 0.03ml epinephrine reagent. It was centrifuged at room temperature for 10mins at 3,000 rpm. The upper clear supernatant layer was transferred into a 1.5ml cuvette and the absorbance was measured against reference blank at 480nm using a spectrophotometer.

Equation:

$$\text{Superoxide dismutase } (\mu\text{mol/ml}) = \frac{OD/\ E_{480}\ X\ V_T/\ Vs\ \ X\ 10^6}{\text{Total Protein Concentration}}$$

Where OD = Optical density (absorbance)

V_T = Total reacting volume

Vs = volume of the sample

E_{480} = Molar extinction coefficient (4020)

Determination of Total Protein:Total concentration of Protein was determined following specifications of Bradford (1976), and Lowry *et al*. (1951). Reagent Preparation: Biuret reagent: 100mmol of sodium hydroxide, 16mmol of Na-k-tartrate, 15mmol of Potassium iodide, 6mmol of Cupric sulphate. Blank: 100mmol of 1 Sodium hydroxide, 16mmol of Na-k-tartrate. The

Biuret reagent was diluted with distilled water at 1:4; the Blank was also diluted with distilled water at the ratio of 1:4.Standard solution: 58.48g/L of (6g/dl) Protein kit.

Procedure: The diluted biuret reagent was added to 0.02ml of the samples while blank reagent was used for the preparation of the protein standard and left at room temperature. The clear supernatant was transferred into 1.5ml cuvette and the absorbance was measured against reference blank at 546 nm using spectrophotometer.

Total Protein Concentration = A sample ⁄ A standard X Standard Concentration

Where; A = Absorbance

Standard Concentration = 58.48g/L

3.4.6.2 Determination of Catalase (CAT):Activity ofCAT was determined according to the procedure of Clairborne (1995) following the absorbance of hydrogen peroxide at 240 nm, pH 7.0 and 25ºC.

Reagent Preparation: The reaction mixture 1.5ml contained; 1.0ml of 0.01M PH 7.0 phosphate buffer, 0.1ml of tissue homogenate and 0.4ml of 2M H_2O_2.The chemicals were weighed using OHAUS sensitive weighing balance.

Procedure: 1.0ml of phosphate-buffer was introduced into 0.1ml of tissue homogenate (Tris-Hcl buffer, pH 7.0) and treated with 0.4ml of hydrogen peroxide. The reaction was stopped by the addition of 2.0ml of dichromate-acetic acid reagent (5% potassium dichromate and glacial acetic acid were mixed in 1:3 ratio). The clear supernatant was put into a 1.5ml cuvette and quantified by measuring the absorbance was against reference blank at 620nm using Spectrumlab S23A spectrophotometer.

Equation:

$$\text{Catalase } (\mu\text{mol/ml}) = \frac{OD / E_{480} \times V_T / V_s \times 10^6}{\text{Total Protein Concentration}}$$

Where OD = Optical density (absorbance)

V_T = Total reacting volume

Vs = volume of the sample

E_{480} = Molar extinction coefficient (4020)

3.4.6.3 *Determination of Glutathione Peroxidase (GPx)*: Quantification of was carried out according to Rotruck *et al*., (1973), and Ellman, (1959).

10% Trichloroacetic acid== 10g of Trichloroacetic acid (TCA) in 100ml of distilled water. The chemicals were weighed using OHAUS sensitive weighing balance.

Procedure: 0.2ml of phosphate buffer and 0.01ml of sodium azide added to 0.2ml of tissue homogenate, 0.2ml glutathione and 0.1ml hydrogen peroxide were also added to the mixture.

Equation:

Glutathione Peroxidase (µmol/ml) = OD / E_{412} X V_T / Vs X 10^3

Where OD = Optical density (absorbance)

V_T = Total reacting volume

Vs = volume of the sample

E_{412} = Molar extinction coefficient (13600)

3.4.6.4 *Determination of Reduced Glutathione*: Reduced glutathione (GSH) activity was determined by the method of Jollow *et al.,* (1974) and Ellman, (1959).

Reagent Preparation: 10% Trichloroacetic acid== 10g of Trichloroacetic acid in 100ml of distilled water. The chemicals were weighed using OHAUS sensitive weighing balance.

Procedure: Reduced glutathione (GSH) was quantified by following specifications of Jollow *et al.,* (1974), Ellman, (1959). 3ml of the 10% TCA was introducedinto 3ml of homogenate and further centrifuged at 3000rpm for 10min. In addition, 1.0 ml of supernatant was treated with 0.5ml of Elman's reagent 20 and 3.0ml of phosphate buffer(0.2M, pH 8.0), after which the absorbance was determined at 412nm with the aid of spectrophotometer.

quation:

Reduced Glutathione (µmol/ml) = OD / E_{412} X V_T / Vs X 10^3

Where OD = Optical density (absorbance)

V_T = Total reacting volume `

Vs = volume of the sample

E_{412} = Molar extinction coefficient (13600)

3.4.6.5 *Determination of Lipid Peroxidation*: Lipid peroxidation as evidenced by the formation of TBARS was measured in Malondialdehyde (MDA) by the method of Yagi (1998), Ohkawa *et al*. (1979) and Niehaus and Samuelsson (1968).

Reagent Preparation: 0.37% thiobarbituric acid, 15% Trichloroacetic acid and 0.25N HCl was prepared and mixed in the ratio 1:1:1 (TBA-TCA-HCl).

Procedure: 0.1ml of tissue homogenate (Tris-Hcl buffer, pH 7.5) was treated with 2ml of (1:1:1 ratio) TBA-TCA-HCl reagent and placed in a water bath (Uniscope Laboratory water bath SM801A) for 15mins, cooled and centrifuged with Axiom Centrifuge-80 at room temperature for 10min at 3,000 rpm. The clear supernatant was transfer into 1.5ml cuvette and the absorbance was determined against reference blank at 535nm using Spectrumlab S23A spectrophotometer.

Equation:

Lipid peroxidase (MDA-mol^{-1}g) = OD ⁄ E_{535} X V_T ⁄ Vs X 10^6

Where OD = Absorbance

V_T = Total reacting volume

Vs = volume of the sample

E_{535} = Molar extinction coefficient (1.56 x10^5 m^{-1}cm^{-1})

3.4.7 Histopathology

The crabwas dissected with forceps, after which the pieces of gill, hepatopancreas, gonads and muscletissues were cut out and placed in Bouin's fluid for about 6hrs to fix tissue which were then later preserved in 10% phosphate buffered formalin prior to further processing at Pathology Unit of Morbid Anatomy Department, Lagos State Teaching Hospital. The gill, hepatopancreas, gonads and muscle tissues weredehydrated in graded series of ethanol dilutions and subsequently cleared in xylene and embedded in paraffin. Sections of 5µm was stained with Delafield haematoxylin/eosin (H&E) and slides was mounted on slides with DPX mountant for microscopy according to the method of Roberts, (2001).

3.5Data analysis

All data were recorded as Mean and Standard Deviation (Mean±SD). The SPSS version 20.0 was used for analysis. Differences in antioxidant biomarker (SOD, GSH, CAT, GPx and MDA) in gill, gonad, hepatopancreas and muscle of *C. amnicola* were determined using ANOVA at $p<0.05$ and $p<0.001$.

ANOVA was used to verify the seasonal variation in antioxidant biomarker and heavy metal of the crab tissues.

Pearson Correlation statistical analysis was also used to test the association between the variables of biomarkers parameters and heavy metals at $p<0.001$

Discriminant function analysis (DFA) was used to check the relationship between the stations and heavy metal concentration in the organs

The seasonal variation;wet season (April- October) and dry season (November-March) were also determined.

Pollution load Index (PLI): PLI represents the number of times by which the heavy metal concentrations in the sediment exceeds the background concentration, and gives a summative indication of the overall level of heavy metal toxicity in a particular sample. Priju, and Narayana (2009).

PLI of the stations was determined according to the procedure of Tomlinson *et al*.,(1980) using the formula;

PLI=n√(CF1xCF2xCF3x…xCFn.

Where, CF(contamination factor) = C metal / C background value,

n = no. of metals/elements; C_{metal} = metal/elementlevels in sediment sample

Biota Sediment Accumulation Factor (BSAF): This refers to the net uptake and retention of a chemical in an organism from all routes of exposure (diet, dermal, respiratory) and any source (water, sediment, food) as typically occurs in the natural environment. Simply put, a BSAF is expressed as the ratio of the concentration in tissue (mg/kg) to the concentration in sediment (mg/kg) as in equation I below:

BSAF = C_b / C_s, …. (I)

WhereC_b is the concentrations of each trace metal in crab organs, and C_s represents the levels of the trace metal within sediment sample.

CHAPTER FOUR
RESULTS

4.1 Physical Observation of Water and Sediment Sample in the Lagos Lagoon

The colour of the sediment in Iddo was black with sandy texture.The Ajah sediment colour was ash while the texture was sandy.The colour and texture of the sediment in Ikoyi was ash and sandy respectively. The colour and texture of the sediment from Mid-lagoon was ash and muddy respectively.

4.2 Physicochemical Parameters of surface water samples from the Lagos Lagoon

4.2.1 Water Temperature

Mean surface water temperature ranged between 29.71±0.49^{0}C at Ajah and 27.14±0.69^{0}C at Mid-lagoon(Table 4). The values were within limit specified by WHO(2011) but exceeded the allowablethreshold specified by NESREA, (2015). However, variation in water temperature in all the sample stations was insignificant ($p>0.05$).

s4.2.2 Dissolved Oxygen (DO)

The mean DO values ranged between 8.89±2.83mg/L at Ajah and 5.23±2.78 mg/L at Makoko. The values were within limits of the NESREA and WHO standards in all stations (Table 4). Analysis of Variance in values obtained for DO showed significant variations ($p<0.05$) in values between the stations.

4.2.3 Salinity

The mean salinity values ranged between 21.00±6.63$^0/_{00}$ at Iddo and 4.96±4.23$^0/_{00}$ at Ajah. The values were within the recommended salinity for fish and shell fish (Table 4). Variation in salinity from one station to the next was significant ($p<0.05$).

4.2.4 Conductivity

The mean conductivity values ranged between 33.15±8.64µS/cm at Iddo and 8.52±6.92µS/cm at Ajah. These values were below NESREA Maximum standard (70µS/cm) (Table 4). Analysis of Variance in conductivity values showed significant differences ($p<0.05$) between the stations.

Table 4: Physicochemical Parameters of Surface Water

Sampling stations	Parameters						
	Temperature	DO*	Salinity*	Conductivity*	pH*	BOD*	Alkalinity*
	(°C)	mg/L	o/∞	µS/cm		mg/L	mg/L
Makoko	29.29±0.49	5.23±2.78	4.2±5.77	22. 20±9.20	7.71±0.32	2.50±1.22	27.00±11.08
Ok obaba	28.71±0.49	5.63±3.62	4.79±4.49	23.66±7.05	7.71±0.23	4.70±2.41	24.14±9.75
Iddo	29.14±0.38	6.19±4.85	21.00±6.63	33.15±8.64	8.03±0.32	2.03±0.63	25.43±3.26
Ajah	29.71±0.49	8.89±2.83	4.96±4.23	8.52±6.92	7.85±0.20	3.07±2.17	2.24±4.07
Ikoyi	29.14±0.38	6.39±3.62	15.07±4.77	21.59±9.74	8.11±0.16	2.17±0.55	14.43±6.24
Mid-lagoon	27.14±0.69	6.16±3.63	6.96±3.62	16.14±9.86	7.73±0.35	2.87±1.90	17.71±8.54
NESREA Limit, (2015)	<26	≥4	__	70	6.5-8.5	30	100
WHO, (2011)	40	**≥6**	__	__	6.8	__	100

*** difference in mean surface water properties are significant at $p<0.05$ between stations.**

4.2.5 Hydrogen Ion Concentration (pH)

The mean pH values ranged between8.11±0.16 at Ikoyi and 7.71±0.23 at Okobaba. These values were found to fall within WHO and NESREA standards limits of 6.8 and 6.5-8.5 respectively.The mean values at different sampling stations were above WHO pH limit of 6.8 (Table 4). Analysis of Variance in pH values showed significant differences ($p<0.05$) between the stations.

4.2.6 Biochemical Oxygen Demanded (BOD)

The mean BOD values ranged between 4.70±2.41mg/L at Okobaba and 2.03±0.63mg/L at Iddo. These values were within NESREA permissible limit (Table 4). The analysis of Variance showed significant variations ($p<0.05$) in BOD values from the sampling stations.

4.2.7 Alkalinity

The mean alkalinity values recorded ranged between 27.00±11.08mg/L at Makoko and 12.24±4.07mg/L at Ajah. These values were within the permissible limit of the NESREA and WHO standards (Table 4). Variation in alkalinity between the sampling stations was significant ($p<0.05$).

4.3 Concentration of Selected Metals in Surface Water

Levels of selected metals concentrations in samples of surface water collected across sampling sites (Table 5) show variations in the distributions of Cd, Pb, Zn, and Cu. Cadmium has the highest mean concentration value in surface water from Okobaba sampling station (0.4±0.02µg/L), the lowest levels of occurrence was found among samples `Makoko and Mid–lagoon (0.02±0.02µg/L). Pb has the highest mean concentration value in surface water from Ikoyi (0.30±0.02µg/L), the lowest concentration was found in Iddo (0.001±0.00µg/L). Zn has highest concentration value in surface water from Ikoyi (0.003±0.008µg/L) while the lowest mean concentrations were found at Mid–lagoon, Iddo and Okobaba (0.001±0.000µg/L). Cu has highest concentration values in surface water from Iddo (0.08±0.08µg/L) while the lowest is (0.01±0.01µg/L) from Makoko.

Cadmium levels in surface water across sampling stations showed consistency with the NESREA permissible limit ex\pt at Okobaba, but they all exceeded WHO permissible limit. Lead concentration in water were within the NESREA permissible limit in Okobaba, Iddo and Mid-lagoon but exceeds this limit in other stations. Concentration of Pb at all sampling stations exceeded WHO permissible limit. Zinc and Cu concentration from all sampling stations were within the NESREA and WHO permissible limits (Table5)

4.4 Trace metal levels in sediment-matrices

The levels of selected metals insediment samples collected across sampling locations are represented in Table 10a. Cadmium concentration in sediment from Makoko was highest (2.73±1.16mg/kg) and lowest in sediment from Mid-lagoon (0.21±0.44mg/kg). mg/kg Zinc concentration was highest in sediment from Okobaba (37.10±27.37mg/kg) and lowest in sediment from Ikoyi (2.28±2.83mg/kg). Copper concentration was highest in sediment from Iddo (60.05±53.89 mg/kg) and lowest in sediment from Ikoyi (2.51±3.88mg/kg) (Table 6a).

Cadmium concentration in sediment from Makoko, Iddo and Ikoyi were within CSOG (0.99-3-moderately polluted) permissible limit except at Mid-lagoon, Okobaba and Ajah, which shows that the sediment from Makoko, Iddo, and Ikoyi was moderately polluted. In contrast, Cd concentration in sediment from all stations were below EPA permissible limit. Lead concentration in sediment from all stations were below CSOG permissible limit (40-non polluted), except at Iddo(51.94±45.16mg/kg) which shows the sediment were not polluted. However, Iddo station was moderately polluted. Pb in sediment from all stations were below EPA permissible limit except at Iddo, which was slightly polluted. Zinc concentration in sediment from all stations were below CSOG and EPA permissible limits, except Okobaba and Iddo which exceed EPA (<25) permissible limit. Copper concentration in sediment from all stations were above CSOG and EPA permissible limits, except at Mid-lagoon Ikoyi and Ajah, thus confirming that shows that Makoko Okobaba and Iddo were moderately or slightly polluted respectively. Copper concentration in sediment from Iddo which was above EPA permissible limit (>50) shows that Iddo station was severely polluted (Table 6b).

Analysis of variance (ANOVA) of Cd, Zn and Cu concentration in the sediment sample shows significant variation at 95% ($p<0.005$) and 99% ($p<0.0001$) within the sediment tested between the sample stations. The variation in Pb concentration shows no significant difference at ($p>0.05$) in all the sample stations.

Table 5: Mean Heavy Metal levels (mg/L) in Surface Water of the Sampling Stations

Sampling stations	Heavy Metals			
	Cd	**Pb**	**Zn**	**Cu***
Makoko	0.02±0.02	0.23±0.25	0.02±0.00	0.02±0.01*
Okobaba	0.4±0.02	0.12±0.14	0.01±0.001	0.02±0.04*
Iddo	0.05±0.03	0.14±0.00	0.01±0.00	0.08±0.08*
Ajah	0.05±0.09	0.26±0.24	0.02±0.02	0.02±0.03*
Ikoyi	0.03±0.02	0.30±0.22	0.03±0.002	0.04±0.06*
Mid-lagoon	0.02±0.02	0.18±0.16	0.001±0.000	0.07±0.06*
NESREA, (2015)	0.1	0.2	2	0.1
WHO, (2011)	0.003	0.01	3	2

***values are significant at P<0.05.**

Table 6a: Mean Heavy metal levels (mg/kg) in Sediment samples from Lagos lagoon

Sampling stations	Heavy Metals types			
	Cd*	**Pb**	**Zn***	**Cu***
Makoko	2.73±1.16	34.24±21.33	20.73.±18.31	32.94±22.67
Okobaba	0.57±0.56	37.10±27.37	37.10±27.37	35.50±30.03
Iddo	1.47±1.37	51.94±45.16	33.78±15.50	60.05±53.89
Ajah	0.98±0.76	21.29±19.90	4.38±2.60	15.73±10.59
Ikoyi	1.99±1.41	12.61±11.34	2.28±1.83	2.51±1.88
Mid-lagoon	0.21±0.14	10.63±8.83	7.21±5.94	5.58±3.41

***values are significant at P<0.05.**

Table 6b: Standard Limits for heavy metal concentration (mg/kg) in Sediment

Standard Category	Cd*	Pb	Zn*	Cu*
CSOG, (2003)				
Non-polluted	<0.99	<40	<90	<25
Moderately polluted	0.99-3	40-70	90-200	25-75
Heavily polluted	>3	>70	>200	>75
EPA,(1999)				
Non-polluted	<40	<40	<25	___
Slightly polluted	___	40-60	90-200	25-50
Severely polluted	>6	>60	>200	>50

CSQG -- Canadian Sediment Quality Guidelines, EPA –Environmental Protection Agency guidelines.

4.5Pollution Load Index (PLI)

The dry season PLI contamination order based on overall concentrations of metals in relation to seasons for the sample stations (Fig 10) was highest at Makoko (1.90) followed by Okobaba (1.19), Iddo (1.09), Ajah (0.72) , at Ikoyi (0.35), and the least was Mid-lagoon (0.30). During the rainy season, highest was at Iddo (1.48), followed by Makoko (0.87), Okobaba (0.6), Ikoyi (0.35), Ajah (0.27) and least (0.24) was at Mid-lagoon. Comparatively Makoko was the most polluted station of the Lagos lagoon in the dry season with about 6 times more metal load than the least PLI (Ikoyi). The Iddo part of the Lagos lagoon was the most polluted during the rainy season with about 5 times more pollutant than the least PLI at Ajah area of the lagoon. Makoko, which was the most polluted station in the dry season (1.90), showed about a 2-fold lower concentration in the rainy season (0.87). Okobaba stations showed overlapping similarities with its PLI-values for the rainy season (0.60) being about half of values obtained for the dry season (1.19). The Iddo station as well as Ikoyi station showed deviation in this trend of more polluted rainy season than dry season, giving its highest PLI (1.48) in the rainy season and lower values (1.09) in the dry season for Iddo and rainy season PLI was 0.33 while dry season PLI was 0.3 for Ikoyi (Fig 2).

4.6Heavy Metals Concentration in samples of *C. amnicola* Organs:

The mean values of some heavy metals concentrations in theHepatopancreas, gills, gonad and muscle of *C.amnicola* sampled across stations generally showed varied distributions of cadmium, Pb, Zn, and Cu. The difference in occurrence were highly significant ($p<0.05$) across the sampling stations.

4.6.1. Hepatopancreas

Cadmium (Cd) concentration in hepatopancreas of crab from Iddo was highest (1.18±0.11mg/L) and lowest in hepatopancreas of crab from Mid-lagoon (0.06±0.22mg/L). Lead (Pb) concentrationwashighest in Hepatopancreas of crab from Okobaba (5.17±7.67mg/L) and lowest in hepatopancreas of crab from Mid-lagoon (0.03±0.15mg/L). Zinc (Zn) concentration was

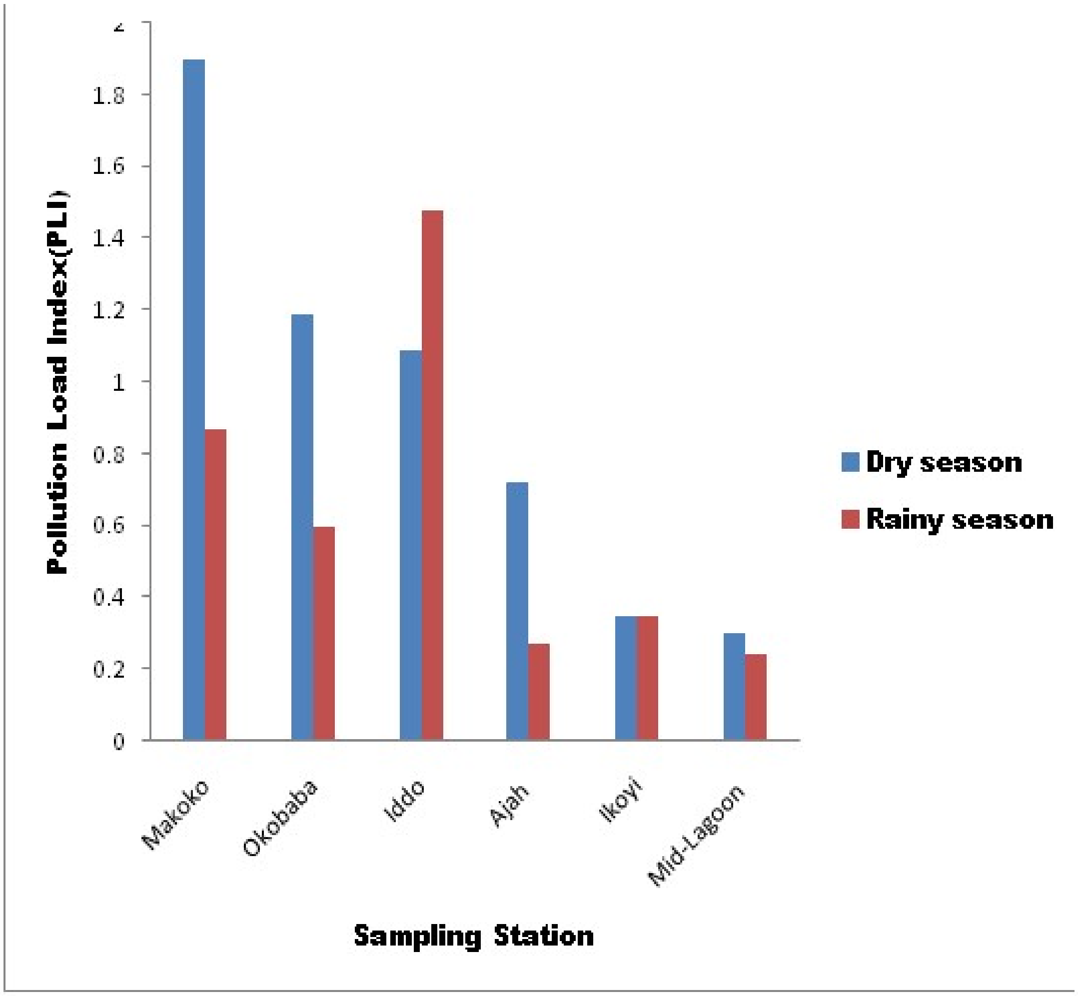


Fig 2: Heavy metal concentration in sediment in relationto the sampling station

highest in hepatopancreas of crab from Ajah (6.83±1.85mg/L) and lowest in hepatopancreas of crab from Mid-lagoon (2.69±1.25mg/L). Copper (Cu) concentration was highest in hepatopancreas of crab from Okobaba (14.64±9.61mg/L) and lowest (3.18±0.82mg/L) in hepatopancreas of crab from Mid-lagoon (Table 7).

Cadmium, Pb, Zn and Cu concentrations in hepatopancreas of crab from all the sample stations except in crab from Mid-Lagoon were above the NESREA and WHO permissible limit (Table 7). However, Mid-lagoon values are also higher, though negligible except in Zn concentration.

The analysis of variance showed that the variation in concentrations of Cd, Pb, Zn, and Cu in the hepatopancreas of *C. amnicola* from across stations were significant at ($p<0.05$).

4.6.2 Gill

Cadmium concentration in gills of crab from Makoko was highest (0.81±1.98mg/L) and lowest in gills of crab from Mid-lagoon (0.10±0.06mg/L). Lead concentrationwashighest in gills of crab from Okobaba (3.17±2.08mg/L) and lowest in gills of crab from Mid-lagoon (0.19±0.79mg/L). Zinc concentration was highest in gills of crab from Iddo (1.68±1.25mg/L) and lowest in gills of crab from Mid-lagoon (1.18±0.23mg/L). Copper concentration was highest in gills of crab from Ajah (34.59±24.54mg/L) and lowest in gills of crab from Mid-lagoon (1.48±2.02mg/L) (Table 8).

Cadmium, Pb, Zn and Cu concentrations in gill tissue of *C. amnicola*across sample stations except Mid-Lagoon were above the NESREA and WHO permissible limit (Table 8).

4.6.3 Gonad

The analysis of variance showed that the variation in concentrations of Cd, Pb, Zn, and Cu in the gills of *C. amnicola* across sampling sites were significant ($p<0.05$).

Cadmium concentration in gonad of crab from Makoko was highest (1.66±1.54mg/L) and lowest in gonad of crab from Mid-lagoon (0.05±0.28mg/L). Lead concentrationwashighest in gonad of crab from Ikoyi (3.83±6.92mg/L) and lowest in gonad of crab from Mid-lagoon (0.14±0.28mg/L). Zinc concentration was highest at Ikoyi (3.89±2.56mg/L) and lowest in gonad of crab from Mid-lagoon (1.23±0.26mg/L). Copper concentrationwas highest in gonad of crab from Makoko (40.24±55.08mg/L) and lowest in gonad of crab from Mid-lagoon (1.64±3.47mg/L) (Table 9).

Table 7: Heavy Metal Concentration (mg/L) in Hepatopancreas of *C.amnicola*

Sampling stations	**Cd ***	**Pb***	**Zn***	**Cu***
Makoko	0.45±0.29	2.82±1.77	6.27±2.04	6.57±6.05
Okobaba	0.39±0.20	5.17±3.67	6.24±2.15	14.64±9.61
Iddo	1.18±0.11	3.07±1.65	5.71±2.91	5.71±2.91
Ajah	0.43±0.40	3.30±1.31	6.83±1.85	8.35±05.57
Ikoyi	0.57±0.28	2.11±02.97	5.71±2.40	13.57±11.51
Mid-lagoon	0.06±0.02	0.03±0.15	2.69±1.25	3.18±0.82
NESREA, (2015)	0.1	0.2	2	0.1
WHO, (2011)	0.003	0.01	3	0.1

*** Significant heavy metals concentration (P<0.05) between stations.**

Table 8: Heavy Metal Concentration (mg/L) in Gill of *C.amnicola*

Sampling stations	Cd*	Pb*	Zn*	Cu*
Makoko	0.81±0.48	1.44±1.01	1.62±1.38	26.08±17.34
Okobaba	0.37±0.21	3.17±2.08	1.67±0.62	29.48±18.59
Iddo	0.45±0.25	1.11±1.01	1.68±1.25	33.44±18.93
Ajah	0.27±0.12	1.56±0.63	1.43±0.55	34.59±24.54
Ikoyi	0.33±0.24	2.04±0.82	1.50±0.50	31.43±15.92
Mid-lagoon	0.10±0.0	60.19±0.09	1.18±0.23	1.48±1.02
NESREA, (2015)	0.1	0.2	2	0.1
WHO, (2011)	0.003	0.01	3	0.1

*** Significant heavy metals concentration (P<0.05) between stations.**

Table 9: Heavy metal Concentration (mg/L) in Gonad of *C.amnicola*

Sampling stations	Cd*	Pb*	Zn*	Cu*
Makoko	1.56±1.44	1.66±1.54	3.00±1.23	40.24±25.08
Okobaba	0.73±0.7	21.14±0.69	2.78±1.66	23.96±19.98
Iddo	0.90±0.76	1.12±1.05	2.93±1.94	19.32±13.20
Ajah	1.35±0.53	1.16±0.80	2.59±2.04	20.83±15.48
Ikoyi	1.02±0.98	3.83±0.92	3.89±2.56	27.37±11.64
Mid-lagoon	0.05±0.02	0.14±0.08	1.23±0.26	1.64±1.47
NESREA, (2015)	0.1	0.2	2	0.1
WHO , (2011)	0.003	0.01	3	0.1

*** Significant heavy metals concentration (P<0.05) between stations.**

Cadmium and Pb concentrations in gonad of crab from all stations were above the NESREA permissible limit except Mid-lagoon. However, the concentrations were above WHO permissible limit in all stations. Zinc concentration from all stations were above and within NESREA and WHO permissible limit except at Mid-Lagoon and Ikoyi respectively. Cu concentration from all stations were above NESREA and WHO permissible limit (Table 9).

The ANOVA showed that metal (Cd, Pb, Zn, and Cu) levels in gonads of *C. amnicola* from across sites were significantly different ($p<0.05$).

4.6.4 Muscle

Cadmium concentration in muscle of crab from Makoko was highest (0.36±0.65mg/L) and lowest in muscle of crab from Mid-lagoon (0.08±0.13mg/L). Lead concentrationwashighest in muscle of crab from Iddo (3.66±7.42mg/L) and lowest in muscle of crab from Mid-lagoon (0.29±0.31mg/L). Zinc concentration was highest in muscle of crab from Makoko (3.28±1.74)mg/L and lowest in muscle of crab from Mid-lagoon (1.24±0.52mg/L). Copper concentrationwas highest in muscle of crab from Ajah (12.06±3.27mg/L) and lowest in muscle of crab from Mid-lagoon (1.18±1.35mg/L) (Table 10).

Cadmium and Pb concentrations in muscle of crab from all stations were above the NESREA permissible limit except at Mid-lagoon, and were all above WHO permissible limit. Zinc concentration from all stations were above WHO permissible limit except Makoko. Copper concentration in muscle of crab from all stations were above the NESREA and WHO permissible limit except Mid-lagoon which was below WHO limit.(Table 10).

The analysis of variance showed that the concentrations of Cd, Pb, Zn, and Cu in the muscle of *C. amnicola* from Makoko, Okobaba, Iddo, Ajah, Ikoyi and Mid-Lagoon were significantly different ($p<0.05$).

4.6.5 Heavy Metals Trend in Select tissues of Crabs across Sampling Stations

Higher mean concentration of Cadmium was found in gonad of blue crab followed by concentration in the hepatopancreas, gill and least concentration was in crab muscle (Fig 3).

Higher Pb concentration was recorded in hepatopancreas followed by gonad, gill and least concentration was in muscle (Fig 3).

Table 10: Heavy metal Concentration (mg/L) in Muscle of *C.amnicola*

Sampling stations	Cd*	Pb*	Zn*	Cu*
Makoko	0.36±0.15	2.30±1.40	3.28±1.74	9.95±4.73
Okobaba	0.32±0.16	1.44±0.83	1.94±1.08	6.67±3.22
Iddo	0.20±0.11	3.66±2.42	2.56±0.81	9.70±3.64
Ajah	0.31±0.13	1.16±1.15	2.31±0.43	12.06±3.27
Ikoyi	0.21±0.20	1.32±0.71	2.78±1.25	11.38±0.82
Mid-lagoon	0.08±0.03	0.29±0.11	1.24±0.52	1.18±1.35
NESREA , (2015)	0.1	0.2	2	0.1
WHO, (2003)	0.003	0.01	3	0.1

*** Significant heavy metals concentration (P<0.05) between stations.**

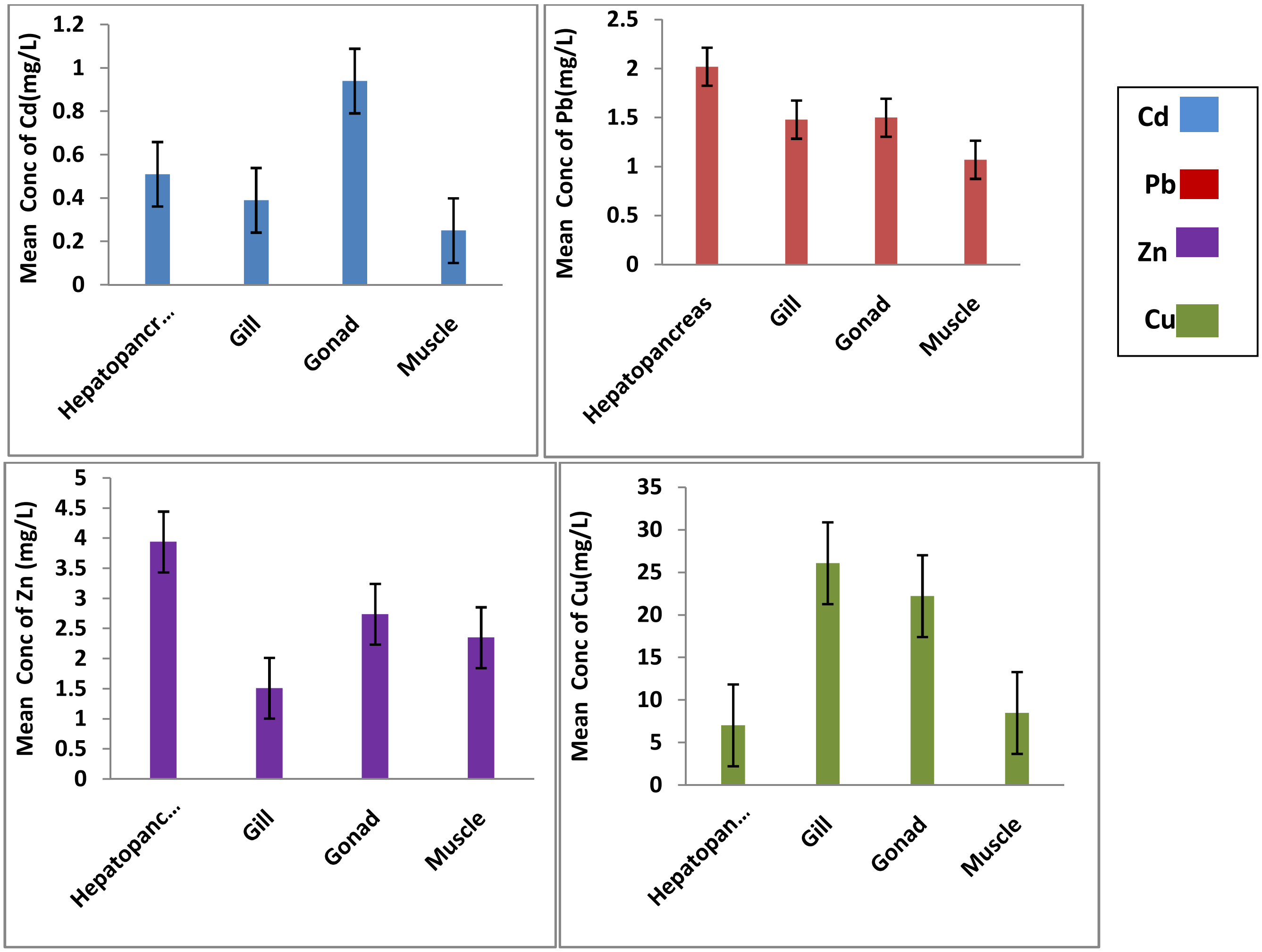


Fig 3: Trend of Heavy Metals Concentration (mg/L) in different Blue Crab Organs from the Sample Stations

The highest occurrence of Zinc was documented in hepatopancreas followed by gonad, muscle and least concentration was in gill (Fig 3).

The highest concentration of Copper was recorded in gill followed by gonad, muscle and least concentration was in hepatopancreas (Fig 3).

Copper has the highest concentrations followed by Zn, Pb and Cd in all examined organs of *C. amnicola*from all the sampling stations.

4.6.6 Uptake Pattern of Heavy Metals Concentration by Organs of *C amnicola* in Relation to the Sample Stations

The ballot of metal concentration in Hepatopancreas (Figure 4), shows a close connection contacting at the tip of ellipses between the mid-lagoon and other stations. This indicates that metal uptake patterns in Hepatopancreas of *C. amnicola* from mid-lagoon may not be as distinct from uptake pattern found in the Hepatopancreas of *C. amnicola* from other stations.

The discriminant ballot for metals in gills shows two distinct ellipses, in near contact. This ballot depicts that metal uptake patterns in crabgills aroundthe mid-lagoon was slightly distinct from metal uptake patterns in gills of crabs from other stations (Figure 5).

Similarly, Figure 6 shows the distinct ellipse between the mid-lagoon cluster and cluster of other stations combined also depicts differences in uptake patterns of metals in gonads of crabs from Mid-lagoon compared with uptake patterns in gonads of crab from all other station.

The ballot of metal concentration in crab muscle (Figure 7) showed an overlap in ellipses between the mid-lagoon cluster and the cluster of other stations. This indicates that levels of metals in muscle may not be distinctly different crab muscles from Mid-lagoon and crab muscles from all other sampling stations.

Analysis of variance shows the variations in concentration of Cadmium, Lead and Zinc, were not significantly difference at 95% ($p>0.05$), however, Cu concentration shows significant difference at 95% ($p<0.05$) between the sample stations.

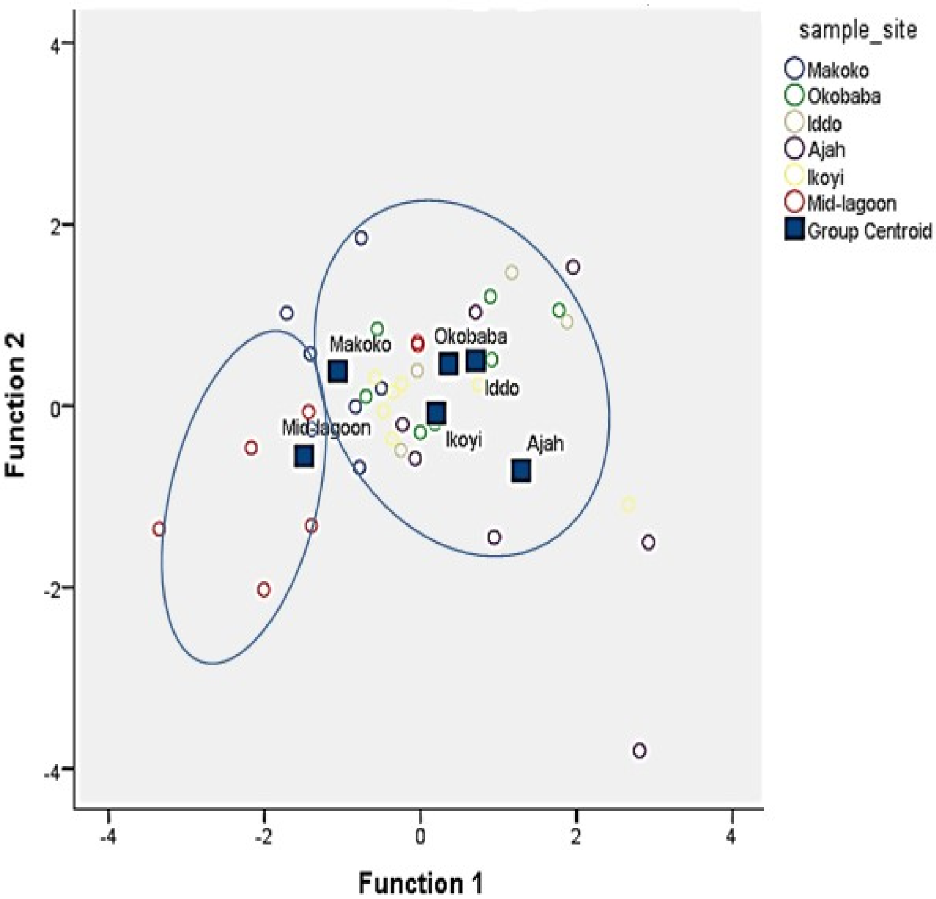


Fig 4: Discriminant Analysis for Heavy metal in Hepatopancreas across the stations

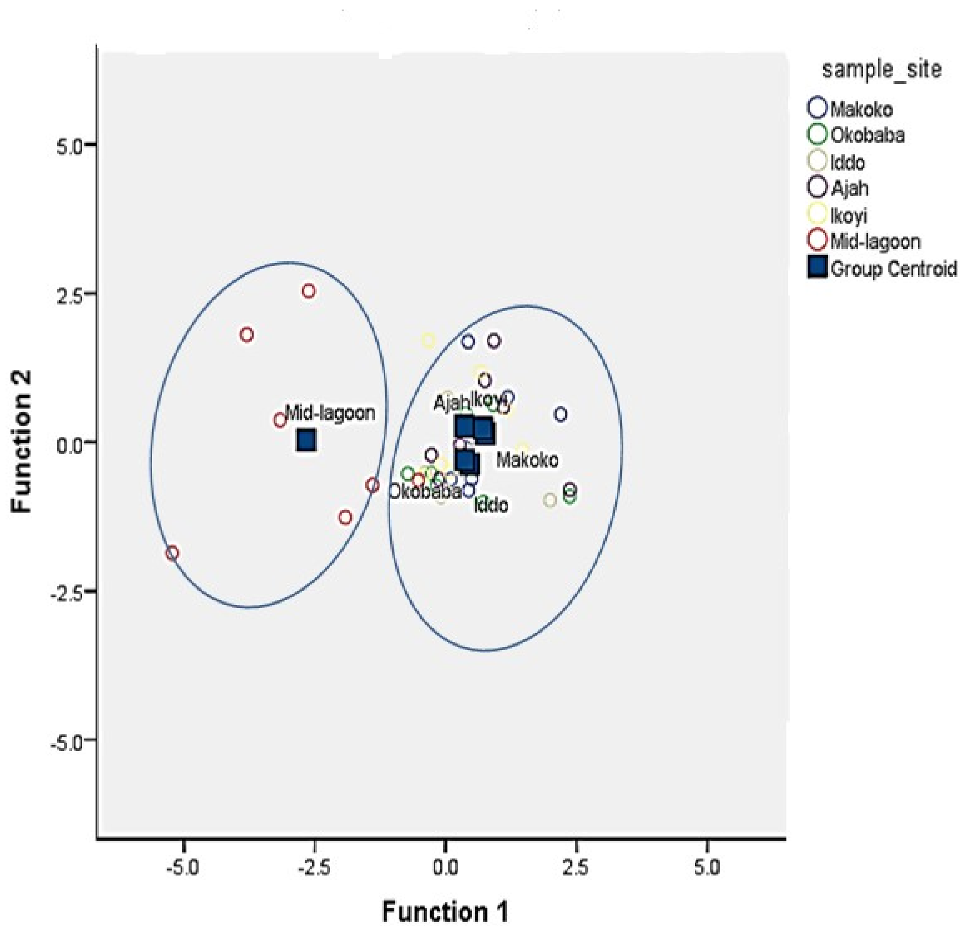


Fig 5: Discriminant Analysis for Heavy metal in gill in all the stations

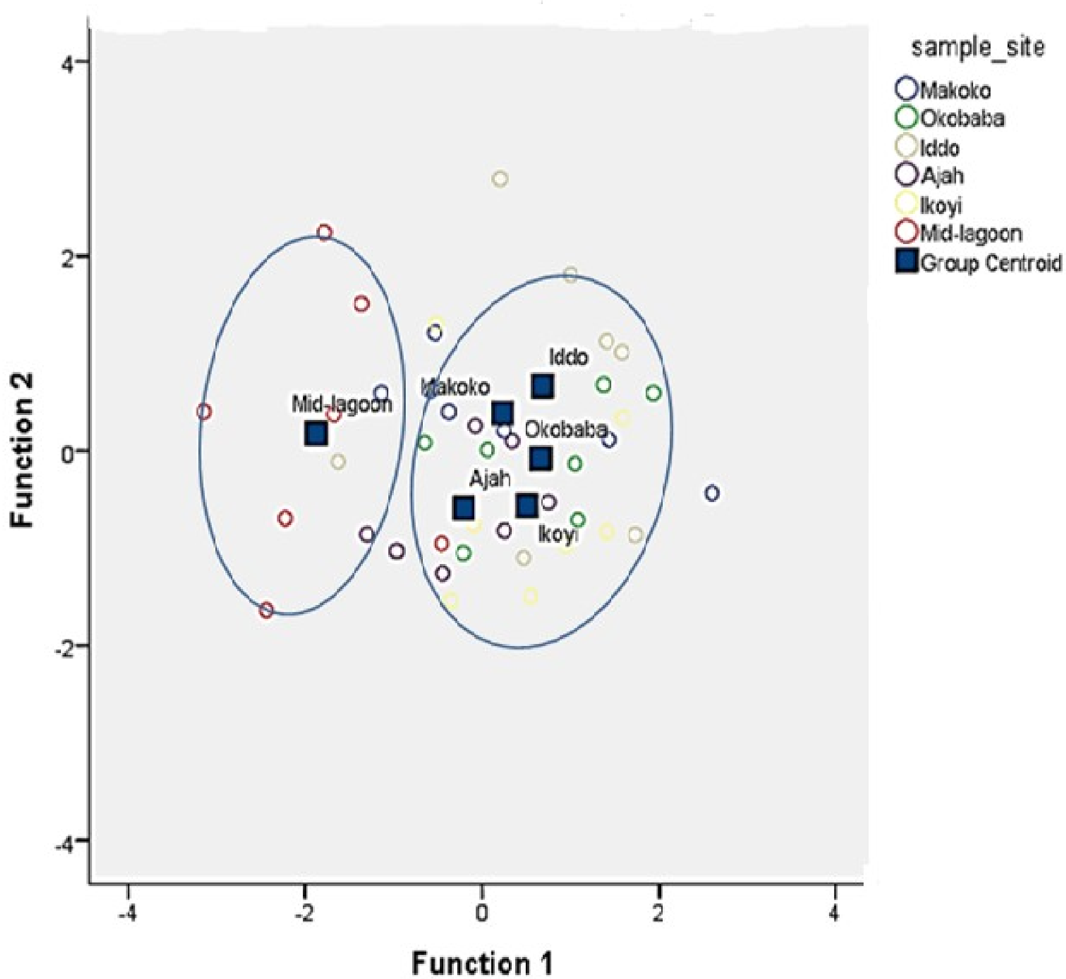


Fig 6: Discriminant Analysis for Heavy metal in gonad tissue across the station

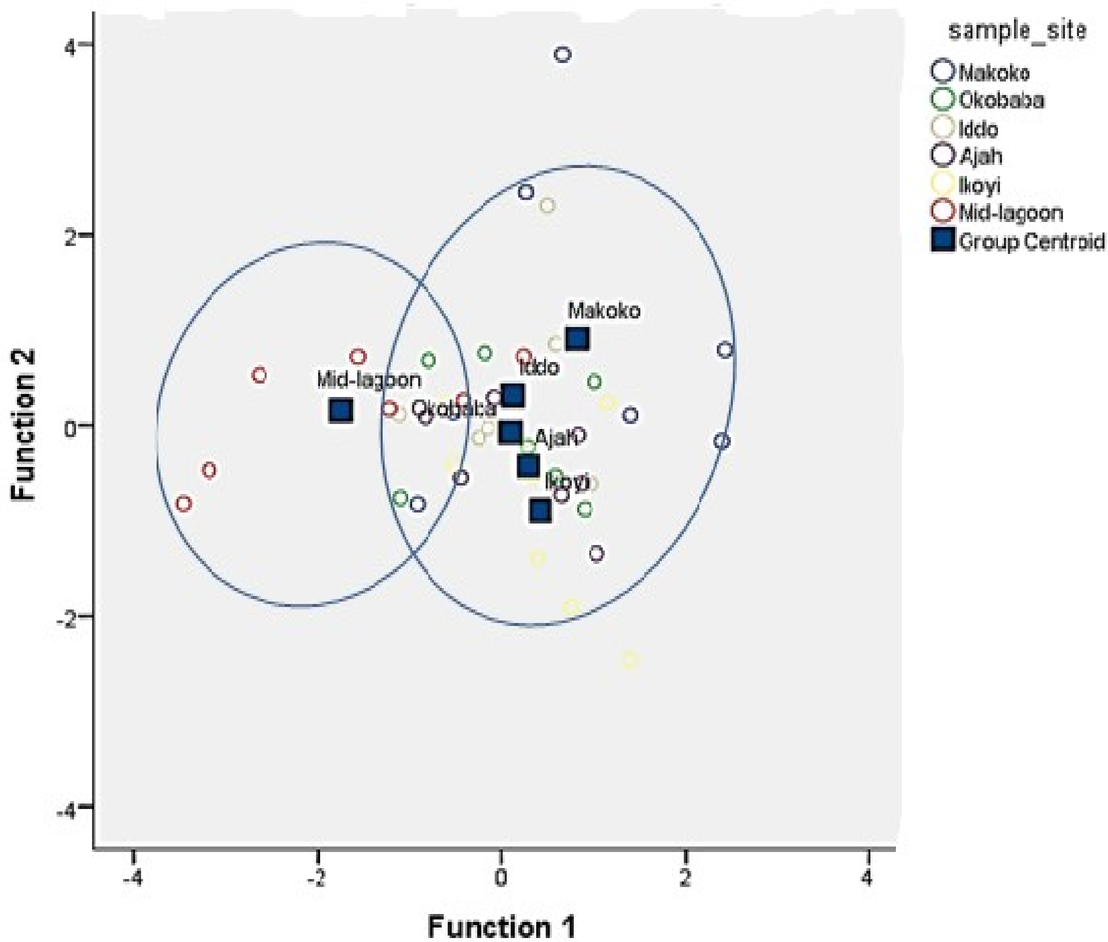


Fig 7: Discriminant Analysis for Heavy metal in muscle across the station

4.7 Biota-Sediment Accumulation Factor (BSAF)

Biota-sediment accumulation factor (BSAF) for crab organs and habitat sediment from the sampling stations showed station specific relationship between metal in crab and sediment.
(Figs. 8-11).

4.7.1 Hepatopancreas

In the hepatopancreas, BSAF for cadmium showed the highest values at Iddo (0.80) while values calculated for Pb, Zn, and Cu showed highest values at Iddo (0.17), Ikoyi (2.50), Makoko (0.20) respectively. Lowest for Cd, Pb, Zn, and Cu was 0.17 at Makoko, 0.04, 0.17, 0.31 at Mid-lagoon, Iddo and Mid-lagoon respectively (Fig. 8).

The BSAF obtained for Cd, Pb, Zn, and Cu in all sampling stations were less than 1.00 and were considered normal, except the BSAF obtained for Zn at Ajah and Ikoyi and Cu at Ajah which were greater than 1.00 indicating that they were highly bioaccumulated and bio-magnified in the hepatopancreas of the crab samples. Cu has the highest BSAF (5.41) at Ikoyi and was therefore, the most bio-magnified compared to other metals in hepatopancreas.

4.7.2 Gill

In the gill, BSAF for cadmium showed the highest values at Okobaba (0.65) while values calculated for Pb, Zn, and Cu, all were highest at Iddo (0.32), Makoko (0.68), Ikoyi (12.52) respectively. Lowest for Cd, Pb, Zn, and Cu was 0.05 at Mid-lagoon, 0.04, 0.05, 0.39 at Makoko, Iddo and Mid-lagoon respectively (Fig. 9).

The BSAF obtained for Cd, Pb, Zn, and Cu in all sampling stations were lower than 1.00 and were categorized as within limits, except the BSAF for Cu at Ajah and Ikoyi which were higher than 1.00 indicating that they were highly bioaccumulated and bio-magnified in the gill of the crab samples. Cu has the highest BSAF (12.52) at Ikoyi indicating greatest bio-magnification compared to the other metals studied.

4.7.3 Gonad

In the gonad, BSAF for cadmium showed the highest values at Ajah (1.38) while values calculated for Pb, Zn, and Cu, all were highest at Iddo (0.30), Ikoyi with 0.30, 1.71 and 1.71

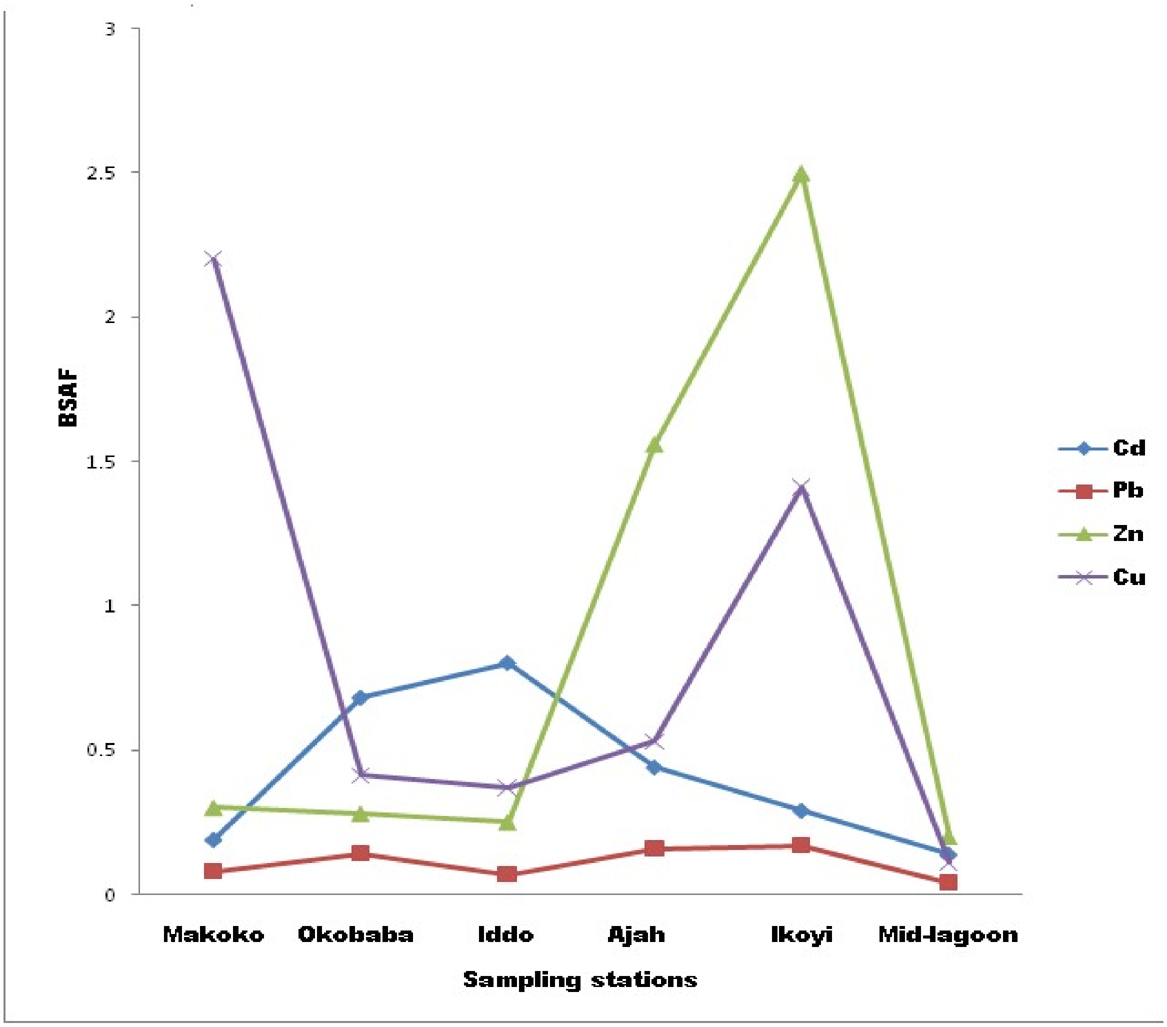


Figure 8: Biota-Sediment accumulation factor of metals between Hepatopancreas of *C. amnicola* and sediments from sampling stations in the Lagos lagoon

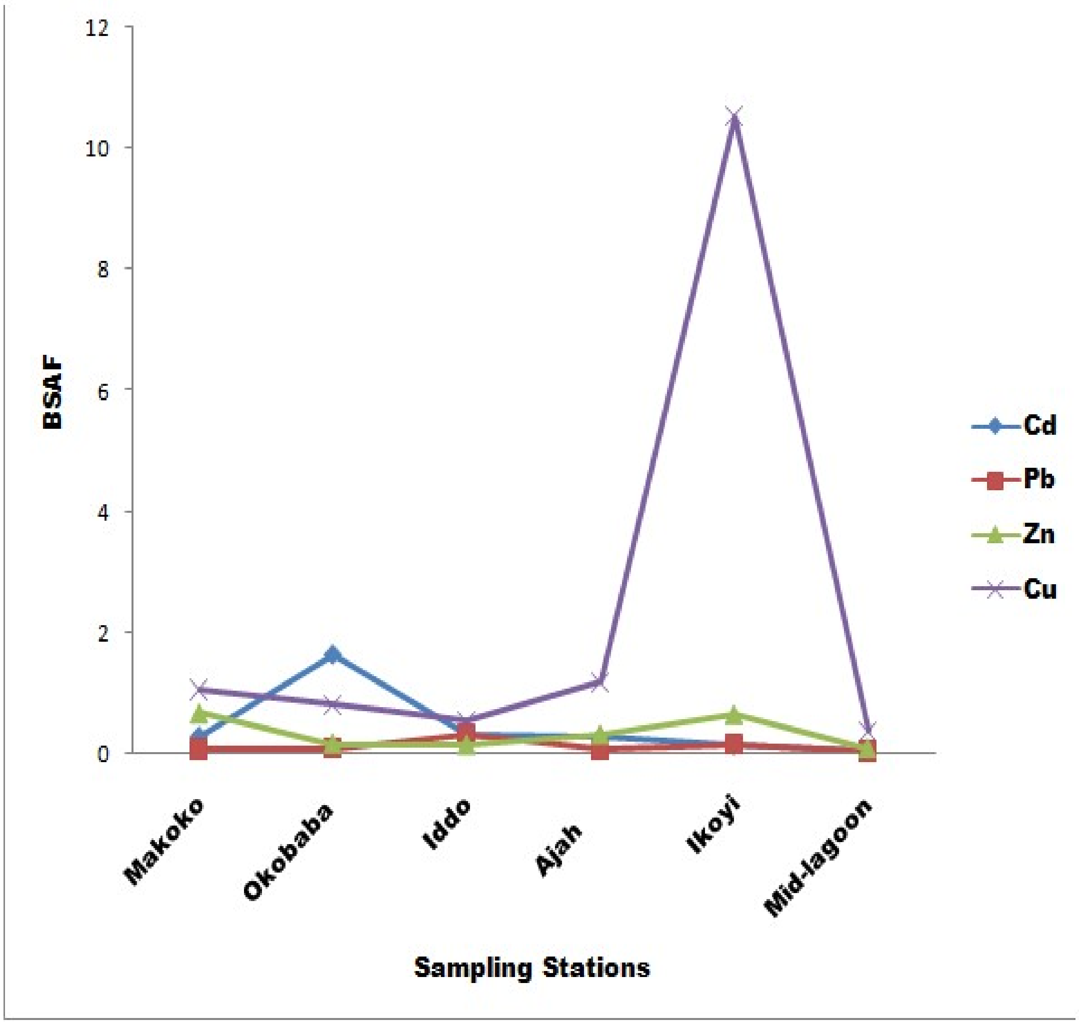


Figure 9: Biota-Sediment accumulation factor of metals between gill of *C. amnicola* and sediments from sampling stations in the Lagos lagoon

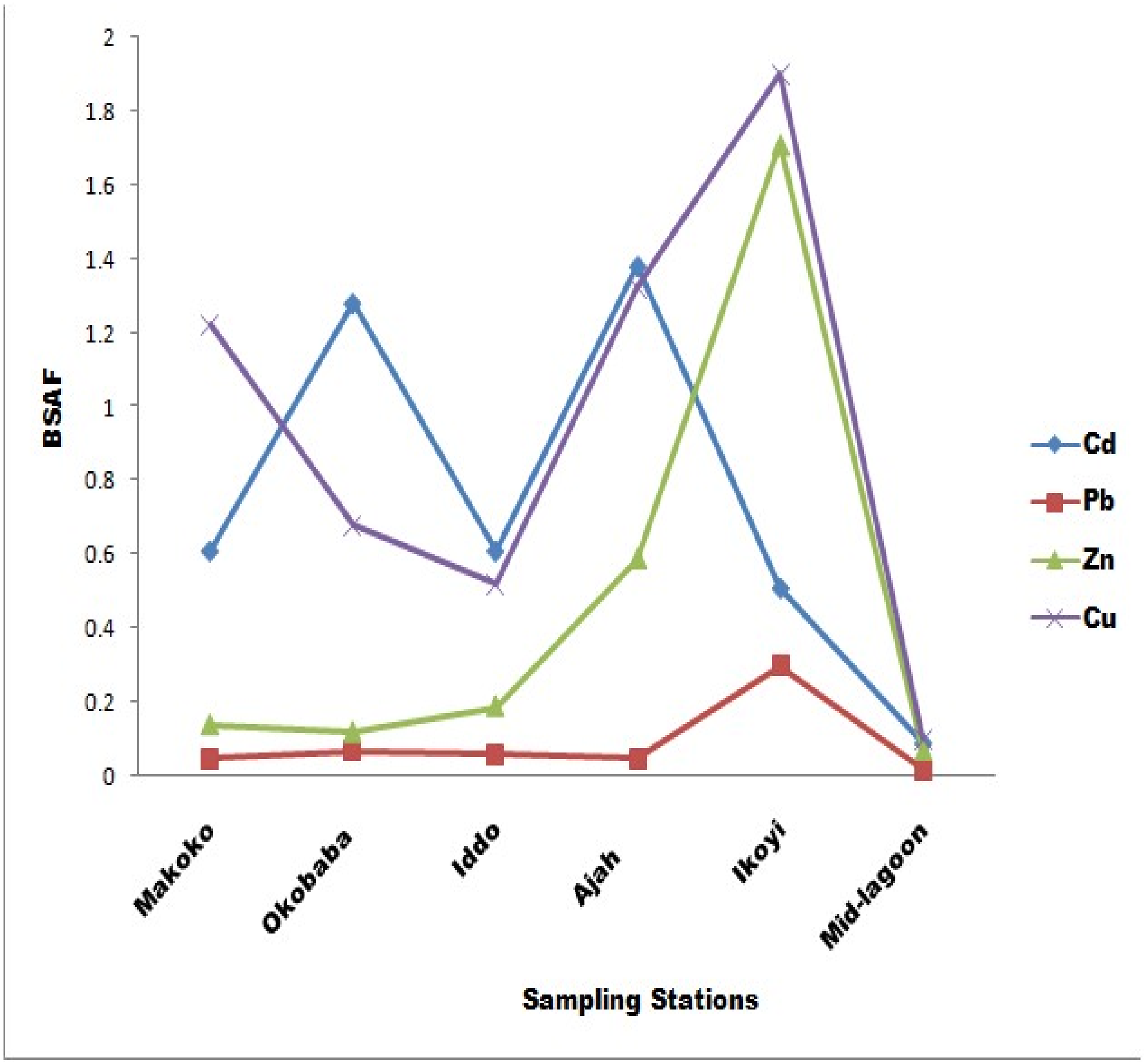


Figure 10: Biota-Sediment accumulation factor of metals between gonad of *C. amnicola* and sediments from sampling stations in the Lagos lagoon

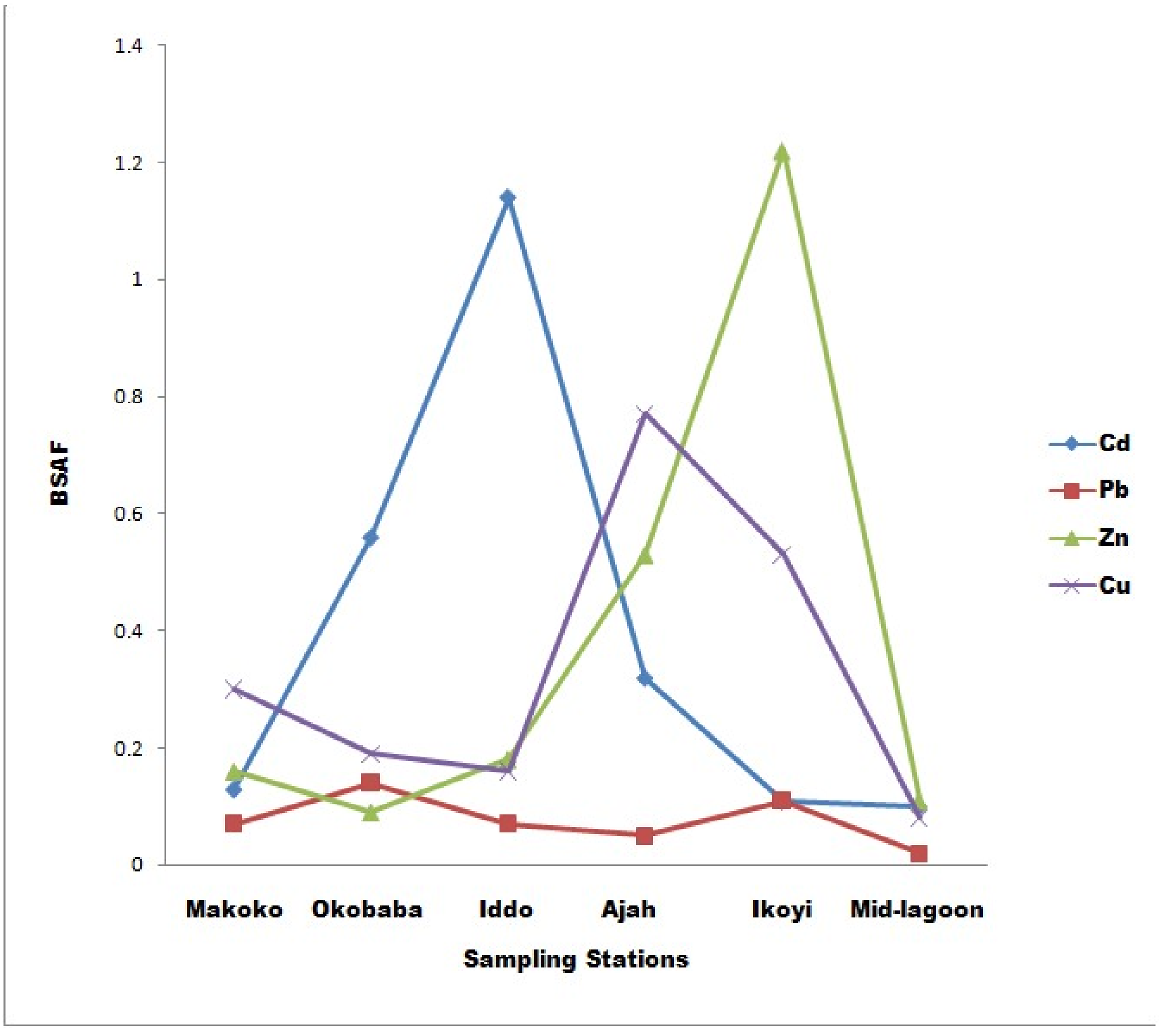


Figure 11: Biota-Sediment accumulation factor of metals between Muscle (flesh) of *C. amnicola* and sediments from sampling stations in the Lagos lagoon

respectively. Lowest for Cd, Pb, Zn, and Cu was 0.09 at Mid-lagoon, 0.02, 0.11, 0.29 at Iddo, Mid-lagoon and Mid-lagoon respectively (Fig. 10).

The BSAF obtained for Cd, Pb, and Zn, in all sampling stations were less than 1.00 and were considered normal, except Cd at Okobaba and Ajah, and Zn at Ikoyi which were greater than 1.00 indicating that they were highly bioaccumulated and bio-magnified in the gonad of the crab samples. However, the BSAF obtained for Cu were less than 1.00, except at Makoko, Ajah and Ikoyi. Cu has the highest BSAF (1.90) at Ikoyi indicating highest incidence of bioaccumulation in gonad

4.7.4 Muscle

In the muscle, BSAF for cadmium showed the highest values at Iddo (1.14) while values calculated for Pb, Zn, and Cu showed highest values at Okobaba (0.14), Ikoyi (1.22), Ajah (0.77) respectively. Lowest for Cd, Pb, Zn, and Cu was 0.10 at Mid-lagoon, 0.02, 0.11, 0.16 at Mid-lagoon, Mid-lagoon and Iddo respectively (Fig. 11).

The BSAF obtained for Cd, Pb, Zn, and Cu in all sampling stations were less than 1.00 and were considered normal, except Cd at Iddo and Zn at Ikoyi which were greater than 1.00 depicting that the metals were highly bioaccumulated and bio-magnified within muscle tissue of the crab samples. Zn showed the higher value of BSAF (1.22) at Ikoyi and indicating incidence of highest bio-magnification in muscle tissue.

4.8: Condition Factor of *C. amnicola* (Blue Crab) in the Sample Stations

The Condition factor of *C. amnicola* ranges from 0.54 -0.78 in the sampling stations with the highest at Mid-Lagoon, followed by 0.59 at Makoko, 0.57, 0.56 and 0.54 at Okobaba, Iddo, Ajah and Ikoyi respectively (Fig 12). The Condition factor of *C. amnicola* in stations with point source pollution and other anthropogenic activities were lower compared with condition factor at Mid-lagoon; the control station with no direct pollution activities.

Measures of condition factor index across study areas (Figure 12) was tested using one way ANOVA which depicted that the mean condition of the blue crab from the mid-lagoon (0.752±0.26) was notably higher($p<0.05$) than Makoko (0.58±0.12), Okobaba (0.57±0.16), Iddo (0.56±0.12), Ajah (0.56±0.11) and Ikoyi (0.53±0.12). Although blue crabs from the land areas

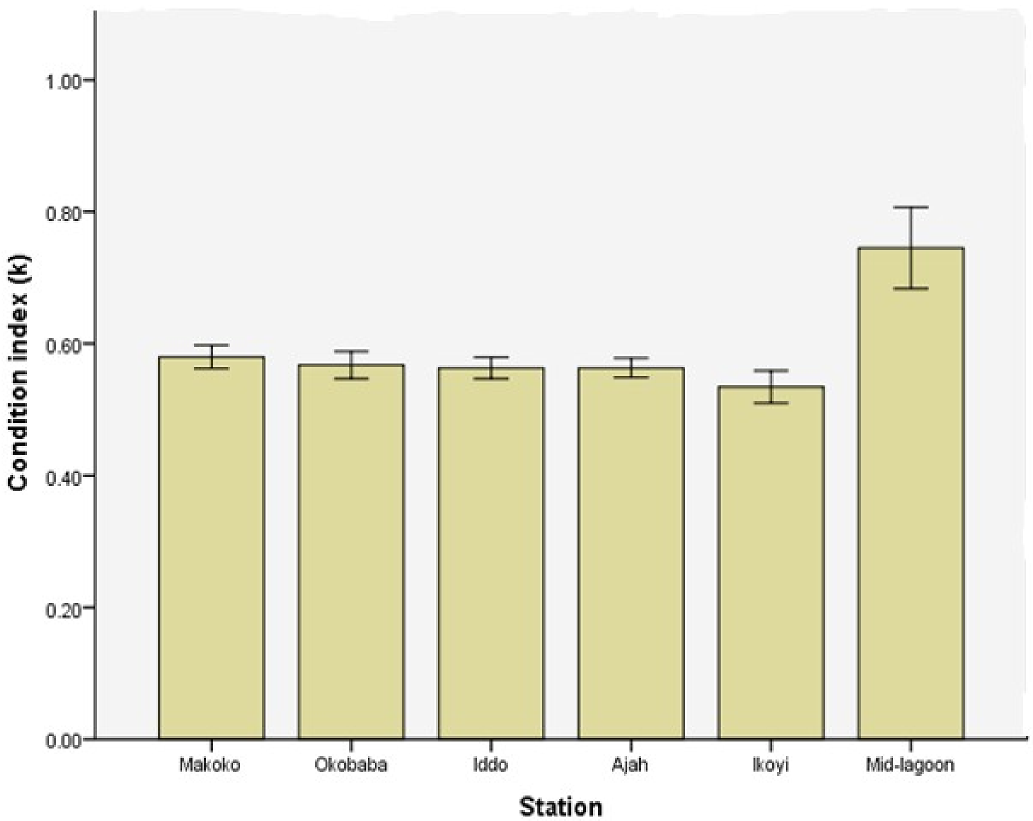


Fig 12:Condition factor of Blue Crab in the sampling stations

adjacent to the Lagos lagoon had similar ranges of condition factor index (CF), sample from the Ikoyi site had the lowest measure of condition.

4.9 Tissue-specific oxidative stress biomarker activity across seasons

4.9.1 *Crab Organs and Superoxide dismuthase Activity*

Superoxide dismutase levels recorded within Hepatopancreas, gill-tissue, gonad and muscle-tissue of blue crab from the sampling stations during dry and rainy season are shown in Fig. 13a&b.

The ANOVA revealed that the variation in activity of SOD in the hepatopancreas, gill, gonad and muscle of *C. amnicola* from Makoko, Okobaba, Iddo, Ajah, Ikoyi and Mid-Lagoon were significant ($p<0.05$).

Hepatopancreas: Activity of SOD was highest in hepatopancreas of crab from Ikoyi ($7.1 \times 10^{-5} \pm 7.76 \times 10^{-5}$ µmol/mgprotein) and lowest in hepatopancreatic tissue of crabs from Mid-lagoon ($1.13 \times 10^{-5} \pm 5.58 \times 10^{-5}$ µmol/mgprotein) during dry season. During rainy season the highest activity were in hepatopancreas of crabs from Makoko ($8.38 \times 10^{-5} \pm 4.34 \times 10^{-5}$ µmol/mgprotein) and lowest at hepatopancreas of crab from Okobaba ($1.1 \times 10^{-4} \pm 5.3910^{-5}$ µmol/mgprotein).

Gill: Superoxide dismuthase activity were highest in gill of crab from Ajah (1.60 ± 1.00 µmol/mgprotein) andlowest at Okobaba (0.89 ± 0.61 µmol/mgprotein) during dry season. In the rainy season the highest activity were in the gill of crabs from Mid-lagoon (1.19 ± 0.31 µmol/mgprotein) and lowest in gill of crab from Ikoyi (0.38 ± 0.09 µmol/mgprotein).

Gonad : Superoxide dismuthase activity were highest in gonad of crab from Mid-lagoon (0.98 ±0.16 µmol/mgprotein) and lowest at Ajah (0.50 ± 0.43 µmol/mgprotein) during dry season. During rainy season the highest activity were in gonad of crabs from Mid-lagoon (1.17 ± 0.19 µmol/mgprotein) and lowest in gonad of crab fromAjah (0.30 ± 0.09 µmol/mgprotein).

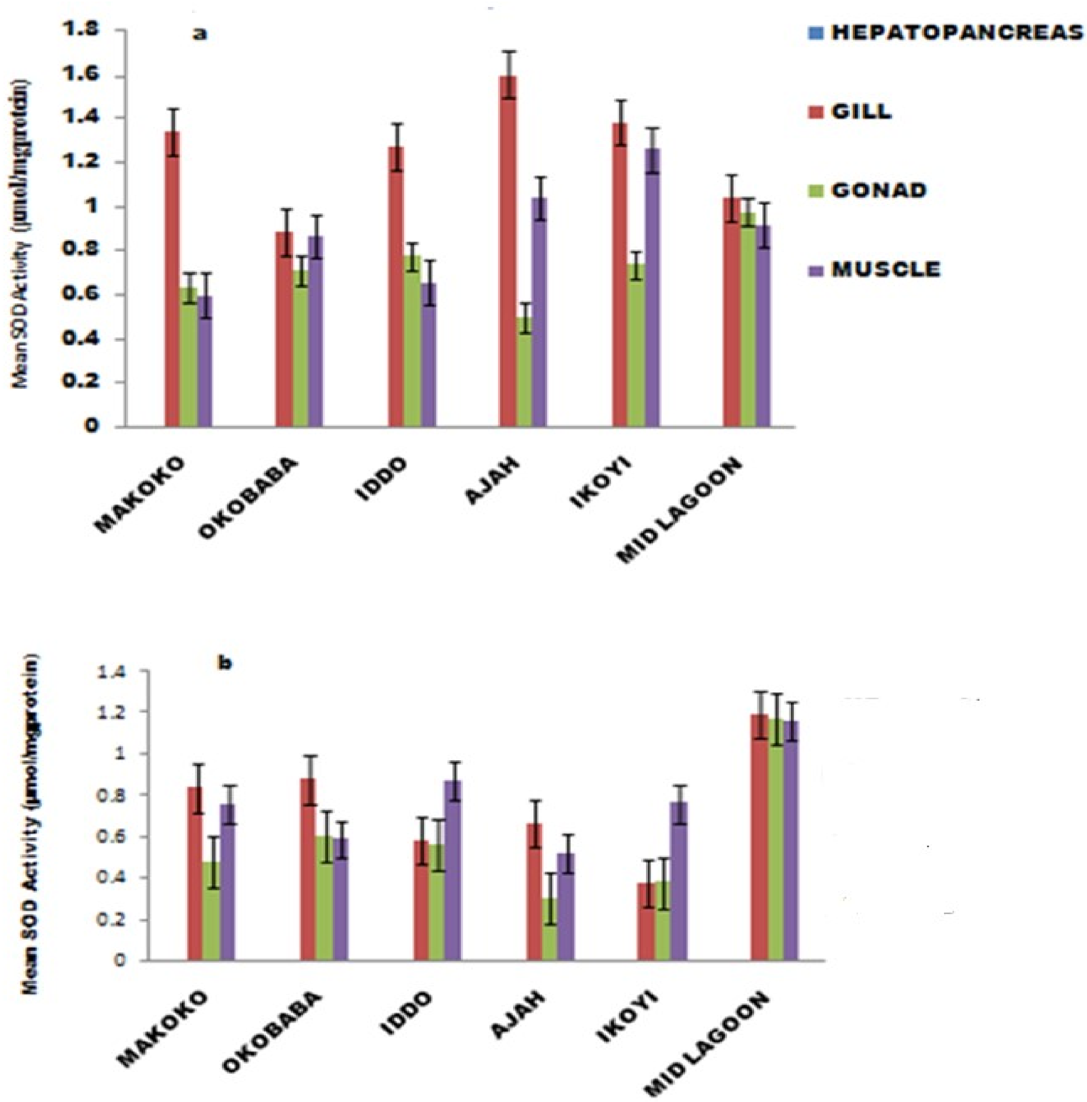


Fig 13: Mean Superoxide dismutase activity in *C. amnicola* in sampling stationsa= dry season and b= rainy season.

Muscle: Superoxide dismuthase activity were highest in in muscle of crab from Ikoyi (1.26 ± 0.71 µmol/mgprotein) and lowest at Makoko (0.60 ± 0.37 µmol/mgprotein) during dry season. During rainy season the highest activity were observed in muscle of crabs sampled from areas of the Mid-lagoon (1.09 ± 0.26 µmol/mgprotein) and lowest in muscle from Ajah (0.52± 0.13 µmol/mgprotein).

4.9.2 *Crab Organs and Catalase Activity*

Catalase (CAT) activity was shown in hepatopancreas, gill, gonad and muscle of blue crab from the sampling stations during dry and rainy season. **(**Fig 14a & b).

The ANOVA revealed that the variation in activity of CAT within the hepatopancreas, gill, `gonad and muscle of *C. amnicola* from Makoko, Okobaba, Iddo, Ajah, Ikoyi and Mid-Lagoon differed significantly ($p<0.05$) and ($p<0.01$) between the stations.

Hepatopancreas: Catalase (CAT) activity in Hepatopancreas were generally low, but the highest was at Mid-lagoon (0.43 ± 0.08umol/mL) and lowest at Okobaba (0.001 ± 0.0007umol/mL) during dry season. In the rainy season the highest value was at Mid-lagoon (0.58 ± 0.08 umol/mL) and lowest at Makoko (0.002 ±0.0009 umol/mL).

Gill: Catalase activity were highest in gill of crab from Ajah (14.18 ± 9.32 umol/mL) and lowest in gill of crab from Okobaba (7.47 ± 6.06 umol/mL) during dry season. In the rainy season the highest activity were in gill of crab from Mid-lagoon (14.55 ± 1.91 umol/mL) and lowest at Ikoyi ‘s crab pancreas (3.65 ± 2.03 umol/mL).

Gonad: Catalase activity were highest in gonad of crab from Mid-lagoon (14.18 ± 5.69 umol/mL) and lowest in gonad of crab from Ajah (6.73 ±3.60 umol/mL) during dry season. During rainy season the highest catalase activity were in the gonad of crab from Mid-lagoon (15.35 ±1.50 umol/mL) and lowest at Ajah‘s crab pancreas (5.77 ±2.04 umol/mL).

Muscle: Catalase activity were highest in muscle of crab from Mid-lagoon (13.16 ± 0.74 umol/mL) and lowest in muscle of crab from Makoko (5.55 ±3.79 umol/mL) during dry season.

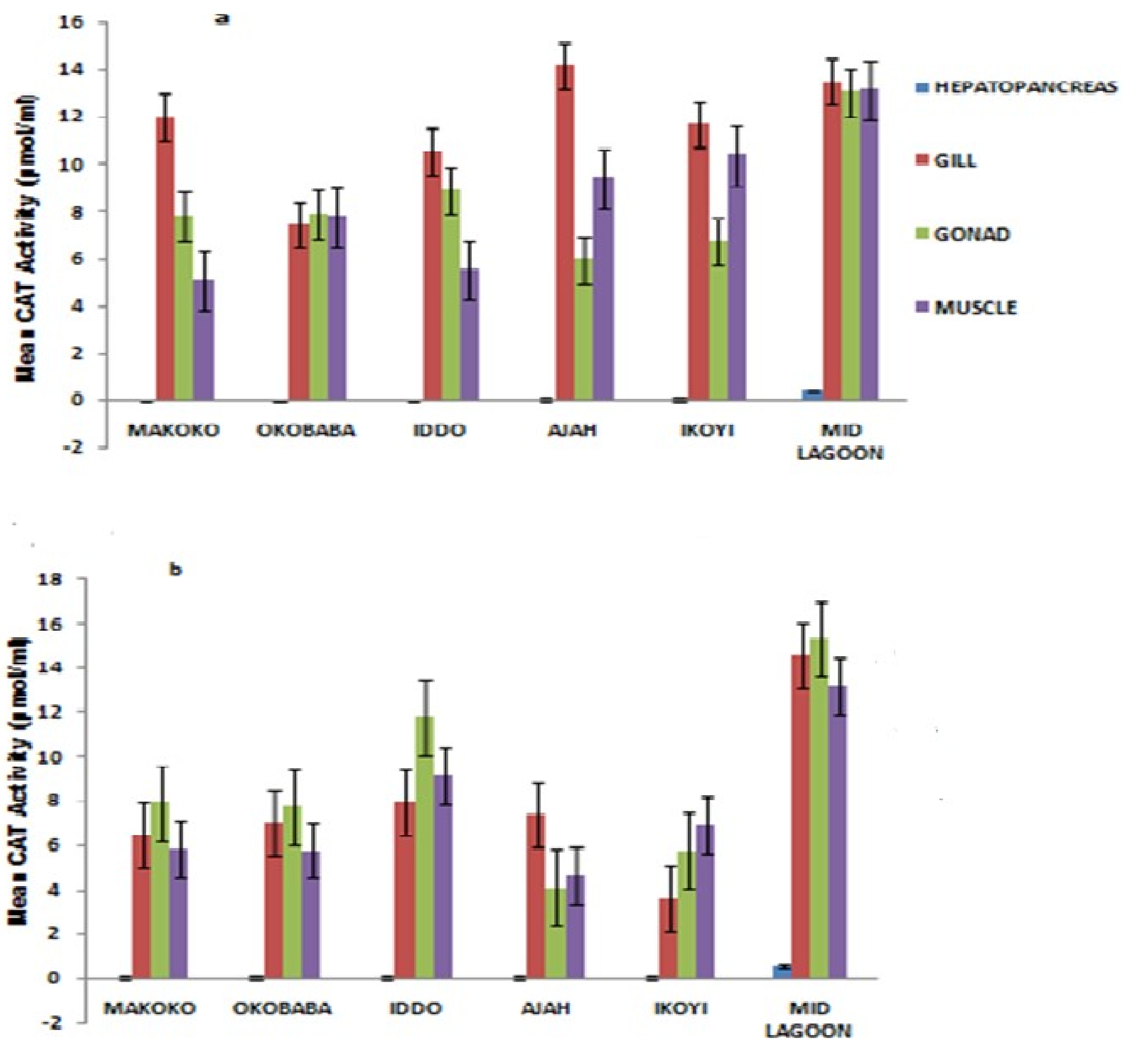


Fig 14: Mean Catalase (CAT) *activity (mol/ml)* in *C. amnicola* in sampling stationsa= dry season and b= rainy season.

In the rainy season, the highest activity were in muscle of crab from Mid-lagoon (13.24 ± 0.92 umol/ml) and lowest at Ajah's crab hepatopancreas (4.67 ± 1.52 umol/mL).

4.8.3 *Crab Organs and Glutathione Peroxidase Activity*

Activity of this enzyme was detected in Hepatopancreas, gill, gonad and muscleof blue crab from the sampling stations during dry and rainy season.(Fig15a&b)

The ANOVA revealed that the variation in activity of GPx in the Hepatopancreas, gill, gonad and muscleof *C. amnicola* from Makoko, Okobaba, Iddo, Ajah, Ikoyi and Mid-Lagoon differed significantly ($p<0.05$) and ($p<0.01$) between the stations.

Hepatopancreas: Glutathione peroxidase (GPx) activity was highest in Hepatopancreas of crab from Ajah (5.68± 0.55µmol/mL) and lowest in Hepatopancreas of crab from Mid-lagoon (4.91± 0.34 µmol/ml) during dry season. During rainy season, the highest activity was in Hepatopancreas of crab from Mid-lagoon (5.05±0.52µmol/mL) and lowest in hepatopancreasof crab from Makoko (4.78± 0.20 µmol/mL).

Gill: Glutathione Peroxidase activity was highest in gill of crab from Makoko (5.37±0.76 µmol/mL) and lowest in gill of crab from Mid-lagoon (5.14± 0.37 µmol/mL) during dry season. In the rainy season, the highest activity were in gill of crab from Mid-lagoon (5.38± 0.39 µmol/mL) and lowest at crab from Okobaba pancreas (4.78 ±0.13 µmol/mL).

Gonad: Glutathione Peroxidase activity were highest in gonad of crab from Makoko (5.47±0.65 µmol/mL) and lowest in the gonad of crab from Mid-lagoon (4.93 ±0.48 µmol/mL) during dry season. During rainy season, the highest activity were in gonad of crab from Iddo (5.25± 0.38 µmol/mL) and lowest at Ajah's crab hepatopancreas (4.89±0.15 µmol/mL).

Muscle: Glutathione Peroxidase activity were highest in muscle of crab from Makoko (5.45± 0.85 µmol/mL) and lowest in muscle of crab from Mid-lagoon (5.21± 0.54 µmol/mL) during dry season. In the rainy season, the highest activity were in muscle of crab from Ajah(4.98± 0.02 µmol/mL) and lowest at Mid-lagoon's crab hepatopancreas (4.87±0.13 µmol/mL).

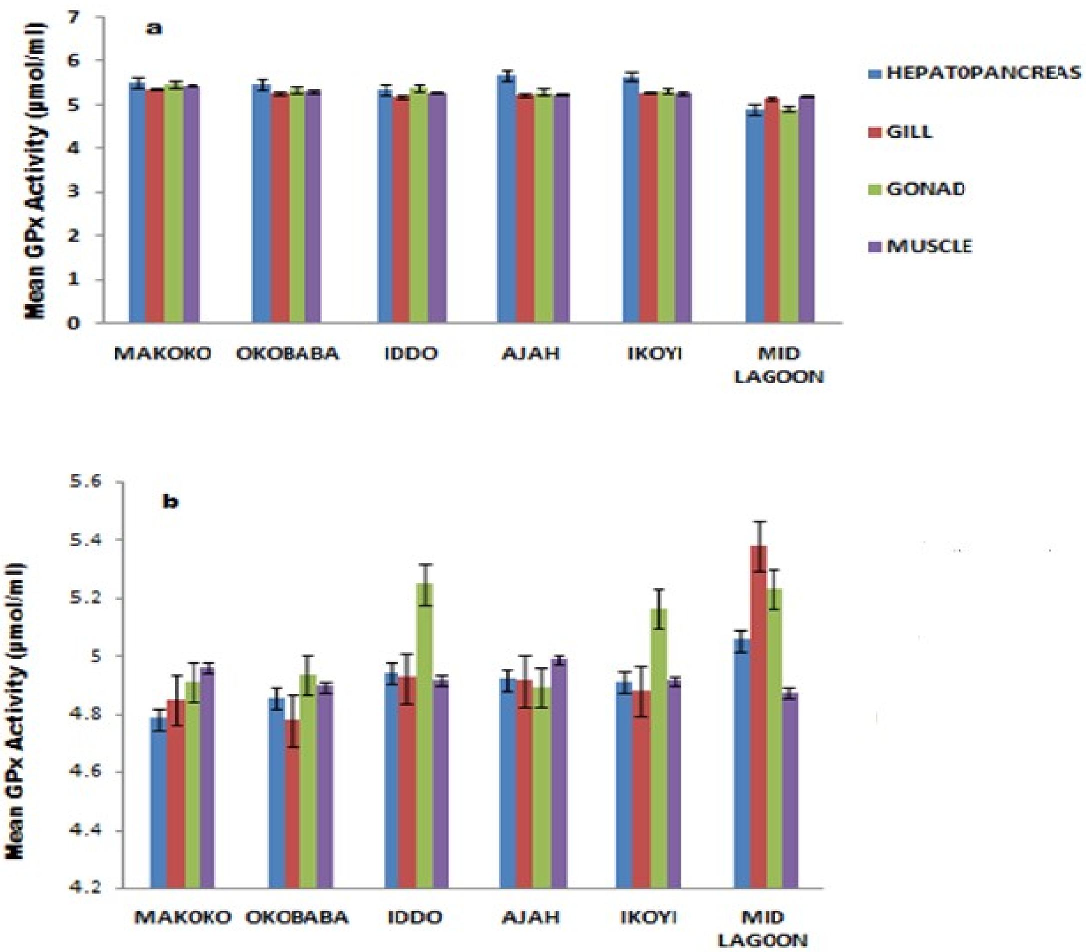


Fig 15: Mean Glutathione Peroxidase (GPx) ***activity (mol/ml)*** **in** ***C.amnicola*** **in sampling stationsa= dry season and b= rainy season.**

4.9.4 *Crab Organs and Reduced Glutathione Activity*

Reduced Glutathione (GSH) concentration was shown in hepatopancreas, gill, gonad and muscleof blue crab from the sampling stations during dry and rainy season **(**Fig 16a&b).

The ANOVA revealed that the variation in activity of GSH in the hepatopancreas, gill, gonad and muscleof *C. amnicola* from Makoko, Okobaba, Iddo, Ajah, Ikoyi and Mid-Lagoon differed significantly ($p<0.05$) and ($p<0.01$) between the stations

Hepatopancreas: Reduced Glutathione (GSH) concentration were highest in Hepatopancreas of crab from Ikoyi (1.92 ± 1.65 Umol/mL) and lowest in hepatopancreas of crab from Iddo (0.77±0.28 Umol/mL) during dry season. During rainy season, the highest concentration were in hepatopancreas of crab from Ajah(1.59± 0.98 Umol/mL and lowest at Mid-lagoon's crab hepatopancreas (0.70± 0.11 Umol/mL).

Gill: Reduced glutathione (GSH) concentration were highest in gill of crab from Ikoyi (0.94±0.32 Umol/mL) and lowest in gill of crab from Mid-lagoon (1.07± 0.21 Umol/mL) during dry season. In the rainy season, the highest concentration were in gill of crab from Mid-lagoon (1.13± 0.30 Umol/mL) and lowest at Okobaba's crab gill (0.54± 0.04 Umol/mL).

Gonad: Reduced glutathione (GSH) concentration were highest in gonad of crab from Ajah (1.47±1.02 Umol/mL) and lowest in gonad of crab from Ikoyi (0.89 ± 0.38 Umol/mL) during dry season. During rainy season, the highest concentration were in gonad of crab from Mid-lagoon (1.12 ± 0.29 Umol/mL) and lowest at Ajah's crab gonad (0.61± 0.04 Umol/mL).

Muscle: Reduced glutathione (GSH) concentration were highest in muscle of crab from Iddo (1.01± 0.32 Umol/mL) and lowest in gill of crab from Ajah (0.84±0.27 Umol/mL) during dry season. During rainy season, the highest concentration were in muscle of crab from Makoko (1.16± 0.69 Umol/mL) and lowest at Ajah's crab muscle (0.64± 0.09 Umol/mL).

4.9.5 *Crab Organs and Lipid Peroxidation* Concentration

Lipid Peroxidation (MDA) concentration shown in hepatopancreas, gill, gonad and muscleof blue crab from the sampling stations during dry and rainy season **(**Fig17 a&b).

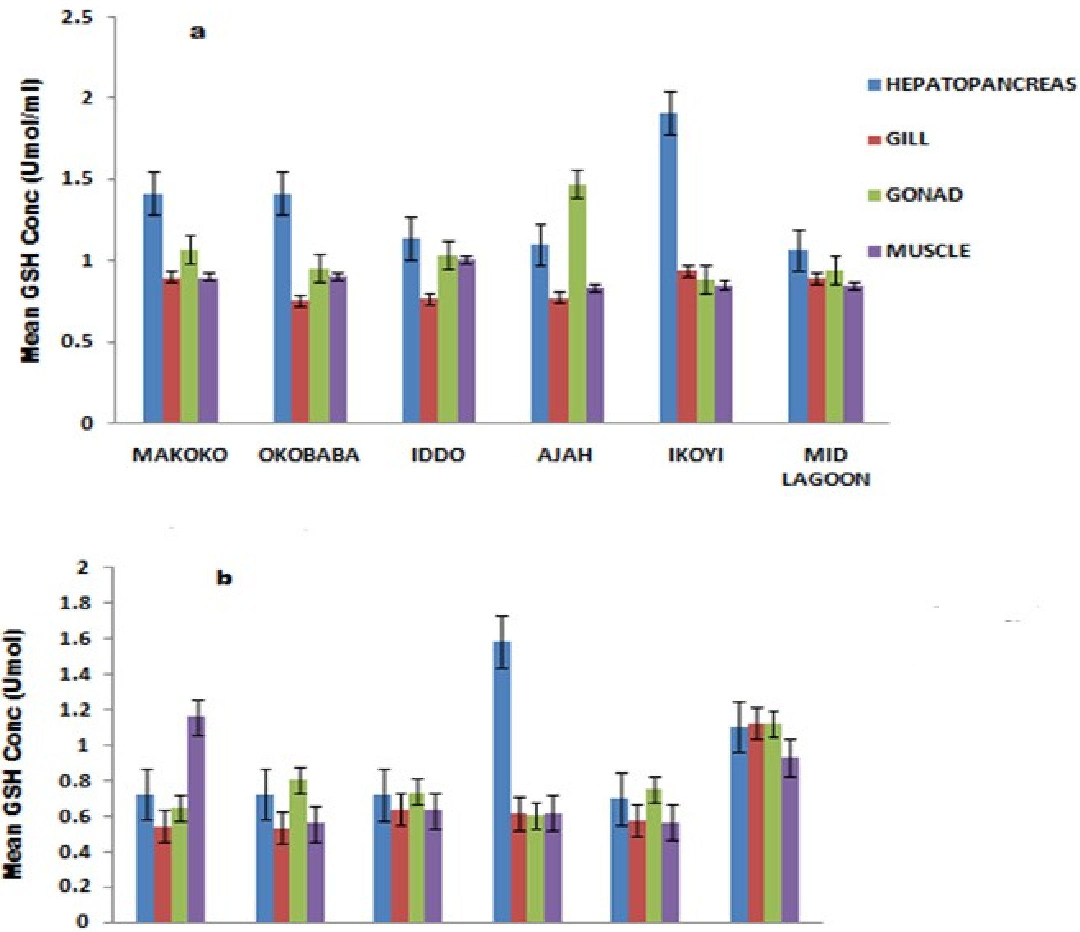
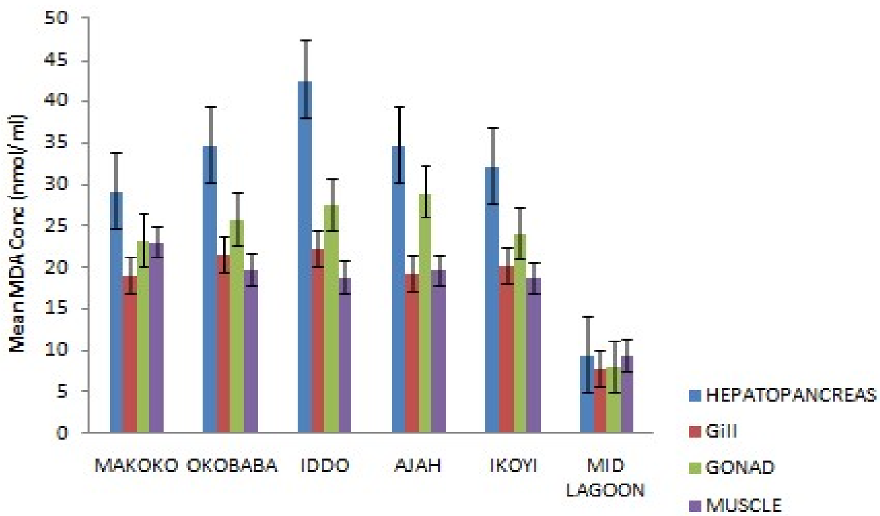


Fig 16: Mean Reduced Glutathione concentration in *C. amnicola* in sampling stations a= dry season and b= rainy season.

a



b

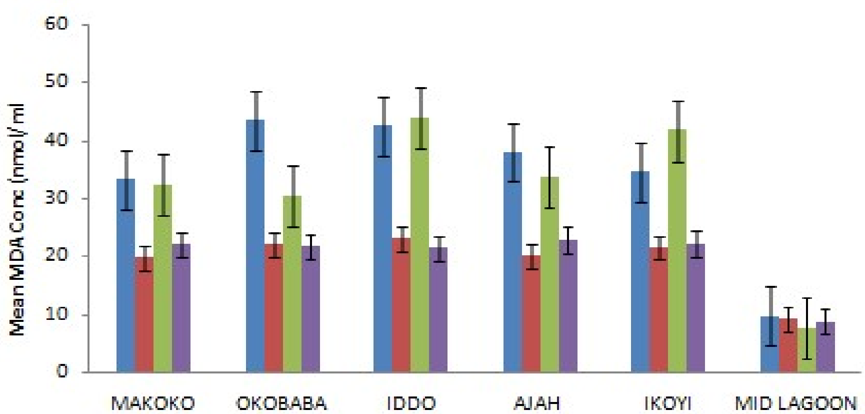


Fig 17: Mean Malondialdehyde (MDA) concentration in *C. amnicola* in sampling stationsa= dry season and b= rainy season.

The ANOVA revealed that the variation in activity of MDA within tissues of the hepatopancreas, gill, gonad and muscleof *C. amnicola* from Makoko, Okobaba, Iddo, Ajah, Ikoyi and Mid-Lagoon differed significanlty ($p<0.05$) and ($p<0.01$) between the stations

Hepatopancreas: Lipid Peroxidation concentration were highest in Hepatopancreas of crab from Iddo (42.74 ± 4.60 nmol/mL) and lowest in hepatopancreas of crab from Mid-lagoon (14.52 ± 0.52 nmol/mL) during dry season. During rainy season, the highest concentration were in hepatopancreas of crab from Okobaba (43.42 ± 4.36 nmol/mL) and lowest at Mid-lagoon's crab pancreas (13.69 ± 1.39 nmol/mL).

Gill: Lipid Peroxidation concentration were highest in gill of crab from Iddo (22.25 ± 2.53 nmol/mL) and lowest in gill of crab from Mid-lagoon (9.87 ± 2.30 nmol/mL) during dry season. In the rainy season, the highest concentration were in gill of crab from Iddo (23.02 ± 3.49 nmol/mL) and lowest at Mid-lagoon's crab gill (11.19 ± 1.66 nmol/mL).

Gonad: Lipid peroxidation concentration were highest in gonad of crab from Ajah (29.14 ± 6.92 nmol/mL) and lowest in gill of crab from Mid-lagoon (10.13 ± 2.30 nmol/mL) during dry season. During rainy season, the highest concentration were in gonad of crab from Iddo (43.95 ± 30.68 nmol/mL) and lowest at Mid-lagoon's crab gonad (12.70 ± 0.79 nmol/mL).

Muscle: Lipid peroxidation concentration were highest in muscle of crab from Makoko (23.09 ± 4.30 nmol/mL) and lowest in muscle of crab from Mid-lagoon (12.46 ± 1.88 nmol/mL) during dry season. During rainy season the highest were concentration were in muscle of crab from Ajah (22.75 ± 4.05 nmol/mL) and lowest at Mid-lagoon's crab muscle (13.73 ± 1.83 nmol/mL)

4.10 Relationship between Oxidative Stress Parameters Activity and Heavy Metal Concentration in Crab Organs.

The relationship between the heavy metal exposures and oxidative stress parameters response in blue crab organs from the sampling stations showed significant correlation especially in hepatopancreas, gill and heavy metal concentration.

Hepatopancreas: In hepatopancreas of sampled crab, zinc showed positive correlation ($p<0.01$ and $p<0.05$) with SOD, CAT, GPx, and MDA, but showed negative association with GSH activity. Lead concentration in crab from sample stations showed positive association ($p<0.01$ and $p<0.05$) with GPx, and MDA, and showed negative correlation with SOD, CAT, GSH activity. Cadmium and Copper showed negative correlation with all the antioxidant biomarker used.

Gill: Zinc and copper of crab from sample stations showed positive correlation ($p<0.01$ and $p<0.05$) with all antioxidant parameters, but Cu showed negative associationto CAT within sample stations. Cadmium and Lead showed negative correlation with all the antioxidant biomarker used.

Gonad: Zinc concentration of crab from sample stations showed positive correlation ($p<0.01$ and $p<0.05$) with SOD, GPx, GSH and MDA of crab and negative correlation with CAT in the sample stations. Cadmium, lead and copper showed negative correlation with all the antioxidant biomarker used

Muscle: Cadmium concentration of crab from sample stations showed positive association ($p<0.01$ and $p<0.05$) with CAT, and MDA of crab and negative correlation with SOD, GPx, and GSH in the sample stations. Zinc concentration of crab showed positive association ($p<0.01$ and $p<0.05$) with MDA, and negative correlation with SOD, CAT, GPx, and GSH,. copper concentration of crab showed positive association ($p<0.01$ and $p<0.05$) with GPx and MDA, and negative correlation with SOD, CAT and GSH. Lead showed negative correlation with all the antioxidant biomarker used in all sample stations.

4.11 Histopathology of Blue Crab (*C. amnicola*) Organs

Sections of tissues from Mid-lagoon, Makoko, Okobaba, Iddo, Ajah, and Ikoyi sampling stations respectively are shown in plate 5 (A-F).

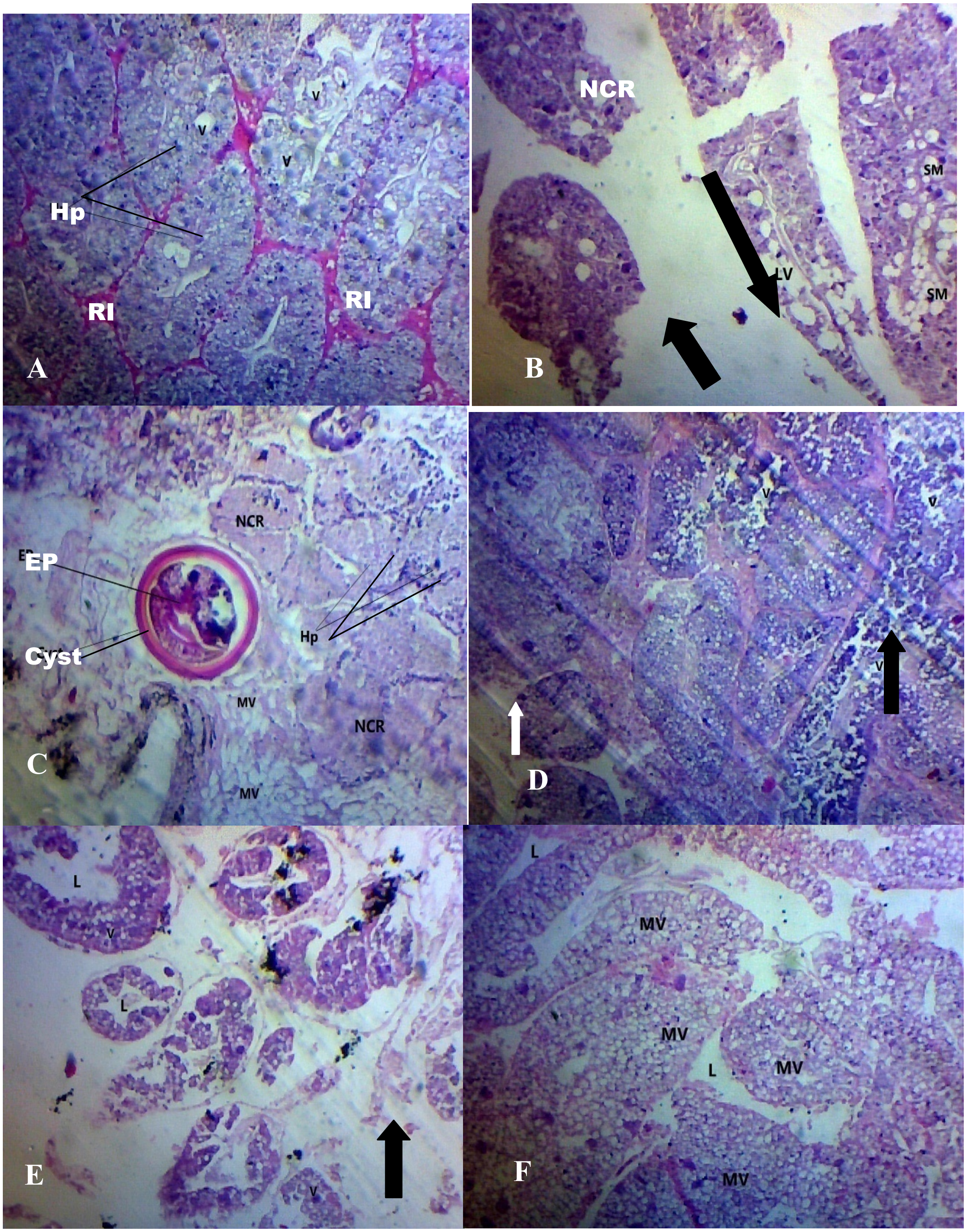


Plate 5A-F: Histopathology of Hepatopancreas of Blue crab from different Sampling Stations: (A) Mid-lagoon (Control), (B) Makoko, (C) Okobaba, (D) Iddo, (E) Ajah, and (F) Ikoyi. MV multiple vacuolations, LV large vacuolation , NCR Necrosis, HP hepatocytes, SM small vacuolation, EP encysted parasite, V vacuolation, L lumen, elongated or distended lumen (long thick arrow), rupture of basal lamella (short thick arrow) , infiltration of hemocytes (short white arrow).

4.11.1 Hepatopancreas

Mid–lagoon: Hepatopancreas of crabs sampled from the Mid-lagoon revealed unaltered lobular and tubular arrangement of hepatocytes with intersperse vacuolations, the hepatocytes were separated with connective tissues rich in red inclusion cells. The tubule epithelium contains blister-like cells, embryonic cells and absorptive cells. (Plate 5A).

Makoko: The pathological changes in hepatopancreas of crabs obtained from Makoko included extensive disruption of tubular and connective tissues with multi-focal occurrences of vacuolation, elongated haemocytes with distended lumen and damage to the myoepithelial layer. (Plate 5B).

Okobaba: The hepatopancreas of crabs from Okobaba reflected the effect of saw-mill waste pollution. The hepatocytes were infected with an encysted parasite (EP); double layer cyst wall diagnostic of hydrated (*Echinococcus granulossus*). The infection contributed to the loss of tubular hepatocytes (HP) arrangement, multiple vacuolation, necrosis of tissue and loss of connective tissues observed (Plate 5C).

Iddo: The vacuolated hepatocytes with lumen structure were still observed in the hepatopancreas of crab from Iddo. The connective tissues with red. inclusions cells were also observed as reported in Hepatopancreas of crab from Mid-lagoon, but vacuolation of the tissue was evident asobserved in crab from Makoko and Okobaba. (Plate 5D).

Ajah: There were less crowded hepatocytes with loss of connective tissues, some other tubule cells, and vacuolation of tissue within the hepatocytes were observed in the hepatopancreas of crab from Ajah (Plate 5E).

Ikoyi: In crabs from Ikoyi, the hepatopancreas showed changes which included loss of normal glandular and tubular shaped hepatopancreas seen in hepatopancreas of crab from Mid-lagoon(Plate 5A), with large and multiple vacuolation and distended lumen, as observed in necrosis, pknotic nucleus and embryonic zone (Plate 5F).

4.11.2 Gill

Mid-lagoon: There were intact gill lamella or filament in the gills of crab from Mid-lagoon; both primary and secondary gill lamella were closely packed and covered with a thin layer of cuticle which attached properly. Ionocytes were also present (Plate 6A).

Makoko: The gills of crab from Makoko showed changes such as collapsed gill lamella due to the distruption of pillar cells with detached cuticle and severe hyperplasic tissue, including lamella necrosis in gill tissue (Plate 6B).

Okobaba: The changes observed in gills of crabfrom Okobaba were thickened gill lamella with detached cuticle caused by distruption of pillar cells, and massive hemocytic infiltration. Hyperplasia, necrosis of filaments, intralamellar space and loss of gill structure are also noticed (Plate 6C).

Iddo: The following changes were observed in gills of crab from Iddo; loss of primary lamella (pillar cells) which resulted in the collapse of gill lamella with detached cuticle and inter lamella space (Plate 6D).

Ajah: Gills of crab from Ajah showed closely packed lamella with pillar cells covered with intact cuticle layer and ionocytes were observed.(Plate 6E).

Ikoyi: The histopathological changes observed in gills of crab from Ikoyi were; epithelial necrosis, hyperplasia and enlargement of secondary gill lamellae. There are evidences of cuticle detachment and ruptured capillaries which released hemocytes at the epithelial lining of the secondary gill lamella (Plate 6F).

4.11.3 Gonad

*Mid-lagoon:*The gonad of crab from Mid-lagoon showed matured vitellogenic oocytes with numerous follicle cells, and highly regular appearance of the oocytes matrix. (Plate 7A).

Makoko: The gonad of crab from Makoko, showed early vitellogenic oocytes, loss of primary oocytes and nucleus in the follicle cells. Infiltration of follicle cell membrane with hemocytes forming thickened wall are visible. Necrosis and abnormal enlargement of follicle cells, ruptured primary oocytes were observed (Plate 7B).

Okobaba, Iddo, Ajah: The gonad of crab from Okobaba, Iddo, Ajah showed epithelium lumen filled with eosinophilic secretions. The gonad tissues showed epithelium lumen filled with eosinophilic secretions; these secretions are dead cells.

Ingonad of crab from Iddo, Ajah, and Ikoyi.The basophilic patches or secretion showed in gonad of Ajah crabs were small packets secreted for separation and compartment (Plate 7D).

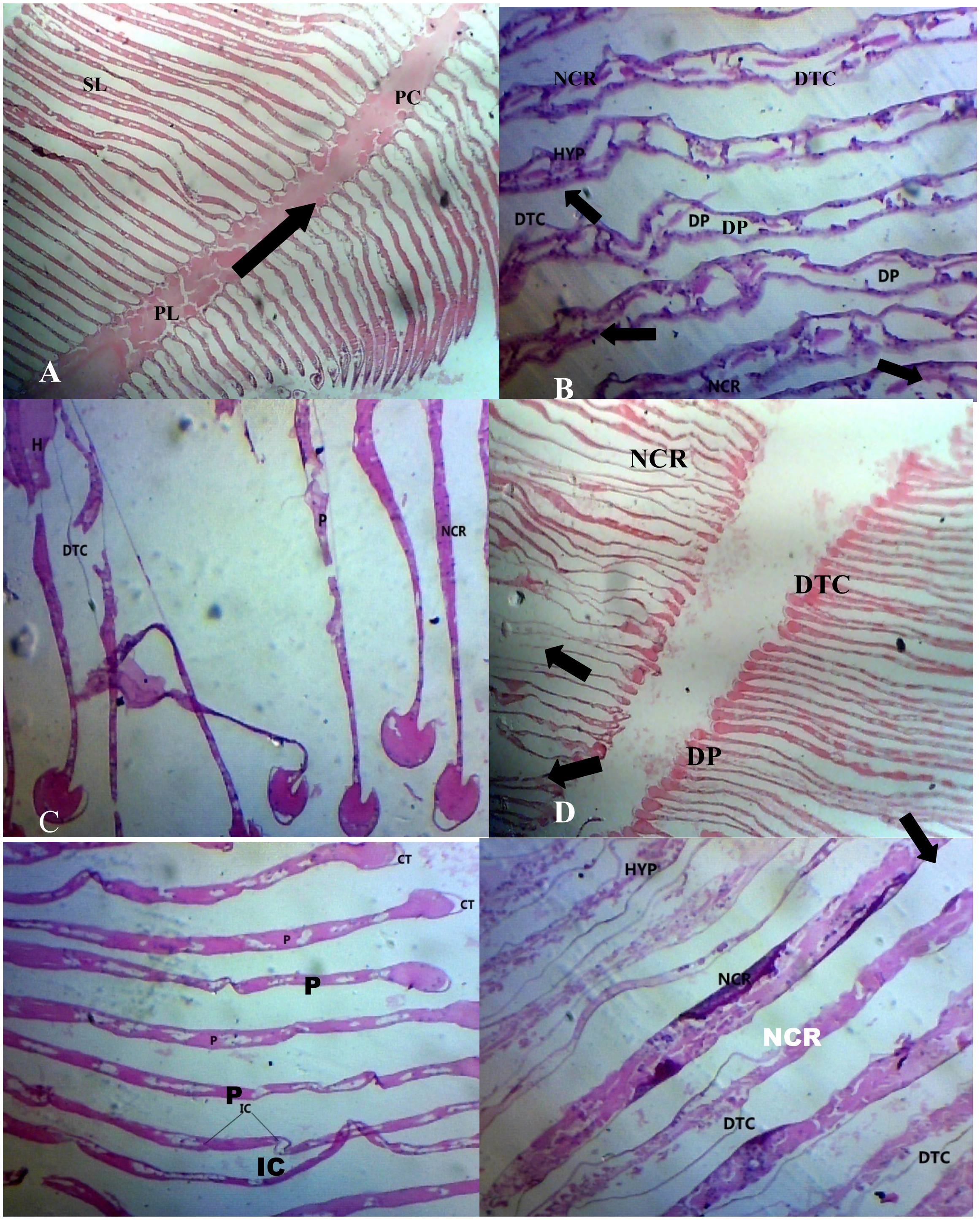


Plate 6A-F: Histopathology of Gills of Blue crab from different Sampling Stations: (A) Mid-lagoon(Control), (B) Makoko, (C) Okobaba, (D) Iddo, (E) Ajah, and (F) Ikoyi. DTC detached cuticle, HYP severe hyperplasia, NCR necrosis, DP distruption of pillar cell, P/PL pillar cell,IC ionocytes, CT cuticle layer intact ,SL secondary lamella or filament, PL primary lamella, H hyperplasia inter lamella space (short arrow).

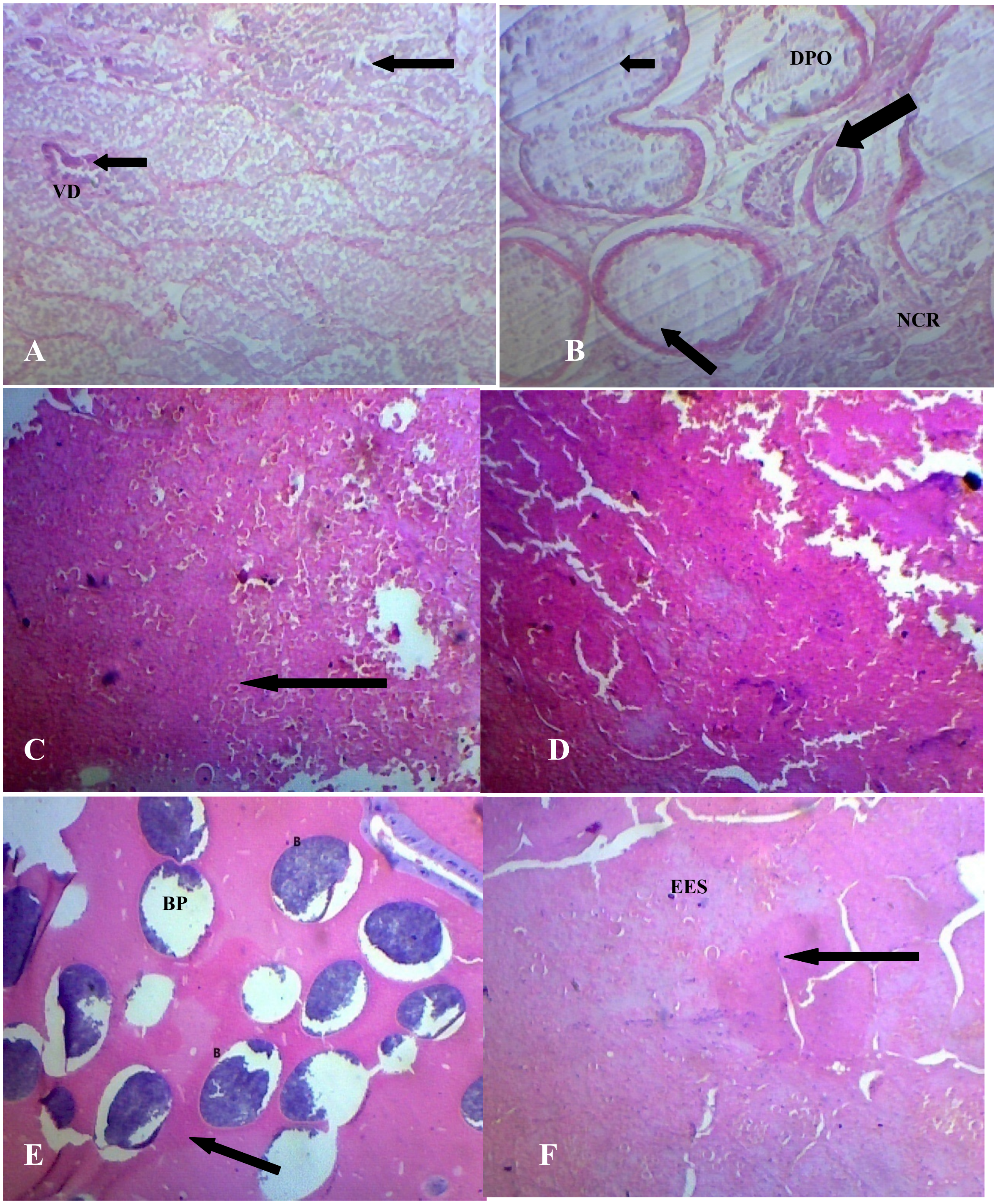


Plate 7A-F: Histopathology of Gonad of Blue crab from different Sampling Stations: (A) Mid-lagoon(Control), (B) Makoko, (C) Okobaba, (D) Iddo, (E) Ajah, and (F) Ikoyi. EES early eosinophilic secretions, BP basophilic patches or secretion,VD vacuolar degeneration, NCR extensive necrosis, DFC deformation of follicle cells, DPO disruption of Primary oocytes.

4.11.4 Muscle

Mid-lagoon:In crab from Mid-lagoon, the muscle tissue showed striated muscle structure with nuclei present and early stage of hyalinization. (Plate 8A).

Makoko: In the muscle of crab from Makoko, the muscle tissues were not intact with loss of striation, loosened muscle bundle, large vacuole, and gap formation between the muscle bundles. (Plate 8B)

Okobaba: Similar changes was also observed in muscle of crab from Okobaba, except fragmentation and fusion of muscle bundle (Plate 8C).

Iddo: The muscleof crab from Iddo crab showed equally spaced muscle bundles with characteristics striation, nuclei were not conspicuous and the epidermis disintegrated (Plate 8D).

Ajah: In muscle of crab from Ajah , the muscle tissue showed large hyalinization, large vacuole and formation of lacunae within muscle strands, and loosened muscle-bundles. (Plate 8E).

Ikoyi: Histopathological changes in muscle from Ikoyi crab showed loss of striated muscle structure and hyalinization.

Fusion and necrosis of muscle tissue, disruption, congestion of fibres with interrupted striation and complete absence of nuclei.(Plate 8F).

Disrupted tissue architecture highlighted in different tissues i.e. the hepatopancreas, gill, gonad and muscle of blue crab from Makoko, Okobaba, Iddo, Ajah and Ikoyi were the reflection of pollution status or toxicity effect of heavy metals in the study area.

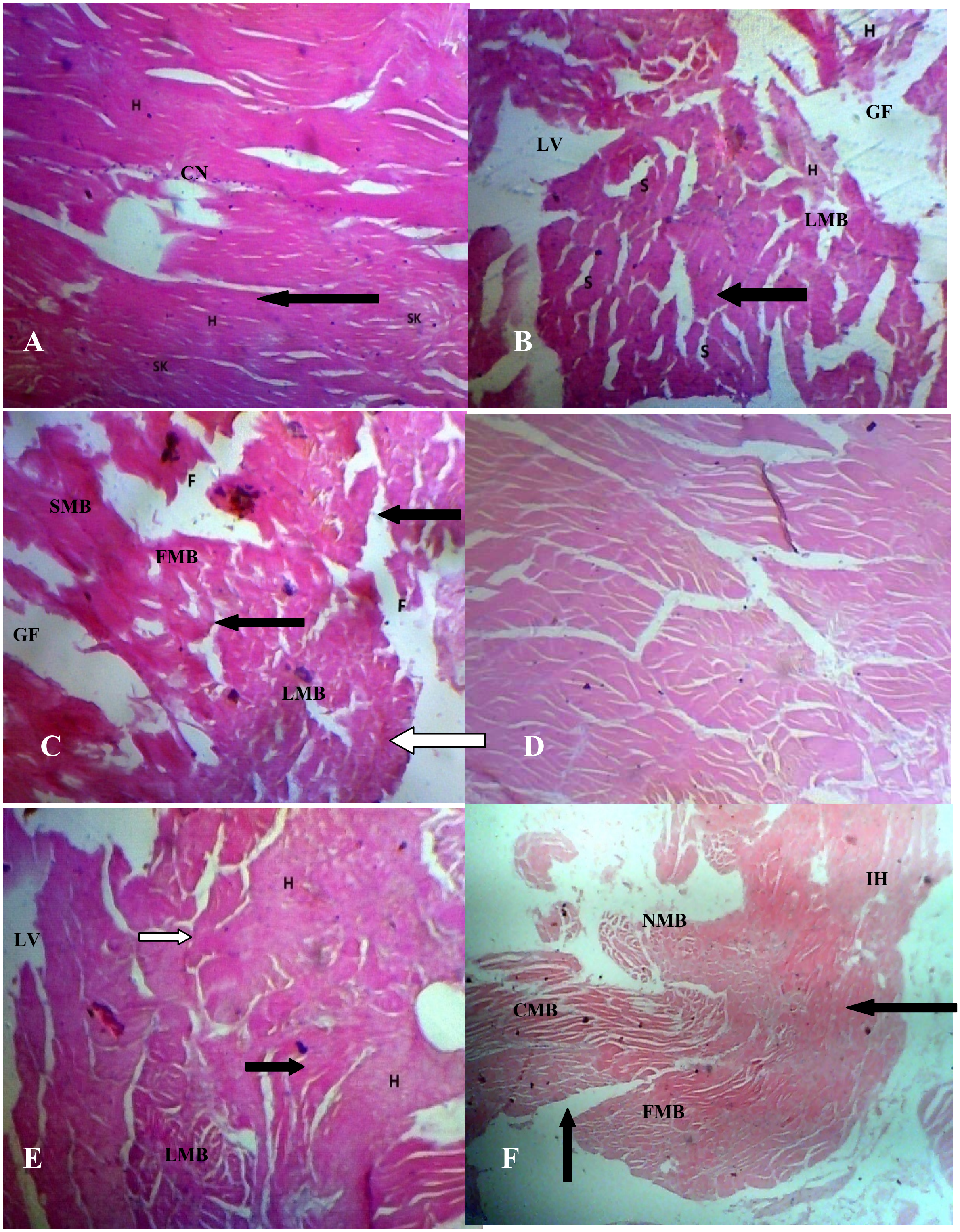


Plate 8A-F: Histopathology of Muscle of Blue crab from different Sampling Stations: (A) Mid-lagoon (Control), (B) Makoko, (C) Okobaba, (D) Iddo, (E) Ajah, and (F) Ikoyi. SK skeletal muscle strand, LMB loosen muscle bundle, FMB fusion of muscle bundle, LV large vacuole, GF gap formation, NMB necrosis of muscle bundle.CMB congestion of muscle bundle, CN congestion of nuclei, DE disintegrated epidermis, SMB striated muscle bundle, H hyalinization, F fragmentation, IH infiltration of hemocytes.

CHAPTER FIVE
DISCUSSIONS

The aquatic environment receives daily substantial amounts of environmental pollutants including heavy metal pollutants from point and non-point sources. These pollutants are capable of inducing oxidative stress in aquatic biotaby means of ROSgenerated within tissue (Halliwell and Gutteridge, 1999), this was confirmed by the findings of the present study. Documented metal contaminants observed presently included Cd, Pb, Zn and Cu, and could therefore be responsible for oxidative stress induced in crab organ. Metals modulate ROS through either redox cycling or antioxidant disruptions (Halliwell and Gutteridge, 1999 and Ercal *et al*., 2001). This situation, where ROS overwhelm antioxidant defenses leading to subcellular damage, is called oxidative stress (Kelly *et al*., 1998).

The uptake of these pollutants by aquatic organisms can be from sediments, suspended particulate matter with toxic properties, and food sources. Exposure to these contaminants will depend on the particular dietary and ecological lifestyles of the aquatic organisms (Livingstone, 2003 and Athanasios *etal.,* 2006). Antioxidants are biomolecules that function as scavengers of free radicals. Most antioxidant biomarkers bind and inactivate the free radicals, forming innocuous end products such as water. Thus, antioxidants protect against oxidative stress and prevent damage to cells bymeans of antioxidant systems which scavenge or eatup free radical in order to abate cellular injury and sustain physiological stability within cells(Chow, 1988; Kirchin, Moore*et al.,* 1992; Filho *et al.,* 2001).

Physico-chemical Parameters

The daily and seasonal fluctuations of physico-chemical properties in aquatic habitats also predispose aerobic organism in such habitat to oxidative stress. These are fluctuations in temperature, salinity and concentration of dissolved oxygen (Lesser, 2006). The season and anthropogenic activities in and around the environment, also influence and vary the range of physico-chemical parameters in aquatic environments, including Lagos Lagoon in particular which is 3m deep except for the dredged areas. The inflow of water from rivers and creeks into the lagoon during raining season, also affect the physical properties (Hill and Webb, 1958). The inflow of salt water from the Atlantic coast through Commondore Channel, also affect the salinity of this part of the Lagoon.

Insignificant increase in water temperature recorded in all the sample stations except Mid-lagoon, may be impinged on heat generated from the decomposition of organic matter and other anthropogenic activities reported in the sample stations except in Mid-lagoon. This was similar to reports given by Fafioye *et al*., (2005) who worked on Omi water body, Ago iwoye, Ogun state, Nigeria. Similar observation was reported by Babalola and Agbebi (2013) who examined the physico-chemical characteristics and water quality of Kuramo Lagoon, Lagos, Nigeria. Surface water temperature influences key biogeochemical processes such aslevels of dissolved oxygen, organic contaminant degradation and resultant availability of metabolites which inturn determine risks to reproductive activities and rate of metabolic function

Significant higher DO recorded in Ajah than recorded in other stations, which is also above NESREA and WHO standard limit are reported common effects of anthropogenic activity, like dredging which was ongoing at the time of the present study; disruption of natural habitat and death of some organisms, biological processes involve in organic matter breakdown could explain the observed rise in DO to level above 6. Dimowo (2013) in studying some physicochemical parameters of surface water in River Ogun (Abeokuta,Ogun State, in Southwestern Nigeria)reported similar findings.

Higher salinity recorded in Iddo, Ikoyi, Okobaba and Makoko respectively than Ajah and Mid-lagoonmay be attributed to influx of sea water from Commondore Channel which is very close to these stations. The significant alkaline pH recorded in all the stations in this study can be attributed to the buffering effect of the coastal waters (Olaniyan, 1969).

The lower Biological Oxygen Demand (BOD) than the reported standard limit, recorded in all sample stations suggest a high variation in dissolved organic matter concentration in all the sample stations. In addition, this may suggest influence of periodic discharge of sewage containing varying amounts of biodegradation substances. This was also reported by Babalola and Agbebi (2013), who examined the physicochemical characteristics and water quality assessment of Kuramo Lagoon, Lagos, Nigeria.

Heavy metal Concentration in Crab organs, sediment and water

The significant variation in Cd, Pb, Zn and Cu concentration in the blue crab organs with increasing concentration ranging from hepatopancreas followed by gonad, gill and muscle, though falling above the NESREA and WHO permissible limit and Zinc concentration in the gills from all the stations, gonad and muscle from Mid-lagoon and muscle from Okobaba falling within NESREA permissible limit were confirmation of the major functional differences in the organs in terms of membrane permeability and enzyme system. The variation is also an indication of the degree to which particular species pick up particulate matter from surrounding water and sediments in particular while feeding.

Higher concentration of metals recorded within hepatopancreatic tissue is in line with its function as a digestive organ that detoxifies pollutants and its high sensitivity to physiological and environmental changes. The hepatopancreas is also a site of bio-transformations. Several reports earlier confirmed that bioaccumulation of heavy metals was more in hepatopancreas than other tissues (Bunt, 1986; Vijayaraman *et al.,*1999among others). The highest Pb concentration found in Hepatopancreas from Okobaba may be attributed to the presence of saw-mill industries that use engines powered by fuel, and burn wood waste (saw-dust), as explained by Narayanan (2011) who reported that such wood industry activities are sources of Pb. Hepatopancreas is a digestive organ that detoxifies contaminants or xenobiotics in the crab organs. Gill as a respiratory tissueinteracts directly with surface water aquatic environment, so they are easily exposed to pollution directly and are responsible for other vital physiological functions like excretion, acid base balance and ion regulation.

The highest Zinc concentration ($p<0.05$) recorded in hepatopancreas from Ajah may be as a result of the sedimemt mixing up due to on-going local and industrial dredging in the station. Narayanan (2011) reported the major sources ofZinc in aquatic environment is through mining and industrial waste. Zinc is a metal of biological importance, but when it is above permissible limit, it becomes toxic to the organism. However, Zn concentration during the present study was above NESREA and WHO standard limit in all stations except control station.

The highest Cu and Cd concentration ($p<0.05$) recorded in gonad of the crab from Makoko station may be attributed to diverse anthropogenic activities being residential areas and domestic waste generated from the activities like direct defecation, disposal of solid waste (like nylon bag, paper bag, plastics and so on), fishing activities, and most of which generate organic and inorganic waste which are possible sources of Cu and Cd. Additionally, higher incidence of Cd in the gonad tissue of blue crabs from Makoko, reflects the polluted state of the environment in the availability, uptake and subsequent retention of trace metals in the tissue of crab. It has been reported that the uptake of pollutants such as trace metals in water can be direct via an integumentary system trapping of adsorbed trace metals in suspended particulate matter, or by preying on organisms with accumulated levels of these trace metals (Brucka-Jastrzebska 2010; Letendre, *et al.*, 2012). Žikić *et al*. (2001) documented the relationship between altered physiological conditions and pathological outcomes in the gold fish *Crassius auratus gibelio* and the induction of oxidative stress following the species' exposure to varied concentrations of Cd. Studies on vertebrates exposed to dietary Cd revealed extensive damage to the ovary (Massányi *et al*., 2005; Yang *et al*., 2012). Other studies demonstrated reprotoxic effects such as follicular atresia in the vertebrate ovary (Massányi and Uhrín, 1996), degenerative alterations in testes (Massányi *et al*., 2002; Toman and Massányi 2002) and decreased motility of spermatozoa (Lukáč *et al*., 2003; Massányi*et al*., 2004). Massányi *et al*. (2005) demonstrated negative effects of cadmium on the ovarian structure and reported a reduction in the primary follicle count after the intraperitoneal administration of cadmium.

The result of the analysis in the present study has shown that the crab *C. amnicola*can be used as a bio-indicator of pollution as it contains variable levels of the metals analyzed with high enrichment of Cu, Pb, Cd and Zn observed. Distribution and accumulation of heavy metals in crab organs vary widely depending on size, sex, growth stage, molting, migration, season of sampling, metal bioavailability, hydrodynamics of the environment, changes in tissue composition and reproductive cycle. Crabs examined in the present study have very similar diets; they are all omnivores which feed mainly on invertebrates like: shrimp, as well as bivalve and vegetation. The difference in the foraging grounds of these crabs could have led to variations in prey size and ultimately, variation in metals intake. Crabs also spend more time in shallow waters, estuaries and coastal areas where anthropogenic metals are infinitesimal. Dissimilar levels of heavy metal in organs of this species is not abnormal, and could be attributed have also contributed to the different functions and metabolic specialty of each organ.

The concentrations of Cu significantly exceeded ($p<0.05$) other metals in the water sampled suggesting the influence of inappropriate disposal of radioactive waste, soil parent materials, sludge, industrial effluents, fertilizers in agricultural runoff and atmospheric fallouts in the surroundings as explained by Adeleye*et al.* (2011).

Expectedly, the highest concentration of Zn, Cu, Cd and Pb ($p<0.05$) found in the sediment, followed by crab organs (Hepatopancreas, gills, gonad and muscle) and least in water may be because sediments, according to Kakulu and Osinbanjo (1988) and Don-Pedro *et al*. (2004), often acts as a receptacle for any waste materials or pollutants dropped in aquatic environments.

Furthermore, the highest concentration of Pb, Zn and Cu ($p<0.05$) recorded in sediment from Iddo may be due to the fact that Iddo is a point source that receives sewage from different residential areas through septic tankers on a daily basis according to reports by Oyewo *et al.* (2009). It may also be due to clay-like materials that form the Iddo sediment structure, as well as the grain size of these materials. This was also reported by Odiete (1999), affirmed the role of sediment as a majorenvironmental matrix for metal retention.

The concentration of Cd was generally low. However, it was notably higher ($p<0.05$) for sediment sampled within Makoko ($p<0.05$) than sediment from other stations. Effluent inflow and artisanal handwork/workmanship from the coastal community may be implicated as possible sources for this metal.

The fact that crabs consume organic substances present in the bottom of sediments of aquatic systems, makes them good biomonitors for pollutants presents in the ecosystems. It is also an important fact that the crab species represents a source of both income and nourishment to the marginal population. Differences in heavy metals concentrations among the crab species is likely to have resulted from metal bioavailability, hydrodynamics of the environment, changes in tissue composition, reproductive cycle, different feeding mechanism, temperature, salinity, stations of collection and sources of pollution within the Lagos Lagoon (Adeleye *et al.*, 2011). The high concentration of heavy metals in commercially important crustaceans sampled from the Lagos Lagoon is a cause of concern and calls for regular monitoring of water quality around the point sources within the Lagos Lagoon. The fact that crab consumption is a main source of heavy metal in-take in people not occupationally exposed, amplifies the need for preventive measures to safeguard public health.

Heavy Metals Uptake Pattern in *C amnicola* in relation tothe Sample Stations

The biplots of metal concentration in hepatopancreas which is closely connected at the tip of ellipses of the Mid-lagoon and other station and the biplots of metal concentration in gill and gonad of *C. amnicola* which are distinct ellipse indicate that the uptake patterns of metals in hepatopancreas, gill and gonad of *C. amnicola* from mid-lagoon was different from uptake patterns of metals in hepatopancreas gill and gonad of crabs from other stations.

The distinct uptake patterns of heavy metals observed in hepatopancreas, gill and gonad of crab from Mid-lagoon and other stations may be attributed to the sediment texture and composition including grain size, organic matter content.Distributionof grain sizes has been reported to influence trace metal levels and uptake in coastal environments (Luoma, 2000). Reports have demonstrated that trace metals reside mostly in the silt/clay matrices of sediment, i.e. particles with size <0.063 mm (Krumgalz *et al.,*1992). Also, the organic matter content of sediments increase as the sediment texture becomes finer (Williamson & Wilcock, 1994; Denton *et al*.,2001). The presence of organic matter can potentially increase metal concentrations in sediment by adsorption of metals from surrounding environment onto organic material (Loomb, 2001). Also dead organisms in sediments may carry the heavy metals with them, either taken in by the organism while alive or sorbed on to the animal before or after death (Fergusson, 1990) and this contribute directly to the metal levels in the sediments.

Pollution Load Index

Reports interpreting the significance of the PLI values of an environment have demonstrated that index-values less than one portray an environment that is relatively undisturbed by human activities, or highly diluted conditions and dispersed metal content with furtherancefrom point sources; values greater than one portray a ongoing environmental deteriorationtraceable to various scale of ongoing anthropogenic activities (Suresh *et al*., 2011). Thus the PLI values recorded for this study which ranged between 0.38 in Ikoyi and 1.9 in Makoko during dry season and between 0.3 in Ajah and 1.5 in Iddo during wet season, depict that the dry season was a more polluted period for the stations compared to the wet season. The differential trend of the PLI for the study stations across seasons suggests that the higher trends in the dry season and lower trends in the wet season observed in the Makoko and Okobaba stations may be attributed to the higher effect of point-sources of pollution during the dry season and dilution of point source effects during the wet season. The difference in indices results in Iddo station which showed a reverse trend with higher pollution load in the wet season compared to the dry season may be attributed to relative sensitivity of indices for contaminants in sediments. The higher PLI (>1) recorded in Makoko, Okobaba and Iddo confirmed that these stations experienced active input of industrial contaminants, sewage and other anthropogenic waste discharged in stations. Elsewhere Kamaldeen and Wahab. (2011), similar indices were applied for regional evaluation of ecological health.

Biota-Sediment Accumulation Factor (BSAF)

The incidence ofrelatively higher concentrations of metals in sediment compared to surface water has been documented by Bower (1979), Fabris *et al*. (1994), Lau *et al*. (1996, 1998), Besada *et al*. (2001) and Eja *et al*. (2003) among others. Sediment functions as a key meatl-depository carrying more than 90 percent of total incidence of metals in aquatic systems (Odiete, 1999). Expressionmetal incidence using the of biota-sediment accumulation factor (BSAF) is useful when comparing the order of uptake of metals. The observed higher BSAF in crab gills indicates that gills has a high potential to concentrate heavy metals (Ademoroti, 1996; Odiete, 1999; Eja *et al*., 2003).

The high BSAF greater than one indicates that a considerable amount of trace metal in gill of *C. amnicola* was taken-up from sediment reserves and were tending towards bioaccumulation. The higher Cu BSAF recorded in gill at Ikoyi may be attributed to the nature and constituent of anthropogenic waste deposited in the station.

The indication of the result is that different environments presented circumstances that allow for the uptake, retention, and loss of contaminants by the blue crab. Reports have shown that although aquatic biota are able to regulate and control metal levels in tissues through selective uptake, storage, detoxification, or any combination (Depledge and Rainbow, 1990; Mason and Jenkins, 1995), the extent or efficiency of regulation achieved by the organism is often a function of bioavailability of the metal within the ecosystem and tissue affinity (Sears, 2013). In addition, biota often actively regulates metal bioaccumulation via saturable kinetics and dynamic feedback systems that respond to environmental loading and maintainance of homeostasis (Wood, 2001). As such, high metal concentrations recorded in most of the crab samples may be a reflection of the organism's inability to regulate uptake efficiently due to the overwhelming concentrations in the Lagos Lagoon environment.

Condition Factor

The significantly higher mean condition factor index (CF) of *C. amnicola*in the mid-lagoon arearelative to the samples in other stations suggest that blue crabs from stations (representing stations adjacent to land areas) other than the mid-lagoon had a significantly lower measure of physiological condition indicating a generally lower physiological fat storage, a possible implication of lower food intake, decreased availability of quality prey organisms, or sub-optimal metabolic capacity y due to environmental stressors. Similar observation was also reported by Adeogun *et al.* (2012) who noted relatively better condition factor in the un-exposed fish relative to fish introduced into serial dilutions of industrial effluent. These authors reported induced oxidative stress and reduction in growth. Fafioye *et al*., (2005) also reported lower and higher condition factor in shellfishes and fin fishes collected from organochlorine pesticide polluted areas of Lagos lagoon.

Oxidative Stess Parameters of crab organs

The significant low activity of SOD recorded in Hepatopancreas, gills, liver and muscle of blue crab during dry and wet season in all stations sample except Mid-lagoon may be attributed to SOD function.

The significant increase activity of catalase observed in gill, gonad and muscle of crab from all the station during dry and wet season may be physiological adjustments following contaminant exposures (Bebianno *et al.,* 2004). Onset of stress reaction triggering massive progression of free radicals which eventual disturb physiological balance in aerobic organism have been documented (Yildirin *et al*. (2011). Similarly, CAT activity in the gill tissue may be on the basis of direct contact with pollutant-laden surface waterand eventual xenobiotic uptake via its thin epithelial cells (Farombi *et al*., 2007).

The low activity of CAT recorded in hepatopancreas from all station except Mid-lagoon, may be attributed to the response of the organ antioxidant enzyme (CAT) to the increased concentration of Pb and Zn recorded within hepatopancreatic tissue. The significant increases in CAT activity and lower activity in SOD, demonstrate a ‘‘disturbance’’ from heavy metal pollutants in the sample stations of Lagos lagoon reflecting the intensity of pollution in the area. But, these induced antioxidant defense enzyme increases were not enough to reduce lipid peroxidation (MDA) levels in the polluted stations, hence the significant increase in MDA.

Superoxide Dismuthase functions in the first line of antioxidant activity by rapidly converting superoxide anion (O2-) to hydrogen peroxide (H2O2) so as to prevent it from participating in the formation of harmful hydroxyl radicals, while CAT and GPx represents the next line of defense aimed at eliminating H2O2 by-product from SOD reaction (Aitken and Roman, 2008).

Although GPx and CAT are notable in peroxidase activity, a higher incidence or predominance of GPx in gonads has been reported (Peltola, *et al*.,1992 and Ziniand Schlegel, 1996) thus suggesting tissue-specific antioxidant potentials and capacity.

The observed significant ($p>0.05$) decrease in the reduced glutathione detoxification system in the crab gill may be attributed to the gill having as a primary contact point with environmental pollutants thus signifying that the gill tissue provided a sensitive biochemical indicator of environmental pollution. However, the decreased ($p<0.05$) GSH concentration observed in gills of C. amnicola from Ikoyi during the dry season and Hepatopancreas of crab from Ajah during the wet season may be hinged on aggravated oxidative damage due to greater trigger in production of free radicals (Figure 8). This concurs with reportsof Doyotte *et al*. (1997) for marine invertebrates interacting with unnatural levels of trace metalswithin ambient environment.

The significant increase of lipid peroxidation levels ($p<0.05$) recorded in different organs of *C.amnicola* collected across the study sites may be attributed to activities of antioxidant enzyme up-regulated in the event of oxidative stress in different crab tissues due to exposure to heavy metals. The significant increase in lipid peroxidation markers may also indicate the susceptibility of lipid molecules to Reactive Oxygen Species and the extent of oxidative damage imposed on these molecules. Therefore, oxidative damage occurs in the crab organs since the higher lipid peroxidation is significant. The fact that CAT activity and lipid peroxidation concentration are both elevated at the all sample stations except Mid-lagoon, suggest that there is excess of H_2O_2 which diffuses into the cells causing oxidative damage. Although CAT removes most of the H_2O_2 by increasing its activity levels, it cannot maintain a homeostasis with high concentration of Cd, Pb, Zn and Cu in the hepatopancreas, gill, and gonad which generate HO^- radicals, thereby causing increased concentration of lipid peroxidation. Therefore we conclude that increase CAT activity in crab organs is not sufficient to eliminate H_2O_2 before the formation of hydroxyl radicals as it has been suggested by others (Bebianno *et al*., 2005).

The low antioxidant (SOD, GPx and GSH)activity documented for all tissues of blue crab examined in this study,explains the high incidence of lipid peroxidation in the tissues of the blue crab. Several studies have demonstrated increased incidence of lipid peroxidation in aquatic biota exposed to elevated pollutant levels (Olakolu *et al.,* 2012) and of contaminated sediments (DiGiulio *etal*., 1993; Livingstone, 1993; Sole *et al*., 1996). Elevated lipid peroxidation was observed by Filho *et al*. (2001) in cichlid fish taken from polluted sites, compared to clean sites. Kamaldeen and Wahab have demonstrated increase in lipid peroxidation in gonads of white suckers exposed to pulp and paper mill effluents as well as municipal sewage treatment plant effluents. Increase in lipid peroxidation was observed in naphthalene exposed marine crab *Scylla serrata* by Sole *et al.* (1996).

Oxidative stress previouslyassociated with several pathological features in mammals (includingmutagenesis, atherosclerosisamong others (Neves *et al.*, 2000) and in molluscs and fish (Di Giulio *et al*., 1993). Various responses of crustaceans to pollutants included the lower activities of SOD, GPx and GSH concentration in alltissues of blue crab examined observed during dry season than wet season compared with activities in all stations except Mid-lagoon; may be attributed to higher anthropogenic activities which increase the heavy metal pollutant whichpenetrate through epithelial cells and increase the rate of ROS production and inhibit homeostasis between the crab organs and the antioxidant enzymes. A similar trend of variation was also reported by Filho*et al*. (2001) and Doherty *et al.* (2010).

The antioxidant enzymes (SOD, CAT and GPx) and the oxyradical scavenger (GSH) appear to be ineffective in providing protection to the tissues of blue crab since significant increases in lipid peroxidation were observed in hepatopancreas, gills, gonad and muscle *C. amnicola* sampled from all the stations. Observations correlate with the report by De Zwarts*et al*. (1997) who demonstrated increase in lipid peroxidation in liver microsomes from rats and salmon exposed to the iron ore mines leachate. Geret, Jouan*et al*.(2002) also investigated increase lipid peroxidation and antioxidant levels in gills of clams exposed to Cd. Peroxidation in muscle and liver tissues of fish was used to measure pollution by a petroleum refinery as reported by Avci *et al*. (2005).

Histopathology of crab organs

The exposure of biota to sub-lethal levels of metals contaminants in their habitatcould disruptcritical biochemical, physiological and histological features of the organism (Hermenean *et al*., 2015).

The histopathological consequences ofparasites in tissue has also been highlighted from this present study where early single-walled cysts were coincided withsevere lesions (granuloma, and necrosis) in hepatopancreatic tissue of blue crabs from Okobaba areas. This feature represents a first documentation of trematode cysts in crab tissue from any part of the Lagos lagoon. Early cyst observed in three walled cyst reported in hepatopancreas of crab from Okobaba was similar to spherical three layered cyst metacercaria of *Microphallus* species described by (Anantaraman and Subramoniam, 1976).

Furthermore, the incidence of parasite cysts in hepatopancreatic tissue of blue crabs from Okobaba may be attributable to decline in habitat quality. The environment is a determinant factor of parasite transmission either directly, by enhancing or encouraging the survival of the transmission stage (e.g. cysts, eggs, or free-living larvae i.e. cercariae) or indirectly, by encouraging the distribution and survival of the host (intermediate or definitive) or vector; in essence poor habitat quality due to the presence of pollutants could increase the likelihood of the parasite and host coming into contact.

Histopathological changes in the hepatopancreas from the Okobaba and Iddo stations could be traceable in theory to its tendency to take up xenobiotics as a detoxifying organ and metabolic centre. The disrupted basal laminae within tubulesofhepatopancreatictissue from Makoko and Ajah suggest that tissue integrity was affected due to the increased Cd,Cu,and Zn pollution status of the stations. Unaturalincidence of hemocytes in the interstitial sinuses of Iddo crab suggest the milited functionality of cellular/host defense in response to the initial stages of tissue damage (Bodhipaksha and Weeks-Perkins, 1994).

The hepatopancreas is key in crustacean metabolic processes. Maharajan *et al*.,(2013) also reported various tissue damage in fresh water crab exposed to high concentration of Chloropyrifos and Cypermethrin (Nurocombi). Stenitiford and Feist (2005) also reported concurrent changes in metabolic condition of the connective tissue storage cell status in the presence of parasite load.

The structure of crab gills across stations differed so was and the severity of lesions; for example, hyperplasic tissues and epithelial lifting indicateshort term encounter with contaminants in surface water. These lesions have also been categoried as adaptive reactionsto reduce xenobiotic uptake (Banerjee, 2007, Maharajan *et al*., 2013)The structural changes (necrosis) in the gills of blue crabs from Okobaba, Iddo and Ikoyi are indicative of chronic stress patterns as described by (Avci et al., 2005). Documented findings by Figueiredo-Fernandes *et al.* (2007) onfirm reports that attribute severity of lesions to levels of toxicant exposure.

The pollution exposure and the effect on the antioxidant system might have resulted in the lesions, haemocytes infiltration and dead cells that were observed in the gonads. Gonads had the highest concentration of Pb and Cu, the histopathological result showed a reflection of the pollution effect on the organ. The adverse effects on the proper function of gonad, was reported to retard reproductive success and atimes prevent successful hatchlings (Lawal-Are and Kusemiju,2010).In view of these past reports, there is possibility of induced oxidative stress in crab following heavy metal pollutant effects in reproductive organ of the blue crab in the present study.

In this study, the alterations in muscle pathology of *C. amnicola* from all the stations except Mid-lagoon could be as a result of abrupt influence of heavy metal pollutants on muscle epidermis abruptly. The observations in the present investigation is in line with a similar observation reported by Tehrani *et al*. (2011). Das and Mukherjee, (2000) also reported induce separation of muscle bundles and intracellular edema in the muscle tissues of fishsubjected to hexachloro cyclohexane.

CONCLUSION

The findings in the present study show that oxidative stress was induced in crab organs (hepatopancreas, gill, gonad and muscle) sampled from the selectedstations from Lagos lagoon (Makoko, Iddo, Okobaba, Ajah, Ikoyi) except Mid-lagoon.This was evidenced in the significant elevationof lipid peroxidation concentration in hepatopancreas, gill, gonad and muscle tissue of the blue crab during the dry and wet season and also, with notablylowconcentrations ofSOD, CAT, GPx and GSH.

The induced oxidative stress observed in this study resulted from heavy metal effects in the blue crab organs,which confirmed the fact that the studied sites around Lagos lagoon (Makoko, Okobaba, Iddo, Ajah and Ikoyi) were polluted.However, blue crabs from these polluted stations may not be ideal for consumption due to the observed concentration of heavy metal in the crab organs which were higher than the acceptablelimit,in addition to the chronic threat it may pose to human health along the food chain.Information on responses by antioxidant defence apparatus including antioxidant enzymesin crab tissuesuggest possible usage of the blue crab as a reliable and sensitive sentinelfor monitoring biota stress and general aquatic health.

Tissue alterations in hepatopancreas, gill and gonad, metal concentrations in tissue and their positive association with lipid peroxidation, highlight the potential for contaminant to negatively impact organism health via oxidative stress. The positive association between tissue alterations, lipid peroxidation, and its negative association with antioxidant activity indicate that lowered antioxidant activity predisposes tissues to oxidative stress. It is anticipated that the findings specific for each area of the lagoon will spur improved management efforts towards better strategies for conservation of the blue crab and other biota in the Lagos lagoon.

CONTRIBUTION TO KNOWLEDGE

This study provided station-specific (as determined by the nature of waste received)information on antioxidant responses, histopathological changes and bioaccumulation in tissues of *Callinectes amnicola*(blue crab).

The study further provides a first report of antioxidant activity in blue crabs from Lagos lagoon as a response to pollutant exposure in the lagoon and also a first report of histopathological changes in the blue crab due to pollution in the Lagos lagoon.

REFERENCES

Abdel-Tawwab, M., Mousa, M.A.A., Ahmad, M.H. and Sakr, S.F.M. 2007. The use of calcium pre-exposure as a protective agent against environmental copper toxicity for juvenile Nile tilapia, *Oreochromis niloticus* (L.). *Aquaculture.* 264: 236–246

Abele, D. 2002. Toxic oxygen: the radical life-giver. *Nature.* 420: 27.

Abele, D. and Puntarulo, S. 2004. Formation of reactive species and induction of antioxidant defence systems in polar and temperate marine invertebrates and fish. *Comparative Biochemistry Physiology*. 138: 405- 415.

Abele, D., Grobpietsch, H. and Pörtner, H.O. 1998. Temporal fluctuations and spatial gradients of environmental O_2, temperature, H_2O_2 and H_2S in its intertidal habitat trigger enzymatic antioxidant protection in the capitellid worm *Heteromastus filiformis*. *Marine Ecology Program Services.* 163: 179-91.

Abulude, F.O, Fapohunda, O.O, and Awanlemhen, B.E., 2006. Determination of some Heavy Metals in *Procambaris clakii, Palaenon sp., Macrobrachium vollenhoventi and Penaus notialis* from the Coastel Water of Ondo State, Nigeria. *Journal of Animal and Veterinary Advances*. 5 (1): 38-41.

Adebayo, O.T., Balogun, A.M. and Olubiyi, O.A.2007.Chemical Analysis of some Industria; Discharge into Lagos lagoon. Nigeria.*Research Journalof Environmental Sciences.*1(4): 196-199.

Adeleye, A.O., Shelle, R.O.D and Akinnigbagbe, A.E. 2011. Pollutant Dynamics and Distribution in Sediments North of Lagos Lagoon Ecosystem. *Nature and Science*. 9(5):13–16.

Ademoroti, C.M.A. 1996. *Environmental Chemistry and Toxicology*. Foludex Press Ltd, Ibadan. p. 217

Adeniyi, A.A. and Yusuf, K.A. 2007. Determination of heavy metals in fish tissues, water and bottom sediments from Epe and Badagry Lagoons, Lagos, Nigeria. *Environmental Monitoring Assessment*. 37: 451-458.

Adeogun, A. O.,Ogidan, I. M., Oju, R.I., Chkwuka, A. V., Adedara, I. A. and Farombi, E.O. 2012. Long-term exposure to industrial effluent induces oxidative stress and affects growth in *Clarias gariepinus. Research Journal of Environmental and Earth Sciences*. 4(7): 738-746

Agarwal, A., Aponte-Mellado, A., Premkumar, B.J., Shaman, A. and Gupta, S. 2012. The Effects of Oxidative Stress On Female Reproduction: A Review. *Reproductive Biology and Endocrinology.*10**:**1.

Ahmad, I., Hamid, T., Fatima, M., Chand, H.S., Jain, S.K., Athar, M. and Raisuddin, S. 2000. Induction of hepatic antioxidants in freshwater catfish (*Channa punctatus* Bloch) is a biomarker of papermill effluent exposure. *Biochimica et Biophysica Acta.*1523: 37–48.

Ahmad, I., Maria, V.L., Oliveira, M., Pacheco, M. and Santos, M.A. 2006. Oxidative stress and genotoxic effects in gill and kidney of *Anguilla anguilla* L. exposed to chromium with or without pre-exposure to beta-naphthoflavone. *Mutation Research.* 608: 16–28

Ahn, I-Y., Lee, S.H., Kim, K.Y., Shim, J.H. and Kim, D-Y. 1996. Baseline heavy metal concentrations in the Antarctic clam *Laternula elliptica* in Maxwell Bay, King George Island, Antarctica. *Marine Pollution Bulletin*. 32: 592-8.

Aitken, R. J. and Roman, S.D.2008. Antioxidant systems and oxidative stress in the testes. *Oxidative Medicine and Cellular Longevity*. 1(1): 15-24.

Ajao, E.A. 1990. The influence of domestic and industrial effluents on populations of sessile and benthic organisms in Lagos Lagoon. Ph.D. Thesis, University of Ibadan, Nigeria. 413p.

Ajao, E.A.1996. Review of the state of pollution in Lagos lagoon. *NIOMR Technical Paper* No 106 pp.

Ajao, E.A. 1996. Review of the state of pollution of Lagos Lagoon. Nigerian Institute of Oceanography and Marine Research (NIOMR) Technical Paper No. 106.

Ajao, E.A. and Fagade, S.O. 1990. The ecology of *Capitella capitata* in Lagos lagoon, Nigeria. *Archive for Hydrobiology.*120(2): 229-239.

Ajayi, A.O. and Akonai, K.A. 2005. Distribution pattern of enteric organisms in the Lagos Lagoon, Nigeria. *African Journal of Biomedical Research.*8(3): 163-168.

Akinpelu, D. 2007.*Environmentalists seek remediation of Apapa canal*. *The Punch.* Monday, June 11th. Page 46.

Akinsanya, C.K. 2003. Recent trends in the pollution load on the Lagos Lagoon. – Lagos state perspective. A paper presented on ecological sustainable industrial development *workshop organized by UNIDO.*

Akpata, T.V.I. 1980. Studies on fungal decomposition of sawdust in Lagos Lagoon. Ph.D Thesis, University of Lagos, Nigeria.

Akpata, T.V.I. 2002.*Aquatic Microbes: Impact on Man and environment*. An inaugural lecture delivered at the University of Lagos on 1st July, 2002. 40pp.

Akpata, T.V.I. and Ekundayo, J.A. 1978. Faecal pollution of the Lagos lagoon. *Nigerian Journal Science.*12: 39-53.

Akpata, T.V.I. and Ekundayo, J.A. 1979. Faecal pollution of the Lagos Lagoon. *Nigerian Journal of Science.*12: 39 – 49.

Alexiadis, A. 2007. Global warming and human activity: a model for studying the potential instability of the carbon dioxide/temperature feedback mechanism. *Ecology Module*. 203: 243-56.

Allakhverdiev, S.I., Kreslavski, V.D., Klimov, V.V., Los, D.A., Carpentier R. and Mohanty, P. 2008. Heat stress: an overview of molecular responses in photosynthesis. *Photosynthesis Resources.* 98: 541-50.

Allen, P. 1995. Chronic Accumulation of Cadmium in The Edible Tissues of Oreochrmis Aureus (Steindachner): Modification by Mercury and Lead. *Archives of Environmental Contamination and Toxicology.*29**:**8-14.

Allen, T., Singhal, R., and Rana, S.V. 2004. Resistance to oxidative stress in a freshwater fish *Channa punctatus* after exposure to inorganic arsenic. *Biological Trace Element Research.* 98, 63–72.

Almeida, E.A., Bainy, A.C.D., De Melo, L., Martinez, G.R., Miyamoto, S., Onuki J., Barbosa L. F., Garcia, C.C.M., Prado, F.M., Ronsein, G.E., Sigolo, C.A., Brochini, C.B., Martins, A.M.G., De Medeiros, M.H.G. and Di Mascio, P. 2007. Oxidative stress in *Perna perna* and other bivalves as indicators of environmental stress in the Brazilian marine environment: Antioxidants, lipid peroxidation and DNA damage. *Comparative Biochemistry and Physiology.*146: 588-600.

Alves de Almeida, E., Miyamoto, S., Bainy, A.C.D., Medeiros, M.H.G. and Di Mascio, P. 2004. Protective effects of phospholipid hydroperoxide glutathione peroxidase (PHGPx) against lipid peroxidation in mussels *Perna perna* exposed to different metals. *Marine pollution Bulletin*. 49: 386-92.

Amiard, J.C., Amiard-Triquet, C. Barka, S. Pellerin, J. and Rainbow, P.S. 2006. Metallothioneins in aquatic invertebrates: Their role in metal detoxification and their use as biomarkers. *Aquatic Toxicology.* 76: 160–202.

Amin, O., Ferrer, L. and Marcovecchio, J. 1996. Heavy metal concentrations in littoral sediments from the Beagle Channel, Tierra del Fuego, Argentina. *Environment Monitoring Assessment*. 4: 219-31.

An, M.I. and Choi, C.Y. 2010. Activity of antioxidant enzymes and physiological responses in ark shell, *Scapharca broughtonii*, exposed to thermal and osmotic stress: effects on hemolymph and biochemical parameters. *Comparative Biochemistry Physiology.* 155: 34-42’

Anantaraman, S. and Subramoniam, T. 1976. On A Microphallid Metacercaria Occurring in The Ovaries of the Sand Crabs*Emerita asiatica* and*Albunea symnista* on the Madras Coast. Proceedings of The Indian Academy of 568 Sciences-Section B. *Springer*. 192-199.

Angel, D.L., Fiedler, U., Eden, N., Kress, N., Adelung, D., and Herut, B. 1999. Catalase activity in macro and micro-organisms as an indicator of biotic stress in coastal waters of the eastern Mediterranean Sea. *Helgol Marine Resources.* 53: 209-18.

Ansaldo, M., Najle, R., and Luquet, C.M. 2005. Oxidative stress generated by diesel seawater contamination in the digestive gland of the Antarctic limpet *Nacella concinna*. *Marine Environmental Resources*. 59(4): 381-90.

APHA-AWWA-WEF. 2005. Standard Methods for the Examination of Water and Wastewater. 21st Edn., American Public Health Association, Washington, D.C.

Aranzamendi, M.C., Sahade, R., Tatián, M. and Chiappero, M.B. 2008. Genetic differentiation between morphotypes in the Antarctic limpet *Nacella concinna* as revealed by inter-simple sequence repeat markers. *Marine Biology*. 154: 875-85.

Arillo, A. and Melodia, F. 1990. Protective effect of fish mucus against Cr (VI) pollution. *Chemosphere.*20: 397–402.

Asagba, S.O., Eriyamremu, G.E. and Igberaese, M.E. 2008. Bioaccumulation of cadmium and its biochemical effect on selected tissues of the catfish (*Clarias gariepinus*). *Fish Physiology and Biochemistry*. 34: 61–69.

Ates, B., Orun, I., Talas, Z.S., Durmaz, G. and Yilmaz, I. 2008. Effects of sodium selenite on some biochemical and hematological parameters of rainbow trout (*Oncorhynchus mykiss* Walbaum) exposed to Pb2+ and Cu2+. *Fish Physiology and Biochemistry.*34: 53–59.

Athanasios, V.,Thomais, V. and Manos, D.M. 2006. Molecular biomarkers of oxidative stress in aquatic organisms in relation to toxic environmental pollutants. *Ecotoxicology and Environmental Safety.* 64: 178–189.

Atli, G. and Canli, M. 2003. Natural Occurrence of Metallothionein-Like Proteins in The Liver of Fish Oreochromis Niloticus and Effects of Cadmium, Lead, Copper, Zinc, and Iron Exposures on Their Profiles. *Bulletin of Environmental Contamination and Toxicology.*70**:**0619-0627.

Avci, A., Kacmaz, M., and Durak, I. 2005. Peroxidation in muscle and liver tissues from fish in a contaminated river due to a petroleum refinery industry. *Ecotoxicology Environment Safety.*60: 101–105.

Awosika, L.F., Dublin-Green, C. O., Folorunsho, R., Adekoya, E. A., Adekanmbi, M. A. and Jim-Saiki, L.2000.Study of the main drainage channelsof Victoria and Ikoyi Islands in Lagos, Nigeria and their response to tidal and sea level changes. *Nigerian Institute for Oceanography and Marine Research Paper*. 108 pp.

Baarschers, W.H. 1996. Eco – facts and Eco – fiction: understanding the environment Debate. London. *Rutledge*.

Babalola, O.A and Agbebi, F.O. 2013. Physico-chemical characterisitics and water quality assessment from Kuramo Lagoon, Lagos, Nigeria. *International Journal of Advanced Biology Research.* 3(1): 98-102

Bagnyukova, T.V., Chahrak, O.I. and Lushchak, V.I. 2006. Coordinated response of gold fish antioxidant defenses to environmental stress. *Aquatic Toxicology.* 78: 325–331.

Baker, R.T.M., Martin, P. and Davies, S.J. 1997. Ingestion of sub-lethal levels of iron sulphate by African catfish affects growth and tissue lipid peroxidation. *Aquatic Toxicology.* 40: 51–61.

Baltova, S., and Veluheva, I. 2005. Some morphological and pathological modification of the blood cells from *Alburuus alburuus* in intoxication with heavy metals (Pb, Zn and Cd) Bulgarian. *Journal Agriculture Science*. 11:577-582.

Banerjee, T. 2007. Histopathology of Respiratory Organs of Certain Air-Breathing Fishes of India. *Fish Physiology and Biochemistry.*33**:**441-454

Barbaro, G., Di Lorenzo, G., Asti, A., Ribersani, M., Belloni, G., Grisorio, B., Filice, G. and Barbarini, G. 1999. Hepatocellular Mitochondrial Alterations in Patients with Chronic Hepatitis C: Ultrastructural and Biochemical Findings. *The American Journal of Gastroenterology.*94**:**2198-2205.

Barbieri, E.S., Villafañe, V.E. and Helbling, E.W. 2002. Experimental assessment of UV effects on temperate marine phytoplankton when exposed to variable radiation regimes. *Limnology Oceanography*. 47: 1648-55.

Baumann, H. and Gauldie, J. 1994. The Acute Phase Response. *Immunology Today.*15**:**74-80.

Beattie, M. 2005. *Permanent Opening Structure for the drainage of Lake Ellesmere, Te Waihora*. 28pp.

Bebianno, J.M. 1998. The determination of heavy metal pollutants in fish samples from River Kaduna. *Journal of Chemical Society*. 23: 21-23.

Bebianno, M.J., Company, R., Serafim, A., Cosson, R.P. and Fiala-Medoni, A. 2005. Antioxidant systems and lipid peroxidation in Bathymodiolusazoricus from Mid-Atlantic Ridge hydrothermal vent fields. *Aquatic. Toxicology*. 75: 354–373

Bebianno, M.J., Geret, F., Hoarau, P., Serafim, M.A., Coelho, M., Gnassia-Barelli, M., and M. Romeo. 2004. Biomarkers in *ruditapes decussatus*: A potential bioindicator species. *Biomarkers*. 9(4-5): 300-305.

Bell, J.L. 1988. Distribution and Abundance of *Dissodactylus mellitae rathbun* (Pinnotheridae) On Mellita Quinquiesperforata (Leske)(Echinodermata). *Journal of Experimental Marine Biology and Ecology.*117**:**93- 114

Besada, V., Fumega, J. and Vaamond, A, 2001. Temporal trends of Cd, Cu,Hg, Pb and Zn in Mussel (*Mytilus galloprovinciatis*) from the Spanish North Atlantic coast 1991-1999. *Science of the Total Environment*. pp1-15

Bhattacharya, A. and Bhattacharya, S. 2007. Induction of oxidative stress by arsenic in *Clarias batrachus*: Involvement of peroxisomes. *Ecotoxicology and Environmental Safety.* 66: 178–187.

Blum, J. and Fridovich, I. 1984. Enzymatic defenses against oxygen toxicity in the hydrothermal vent animals *Riftia pachyptila* and *Calyptogena magnifica*. *Archeology Biochemistry*. 228(2):617-20

Bodhipaksha, N. and Weeks Perkins B.A., 1994. The Effects of Methyl Parathion on Phagocytosis and Respiratory Burst Activity of Tiger Shrimp (Penaeus Monodon) Phagocytosis. In: Stolen, J.S., Fletcher, T.C. (Eds.), Modulators of Fish Immune Responses, Models For *Environmental Toxicology Biomarkers Immunostimulators*. 1:11–12.

Bodkhe, M. K. 1983. Effect of Some Pesticidal Pollutant on the Physiology of *Barytelphusa cunicularis*(Ph.D. Thesis). Dr. B. A. M. U, Aurangabad.

Boening, D.W. 1999. *Environmental Monitoring Assessment*. 55; 459.

Bonada, N., Prat, N., Resh, V.H.and. Statzner, H. 2006. *Annual. Revise Entomology*. 51: 495.

Boveris, A. and Chance, B.1973. The mitochondrial generation of hydrogen peroxide. *Biochemistry Journal.*134: 707-16.

Bower, H. J. 1979. Heavy metals in the sediments of foundary cover cold spring. New York; *Environmental Science Technology*. 13, 683-689

Box, A., Sureda, A., Galgani, F., Pons, A. and Deudero, S. 2007. Assessment of environmental pollution at Balearic Islands applying oxidative stress biomarkers in the mussel *Mytilus galloprovincialis*. *Comparative Biochemistry Physiology.* 146: 531-9.

Bradford, M. M. 1976. A Rapid and Sensitive Method for the Quantitation of Microgram Quantities of Protein Utilizing the Principle of Protein-Dye Binding Reproduction. *Analytical Biochemistry.* 72: 248-254

Brucka-Jastrzębska, E.2010. The Effect of Aquatic Cadmium and Lead Pollution on Lipid Peroxidation and Superoxide Dismutase Activity in Freshwater Fish.” *Polish Journal of Environmental Studies.* 19(6): 1139–1150.

Bunt, A.H. 1986. An ultrastructural study of the hepatopancreas of *Procambarus clarkia* (Girard) (Decapoda: Astacides). *Crustaceana.* 15: 282-288.

Busacker, G.P., Adelman, I.R. and Goolish, E.M. 1990. Growth Methods for fish biology (Eds. Schreck, C.B. and Moyle, P.B.). *American Fisheries Society*, Bethesda, Maryland, USA. 446, pp: 363.

Caceci, T., Neck, K.F., Lewis, D.H. and Sis R.F. 1988. Ultrastructure of the Hepatopancreas of The Pacific White Shrimp, Penaeus Vannamei (Crustacea: Decapoda). *Journal Marine Biology Association.* 68: 323–337

Cajaraville, M. P., Bebianno, M. J., Blasco, J., Porte, C., Sarasquete, C. and Viarengo, A., 2000. The use of biomarkers to assess the impact of pollution in coastal environments of the Iberian Peninsula: a practical approach. *Science Total Environment.* 247: 295-311.

Callender, E.2003. Heavy Metals in the Environment--Historical Trends', in H. D. Holland & K. K. Turekian (ed.), Treatise on Geochemistry. *Environmental Geochemistry*. 9: 67-105pp.

Campana, O., Sarasquete, C. and Blasco, J. 2003. Effect of lead on ALA-D activity, metallothionein levels, and lipid peroxidation in blood, kidney, and liver of the toadfish *Halobatrachus didactylus*. *Ecotoxicology and Environmental Safety.* 55: 116–125.

Camus, L., Birkely, S.R., Jones, M.B.,Børseth, J.F., Grøsvik, B.E.,Gulliksen, B., Lønne, O.J.,Regoli, F. and Depledge, M.H. 2003. The Biomarker responses and PAH uptake in *Mya truncata* following exposure to oil-contaminated sediment in an Arctic fjord (Svalbard).*Science of the Total Environment.* 308: 221–234.

Canli,M., Ay, O. and Kalay, M. 1998. Levels of heavy metals (Cd, Pb,Cu ,Cr and Ni) in tissues of *Cyprinus carpio, Barbus capito* and *Chondrostoma* regionfrom the seyhan river Turkey. *Trurkey Journal Zoology*. 22:149-157.

Canterford, G.S., Bichanan, A.S. and Ducker, S.C. 1978. Accumulation of heavy metals by the marine diatom *Ditylum brightrelli*(west) Grunow. *Australian Journal of Marine and freshwater Research.*29: 611-622

Cao, L., Huang, W., Liu, J., Yin, X. and Dou, S. 2010. Accumulation and oxidative stress biomarkers in Japanese flounder larvae and juveniles under chronic cadmium exposure. C 151: 386–392

Cardoso, E., Chiarini-Gracia, L.H., Ferreira, R.M.A. and Poli, C.R. 1996. Morphological Changes in The Gills of Lophiosilurus Alexandri Exposed to Unionized Ammonia. *Journal of Fish Biology.* 49: 778–787.

Carney, A. B. 2008. Oxidative damage in fish used as biomarkers in field and Laboratory Studies. *Dissertation* Published by the Department of Zoology/Zoophysiology Göteborg University, Sweden.

Chakravarty, M. and Patgiri, A.D.2009. Metal pollution assessment in sediments of the Dikrong River, N.E. India. *Journal of Human Ecology.*27: 63–67.

Chambon, C., Legeay, A., Durrieu, G., Gonzalez, P., Ciret, P. and Massabuau, J.C. 2007. Influence of the parasite worm *Polydora* sp. on the behavior of the oyster *Crassostrea gigas*: a study of the respiratory impact and associated oxidative stress. *Marine Biology*. 152: 329-38.

Chandel, N.S. and Schumacker, P.T. 2000. Cellular oxygen sensing by mitochondria: old questions, new insight. *Journal Applied Physiology*. 88: 1880-9.

Chandel, N.S., Maltepe, E., Goldwasser, E., Mathieu, C.E., Simon, M.C. and Schumacker, P.T. 1998. Mitochondrial reactive oxygen species trigger hypoxia-induced transcription. *National Academic Science.* 95: 11715-20.

Chapman, P.M., Allen, H.E. and Godtfredsen, K. 1996. Evaluation of bioaccumulation factors in regulating metals. *Environmental Science Technology.* 30:448–452

Cheung, C.C.C., Zheng, G.J., Li, A.M.Y., Richardson, B.J. and Lam, P.K.S. 2001. Relationships betweentissue concentrations of polycyclic aromatic hydrocarbons and antioxidative responses of marine mussels, *Perna viridis*.*Aquatic Toxicology.* 52: 189–203.

Chindah, A.C.and Braide, S.A. 2003. Cadmium and Lead concentrations in fish species of a brackish wetland / upper Bonny Estuary, Niger Delta. *Journal of Nigerian Environmental Society.*1(3): 399-405.

Chiu, H., Brittingham, J.A. and Laskin, D.L. 2002. Differential Induction of Heme Oxygenase-1 in Macrophages and Hepatocytes During Acetaminophen-Induced Hepatotoxicity in the Rat: Effects of Hemin and Biliverd in. *Toxicology and Applied Pharmacology.*181**:** 106-115

Choi, C.Y., An, K.W. and An, M.I. 2008. Molecular characterization and mRNA expression of glutathione peroxidase and glutathione S-transferase during osmotic stress in olive flounder (*Paralichthys olivaceus*). Comparative Biochemistry and Physiology. 149: 330-7.

Choi, J., and Oris, J.T. 2000. Evidence of oxidative stress in bluegill sunfish (*Lepomis macrochirus*) liver microsomes simultaneously exposed to solar ultraviolet radiation and anthracene. *Environmental Toxicology Chemistry*. 19: 1795-1799.

Chourpagar, A.R.and Kulkarani, G.K. 2014. Effect of Mercuric Chloride on Gill Structure of A Freshwater Female Crab, Barytelphusa Cunicularis (Westwood). *Journal of Global. Bioscience*. 3(2): 423–427.

Chow, C.K. 1988. *Cellular Antioxidant Defense Mechanisms*, vol. I and II. CRC Press, Boca Raton, FL. Accra, Ghana 26pp.

Ciardullo, S., Aureli, F., Raggi, A. and Cubadda, F. 2010. Arsenic speciation in freshwater fish: Focus on extraction and mass balance. *Talanta* 81: 213–221.

Clairborne, A., 1995. *CRC Handbook of Methods for Oxygen Radical Research*. *CRC Press*, Boca Raton. 447pp.

Cossins, A.R., In: Morris G.J. and Clarke A. 1981. *Effects of low temperatures on biological membranes*. London: *Academic Press*. 83-106pp.

Courtney, L.A. and Clements, W.H.2002. Assessing the influence of water and substratum quality on benthic macroinvertebrate communities in a metal-polluted stream: An experimental approach. *Freshwater Biology*. 47: 1766-1778.

Covacia, A., Gheorgheb, A., Voorspoelsa, S., Maervoeta, J., Redekere, E.S., Blustc, R. and Schepensa, P.2005. Polybrominated Diphenyl Ethers, Polychlorinated Biphenyls and Organochlorine Pesticides in Sediment Cores from the Western Scheldt River (Belgium): Analytical Aspects and Depth Profiles. *Environment International*. 31: 367-375.

Das, B.K. and Mukherjee, S.C. 2000. Histopathological Study of Carp (Labeo Rohita) Exposed To Hexachlorocyclohexane. *Veterinarski Arhive.* 70 (4): 169–180.

Davenport, J. 2001. Meltwater effects on intertidal Antarctic limpets. *Journal of Marine Biology Association*. 81: 643-49.

De Smet, H., De Wachter, B., Lobinski, R. and Blust, B. 2001. Dynamics of (Cd,Zn)-metallothioneins in gills, liver and kidney of common carp *Cyprinus carpio* during cadmium exposure. *Aquatic Toxicology.* 52: 269–281.

De Zwart, L.L., Venhorst, J., Groot, M., Commandeur, J.N.M., Hermanns, R.C.A., Meerman, J.H.N., Van Baar, B.L.M. and Vermeulen, N.P.E. 1997. Simultaneous determination of eight lipidperoxidation degradation products in urine of rats treated with carbon tetrachloride using gas chromatography with electroncapture detection. *Journal Chromatography*. 694, 277–288. Chemistry Series.

Deas, M. L. and Lowney, C. L. 2000. *Water Temperature Modeling Review.* California Water modeling forum. 117pp.

Denton, G.R.W., Bearden, B.G., Concepcion, L.P., Siegrist, H.G., Vann, D.T. and Wood, H.R. 2001. Contaminant Assessment of Surface Sediments from Tanapag Lagoon,Saipan, Water and Environmental Research Institute of the Western Pacific, *Technical Report* No. 93, University of Guam, Mangilao, Guam.

Denton, G.R.W., Wood, H.R., Concepcion, L.P., Siegrist, H.G., Eflin, V. S., Narcis, D. K. and Pangelinan, G. T. 1997. Analysis of In-Place Contaminants in Marine Sediments from Four Harbor Locations on Guam: A Pilot Study, Water and Environmental Research Institute of the Western Pacific, *Technical Report* No. 87, University of Guam, Mangilao, Guam.

Depledge, M.H. and Fossi, M.C. 1994. The role of biomarkers in environmental assessment. *Invertebrates Ecotoxicology.*3:161–172.

Depledge, M.H. and Rainbow, P.S. 1990. Models of regulation and accumulation of trace metals in marine invertebrates. *Comparative Biochemistry Physiology.* 96:1–7

Deshpande, J.R. 1985. Effect of Some Pollutants on the Reproduction in Crustaceans (Ph.D. Thesis*).* Dr. Babasaheb Ambedkar Marathwada University, Aurangabad

Di Giulio, R.T. and Meyer, J.N. 2008. *Reactive oxygen species and oxidative stress.* In: Di Giulio RT, Hinton DE (eds.): The Toxicology of Fishes. CRC Press, *Taylor and Francis* Group. 273–324pp.

Di Giulio, R.T., Habig, C. and Gallagher, E.P. 1993. Effects of Black Rock Harbor sediments on indexes of biotransformation, oxidative stress, and DNA integrity in channel cat-fish. *Aquatic Toxicology*. 26: 1-22

Dierssen, H.M., Smith, R.C. and Vernet, M. 2002. Glacial melt water dynamics in coastal waters west of Antarctic Peninsula. *Proceedings National Academy Science*. 99: 1790-5.

Dimowo, B.O. 2013. Assessment of Some Physico-chemical Parameters of River Ogun (Abeokuta, Ogun State, Southwestern Nigeria) in Comparison with National and International Standards.*International Journal of Aquaculture*. 3(15): 79-84

Dinarello, C. A.1984. Interleukin-1 and the Pathogenesis of The Acute-Phase Response. *New England Journal of Medicine.*311**:**1413-1418.

Dissanayake, A., Galloway, T. S. and Jones, M.B. 2008. Physiological responses of juvenile and adult shore crabs *Carcinus maenas* (Crustacea: 90 Decapoda) to pyrene exposure. *Marine Environmental Research*. 66: 445–450.

Dirk, G.B. 1993. Explanatory Mining for Gold: Contrasting Evidence from Simple and Multiple Reaction. *Resources Policy*. 36(3): 265-275.

Doherty, V.F., Ogunkuade, O.O. and Kanife, U.C. 2010. Biomarkers of oxidative stress as indicators of Environmental pollution in some selected fishes in Lagos, Nigeria. American-*Eurasian Journal of Agricultural Environmental Science*. 7(3): 359-365.

Don-Pedro, K.N., Oyewo, E.O. and Otitoloju, A.A. 2004. Trend of heavy-metals concentrations in Lagos Lagoon Ecosystem, Nigeria. *West African Journal Applied Ecology*.5: 103–114.

Dosunmu, O.O. and Ajayi, A.B. 2002. Problems and management of sawmill waste in Lagos.Proceedings of International Symposium on Environmental Pollution Control and Waste Management. Tunis (EPCOWM'). 271-278pp.

Doyotte, A.,Cossu, C., Jacquin, M.C., Babut, M. and Vasseur, P. 1997. Antioxidant enzymes, glutathione and lipid peroxidation as relevant biomarkers of experimental or field exposure in the gills and the digestive gland of the freshwater bivalve Unio tumidus. *Aquatic Toxicology*. 39:93-110.

Duarte, C.A., Giarratano, E., Amin, O.A. and Comoglio, L.I. 2011. Heavy metal concentrations and biomarkers of oxidative stress in native mussels (*Mytilus edulis chilensis*) from Beagle Channel coast (Tierra del Fuego, Argentina). *Marine Pollution Bulletin*. 62: 1895-904.

Dungan, J.E., Ichikawa, G., Stephenson, M., Crane, D.B., Mccall, J. and Regalado, K. 2005. *Monitoring of coastal contaminants using sand crabs*. *Relatório Final*, Central Coast Regional Water Quality Control Board, Califórnia. 37pp.

Dunlap, W.C., Fujisawa, A., Yamamoto, Y., Moylan, T.J. and Sidell, B.D. 2002. Notothenioid fish, krill and phytoplankton from Antarctica contain a vitamin E constituent (a-tocomonoenol) functionally associated with cold-water adaptation. *Comparative Biochemistry Physiology*. 133: 299-305.

Duranteau, J., Chandel, N.S., Kulisz, A., Shao, Z. and Schumacker, P.T. 1998. Intracellular signaling by reactive oxygen species during hypoxia in cardiomyocytes. *Journal Biological Chemistry*. 273: 11619-24.

Ecological Indicators. 1: 101-112.

Edokpayi, C.A. and Olowoporoku, A. O. 2010. The Hydrochemistry and Macro benthic Fauna Characteristics of an Urban Draining Creek.*International Journal of Biodiversity and Conservation.* 2(8): 196-203

Eickhoff, C.V.,Gobas, F.A.P.C. and Law, F.C.P. 2003. Screening pyrene metabolites in the hemolynph of dungeness crabs (*Cancer magister*) with Synchronous Fluorescence

Spectrometry: Method development and application. *Journal of Environmental Toxicology and Chemistry* 22: 59-66.

Eja, M. E., Ogri, O.R.A. and Arikpo, G.E. 2003. Bioconcentration of heavy metals in surface sediments from the Great kwa Rivers Estuary, Calabar, South Eastern Nigerian. *Journal of Nigeria Environment Society*. 2: 247-256.

Ekundayo, J.A. 1977. Environmental consequences of the pollution of the Lagos lagoon. *Bulletin Science. Association*. Nigeria 3: 290–299.

Ekundayo, J.A., Akpata, T.V.I., Ogunsanya, C.O., and Ibe, S.I. 1978. Degradation of faecal matter and associated micro-flora in the Lagos Lagoon. Journal of West Africa Science Associations.

Elia, A.C., Dorr, A.J.M., Mantilacci, L., Taticchi, M.I. and Galarini, R. 2000. Effects of mercury on glutathione and glutathione-dependent enzymes in catfish (*Ictalurus melas* R.). In: Markert B, Friese K (eds.): Trace Elements-Their Distribution and Effects in the Environment: Trace Metals in the Environment. *Elsevier Science*. 411–421.

Elia, A.C., Galarini, R., Taticchi, M.I., Dorr, A.J.M. and Mantilacci, L. 2003. Antioxidant responses and bioaccumulation in *Ictalurus melas* under mercury exposure. *Ecotoxicology and Environmental Safety.* 55: 162–167.

Ellman, G.L. 1959. Tissue sulfhydryl groups. Archives of *Biochemistry and Biophysics.* 82.

Emerit, J., Beaumont, C. and Trivin, F. 2001. Iron metabolism, free radicals, and oxidative injury. Biomedicine and Pharmacotherapy 55: 333–339.

Environmental Protection Agency. 1997. Toxicological Benchmarks for Screening Contaminants of Potential Concern for Effects on Sediment-Associated Biota, Report of the Sediment Criteria Subcommittee, *Science Advisory Board*. ES/ER/TM-95/R4.

Ercal, N., Gurer-Orhan, H.and Aykin-Burns, N. 2001. Toxic metals and oxidative stress part I: mechanisms involved in metal-induced oxidative damage. *Current Topics Medical Chemistry*. 1: 529–539.

Erondu, E.S. and Chindah, A.C. 1991. Physicochemical and plankton changes in a tidal fresh water station of the New Calabar River, South-eastern Nigeria. *Environment and Ecology.* 9(3): *561-570.*

Esteves, J.L., Commendatore, M., Nievas, M., Paletto, M.M. and Amin, O. 2006. Hydrocarbon pollution in coastal sediments of Tierra del Fuego Island, Patagonia Argentina. *Marine Pollution Bulletin*; 52: 572-97.

Ezemonye, L. and Ikpesu, T.2011. Evaluation of Sub-Lethal Effects of Endosulfan on Cortisol Secretion, Glutathione S-Transferase and Acetylcholinesterase Activities in *Clarias gariepinus*. *Food and Chemical Toxicology.*49**:** 1898-1903.

Fabris, J.G., Richardson, B.J., O'Sullivan, J.F. and Brown, F.C. 1994. Estimation of Cadmium, Lead and Mercury concentration in estuarine waters using the Mussel *Mytilus edulisplanutatus* L. *Environmental Toxicology*. 9: 183-192

Fafioye, O., Adebisi, A. and Fagade, S.2004. Toxicity of*Parkia biglobosa* and *Raphia vinifera* Extracts on *Clarias gariepinus* juveniles. *African Journal of Biotechnology.*3**:**627-630.

Fafioye, O.O., Olurin, K.B. and Sowunmi, A.A. 2005. Studies on the physico-chemical parameters of Omi water body of Ago-Iwoye, Nigeria, *African Journal of Biotechnology*. 4 (9):1022-1024.

FAO. 1991. Analyses of metals and organochlorinesin fish. African fisheries and the Environment FAO Regional Office, Accra, RAFR/91/02.

FAO/SIDA. 1983. Manual of Methods in AquaEffects of seagrass habitat fragmentation on juvenile blue crab survival and abundance. 271(1): 75-98

FAO/SIDA. 2003. Manual of Methods in Aquatic Environmental Research, Part 9. Analyses of Metals and Organo-chlorines in Fish. FAO Fisheries *Technical Paper*. 212pp.

Farag, A.M., May, T., Marty, G.D., Easton, M., Harper, D.D., Little, E.E. and Cleveland, L. 2006. The effect of chronic chromium exposure on the health of Chinook salmon (*Oncorhynchus tshawytscha*). *Aquatic Toxicology.*76: 246–257

Farombi, E.O., Adelowo, O.A. and Ajimoo, Y.R. 2007.Biomarker of oxidative stress in heavy metals as indicator of pollution in Africa catfish(*C. gariepinus*) from Ogun river. Nigeria *International Journal of Environment Research and Public Health*. 4(2):158-165.

Federal Environmental Protection Agency. 1991. Guidelines and Standards for Environmental Pollution Control in Nigeria. 1-238

Federal Environmental Protection Agency. 1992. Amendment of Decree No 59, Laws of the Federation of Nigeria.

Federal Office for the Environment (FOEN).2011. Indicator Water temperature of surface waters, Department of the Environment, Transport, Energy and Communications, www.bafu.admin

Fergusson, J.E.1990. *The Heavy Elements: Chemistry, Environmental Impact and Health Effects*, Pergamon Press, Oxford, England.

Figueiredo-Fernandes, A., Ferreira-Cardoso, J.V., Garcia-Santos, S., Monteiro, S.M., Carrola, J., Matos, P. and Fontaínhas-Fernandes, A.2007. Histopathological Changes in Liver and Gill Epithelium Of Nile Tilapia, Oreochromis Niloticus, Exposed To Waterborne Copper. *Pesquisa Veterinária Brasileira.*27**:**103- 109.

Filho, D.W., Tribess, T., Gaspari, C., Claudio, F.D, Torres, M.A. and Magalhaes, A.R.M. 2001. Seasonal changes in antioxidant defenses of the digestive gland of the brown mussel *Perna perna*. *Aquaculture.* 203: 149-158.

Finkelman, R.B.2005. 'Sources and Health Effects of Metals and Trace Elements in our Environment: An Overview', in Moore, T.A., Black, A., Centeno, J.A., Harding J.S. and Trumm D.A., (ed.), Metal Contaminants in New Zealand, *Resolution Press*, Christchurch, New Zealand, pp. 25-46.

Firat, O., Cogun, H.Y., Aslanyavrusu, S. and Kargin, F. 2009. Antioxidant responses and metal accumulation in tissues of Nile tilapia Oreochromis niloticus under Zn, Cd and Zn+Cd exposures. *Journal of Applied Toxicology.* 29: 295–301.

Fitzgerald, W.F., Lamborg, C.H. and Heinrich, K.K.T.2003. 'Geochemistry of Mercury in the Environment', in H. D. Holland and K. K. Turekian (ed.), *Treatise on Geochemistry Environmental Geochemistry*. 9: 107-148.

Fitzpatrick. P.J., O'Halloran, J., Sheehan, D. and Walsh, A.R. 1997. Assessment of a glutathione *S* - transferase and related proteins in the gill and digestive gland of *Mytilus edulis* (L.), as potential organic pollution biomarkers. *Biomarkers*. 2: 51-56.

Fridovich, I. 1986. Superoxide dismutases. *Advanced Enzymology*. 58: 61- 97.

Fung, C.N., Lam, J.C.W. Zheng, G.J, Connell, D.W., Monirith, I.and Tanabe, S. 2004. Mussel-based monitoring of trace metal and organic contaminants along the east coast of China using *Perna viridis* and *Mytilus edulis*, *Environmental Pollution.* 127:203–216.

Gabryelak, T. and Klekot J. 1985. The effect of paraquat on the peroxide metabolism enzymes in erythrocytes of freswater fish species.*Comparative Biochemistry and Physiology*. 81: 415- 418.

Gao, K.S., Li, P., Walanabe, T. and Helbling, E.W. 2008. Combined effects of ultraviolet radiation and temperature on morphology, photosynthesis, and DNA of *Arthrospira (Spirulina) platensis*(Cyanophyta). *Journal of Phycology*. 44: 777-86.

Gao, K.S., Wu, Y.P., Li, G., Wu, H.Y., Villafañe, V.E. and Helbling, E.W. 2007. Solar UV radiation drives CO2 fixation in marine phytoplankton: a double-edged sword. *Plant Physiology*. 144: 54-9.

Garcia-Medina, S., Razo-Estrada, A.C., Gomez-Olivan, L.M., Amaya-Chavez, A., Madrigal-Bujaidar, E. and Galar-Martinez, M. 2010. Aluminium-induced oxidative stress in lymphocytes of common carp (*Cypri`nus carpio*). *Fish Physiology and Biochemistry.* 36: 875–882.

Geret, F., Jouan, A., Turpin, V., Bebianno, M.J. and Cosson, R.P. 2002. Influence of metal exposure on metallothionein synthesis and lipid peroxidation in two bivalve mollusks: the oyster (Crassostrea gigas) and the mussel (Mytilus edulis). *Aquatic Living Resources.*15: 61–66.

Geret, F., Serafim, A., Barreira, L.and Bebianno, M.J. 2002.Effect of cadmium on antioxidant enzyme activities and lipid peroxidation in the gills of the clam *Ruditapes decussatus*. *Biomarkers.*7: 242-256.

Ghedira, J., Jebali J., Bouraoui, Z., Banni, M., Guerbej, H. and Bousetta, H. 2010. Metallothionein and metal levels in liver, gills and kidney of *Sparus aurata* exposed to sublethal doses of cadmium and copper. *Fish Physiology and Biochemistry*36: 101–107.

Giarratano, E., Duarte, C.A. and Amin, O.A. 2010. Biomarkers and heavy metal bioaccumulation in mussels transplanted to coastal waters of the Beagle Channel. *Ecotoxicology Environmental Safety*. 73: 270-79.

Giarratano, E., Gil, M.N. and Malanga, G. 2013. Assessment of antioxidant responses and trace metal accumulation by digestive gland of ribbed mussel *Aulacomya atra atra* from Northern Patagonia.*Ecotoxicology Environmental Safety*. 92: 39-50.

Giordani, T., Natali, L., Maserti, B.E., Taddei, S. and Cavallini, A. 2000. Characterization and expression of DNA sequences encoding putative type-II metallothioneins in the seagrass *Posidonia Oceanica*. *Plant Physiology*. 123: 1571-81.

Gómez-Mendikute, A. and Cajaraville, M.P. 2003. Comparative effects of cadmium, copper, paraquat and benzo[a]pyrene on the actin cytoskeleton and production of reactive oxygen species (ROS) in mussel haemocytes. *Toxicology in Vitro*. 17: 539-46.

Gonç alves, R.J., Villafañe, V.E. and Helbling, E.W. 2002. Photorepair activity and protective compounds in two freshwater zooplankton species (*Daphnia menucoensis* and

Metacyclops mendocinus) from Patagonia, Argentina. *Photochemistry and Photobiology Science*. 1: 996-1000.

González, P.M. and Puntarulo, S. 2011. Iron and nitrosative metabolism in the Antarctic mollusk *Laternula elliptica*. *Comparative Biochemistry and Physiology*. 153: 243-50.

González, P.M., Abele, D. and Puntarulo, S. 2010. Exposure to excess dissolved iron in vivo affects oxidative status in the bivalve *Mya arenaria*. *Comparative Biochemistry and Physiology*. 152: 167-74

Grad, G., Burnett, B.J. and Williamson, C.E. 2003. UV damage and photoreactivation: timing and age are everything. *Photochemistry and Photobiology*. 78: 225-7.

Graham, L.E. and Wilcox LW. 2000.Algae. New York: Prentice Hall.

Gravato, C., Teles, M., Oliveira, M. and Santos, M.A. 2006. Oxidative stress, liver biotransformation and genotoxic effects induced by copper in *Anguilla anguilla* L. – the influence of pre-exposure to β-naphthoflavone. *Chemosphere.* 65: 1821–1830.

Grzymski, J., Orrico, C. and Schofield, O.M. 2001. Monochromatic ultraviolet light induced damage to Photosystem II efficiency and carbonfixation in the marine diatom *Thalassiosira pseudonana* (3H*). Photosynthensis Resources*. 68: 181-92.

Gül, Ş., Belge-Kurutaş, E., Yıldız, E., Şahan, A. and Doran, F.2004. Pollution 641 Correlated Modifications Of Liver Antioxidant Systems And Histopathology Of Fish (Cyprinidae) Living In Seyhan Dam Lake, Turkey. *Environment International.*30**:**605-609.

Gutteridge, J.M. 1985. Inhibition of the Fenton reaction by the protein ceruloplasmin and other copper complexes. Assessment of ferrioxidase and radical scavenging activities. *Chemico-Biological Interactions.* 56: 113–120

Hageman, J.J., Bast, A., Vermeulen, N.P.E. 1992. Monitoring of oxidative free radical damage in vivo: analytical aspects. *Chemistry and Biology Interaction.* 82: 243-293

Hai, D.Q., Varga, S.I.and Matkovics, B. 1997. Organophosphate effects on antioxidant system of carp (Cyprinus carpio) and catfish(Ictahrus nebulosus). *Comparative Biochemistry and Physiology*. 117: 83–88.

Halliwell, B.and Gutteridge, J.M.C. 1999. *Free Radicals in Biology and Medicine.Third Edition*. Oxford University Press, Oxford, UK.

Halliwell, B.and Gutteridge, J.M.C. 2007. *Free Radicals in Biology and Medicine*. 4th ed. London: Oxford: Oxford University Press.

Halliwell, B. and Whiteman, M. 2004. Measuring Reactive Species and Oxidative Damage in Vivo and in Cell Culture: How Should You Do It and What Do the Results Mean? *British Journal of Pharmacology.*142**:** 231-255.

Hamer, B., Jaki, and Pavii-Hamer, D. 2008. Effect of hypo-osmotic stress by low salinity acclimation of Mediterranean mussels *Mytilus galloprovincialis* on biological parameters used for pollution assessment. *Aquatic Toxicology*. 89: 137-51.

Hamer, D.H. 1986. Metallothionein. Annual Review *Biochemistry.* 55: 913–951.

Han, Q., Fang, J., Ding, H., Johnson, J.K., Christensen, B.M. and Li, J. 2002. Identification of Drosophila melanogaster yellow-f and yellow-f2 proteins as dopachrome-conversion enzymes. Biochem. J. 368(1): 333--340.

Harkabusova, V., Macharackova, B., Celechovska, O. and Vitoulova, E. 2009. Determination of arsenic in the rainbow trout muscle and rice samples. Czech *Journal of Food Sciences.* 27: 404–406.

Harold, R. 1997. Environmental issues and the interaction of aquaculture with other compelling resource users. *Proceedings of the Huntsmas Marine Science Centre Symposium.Aquaculture Association of Canada Special Publication*.2(1) 13-22.

Harriet, P. 2001. *Activity: Blue crab dissection, Project Oceanography*: Unit Five COAST/ Blue crabs; p75.

Hasselberg, L., Meier, S. and Svardal, A. 2004. Effects of alkylphenols on redox status in first spawning Atlantic cod (*Gadus morhua*). *Aquatic Toxicology*. 9: 95-105.

Hazel, J.R. and Williams, E.E. 1990. The role of alterations in membrane lipid composition in enabling physiological adaptations of marine organisms to their physical envirionment. Program Lipid Reservatives. 29: 167-227.

Heather, R. C. 2003. Enzymatic Response of *Callinectes sapidus* and *Geukensia demissa* as Biomarkers for Pesticide Exposure. *Master Thesis,* Department of Chemistry; University of North Carolina.

Helbling, E.W., Villafañe, V.E., Ferrario, M.E. and Hansen, O.H. 1992. Impact of natural ultraviolet radiation on rates of photosynthesis and on specific marine phytoplankton species. *Marine Ecology Program* Services. 80: 89-100.

Heraud, P. and Beardall, J. 2000. Changes in chlorophyll fluorescence during exposure of *Dunaliella tertiolecta* to UV radiation indicate a dynamic interaction between damage and repair processes. *Photosynthensis Resources*. 63: 123-34.

Hermenean, A., Damache, G., Albu, P., Ardelean, A., Ardelean, G., Ardelean, D. P., Horge, M., Nagy, T., Braun, M. and Zsuga M. 2015. Histopatological Alterations And Oxidative Stress In Liver And Kidney Of 652 Leuciscus Cephalus Following Exposure To Heavy Metals In The Tur River, North Western Romania.*Ecotoxicology And Environmental Safety.*119**:**198-205.

Hermes-Lima, M.and Zenteno-Savín, T. 2002. Animal response to drastic changes in oxygen availability and physiological oxidative stress. *Comparative Biochemistry and Physiology.* 133: 537-556.

Hernando, M., Malanga, G., Puntarulo, S. and Ferreyra, G. 2011. Non-enzymatic antioxidant photoprotection against potential UVBR-induced damage in an Antarctic diatom (*Thalassiosira* sp.).*Latin American Journal of Aquatic Resources.* 39(3): 397-408.

Hill, W.B. and Webb, J.E. 1958. The ecology of Lagos lagoon II. The topography andphysical features of Lagos Harbour and Lagos lagoon. *Philosophical Transactions of the Royal Society of London*. 241: 307-419.

Hinton, D.E. 1994. Cells, Cellular Responses and their Markers on Chronic Toxicity of fishes. In: Aquatic Toxicology: *Molecular, Biochemical and Cellular Perspectives*. Malins D.C.,and Ostrander G.K.(Eds). Lewis Publishers, Boac Rato, USA. pp: 207-239.

Hinton, D.E., Baumann, G.R.., Gardner, W.E., Hawkins, and Hendricks, J.D. 1992. Histopathologic Biomarkers. In: *Biomarker, physiological and Histological Markers of Anthropogenic Stress*, Huggett,R.J.,Kimerie,R.A., Mehrie P.M. and Bergman H.L(Eds). Lewis Publishers, Boac Rato, USA. pp155-210.

Houghton, J.T., Ding, Y. and Griggs, D.J. 2001. *Climate change: the scientific basis.* UK: Cambridge University Press 2001.

Houserova, P., Kuban, V., Spurny, P. and Habarta, P. 2006. Determination of total mercury and mercury species in fish and aquatic ecosystems of Moravian rivers. *Veterinarni Medicina* 51: 101–110.

Hovel, K.A. and Lipcius Romuald, N. 2001. Habitat Fragmentation In A Seagrass Landscape: Patch Size and Complexity Control Blue Crab Survival. 82:1814–1829.1890/0012-9658.Ecology in environmental risk assessment: a Review. *Environmental Toxicology and Pharmacology.* 13.

Hovel, K. A. and Lipcius,R. N. 2002. Show more *Journalof Experimental Marine Biology* and ecology. 0981(02)00043-6.

Javed, M. 2005. Heavy metal contamination of fresh water fish and bed sediments in the river rari stretch and related tributaries. *Pakistan Journal of Biological Science*. 8:1337-1341.

Jayasree, Sr, L., Janakiram, P. and Madhavi, R. 2001. Epibionts And Parasites Of Macrobrachium Rosenbergii And Metapenaeus Dobsoni From Gosthani Estuary. *Journal Of Natural History*.35**:** 157-167.

Jerome, F. C. and Chukwuka, A. V. 2016. Metal residues in flesh of edible blue crab, Callinectes amnicola, from a667 tropical coastal lagoon: Health implications. *Human and Ecological Risk Assessment: An InternationalJournal*.22, 1708-1725.

Jebali, J., Banni, M., Guerbej, H., Almeida, E.A., Bannaoui, A. and Boussetta, H. 2006. Effects of malathion and cadmium on acetylcholine esterase activity and metallothionein levels in the fish *Seriola dumerilli*. *Fish Physiology and Biochemistry.* 32: 93–98

Jena, S.D., Behera, M., Dandapat, J. and Mohanty, N. 2009. Non-enzymatic antioxidant status and modulation of lipid peroxidation in the muscles of *Labeo rohita* by sub-lethal exposure of $CuSO_4$. *Veterinary Research Communications.* 33: 421–429.

Jia, X., Zhang, H. and Liu, X. 2011. Low levels of cadmium exposure induce DNA damage and oxidative stress in the liver of Oujiang colored common carp *Cyprinus carpio* var. *color*. *Fish Physiology and Biochemistry.* 37: 97–103.

Jiang, H.B. and Qiu, B.S. 2011. Inhibition of photosynthesis by UV-B exposure and its repair in the bloom-forming cyanobacterium *Microcystis aeruginosa*. *Journal of Applied Phycology*. 23: 691-96.

Jollow, D.J., Michell, J.R. Zampaglione, N. and Gillete, J.R. 1974. Bromobenzene induced live necrosis: Protective role of GSH and evidence of 3, 4-bromobenzene oxide as the hepatotoxic metabolite. *Pharmacology*. 11: 151-169.

Kadiri, M.O. 1999. Phytoplankton distribution in some coastal waters of Nigeria. *Nigerian Journal of Botany*. 12(1): 51-62.

Kakulu, S.E. and Osibanjo, O. 1988. Trace heavy metal pollutional status in sediments of the Niger Delta Area of Nigeria. *Journal Chemical Society of Nigeria*. 13:9-11.

Kamaldeen, O. S. and Wahab, B. 2011. The Impact of Excreta Disposal into Lagos Lagoon on The Lagoon Ecosystem at Iddo Discharge Point in Apapa Local Government Area of

Lagos State Nigeria. *Journal of "Sustainable Development and Environmental Protection.* 1: 1.

Kandemir, S., Dogru, M.I, Orun, I., Dogru, A., Altas, L., Erdogan, K., Orun, G. and Polat, N., 2010. Determination of heavy metal levels, oxidative status, biochemical and hematological parameters in *Cyprinus carpio* L., 1785 from Bafra (Samsun) fish lakes. *Journal of Animal and Veterinary Advances.* 9: 617–622.

Kappus, H.1987. Oxidative stress in chemical toxicity. *Archeology Toxicology*. 60:144 –149.

Kelly, S. A., Havrilla, M., Brady, T.C., Abramo, K.H. and Levin E.D. 1998. "Oxidative Stress in Toxicology: Established Mammalian and Emerging Piscine Model Systems." *Environmental Health Perspectives.* 106: 375–384.

Khessiba, A., M. Roméo, and P. Aïssa, 2005. Effects of some environmental parameters on catalase activity measured in the mussel (*Mytilus galloprovincialis*) exposed to lindane *Environmental Pollution.* 133: 275-281.

Kidder, G. 2002. Using Waste Products in Forage Production. *The Florida Forage Handbook*, an electronic publication of the Agronomy Department,University of Florida, Gainesville, 32611-0290.

Kinnberg, K., Korsgarrd, B. and Bjerregaard, P. 2000. Concentration Dependent Effects Of Nonylphenol On Testis Structure In Adult Platy Fish, Xiphophorus Maculates. *Marine Environmental Resources.* 50: 169–173.

Kirchin, M.A., Moore, M.N., Dean, R.T. and Winston, G.W. 1992. The role of oxyradicals in intracellular proteolysis and toxicity in mussels. *Marine Environmental Resources*. 34: 315-20.

Kirchin, M.A., Wiseman, A. and Livingstone, D.R.1992. Seasonal and sex variation in themixed function oxygenase system of digestive gland microsomes of the common mussel,*Mytilus edulis* L. *Comparative Biochemistry and Physiology.* 101: 81-91

Koenig, S., Svage, C. and Kim, J.P. 2008. Non-destructive assessment of polycyclioc aromatic hydrocarbon (PAH) exposure by fluorimetric analysis of crab urine. *Marine Pollution Bulletin.* 56p.

Kohen, R. and A. Nyska, 2002. Oxidation of biological systems: oxidative stress phenomena, antioxidants, redox reactions and methods for their quantification. *Toxicology Pathology,* 30(6): 620-650.

Kono, Y. and Fridovich, I. 1982. Superoxide radical inhibits catalase. *Journal of Biological Chemistry*. 257: 5751–5754.

Kori-Siakpere, O. and Ubegu, E.O. 2008. Sublethal haematological effects of zinc on the fresh water fish. *Heteroclarias sp*. (Osteichthyes: Clariidae).*African. Journal. Biotechnol*. 7:2068-2073.

Kovarova, J., Celechovska, O., Kizek, R., Adam, V., Harustiakova, D. and Svobodova, Z. 2009. Effect of metals, with special attention of Cd, content of the Svitava and Svratka rivers on levels of thiol compounds in fish liver and their use as biochemical markers.*Neuroendo-crinology Letters* 30: 167–169.

Krishnamoorthy, P., and Subramanian, P. 1996. Effect of Sub Lethal Doses of Copper on The Hepatopancreas of The Freshwater Prawn, Macrobrachium Lamerrei Lamerrei (H.M. Edwards). *Geobios.* 23(1): 16–18.

Krumgalz, B. S., Fainshtein, G. and Cohen, A.1992. 'Grain size effect on anthropogenic trace metals and organic matter distribution in marine sediments', *The Science of the Total Environment, Elsevier Science Publishers*.116: 15 - 30.

Kubrak, O.I., Lushchak, O.V., Lushchak, J.V., Torous, I.M., Storey, J.M., Storey, K.B. and Lushchak, V.I. 2010. Chromium effects on free radical processes in gold fish tissues: Comparison of Cr(III) and Cr(VI) exposures on oxidative stress markers, glutathione status and antioxidant enzymes. *Comparative Biochemistry and Physiology.* 152: 360–370.

Kumar, M., Kumari, P., Gupta, V., Reddy, C.R.K. and Jha, B. 2010. Biochemical responses of red alga *Gracilaria corticata* (Gracilariales, Rhodophyta) to salinity induced oxidative stress. *Journalof Experimental Marine Biological Ecology*. 391: 27-34.

Kuykendall, J..R, Miller, K.L., Mellinger, K.N. and Cain, A.V. 2006. Waterborne and dietary hexavalent chromium exposure causes DNA-protein crosslink (DPX) formation in erythrocytes of largemouth bass (*Micropterus salmoides*). *Aquatic Toxicology.* 78: 27–31.

Ladigbolu, I.A., Balogun, K.J. and Shelle, R.O. 2011. Hydrochemistry and levels of some heavy metals in samples of Ibeshe, Lagos Lagoon Complex, *Nigeria. Journal of American Science*. 7(1).

Laffon, B., Rábade, T., Pásaro, E. and Méndez, J. 2006. Monitoring of the impact of Prestige oil spill on *Mytilus galloprovincialis* from Galicia coast. *Environmental Int*ernational. 32: 342-8.

Lalyeye and Morean, 2005. Resources and constraints of West African coast at waters for fish production. *World Fish Centre Conference proceedings*. 983: 2346 – 32.

Lam, P.K.S.and Gray, J.S. 2003. The use of biomarkers in environmental monitoring programs, *Marine Pollution Bulletin.* 46: 182–186.

Larose, C., Canuel, R., Luccote, M., Di Giulio, R. 2008. Toxicological effects of methylmercury on walleye (*Sander vitreus*) and perch (*Perca flavescens*) from lakes of the boreal forest. *Comparative Biochemistry and Physiology*. 147: 139–149.

Lattuca, M.E., Pérez, A.F., Giarratano, E. and Malanga, G. 2013. Baseline levels of biomarkers of oxidative damage in *Odontesthes nigricans* (Pisces, Atherinopsidae) from two coastal areas of the Beagle Channel, *Argentina. Revise Chil Histology Nat*ure. 86: 453-64.

Lau, S., Mohammed, M., Tanchi, YA. and Su'ut, S. 1998. Accumulation of heavy metals in freshwater mollusks. Science of the *Total Environment.* 214: 113-121.

Lau, S.M., and Sabtutah, S. 1996. Heavy metals in sediment as a tracer for sources of pollution in Sg- Saawak. *Malaysian Journal of Analytical Science 2(2)*: 365-371.

Lawal-Are, A. and Kusemiju, K.2000. Size Composition, Growth Pattern and Feeding Habits of The Blue Crab, *Callinectes amnicola* (De Rocheburne) in the Badagry Lagoon, Nigeria. *Journal Science Resources Development.*5.

Lawal-Are, A. O. and Kusemiju, K. 2010. Effect of salinity on survival and growth of blue crab, Callinectes amnicolafrom Lagos Lagoon, Nigeria.*Journal of Environmental Biology.* 31(4): 461-464.

Lenartova, V., Holovska, K., Pedrajas, J.R., Martinez-L ara, E., Peinado, J. and Lopez-Barea, J. 1970. Antioxidant and detoxifying fish enzymes as biomarkers of river pollution. *Biomarkers*. 2: 247-252.

Lesser, M.P. 2006. Oxidative stress in marine environments. Biochem Physiol Ecol Annu Rev Physiol; 68: 253-78. Lukáč, N., P. Massányi, R. Toman, and J. Trandžík. 2003. "Effect of Cadmium on Spermatozoa Motility." *Savremena Poljoprivreda.* 3(4): 215–217.

Lesser, M.P., Kruse, V.A. and Barry, T.M. 2003. Exposure to ultraviolet radiation causes apoptosis in developing sea urchin embryos. *Journal Experimental Biology*. 206: 4097-103.

Letendre, J., Leboulenger, F. and Durand, F. 2012. "Oxidative Challenge and Redox Sensing." In Mollusks: Effects of Natural and Anthropic Stressors: In Oxidative Stress in Vertebrates and Invertebrates: Molecular Aspects on Cell Signaling, edited by T. Farooqui and A. A. Farooqui, 398. Hoboken, NJ: *John Wiley and Sons*.

Li, H.C., Zhou, Q., Wu, Y., Fu, J., Wang, T. and Jiang, G. 2009. Effects of waterborne nano-iron on medaka (*Oryzias latipes*): Antioxidant enzymatic activity, lipid peroxidation and histopathology. *Ecotoxicology and Environmental Safety.*72: 3684–3692

Li, Y., Gao, K., Villafañe, V.E. and Helbling, E.W. 2012. Ocean acidification mediates photosynthetic response to UV radiation and temperature increase in the diatom *Phaeodactylum tricornutum*. Biogeoscience Discusion. 9: 7197-226.

Liping, W. and Binghui, Z. 2008. Toxic effects of fluoranthene and copper on marine diatom *Phaeodactylum tricornutum*. *Journalof Environmental Science.* 20: 1363-72.

Liu, J., Qu, W. and Kadiiska, M.B. 2004. Role of oxidative stress in cadmium toxicity and carcinogenesis, *Toxicology and Applied Pharmacology.* 238: 209-214.

Liu, Y., Wang, W.N., Wang, A.L., Wang, J.M. and Sun, R.Y. 2007. Effects of dietary vitamin E supplementation on antioxidant enzyme activities in *Litopenaeus vannamei*(Boone, 1931) exposed to acute salinity changes.*Aquaculture.* 265: 351-8.

Livingstone, D., Lips, F., Martnez, P. and Pipe, R.1992. Antioxidant enzymes in the digestive gland of the common mussel (*Mytilus edulis* L.). *Marine Biology.* 112: 265- 276,

Livingstone, D.R. 1991. Induction of enzymes as a mechanism for the seasonal control of metabolism in marine invertebrates:glucose-6-phosphate dehydrogenases from the mantle and hepatopancreas of the common mussel *Mytilus edulis* (L). *Comparative Biochemistry and Physiology*. 69: 147-156.

Livingstone, D.R. 1993. Biotechnology and pollution monitoring - use of molecular bio-markers in the aquatic environment. *Journal of Chemistry, Technology and Biotechnol*ogy. 57: 195-211.

Livingstone, D.R. 2001. Contaminant-stimulated reactive oxygen species production and oxidative damage in aquatic organisms.*Marine Pollution Bulletin.* 42: 656-666

Livingstone, D.R. 2003. "Oxidative Stress in Aquatic Organism in Relation to Pollution and Agriculture." *Revue de Medecine Veterinaire*. 154: 427–430.

Loguercio, C., Piscopo, P., Guerriero, C., Girolamo, V. D., Disalvo, D. and Del Vecchio Blanco, C. 1996. Effect of Alcohol Abuse and Glutathione Administration on the Circulating

Levels of Glutathione and on Antipyrine Metabolism in Patients with Alcoholic Liver Cirrhosis. *Scandinavian Journal of Clinical and Laboratory Investigation.*56**:**441-447.

Loomb, C.A.M. 2001.*Muddy Sedimentation in a Sheltered Estuarine Marine*, Westpark Marina, Auckland, New Zealand, Thesis, The University of Waikato. 357-369pp.

Lopez, E., Arce, C., Oset-Gasque, M.J., Canadas, S.and Gonzalez, M.P. 2006. Cadmium induces reactive oxygen species generation and lipid peroxidation in cortical neurons in culture. *Free Radical, Biology and Medicine.* 40: 940–951.

Loschen, G., Azzi, A. and Flohe,´ L. 1973. Mitochondrial H_2O_2 formation: relationship with energy conservation. *FEBS Letters*. 33: 84-8.

Lowe C. G.ToppingD. T. CartamilD. P.PapastamatiouY. P. 2003. Movement patterns, home range, and habitat utilization of adult kelp bass *Paralabrax clathratus* in a temperate no-take marine reserve*Marine Ecology Progress Series.* 256 :206-216.

Lowry, O.H., Rosenbrough, N.M. Farr A.L. and Randall, J. 1951.Protein measurement with folin phenolreagent. *Journal of Biological Chemistry.*193: 265-275

Lu, I-F., Ming-Shiuan, S. and Lee, T.M. 2006. Salinity stress and hydrogen peroxide regulation of antioxidant defense system in *Ulva fasciata*. *Marine Biology.* 150: 1-15.

Lukáč, N., P. Massányi, R. Toman, and J. Trandžík. 2003. “Effect of Cadmium on Spermatozoa Motility.” *Savremena Poljoprivreda.* 3(4): 215–217

Luoma, S.N. 2000, 'Processes Affecting Metal Concentrations in Estuarine and Coastal Marine Sediments', in R. W. Furness & P. S. Rainbow (ed.), Heavy Metals in the Marine Environment, CRC Press, Inc, Florida, United States of America, pp. 51-66.*Environments, Elsevier Applied Science*. Publishers.

Luoma, S.N. and Rainbow, P.S. 2008.*Sources and cycles of trace metals. In: Metal Contamination in Aquatic Environments:* Science and Lateral Management. *Cambridge University Press*, Cambridge. 47–66pp.

Lushchak, V.I. 2011. Environmentally induced oxidative stress in aquatic animals. *Aquatic Toxicology.* 1: 13–30.

Lushchak, V.I., Lushchak, L.P.A. and Mota, M. 2001. Oxidative stress and antioxidant defenses in goldfish *Carassius auratus*during anoxia and re-oxygenation. *American Journal of Physiology*. 280: 100-107.

Luza, S.C. and Speisky, H.C. 1996. Liver copper storage and transport during development: Implications for cytotoxicity. *American Journal of Clinical Nutrition*. 63: 812–820

Magwere, T., Y Naik .S. and Hasler, J.A. 1997. Effects of chloroquine treatment on antioxidant enzymes in rat liver and kidney. *Free Radical Biological Medicine.* 22(1-2):321-327.

Maharajan, A., Neelakandamoorthy, N., Kumarasamy, P. 2012a. Impact of Profenofos on Oxygen Consumption and Gill Histopathology of the Fresh Water Crab, *Paratelphusa jacquemontii* (Rathbun). *Resources Journalof Toxicology.* 2(2): 46–55.

Maharajan, A., Rajalakshmi, S., Vijayakumaran, M. and Kumarasamy, P. 2012b. Sublethal Effect of Copper Toxicity Against Histopathological Changes in the Spiny Lobster, Panulirus Homarus (Linnaeus, 1758). *Biology Trace Element. Resources*. 145: 201–210.

Maharajan, A., Usha, R., Paruruckmani, P.S., Vijaykumar, B.S., Ganapiriya, V. and Kumarasamy, P. 2013. Sublethal Effect Of Profenofos On Oxygen Consumption And Gill Histopathology of The Indian Major Carp, Catla Catla (Hamilton) *International Journal of Pure and Applied Zool*ogy. 1(1): 196–204.

Maheswaran, R., Devapaul, A., Muralidharan, S., Velmurugan, B., Ignacimuthu, S. 2008. Haematological study of fresh water fish, *Clarias batrachus* (L) exposed to mercuric chloride. *International Journal Integrated Biology.* 2:19-54.

Maiti, A.K., Saha, N.K. and Paul, G. 2010. Effect of lead on oxidative stress, Na+K+ATPase activity and mitochondrial electron transport chain activity of the brain of *Clarias batrachus* L. *Bulletin of Environmental Contamination and Toxicology.* 84: 672–676.

Manney. G.L., Santee, M.L. and Rex, M. 2011.Unprecedented Arctic ozone loss in 2011. *Nature.* 478: 469-75.

Manosathiyadevan, M., Selvisabhanayakam, V. and Divya, H. 2012. Morphological Alterations And Biochemical Contents Of The Testis Of Adult Male Freshwater Prawn Macrobrachium Malcolmsonii. *Indian Jounal of Fundamental Applied Life Sciences*. 2(4): 104–113.

Martinez, C., Nagae, M., Zaia, C. and Zaia, D. 2004. Acute Morphological And Physiological Effects Of Lead In The Neotropical Fish Prochilodus Lineatus. *Brazilian Journal of Biology.*64**:**797-807.

Mason, A.Z. and Jenkins, K.D.1995. *Metal detoxification in aquatic organisms*. In: Tessier A and Turner A (eds), Metal Speciation and Bioavailability in Aquatic Systems. 479–608pp. *John Wiley*, Chichester, UK.

Massányi, P. and Uhrín, V.1996. "Histological Changes in the Ovaries of Rabbits after an Administration of Cadmium."*Journal of Environmental Science and Health.*32 (5):1459–1446.

Massányi, P., Toman, R. and Trandžík J.2004. "Concentration of Copper, Zinc, Iron, Cadmium, Lead and Nickel in Bull, Ram, Boar, Stallion and Fox Semen." *Trace Elements and Electrolytes.* 21(1): 45–49.

Massányi, P., V. Uhrín, R. Toman, J. Pivko, N. Lukáã, Zs. Forgács, Z. Somosy, M. Fabis, and Danko, J.2005. "Ultrastructural Changes of Ovaries in Rabbits Following Cadmium Administration." *Acta Veterinaria Brno.* 74: 29–35.

Massányi, P., Z. Kiss, R. Toman, and L. Bardos. 2002. "Effect of Acute Cadmium Exposure on Testicular Tissue and Testicular Retenoid and Beta-carotene Content." *Magyar Allatorvosok Lapja*. 124 (11): 688–692.

Mc Gaw, I.J. and Naylor, E. 1992b.Salinity preference of shore crab *Carcinus maenas* in relation to colouration during intermoult and to prior acclimation. *Journal of experimental marine Biology and Ecology.* 155:145-159.

Mc Gaw, I.J., Kaiser, M.J., Naylor, E. and Hughes, R.N. 1992a. Intraspecific morphological variation related to moult-cycle in colour forms of the shore crabs *Carcinus maenas. Journal of Zoology.* 228: 351-359.

McCarthy, J.F., Halbrook, R.S. and Shugart, L.R. 1991. Conceptual strategy for design, implementation, and validation of a biomarkerbased biomonitoring capability. Environmental Sciences Division.*Annual Review of Biochemistry.* 52: 711-760.

McCord, J. M. and Fridovish, I. 1989. Superoxide dismuthase. An enzymatic function for erythrocuprien (hemocuprein). *Journal of Biological Chemistry.* 244:4039-4045.

McCord, J.M, andFridovich, I. 1969. Superoxide dismutase: an enzymatic function for erythrocuprein (hemocuprein). *Journal of Biological Chemistry.* 244: 6049-6055.

McDonald, D.G. and Wood, C.M. 1993. Branchial mechanisms of acclimation to metals in freshwater fish. In: Rankin J.C. and Jensen F.B. (eds), *Fish Ecophysiology*, pp297–321. Chapman & Hall, London, UK.

Meister, A. and Anderson, M.E.1983. Glutathione, metallothionein synthesis and lipid peroxidation in two bivalve mollusks: the oyster (*Crassostreagigas*) and the mussel (*Mytilus edulis*). *Aquatic Living Resources.*15: 61-66.

Mela, M., Randi, M., Ventura, D., Carvalho, C., Pelletier, E. and Ribeiro, C. O. 2007. Effects of Dietary Methylmercury on Liver and Kidney Histology in the Neotropical Fish *Hoplias malabaricus*. *Ecotoxicology and Environmental Safety.*68**:**426-435.

Melville, F. 2005.Mangrove algae in the assessment of estuarine pollution. Ph.D. thesis. Department of Environmental Sciences, University of Technology, Sydney, Australia.

Melville, F., Burchett, M.D. and Pulkownik, A. 2004. Genetic variation among age-classes of the mangrove *Avicennia marina* in clean and contaminated sediments. *Marine Pollution Bulletin*. 49: 695.703.

Messick, G. A. 1998. Diseases, Parasites, and Symbionts of Blue Crabs (*Callinectes sapidus*) Dredged From 715 Chesapeake Bay. *Journal of Crustacean Biology.*18**:**533-548.

Micheli, F.andPeterson, C.H. 1999. Estuarine Vegetated Habitats as Corridors for Predator Movements. *Conservation Biology*. 13:869-881.

Mieiro, C.L., Ahmad, I., Pereira, M.E., Duarte, A.C. and Pacheco, M. 2010. Antioxidant system breakdown in brain of feral gulden grey mullet (*Liza aurata*) as an effect of mercury exposure. *Ecotoxicology.*19: 1034–1045.

Mieiro, C.L., Bervoets, L., Joosen, R., Blust, R., Duarte, A.C., Pereira, M.E. and Pacheco, M. 2011. Metallothioneins failed to reflect mercury external levels of exposure and bioaccumulation in marine fish – Considerations on tissue and species specific responses. *Chemosphere.* 85: 114–121.

Miller, G.T. 2000.*Living in the Environment: Principles, Connections and Solutions*. New York. Brooks.

Miller, L.L., Wang, F., Palace, W.P. and Hontela, A. 2007. Effects of acute and sub-chronic exposures to waterborne selenite on the physiological stress response and oxidative stress indicators in juvenile rainbow trout. *Aquatic Toxicology.* 83: 263–271

Monirith, I., Ueno, D., Takahashi, S., Nakata, H., Sudaryanto, A. and Subramanian, A. 2003. Asia-Pacific mussel watch: monitoring contamination of persistent organochlorines compounds in coastal waters of Asian countries, *Marine Pollution Bulletin.* 46: 281–300.

Monteiro, D.A., Rantin, F.T. and Kalinin, A.L. 2010. Inorganic mercury exposure: toxicological effects, oxidative stress biomarkers and bioaccumulation in the tropical freshwater fish matrinxa, *Brycon amazonicus* (Spix and Agassiz, 1829). *Ecotoxicology.* 19: 105–123.

Monteiro, S., Rocha, E., Fontaínhas-Fernandes, A. and Sousa, M. 2008. Quantitative Histopathology Of Oreochromis Niloticus Gills After Copper Exposure. *Journal of Fish Biology.*73**:**1376-1392.

Moore, J.K., Doney, S.C. and Lindsay, K. 2004. Upper ocean ecosystem dynamics and iron cycling in a global three dimensional model. *Global Biogeochemistry*. 18(4): 1-21.

Mopper, K., Kieber, D.J., In: De Mora S., Demers S., Vernet M., Eds. 2000. *The effects of UV radiation in the marine environment.* Cambridge, UK: *Cambridge University Press;* pp. 101-30.

Morales-Caselles, C., Martín-Díaz, M.L., Riba, I., Sarasquete, C. and Delvalls, T.A. 2008. Sublethal responses in caged organisms exposed to sediments affected by oil spills. *Chemosphere*. 72: 819-825.

Morillo, J. and Usero, J. 2008. Trace metal bioavailability in the waters of two different habitats in Spain: Huelva estuary and Algeciras Bay. *Ecotoxicology Environmental Saf*ety. 71(3): 851-9.

Morris, C.A., Nicolaus, B., Sampson, V., Harwood, J.L. and Kille, P. 1999. Identification and characterization of a recombinant metallothionein protein from a marine alga, (*Fucus vesiculosus*). *Biochemistry Journal*. 338: 553-60.

Mukaddes, E. 2012. Oxidative Stress and Benefits of Antioxidant Agents in Acute and Chronic Hepatitis. *Hepatitis Monthly.*160-167.

Muller, K.E., 1988. Applied regression analysis and other multivariate methods, PWS–KENT Publishing Company, USA. 2nd Edn.

Narayana, 2011.Environmental Pollution; Principles, Analysis and Control.1st Edition., Indian Binding House (UP)

National Environmental *Standards* and Regulations and Regulation Enforcement Agency (NESREA), 2015.The National *Guidelines* and *Standards* for *Water* Quality in Nigeria.

National Research Council, Committee (NRC), 1987. Issues on biological markers, *Environmental Health Perspectives.* 74: 3–9.

Navarro, A., Quiros, L., Casado, M., Faria, M., Carrasco, L., Benejam, L., Benito, J., Diez, S., Raldua, D., Barata, C., Bayona, J.M., and Pina, B. 2009. Physiological responses to mercury in feral carp populations inhabiting the low Ebro River (NE Spain), a historically contaminated site. *Aquatic Toxicology*. 93: 150–157.

Nebel, B. J. and Wright, R. T. 2002.*Environmental Science: Toward A Sustainable Future* (8th Edition). 13: 978-0130325389.

Neff, J.M. 1979. *Polycyclic Aromatic Hydrocarbons in the Aquatic Environment*. London: *Applied Science Publishers* Ltd.

Neogrohati, S. 2006. Bioaccumulation dynamic of heavy metals in *Oreochromis nilotycus* (predicted through a bioaccumulation modelconstructed based on biotic ligand model(BLM). Sri. Noegrohati Bioaccumulation Dyn. *Heavy*. 16:29-40.

Nesto, N., Romano, S., Moschino, V., Mauri, M. and Da Ros, L. 2007. Bioaccumulation and biomarker responses of trace metals and micro-organic pollutants in mussels and fish from the Lagoon ofVenice, Italy.*Marine Pollution Bulletin*. 55:169-184.

Neves, C.A., Santos, E.A. and Bainy, A.C.D. 2000. Reduced superoxide dismutase activity in *Palaemonetes argentinus* (Decapoda, Palemonidae) infected by *Probopyrus ringueleti* (Isopoda, Bopyridae). Diseases of Aquatic Organisms. 39: 155-8.

Niehaus, W. G. and Samuelsson, B. 1968. Formation of malondialdehyde from phospholipid arachidonate during microsomal lipid peroxidation. *European Journalof Biochemistry.*6:126-130.

Nielsen, T., Siigur, K., Helweg, C., Jorgensen, O., Hansen, P.E. and Kirso, U. 1997. Sorption of polycyclic aromatic compounds to humic acid as studied by high performance liquid chromatography. *Environmental Science Technology*. 31: 1102-8.

Nies, D. H. 2003. Efflux-Mediated Heavy Metal Resistance in Prokaryotes. *Fems Microbiology Reviews.*27**:** 313-339.

Nikinmaa, M. 2002. Oxygen-dependent cellular functions - why fishes and their aquatic environment are a prime choice of study. *Comparative Biochemistry Physiol*ogy. 133: 1-16.

Nilawati, J., Greenberg, B.M. and Smith, R.E.H. 1997. Influence of ultraviolet radiation on growth and photosynthesis of two cold ocean diatoms. *Journal of Phycology*. 33: 215-24.

Nishida, Y. 2011. The chemical process of oxidative stress by copper (II) and iron (III) ions in several neurodegenerative disorders. *Monatshefte fur Chemistry* 142: 375–384.

Nkono, N.A., Asubiojo, O.L. and Ogunsua O. 1999. Levels, sources and speciation of trace elements in the surface waters of the Lagos Lagoon.*International Journal of American Science.*

Nkwoji, J. A., Yakub, A., Ajani, G.E., Balogun, K.J., Renner, K.O., Igbo, J.K., Ariyo,A.A. and Bello, B.O.2010. Seasonal Variations in the Water Chemistry and Benthic Macroinvertebrates of a South Western Lagoon, Lagos, Nigeria. *Journal of American Science.* 6(3).

Nkwoji, J.A.M. 2017.The Impacts of Organic Pollution on the Hydrochemistry and Community Structure of Benthic Macro-fauna of Lagos Lagoon, Southwest Nigeria. *Journal of Applied Sciences and Environmental Management.* 21: 2

Nubi, O.A., Ajao, E.A. and Nubi, A.T. 2008. Pollution Assessment of the Impact of Coastal Activities On Lagos Lagoon, Nigeria.*Science World Journal.* 3:2

Nwadozie, J.M. 1998. The determination of heavy metal pollutants in fish samples from River Kaduna. *Journal of Chemistry Society*. 23: 21–23.

Nwankwo, D. I. 1998. The influence of saw-mill woodwastes on Diatom population atOkobaba Lagos, Nigeria. *Journal of Botany*. 11: 16-24.

Nwankwo, D.I. and Akinsoji, A. 1989. The Benthic Algal community of a sawdust Deposition Site in Lagos lagoon. *International Journal of Nigerian Environmental Sciences*. 15: 197-204.

Nwankwo, D.I. 1984. Seasonal changes of phytoplankton of Lagos lagoon and the adjacent sea in relation to environmental factors. Ph.D. Thesis, University of Lagos

Nwankwo, D.I. and Akinsoji, A. 1992.Epiphyte community on water hyacinth *Eichhornia crassipes* (Mart.).Solms. in coastal waters of southwestern Nigeria. *Archive for Hydrobiology.* 124(4): 501-511.

Nwankwo, D.I., Abosede, A.O. and Abdulrasaq, Y.1994. Floating timber logs as a substrate for periphyton algae in the Lagos Lagoon, Nigeria. *Pollution Archive for Hydrobiology* 4(4): 419 – 430.

Obermüller, B. Karsten, U. and Abele D. 2005. Response of oxidative stress parameters and sunscreening compounds in Arctic amphipods during experimental exposure to maximal natural UVB radiation. *Journalof Experimental Marine Ecology*. 323: 100-17.

Obire, O. and Aguda, M. 2002. Bacterial Community of Leachate from a Waste-Dump and an Adjacent Stream. *Journal of Applied Sciences and Environmental Management*. 6(2): 71-75.

Odiete, W. O. 1999.Environmental Physiology of animals and pollution Diversified resources, Ltd. Lagos., pp. 261.

Odiete, W.O. 1999.*Environmental Physiology of Animals and Pollution.* Diversified resources, Ltd. Lagos., pp. 261.

Odumuyiwa, O.F. 2010: Metals in Two Common Species (*Soleasoleand Pseudolithus spp*) from Lagos and Cocoa Lagoons in Lagos and Delta States. 5: 31.

Ohkawa, H., Ohishi, N., Yagi, K. 1979. Assay for lipi peroxides in animal tissues by thiobituric acid reaction. *Analytical Biochemistry* 95(2): 351 - 358

Okamoto,K.O. Pinto, E. Latorre, L.R. Bechara, E.J.H. and Colepicolo, P. 2001. Antioxidant modulation in response to metal-induced oxidative stress in algal chloroplasts. *Archive Environmental Contaminant Toxicology.* 40: 18-24.

Oketola, A.A. and Osibanjo, O.2009a. Estimating sectoral pollution load in Lagos by Industrial Pollution Projection System (IPPS): Employment versus Output. *Toxicological & Environmental Chemistry*. 91(5): 799-818.

Oketola, A.A. and Osibanjo, O. 2009b. Industrial pollution load assessment by Industrial Pollution Projection System (IPPS). *Toxicological & Environmental Chemistry*. 91(5).

Okoye, B.C.O. 1991. Heavy metals and organisms in the Lagos Lagoon. *International. Journal of Environment Study*. 37: 285 – 292.

Olakolu, F. C. and Chukwuka, A. V. 2014. Trace metal concentrations and antioxidant activity in ovarian tissue of blue crab *Callinectes amnicola* from Lagos lagoon and implications for reproductive success. *Zoology and Ecology*24**:**278-284.

Olakolu, F.C., Hassan, A.A. and Akindele S.K. 2012. Seasonal variation of oxidative stress biomarkers in gills and muscle of blue crabs *Callinectes amnicola* from Lagos Lagoon. *European Journal of Scientific Research*. 5:406 – 415.

Olaniyan, C.I.O. 1969. The seasonal variation in the hydrology and total plankton of the Lagoons of South West-Nigeria. *Nigerian Journal of Science*. 3(2): 101-119.

Olowu, R. A., Ayejuyo, O. O., Adewuyi, G. O., Adejoro, I. A., Denloye, A. A. B., Babatunde, A. O. and Ogundajo A. L. 2010. Determination of Heavy Metals in Fish Tissues, Water and Sediment from Epe and Badagry Lagoons, Lagos, Nigeria.*European Journal of Chemistry.*7(1):215-221.

Onyema, I. C. and Popoola, R. T. 2013. The Physico-Chemical Characteristics, Chlorophyll Alevels and Phytoplankton Dynamics of the East Mole Area of the Lagos Harbour, Lagos. Department of Marine Sciences, *Scientific Research Journal of Asian Scientific Research*, 3(10):995-1010

Onyema, I.C., Nkwoji, J.A., and. Eruteya, O.J. 2010. The water chemistry and plankton Science, 6(1): 111-122.

Orbea, A., Ortiz-Zarragoitia, M., Solé, M., Porte, C. and Cajaraville, M.P. 2002. "Antioxidant Enzymes and PeroxisomeProliferation in Relation to Contaminant Body Burdens of PAHs and PCBs in Bivalve Molluscs, Crabs and Fish from the Urdaibai and Plentzia Estuaries (Bay of Biscay). *Aquatic Toxicology.* 58(1–2): 75–98.

Orun, I., Talas, Z.S., Ozdemir, I., Alkan, A., Erdogan, K. 2008. Antioxidative role of selenium on some tissues of (Cd2+, Cr3+)-induced rainbow trout. *Ecotoxicology and Environmental Safety.* 71: 71–75.

Osibanjo, O. 2006. Global and National POPs Situation-Effort and challenges. A commissioned paper presented at NES-IPEP Awareness *Raising Workshop,* Lagos. www.ipen.org 69p

Osibanjo, O., Daso, A.P. and Gbadebo, A.M. 2011. The impact of industries on surface water quality of River Ona and River Alaro in Oluyole industrialestate, Ibadan, *Nigeria. African Journal of Biotechnology*. 10(4): 696-702.

Overstreet, R.M. 1983. Metazoan Symbionts of Crustaceans. *The Biology of Crustacea: Pathobiology,* 6**:** 155-250.

Oyelola, O.T. and Babatunde, A.I. 2008. Effect of Municipal Solid Waste on the Levels of Heavy Metals in Olusosun Dumpsite Soil, Lagos State, *Nigeria. International Journal Applies Sciences*. 2(1):17–21.

Oyewo, E.O. 1998. Industrial sources and distribution of heavy metals in Lagos lagoon and their biological effects on estuarine animals. Ph.D Thesis, University of Lagos, Nigeria. 279pp.

Oyewo EO., Don Pedro K.N. and Otitoloju, A. 2009. Trend of heavy metal concentration in Lagos Lagoon ecosystem, Nigeria. *West African Journal of Applied Ecology*. 5(1): 4314-45601

Palace, V.P. and Klaverkamp, J.F. 1993. Variation of hepatic enzymes in three species of freshwater fish from precambrian shield lakes and the effect of cadmium exposure. *Comparative Biochemistry and Physiology*. 104: 147-54.

Pandey, S., Ahmad, I., Parvez, S., Bin-Hafeez, B., Haque, R. and Raisuddin, S. 2001. Effect of endosulfan on antioxidants of freshwater fish *Channa punctatus* Bloch: 1. Protection against lipid peroxidation in liver by copper pre-exposure. *Archives of Environmental Contamination and Toxicology.*41: 345–352.

Parikh, H., Rangrez, P., Bagchi, A., Desai, B.N. 2010. Effect of Dimethoate on Some Histoarchitecture of Freshwater Fish, *Oreochromis mossambicus* (Peters, 1852). *Bioscan* 5 (1): 55–58.

Paruruckumani, P.S., Maharajan, A., Ganapiriya, V., Narayanaswamy, Y., Raja Jeyasekar, R. 2015. Surface Ultrastructural Changes in The Gill And Liver Tissue Of Asian Sea Bass Lates Calcarifer (Bloch) Exposed To Copper. *Biological Trace Element Research.*94;3(3):157-60.

Parvez, S. and Raisuddin, S. 2006. Copper modulates non-enzymatic antioxidants in the freshwater fish *Channa punctata* (Bloch) exposed to deltamethrin. *Chemosphere.* 62: 1324–1332.

Parvez, S., Sayeed, I., Pandey, S., Ahmad, A., Bin-Hafeez, B., Haque, R., Ahmad, I. and Raisuddin, S. 2003. Modulatory effect of copper on non-enzymatic antioxidants in freshwater fish *Channa punctatus* (Bloch.). *Biological Trace Element Research.* 93:237–248.

Paulino, M., Souza, N. and Fernandes, M. 2012. Subchronic Exposure to Atrazine Induces Biochemical And Histopathological Changes In The Gills of A Neotropical Freshwater Fish, Prochilodus Lineatus. *Ecotoxicology and Environmental Safety.*80**:**6-13.

Peace, D. 2006.*Water transporters link boat mishaps to pollution*. Daily Sun. Saturday November 11. Pg 29.

Peakall, D.B.1994. The role of biomarkers in environmental assessment. Sep; 3(3):157-60. doi: 10.1007/BF00117080

Pedersen, S.N, Lundebye, A-K and Depledge, M.H.1998. Field application of metallothioein and stress protein biomarkers in the shore crab (*Carcinus maenas*) exposed to trace metals. *Aquatic Toxicology Journal.* 37: 183-200.

Pellerin-Massicote, J. 1994. Oxidative process as indicators of chemical stress in marine bivalves. *Journal of Aquatic Ecosystem and Health*. 3(2): 101-11.

Peltola, V., Huhtaniemi, I. and Ahotupa, M. 1992. "Antioxidant Enzyme Activity in the Maturing Rat Testis." *Journal of Andrology* 13: 450–455.

Perkins-Visser, E., Wolcott, T.G. and Wolcott, D.L.1996. Nursery role of seagrass beds: enhanced growth of juvenile blue crabs (*Callinectes sapidus* Rathbun). *Journal of Experimental Marine Biology and Ecology.* 198: 155–173.

Pina, S., Russell-Pinto, F. and Rodrigues, P. 2011. Morphological and Molecular Study Of Microphallus Primas (Digenea: Microphallidae) Metacercaria, Infecting the Shore Crab *Carcinus maenas*from Northern Portugal. *Folia Parasitological*58**:** 48.

Pinto, E., Sigaud-Kutner, T.C.S., Leitão, M.A.S., Okamoto, O.K. and Morse, D. 2003. Colepicolo P. Heavy metal-induced oxidative stress in algae. *Journal of Phycology.* 39: 1008-18.

Playle, R.C.1998. Modelling metal interactions at fish gills. *Science of Total Environment* 219:147–63

Ploch, S.A., Lee, Y.-P., MacLean, E. and Di Giulio, R.T. 1999. Oxidative stress in liver of brown bullhead and channel catfish following exposure to tert-butyl hydroperoxide. *Aquatic Toxicology Journal.* 46: 231-240.

Pörtner, H.O. 2002. Climate variations and the physiological basis of temperature dependent biogeography: systemic to molecular hierarchy of thermal tolerance in animals. *Comparative Biochemistry and Physiology*. 132: 739-61.

Priju, C.P. and Narayana, A.C. 2009. *Coastal landform changes in and around Cochin and their implications in coastal zone management*. In: K.S.Jayappa and A.C.Narayana (Eds.), Coastal Environments: Problems and Perspectives.pp108-117.

Puntarulo, S. and Cederbaum, A.I. 1988. Comparison of the ability of the ferric complexes to catalyze microsomal chemiluminescence, lipid peroxidation and hydroxyl radical generation. *Archive Biochemistry and Biophysiology.* 264: 482-91.

Quinlan, G.J., Halliwell, B., Moorhouse, C.P. and Gutteridge, J.M.C. 1988. Action of lead (II) and aluminium (III) ions on ironstimulated lipid peroxidation in liposomes, erythrocytes and rat liver microsomal fractions. *Biochemistry and Biophysiology Acta*. 962: 196-200.

Qunfang, Z, Jianbin, Z., Jianjie, F., Jianbo, S. and Guibin, J. 2008. Biomonitoring: An appealing tool for assessment of metal pollution in the aquatic ecosystem. *Analytica Chimica Acta* 606:135–150.

Rainbow, P.S. 1998.*Phylogeny of trace metal accumulation in crustaceans*. In: Angston WJ, Bebianno MJ (eds) Metal metabolism in aquatic environments. Chapman & Hall, London, 285−319pp.

Rakhi, S.F., Reza, A.H.M.M., Hossen, M.S., Hossain, Z. 2013. Alterations In Histopathological Features And Brain Acetylcholinesterase Activity In Stinging Catfish, Heteropneustes FossilisExposed To Polluted River Water. *Internationalof Aquatic Resources*. 5(7): 1–18

Ramesh, F. and Nagarajan, K.2007. Histopathological Changes In Gills of *Clarias batrachus* Treated with Sago Effluent. *Journal of Experimental Zoology.*10**:**169-171.

Rana, S.V.S, Singh R. and Verma, S. 1995. Mercury-induced lipid peroxidation in the liver, kidney, brain and gills of a fresh water fish *Channa punctatus*. *Japanese Journal of Ichthyology.*42: 255–259.

Raufu, A. 2006. Negative echoes from Lagos lagoon. www.ecotopics.com/archives.htm-60k.12/21/2006.7/29/2007.

Reddy, S.J., Reddy, B.V., and Ramamurthi, R. 1991. Impact of chronic phosalone toxicity on erythropoietic activity of fish, Oreochromis mossambicus. *Biochemistry International.* 25: 547-552.

Regoli F., (1992). Lysosomal responses as a sensitive stress index in biomonitoring heavy metal pollution. *Marine Ecology Programme Ser*ology. 84: 63-9.

Regoli, F. and Principato, G. 1995. Glutathione, glutathione-dependent and antioxidant enzymes in mussel, *Mytilus galloprovincialis*, exposed to metals under field and laboratory conditions: Implications for the use of biochemical biomarkers. *AquaticToxicology.* 31:143-164.

Regoli, F. 1998. Trace metals and antioxidant enzymes in gills and digestive gland of the Mediterranean mussel *Mytilus galloprovincialis*. *Archive of EnvironmentalContamination and Toxicology.* 34: 48–63.

Regoli, F. Hummel, C., Amiard-Triquet, C., Larroux A and Sukhotin A. 1998. "Trace metals and variations of antioxidant enzymes in Arcticbivalve populations." *Archive of Environmental Contamination and Toxicology.* 35(4): 594-601.

Regoli, F., Gorbi, S. and Frenzilli, G. 2002. Oxidative stress in ecotoxicology: from the analysis of individual antioxidants to a more integrated approach. *Marine Environmental Resources*. 54: 419-23.

Regoli, F., Principato, G.B., Bertoli, E., Nigro, M. and Orlando, E. 1997. Biochemical characterization of the antioxidant system in the scallop *Adamussium colbecki*, a sentinel organism for monitoring the Antarctic environment. *Polar Biol*ogy. 17: 251-58.

Reméo, D., Bennani, N., Gnassia-Barelli, M., Lafaurie, M., Girard, J.P. 2000. Cadmium and copper display different responses towards oxidative stress in the kidney of the sea bass Dicentrarchus labrax. *Aquatic Toxicology.* 48: 185–194.

Richmonds, C., Dutta, H.M., (1989). Histopathological Changes Induced By Malathion In The Gills Of Bluegill *Lepomis macrochirus*. *Bulletin Environment Contamination Toxicolog* 43: 123–130.

Rijstenbil, J.W. 2003. Effects of UVB radiation and salt stress on growth, pigments and antioxidative defence of the marine diatom *Cylindrotheca closterium*. *Marine Ecology Program Serology.* 254: 37-48.

Ringwood, A.H., Conner, D.E., Keppler, C.J. and Dinovo, A. 1999. Biomarker studies with juvenile oysters (*Crassostrea virginica*) deployed *in situ*. *Biomarkers*. 4: 400-414.

Ringwood, A.H., Conners, D.E., Hoguet, J., and Ringwood, L.A.2005. Lysosomal Destabilization Assays in Estuarine Organisms. *Aquatic Toxicology*. 2: 287-300.

Rivera-Ingraham, G.A., Malanga, G. and Puntarulo, S. 2013. Antioxidantdefenses and trace metal bioaccumulation capacity of *Cymbula nigra* (Gastropoda: Patellidae). *Water, Air and Soil Pollut*ion. 224: 1458-66.

Robaldo, R.B., Monserrat, J.M., Cousin, J.C.B. and Bianchini, A. 1999. Effects of Metacercariae (Digenea: Microphallidae) on the Hepatopancreas of Chasmagnathus Granulata (Decapoda: Grapsidae).

Roesijadi, G. 1996. Metallothionein and its role in toxic metal regulation. *Comparative Biochemistry and Physiology.* 113: 117–123.

Robert, N. 2001. Habitat Fragmentation in a Seagrass Landscape: Patch Size and Complexity Control Blue Crab Survival. *Ecology.* 82(7):1814-1829 ·

Roganovic – Zafirova, D., Jordanova, M., Panov, S. and Velkova - Jordanoska, L. 2003. Hepatic capillariasis in the Mediterranean barbell (Barbus meridionalis petenyi heck.) from lake Ohrid. *Folia Veterinaria*. 47(1), 35 – 37.

Romeo, M. Bennani, M. Gnassia-Barelli, M. Lafaurie, M. and Girard, J.P. 2000. Cadmium and copper display different response towards oxidative stress in the kidney of the sea bass *Dicentrarchus labrax*. *Aquatic Toxicology.* 48: 185–194.

Romero, M.C., Tapella, F., Sotelano, M, Pa., Ansaldo, M. and Lovrich, G.A. 2011. Oxidative stress in the subantarctic false king crab *Paralomis granulosa* during air exposure and subsequent re-submersion. *Aquaculture*. 319: 205-10.

Rotruck, J.T., Pope, A.L., Ganther, H.E. and Swanson, A.B. 1973: Selenium biochemical role as a component of glutathione peroxideea. *Public Medical* 179(4073): 588 – 90.

Ruas, C.B.G. Carvalho, C.D. Araujo, H.S.S. Espindola, E.L.G. and Fernandes, M.N. 2008. Oxidative stress biomarkers of exposure in the blood of cichlid species from a metal-contaminated river. *Ecotoxicology and Environmental Safety.* 71: 86–93.

Ryer, C.H.,Van Montfrans, J. and Moody, K.E.1997. Cannibalism, refugia and the molting blue crab. *Marine Ecology Progress Series.* 147:77- 85.

Sahan, A., E. Belge and T. Altun, 2010. The determination of biochemical indicators (Biomarkers) in the common Carp (*Cyprinu carpio*) to the Physico-chemical parameters of Ceyhan river (Adana-Turkey). *Ekoloji.* 19(76): 8-14.

Salih, H.M. and Aljabre. H. 2002. Hospital Generated Waste: A Plan for Its Proper Management. *Journal of Family Community Med*icine. 9(2): 61–65

Sanchez, W., Palluel, O., Meunier, L., Coquery, M., Porcher, J.M. and Aït-Aïssa, S. 2005. Copper-induced oxidative stress in three-spined stickleback: relationship with hepatic metal levels. *Environmental Toxicology and Pharmacology.* 19: 177–183.

Saravana Bhavan, P. and Geraldine, P. 2000. Histopathology of The Hepatopancreas And Gills Of The Prawn Macrobrachium Malcolmsonii Exposed To Endosulfan. *Aquatic. Toxicology*. 50: 331–339.

Saravana Bhavan, P. snd Geraldine, P. 2009. Manifestation of Carbaryl Toxicity On Soluble Protein And Histopathology In The Hepatopancreas And Gills of The Prawn, Macrobrachium Malcolmsonii. *Journal of Environmental Biology.* 30(4): 533–538.

Saville, D. and Irwin, S. 2005. A Study of the Mechanisms by Which the Cercariae of *Microphallus primas* (Jag, 1909) Stunkard, 1957 Penetrate The Shore Crab, *Carcinus maenas* (L). *Parasitology-Cambridge.*131**:**521.

Sawyer, T. K., Lewis, E. J., Galasso, M. E., Ziskowski, J. J., Pacheco, A. L. and Gorski, S. W. 1985. Gill Blackening 761 And Fouling in The Rock Crab, Cancer Irroratus, As An Indicator Of Coastal Pollution. *Environmental Science and Technology.*6**:** 113-129

Schweikert, K. and Burritt, D.J. 2012. The organophosphate insecticide Coumaphos induces oxidative stress and increases antioxidant and detoxification defences in the green macroalgae *Ulva pertusa. Aquatic Toxicol*ogy. 122(123): 86-92.

Sears, M.E. 2013. Chelation: harnessing and enhancing heavy metal detoxification—a review. *Science of World Journal*. 219840. 18i:10.1155

Seok, S.H., Baek, M.W., Lee, H.Y., Kim, D.J., Na, Y.R., Noh, K.J., Park, S.H., Lee, H.K., Lee, B.H., Ryu, D.Y. and Park, J.H. 2007. Arsenite-induced apoptosis is prevented by antioxidants in zebrafish liver cell line. *Toxicology* in Vitro. 21: 870–877.

Shams, T. and Ahmad M. 2011. Some enzymatic/non enzymatic antioxidants as potential biomarkers of trichloroethylene, heavy metals mixture and ethyl alcohol in rat tissues. *Environmental Toxicology.* 26(2):207-216

Sies, H. 1993. Damage to Plasmid Dna by Singlet Oxygen and Its Protection. *Mutation Research/Genetic Toxicology.*299**:**183-191.

Simboura, N. A. Zenetos, P. Panayotidis,and Makra, A.1995. Changes of benthic community structure along an environmental pollution gradient *Marine Pollution Buletin*. 30(7): 470-474.

Smirnov, L.P., Sukhovskaya, I.V. and Nemova, N.N. 2005. Effects on environmental factors on low-molecular-weight peptides of fishes: A Review. *Russian Journal of Ecology.* 36:41–47.

Sole, M., Porte, C., Biosca, X., Mithcelmore, C.L., Chipman, J.K., Livingstone, D.R., Albaiges, T., (1996). Effects of the Aegean Sea oilspill on biotransformation enzymes, oxidative stress and DNA adducts in the digestive glands of the muscle (*Mytillus edulis* L.). *Comparative Biochemistry and Physiology.*113: 257–265.

Soyinka, A. (2007). Simple hygiene can prevent 45% of recoded deaths.*The Punch*. Wednesday May 30.pg42.

Stegeman, J.J., Brouwer, M., Richard, T.D.G., Forlin, L., Fowler, B.A., Sanders, B.M., van Veld, P.A. 1992. Molecular responses to environmental contamination: enzyme and protein systems as indicators of chemical exposure and effect. In: Huggett, R.J.,Kimerly, R.A., Mehrle,Suter, G.W., II, 1993. *Ecological Risk Assessment*. Lewis Publishers, Boca Raton, FL, USA, p. 538.

Stentiford, G., Longshaw, M., Lyons, B., Jones, G., Green, M. and Feist, S.2003. Histopathological Biomarkers in Estuarine Fish Species For The Assessment Of Biological Effects Of Contaminants. *Marine EnvironmentalResearch.*55**:**137-159.

Stentiford, G.D. and Feist, S.W. 2005. A Histopathological Survey of Shore Crab (*Carcinus Maenas*) And Brown Shrimp (*Crangon Crangon*) From Six Estuaries In The United Kingdom. *Journal of Invertebrate Pathology.*88:136–146.

Stohs, S.J. and Bagghi, D. 1995. Mechanisms in the toxicity of metal ions. *Free Radical Biology Medicine.* 18: 321-36.

Stumm, W. and Morgan, J.J. 1994. *Aquatic Chemistry*. John Wiley, New York, NY

Sturve, J., Hasselberg, L., Fälth, H., Celander, M. and Förlin, L. 2006. Effects of North Sea oil and alkylphenols on biomarker responses in juvenile Atlantic cod (*Gadus morhua*). *Aquatic Toxicology.*78: 73-8.

Suhel, P.Suwarna P., Mehboob A. andSheikh, R. 2006.Biomarkers of oxidative stress in *Wallago attu* (Bl. and Sch.) during and after a fish-kill episode at Panipat, *India.Science of Total Environment*.368(2–3)**:** 627-636

Sureda, A., Box, A., Tejada, S., Blanco, A., Caixach, J. and Deudero, S. 2011. Biochemical responses of *Mytilus galloprovincialis* as biomarkers of acute environmental pollution caused by the Don Pedro oil spill (Eivissa Island, Spain). *Aquatic Toxicol*ogy. 101: 540-9.

Suresh, B., Steiner, W., Rydlo, M. 1999. Concentration of 17 elements in zebra mussel (Dreissena polymorpha). *Environmental Toxicology and Chemistry. 18:2574–9.*

Suresh, G., Ramasamy, V., Meenakshisundaram, V., Venkatachalapathy, R., Ponnu- samy, V., 2011. Influence of mineralogical and heavy metal composition on natural radionuclide contents in the river sediments. *Applied Radiation.* 69: 1466–1474.

Suter, G.W. 1990. *Use of biomarkers in ecological risk assessment*. In: McCarthy, J.F., Shugart, L.R. (Eds.), Biomarkers of Environmental Contamination. Lewis Publishers, Boca Raton, FL, USA,pp. 419-428.

Suter, G.W. 2001. Applicability of indicator monitoring to ecological risk assessment.

Talling, J.F. and I.B. Talling, 1965. The chemical composition of African Lake Waters. *Hydrobiogia*. 50: 421-463.

Tanabe, S.and Subramanian, A. 2003. Biomarkers and Analytical Methods for the Analysis of POPs in Developing Countries. STAP/GEF and Ministry of Environment, *STAP Workshop on The Use of Bioindicators,* 2003, p. 1.

Tao, S., Wen, Y., Long, .A, Dawson, R., Cao, and Xu, F. 2001. Simulation of acid-base condition and copper speciation in fish gill microenvironment. *Computers and Chemistry*. 25: 215–222.

Tapley, D.W., Buettner, G.R. and Shick J.M. 1999. Free radicals and chemiluminescence as products of the spontaneous oxidation of sulfide in seawater, and their biological implications. *Biology Bulletin*. 196: 52-6.

Tatur, A., Valle, R. and Barczuk, A. 1999. *In: Polish Polar Studies*, Proceedings of XXVI Polar Symposium. 305-21pp.

Tavakoly Sany, B., Sulaiman, A.H., Monazami G.H. and Salleh A. 2011. Assessment of Sediment Quality According To Heavy Metal Status in the West Port of Malaysia. World

Academy of Science, Engineering and Technology *International Journal of Biological*, Biomolecular, Agricultural, Food and Biotechnological Engineering 5:2

Tehrani, A.A.G., Sadeghi, Z., Badamchi Sanjou, N.H., Mansoub Azhari, A. 2011. Effect of Carbamates Pesticides On Instar I-Ii Larvae And Adult Artemia Urmiana. *Annual Biology Resources*. 2(3): 515–525.

Ternjej, I, Mihaljevic, Z, Stankovic, I, Kerovec, M, Sipos, L, Zeljezic, D. and Kopjar, N. 2010. Estimation of DNA integrity in blood cells of eastern mosquitofish (*Gambusia holbrooki*) inhabiting an aluminium-polluted water environment: an alkaline comet assay study. *Archives of Environmental Contamination and Toxicology*. 59: 182–193.

Thomas, P. and Wofford, H. W. 1984. Effects of metals and organic compounds on hepatic glutathione, cysteine, and acid-soluble thiol levels in mullet (*Mugil cephalus* L.). *Toxicology and Applied Pharmacology*. 76: 172.

Thomas, P., and Wofford, H.W. 1993. Effects of cadmium and Aroclor 1254 on lipid peroxidation, glutathione peroxidase activity, and selected antioxidants in Atlantic croaker tissues. *Aquatic Toxicology*. 27: 159–178.

Threlfall, W. 1968. A Mass Die-Off of Three-Spined Sticklebacks (*Gasterosteus aculeatus* L.) Caused by Parasites. *Canadian Journal of Zoology.*46**:**105-106.

Toman, R. and Massányi,P. 2002. “Changes in the Testis and Epididymis of Rabbits After an Intraperitoneal and Peroral Administration of Cadmium.” *Trace Elements and Electrolytes.* 19: 114–117.

Tomlinson, D.L., Wilson, J.G., Harris, C.R. and Helgolander. 1980. Problems in Assessment of Heavy Metals in Estuaries and the Formation of Pollution.*Helgoland Marine Research.*33(1):566-575 ·

Torres, M.A., Barros, M.P. and Campos, S.C.G. 2008. Biochemical biomarkers in algae and marine pollution: A review. *Ecotoxicology and Environmental Safety.* 71: 1-15.

Tschischka, K., Abele, D. and Pörtner, H.O. 2000. Mitochondrial oxy-conformity and cold adaptation in the polychaete *Nereis pelagica* and the bivalve *Arctica islandica* from Baltic and White Seas. *Journal of Experimental Biology*. 203: 3355-68.

Ukwe, C.N, Ibe, C.A, Nwilo, P.C, and Huidobro, P.A. 2006.Contribution to the WSSD Targets on Oceans and Coasts in West and Central Africa: The Guinea Current Large Marine Ecosystem Project". *International Journal of Oceanography.*1(1): 21-44.

UNESCO. 2000. Reducing megacity impacts on the coastal environment: Alternative livelihoods and waste management in Jakarta and the Seribu Islands. Coastal region and small island papers 6, UNESCO, Paris, 59 pp.

Valavanidis, A., Vlachogianni, T., Dassenakis, E. and Scoullos M. 2006. Molecular biomarkers of oxidative stress in aquatic organisms in relation to toxic environmental pollutants. *Ecotoxicology and Environmental Safety.*46:178-189.

Valko, M., Morris, H. and Cronin, M.T.D. 2005. Metals, toxicity and oxidative stress. *Current Medicinal Chemistry.*12: 1161–1208.

Van Der Oost, R., Beyer, J. and Vermeulen, N. P.2003. Fish Bioaccumulation And Biomarkers In Environmental Risk Assessment: A Review. *Environmental Toxicology and Pharmacology.*13**:**57-149.

Verlecar, X.N., Jena, K.B. and Chainy, G.B.N. 2008. Biochemical markers of oxidative stress in *Perna viridis* exposed to mercury and temperature, *Chemico-Biological Interactions* 167: 219- 226.

Viarengo, A. 1989. Heavy metals in marine invertebrates, mechanisms of regulation and toxicity at cellular concentrations. *Revelation Aquatic Sciences.* 1: 295-317.

Viarengo, A., Burlando, B., Cavaletto, M., Marchi, B. and Ponzano, E. 1999. Blasco J. Role of metallothionein against oxidative stress in the mussel *Mytilus galloprovincialis. American Journal Physiol Regulatory Integrative Comparative Physiology.* 277: 1612-9.

Viarengo, A., Canesi, L., Martinez, P.G., Peters, L.D. and Livingstone, D.R. 1995. Prooxidant processes and antioxidant defence systems in the tissues of the Antarctic scallop (*Adamussium colbecki*) compared with the Mediterranean scallop (*Pecten jacobaeus*). *Comparative Biochemistry and Physiology.* 111: 119-26.

Viarengo, A., Canesi, L., Pertica, M., Poli, G., Moore, M.N., Orunesu, M. 1990. Heavy metal effects on lipid peroxidation in the tissues of Mytilus galloprovincialis Lam. *Comparative Biochemistry and Physiology.* 97: 37–42.

Viarengo, A., L. Canesi, M. andPertica, D.R.1991. Livingstone, Seasonal variations in the antioxidant defence systems and lipid peroxidation of the digestive gland of mussels, *Comparative Biochemistry and Physiology C: Pharmacology, Toxicology and Endocrinology.* 100: 187–190.

Viarengo, A., Lowe, D., Bolognesi, C., Fabbri, E. and Koehler, A. 2007. The use of biomarkers in biomonitoring: a 2-tier approach assessing the level of pollutant-induced stress syndrome in sentinel organisms. *Comparative Biochemistry and Physiology.* 146: 281-300.

Victor, B. 1984. Reproductive Biology of Freshwater Prawn, Caridinarajadhari (Ph.D. Thesis). Marathwada University, Aurangabad.

Vijayaraman, K., John, G., Sivakumar, P. and Mohamed, R.R. 1999. Uptake and less of heavy metal by the freshwater prawn, Macrobrachium malcolmsonii. *Journal of Enviromental Biological*. 20(3): 217-222.

Vijayavel, K., Gopalakrishnan, S., Thilagam.H and Balasubramanian,M.P. 2006. Dietary ascorbic acid and -tocopherol mitigates oxidative stress induced by copper in the thorn fish *Terapon jarbua.Scienceof Total Environment.* 372: 157–163.

Vijayavel, K., R.D. Gomathi, K. Durgabhavani and M.P. Balasubramanian. 2004. Sublethal effect of naphthalene on lipid peroxidation and antioxidant status in the edible marine crab *Scylla serrata. Marine Pollution Bulletin*. 48(5-6): 429-433.

Vinodhini, R. and Narayanan, M. 2008. Bioaccumulation of heavy metals in organs of fresh

Viswanathan, S. and Manisseri, M.K. 1995. Histopathological Studies on Zinc Toxicity in Penaeus Indicus H. Milne Edwards. In: Rengarajan, K. (Ed.). In: Mariculture Research Under the *Postgraduate Programme in Mariculture Pa*rt-6, Vol. 61. Cmfri, Cochin, India. 25–29pp.

Voigt, H.-R. 2003. Concentrations of mercury and cadmium in some coastal fishes from the Finnish and Estonian parts of the Gulf of Finland. *Proceedings of the Estonian Academy of Sciences Biology Ecology*. 52(3): 305-318

Wagner,M.,Klein.C.L., Van, T.C., Kooten, and Kirkpatrick C.J. 1998. Mechanism of cell activation by heavy metal ions *Journal of Biomedical Materials. Resources.* 3:395-399.

Wang, C.H., Zhao, Y., Zheng, R., Ding, X., Wei, W., Zuo, Z. and Chen, Y. 2006. Effects of tributyltin, benzo[a]pyrene, and their mixture on antioxidant defense systems in *Sebastiscus marmoratus.Ecotoxicology and Environmental Safety.*65: 381–387.

water fish *Cyprinus carpio* (Common carp).*International Journal of Environmental. Science Technology*. 5:179-182.

Weber, L.J and Gingerich, W.H. 1982. Hepatic Toxicology of Fishes. In: Aquatic Toxicology, Weber L.J.(Ed.) *Raven press,* New York, pp:55-105.

Weber, L.J.and Gingerich, W.H. 1982. Hepatic Toxicology of Fishes. In: Aquatic Toxicology, Weber L.J.(Ed.) *Raven press*, New York, pp:55-105.

Weihe, E. and Abele, D. 2008. Differences in the physiological response of inter- and subtidal Antarctic limpets *Nacella concinna* to aerial exposure. Aquatic Biology. 4: 155-66.

Weihe, E., Kriews, M. and Abele, D. 2010. Differences in heavy metal concentrations and in the response of the antioxidant system to hypoxia and air exposure in the Antarctic limpet *Nacella concinna*. *Marine Environmental Resources.*69(3): 127-35.

Werner, I., Broeg, K, Cain, D, Wallace, W, and Hornberger, M., 1999, Biomarkers of heavy metal effects in two species of caddisfly larvae from Clark Fork River, Montana: stress proteins (HSP70) and lysosomal membrane integrity, in: Presented at 20th Annual Meeting Society of *Environmental Toxicology and Chemistry.*

WHO. 1998. Environmental health criteria, Copper.WHO, Geneva WHO International Programme on Chemical Safety (IPCS). 1993. Biomarkers and risk assessment: concepts and principles. *Environmental Health Criteria* 155, World Health Organization, Geneva.

World Health Organization (WHO). 2011. *Guidelines for Drinking Water*. 4th ed. Geneva: Taylor & Francis.

Wilce, M. C. & Parker, M. W. 1994. Structure and Function of Glutathione S-Transferases. *Biochimica Et Biophysica Acta (Bba)-Protein Structure and Molecular Enzymology,* 1205**,** 1-18.

Williamson, R. B. and Wilcock, R. J. 1994, The Distribution and Fate of Contaminants in Estuarine Sediments: Recommendations for environmental monitoring and assessment, *Technical Publication No*. 47, Auckland Regional Council, Auckland, New Zealand.

Williamson, R. B., Blom, A., Hume, T. M., Glasby, G. P. and Larcombe, M. (1992). Heavy Metals in Manukau Harbour Sediments, Water Quality *Center Publication* 23, 23, Hamilton, New Zealand.

Winston, G.W. and Digiulio, R.T.1991. Pro-oxidant and antioxidant mechanisms in aquatic organisms. *Aquatic Toxicology.* 19: 137-161.

Wolke, R.E. 1992. Piscine macrophage aggregates: A Review, Ann. Rev Fish Dis., 2:91-108.

Wood, C.M. 2001. Toxic responses of the gill. In Schlenk DW and Benson WH (eds), Target Organ. Toxicity in Marine and Freshwater Teleosts, vol. 1—Organs. *Taylor & Francis*, Washington, DC

Woodward, B. 1994. Dietary vitamin requirements of cultured young fish, with emphasis on quantitative estimates for salmonids. *Aquaculture*. 124: 133-68.

World Health Organization, WHO. 1972. Evaluation of certain food additives and contaminants- mercury, lead and cadmium. WHO Expert Committee on Food Additives. WHO Technical Report Series No.

Wright, D.A. and Welbourn, P. 2002. *Environmental Toxicology*. Cambridge: *Taylor & Francis*;

Wu, H.F. and Zhao,B.S. 2006. The Analytical Methods in the Monitoring of Water and Wastewater, China Environmental Science Press, Beijing. P.G.C. Campbell, *Environmental Chemistry*. 3:387.

Wu, R.S.S., Zhou, B.S., Randall, D.J., Woo, N.Y.S and Lam, P.K.S. 2003. Hypoxia is an endocrine disruptor and impairs fish reproduction, *Environmental Science and Technology*. 37: 1137–1141.

Xiong, F.S. 2001. Evidence that UV-B tolerance of the photosynthetic apparatus in microalgae is related to the D1-turnover mediated repair cycle *in vivo*. *Journal of Plant Physiology*. 158: 285-94.

Xu, J.L. and Yang, J.R.1996. Heavy Metals in Terrestrial Ecosystem, China *Environmental Science* Press, Beijing.

Yakovleva, I.M., Baird, A.H., Yamamoto, H.H. Bhagooli, R., Nonaka Hidaka, M. 2009. Algal symbionts increase oxidative damage and death in coral larvae at high temperatures. *Marine Ecology Programe Ser*ology. 378: 105-12.

Yamamoto, Y., Fujisawa, A., Hara, A. and Dunlap, W.C. 2001. An unusual vitamin E constituent provides antioxidant protection in marine organisms adapted to coldwater environments. *Proc Natl Acad Sci* USA; 98: 13144-48.

Yagi ,T., Inutsuka , S., Kondo, T. 1998. *Isothermal Compression Curve of Al_2SiO_5 Kyanite:* 10: 1029. Pp 0281.

Yang, S., Z. Zhang, J. He, J. Li, J. Zhang, H. Xing, and Xu.S. 2012. "Ovarian Toxicity Induced by Dietary Cadmium in Hen." *Biological Trace Element Research*. 148(1): 53–60.

Yildrin, E .Y., Akalp, Y., Aytac, S., Bayram, N. 2011. Factors influencing information security management in small and medium-sized enterprises: A case study from Turkey. *International Journal of Information Management.* 31(4):360-365 ·

Zhao, S.C., Feng, W.,Quan, X., Chen, J., Niu, and Shen, Z. 2012. Role of living environments in the accumulation characteristics of heavy metals in fishes and crabs in the Yangtse River Estuary, China. *Marine Pollution Bull*etin. 64:1163-1171.

Zhou, Q., Zhang, J., Fu, J., Shi, J.and Jiang, G. 2008.Biomonitoring: An appealing tool for assessment of metal pollution in the aquatic ecosystem. State Key Laboratory of Environmental Chemistry and Ecotoxicology.*Analytica Chimica Acta.*606: 135–150

Žikić, R.V., S. Štajn, S.Z. Pavlović, I. Ognjanović, and Saićić, Z.S. 2001. "Activities of Superoxide Dismutase and Catalase in Erythrocytes and Plasma Transaminases of Goldfish (Carassius auratus gibelio Bloch.) Exposed to Cadmium."*Physiology Research.*50: 105–111.

Zini, A. and Schlegel, P.N. 1996. "Catalase mRNA Expression in the Male Rat Reproductive Tract." *Journal of Andrology.* 17: 473–4.

Zutshi, B. and Murthy, P.S. 2001. Ultrastructural Changes in Testis of Gold Fish *Glossogobius giuris* (Ham.) Induced by Fenthion. *Indian Journal of Experimental Biology.* 39: 170–173.

APPENDIX

Appendix 1: Occurrence of Pollution Load Index (PLI)

Study Stations	PLI dry season	PLI rainy season
Makoko	1.90	0.87
Okobaba	1.19	0.6
Iddo	1.09	1.48
Ajah	0.72	0.27
Ikoyi	0.35	0.35
Mid-Lagoon (Control station)	0.30	0.24

Appendix 2a: Biota-Sediment accumulation factor of metals between Hepatopancreas of *C. amnicola* and sediments from sampling stations in the Lagos lagoon

	Makoko	Okobaba	Iddo	Ajah	Ikoyi	Mid-lagoon
Cd	0.19	0.68	0.80	0.44	0.29	0.14
Pb	0.08	0.14	0.07	0.16	0.17	0.04
Zn	0.30	0.28	0.25	1.56	2.50	0.20
Cu	2.20	0.41	0.37	0.53	1.41	0.11

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Highlighted values indicate values higher than hazard threshold i.e. BSAF=1

Appendix 2b: Biota-Sediment accumulation factor of metals between gill of *C. amnicola* and sediments from sampling stations in the Lagos lagoon

	Makoko	Okobaba	Iddo	Ajah	Ikoyi	Mid-lagoon
Cd	0.30	1.65	0.31	0.28	0.16	0.05
Pb	0.08	0.09	0.32	0.07	0.16	0.04
Zn	0.68	0.17	0.15	0.32	0.66	0.09
Cu	1.08	0.83	0.56	1.20	10.52	0.39

Highlighted values indicate values higher than hazard threshold i.e. BSAF=1

Appendix 2c: Biota-Sediment accumulation factor of metals between gonad of *C. amnicola* and sediments from sampling stations in the Lagos lagoon

	Makoko	Okobaba	Iddo	Ajah	Ikoyi	Mid-lagoon
Cd	0.61	1.28	0.70	1.38	0.51	0.09
Pb	0.05	0.07	0.06	0.05	0.30	0.02
Zn	0.14	0.12	0.19	0.59	1.71	0.07
Cu	1.22	0.68	0.52	1.32	1.90	0.10

Highlighted values indicate values higher than hazard threshold i.e. BSAF=1

Appendix 2d: Biota-Sediment accumulation factor of metals between Muscle (flesh) of *C. amnicola* and sediments from sampling stations in the Lagos lagoon

	Makoko	Okobaba	Iddo	Ajah	Ikoyi	Mid-lagoon
Cd	0.13	0.56	1.14	0.32	0.11	0.10
Pb	0.07	0.14	0.07	0.05	0.11	0.02
Zn	0.16	0.09	0.18	0.53	1.22	0.11
Cu	0.30	0.19	0.16	0.77	0.53	0.08

Highlighted values indicate values higher than hazard threshold i.e. BSAF=1

Appendix 3: ANOVA AND Descriptive Statistics for Physicochemical characteristics

ONEWAY MONTH AIR_TEMPERATURE WATER_TEMPERATURE DISSOLVED_OXYGEN SALINITY CONDUCTIVITY pH BOD ALKALINITY BY STATION/STATISTICS HOMOGENEITY BROWNFORSYTHE WELCH /PLOT MEANS/MISSING ANALYSIS (p<0.05).

ANOVA

		Sum of Squares	df	Mean Square	F	Sig.
MONTH	Between Groups	.000	5	.000	.000	1.000
	Within Groups	858.000	66	13.000		
	Total	858.000	71			
AIR_TEMPERATURE	Between Groups	2.958	5	.592	1.782	.129
	Within Groups	21.917	66	.332		
	Total	24.875	71			
WATER_TEMPERATURE	Between Groups	6.792	5	1.358	1.543	.189
	Within Groups	58.083	66	.880		
	Total	64.875	71			
DISSOLVED_OXYGEN	Between Groups	131.760	5	26.352	2.919	.019
	Within Groups	595.759	66	9.027		
	Total	727.519	71			
SALINITY	Between Groups	1800.077	5	360.015	13.028	.000
	Within Groups	1823.830	66	27.634		
	Total	3623.907	71			
CONDUCTIVITY	Between Groups	3720.448	5	744.090	13.693	.000
	Within Groups	3586.395	66	54.339		
	Total	7306.843	71			
pH	Between Groups	2.190	5	.438	5.913	.000
	Within Groups	4.890	66	.074		
	Total	7.080	71			
BOD	Between Groups	34.545	5	6.909	3.397	.009
	Within Groups	134.230	66	2.034		
	Total	168.775	71			
ALKALINITY	Between Groups	1859.325	5	371.865	8.198	.000
	Within Groups	2993.670	66	45.359		
	Total	4852.995	71			

Robust Tests of Equality of Means

		Statistic[a]	df1	df2	Sig.
MONTH	Welch	.000	5	30.800	1.000
	Brown-Forsythe	.000	5	66.000	1.000
AIR_TEMPERATURE	Welch	1.604	5	30.647	.189
	Brown-Forsythe	1.782	5	59.032	.131
WATER_TEMPERATURE	Welch	1.575	5	30.760	.196
	Brown-Forsythe	1.543	5	63.464	.189
DISSOLVED_OXYGEN	Welch	4.490	5	30.635	.003
	Brown-Forsythe	2.919	5	55.977	.021
SALINITY	Welch	14.679	5	30.546	.000
	Brown-Forsythe	13.028	5	52.464	.000
CONDUCTIVITY	Welch	16.169	5	30.696	.000
	Brown-Forsythe	13.693	5	61.908	.000
pH	Welch	5.337	5	30.704	.001
	Brown-Forsythe	5.913	5	61.519	.000
BOD	Welch	3.439	5	29.565	.014
	Brown-Forsythe	3.397	5	40.690	.012
ALKALINITY	Welch	20.738	5	30.086	.000
	Brown-Forsythe	8.198	5	41.189	.000

a. Asymptotically F distributed.

Appendix 4a: ANOVA AND Descriptive Statistics for Heavy Metals in Crab Organs (Hepatopancreas, Gill, Gonad and muscle)
CADMIUM WITH THE BLUE CRAB

Descriptive Statistics

	N	Range	Minimum	Maximum	Mean		Std. Deviation	Variance
	Statistic	Statistic	Statistic	Statistic	Statistic	Std. Error	Statistic	Statistic
HEPA	60	2	0	2	.38	.057	.439	.192
GILL	60	6	0	6	.41	.110	.850	.722
GONAD	60	9	0	9	1.02	.162	1.252	1.568
MUSCLE	60	2	0	2	.27	.043	.336	.113
STATION	60	5.00	1.00	6.00	3.5000	.22234	1.72224	2.966
Valid N (listwise)	60							

Means

Eport

STATION		HEPA	GILL	GONAD	MUSCLE
MAKOKO	Mean	.45	.81	1.66	.36
	N	10	10	10	10
	Std. Deviation	.291	1.976	2.542	.649
	Minimum	0	0	0	0
	Maximum	1	6	9	2
	Std. Error of Kurtosis	1.334	1.334	1.334	1.334
	Std. Error of Mean	.092	.625	.804	.205
	Range	1	6	8	2
OKOBABA	Mean	.39	.38	.73	.32
	N	10	10	10	10
	Std. Deviation	.589	.408	.716	.462
	Minimum	0	0	0	0
	Maximum	2	1	2	2
	Std. Error of Kurtosis	1.334	1.334	1.334	1.334
	Std. Error of Mean	.186	.129	.226	.146
	Range	2	1	2	2

IDDO	Mean	.18	.45	.90	.19
	N	10	10	10	10
	Std. Deviation	.112	.549	.958	.108
	Minimum	0	0	0	0
	Maximum	0	2	3	0
	Std. Error of Kurtosis	1.334	1.334	1.334	1.334
	Std. Error of Mean	.035	.173	.303	.034
	Range	0	2	3	0
AJAH	Mean	.43	.27	1.35	.31
	N	10	10	10	10
	Std. Deviation	.448	.219	.525	.127
	Minimum	0	0	0	0
	Maximum	2	1	2	1
	Std. Error of Kurtosis	1.334	1.334	1.334	1.334
	Std. Error of Mean	.142	.069	.166	.040
	Range	1	1	2	0
IKOYI	Mean	.57	.33	1.02	.21
	N	10	10	10	10
	Std. Deviation	.677	.243	.981	.187
	Minimum	0	0	0	0
	Maximum	2	1	3	0
	Std. Error of Kurtosis	1.334	1.334	1.334	1.334
	Std. Error of Mean	.214	.077	.310	.059
	Range	2	1	3	0
MID-LAGOON	Mean	.26	.22	.44	.23
	N	10	10	10	10
	Std. Deviation	.224	.062	.283	.127
	Minimum	0	0	0	0
	Maximum	1	0	1	0
	Std. Error of Kurtosis	1.334	1.334	1.334	1.334
	Std. Error of Mean	.071	.020	.090	.040
	Range	1	0	1	0
Total	Mean	.38	.41	1.02	.27
	N	60	60	60	60
	Std. Deviation	.439	.850	1.252	.336
	Minimum	0	0	0	0
	Maximum	2	6	9	2
	Std. Error of Kurtosis	.608	.608	.608	.608
	Std. Error of Mean	.057	.110	.162	.043
	Range	2	6	9	2

ANOVA Table

			Sum of Squares	df	Mean Square	F	Sig.
HEPA * STATION	Between Groups	(Combined)	.972	5	.194	1.011	.420
		Linearity	.003	1	.003	.018	.893
		Deviation from Linearity	.969	4	.242	1.260	.297
	Within Groups		10.381	54	.192		
	Total		11.353	59			
GILL * STATION	Between Groups	(Combined)	2.241	5	.448	.600	.700
		Linearity	1.542	1	1.542	2.064	.157
		Deviation from Linearity	.699	4	.175	.234	.918
	Within Groups		40.343	54	.747		
	Total		42.584	59			
GONAD * STATION	Between Groups	(Combined)	9.577	5	1.915	1.248	.300
		Linearity	3.238	1	3.238	2.109	.152
		Deviation from Linearity	6.339	4	1.585	1.032	.399
	Within Groups		82.909	54	1.535		
	Total		92.486	59			
MUSCLE * STATION	Between Groups	(Combined)	.218	5	.044	.367	.869
		Linearity	.096	1	.096	.805	.374
		Deviation from Linearity	.123	4	.031	.258	.904
	Within Groups		6.429	54	.119		
	Total		6.647	59			

LEAD (Pb)
Means

Report

STATION		HEPA	GILL	GONAD	MUSCLE
MAKOKO	Mean	2.82	1.44	1.27	2.32
	N	10	10	10	10
	Std. Deviation	6.770	2.011	1.421	3.396
	Minimum	0	0	0	0
	Maximum	22	6	4	11
	Std. Error of Kurtosis	1.334	1.334	1.334	1.334
	Std. Error of Mean	2.141	.636	.449	1.074
	Range	22	6	4	11
OKOBABA	Mean	5.17	3.17	1.14	1.44
	N	10	10	10	10
	Std. Deviation	7.668	2.080	.693	1.828
	Minimum	0	0	0	0
	Maximum	24	8	2	6
	Std. Error of Kurtosis	1.334	1.334	1.334	1.334
	Std. Error of Mean	2.425	.658	.219	.578
	Range	24	7	2	6
IDDO	Mean	3.07	1.10	1.12	3.66
	N	10	10	10	10
	Std. Deviation	5.646	1.412	1.954	7.417
	Minimum	0	0	0	0
	Maximum	19	4	6	23
	Std. Error of Kurtosis	1.334	1.334	1.334	1.334
	Std. Error of Mean	1.785	.447	.618	2.346
	Range	18	4	6	23
AJAH	Mean	3.30	1.56	1.16	1.16
	N	10	10	10	10
	Std. Deviation	5.309	2.625	2.803	2.153
	Minimum	0	0	0	0
	Maximum	17	9	9	7
	Std. Error of Kurtosis	1.334	1.334	1.334	1.334
	Std. Error of Mean	1.679	.830	.886	.681
	Range	17	9	9	7

IKOYI	Mean	2.11	2.04	3.83	1.32
	N	10	10	10	10
	Std. Deviation	2.970	2.830	6.916	1.596
	Minimum	0	0	0	0
	Maximum	9	9	22	5
	Std. Error of Kurtosis	1.334	1.334	1.334	1.334
	Std. Error of Mean	.939	.895	2.187	.505
	Range	9	9	22	5
MID-LAGOON	Mean	.81	1.18	1.00	.49
	N	10	10	10	10
	Std. Deviation	.714	.790	.542	.310
	Minimum	0	0	0	0
	Maximum	3	3	2	1
	Std. Error of Kurtosis	1.334	1.334	1.334	1.334
	Std. Error of Mean	.226	.250	.172	.098
	Range	2	3	2	1
Total	Mean	2.88	1.75	1.59	1.73
	N	60	60	60	60
	Std. Deviation	5.319	2.110	3.245	3.581
	Minimum	0	0	0	0
	Maximum	24	9	22	23
	Std. Error of Kurtosis	.608	.608	.608	.608
	Std. Error of Mean	.687	.272	.419	.462
	Range	24	9	22	23

ANOVA Table

			Sum of Squares	df	Mean Square	F	Sig.
HEPA * STATION	Between Groups	(Combined)	103.194	5	20.639	.712	.617
		Linearity	51.446	1	51.446	1.774	.189
		Deviation from Linearity	51.748	4	12.937	.446	.775
	Within Groups		1566.287	54	29.005		
	Total		1669.481	59			
GILL * STATION	Between Groups	(Combined)	29.768	5	5.954	1.380	.246
		Linearity	2.556	1	2.556	.592	.445
		Deviation from Linearity	27.212	4	6.803	1.577	.194
	Within Groups		232.989	54	4.315		
	Total		262.757	59			
GONAD * STATION	Between Groups	(Combined)	60.710	5	12.142	1.169	.336
		Linearity	6.547	1	6.547	.631	.431
		Deviation from Linearity	54.164	4	13.541	1.304	.280
	Within Groups		560.668	54	10.383		
	Total		621.379	59			
MUSCLE * STATION	Between Groups	(Combined)	61.935	5	12.387	.963	.449
		Linearity	20.578	1	20.578	1.600	.211
		Deviation from Linearity	41.357	4	10.339	.804	.528
	Within Groups		694.523	54	12.862		
	Total		756.458	59			

For ZINC (Zn)

Descriptives

Descriptive Statistics

	N	Range	Minimum	Maximum	Mean		Std. Deviation	Variance
	Statistic	Statistic	Statistic	Statistic	Statistic	Std. Error	Statistic	Statistic
HEPA	60	8	1	9	5.86	.278	2.156	4.648
GILL	60	5	1	5	1.51	.108	.840	.706
GONAD	60	8	1	9	2.74	.242	1.872	3.506
MUSCLE	60	7	0	7	2.35	.172	1.331	1.772
STATION	59	5.00	1.00	6.00	3.4576	.22200	1.70519	2.908
Valid N (listwise)	59							

Means

Report

STATION		HEPA	GILL	GONAD	MUSCLE
MAKOKO	Mean	6.27	1.62	3.00	3.28
	N	10	10	10	10
	Std. Deviation	2.044	1.380	1.228	1.739
	Minimum	3	1	2	0
	Maximum	9	5	6	7
	Std. Error of Kurtosis	1.334	1.334	1.334	1.334
	Std. Error of Mean	.646	.437	.388	.550
	Range	5	5	4	7
OKOBABA	Mean	6.24	1.62	2.78	1.94
	N	10	10	10	10
	Std. Deviation	2.145	.620	1.665	1.077
	Minimum	3	1	1	0
	Maximum	8	3	6	3
	Std. Error of Kurtosis	1.334	1.334	1.334	1.334
	Std. Error of Mean	.678	.196	.526	.341
	Range	5	2	5	3
IDDO	Mean	5.71	1.68	2.93	2.56
	N	10	10	10	10
	Std. Deviation	2.909	1.246	1.942	.811
	Minimum	1	1	1	1
	Maximum	9	5	7	3
	Std. Error of Kurtosis	1.334	1.334	1.334	1.334
	Std. Error of Mean	.920	.394	.614	.257
	Range	8	4	6	2
AJAH	Mean	6.53	1.43	2.59	2.31
	N	10	10	10	10
	Std. Deviation	1.852	.550	2.043	1.427
	Minimum	3	1	1	0
	Maximum	9	2	7	4
	Std. Error of Kurtosis	1.334	1.334	1.334	1.334
	Std. Error of Mean	.586	.174	.646	.451
	Range	6	2	6	4
IKOYI	Mean	5.71	1.50	3.89	2.78
	N	10	10	10	10
	Std. Deviation	2.408	.488	2.557	1.303
	Minimum	2	1	1	1
	Maximum	9	2	9	5
	Std. Error of Kurtosis	1.334	1.334	1.334	1.334
	Std. Error of Mean	.762	.154	.809	.412
	Range	6	2	7	4

MID-LAGOON	Mean	4.95	1.17	1.25	1.32
	N	9	9	9	9
	Std. Deviation	1.006	.241	.265	.465
	Minimum	3	1	1	1
	Maximum	7	2	2	2
	Std. Error of Kurtosis	1.400	1.400	1.400	1.400
	Std. Error of Mean	.335	.080	.088	.155
	Range	4	1	1	1
Total	Mean	5.92	1.51	2.77	2.38
	N	59	59	59	59
	Std. Deviation	2.124	.847	1.875	1.319
	Minimum	1	1	1	0
	Maximum	9	5	9	7
	Std. Error of Kurtosis	.613	.613	.613	.613
	Std. Error of Mean	.277	.110	.244	.172
	Range	8	5	8	7

ANOVA Table

			Sum of Squares	df	Mean Square	F	Sig.
HEPA * STATION	Between Groups	(Combined)	15.396	5	3.079	.662	.653
		Linearity	7.115	1	7.115	1.531	.221
		Deviation from Linearity	8.281	4	2.070	.445	.775
	Within Groups		246.351	53	4.648		
	Total		261.747	58			
GILL * STATION	Between Groups	(Combined)	1.659	5	.332	.441	.818
		Linearity	1.073	1	1.073	1.425	.238
		Deviation from Linearity	.586	4	.147	.195	.940
	Within Groups		39.911	53	.753		
	Total		41.570	58			
GONAD * STATION	Between Groups	(Combined)	34.434	5	6.887	2.154	.073
		Linearity	3.677	1	3.677	1.150	.288
		Deviation from Linearity	30.757	4	7.689	2.405	.061
	Within Groups		169.420	53	3.197		
	Total		203.854	58			
MUSCLE * STATION	Between Groups	(Combined)	21.976	5	4.395	2.951	.020
		Linearity	7.184	1	7.184	4.824	.032
		Deviation from Linearity	14.792	4	3.698	2.483	.055
	Within Groups		78.934	53	1.489		
	Total		100.910	58			

FOR COPPER (Cu)

Descriptive Statistics

	N	Range	Minimum	Maximum	Mean		Std. Deviation	Variance
	Statistic	Statistic	Statistic	Statistic	Statistic	Std. Error	Statistic	Statistic
HEPA	60	43	1	44	10.45	1.008	7.809	60.975
GILL	60	79	0	79	27.75	2.401	18.599	345.929
GONAD	60	177	1	178	23.40	3.726	28.859	832.825
MUSCLE	60	21	0	21	9.66	.635	4.917	24.172
STATION	60	5.00	1.00	6.00	3.5167	.22360	1.73197	3.000
Valid N (listwise)	60							

Means

Report

STATION		HEPA	GILL	GONAD	MUSCLE
MAKOKO	Mean	6.57	26.08	40.24	9.95
	N	10	10	10	10
	Std. Deviation	5.054	17.343	55.079	4.728
	Minimum	2	7	8	2
	Maximum	18	62	178	18
	Std. Error of Kurtosis	1.334	1.334	1.334	1.334
	Std. Error of Mean	1.598	5.484	17.417	1.495
	Range	16	55	170	16
OKOBABA	Mean	14.64	29.48	23.96	6.67
	N	10	10	10	10
	Std. Deviation	9.609	18.591	19.983	8.215
	Minimum	6	0	4	0
	Maximum	38	67	70	21
	Std. Error of Kurtosis	1.334	1.334	1.334	1.334
	Std. Error of Mean	3.039	5.879	6.319	2.598
	Range	32	67	66	21
IDDO	Mean	10.41	33.44	19.32	9.70
	N	10	10	10	10
	Std. Deviation	5.524	18.926	13.199	3.640
	Minimum	2	12	9	4
	Maximum	20	75	51	15
	Std. Error of Kurtosis	1.334	1.334	1.334	1.334
	Std. Error of Mean	1.747	5.985	4.174	1.151
	Range	18	63	42	11

AJAH	Mean	9.12	37.10	21.82	12.16
	N	9	9	9	9
	Std. Deviation	7.609	24.637	16.077	3.458
	Minimum	2	3	3	6
	Maximum	27	79	46	16
	Std. Error of Kurtosis	1.400	1.400	1.400	1.400
	Std. Error of Mean	2.536	8.212	5.359	1.153
	Range	24	77	43	10
IKOYI	Mean	12.47	29.67	25.96	11.36
	N	11	11	11	11
	Std. Deviation	11.788	16.192	30.377	4.317
	Minimum	1	8	8	7
	Maximum	44	54	100	21
	Std. Error of Kurtosis	1.279	1.279	1.279	1.279
	Std. Error of Mean	3.554	4.882	9.159	1.302
	Range	43	46	92	14
MID-LAGOON	Mean	9.18	11.48	8.67	8.18
	N	10	10	10	10
	Std. Deviation	.823	2.023	3.468	1.347
	Minimum	8	9	1	5
	Maximum	10	16	12	10
	Std. Error of Kurtosis	1.334	1.334	1.334	1.334
	Std. Error of Mean	.260	.640	1.097	.426
	Range	2	7	11	5
Total	Mean	10.45	27.75	23.40	9.66
	N	60	60	60	60
	Std. Deviation	7.809	18.599	28.859	4.917
	Minimum	1	0	1	0
	Maximum	44	79	178	21
	Std. Error of Kurtosis	.608	.608	.608	.608
	Std. Error of Mean	1.008	2.401	3.726	.635
	Range	43	79	177	21

ANOVA Table

			Sum of Squares	df	Mean Square	F	Sig.
HEPA * STATION	Between Groups	(Combined)	403.107	5	80.621	1.363	.253
		Linearity	5.012	1	5.012	.085	.772
		Deviation from Linearity	398.095	4	99.524	1.682	.167
	Within Groups		3194.423	54	59.156		
	Total		3597.530	59			
GILL * STATION	Between Groups	(Combined)	3853.908	5	770.782	2.514	.041
		Linearity	675.363	1	675.363	2.203	.144
		Deviation from Linearity	3178.545	4	794.636	2.592	.047
	Within Groups		16555.912	54	306.591		
	Total		20409.821	59			
GONAD * STATION	Between Groups	(Combined)	5268.324	5	1053.665	1.297	.279
		Linearity	3111.031	1	3111.031	3.830	.056
		Deviation from Linearity	2157.293	4	539.323	.664	.620
	Within Groups		43868.343	54	812.377		
	Total		49136.667	59			
MUSCLE * STATION	Between Groups	(Combined)	199.964	5	39.993	1.761	.137
		Linearity	8.949	1	8.949	.394	.533
		Deviation from Linearity	191.015	4	47.754	2.103	.093
	Within Groups		1226.193	54	22.707		
	Total		1426.157	59			

Appendix 4b: ANOVA AND Descriptive Statistics for Heavy Metals in Surface Water and Sediment

ONEWAY Cd.sediment Cd.water Pb.sediment Pb.water Zn.sediment Zn.water Cu.sediment Cu.water
BY Station
/STATISTICS DESCRIPTIVES/PLOT MEANS/MISSING ANALYSIS

Descriptives

		N	Mean	Std. Deviation	Std. Error	95% Confidence Interval for Mean Lower Bound	95% Confidence Interval for Mean Upper Bound	Minimum	Maximum
Cd.sediment	Makoko	10	2.7250	1.15603	.36557	1.8980	3.5520	.00	3.92
	Okobaba	10	.5686	.55558	.17569	.1712	.9660	.00	1.66
	Iddo	10	1.4711	2.37462	.75092	-.2276	3.1698	.00	7.28
	Ajah	10	.9783	.75973	.24025	.4348	1.5218	.00	1.87
	Ikoyi	10	1.9870	1.40958	.44575	.9786	2.9954	.00	4.98
	Mid Lagoon	10	.2103	.44355	.14026	-.1070	.5276	.00	1.08
	Total	60	1.3234	1.50675	.19452	.9341	1.7126	.00	7.28
Cd.water	Makoko	10	.0244	.01545	.00489	.0133	.0355	.00	.05
	Okobaba	10	.0362	.02057	.00650	.0215	.0509	.01	.07
	Iddo	10	.0468	.03100	.00980	.0246	.0689	.00	.09
	Ajah	10	.0522	.09103	.02879	-.0129	.1174	.00	.31
	Ikoyi	10	.0344	.02306	.00729	.0179	.0509	.00	.06
	Mid Lagoon	10	.0216	.01966	.00622	.0075	.0356	.00	.05
	Total	60	.0359	.04213	.00544	.0250	.0468	.00	.31
Pb.sediment	Makoko	10	34.2355	41.33779	13.07216	4.6642	63.8068	.00	109.27
	Okobaba	10	37.1007	27.36824	8.65460	17.5226	56.6788	.00	98.82
	Iddo	10	51.9395	45.15791	14.28019	19.6355	84.2435	.00	144.06
	Ajah	10	21.2917	19.90104	6.29326	7.0554	35.5280	.00	55.09
	Ikoyi	10	12.6142	19.33531	6.11436	-1.2174	26.4458	.00	49.10
	Mid Lagoon	10	20.6252	22.82808	7.21887	4.2950	36.9554	.00	63.30
	Total	60	29.6345	32.47991	4.19314	21.2440	38.0249	.00	144.06
Pb.water	Makoko	10	.2333	.25457	.08050	.0512	.4154	.00	.64
	Okobaba	10	.1224	.13696	.04331	.0244	.2204	.00	.41
	Iddo	10	.1681	.15141	.04788	.0598	.2764	.00	.48
	Ajah	10	.2577	.24432	.07726	.0830	.4325	.00	.84
	Ikoyi	10	.3041	.46149	.14594	-.0260	.6343	.00	1.53
	Mid Lagoon	10	.1791	.16227	.05132	.0630	.2952	.00	.47
	Total	60	.2108	.25611	.03306	.1446	.2770	.00	1.53
Zn.sediment	Makoko	10	20.7325	18.31502	5.79172	7.6307	33.8343	.00	44.72
	Okobaba	10	22.6939	13.48368	4.26391	13.0483	32.3395	.00	45.36
	Iddo	10	33.7804	15.49608	4.90029	22.6952	44.8656	.00	51.60
	Ajah	10	4.3757	4.60136	1.45508	1.0841	7.6673	.00	12.94
	Ikoyi	10	2.2823	2.83154	.89541	.2567	4.3079	.00	10.15
	Mid Lagoon	10	11.2190	8.93999	2.82707	4.8237	17.6143	.00	27.88
	Total	60	15.8473	15.99368	2.06478	11.7157	19.9789	.00	51.60

Zn.water	Makoko	10	.0000	.00000	.00000	.0000	.0000	.00	.00
	Okobaba	10	.0005	.00121	.00038	-.0004	.0013	.00	.00
	Iddo	10	.0001	.00032	.00010	-.0001	.0003	.00	.00
	Ajah	10	.0000	.00000	.00000	.0000	.0000	.00	.00
	Ikoyi	10	.0027	.00826	.00261	-.0032	.0086	.00	.03
	Mid Lagoon	10	.0000	.00000	.00000	.0000	.0000	.00	.00
	Total	60	.0006	.00341	.00044	-.0003	.0014	.00	.03
Cu.sediment	Makoko	10	32.9445	32.66919	10.33091	9.5744	56.3146	.00	91.18
	Okobaba	10	35.4978	30.03009	9.49635	14.0156	56.9800	.00	96.84
	Iddo	10	60.0521	53.88456	17.03980	21.5054	98.5988	.00	155.70
	Ajah	10	15.7249	25.58855	8.09181	-2.5800	34.0298	.00	66.42
	Ikoyi	10	2.5100	3.88036	1.22708	-.2658	5.2858	.00	12.25
	Mid Lagoon	10	29.5763	23.40721	7.40201	12.8318	46.3208	.00	68.72
	Total	60	29.3843	35.36907	4.56613	20.2475	38.5211	.00	155.70
Cu.water	Makoko	10	.0087	.00808	.00255	.0029	.0145	.00	.02
	Okobaba	10	.0200	.04362	.01380	-.0112	.0512	.00	.14
	Iddo	10	.0839	.08126	.02570	.0258	.1420	.00	.23
	Ajah	10	.0236	.03206	.01014	.0007	.0466	.00	.11
	Ikoyi	10	.0389	.05658	.01789	-.0016	.0793	.00	.16
	Mid Lagoon	10	.0732	.06737	.02130	.0250	.1214	.00	.16
	Total	60	.0414	.05861	.00757	.0262	.0565	.00	.23

ANOVA

		Sum of Squares	df	Mean Square	F	Sig.
Cd.sediment	Between Groups	43.545	5	8.709	5.202	.001
	Within Groups	90.403	54	1.674		
	Total	133.948	59			
Cd.water	Between Groups	.007	5	.001	.804	.552
	Within Groups	.097	54	.002		
	Total	.105	59			
Pb.sediment	Between Groups	10148.868	5	2029.774	2.104	.079
	Within Groups	52092.875	54	964.683		
	Total	62241.743	59			
Pb.water	Between Groups	.221	5	.044	.653	.660
	Within Groups	3.649	54	.068		
	Total	3.870	59			
Zn.sediment	Between Groups	7293.652	5	1458.730	10.101	.000
	Within Groups	7798.426	54	144.415		
	Total	15092.077	59			
Zn.water	Between Groups	.000	5	.000	1.002	.425
	Within Groups	.001	54	.000		
	Total	.001	59			
Cu.sediment	Between Groups	18994.079	5	3798.816	3.742	.006
	Within Groups	54813.218	54	1015.060		
	Total	73807.297	59			
Cu.water	Between Groups	.047	5	.009	3.227	.013
	Within Groups	.156	54	.003		
	Total	.203	59			

Appendix 5: ANOVA and Descriptive statistics for Oxidative Enzymes and Cellular Damage Biomarker in Crab Organs (Hepatopancreas, Gill, Gonad and muscle).

BIOMARKERS

Descriptives

Season			N	Mean	Std. Deviation	Std. Error	95% Confidence Interval for Mean		Minimum	Maximum
							Lower Bound	Upper Bound		
Dry season	MDA_hepa	Makoko	4	29.2788	3.29831	1.64915	24.0305	34.5272	24.37	31.37
		Okobaba	4	34.8654	9.25295	4.62648	20.1419	49.5889	22.08	44.15
		Iddo	4	42.7404	5.30608	2.65304	34.2972	51.1835	34.87	46.31
		Ajah	4	34.8654	8.25958	4.12979	21.7225	48.0082	23.02	41.73
		Ikoyi	4	32.2067	5.30821	2.65411	23.7602	40.6533	26.25	38.37
		Mid-lagoon	4	20.8990	7.35956	3.67978	9.1883	32.6097	14.00	27.46
		Total	24	32.4760	9.01694	1.84057	28.6684	36.2835	14.00	46.31
	MDA_gill	Makoko	4	19.0144	1.01558	.50779	17.3984	20.6304	18.31	20.46
		Okobaba	4	21.6731	1.23866	.61933	19.7021	23.6441	19.92	22.62
		Iddo	4	22.2452	2.92330	1.46165	17.5936	26.8968	19.25	26.12
		Ajah	4	19.3173	.78108	.39054	18.0744	20.5602	18.58	20.06
		Ikoyi	4	20.1923	1.84575	.92288	17.2553	23.1293	17.90	21.94
		Mid-lagoon	4	27.7308	9.67734	4.83867	12.3320	43.1296	13.46	34.46
		Total	24	21.6955	4.81691	.98325	19.6615	23.7295	13.46	34.46
	MDA_gonad	Makoko	4	23.3558	2.24314	1.12157	19.7864	26.9251	21.00	26.38
		Okobaba	4	25.8798	5.62425	2.81213	16.9304	34.8293	21.81	34.19
		Iddo	4	27.5962	7.68841	3.84421	15.3622	39.8301	22.21	38.90
		Ajah	4	29.1442	7.98478	3.99239	16.4387	41.8498	20.33	37.29
		Ikoyi	4	24.1635	4.31120	2.15560	17.3034	31.0235	19.25	29.21
		Mid-lagoon	4	29.3798	1.56746	.78373	26.8856	31.8740	28.13	31.63
		Total	24	26.5865	5.39894	1.10205	24.3068	28.8663	19.25	38.90
	MDA_muscle	Makoko	4	23.0865	4.95280	2.47640	15.2055	30.9675	16.69	27.33
		Okobaba	4	19.8221	3.57069	1.78535	14.1403	25.5039	16.96	25.04
		Iddo	4	18.9135	2.53158	1.26579	14.8852	22.9418	16.42	21.54
		Ajah	4	19.7212	2.19963	1.09982	16.2210	23.2213	18.04	22.88
		Ikoyi	4	18.7788	2.17755	1.08878	15.3139	22.2438	16.56	21.54
		Mid-lagoon	4	18.2067	3.38128	1.69064	12.8264	23.5871	14.67	22.48
		Total	24	19.7548	3.32813	.67935	18.3495	21.1602	14.67	27.33
	GSH_hepa	Makoko	4	1.4178	.89392	.44696	-.0046	2.8403	.77	2.69
		Okobaba	4	1.4178	.89392	.44696	-.0046	2.8403	.77	2.69
		Iddo	4	1.1424	.25416	.12708	.7379	1.5468	.88	1.41
		Ajah	4	1.1027	.26737	.13369	.6772	1.5281	.75	1.39
		Ikoyi	4	1.9175	1.90434	.95217	-1.1128	4.9477	.75	4.74
		Mid-lagoon	4	.6427	.03013	.01506	.5948	.6907	.61	.68
		Total	24	1.2735	.92519	.18885	.8828	1.6642	.61	4.74
	GSH_gill	Makoko	4	.9041	.36796	.18398	.3186	1.4896	.60	1.36
		Okobaba	4	.7552	.35629	.17815	.1883	1.3222	.53	1.29
		Iddo	4	.7685	.32821	.16410	.2462	1.2907	.54	1.25
		Ajah	4	.7776	.31995	.15997	.2685	1.2867	.53	1.25
		Ikoyi	4	.9414	.37379	.18689	.3466	1.5361	.59	1.29
		Mid-lagoon	4	.6494	.10885	.05443	.4761	.8226	.57	.81
		Total	24	.7994	.30217	.06168	.6718	.9270	.53	1.36

	Makoko	4	1.0704	.47128	.23564	.3205	1.8203	.66	1.56
	Okobaba	4	.9604	.31357	.15678	.4614	1.4593	.66	1.33
	Iddo	4	1.0365	.40160	.20080	.3975	1.6755	.62	1.39
GSH_gonads	Ajah	4	1.4741	1.18264	.59132	-.4078	3.3559	.64	3.18
	Ikoyi	4	.8892	.44047	.22024	.1884	1.5901	.54	1.53
	Mid-lagoon	4	.3747	.04359	.02180	.3054	.4441	.35	.44
	Total	24	.9676	.61668	.12588	.7072	1.2280	.35	3.18
	Makoko	4	.9041	.35963	.17981	.3319	1.4764	.58	1.28
	Okobaba	4	.9083	.36858	.18429	.3218	1.4948	.57	1.30
	Iddo	4	1.0084	.37208	.18604	.4163	1.6004	.65	1.40
GSH_muscle	Ajah	4	.8363	.31204	.15602	.3398	1.3328	.61	1.27
	Ikoyi	4	.8562	.32763	.16381	.3348	1.3775	.61	1.31
	Mid-lagoon	4	.5013	.02612	.01306	.4597	.5429	.48	.53
	Total	24	.8358	.32538	.06642	.6984	.9732	.48	1.40
	Makoko	4	5.5233	1.15000	.57500	3.6933	7.3532	4.63	7.03
	Okobaba	4	5.4744	.88924	.44462	4.0595	6.8894	4.69	6.41
	Iddo	4	5.3520	.63420	.31710	4.3429	6.3612	4.78	5.96
GPx_hepa	Ajah	4	5.6763	.63963	.31982	4.6585	6.6941	4.78	6.29
	Ikoyi	4	5.6664	.64642	.32321	4.6378	6.6950	4.71	6.14
	Mid-lagoon	4	4.4140	.45596	.22798	3.6884	5.1395	4.12	5.08
	Total	24	5.3511	.81197	.16574	5.0082	5.6939	4.12	7.03
	Makoko	4	5.3735	.87665	.43832	3.9786	6.7685	4.51	6.36
	Okobaba	4	5.2776	.68727	.34363	4.1840	6.3712	4.58	5.96
	Iddo	4	5.1817	.67922	.33961	4.1009	6.2625	4.55	5.83
GPx_gills	Ajah	4	5.2379	.58492	.29246	4.3071	6.1686	4.67	5.79
	Ikoyi	4	5.2875	.64108	.32054	4.2674	6.3076	4.65	5.98
	Mid-lagoon	4	4.3908	.16402	.08201	4.1298	4.6518	4.20	4.53
	Total	24	5.1248	.66308	.13535	4.8448	5.4048	4.20	6.36
	Makoko	4	5.4662	.75459	.37729	4.2655	6.6669	4.82	6.33
	Okobaba	4	5.3471	.61945	.30972	4.3614	6.3327	4.81	5.97
	Iddo	4	5.4000	.69263	.34632	4.2979	6.5021	4.76	6.09
GPx_gonads	Ajah	4	5.3007	.57800	.28900	4.3810	6.2205	4.76	5.80
	Ikoyi	4	5.3371	.66435	.33218	4.2800	6.3943	4.74	5.99
	Mid-lagoon	4	4.4256	.31776	.15888	3.9199	4.9312	3.99	4.67
	Total	24	5.2128	.65835	.13438	4.9348	5.4908	3.99	6.33
	Makoko	4	5.4521	.97709	.48855	3.8973	7.0069	4.57	6.63
	Okobaba	4	5.3123	.72518	.36259	4.1584	6.4662	4.60	5.99
	Iddo	4	5.2892	.68401	.34200	4.2007	6.3776	4.65	5.92
GPx_muscle	Ajah	4	5.2544	.64864	.32432	4.2223	6.2865	4.67	5.88
	Ikoyi	4	5.2743	.65913	.32957	4.2254	6.3231	4.67	5.90
	Mid-lagoon	4	4.2634	.16437	.08218	4.0019	4.5250	4.02	4.35
	Total	24	5.1409	.73092	.14920	4.8323	5.4496	4.02	6.63
	Makoko	4	.2460	.11095	.05548	.0695	.4226	.16	.40
	Okobaba	4	.2562	.13255	.06628	.0453	.4671	.16	.45
	Iddo	4	.2141	.02125	.01062	.1802	.2479	.19	.24
CAT_hep	Ajah	4	.2083	.03438	.01719	.1536	.2630	.16	.24
	Ikoyi	4	.3431	.30945	.15473	-.1493	.8355	.16	.80
	Mid-lagoon	4	.1511	.01285	.00643	.1307	.1716	.14	.17
	Total	24	.2365	.14204	.02899	.1765	.2964	.14	.80
	Makoko	4	11.9900	9.70216	4.85108	-3.4483	27.4283	3.82	25.10
	Okobaba	4	7.4737	6.99449	3.49724	-3.6561	18.6035	3.14	17.91
	Iddo	4	10.5221	7.45023	3.72511	-1.3329	22.3771	3.75	18.11
CAT_gills	Ajah	4	14.1878	10.75654	5.37827	-2.9282	31.3039	2.79	26.23
	Ikoyi	4	11.7070	8.14984	4.07492	-1.2612	24.6753	4.04	19.86
	Mid-lagoon	4	9.0291	6.22915	3.11458	-.8829	18.9410	.35	14.95
	Total	24	10.8183	7.71905	1.57564	7.5588	14.0778	.35	26.23

	Makoko	4	7.7903	6.51518	3.25759	-2.5768	18.1574	2.07	15.46
	Okobaba	4	7.8738	7.85732	3.92866	-4.6290	20.3765	1.78	19.03
	Iddo	4	8.9028	7.39459	3.69730	-2.8636	20.6693	3.08	19.09
CAT_gonads	Ajah	4	5.9454	3.77808	1.88904	-.0664	11.9572	2.43	11.24
	Ikoyi	4	6.7308	4.16621	2.08311	.1014	13.3601	3.02	11.66
	Mid-lagoon	4	5.5416	4.30562	2.15281	-1.3096	12.3928	1.82	10.12
	Total	24	7.1308	5.35673	1.09344	4.8688	9.3927	1.78	19.09
	Makoko	4	5.1005	4.14927	2.07463	-1.5019	11.7029	1.86	11.19
	Okobaba	4	7.7839	5.48063	2.74031	-.9370	16.5048	3.18	15.54
	Iddo	4	5.5457	4.37193	2.18596	-1.4110	12.5025	1.86	11.88
CAT_muscle	Ajah	4	9.4279	5.91457	2.95729	.0165	18.8393	3.86	16.35
	Ikoyi	4	10.3626	7.70031	3.85015	-1.8903	22.6155	3.81	19.34
	Mid-lagoon	4	3.9067	1.26816	.63408	1.8888	5.9246	2.48	5.47
	Total	24	7.0212	5.18852	1.05910	4.8303	9.2121	1.86	19.34
	Makoko	4	.0000	.00003	.00001	.0000	.0001	.00	.00
	Okobaba	4	.0000	.00005	.00003	.0000	.0001	.00	.00
	Iddo	4	.0001	.00006	.00003	.0000	.0001	.00	.00
SOD_hep	Ajah	4	.0001	.00006	.00003	.0000	.0002	.00	.00
	Ikoyi	4	.0001	.00009	.00004	-.0001	.0002	.00	.00
	Mid-lagoon	4	.0001	.00004	.00002	.0000	.0001	.00	.00
	Total	24	.0001	.00005	.00001	.0000	.0001	.00	.00
	Makoko	4	1.3436	.87830	.43915	-.0540	2.7411	.30	2.35
	Okobaba	4	.8866	.70376	.35188	-.2333	2.0064	.30	1.91
	Iddo	4	1.2722	.77082	.38541	.0456	2.4987	.33	1.95
SOD_gill	Ajah	4	1.6038	1.15853	.57927	-.2397	3.4473	.25	2.65
	Ikoyi	4	1.3854	.99199	.49600	-.1931	2.9639	.19	2.40
	Mid-lagoon	4	.6438	.59394	.29697	-.3013	1.5889	.04	1.46
	Total	24	1.1892	.83770	.17099	.8355	1.5430	.04	2.65
	Makoko	4	.6337	.66698	.33349	-.4276	1.6950	.14	1.59
	Okobaba	4	.7120	.83480	.41740	-.6164	2.0403	.12	1.95
	Iddo	4	.7757	.67089	.33545	-.2918	1.8432	.22	1.71
SOD_gonad	Ajah	4	.4968	.49937	.24968	-.2978	1.2914	.05	1.15
	Ikoyi	4	.7359	.50110	.25055	-.0614	1.5333	.28	1.27
	Mid-lagoon	4	.2256	.08152	.04076	.0959	.3554	.11	.31
	Total	24	.5966	.55763	.11383	.3612	.8321	.05	1.95
	Makoko	4	.5957	.42161	.21081	-.0752	1.2666	.18	1.09
	Okobaba	4	.8666	.40029	.20015	.2296	1.5035	.27	1.11
	Iddo	4	.6559	.45647	.22824	-.0705	1.3822	.19	1.14
SOD_muscle	Ajah	4	1.0407	.54642	.27321	.1712	1.9102	.27	1.52
	Ikoyi	4	1.2612	.81903	.40952	-.0420	2.5645	.36	2.06
	Mid-lagoon	4	.4943	.24466	.12233	.1050	.8836	.24	.79
	Total	24	.8191	.52924	.10803	.5956	1.0425	.18	2.06
	Makoko	4	129.1097	100.84377	50.42188	-31.3552	289.5747	51.02	273.04
	Okobaba	4	71.0834	26.81559	13.40779	28.4139	113.7530	49.61	110.31
	Iddo	4	87.1654	11.26911	5.63455	69.2338	105.0971	77.44	102.44
Protein_hep	Ajah	4	84.6952	45.06471	22.53235	12.9872	156.4032	42.55	135.92
	Ikoyi	4	91.2994	58.60905	29.30453	-1.9607	184.5595	38.31	147.61
	Mid-lagoon	4	68.5628	32.93015	16.46508	16.1635	120.9620	28.23	108.89
	Total	24	88.6527	52.00045	10.61455	66.6948	110.6105	28.23	273.04
	Makoko	4	9.8811	4.82289	2.41145	2.2068	17.5554	4.44	14.52
	Okobaba	4	14.3175	6.54681	3.27340	3.9001	24.7350	5.44	19.36
	Iddo	4	9.6290	4.62013	2.31007	2.2774	16.9807	5.44	14.12
Protein_gill	Ajah	4	9.6794	6.63728	3.31864	-.8820	20.2408	3.83	17.34
	Ikoyi	4	14.5696	13.95785	6.97893	-7.6405	36.7796	4.64	34.68
	Mid-lagoon	4	39.8773	59.45976	29.72988	-54.7364	134.4911	9.07	129.06
	Total	24	16.3257	24.98243	5.09952	5.7765	26.8748	3.83	129.06

		Makoko	4	38.6674	45.75170	22.87585	-34.1338	111.4685	6.45	106.47
		Okobaba	4	28.5342	17.74451	8.87225	.2987	56.7697	5.24	44.36
		Iddo	4	20.7201	14.48120	7.24060	-2.3227	43.7629	6.05	40.33
	Protein_gonad	Ajah	4	55.7577	48.38197	24.19099	-21.2289	132.7442	8.87	104.05
		Ikoyi	4	18.4010	14.48026	7.24013	-4.6403	41.4424	8.27	39.73
		Mid-lagoon	4	37.1550	12.42909	6.21454	17.3775	56.9324	23.19	48.80
		Total	24	33.2059	29.31978	5.98487	20.8252	45.5865	5.24	106.47
		Makoko	4	27.3747	20.68146	10.34073	-5.5341	60.2835	13.51	58.08
		Okobaba	4	12.7547	2.39381	1.19691	8.9456	16.5638	9.68	15.33
		Iddo	4	23.5937	21.26482	10.63241	-10.2434	57.4307	11.90	55.46
	Protein_muscle	Ajah	4	11.4439	3.95976	1.97988	5.1431	17.7448	6.86	16.33
		Ikoyi	4	10.1836	5.29577	2.64788	1.7568	18.6103	5.04	16.74
		Mid-lagoon	4	15.8803	3.79903	1.89951	9.8352	21.9254	12.30	21.17
		Total	24	16.8718	12.89067	2.63130	11.4286	22.3151	5.04	58.08
		Makoko	3	33.8782	1.40112	.80894	30.3976	37.3588	32.31	35.00
		Okobaba	3	45.0962	4.58286	2.64591	33.7117	56.4806	40.38	49.54
		Iddo	3	41.4167	2.42806	1.40184	35.3850	47.4483	38.90	43.75
	MDA_hepa	Ajah	3	39.2628	15.14548	8.74425	1.6394	76.8863	28.27	56.54
		Ikoyi	3	34.6410	2.30948	1.33338	28.9040	40.3781	32.17	36.75
		Mid-lagoon	3	38.9487	4.38551	2.53198	28.0545	49.8429	33.92	42.00
		Total	18	38.8739	6.98850	1.64721	35.3986	42.3492	28.27	56.54
		Makoko	3	19.6987	.95504	.55139	17.3263	22.0712	18.85	20.73
		Okobaba	3	21.8526	.54404	.31410	20.5011	23.2040	21.27	22.35
		Iddo	3	22.7051	.33877	.19559	21.8636	23.5467	22.35	23.02
	MDA_gill	Ajah	3	20.5962	1.32581	.76546	17.3027	23.8896	19.52	22.08
		Ikoyi	3	21.2692	1.52894	.88273	17.4711	25.0673	20.19	23.02
		Mid-lagoon	3	34.1923	20.98446	12.11538	-17.9360	86.3206	22.08	58.42
		Total	18	23.3857	8.83804	2.08315	18.9906	27.7807	18.85	58.42
		Makoko	3	31.9487	4.74284	2.73828	20.1668	43.7306	26.92	36.35
		Okobaba	3	31.1859	3.08247	1.77966	23.5286	38.8432	27.73	33.65
		Iddo	3	50.0769	40.72065	23.51008	-51.0788	151.2326	23.15	96.92
	MDA_gonad	Ajah	3	34.1923	15.48779	8.94188	-4.2815	72.6661	23.56	51.96
		Ikoyi	3	38.9038	9.21009	5.31745	16.0247	61.7830	30.96	49.00
		Mid-lagoon	3	39.9808	3.49741	2.01923	31.2927	48.6688	35.94	42.00
		Total	18	37.7147	16.79838	3.95942	29.3611	46.0684	23.15	96.92
wet season		Makoko	3	21.4038	.48536	.28022	20.1981	22.6096	21.00	21.94
		Okobaba	3	21.2244	2.39424	1.38231	15.2767	27.1720	19.52	23.96
		Iddo	3	21.9872	1.38159	.79766	18.5551	25.4192	20.46	23.15
	MDA_muscle	Ajah	3	23.2885	2.64820	1.52894	16.7100	29.8669	20.87	26.12
		Ikoyi	3	22.6603	1.60601	.92723	18.6707	26.6498	20.87	23.96
		Mid-lagoon	3	24.3654	3.26425	1.88462	16.2565	32.4742	22.48	28.13
		Total	18	22.4882	2.14068	.50456	21.4237	23.5528	19.52	28.13
		Makoko	3	.6386	.03729	.02153	.5460	.7312	.60	.66
		Okobaba	3	.6386	.03729	.02153	.5460	.7312	.60	.66
		Iddo	3	.6915	.10364	.05983	.4341	.9490	.62	.81
	GSH_hepa	Ajah	3	1.8452	1.22596	.70781	-1.2002	4.8907	.63	3.08
		Ikoyi	3	.6419	.03685	.02127	.5504	.7334	.61	.68
		Mid-lagoon	3	.7566	.11464	.06619	.4718	1.0414	.65	.88
		Total	18	.8687	.61961	.14604	.5606	1.1769	.60	3.08
		Makoko	3	.5658	.01324	.00764	.5329	.5987	.55	.58
		Okobaba	3	.5592	.00573	.00331	.5450	.5734	.55	.56
		Iddo	3	.5989	.11068	.06390	.3239	.8739	.52	.72
	GSH_gill	Ajah	3	.6187	.02498	.01442	.5567	.6808	.60	.65
		Ikoyi	3	.5956	.02066	.01193	.5443	.6469	.57	.61
		Mid-lagoon	3	.5890	.03816	.02203	.4942	.6838	.55	.62
		Total	18	.5879	.04682	.01104	.5646	.6112	.52	.72

	Makoko	3	.6563	.01632	.00942	.6157	.6968	.65	.68
	Okobaba	3	.6971	.08985	.05187	.4739	.9203	.64	.80
	Iddo	3	.7125	.03179	.01836	.6335	.7915	.68	.74
GSH_gonads	Ajah	3	.6254	.03455	.01994	.5396	.7112	.59	.65
	Ikoyi	3	.7125	.05065	.02924	.5867	.8383	.68	.77
	Mid-lagoon	3	.6607	.09983	.05764	.4127	.9087	.55	.73
	Total	18	.6774	.06184	.01458	.6466	.7081	.55	.80
	Makoko	3	1.1658	.96913	.55953	-1.2416	3.5733	.55	2.28
	Okobaba	3	.5691	.02626	.01516	.5039	.6344	.55	.60
	Iddo	3	.5868	.02674	.01544	.5203	.6532	.56	.62
GSH_muscle	Ajah	3	.5526	.03156	.01822	.4742	.6310	.52	.58
	Ikoyi	3	.5746	.00764	.00441	.5557	.5936	.57	.58
	Mid-lagoon	3	.5625	.04452	.02570	.4519	.6731	.51	.60
	Total	18	.6686	.40434	.09530	.4675	.8696	.51	2.28
	Makoko	3	4.8750	.17625	.10176	4.4372	5.3128	4.67	4.99
	Okobaba	3	4.8728	.09130	.05271	4.6460	5.0996	4.80	4.98
	Iddo	3	4.9919	.01665	.00962	4.9505	5.0333	4.98	5.01
GPx_hepa	Ajah	3	4.9346	.08379	.04838	4.7264	5.1427	4.87	5.03
	Ikoyi	3	4.9996	.01442	.00833	4.9638	5.0355	4.98	5.01
	Mid-lagoon	3	4.7007	.33875	.19558	3.8592	5.5422	4.33	4.99
	Total	18	4.8958	.17234	.04062	4.8101	4.9815	4.33	5.03
	Makoko	3	4.8882	.15240	.08799	4.5097	5.2668	4.73	5.04
	Okobaba	3	4.8463	.08005	.04622	4.6475	5.0452	4.78	4.94
	Iddo	3	4.9324	.02674	.01544	4.8659	4.9988	4.90	4.96
GPx_gills	Ajah	3	4.9765	.00000	.00000	4.9765	4.9765	4.98	4.98
	Ikoyi	3	4.9125	.05054	.02918	4.7869	5.0381	4.86	4.96
	Mid-lagoon	3	4.6059	.39612	.22870	3.6219	5.5899	4.15	4.86
	Total	18	4.8603	.19418	.04577	4.7637	4.9569	4.15	5.04
	Makoko	3	4.8893	.14241	.08222	4.5356	5.2431	4.73	4.98
	Okobaba	3	4.9743	.01910	.01103	4.9268	5.0217	4.96	5.00
	Iddo	3	5.0294	.06618	.03821	4.8650	5.1938	4.96	5.10
GPx_gonads	Ajah	3	4.9368	.18339	.10588	4.4812	5.3923	4.83	5.15
	Ikoyi	3	4.9555	.06032	.03483	4.8057	5.1054	4.89	5.01
	Mid-lagoon	3	4.7537	.19868	.11471	4.2601	5.2472	4.64	4.98
	Total	18	4.9232	.14112	.03326	4.8530	4.9933	4.64	5.15
	Makoko	3	4.9743	.07259	.04191	4.7939	5.1546	4.92	5.06
	Okobaba	3	4.9213	.13405	.07739	4.5883	5.2543	4.77	5.03
	Iddo	3	4.9213	.01665	.00962	4.8800	4.9627	4.90	4.94
GPx_muscle	Ajah	3	4.9897	.02386	.01378	4.9304	5.0490	4.97	5.02
	Ikoyi	3	4.9831	.02292	.01324	4.9261	5.0400	4.96	5.00
	Mid-lagoon	3	4.8827	.18305	.10568	4.4280	5.3374	4.67	5.00
	Total	18	4.9454	.09208	.02170	4.8996	4.9912	4.67	5.06
	Makoko	3	.1284	.00731	.00422	.1102	.1465	.12	.13
	Okobaba	3	.1299	.00912	.00527	.1072	.1525	.12	.14
	Iddo	3	.1406	.02152	.01242	.0871	.1940	.13	.17
CAT_hep	Ajah	3	.3696	.24588	.14196	-.2412	.9804	.13	.62
	Ikoyi	3	.1288	.00731	.00422	.1107	.1470	.12	.14
	Mid-lagoon	3	.1556	.02924	.01688	.0830	.2283	.13	.19
	Total	18	.1755	.12396	.02922	.1138	.2371	.12	.62
	Makoko	3	4.8284	1.67160	.96510	.6759	8.9809	3.73	6.75
	Okobaba	3	5.8250	1.08254	.62500	3.1358	8.5141	4.78	6.94
	Iddo	3	4.4020	.94196	.54384	2.0620	6.7419	3.56	5.42
CAT_gills	Ajah	3	5.4855	2.78912	1.61030	-1.4431	12.4140	3.58	8.69
	Ikoyi	3	3.8191	1.96786	1.13614	-1.0693	8.7076	1.56	5.15
	Mid-lagoon	3	5.7963	1.66269	.95996	1.6659	9.9266	3.88	6.91
	Total	18	5.0260	1.68946	.39821	4.1859	5.8662	1.56	8.69

CAT_gonads	Makoko	3	8.1368	1.11324	.64273	5.3713	10.9022	7.23	9.38
	Okobaba	3	8.8935	5.35257	3.09031	-4.4031	22.1900	4.14	14.69
	Iddo	3	10.3008	7.28930	4.20848	-7.8069	28.4084	2.10	16.06
	Ajah	3	4.3886	1.96729	1.13581	-.4984	9.2756	2.90	6.62
	Ikoyi	3	5.7566	2.89423	1.67098	-1.4331	12.9462	2.96	8.74
	Mid-lagoon	3	6.7436	3.18310	1.83777	-1.1636	14.6509	3.56	9.92
	Total	18	7.3700	4.06529	.95820	5.3483	9.3916	2.10	16.06
CAT_muscle	Makoko	3	5.4916	1.47604	.85219	1.8249	9.1583	4.12	7.05
	Okobaba	3	3.6324	1.06168	.61296	.9951	6.2698	2.72	4.80
	Iddo	3	5.3967	1.92971	1.11412	.6030	10.1903	3.19	6.78
	Ajah	3	4.1213	1.67832	.96898	-.0479	8.2905	2.37	5.71
	Ikoyi	3	6.0895	3.85133	2.22357	-3.4777	15.6568	3.55	10.52
	Mid-lagoon	3	3.5400	.72997	.42145	1.7267	5.3533	2.70	4.03
	Total	18	4.7119	2.00015	.47144	3.7173	5.7066	2.37	10.52
SOD_hep	Makoko	3	.0001	.00005	.00003	.0000	.0002	.00	.00
	Okobaba	3	.0001	.00006	.00003	.0000	.0003	.00	.00
	Iddo	3	.0001	.00003	.00002	.0000	.0002	.00	.00
	Ajah	3	.0001	.00002	.00001	.0000	.0001	.00	.00
	Ikoyi	3	.0001	.00006	.00004	.0000	.0003	.00	.00
	Mid-lagoon	3	.0001	.00002	.00001	.0000	.0001	.00	.00
	Total	18	.0001	.00004	.00001	.0001	.0001	.00	.00
SOD_gill	Makoko	3	.5523	.29933	.17282	-.1913	1.2959	.37	.90
	Okobaba	3	.6630	.36564	.21110	-.2453	1.5713	.45	1.09
	Iddo	3	.4540	.07240	.04180	.2742	.6339	.37	.51
	Ajah	3	.4750	.28137	.16245	-.2240	1.1739	.29	.80
	Ikoyi	3	.3785	.19455	.11232	-.1047	.8618	.15	.49
	Mid-lagoon	3	.5276	.23156	.13369	-.0476	1.1028	.38	.79
	Total	18	.5084	.23511	.05542	.3915	.6253	.15	1.09
SOD_gonad	Makoko	3	.4648	.23090	.13331	-.1088	1.0384	.29	.73
	Okobaba	3	.7272	.52875	.30527	-.5863	2.0407	.31	1.32
	Iddo	3	.5063	.37696	.21764	-.4302	1.4427	.10	.84
	Ajah	3	.3175	.12155	.07018	.0155	.6194	.21	.45
	Ikoyi	3	.3559	.11299	.06524	.0753	.6366	.26	.48
	Mid-lagoon	3	.2779	.04451	.02570	.1674	.3885	.25	.33
	Total	18	.4416	.28865	.06804	.2981	.5852	.10	1.32
SOD_muscle	Makoko	3	.6049	.18197	.10506	.1529	1.0569	.40	.73
	Okobaba	3	.3869	.18938	.10934	-.0835	.8574	.24	.60
	Iddo	3	.5036	.16002	.09239	.1061	.9011	.32	.63
	Ajah	3	.4683	.12499	.07216	.1578	.7788	.35	.60
	Ikoyi	3	.7243	.42160	.24341	-.3230	1.7717	.37	1.19
	Mid-lagoon	3	.4000	.07522	.04343	.2132	.5869	.35	.49
	Total	18	.5147	.22212	.05235	.4042	.6251	.24	1.19
Protein_hep	Makoko	3	93.2459	20.63563	11.91398	41.9842	144.5076	74.97	115.63
	Okobaba	3	107.4077	16.91916	9.76828	65.3782	149.4372	90.80	124.62
	Iddo	3	75.9740	31.81983	18.37119	-3.0708	155.0189	40.15	100.97
	Ajah	3	65.9219	5.92656	3.42170	51.1995	80.6443	59.15	70.14
	Ikoyi	3	68.5877	48.97569	28.27613	-53.0747	190.2500	39.15	125.12
	Mid-lagoon	3	90.1914	43.86056	25.32291	-18.7643	199.1470	40.49	123.46
	Total	18	83.5548	30.73811	7.24504	68.2691	98.8405	39.15	125.12
Protein_gill	Makoko	3	11.1073	2.14230	1.23686	5.7855	16.4291	8.66	12.66
	Okobaba	3	9.3301	1.49949	.86573	5.6052	13.0551	7.83	10.83
	Iddo	3	10.7741	1.67717	.96831	6.6078	14.9404	9.00	12.33
	Ajah	3	14.1618	4.04350	2.33452	4.1172	24.2064	9.50	16.66
	Ikoyi	3	19.4378	9.40472	5.42982	-3.9248	42.8004	11.50	29.82
	Mid-lagoon	3	10.4409	2.83889	1.63903	3.3887	17.4931	7.16	12.16
	Total	18	12.5420	5.17510	1.21978	9.9685	15.1155	7.16	29.82

	Makoko	3	17.7717	5.59157	3.22829	3.8815	31.6619	12.00	23.16
	Okobaba	3	11.1073	4.85556	2.80336	-.9546	23.1692	6.00	15.66
	Iddo	3	29.1012	34.57315	19.96082	-56.7833	114.9856	7.50	68.98
Protein_gonad	Ajah	3	27.8238	6.21836	3.59017	12.3766	43.2711	22.49	34.65
	Ikoyi	3	21.3816	2.93504	1.69455	14.0905	28.6726	17.99	23.16
	Mid-lagoon	3	29.6565	9.25846	5.34538	6.6572	52.6558	23.66	40.32
	Total	18	22.8070	14.54041	3.42721	15.5762	30.0378	6.00	68.98
	Makoko	3	10.6630	2.45431	1.41700	4.5662	16.7599	8.00	12.83
	Okobaba	3	20.1598	13.42527	7.75108	-13.1904	53.5100	8.83	34.99
	Iddo	3	13.2732	2.92557	1.68908	6.0057	20.5408	10.50	16.33
Protein_muscle	Ajah	3	13.8841	2.05635	1.18724	8.7759	18.9924	12.16	16.16
	Ikoyi	3	10.6630	3.98124	2.29857	.7731	20.5530	7.16	14.99
	Mid-lagoon	3	16.9942	3.67676	2.12278	7.8606	26.1278	13.50	20.83
	Total	18	14.2729	6.25279	1.47380	11.1635	17.3823	7.16	34.99

Superoxide Dismutase (SOD)
Oneway

ANOVA

		Sum of Squares	df	Mean Square	F	Sig.
HEPATOPANCRESE.DRY	Between Groups	.000	5	.000	.	.
	Within Groups	.000	0	.		
	Total	.000	5			
HEPATOPANCREASE.WET	Between Groups	.000	5	.000	.	.
	Within Groups	.000	0	.		
	Total	.000	5			
GLL.DRY	Between Groups	.329	5	.066	.	.
	Within Groups	.000	0	.		
	Total	.329	5			
GLL.WET	Between Groups	.397	5	.079	.	.
	Within Groups	.000	0	.		
	Total	.397	5			
GONAD.DRY	Between Groups	.126	5	.025	.	.
	Within Groups	.000	0	.		
	Total	.126	5			
GONAD.WET	Between Groups	.477	5	.095	.	.
	Within Groups	.000	0	.		
	Total	.477	5			
MUSCLE.DRY	Between Groups	.303	5	.061	.	.
	Within Groups	.000	0	.		
	Total	.303	5			
MUSCLE.WET	Between Groups	.258	5	.052	.	.
	Within Groups	.000	0	.		
	Total	.258	5			

Catalase (CAT)

ANOVA

		Sum of Squares	df	Mean Square	F	Sig.
HEPATOPANCRESE.DRY	Between Groups	.151	5	.030	.	.
	Within Groups	.000	0	.		
	Total	.151	5			
HEPATOPANCREASE.WET	Between Groups	.275	5	.055	.	.
	Within Groups	.000	0	.		
	Total	.275	5			
GLL.DRY	Between Groups	28.764	5	5.753	.	.
	Within Groups	.000	0	.		
	Total	28.764	5			
GLL.WET	Between Groups	65.221	5	13.044	.	.
	Within Groups	.000	0	.		
	Total	65.221	5			
GONAD.DRY	Between Groups	31.255	5	6.251	.	.
	Within Groups	.000	0	.		
	Total	31.255	5			
GONAD.WET	Between Groups	84.779	5	16.956	.	.
	Within Groups	.000	0	.		
	Total	84.779	5			
MUSCLE.DRY	Between Groups	46.789	5	9.358	.	.
	Within Groups	.000	0	.		
	Total	46.789	5			
MUSCLE.WET	Between Groups	49.769	5	9.954	.	.
	Within Groups	.000	0	.		
	Total	49.769	5			

Glutathione Peroxidase (GPx)

ANOVA

		Sum of Squares	df	Mean Square	F	Sig.
HEPATOPANCRESE.DRY	Between Groups	.399	5	.080	.	.
	Within Groups	.000	0	.		
	Total	.399	5			
HEPATOPANCREASE.WET	Between Groups	.041	5	.008	.	.
	Within Groups	.000	0	.		
	Total	.041	5			
GLL.DRY	Between Groups	.034	5	.007	.	.
	Within Groups	.000	0	.		
	Total	.034	5			
GLL.WET	Between Groups	.231	5	.046	.	.
	Within Groups	.000	0	.		
	Total	.231	5			
GONAD.DRY	Between Groups	.181	5	.036	.	.
	Within Groups	.000	0	.		
	Total	.181	5			
GONAD.WET	Between Groups	.142	5	.028	.	.
	Within Groups	.000	0	.		
	Total	.142	5			
MUSCLE.DRY	Between Groups	.034	5	.007	.	.
	Within Groups	.000	0	.		
	Total	.034	5			
MUSCLE.WET	Between Groups	.009	5	.002	.	.
	Within Groups	.000	0	.		
	Total	.009	5			

Reduced Glutathione (GSH)

ANOVA

		Sum of Squares	df	Mean Square	F	Sig.
HEPATOPANCRESE.DRY	Between Groups	.515	5	.103	.	.
	Within Groups	.000	0	.		
	Total	.515	5			
HEPATOPANCREASE.WET	Between Groups	.642	5	.128	.	.
	Within Groups	.000	0	.		
	Total	.642	5			
GLL.DRY	Between Groups	.034	5	.007	.	.
	Within Groups	.000	0	.		
	Total	.034	5			
GLL.WET	Between Groups	.252	5	.050	.	.
	Within Groups	.000	0	.		
	Total	.252	5			
GONAD.DRY	Between Groups	.223	5	.045	.	.
	Within Groups	.000	0	.		
	Total	.223	5			
GONAD.WET	Between Groups	.168	5	.034	.	.
	Within Groups	.000	0	.		
	Total	.168	5			
MUSCLE.DRY	Between Groups	.020	5	.004	.	.
	Within Groups	.000	0	.		
	Total	.020	5			
MUSCLE.WET	Between Groups	.303	5	.061	.	.
	Within Groups	.000	0	.		
	Total	.303	5			