CONCENTRATION AND DISTRIBUTION OF PESTICIDE METABOLITES IN THE BLOOD AND URINE OF SOME SELECTED AGROCHEMICALS RETAILERS IN TARABA STATE, NIGERIA

BY

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CHAPTER ONE
INTRODUCTION

1.0 GENERAL INTRODUCTION

Food is a basic surrogate for man's existence, as man multiply to replenish and subdue the earth, the demand for food increase in order to meet the growing population. However, the need to cultivate lands and domesticate animal for food becomes paramount and also faced with the challenge of insect infestation, rodents, weed, fungi etc that plays a vital role in diminishing crop productivity and yield. In an attempt to combat the army of pest and weeds that retard crop yield, pesticides were developed to help provide a plentiful supply of healthy and affordable year round food to meet the demand of consumers.

Many pesticides were developed under the secrecy of wartime and with military purpose in mind in World War II (Cox and Surgan, 2006). As the agricultural production system moves more and more from subsistence to market oriented large scale farming, a concomitant increase in pesticide usage seem inevitable (Sosan *et al*., 2008). Agricultural workers and families are exposed to pesticide primarily through, storage, mixing of chemicals, loading into dispensers, application, clean-up and disposal of empty chemical containers (Reynolds *et al*., 2005). Pesticides primarily are designed to kill living organism and are toxic and also deliberately released to the environment.

These twin properties make pesticide unique and constitute threat to man's health and environment (Cox *et al*., 2006). Pesticides are toxic, or poisonous to be effective against the pest they are intended to control, Centre for Disease control and Prevention (CDC, 2005). Because pesticides are toxic, they are also potentially hazardous to human and animals.

Toxicity of pesticide is a measure of the capacity of a pesticide to cause injury; it is a property of the chemical itself. Pesticide means any chemical substance or mixture of substances intended for preventing, destroying, repelling or mitigating the effect of any pest of plants and animal and shall include herbicides, insecticide rodenticides, fungicides, molluscides, nematucides, repellants, attractants, insect

growth regulators used in agriculture, public health, horticulture, food storage or a chemical substance use for similar purpose (NAFDAC Decree 2004).

Occupational exposure may be via inhalation, dermal or oral route. Several studies have shown that children have higher concentration because of their body weight (Quandi *et al.,* 2005). Pesticide storage, handling and usage are fraught with problems of undesirable side effects and food chain involvement. A National survey by the US Geological survey found pesticide residue in every stream monitored (Gilliom *et al*., 2007). Pesticide residues are present in more than 70 % of fruits and vegetable, > 60 % of wheat samples and 99 % of milk samples analyzed United State Department of Agriculture (USDA, 2006). Pesticides are found in the bodies of nearly all U.S adult and children (CDC, 2005).

Monitoring studies indicated that pesticide exposures are wider spread in the U.S population including pregnant women and children (Barr *et al*., 2004; Berkowrtz *et al*., 2003, Bradman *et al*., 2005; CDC 2006; Whyatt *el at*., 2003).There is evidence that fetuses can be exposed to pesticide, pesticides pass through the blood- brain barrier and placenta and have also been found in amniotic fluid (Bradman and whitaker 2006). Children of farm workers can be exposed through multiple pathways including agricultural take home and drift as well as residential application. Biological monitoring of exposure can be carried out by determine intact compounds or their metabolites in the blood serum, plasma or urine (Aprea *et al*., 2002). The whole range of workers associated with pesticide – treatment of crops or premises are also liable to exposure as workers, their families and segments of the population during disease vector control procedures that have a common mechanism of toxicity (USEPA, 2006b).

Pesticides in ambient air result in inhalation exposure either when a person is out door or breaths contaminated air or when contaminants in the air are transported indoors which may results during storage or aerial sprays of dispersions pesticide to farmland. Acute health effects of organophosphorus (Op) pesticide on coffee farm workers in 1991-1992 in Tanzania ware reported to provide a basis for concern over farm workers being overexposed during application. Farm workers children, like their parents, are exposed to acute health effect (Strong *et al*., 2004; Quandt *et al.,* 2005; Thompson *et al*., 2003).

According to industry estimate, the pesticide use has high growth potential in Nigeria as the use of agricultural pesticide is markedly low at 0.25kg/ha as against 0.54 kg/ha in India, 3.7 kg/ha in USA and 2.7 kg/ha in China (Mathur *et al*., 2005). Notwithstanding the fact that the overall consumption in Nigeria is low than that used in the developed countries of the world, there is increased wider spread of pesticide poisoning among workers, consumers of Agricultural produce preserved with pesticide e.g grains, beans e.t.c. Farm workers children like their parent are exposed to pesticide (Arcury *et al*., 2005; 2006; Coronado *et al*., 2006; Lambert *et al.,* 2005 and Thompson *et al*., 2003). Few studies had been documented in developing countries such as Pakistan; the presence of pesticide residues in the blood of Karachi people (Azmi *et al.,* 2005), Azmi *et al.,* (2006) reported the effect of pesticide residues on health and different enzyme level in the blood of farm workers from Karachi, Pakistan. Cruz *et al.,* 2003 also reported the presence of pesticide residue in an urban and two rural populations in Portugal.

In Nigeria, apart from the work of Ivbijaro (1977) which evaluated insecticide residue in Kalanuts, Sosan *et al.* (2008) from Ile-Ife, that examined the insecticide residue in the blood serum and domestic water source of cacao farmers in south-western Nigerian and that of Hotton *et al.,* 2010a; 2010b and 2011 that monitors cholinesterase inhibition among retailers of agrochemicals, evaluated pesticide urinary metabolite and deposit on retail outlets and analysed hexachlorocyclohexane isomers in the blood of agrochemicals retailers in Taraba Nigeria respectively, there seems to be no evaluation of pesticides residue in the country. Greater percentage of the pesticide retail outlets and corner shops in Northern Nigeria are stores with a chair and a table for the retailers, with no cross ventilation, good storage conditions and safety measures. Some are hawked from one village market to another (Hotton *et al*., 2010a).

1.1 **Regulation and Monitoring of Pesticide in Nigeria**

The National Agency for Food and Drug Administration and Control (NAFDAC), was established by Decree 15 of 1993 as amended by Decree 19 of 1999 and now Act Cap N1 Laws of the Federation of Nigeria (LFN) 2004, to regulate and control the manufacture, importation, exportation, distribution, advertisement, sale and use of food, drugs, cosmetics, chemicals, medical devices and packaged water (known as regulated products). The power to regulate and monitor narcotic drugs and psychotropic substances and precursor chemical (pesticide) are conferred under the NAFDAC Act Cap N1 Laws of the Federation of Nigeria (LFN) 2004 sections 5a.5c Pesticide Registration Regulation 2005 and various articles of UN conventions of 1961, 1971 and 1988. Precisely Article 43 of 1961 Convention Article 15 of 1971 Convention and Article 12 paragraphs 9 of 1988 Convention states that each country should establish and maintain a system to monitor international trade in order to facilitate the identification of suspicious transaction.

NAFDAC undertake measures to ensure that the use of narcotic drugs and psychotropic substances are limited to medical and scientific purposes. The Agency grant authorizations for the import and export of narcotic drugs and psychotropic substances and other controlled, conduct the monitoring of pesticide in close collaboration with manufacturers, importers, exporters, wholesale and retailers. Before pesticide are approved they are rigorously assessed to ensure they do not pose an unacceptable risk to users of the pesticide, the environment, non-target animal and that any pesticide residues left in food will not be harmful to consumers. Pre import inspection of warehouses of importers of chemicals before import permit is issued. Verification of LPO's issued by legitimate end users to marketers of precursor chemicals before permit to import is issued .Cross checking the disposal records of marketers before issuance of permits for restricted and precursor chemicals to prevent diversion and stockpiling .Routine monitoring inspection of the warehouses of marketers (importers and retailers) and end users that were issued permits to ensure proper storage, handling and disposal (NAFDAC Act, 2004).

Pesticide residues in the food chain, water and water based product are also monitored to check they are within legal and safe limit. Before Marketing Company obtain permit from the Agency to import precursor chemicals, the company is required to submit local purchase order (LPO) among other documents from end user companies.This is to ensure that distribution outlets are known. NAFDAC by the enabling decree is the regulatory authority in the country with the mandate to regulate the importation, exportation, manufacture, advertisement distribution and sales of food, drugs cosmetics, medical devices, chemical (pesticide) and packaged water. Despite the stringent regulation, pesticide poisoning and exposure has been a threat to public health in Nigeria and a concern to Regulatory Agencies like NAFDAC, empowered by law to safe guard the health of the Nation. The wider spread, unethical sales, storage, applications, acute toxicity and neurotoxic properties needed to be investigated.

1.2 Statement of the Problem.

After the flagging off, of Nigerian Green Revolution, the consumption of pesticide and fertilizer in Nigeria has increased several thousand folds from 30 metric ton (MT) in 1977 to 20,000MT in 2003 – 2005. These however, led to the indiscriminate proliferation and unethical sales and hawking of pesticide in open markets, residential areas, stores, streets and villages.

To this end, there were several uninvestigated cases of threat to public health constituted by pesticide poisoning from employees assigned to retail pesticide in rented shops whose design is to accommodate the pesticide and their sales desk not minding the high temperature profile of the Northern Region and the volatility of the pesticide. Despite the common use of pesticide in residential environment and in agriculture, few studies have measured children exposure levels, some have focused on acute poisoning incident with known or probable sources (Fenske *et al.*,2005), others have examined low level chronic pesticide exposure in agricultural communities (Quandt *et al.,* 2006).

There are few published studies identified to date that examines pesticide exposure among retailers of agrochemicals including that of Hotton *et al.,* 2010a; 2010b; 2011.These indiscriminate retailing of pesticide in unventilated stores, open-markets residential areas, the incessant cases of pesticide poisoning and it prevalence health hazard posed by handling, storage and sales of pesticide rekindle the quest for this research work.

1.3 Aim and Objectives

The aim is to assess the health hazard associated with the exposure of agrochemical retailers to agrochemicals in an indoor environment. The specific objectives are to:

(i) Identify and evaluate organophosphurus pesticide metabolites in the urine of agrochemical retailers.

(ii) Assess the concentration and distribution of pesticide deposit in various retail environments.

(iii) Assess the levels of Organochlorine metabolites (DDT metabolites and HCH isomers) in the blood of agrochemical retailers

(iv) Monitor the level of cholinesterase inhibition and relate Common symptoms prevalence among the retailers with inhibition.

(v) Develop a regulatory control measure on the reduction of pesticide exposure pathways using activated carbon made from coconut shell and born charcoal as Absorbent.

1.4 Significance of the Study

The information obtained from this research is expected to;

(i) Provide some benchmark information about the accumulation of pesticide in body system associated with retailing which may guide Government, Regulatory bodies and policy makers in evaluating risk assessment.

(ii) Foster understanding of the nature and net impact of pesticide drift and distribution in various retail outlets.

(ii) Provide an insight to the possible mitigating measure to minimize risk of pesticide exposure using locally sourced absorbent.

1.5 Scope of the Study.

The scope of this research work covers the following;

(i) 50 selected pesticide retail stores in the Northern and Southern Senatorial districts of Taraba, Nigeria were investigated.

(ii) Pesticide metabolites analysis was limited to blood, on the spot urine sample and wipe surface sampling using gas chromatograph.

(iii) The analysis of the local absorbent for adsorption was limited to the surface characterization and concentration of the pesticide residue on each absorbent.

(iv) The preliminary survey questionnaires covered fifty (50) retailers of agrochemicals and fifty (50) retailers of retailers of animal feed as control.

CHAPTER TWO
LITERATURE REVIEW

2.0 INTRODUCTION

Across the globe, pesticides have been found in human blood, urine, breast milk, semen, infant and umbilical cord blood (Mathur *el al.,* 2005). In recent years, there have been increasing concern regarding the widespread and use of pesticide and potential impacts on public health (Rothlein *el al.,* 2006). Their intensive use throughout decades and their high persistence, accumulation in food chains and in the human body especially in lipid-rich tissues has raised the interest in knowing the extent of their spread leading to investigation of the magnitude of their residues in all compound of the human environment .Insecticides, herbicides and fungicides constitute the major pesticides used in Nigeria.

According to Sosan, *et al* (2008), pesticide importation raised steadily from about 13 million dollars in 2001 to 28 million dollars in 2003 with insecticides accounting for about 32 % of the imports. The bulk of the pesticides are used in respect of agricultural production for the control of noxious weeds, insect, pest and diseases of crop. As the agricultural production system moves more and more from subsistence to market-oriented large scale farming, a concomitant increase in pesticide usage seems inevitable. Besides, pesticides are used in urban and sub-urban dwellings rather extensively as aerosols for the control of pest (e.g ants, cockroaches, mosquitoes and other nuisance flies). (Sosan *et al.,* 2008).

In tropical countries like Nigeria, the spread and proliferation of the sales of pesticide in traditional local markets and small – scale retailing in an indoor rented stores located in open markets, streets and residential areas have continue to dominate the Northeastern Nigeria retail section (Hotton *et al.,* 2010).

Pesticide, storage, handling and usage are fraught with problems of undesirable side effects and food chain involvement. A worker exposed to a chemical receives a dose of that chemical only if it is absorbed into the body. Absorption can occur after dermal contact, inhalation, ingestion, or from a combination of those

routes. The extent of absorption from an exposure and the rate of absorption depend on the properties of the chemical (especially its solubility in lipids and water) and the route of exposure. Once absorbed, a chemical is distributed and partitions into various tissues due to tissue variations in pH, permeability, etc. Highly water-soluble chemicals may be distributed throughout the total body water, while more lipophilic substances may concentrate in the body fat or other lipid rich tissues, such as the brain. The loss of chemical from the body can loosely be defined as elimination, which depends on metabolism and excretion.

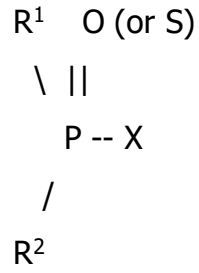
Chemicals may be eliminated by numerous routes, including fecal, urinary, exhalation, perspiration, and lactation. A chemical can be excreted from the body without metabolism, in which case the parent compounds may be detectable in the urine, breath, or fecal material. In other cases, the chemical may be metabolized through oxidation, reduction, hydrolysis, or a combination of these processes, often followed by conjugation with an endogenous substrate. Conjugation of a chemical or metabolite is a pathway for excretion.

The kidney is the major organ of excretion and is the primary route for water-soluble substances. These substances enter the urine by either glomerular filtration, tubular secretion, or sometimes both mechanisms. Biological monitoring has the potential to assess worker exposure to industrial chemicals by all routes, including inhalation, skin absorption, and ingestion. Often, most of the toxicological and pharmacological information available is from experimental animals and, thus, not always directly applicable to humans indicated that pesticide exposures are wider spread in the U.S population including pregnant women and children (Barr *et al.,* 2004; Berkowrtz *et al.,* 2003; Bradman *et al.,* 2005; Centre for Disease control and prevention (CDC) 2006; Whyatt *el. at*., 2003). Pesticides are multifaceted in use, chemical composition, structural diversity, application and in grouping. Pesticide can be group into organophosphate, organochlorine, carbamate, and natural and synthetic pyrethroids with different varying properties among member groups.

2.1 TYPES OF PESTICIDE

2.1.1 Organophosphorus Pesticides

A class of pesticides derived from phosphoric acid. They tend to have high acute toxicity to vertebrates. Organophosphate insecticides represent one of the most widely used classes of pesticides with high potential for human exposure in both rural and residential environment (Lu *et al.,* 2001). Although readily metabolized by vertebrates, some metabolic products are more toxic than the parent compound. Organophosphorous insecticides represent one group of pesticides that is widely used and has been shown to have toxic effects in human and animals. Various structures of organophosphorus insecticides are illustrated in Table 2.1 The compounds are normally esters, amides, or thiol derivatives of phosphoric or phosphonic acid:



Where R^1 and R^2 are usually simple alkyl or aryl groups, both of which may be bonded directly to phosphorus (in phosphonates), or linked via -O-, or -S- (in phosphates), or R^1 may be bonded directly and R^2 bonded via one of the above groups (phosphonates). In phosphoramidates, carbon is linked to phosphorus through an -NH group. The group X can be any one of a wide variety of substituted and branched aliphatic, aromatic, or a heterocyclic group linked to phosphorus via a bond of some liability (usually -O- or -S-) and is referred to as the leaving group. The double-bonded atom may be oxygen or sulfur and related compounds would, for example, be called phosphates or phosphorothioates (the nomenclature "thiophosphate" or "thionophosphate" is now less used).

The P=O form of a thioate ester may be referred to as the oxon, and this is often incorporated in the trivial name (e.g., parathion is the parent P=S compound

of paraoxon). The variations in the phosphorus group for the insecticides that have been developed are shown in Table 2.1 together with the common or other names for some pesticides falling into this classification. Organophosphates are widely employed both in natural and synthetic applications because of the ease with which organic groups can be linked together.

Table2.1: Evaluation in the chemical structure of Organophosphorus pesticides

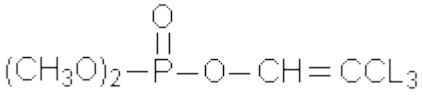
Types of phosphorus group	structure	Common or other name
Phosphate	$(R\text{-}O)_2 – P\text{-}O\text{-}X$	chlorfenvinphos, dichlorvos, dicrotophos, mevinphos, monocrotophos, phosphamidon, TEPP, tetrachlorvinphos, triazophos
O-alkyl phosphorothioate	$(R\text{-}O)_2 – P\text{-}S\text{-}X$	amiton, demeton-S-methyl, omethoate, oxydenmetonmethyl, phoxim, vamidothion
	$(R\text{-}O)_2 – P\text{-}O\text{-}X$	azothoate, bromophos, bromophos-ethyl, chlorpyriphos, chlorpyriphos-methyl, coumaphos, diazinon, dichlofenthion, fenchlorphos, fenitrothion, fenthion, iodofenphos, parathion, parathion-methyl, pyrazophos, pyrimiphos-ethyl, pyrimiphos-methyl, sulfotep, temephos, thionazin
Phosphorodithioate	$(R\text{-}O)_2 – P\text{-}S\text{-}X$	

These compounds must be reasonably stable at neutral pH, since many are formulated as concentrated in oil, in water-miscible solvent. Such as ethylene glycol monomethyl ether, or are absorbed on to inert granules for application directly or after dispersion in water. However, nearly all are rapidly hydrolysed by alkali and many are also instable at pH levels below 2.

2.1.1.1: Dichlorvos Pesticide

Dichlorvos is organophosphates pesticide used to control household, public health, and stored product insects. It is effective against mushroom flies, aphids, spider mites, caterpillars, thrips, and white flies in greenhouse, outdoor fruit, and vegetable crops (Falcon *el al*. 2004). It is a light-yellow or yellowish brown transparent and volatile liquid with a slight aromatic odour.

Chemical name: 2,2-dichlorovinyl dimethyl phosphate

structural formula: 

2.1.1.1.1 Toxicological effects

Toxicity: The amount of a chemical that is lethal to one-half (50%) of experimental animals fed the material is referred to as its acute oral lethal dose fifty, or LD_{50}. Acute oral LD_{50} to rats is 56-108mg/kg, acute dermal LD_{50} to rats is 75-210 mg/kg. These chemicals act by interfering with the activities of cholinesterase, an enzyme that is essential for the proper working of the nervous systems of both humans and insects.

Acute Toxicity: Dichlorvos is highly toxic by inhalation, dermal absorption and ingestion. Because dichlorvos is volatile, inhalation is the most common route of exposure. As with all organophosphates, dichlorvos is readily absorbed through the skin. Skin which has come in contact with this material should be washed immediately with soap and water and all contaminated clothing should be removed. Acute illness from dichlorvos is limited to the effects of cholinesterase inhibition. Compared to poisoning by other organophosphates, dichlorvos causes a more rapid onset of symptoms, which is often followed by a similarly rapid recovery (Fabra *el al.,* 2005). This occurs because dichlorvos is rapidly metabolized and eliminated from the body. Persons with reduced pulmonary (lung) function, convulsive disorders, liver disorders, or recent exposure to cholinesterase inhibitors will be at increased risk from exposure to dichlorvos.

Alcoholic beverages may enhance the toxic effects of dichlorvos. High environmental temperatures or exposure of dichlorvos to visible or UV light may enhance its toxicity. Dichlorvos is mildly irritating to skin. Concentrates of dichlorvos may cause burning sensations, or actual burns. Dichlorvos can be very toxic if it is not immediately washed off, but instead left on the skin long enough for it to become absorbed through the skin and into the bloodstream. The organophosphate insecticides are cholinesterase inhibitors. They are highly toxic by all routes of exposure. When inhaled, the first effects are usually respiratory and may include bloody or runny nose, coughing, chest discomfort, difficult or short breath, and wheezing due to constriction or excess fluid in the bronchial tubes.

Skin contact with organophosphates may cause localized sweating and involuntary muscle contractions. Eye contact will cause pain, bleeding, tears, pupil constriction, and blurred vision. Following exposure by any route, other systemic effects may begin within a few minutes or be delayed for up to 12 hours. These may include pallor, nausea, vomiting, diarrhea, abdominal cramps, headache, dizziness, eye pain, blurred vision, constriction or dilation of the eye pupils, tears, salivation, sweating, and confusion. (Torres *el al.,* 2007).Severe poisoning will affect the central nervous system, producing in coordination, slurred speech, loss of reflexes, weakness, fatigue, involuntary muscle contractions, twitching, tremors of the tongue or eyelids, and eventually paralysis of the body extremities and the respiratory muscles. In severe cases there may also be involuntary defecation or urination, psychosis, irregular heartbeats, unconsciousness, convulsions and coma. Death may be caused by respiratory failure or cardiac arrest International Programme on Chemical Safety (IPCS, 1999).

Some organophosphates may cause delayed symptoms beginning 1 to 4 weeks after an acute exposure which may or may not have produced immediate symptoms. In such cases, numbness, tingling, weakness and cramping may appear in the lower limbs and progress to in-coordination and paralysis. Improvement may occur over months or years, but some residual impairment will remain (WHO, 2006). Dichlorvos is very volatile, meaning that it readily forms vapours which may be

inhaled. Inhalation is the most common way to be exposed to dichlorvos. Low, repeated doses may be non-toxic. High doses of dichlorvos may be very toxic, especially if inhalation exposure is continuous. Dichlorvos produces irritating gases, such as phosphorous and chlorine oxides, when heated.

Eye protection should be worn when handling dichlorvos. Application of 1.67 mg/kg in rabbits' eyes produced mild redness and swelling, but no injury to the cornea. Dichlorvos may cause eye burns. Organophosphates cause the pupils to constrict (pin point pupils). The amount of a chemical that is lethal to one-half (50%) of experimental animals fed the material is referred to as its acute oral lethal dose fifty, or LD_{50}. The oral LD_{50} for dichlorvos in mice is 61 to 175 mg/kg, 100 to 1090 mg/kg in dogs, 15 mg/kg in chickens, 25 to 80 mg/kg in rats, 157 mg/kg in pigs, and 11 to 12.5 mg/kg in rabbits. The dermal LD_{50} for dichlorvos in rats is 70.4 to 250 mg/kg, 206 mg/kg in mice, and 107 mg/kg in rabbits (Torres *el al* (2007).

The lethal concentration fifty, or LC_{50}, is that concentration of a chemical in air or water that kills half of the experimental animals exposed to it for a set time period. Chronic toxicity, feeding studies indicate that a dosage of dichlorvos very much larger than doses which inhibit cholinesterase are needed to produce illness. Rats tolerated dietary doses as high as 62.5 mg/kg/day for 90 days with no visible signs of illness, while a dietary level of 0.25 mg/kg/day for only 4 days produced a reduction in cholinesterase levels. Rats were exposed to air concentrations of 0, 0.05, 0.5 and 5 mg/m^3 of dichlorvos over a 5 week period. Rats in the 0.5 and 5 mg/kg groups exhibited significantly decreased cholinesterase activity in the plasma, red blood cells, and brain. The NOAEL for this study was 0.05 mg/m^3. In dogs fed dietary doses of 0.0095, 0.016, 0.16, 1.6 or 12.5 mg/kg/day for 2 years, decreased red blood cell cholinesterase activity, increased liver weights and increased liver cell size occurred in the two highest doses tested with a NOAEL of 0.08 mg/kg/day. (WHO, 1992).

Repeated or prolonged exposure to organophosphates may result in the same effects as acute exposure including the delayed symptoms. Other effects reported in workers repeatedly exposed include impaired memory and concentration,

disorientation, severe depressions, irritability, confusion, headache, speech difficulties, delayed reaction times, nightmares, sleepwalking and drowsiness or insomnia. Influenza-like condition with headache, nausea, weakness, loss of appetite, and malaise has also been reported. Dichlorvos can bind to molecules such as DNA. For this reason, there has been extensive testing of dichlorvos for mutagenicity.

Several studies reviewed by United Nation Environmental Protection Agency have shown dichlorvos to be a mutagen. Dichlorvos is reported positive in the Ames mutagenicity assay and in other tests involving bacterial or animal cell cultures. However no evidence of mutagenicity has been found in tests performed on live animals. Its lack of mutagenicity in live animals may be due to rapid metabolism and excretion of dichlorvos.

2.1.1.1.2 Carcinogenic Effects

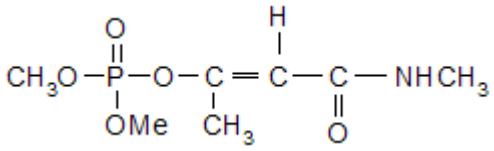
Dichlorvos has been classified as a possible human carcinogen by EPA because of the results of tests on rats and mice (WHO, 1992). When dichlorvos was administered to mice for 5 days per week for 103 weeks at doses of 10 or 20 mg/kg to males and 20 or 40 mg/kg to females, there was an increased incidence of benign tumours in the lining of the stomach at the high dose for both sexes. When rats given daily doses of 0, 4 or 8 mg/kg for five days per week for 103 weeks, there was an increased incidence of benign tumours of the pancreas and of leukaemia in male rats at both doses. At the highest dose, there was also an increased incidence of benign lung tumours in males. In female rats, there was an increase in the incidence of benign tumours of the mammary gland (CDC, 2003).

A few tumours were found in the oesophagus of mice given dichlorvos orally, even though tumours of this kind are normally rare. Dichlorvos primarily affects the nervous system through cholinesterase inhibition, by which there is a deactivation of cholinesterase, an enzyme required for proper nerve functioning. Dichlorvos causes fluid to accumulate in the lungs. Liver enlargement has occurred in pigs maintained for long periods of time on high doses (500 ppm). Dichlorvos caused adverse liver

effects in dogs Cholinesterase inhibition may affect the nervous system. In mice, a single oral dose of 40 micrograms (ug)/kg caused changes in the testes. In male rats, repeated doses caused abnormalities in the tissues of the lungs, heart, thyroid, liver and kidneys. Dichlorvos does not accumulate in body tissues and has not been detected in the milk of cows or rats, even when the animals were given doses high enough to produce symptoms of severe poisoning (Fabra *el al.,* 2005).

2.1.1.2 Monocrotophos

Monocrotophos is an organophosphate insecticide. It is acutely toxic to birds and humans, and for that reason has been banned in the U.S, Nigeria and many other countries. Monocrotophos is rapidly absorbed, metabolized and eliminated by mammals. The organophosphate insecticides are cholinesterase-inhibitors. They are highly toxic by all routes of exposure. When inhaled, the first effects are usually respiratory and may include bloody or runny nose, coughing, chest discomfort, difficult or short breath and wheezing due to constriction or excess fluid in the bronchial tubes. Skin contact with organophosphates may cause localized sweating and involuntary muscle contractions. Eye contact will cause pain, bleeding, tears, pupil constriction and blurred vision. Following exposure by any route, other systemic effects may begin within a few minutes or be delayed for up to 12 hours (Fabra *el al.,* 2005)

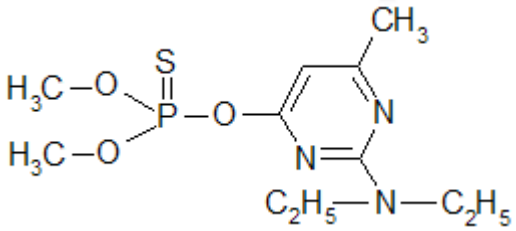
Chemical Name	Dimethyl(E)-1-methyl-2-(methylcarbamoyl)vinyl phosphate
Empirical Formula	$C_7H_{14}NO_5P$
Molecular Weight	223.2
Structural Formula	
Solubility	1kg/kg in water, 700g/kg in acetone, 800g/kg in dichloromethane, 1kg/kg in methanol, 60g/kg in toluene

Acute toxicity

Monocrotophos is classified in group Ib by WHO, as highly hazardous, and has been responsible for deaths resulting from accidental or intentional exposure. It is highly toxic orally, as well by inhalation or absorption through the skin. Early symptoms of poisoning may include excessive sweating, headache, weakness, giddiness, nausea, vomiting, hyper salivation, abdominal cramps, diarrhoea, blurred vision and slurred speech. Inhalation or skin contact may increase the susceptibility to the pesticide without showing immediate symptoms. The acute oral toxicity for rats (LD_{50}) is 23mg/kg (males) and 18 mg/kg (females). The acute dermal toxicity for a rat is 354 mg/kg. Tests on rabbits indicate that it is irritating to eyes and causes reversible corneal opacity and reports on human health indicate eye contact will cause pain, bleeding, tears, pupil constriction and blurred vision.

2.1.1.3 Pirimiphos-methyl

Pirimiphos-methyl (0,2-diethylamino-6-methylpirimidin-4-yl O, O-dimethyl phosphorothioate) is a broad spectrum, non-cumulative organophosphorous pesticide responsible for the hydrolysis of body choline esters including acetylcholine at cholinergic synapses. Pirimiphosmethyl is a cheap pesticide widely used in the world and particularly in Africa to protect food against pests.

Chemical Name	O-(2-diethylamino-6-methylpyrimidin-4-yl) O,O-dimethyl phosphorothioate
Empirical Formula	$C_{11}H_{20}N_3O_3PS$
Molecular Weight	305
Structural Formula	

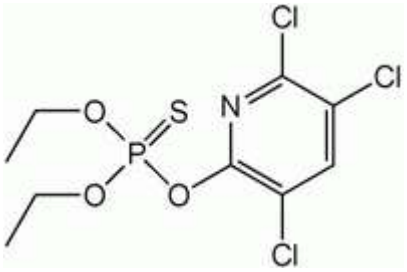
Molecular formula: $C_{11}H_{20}N_3O_3PS$

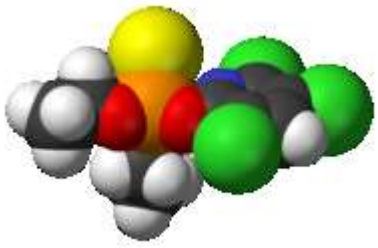
2.1.1.4 Chlorpyrifos

Chlorpyrifos is a crystalline organophosphate insecticide that inhibits acetylcholinesterase and is used to control insect pest. Chlorpyrifos is a neurotoxin and suspected endocrine disruptor, and it has been associated with asthma, (Rauh *et al.,* 2006) reproductive and developmental toxicity, and acute toxicity. For acute effects, the EPA classifies chlorpyrifos as Class II: moderately toxic.

Properties

Molecular formula $C_9H_{11}Cl_3NO_3PS$,Molar mass 350.59 g/mol,





IUPAC name O,O-diethyl O-3,5,6-trichloro-2-pyridyl phosphorothioate

A body burden study conducted by the Centre for Disease Control and Prevention found a metabolite specific to chlorpyrifos, in the urine of 91% of people tested. (CDC, 2003) An independent analysis of the CDC data claims that Dow has contributed 80% of the chlorpyrifos body burden of people living in the US. A 2008 study found dramatic drops in the urinary levels of chlorpyrifos metabolites when children switched from conventional to organic diets (Lu *et al.,* 2008). Research indicated also that children exposed to chlorpyrifos while in the womb have an increased risk of delays in mental and motor development at age 3 and an increased occurrence of pervasive developmental disorders such as ADHD.(Rauh *et al.,*2006). An earlier study demonstrated a correlation between prenatal chlorpyrifos exposure and lower weight and smaller head circumference at birth.(Whyatt *et al.,* 2004). A 2010 study found that each 10-fold increase in urinary concentration of

organophosphate metabolites was associated with a 55 % to 72 % increase in the odds of ADHD in children.

2.1.1.5 Acetyl Cholinesterase: Its action and inhibition

The nervous system of the human body, as well as other vertebrates and insects, uses electrical switching centers to make the system work. These nerve endings are constantly sending signals carried in a chemical called acetycholine. Cholinesterase is an enzyme that is needed for the proper function of the human body's nervours system. When acetylcholine completes its function cholinesterase in the body breaks down the acetylcholine, terminating that function of the electrical command. Organophosphorus and carbarmate pesticide contain a cholinesterase inhibitor, which makes them effective in controlling insect and pest. Unfortunately, when people breathe in those pesticide or it get on their skin, they are subjected to the same negative effect. (Mulchandani *et al.,* 2001). If the body's cholinesterase level is decreased because of exposure to organophosphates or carbamates, the cholinesterase fails to function properly, causing the body's nervous system to become "jammed up" with unnecessary commands. The result of this jamming is that the nervous system is constantly stimulated with commands instructing it to perform certain function, but not necessarily in the proper sequence or at the right time. The health effects of this jamming from cholinesterase inhibition can include headaches, dizziness, nausea, restlessness, anxiety, mental confusion, and shortness of breath, diarrhea, convulsions, coma and death. Acetylcholine binds to cholinesterase at two attachment sites. The ester-forming site, containing residue in the protein chain, and the negative or anionic site, contain glutamic acid residue. The carbon atom of the carbonyl group of the substrate carries a slight positive charge and makes an electrophilic attack on the hydroxyl group of the service. This results in acetylation of the enzymes and in the splitting and deactivation of the acetylcholine. The free choline readily leaves the enzymes surface. The esteratic bond (EB) is weak and is rapidly hydrolyzed during the recovery stage of the enzymes, the hydrolysis being facilitated by a basic histidine residue nearby. The surface of the enzyme is then free to accept another molecule of acetylcholine.

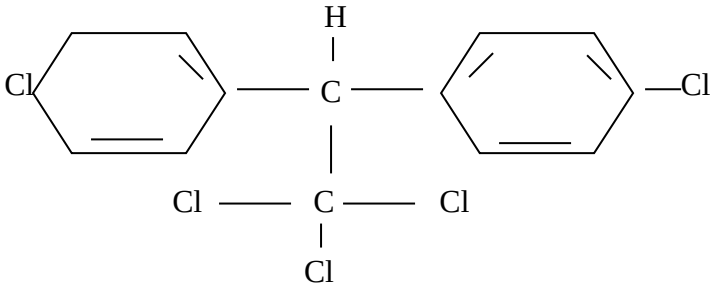
About 300,000 molecules of acetylcholine are destroyed by one molecules of enzyme per minute at 37 ^{0}C (Mulchandani *et al*., 2001)

Organophosphorus compound have similar structure to the natural substrate, acetylcholine. The phosphate group is attracted to the esteratic site and the rest of the molecule is aligned by interaction with the numerous side-groups of amino acids, which form the total active area of the enzyme. Most organophosphorus compounds have no positively changed groups to associate with the anionic site. However, they phosphorylate the enzyme rather than acetylating.The esteratic bond (EB) is relatively stable to hydrolyses, it takes about 80 min for half the enzyme molecules to be dephosphorylated when dimethylphosphate is attached to it. But some six times longer when it is phosphorylated by diethyl/ phosphate .These times are such that it takes up to a million times longer for a molecule of the enzymes to become operative again than it does when it undergoes the natural process of acetylation (Praveen *et al* 2009).

2.1.2 Organochlorine Pesticide

2.1.2.1 Structural Diversity and Properties

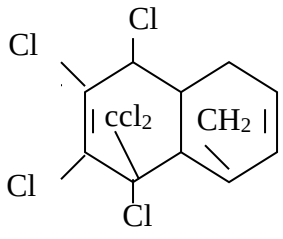
Organochlorine pesticides are characterized by their long-term environmental efforts of highly persistent compounds store in fatty tissue and blood. They are usually divided into three main families, comprising respectively compound related to DDT,γ-HCH and compound related to aldrin



(a) DDT family, eg DDT

(b) HCH family

(only γ - HCH)



(c) Chlorinated cyclodiene family , eg aldrin

Some members of the first and third families contain elements other than carbon, hydrogen and chlorine e.g oxygen or sulphur or both structurally, one of the few features members of the three have in common is that the molecule contain one or more chlorinated carbon cyclic rings. Members of the different families do, however have important physicochemical characteristics in common. The chemical stability of many members of the group is high because their molecules are constructed entirely or largely, from C-C, C-H and C-Cl bond, all of which tend to be chemically rather inactive under normal environmental conditions. Another physico chemical feature of great importance is the low solubility in water of most members of the group couples with this strategy lipophilic. Most organochlorine pesticides are waxy solid at room temperature (CDC, 2003).

2.1.2.1.1 DDT (dichlorodiphenyltrichloroethane)

Persistent organochlorine pesticide such has DDT (dichlorodiphenyltrichloroethane) and its metabolites have provided great benefits to human since their introduction in public health to control mosquito-borne malaria and in agriculture. DDT is highly hydrophobic, colorless, and with a weak chemical odor. It has a good solubility in most organic solvents which is readily absorbed through the gastrointestinal tract with increase absorption in fat. Their intensive use throughout decades and their high persistence, accumulation in food chains and in the human body especially in lipid-rich tissues has raised the interest in knowing the extent of their spread leading to investigation of the magnitude of their residues in all compound of the human environment.Commercial DDT is a mixture of several closely-related compounds.

The major component (77 %) is the p, p' isomers and 15 % o, p' isomer. Dichlorodiphenyldichloroethylene (DDE) and dichlorodiphenyldichloroethane ethane (DDD) are major metabolites and breakdown products in the environment (WHO, 1979). Potential mechanisms of action on humans are genotoxicity and endocrine disruption. DDT is moderately to slightly toxic to studied mammalian species via the

oral route. Reported oral LD_{50} range from 113 to 800 mg/kg in rats, 150-300 mg/kg in mice. 300 mg/kg in guinea pigs ; 400 mg/kg in rabbits ; 500-750 mg/kg in dogs and greater than 1,000 mg/kg in sheep and goats. Toxicity will vary according to formulation. DDT is readily absorbed through the gastrointestinal tract, with increased absorption in the presence of fats. DDT may be directly genotoxic but may also induce enzymes to produce other genotoxic intermediates and DNA adducts (Coha *et al.,* 2007). DDT and DDE, specifically, have been linked to diabetes.

A number of studies from the US, Canada, and Sweden have found that the prevalence of the disease in a population increase with serum DDT or DDE levels (Jones *et al.,*2008; Turyk M.,2009;Codru *et al.,* 2007; Cox *et al.,*2007and Philibert *et al.,* 2009). Toxicological evidence shows endocrine – disrupting properties; human data also indicates possible disruption in semen quality, menstruation, gestational length and duration of lactation (Rogan and Chen, 2005). Several recent studies demonstrate a link between in utero exposure to DDT or DDE and development neurotoxicity in humans. A 2006 University of California, Berkeley study suggests that children exposed while in the womb have a greater chance of development problem and other studies document decreases in semen quality among men with high exposures (Jurewiez *et al*., 2010; Aneck *et al*.,2007; Dejar *et al*., 206). Occupational exposure in agriculture and malaria control has been linked to neurological problem such as Parkinson and asthma.

Epidemiological studies that indicates DDT causes cancers of the liver, Pancreas and breast. There is mixed evidence that it contributes to leukemia, Lymphoma (Spinelli *et al*. 2007) and testicular cancer (McGlym *et al*., 2008). Breast milk in regions where DDT is used against malaria greatly exceeds the allowable standards for breast feeding infants (Bovwman *et al.,* 2006; Ntow *et al.,* 2008; Spicer and Kereu. 1993). The Stockholm Convention, which took effect in 2004, outlawed several persistent organic pollutants, and restricted DDT use to vector control. The Convention has been ratified by more than 160 countries and is endorsed by most environmental groups. Despite the world wide banned agricultural use continues in India North Korea, and possibly Africa (Van den Berg *et al.,* 2008). Most studies of DDTs human health effects have been conducted in developed countries where DDT is not used and exposure is relatively low (Rogan and Chen.2005). Farm workers and

retailers of agrochemicals in the third world counties are at high risk for pesticide related illness.

2.1.2.1.2 Hexachlorocyclohexane (HCH)

Hexachlorocyclohexane (HCH) and lindane were used as organochlorine insecticide since 1949's, against sucking and biting pests and as smoke for control of pests in grain stores, as dust to control various soil pest such as flea beetles and mushroom flies. HCH were progressively banned in most of the nations in the worlds, because of their persistence and their potential toxicity and carcinogenicity (Fabra *el al.*, 2005). HCH consists of eight isomers, only γ –HCH, α-HCH, ß–HCH, and δ- HCH are of commercial significant and considered in this profile as well as technical. Technical grade HCH is not an isomer of HCH but rather a mixture of several isomers. It constituted of approximately 60- 70 % of α-HCH, 10-15 % of γ-HCH, 6 -10 % of δ- HCH, 5-12 % ß –HCH, 3-4 % of ε – HCH and traces of λ-HCH. It has insecticide properties because of the γ- HCH isomer. More than 10 million tons of technical HCH were used in the world (Fabra *el al.,* 2005).Research have documented the presence of biological markers of pesticide in adults and children in agricultural communities (Needham *et al*,2006; Arcury *el al.,* 2007, 2006; Perera *el al.,* 2006; Rauh *el al.,* 2006; USEPA, 2006 ; Lu *el al.,* 2008).

HCH isomers were also recovered in human and animal tissues. The toxicity, bioaccumulation and the persistence of the HCH isomers are the problem of human and animal health. When a person is exposed to them, body detoxification mechanisms are activated. Some are metabolized into different chemical and excreted and some are store in fatty tissue in the body.(Hotton *et al.,* 2011) The wider spread sales and retailing of pesticide in stores located in Open Markets Street and residential area of Taraba Nigeria is at increase. Whose stores were designed to accommodate the pesticide and sales attendance with poor or no cross ventilation not minding the high temperature profile of the region, pesticide volatility and exposure pathways? To this end there were several uninvestigated cases of threat to public health associated with the retailing of pesticide in Nigeria (Hotton *et al.*, 2010).

2.1.3 Carbamates pesticide

Carbamate pesticides are used in agriculture, as insecticide, fungicides, herbicides, nematocides, or sprout inhibitors. They are used as biocides for industrial or other application and in household product. Carbamates are *N* - substituted esters of carbamic acid.

Their general formula is:

$$R^1NH - \underset{\displaystyle O}{\overset{\displaystyle ||}{C}} - OR^2$$

where R^2 is an aromatic or aliphatic moiety. Three main classes of carbamate pesticides are known:

(a) carbamate insecticides; R^1 is a methyl group;

(b) carbamate herbicides; R^1 is an aromatic moiety; and

(c) carbamate fungicides; R^1 is a benzimidazole moiety.

2.1.3.1 Physical and Chemical Properties

In general, simple esters or *N*-substituted derivatives of carbamic acid are unstable compounds, especially under alkaline conditions. Decomposition takes place, and the parent alcohol, phenol, ammonia, amine, and carbon dioxide are formed. The salts and esters of substituted carbamic acid are more stable than carbamic acid. This enhanced stability is the basis for the synthesis of many derivatives that are biologically active pesticides. Carbamate ester derivatives are crystalline solids of low vapour pressure with variable, but usually low, water solubility. They are moderately soluble in solvents such as benzene, toluene, xylene, chloroform, dichloromethane, and 1,2-dichloroethane. In general, they are poorly soluble in nonpolar organic solvents such as petroleum hydrocarbons but highly soluble in polar organic solvents such as methanol, ethanol, acetone,

dimethylformamide, etc. The carbamate derivatives with herbicidal action (such as pyrolan and dimetilan) are substantially more stable to alkaline hydrolysis than the methyl carbamate derivatives (carbaryl and propoxur), which have an insecticidal action. (Needham *et al*, 2005b).

2.1.3.2 Effects on man

Health hazards for man occur mainly from occupational over-exposure to carbamate insecticides resulting in poisoning characterized by cholinergic symptoms caused by inhibition of the enzyme acetyl cholinesterase. Various cases of intoxication have been described. Most of them were spraymen applying insecticides inside houses in the tropics to control mosquito vectors of malaria, or plant protection workers. The main routes of exposure are inhalation and skin. From controlled human studies, it is clear that poisoning symptoms can be seen a few minutes after exposure, and can last for a few hours. Thereafter, recovery starts and within hours, the symptoms disappear, and the cholinesterase (ChE) activity in erythrocytes and plasma returns to normal, because the carbamate is rather rapidly metabolized and the metabolites excreted. The appearance of these metabolites in the urine may be used for biological monitoring. Apart from the symptoms indicative of cholinesterase (ChE) poisoning, other signs and symptoms induced by certain carbamates have been described, such as skin and eye irritation, hyperpigmentation, and influence on the function of testes (slight increase of sperm abnormalities). These signs and symptoms were found in a few studies and should be confirmed before it can be stated that they were induced by carbamates. Epidemiological studies with persons primarily exposed to carbamates are not available.

2.1.4 Natural and Synthetic Pyrethroids

2.1.4.1 Natural Pyrethroids

The pyrethroid pesticide esters are only liquids soluble in alcohol, acetone and petroleum but insoluble in water. The esters are unstable, even in he dust from the dried flower heads, but can be protected by anti-oxidants as long as light and water are excluded (Lu *et al.*, 2006a). In water, they are hydrolysed, the reaction being both acid- and base catalysed. In consequence they are unable to kill insects as stomach poisons and are also very ephemeral contact poisons.

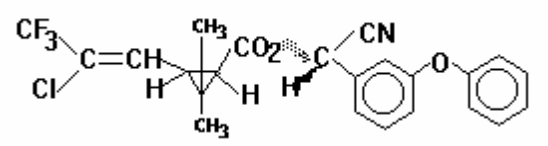
2.1.4.2 Synthetic Pyrethroids

Modeled after the natural product found in chrysanthemums these insecticide are highly toxic to insect yet relatively safe to humans. These compounds act as nerve, poisons and they appear to disrupt sodium transport in axons in much the same way as some organochlorines. The elucidation of the structure of the natural pyrethroids made possible the synthesis of related compounds which while possessing similar or higher insecticidal activities then the natural compounds are also more stable to light and to air. Synthetic pyrethroids modeled after the natural product found in chrysanthemums these insecticides are highly toxic to insects yet relatively safe to human. Natural pyrethrum is nearly useless outdoors because it breaks down rapidly in sunlight.

2.1.5 Lambda-cyhalothrin

Molecular Formula: $C_{23}H_{19}ClF_3NO_3$ Molecular Weight: 449.86

Structural Formula:



2.1.5.1. Toxicological Information

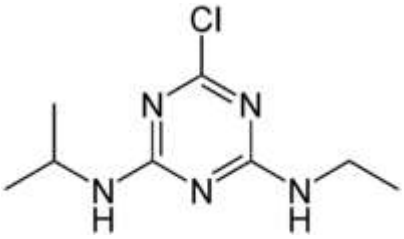
Acute toxicity/irritation studies (Finished Product) Ingestion: Oral (LD_{50} Rat): > 5000 mg/kg body weight Dermal: Dermal (LD50 Rat) : > 2000 mg/kg body weight Inhalation: Inhalation (LD_{50} Rat) : > 4.62 mg/l air - 4 hours Reproductive Effects: In two studies, lambda cyhalothrin caused reduced body weight gain at doses of 15 mg/kg/day in pregnant rats (highest dose tested) and at doses of 30 mg/kg/day in pregnant rabbits (also the highest dose tested), but these doses produced no observable reproductive effects.There were reduced numbers of viable offspring at doses of 50 mg/kg/day in the second and third generations in the three-generational rat study noted above. It is unlikely that lambda cyhalothrin would cause reproductive effects in humans under normal conditions.

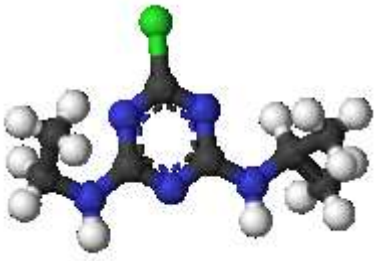
2.1.5.3. Ecological and Ecotoxicological Information

Effects on Birds: Lambda cyhalothrin's toxicity to birds ranges from slightly toxic to practically non-toxic. In the mallard duck, the reported oral LD_{50} is greater than 3,950 mg/kg, and the reported dietary LC_{50} is 3,948 ppm. Inbobwhite quail the reported dietary LC_{50} is greater than 500 ppm. There is evidence that it does not accumulate in the eggs or tissues of birds.

2.1.6 Atrazine

Atrazine, 2-chloro-4-(ethylamino)-6-(isopropylamino)-s-triazine, an organic compound consisting of an s-triazine-ring is a widely used herbicide. Its use is controversial due to widespread contamination in drinking water and its associations with birth defects, menstrual problems, and cancer when consumed by humans at concentrations below government standards. It is still one of the most widely used herbicides in the world.





IUPAC name 1-chloro-3-ethylamino-5-isopropylamino-2,4,6-triazine

Molecular formula $C_8H_{14}ClN_5$

Atrazine is used to stop pre- and post-emergence broadleaf and grassy weeds in major crops. Atrazine is the most widely used herbicide in conservation tillage systems, which are designed to prevent soil erosion. According to Extension Toxicology Network in the U.S., "The oral median Lethal Dose or LD_{50} for atrazine is 3090 mg/kg in rats, 1750 mg/kg in mice, 750 mg/kg in rabbits, and 1000 m_{50}g/kg in hamsters. The dermal LD_{50} in rabbits is 7500 mg/kg and greater than 3000 mg/kg in rats. The 1-hour inhalation LC is greater than 0.7 mg/L in rats. The 4-hour inhalation LC_{50} is 5.2 mg/L in rats (CDC, 2003).

Atrazine was prominently featured in the New York Times as a potential cause of birth defects, low birth weights and menstrual problems when consumed at concentrations below Federal standards. A Natural Resources Defense Council's Report on Atrazine suggested that the EPA is ignoring atrazine contamination in surface and drinking water in the central United States. (IPCS, 2002).

2.2 Monitoring of Pesticide Metabolites in Work Places

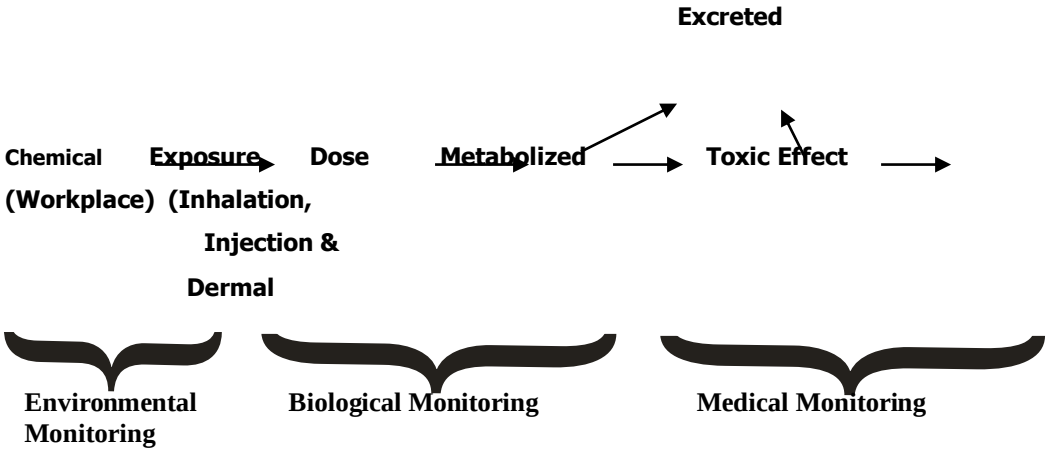
Monitoring is the science of studying, evaluating and checking of situation, event or process in order to determine how it changes or progresses over a period of time. The evaluation of health risk is a multifaceted process and can be group into, biological, medical and environmental monitoring;

i. Biological monitoring is a tool use to determine exposure by looking at the concentration of the chemical in work environment via surface wipe samples of work place or urine and blood of workers.

ii. This gives a pre-clinical measurement of the presence of chemical, estimating the amount which has proceeded from the external to the internal environment.

ii. Medical monitoring is conducted on exposed individual to evaluate any adverse health effect of those exposures or occupational illness is in a particular individual

iii. Environmental Monitoring: This is carried out if inhalation is the only significant route of entry to the body.



2.3 Blood

Blood is composed of a yellowish fluid, called plasma (55 %), in which are suspended millions of cells that constitute 45 % by volume of whole blood. In an average adult, the volume of the blood is about one –eleventh of the body weight and the volume of blood in an average adult is 4.7-5.0 litres. (http://hypertextbook.com/facts/1998/LanNaLee.shtml). The density of blood plasma is 1.025 g/mL and density of blood cells circulating in the blood is 1.125 g/mL. Blood plasma and its content is known as whole blood.

The average density of whole blood for a human is 1.060 g/mL. Whole blood contains on an average about 0.5% lipid. (Covaci *et al*., 2002). Plasma is the straw-colored liquid in which the blood cells are suspended composed of water (90-92 %), proteins (6-8 %), salts (0.8 %), lipids (0.6 %) and glucose-blood sugar (0.1%). Serum lipids (0.6 %) include high density lipoprotein cholesterol, Low Density Lipoprotein cholesterol and 20 % of the triglyceride value. Plasma transports materials needed by cells and materials that must be removed from cells, various ions (Na+, Ca^{2+} , HCO_3, etc.) glucose and traces of other sugars, amino acids, other organic acids, cholesterol and other lipids, hormones, urea and other wastes. Serum proteins make up 6–8% of the blood.

They are about equally divided between serum albumin and a great variety of serum globulins. After blood is withdrawn from a vein and allowed to clot, the clot slowly shrinks. As it does so, a clear fluid called serum is squeezed out. Serum is blood plasma without fibrinogen and other clotting factors. Suspended in the watery plasma are seven types of cells and cell fragments-red blood cells (RBCs) or erythrocytes, platelets or thrombocytes, five kinds of white blood cells (WBCs) or leukocytes, three kinds of granulocytes –neutrophils, eosinophils and basophils and two kinds of leukocytes without granules in their cytoplasm – lymphocytes and monocytes.

2.4 Studies that Examines Children Exposure to Pesticide

Children's exposure to pesticide has been documented in several "Snapshot" studies that tested for a range of pesticide residue in single urine, or blood samples. (Arcury *et al.,* 2005; 2006, Coronado *et al.,* 2006, Lambett *et al.* 2005, Lu *et al.,* 2006). Pesticides pose a greater health risk for children than for adults because of their small body size and rapid development (Weiss *et al.,* 2004). Research demonstrated that multiple types of pesticide are present in many dwellings in the United States, particularly in low-income, urban neighborhoods (Berkowitz *et al*., 2003, Pang *et al*., 2002 and whyatt *et al.,* 2003).

Cumulative exposure to pesticides may come from food, water, air, dust, soil etc. Pesticides can be absorbed through skin contact, inhalation or accidental ingestion. Farm workers come into direct contact with pesticides at work as well and are occupationally exposed to them. When a person is exposed to pesticides, body's detoxification mechanisms are activated. Some pesticides are metabolized into different chemicals and excreted and some are stored in fatty tissues in the body. Body burden data from analysis of blood provides evidence of exposure to chemicals stored in our body. There are several studies including international and national on pesticide residues found in blood samples. A study on the effect of prenatal exposure to air bone hydrocarbon on neurodevelopment among inner-city children shows that high prenatal exposure to was associated with lower mental development index at age 3 (beta = -5.69; 95% confidence interval, P $<$0.01). The odds of cognitive development delay were also significantly greater for children with high prenatal exposure (P=0.01) (Perera and Viswanathan*,* 2006 ; Rauh *et al.*, 2006).

Analysis of 60 urine samples were collected from latino farm workers children 1-6 years of age results reveals that substantial proportion of children, particularly metabolites of methyl parathion (90 %, geometric mean 1.00ug/L) chlorpyrifos / chlorpyrifos methyl (83.3 %; geometric mean

1.92 ug/L) and diazinon (55.0 %; geometric mean 10.56ug/L). The number of metabolites detected ranged form 0 to 7, with a mode of 4 detected (28.3 %). Boy, children living in rented housing and children with mothers working part-time had more metabolites detected (Arcury *et al.*, 2007). National population-based surveys of pesticide urinary metabolites in adults indicate widespread exposure to pesticides, including organophosphates, carbamates, wood preservatives, and fungicides detected chlorpyrifos, an OP pesticide, in 82 % of 993 adults tested through the National Health and Nutrition Examination Survey, and found a 5-fold increase in the proportion of adults with levels over 5 pg/L compared to earlier surveys, suggesting increasing exposure in the general population. OPs are eliminated from the body after 3-6 days, so the widespread detection of these compounds indicates continuing exposure.

Biologic information on children's pesticide exposure is very limited. Preliminary results from the federal Agricultural Health Study indicate detectable pesticide residues in children's urine. Loewenherz *et al.,* 2004, working in Washington State, found that 44 % of children of pesticide applicators and 27 % of nonfarm, rural children had detectable OP residues. Preliminary data from Arizona, chlorpyrifos was detected in 100 % of about 40 children > 6 years of age sampled in a population-based survey, and approximately 25 % of 150 children <6 years of age sampled in an agricultural area. Detection limits in the second survey were higher. Comparison of these studies to each other and to data reported was difficult because of differences in detection limits, sample type (spot samples vs first morning void), and ages of participants.Investigation has proposed a model of farm worker child pesticide exposure that includes Para occupational, residential and environmental factors (Fenske *et al.*, 2005; Quandt *et al.,* 2006). However, measurement of the alkylphosphate metabolites does not provide information on the specific OP pesticide to which these children are exposed (Needham *et al.*, 2005b). A United States study examined the aggregate exposure of pre-school children to clorpyrifor and its degradation product, 3, 5, 6 – trichloro -2- pyridinol (TCP);

Samples that were collected included duplicate diet, indoor and outdoor air, urine, solid and liquid food, indoor floor dust, play area soil, and surface wipes. Generally, level of chlorpyrifos were higher than level of TCP in all media. The median urinary TCP concentration for the preschool children was 5.3 ng/ml and the maximum value was 104 ng/ml. the median potential aggregate absorbed dose of chlorpyrifos for these preschool children was estimated to be 3.0 ng/kg of body weight per day (Morgan *et al*., 2005).

In order to control malaria, DDT was used in Mexico until the year 2000. As a result, DDT contamination was widespread, and it has been shown that children can be exposed to this insecticide by soil ingestion, household dust ingestion / inhalation, fish consumption and human milk. In these areas, DDT level in blood in children are higher than those in adults (Carrizales *et al*., 2006). Exposures by different pathways may result in differential absorption, metabolism, and toxic response even for the same chemical. Cumulative risks are different to assess, and methods are still under development (USEPA, 2003b, USEPA, 2006b).

In a study done in an endemic malaria area in Oaxaca, Mexico, it was found that children were exposed to deltamethrin i.e pesticide used in the control programme for malarial (Yanez *et al.,* 2002b). Moreover, a negative correlation between urinary 3 – phen – oxybenzoic acid (a biomarker for deltamethrin) and age was found in these children (Yanez *et al*., 2002). A community intervention trial to reduce organophosphate pesticide exposure in children of farm workers was conducted and the median concentration of urinary metabolites was higher in year than in year 1 for dimethylthiophosphate (DMTP) and dimethyldithiophosphate in adults and for DMTP for children. There were significant increases within both the intervention and control communities between year 1 and year 4. However, the differences were not significant between study communities after adjusting for year (P = 0.21). The dust residue data showed aziphos-methyl having the highest percentage of detects in vehicles (86 % and 84 % in years 1 and 4, respectively) and in house dust (85 % and 83 % in years 1 and 4, respectively).

There were no significant differences between lecturers intervention and control communities after adjusting for year (P = 0.49) (Thompson *et al.,* 2008). An increasing body of scientific literature has shown that children have a greater vulnerability to the effects of chemical exposure than adults. This can be attributed to a variety of factors including development of sensitive organ system (i.e. brain, immune, respiratory) as well as different capacity to metabolize and eliminate compounds than adults (IPCS, 2007.)

Furthermore children can experience greater environmental exposure by engaging in hand-to-mouth behaviors that increase their risk of ingesting pesticide found in the home (Mc Cauley *et al.,* 2006b and Quandt *et al*., 2004). Studies of children have identified possible risk for the development of cancers, birth defects, and abnormal reflexes, as well as neurological impairments after environmental exposures (Mill and Yang 2003; Rohlman *et al*., 2005; Young *et al*., 2005). Data from Furlong *et al.,* (2006) suggest that children may be much more susceptible to the effects of pesticides than adults. Adverse health effects associated with pesticide exposure from residential use include altered fetal growth from prenatal exposure (Berkowitz *et al.,* 2004), childhood cancer (Flower *et al*., 2004) and asthma (Salam *et al*., 2004).

2.5 Studies that Examine Women Exposure

Most epidemiological studies of breast cancer among women farmers and farm workers have relied on estimations of the past exposure, which can be subjected to exposure misclassification. They have often presented results for all pesticide which makes it different to evaluate the role of an individual compound, but accounts for the fact that exposure generally does not occur to individual pesticide (Engel *et al*., 2005).A nested case – control study of 128 newly diagnosed breast cancer cases within a cohort of Hispanic women farm workers found increased breast cancer risk among younger women and those with early-onset breast cancer (Mills *et al*., 2005). A total of 96 serum and 46 adipose tissue samples collected from infertile women attending centre for reproductive medicine in Belgium from 1996-98 ware analysed for seven organochlorine pesticide and seven

polychlorinated biphenyls. There was a strong association between adipose tissue and serum residues. (Pauwels *et al*., 2000).

In a study from Vera Cruz, Mexico maternal adipose tissue material blood serums from 64 volunteer mothers were analyzed for pesticide residue. The concentration of t-HCl in maternal adipose tissue, maternal serum was 0.17 and 0.22 mg/kg on fat basis respectively and t-DDT was 5.881, 5226 mg/kg and hexachloro benzene was 0.065 and 0.18 mg/kg respectively (Waliszewk *et al*., 2003). A 2005 study found no evidence that California women living in areas of recent, high agricultural pesticide use, experience higher rates of breast cancer (Reynolds *et al*, 2005) similarly, a 2004 study found no association between residential proximity to recent agricultural pesticide use and invasive breast cancer incidents among members of the California teacher study cohort, which has been followed for cancer incidence since 1995 (Reynold *et al.,* 2004)

However, organophosphate urinary metabolites levels were examined during pregnancy and after delivery in women living in an agricultural community in the Salinas valley, California. A total of 28 % were employed as farm field workers during pregnancy, and 81% had at least one household member who worked in agriculture. Samples were collected twice during pregnancy and just after delivery. Total diakylphosphate metabolites levels in samples collected after delivery were higher than in sample collected during pregnancy. Both prenatal and postpartum metabolites levels were higher in these Salinas valley women than in a sample of women of child bearing age in the general U.S population (Brandman *et al.,* 2005).Higher DAP metabolites levels in the immediate postpartum period may have implications for estimating dose during pregnancy and for exposure during lactation (Bradman *et al.,* 2006).

In a study conducted in U.S.A, prenatal insecticide exposure and birth weight and length among an urban minority cohort by analyzing organophosphate diazinon and the carbamate propoxur and insecticide measurement in maternal air during pregnancy as well as in umbilical cord plasma at delivery. Among newborns born after January 2001, exposure levels were substantially lower, and no association with fatal growth was apparent ($P>0.8$). The propoxur metabolite 2-150 propoxyphenol in cord plasma was inversely associated with birth length; a finding

of borderline significance (P = 0.05) after controlling for chlorpyrifos and diazinon. Results indicate that prenatal chlorpyrifos exposures have impaired fetal growth among this minority cohort and that diazinon exposures may have contributed to the effects (Whyath *et al*., 2004). Findings support recent regulatory action to phase out residential use of the insecticides (Zhao *et al*., 2005; Zhao *et al*., 2007). However, the results of most recent case control studies which have focused on white, Western adult women have been largely negative (Brody *et al*., 2007) it is still unclear if particular subpopulations of women of different racial or ethnic backgrounds, for example many have higher breast cancer risks from post or current organochlorine pesticide exposure (Snedekar, 2001).

Among currently-used pesticides, researchers have established connection in some studies, but not all to breast cancer and atrazine, 2,4 D and malathion (Engel *et al*., 2005; Mills *et al*., 2005).

2.6 Studies that examine Animal Exposure

There is a strong and growing body of evidence linking exposure to OP pesticides during gestation or the early postnatal period and neurodevelopmental effects in animals. These effects may be due to the direct impact of OPs on the cholinergic system of the fetus, although effects on cellular intermediates such as adenylyl cyclise and altered DNA synthesis in the brain through noncholinergic mechanisms have been hypothesized (Eskenazi *et al*., 1999).Animal studies revealed that atrazine pesticide can affect mammary gland development. Early-life exposure to atrazine delays mammary gland differentiation in ways that prolong the presence of terminal end buds in the gland (Brody *et al*., 2007).

A 2010 study from Ile-Ife was aimed at determining the effects of an insecticide, 'Raid' contained in feed on the survival, growth and activity of alkaline phosphatase (ALP), glutathione reductase (GR), catalase (CAT), superoxide dismutase (SOD) activities, as well as the effects on glutathione content (GSH), lipid peroxidation (LPO) and utilization of basal energy in Wistar rats. The results indicate that mortality was observed at higher concentrations of the pesticide. Mean total body weight of rats fed 430 µg/g Raid was not significantly different from controls ($p > 0.05$). Significant effects of insecticide Raid on survival and growth were observed

with concentration of 961 µg/g compared to control ($p < 0.05$) after 10 days. Alkaline phosphatase in serum and lipid peroxidation in the liver tissue significantly increased whereas glutathione contents, glutathione reductase, catalase, and superoxide dismutase activities were significantly decreased in the liver tissue, while there was substantial residual glucose level, in both plasma and liver. Increases in ALP and LPO suggest that Raid induced hepatotoxicity and oxidative stress in animals and effect on glucose utilization suggests liver functions may have been impaired in the insecticide-fed rats, resulting in abnormal uptake of glucose. (Achudume *et al*., 2010).Among long-Evans rat, very low levels of atrazine metabolite mixtures administered during late pregnancy were able to perturb mammary gland development of female offspring in ways that persisted into adulthood and that were unrelated to pubertal timing (Enoch *et al*., 2007; Calaf *et al.,* 2007, Moser *et al.,* 2006; Perara *et al*., 2006). By contrast, in human reproductive aging is associated with lower estrogen level. However, data do not exist to support this presumption (Rudel *et al*., 2007).

Gene-expression profiling in some, but not all assays, has shown that the activity of genes related to proliferation was altered after treatment with 2,4 D pesticide (Terasaka *et al*., 2006). In addition to atrazine, the pesticide demonstrated to cause mammary gland tumors in animal studies are 1, 2 – dibromo – 3 – chloropropane (DBCP), captifol, chlordane, clonitralid, dischlorros, fenvalerate, niturthiazole, simazine and suffate (Rudel *et al.,* 2007) of these terms seven are banned or restricted. DBCP is a fumigant that was heavily used on grapes, tomatoes and pineapples until its ban in 1985 (USEPA, 2006). However, there is widespread potential for human exposure in the great lakes area, nifurthiazole is an antibacterial agent no longer produced in the U.S (Brody *et al*., 2007). Califonia receives the highest use of simazine (atriazine herbicides used as a soil sterilant and weed killer) in U.S (USEPA, 2007), several pesticide have been shown to exhibit estrogenicity in the screen assay developed by solo and other (Soto *et al*., 1995). Neurobehavioral tests given postnatally found that animals exposed in utero demonstrated decreased balance, increased righting reflex time, and poorer cliff avoidance. When exposure occurred in the early postnatal period, there was a lowered threshold for convulsions as well as increased gait abnormalities and tremors and deficits in delayed

alternation on mazes. Some studies suggest that early gestation may be a critical period for the neurodevelopmental effects of certain pesticides.

A number of the animal studies reported above have demonstrated a decrease in birth weight or body weight in developing animals exposed to OPs. Anticholinesterase agents such as OPs may have a nonspecific regulatory effect on growth, perhaps by an influence on placental transport of nutrients (Eskenazi *et al*., 1999).

2.7 Dietary intake of pesticide

Many pesticide contaminants are concentrated up the food – chain and result in dietary exposure (WHO, 2006). Infants and young children are often targeted for pesticide exposure assessment because of their susceptibility to possible neurologic and developmental effect (Eskenazi *et al.,* 2007; Whyatl *et al*., 2004). The establishment of the relation between neurological impairments and repeated low-level pesticide exposure that does not induce symptoms of acute poisoning in humans is far less.

Lu *et al*., 2008 carried out a study on dietary intake by substituting organic fresh fruits and vegetables for corresponding conventional food items, the median urinary metabolite concentration were reduced to non-detected level for malathion and chlorpyrifos at the end of the 5 – day organic diet intervention period in both summer and fall season. 750 whole blood samples from resident, of large and medium to small urban centers across Ontario showed a mean concentration of PCBs up to 9.2 ug/kg and DDE up to 3.7ug/kg. Dietary level of PCBS and DDE in food consumed by Ontario residents in whole food basis ranged from 0.1-3.0 ug/kg and 0.05-077 ug/kg respectively.

2.8 Organophosphorus Metabolite in Blood and Urine Samples

Organophosphates exert their toxic effect by inhibiting the enzymatic degradation of the neurotransmitter acetylcholine, at extremely high does. Inhibition of acetylcholine esterase results in the continued firing of the neuron and subsequent paralysis or death of the affected organism (IPCS, 2002). According to Mathur et al., 2005, a total of pesticide detected in blood samples from four villages in Punjab(15 of 28 analysed) which indicated that each person is exposed to and carries a body of multiple pesticide which might be due to a combination of direct and indirect exposure to these pesticide.

Presence of organophosphorus pesticide in blood means that they do persist in the body for good amount of time. In a study conducted in USA, plasma samples collected at birth between 1999 and 2001 from 230 mother and newborn pairs enrolled in the Columbia center for children's environmental health were analysed for 29 organophosphates pesticides. Seven pesticide were detected in 48-83 % of plasma samples (range, 1-270 nglg) the organophosphate chlorpyrifos and diazinon, carbamates bendiocarb and 2 – 150 propoxyphenol (metabolite of propoxur) and fungicides – diclorani maternal and cord plasma levels were similar, except for phthalimide and were highly correlated)$P<0.001$) (whyatt, *et al.,* 2003). Centre for Disease Control (CDC) found that among the people who had their blood and urine tested, 100 percent showed pesticide residues. The average person carried a toxic cocktail of 13 of 23 pesticide analyzed. Two pesticide chlorpyrifos and methyl parathion – were found at levels up to 4.5 times higher than what U.S government deems acceptable. Children, women and Mexican Americans shouldered the heaviest pesticide burden children, the population most vulnerable are exposed to a higher level of nerve damaging organophosphatus pesticide (Schafer *et al.,* 2004).

A study carried out by Harnly *et al.,* 2005; correlating agricultural use of organophosphates without door air concentrations. It was observed that Agricultural use within a 3 – mile radius on the monitoring day and use on the 2 – 4 prior days were significantly associated with air concentration ($P<0.01$) for all analyses except malathion; chlorpyrifos oxon showed the strongest association ($P<0.01$) for malathion to 0.65 ($P<0.01$) for diazinon. Recent cellular animal and human evidence of toxicity, particularly in newborns, supports the public health concern indicated by

initial risk estimate (CDC, 2005). Agricultural applications of organophosphates and their oxon products may have substantial vitalization and off-field movement and are a probable source of exposures of public health concern. (Harnly *et al.*, 2005).

A survey conducted on the Neurological symptoms in licensed private pesticide application in the agricultural health study exposure to high level of many pesticide that has both acute and long term neurological consequences, 18,782 white male licensed private pesticide application were analyzed. Applications provided information on lifetime pesticide use and 23 neurological symptoms typically associated with pesticide intoxication. An indicator of more symptoms ($>$ or = 10 vs $<$10) during the year before enrolment was associated with cumulative lifetime days of insecticide use; it was observed that pesticide use within the year before enrolment was not associated with symptom count only association with insecticide and fumigants persisted when all four pesticide group were examined simultaneously (Kamel *et al.,* 2005).

2.9 Organochlorine Metabolite in Blood and Urine Sample

Organochlorine are persistent chemicals that are stored in body fat, exposures can be measured in blood or fat samples, often many years after exposure. By the early 1990's several descriptive studies had suggested that blood levels or adipose levels of DDT and its DDE metabolites as well as that of other organochlorine pesticide, might predict breast cancer risk (Saedeker *et al.,* 2001). In a study conducted in Delhi, samples of maternal blood, breast milk and cord blood from 25 mothers (23.4$\pm$1.085 years of age with a range of 18-40 years) and their newborn from Irwin Hospital, Delhi showed the presence of t-DDT at an average level of 1.27, 0.27 and 0.14mg/l respectively. Breast milk contained four and a half times more DDT than the maternal serum.

Levels of different metabolites of DDT in maternal serum were more than those in cord serum. HCH isomers were present in smaller amounts than those of DDT residues. Average value of t-HCH in maternal blood, breast milk and cord blood was 0.327, 0.050 and 0.033mg/l β - isomer was the predominant isomer accounting for more than 60 percent of the various isomers (Mathur *et al.*, 2005). A total of 41 samples of maternal blood, milk subcutaneous fat and umbilical cord blood were

analysed from mothers giving birth by caesarean operation at Kenyatta National Hospital in Nairobi. The main contaminants found in all the samples were PP^1 – DDT (100 %), PP^1 – DDE (100 %), PP^1 – DDT (59 %), dieldrin (27 %), transnonachlor (15 %), β-HCH (12 %) and lindane (2 %) of all the samples analyzed. The mean level (mg/kg fat) of t-DDT was 5.9 in subcutaneous fat, 4.86 in mother milk, 2.75 in maternal serum and 1.9 in umbilical cord serum. The mean level of beta-hexachlorocy hexane (β- HCH) in subcutaneous fat and milk fat were 0.034 and 0.26 mg/kg fat, respectively (Kanja *et al.*, 2002). Blood samples of 135 residents living near the estuary of the Eib river (Schleswig Holstein, Germany) were analysed for organochlorines (e.g, P-HCH – 0.5 -222.09 ng/mL) benzene hexachloride (HCB – 0.8 – 55.2 ng/mL), DDE – 0.5 – 29.2 ng/mL and octachlorostyrene (n.d -9.2 ng/mL) (Lommel *et al.,* 2003).

Blood and abdominal tissue from 126 adult cadavers submitted for autopsy at the institute of forensic medicine of the University of Vera Cruz, Mexico were analyzed for HCB, β - HCH, PP^1 – DDE, OP^1 – DDT, and PP^1 – DDT. The comparison of mean and standard deviation values for al organochlorine pesticides between both sample groups indicated significantly higher values of serum lipids Vs. adipose lipids expressed as mg/kg on lipid basis (HCB 0.178 Vs 0.0055, β-HCH 0.504 Vs 0.216 PP^1 DDE 2.789 Vs 1.063, OP^1 – DDT 0.130 Vs 0.062, OP^1 – DDT 0.340 Vs 0.585 and t – DDT – 3.258 Vs. 1.706). 0nly PP^1 – DDT reveals inverse levels which could be due to higher accumulation in adipose fats. The higher levels in blood serum lipids express that these organochlorine are inclined to blood lipids as a body compartment and that he equilibrium pattern favours blood serum lipids (Waliszewski *et al.*, 2004).

Human blood samples from 18 male healthy volunteers of Ahmedabad (urban) area showed the presence of PP^1 – DDE, PP^1 – DDD, PP^1 – DDT and t- DDT an average value of 20.85, 1.15. 2.03, 9.28 and 32.61 ug/L in serum samples respectively. The concentration of α, β and γ - and t-HCH in serum samples was 4.49, 35.06, 1.69 ug/L and 41.23 ug/L respectively. Hexachlorobenzene was present in 7 samples at an average concentration of 0.2 ug/L (Bhatnagar *et al.*, 2004).A survey of blood samples of general population of occupationally exposed population from Delhi showed levels of DDT several times higher than that from other countries. Total DDT ranged from 0.053 – 0.663ppm with a mean value of

0.301ppm, mean total DDT in males (0.344 ppm) was higher than of females (0.229ppm) (Mathur *et al.,* 2005)

In a study from Ile-Ife Nigerian, blood serum of cacao farmers and their domestic water sources were anlaysed for pesticide residues in selected cacao growing communities of South Western Nigeria. The residue analyses revealed that 42 out of the 76 farmers had residues of diazinon, endosufan, propoxur and lindane in their blood; and 47.6 % out of these farmers belong in the > 20 years exposure duration period. About 34 % of the farmers had diazinon with a man concentration of 0.067mgkg, 29 % endosufan (mean = 0.033 $mgkg^{-1}$ 23 % propoxur (mean = 9.095$mgkg^{-1}$) and 17 % indane (mean = 0.080$mgkg^{-1}$) in their blood. The study also revealed that the sources of drinking water had been contaminated with dazinon and proposxur in some of the farmers' localities (Sosan *et al.,* 2008).

2.10 Acute Toxicity and Acute Effects

The acute toxicity of a pesticide refers to the ability of the chemical to cause injury to a person or animal from a single exposure, generally of short duration. Acute toxicity is determined by at least three methods: dermal toxicity is determined by exposing the skin to the chemical; inhalation toxicity is determined by permitting test animals to breathe vapours of the chemical; and oral toxicity is determined by feeding the chemical to test animals. The harmful effects that occur from a single exposure by any route of entry (dermal, inhalation, oral) are termed acute effects. In addition, the effect of the chemical as an irritant to the eyes and skin is examined under laboratory conditions. Acute toxicity is usually expressed as LD_{50} (lethal dose 50) or LC_{50} (lethal concentration 50), which is the amount or concentration of a toxicant required to kill 50 percent of a test population of animals under a standard set of conditions. LD_{50} values of pesticides are recorded in milligrams of pesticide per kilogram of body weight of the test animal (mg/kg bw). LC_{50} values of pesticides are recorded in milligrams of pesticide per volume of air or water (ppm). The lower the LD_{50} value of a pesticide, the less it takes to kill 50 percent of the test population, and therefore the greater the toxicity of the chemical. Parathion, for example, is considered to be highly toxic because the oral lethal dose is less than 4

milligram per kilogram (mg/kg) of body weight, compared with 1200 mg/kg for Malathion, or 5,000 mg/kg for methoxychlor (Mathur *et al.,* 2005).

2.10.1 Chronic Toxicity and Chronic Effects:

Chronic toxicity is determined by subjecting test animals to long-term exposure to a pesticide. The harmful effects that occur from small doses repeated over a period of time, usually years, are termed chronic effects. Some of the chronic effects found in test animals exposed to certain pesticides include birth defects (teratogenesis); toxicity to a fetus (fetotoxic effects); production of tumors (oncogenesis), either benign (noncancerous) or malignant (cancerous/carcinogenesis); genetic changes (mutagenesis); blood disorders (hemotoxic effects); nerve disorders (neurotoxic effects); endocrine disruption; and reproductive effects. The chronic toxicity of a pesticide is more difficult to determine through laboratory analysis than the acute toxicity.**(**Mathur *et al.,* 2005)

2.10.2 Chronic Pesticide Exposure and Diseases

Studies indicate a strong linkage between pesticide exposure and chronic diseases, as very low-level exposures can result in effect long after the initial exposure occurs.

2.10.2 .1 Mutagenesis (genetic changes).

Pesticides have been shown to cause genetic changes. DDT and its metabolites induced DNA damaged in peripheral blood mononuclear cell shown by comet assay. A significant correlation between blood levels of DDT, DDD, DDE and DNA damage was found in women with different amount of environmental exposure to DDT and its metabolites (Yanaz *et al.,* 2004). Monocrotophos was shown to induce single / double strand DNA breaks in mice in vivo using comet assay when oral dose of 0.046, 0.186, 0.373 and 0.746 mg/kg body weight and assay was performed on whole blood (Mohboob *et al.,* 2002).

Malathion was found to cause DNA abnormalities at all doses (0.02, 0.2, 2 and 20 ug/L) when added to human blood cell drawn from three healthy non-smoking men, age 23, 24, & 25 years. It causes a dose dependent increase in chromosomal aberration as well as sister chromated exchanges in human leukocyte cultures. A dose dependent decreases in mitotic under was also observed which

suggested that malathion is a mild mutagen and at higher concentration it may cause genotoxicity in human (Kamel *et al*., 2005).

2.10.2.2 Fetototxic (toxicity to fetus)

A strong relationship has been found between prematurely delivered and low birth weight babies and mothers level of DDE, metabolic break down product of DDT. Studies carried out to evaluate potential toxicological effects of clorpyrifos in rats showed that repeated exposure to sub – threshold doses of chlorpyrifos may lead to growth retardation, behavioral abnormalities and muscle weakness (Mathur *et al.,* 2005). Chlorpyrifos was evaluated for potential development toxicity in rat and was found to show fetotoxic and terratogenic effects at maternal dose of 25mg / kg per day, a dose that also produced maternal toxicity, fetal weight and viability were decreased and fetal death and early resorption increased at this dose (Farag *el al.,* 2005).

2.10.2.3 Terratogenic (birth defects) Effect:

Children born to women who live in a high pesticide use area while pregnant have an increased risk of birth defects –deft lip, limb reduction defects and neural tube defects (Mathur, 2005). Studies suggest that chlorpyrifos, is a suspected neuroteratogen has a shifting cellular target, initially impacting the development of neurons and subsequently affecting the glia, which develop much later (Garcia *et al*., 2003). Malathion has been shown to cause birth defects in a variety of wildlife and at levels lower than other pesticides. When administered to adult animals malathion and related thiophosphonates stimulates and subsequently inhibit the nicotinic sites in skeletal muscle, resulting in muscle weakness and paralysis. Neonates (newborn babies) are far more sensitive to these agents than adults, mainly because of a slower rate of detoxification of the metabolite (the metabolite in this case would be the liver breakdown product of malathion – malaoxon) which has been shown to be far more toxic than malathion itself. Defects in the development of fertilized hen eggs, injected with various organophosphates, are known, but many of these are associated with the inhibition of the enzymes kynurine formamidase and a depression levels critical to development (IPCS, 2000).

2.10.2.4 Effect on the Immune System

Association of DDT, dichlorodiphenyldichloroethylene (DDE), and dichlorodiphenyldichloroethane (DDD) in levels of blood with several immune parameters in patients occupationally exposed to insecticides have been reported. The majority of 49 patients who worked as farmers or farm hands in the former German Democratic Republic were contaminated with more than 1 chemical most commonly DDE, PCBS and HCB. Occupational exposure to insecticide for at least 6 months, 80 % of them had been exposed for more than 20 years had resulted in frequent infections and immunological abnormalities. (Daniel *et al* ., 2002).

Chronic exposure to chlorpyrifos has been shown to cause immunological change. Comparison of chronic health with respects to peripheral lymphocyte phenotypes; auto antibodies (nucleic acids and nucleoproteins, parietal cell, brush border, mitochondria, smooth muscle, thyroid gland, and central nervous system / peripheral nervous system myelin); mitogenesis to phytohemaggtutimin and concanavillin and compared with three control groups (i.e.1 positive, 2 negative) showed an increase in CD 2.6 expression, a decrease in percentage of CD5 phenotype, decreased mitogenesis in response to phytohemagglutinin and concanavillin, and an increased frequency of auto antibodies. The alterations in these peripheral blood markers were unaffected by medication, age, sex, or season. (Thrasher *et al*., 2002).

2.10.2.5 Lymphoma

In utero and early childhood exposure to pesticide is associated with a significantly increased irks for developing non-Hodgkins lymphoma. Non-Hodgkins lymphoma is a cancer that begins in cell of the lymph system (Zahm, 1992) studies indicate an association between pesticide exposure and NHL in children exposed to lindane, DDT, organophosphorus pesticides. The lymph system includes the spleen, thymus, tonsils, bone marrow, lymph nodes and circulating white blood cells, called lymphocytes, lymphocytes and the lymph system are part of the immune system, which protects the body from disease and infection. Non-Hodgkins Lymphoma is characterized by the exercise accumulation of typical lymphocytes. These lymphocytes crowd the lymph system and suppress the formation and function of

blood and immune cells. This leads to a diminished ability of the body to fight infection. (Alavanga *et al*., 2004).

2.10.2.6 Parkinson Disease

Epidemiological studies have suggested an etiologic relationship between pesticide exposure and Parkinson disease. Organochlorine pesticides were assayed in postmortem brain samples from 20 parkinson's disease, 7 Alzheimers disease, and 14 non-neurological control cases. Dieldrin, a lipid – soluble, long – lasting mitochondrial poison, was investigated as a potential etiological of Parkinsonism (Mathur *et al.,* 2005).

2.10.2.7 Behavioural and other Effects on Nervous System

Numerous research workers have reported changes at doses that affect level of ACHE, but without overt signs of intoxication.Developmental neurotoxicity of chlorpyrifos is thought to involve both neuros and glia, incrasing the vulnerability of developing brain (Eskenazi *et al.,* 2007). A study carried out by Kamel *et al.,* 2005 on the Neurologic symptoms in licensed private pesticide applicators in the agricultural health study reveals an indicator or more symptoms during the year before enrolment was associated with cumulative life time days of pesticide use.

Perera *et al*., 2006 used the Bayley scales of infant development to evaluate the effects on child mental and psychomotor development of parental exposure to pesticide during pregnancy in the pathogenesis of neurobehavioral disorders. The result shows that high prenatal exposure (upper quartile) was associated with lower mental development index. A study on the impact of prenatal chlorpyrifos exposure on neurodevelopment among inner-city children reveals a significant effect on the developmental pattern (Rauh *et al*., 2006; Rohlman *et al*., 2006).

The vulnerability increases from the gestational exposure through later periods of development which glial neuronal interaction influence brain architectural, cirultary and function. Exposures occurring during children are as important at those occurring prenatally (Barone *et al*., 2000; Rohlman *et al.,* 2005). Fetal and childhood exposure to chlorpyrifos has raised concerns about development neurotoxicity.

Exposure to chlorpyrifos resulted in adverse effects on brain cell development and cholinergic bio markers.

2.11 Absorption

Adsorption is the adhesion of atoms, ions, bio molecules or molecules of gas, liquid, or dissolved solids to a surface. This process creates a film of the adsorbate (the molecules or atoms being accumulated) on the surface of the adsorbent. It differs from absorption, in which a fluid permeates or is dissolved by a liquid or solid. (Sivaraman *et al*., 2009) The term *sorption* encompasses both processes, while *desorption* is the reverse of adsorption. It is a *surface phenomenon*.

Similar to surface tension, adsorption is a consequence of surface energy. In a bulk material, all the bonding requirements (be they ionic, covalent, or metallic) of the constituent atoms of the material are filled by other atoms in the material. However, atoms on the surface of the adsorbent are not wholly surrounded by other adsorbent atoms and therefore can attract adsorbates. The exact nature of the bonding depends on the details of the species involved, but the adsorption process is generally classified as physisorption (characteristic of weak van der Waals forces) or chemisorption (characteristic of covalent bonding). It may also occur due to electrostatic attraction. (Sivaraman *et al*., 2009) . Adsorption is present in many natural physical, biological, and chemical systems, and is widely used in industrial applications such as activated charcoal, capturing and using waste heat to provide cold water for air conditioning and other process requirements (adsorption chillers), synthetic resins, increase storage capacity of carbide-derived carbons for tunable nanoporous carbon, and water purification.

Adsorption, ion exchange, and chromatography are sorption processes in which certain adsorbates are selectively transferred from the fluid phase to the surface of insoluble, rigid particles suspended in a vessel or packed in a column. In chemistry, is a physical or chemical phenomenon or a process in which atoms, molecules, or ions enter some bulk phase - gas, liquid, or solid material. This is a different process from adsorption, since molecules undergoing absorption are taken up by the volume, not by the surface (as in the case for adsorption). A more general term is "sorption", which covers absorption, adsorption, and ion exchange. Absorption is a condition in which something takes in another substance (McMurry , 2003).

2.11.1 Activated carbon

Activated carbon is a highly porous, amorphous solid consisting of microcrystallites with a graphite lattice, usually prepared in small pellets or a powder. It is non-polar and cheap. One of its main drawbacks is that it is reacts with oxygen at moderate temperatures (over 300 °C).

Activated carbon can be manufactured from carbonaceous material, including coal (bituminous, subbituminous, and lignite), peat, wood, or nutshells (e.g., coconut). The manufacturing process consists of two phases, carbonization and activation. The carbonization process includes drying and then heating to separate by-products, including tars and other hydrocarbons from the raw material, as well as to drive off any gases generated. The process is completed by heating the material over 400 °C (750 °F) in an oxygen-free atmosphere that cannot support combustion. The carbonized particles are then "activated" by exposing them to an oxidizing agent, usually steam or carbon dioxide at high temperature. This agent burns off the pore blocking structures created during the carbonization phase and so, they develop a porous, three-dimensional graphite lattice structure.

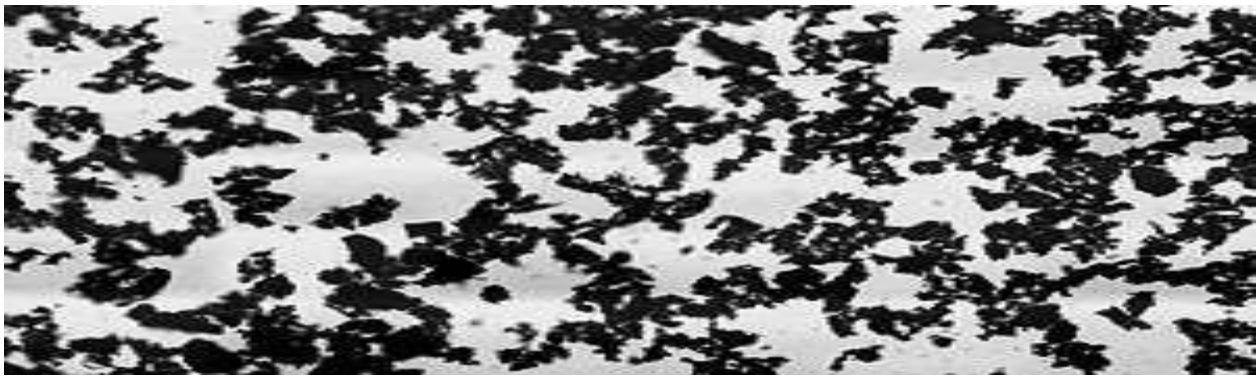
The size of the pores developed during activation is a function of the time that they spend in this stage. Longer exposure times result in larger pore sizes. The most popular aqueous phase carbons are bituminous based because of their hardness, abrasion resistance, pore size distribution, and low cost, but their effectiveness needs to be tested in each application to determine the optimal product. (Scopelliti , 2010)

Activated carbon is used for adsorption of organic substances and non-polar adsorbates and it is also usually used for waste gas (and waste water) treatment. It is the most widely used adsorbent since most of its chemical (e.g. surface groups) and physical properties (e.g. pore size distribution and surface area) can be tuned according to what is needed. Its usefulness also derives from its large micropore (and sometimes mesopore) volume and the resulting high surface area.

2.11.2 Classicification of Activated Carbon

Activated carbon is used for adsorption of organic substances and non-polar adsorbates and it is also usually used for waste gas (and waste water) treatment. It is the most widely used adsorbent since most of its chemical (e.g. surface groups) and physical properties (e.g. pore size distribution and surface area) can be tuned according to what is needed. Its usefulness also derives from its large micropore (and sometimes mesopore) volume and the resulting high surface area. Activated carbons are complex products which are difficult to classify on the basis of their behaviour, surface characteristics and preparation methods. However, some broad classification is made for general purpose based on their physical characteristics. (Wilson , 2005)

2.11.2.1 Powdered activated carbon (PAC)



A micrograph of activated charcoal under bright field illumination on a light microscope.

The fractal-like shape of the particles hinting at their enormous surface area. Each particle in this image, despite being only around 0.1 mm wide, has a surface area of several square metres. This image of activated charcoal in water is at a scale of 6.236 pixels/µm, the entire image covers a region of approximately 1.1 by 0.7 mm.

Traditionally, active carbons are made in particulate form as powders or fine granules less than 1.0 mm in size with an average diameter between 0.15 and 0.25 mm. Thus they present a large surface to volume ratio with a small diffusion distance. PAC is made up of crushed or ground carbon particles, 95–100 % of which will pass through a designated mesh sieve or sieve. Granular activated carbon is defined as the activated carbon being retained on a 50-mesh sieve (0.297 mm) and PAC material as finer material, while ASTM classifies particle sizes corresponding to an 80-mesh sieve (0.177 mm) and smaller as PAC. PAC is not commonly used in a dedicated vessel, owing to the high head loss that would occur. PAC is generally added directly to other process units, such as raw water intakes, rapid mix basins, clarifiers, and gravity filters. (Ferrari *et al.,* 2010)

2.11.2.2 Granular activated carbon (GAC)

Granular activated carbon has a relatively larger particle size compared to powdered activated carbon and consequently, presents a smaller external surface. Diffusion of the adsorbate is thus an important factor. These carbons are therefore preferred for all adsorption of gases and vapors as their rate of diffusion are faster. Granulated carbons are used for water treatment, deodorization and separation of components of flow system. GAC can be either in the granular form or extruded. GAC is designated by sizes such as 8×20, 20×40, or 8×30 for liquid phase applications and 4×6, 4×8 or 4×10 for vapor phase applications. A 20×40 carbon is made of particles that will pass through a U.S. Standard Mesh Size No. 20 sieve (0.84 mm) (generally specified as 85% passing) but be retained on a U.S. Standard Mesh Size No. 40 sieve (0.42 mm) (generally specified as 95% retained). The most popular aqueous phase carbons are the 12×40 and 8×30 sizes because they have a good balance of size, surface area, and head loss characteristics.(Ferrari *et al.*, 2010)

2.11.2.3 Extruded activated carbon (EAC)

Extruded activated carbon combines powdered activated carbon with a binder, which are fused together and extruded into a cylindrical shaped activated carbon block with diameters from 0.8 to 130 mm. These are mainly used for gas phase applications because of their low pressure drop, high mechanical strength and low dust content.

2.11.2.4 Bead activated carbon (BAC)

Bead activated carbon is made from petroleum pitch and supplied in diameters from approximately 0.35 to 0.80 mm. Similar to EAC, it is also noted for its low pressure drop, high mechanical strength and low dust content, but with a smaller grain size. It's spherical shape makes it preferred for fluidized bed applications such as water filtration.

2.11.2.5 Impregnated carbon

Porous carbons containing several types of inorganic impregnant such as iodine, silver, cations such as Al, Mn, Zn, Fe, Li, Ca have also been prepared for specific application in air pollution control especially in museums and galleries. Due to antimicrobial/antiseptic properties, silver loaded activated carbon is used as an adsorbent for purification of domestic water. Drinking water can be obtained from natural water by treating the natural water with a mixture of activated carbon and $Al(OH)_3$, a flocculating agent. Impregnated carbons are also used for the adsorption of H_2S and thiols. Adsorption rates for H_2S as high as 50 % by weight have been reported.

2.11.2.6 Polymer coated carbon

This is a process by which a porous carbon can be coated with a biocompatible polymer to give a smooth and permeable coat without blocking the pores. The resulting carbon is useful for hemoperfusion. Hemoperfusion is a treatment technique in which large volumes of the patient's blood are passed over an adsorbent substance in order to remove toxic substances from the blood. If absorption is a physical process and not accompanied by any other physical or chemical process, it usually follows the Nernst partition la. The law states that:"the ratio of concentrations of some solute species in two bulk phases in contact is constant for a given solute and bulk phases:

$$\frac{[x]_1}{[x]_2} = \text{constant} = K_{N(x,12)}$$

The value of constant K_N depends on temperature and is called partition coefficient. This equation is valid if concentrations are not too large and if the species "x" does not change its form in any of the two phases "1" or "2". If such molecule undergoes association or dissociation then this equation still describes the equilibrium between "x" in both phases, but only for the same form - concentrations of all remaining

forms must be calculated by taking into account all the other equilibria (McMurry , 2003).

In the case of gas absorption, one may calculate its concentration by using, e.g., the ideal gas law, c = p/RT. In alternative fashion, one may use partial pressures instead of concentrations. In many important processes in technology, the chemical absorption is used in place of the physical process, e.g., absorption of carbon dioxide by sodium hydroxide - such acid base processes do not follow the Nernst partition law. Some examples of this effect, include liquid-liquid extraction. It is possible to extract from one liquid phase to another a solute without a chemical reaction. Examples of such solutes are noble gases and osmium tetroxide (McMurry , 2003).

2.11.2.6 Adsorption chillers

Combining an adsorbent with a refrigerant, adsorption chillers use heat to provide a cooling effect. This heat, in the form of hot water, may come from any number of industrial sources including waste heat from industrial processes, prime heat from solar thermal installations or from the exhaust or water jacket heat of a piston engine or turbine. Although there are similarities between absorption and adsorption refrigeration, the latter is based on the interaction between gases and solids. The adsorption chamber of the chiller is filled with a solid material (for example zeolite, silica gel, alumina, active carbon and certain types of metal salts), which in its neutral state has adsorbed the refrigerant. When heated, the solid desorbs (releases) refrigerant vapour which subsequently is cooled and liquefied. This liquid refrigerant then provides its cooling effect at the evaporator, by *ab*sorbing external heat and turning back into a vapour. In the final stage the refrigerant vapour is (re)adsorbed into the solid. (Pilatowsky *et al.*, 2011) As an adsorption chiller requires no moving parts, it is relatively quiet.

2.11.3 Adsorption Isotherm

Adsorption is usually described through isotherms, that is, the amount of adsorbate on the adsorbent as a function of its pressure (if gas) or concentration (if liquid) at constant temperature. The quantity adsorbed is nearly always normalized by the mass of the adsorbent to allow comparison of different materials.

2.11.3.1 Freundlich adsorption isotherm

A sorption isotherm (also adsorption isotherm) describes the equilibrium of the sorption of a material at a surface (more general at a surface boundary) at constant temperature. It represents the amount of material bound at the surface (the sorbate) as a function of the material present in the gas phase and/or in the solution. Sorption isotherms are often used as empirical models, which do not make statements about the underlying mechanisms and measured variables. They are obtained from measured data by means of regression analysis. The most frequently used isotherms are the linear isotherm, Freundlich isotherm, the Langmuir isotherm, and the BET model.

The **Freundlich equation** or **Freundlich adsorption isotherm** is an adsorption isotherm, which is a curve relating the concentration of a solute on the surface of an adsorbent, to the concentration of the solute in the liquid with which it is in contact. The first mathematical fit to an isotherm was published by Freundlich and Küster and is a purely empirical formula for gaseous adsorbates,

$$\frac{x}{m} = kP^{\frac{1}{n}}$$

where x is the quantity adsorbed, m is the mass of the adsorbent, P is the pressure of adsorbate and k and n are empirical constants for each adsorbent-adsorbate pair at a given temperature. The function has an asymptotic maximum as pressure increases without bound. As the temperature increases, the constants k

and *n* change to reflect the empirical observation that the quantity adsorbed rises more slowly and higher pressures are required to saturate the surface.

In 1909, Freundlich gave an empirical expression representing the isothermal variation of adsorption of a quantity of gas adsorbed by unit mass of solid adsorbent with pressure. This equation is known as Freundlich Adsorption Isotherm or Freundlich Adsorption equation. There are basically two well established types of adsorption isotherm: the Freundlich adsorption isotherm and the Langmuir adsorption isotherm. Here the amount of mass that is adsorbed is plotted against the temperature which gives an idea about the variation of adsorption with temperature.

2.11.3.2 Langmuir Adsorption Isotherm

In 1916, Irving Langmuir published a new model isotherm for gases adsorbed to solids, which retained his name. It is a semi-empirical isotherm derived from a proposed kinetic mechanism. It is based on four assumptions:

1. The surface of the adsorbent is uniform, that is, all the adsorption sites are equivalent.
2. Adsorbed molecules do not interact.
3. All adsorption occurs through the same mechanism.
4. At the maximum adsorption, only a monolayer is formed: molecules of adsorbate do not deposit on other, already adsorbed, molecules of adsorbate, only on the free surface of the adsorbent.

These four assumptions are seldom all true: there are always imperfections on the surface, adsorbed molecules are not necessarily inert, and the mechanism is clearly not the same for the very first molecules to adsorb to a surface as for the last. The fourth condition is the most troublesome, as frequently more molecules will adsorb to the monolayer; this problem is addressed by the BET isotherm for relatively flat (non-microporous) surfaces. The Langmuir isotherm is nonetheless the

first choice for most models of adsorption, and has many applications in surface kinetics (usually called Langmuir-Hinshelwood kinetics) and thermodynamics.

Langmuir suggested that adsorption takes place through this mechanism:

$$A_g + S \rightleftharpoons AS,$$

where A is a gas molecule and S is an adsorption site. The direct and inverse rate constants are k and k_{-1}. If we define surface coverage, θ, as the fraction of the adsorption sites occupied, in the equilibrium we have

$$K = \frac{k}{k_{-1}} = \frac{\theta}{(1-\theta)P}, \quad \theta = \frac{KP}{1+KP}.$$

Where *P* is the partial pressure of the gas or the molar concentration of the solution. For very low pressures $\theta \approx KP$ and for high pressures $\theta \approx 1$

θ is difficult to measure experimentally; usually, the adsorbate is a gas and the quantity adsorbed is given in moles, grams, or gas volumes at standard temperature and pressure (STP) per gram of adsorbent. If we call v_{mon} the STP volume of adsorbate required to form a monolayer on the adsorbent (per gram of adsorbent), $\theta = \frac{v}{v_{\mathrm{mon}}}$ and we obtain an expression for a straight line

$$\frac{1}{v} = \frac{1}{Kv_{\mathrm{mon}}}\frac{1}{P} + \frac{1}{v_{\mathrm{mon}}}.$$

Through its slope and *y*-intercept we can obtain v_{mon} and *K*, which are constants for each adsorbent/adsorbate pair at a given temperature. v_{mon} is related to the number of adsorption sites through the ideal gas law. If we assume that the number of sites is just the whole area of the solid divided into the cross section of the adsorbate molecules, we can easily calculate the surface area of the adsorbent. The surface area of an adsorbent depends on its structure; the more pores it has,

the greater the area, which has a big influence on reactions on surfaces.If more than one gas adsorbs on the surface, we define θ_E as the fraction of empty sites and we have

$$\theta_E = \frac{1}{1 + \sum_{i=1}^{n} K_i P_i}$$

and

$$\theta_j = \frac{K_j P_j}{1 + \sum_{i=1}^{n} K_i P_i}$$

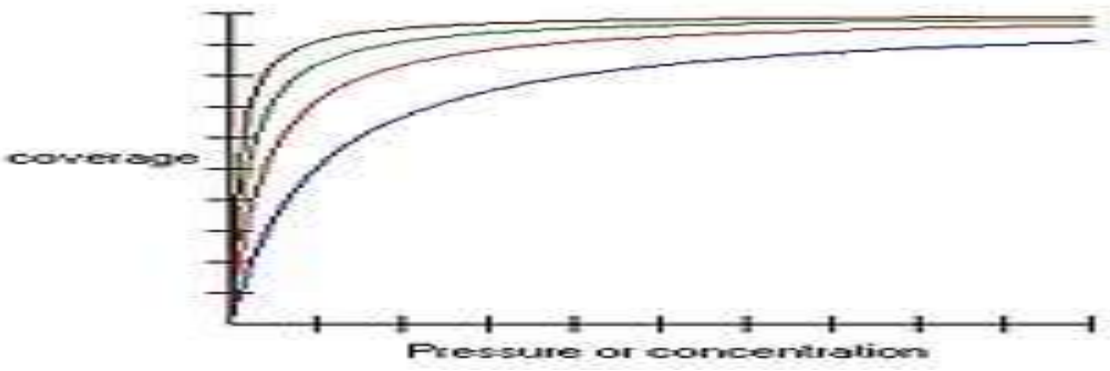
where *i* is each one of the gases that adsorb.

The Langmuir equation (also known as the Langmuir isotherm, Langmuir adsorption equation or Hill-Langmuir equation) relates the coverage or adsorption of molecules on a solid surface to gas pressure or concentration of a medium above the solid surface at a fixed temperature. The equation was developed by Irving Langmuir in 1916. The equation is stated as:

$$\theta = \frac{\alpha \cdot P}{1 + \alpha \cdot P}$$

θ or theta is the fractional coverage of the surface, **P** is the gas pressure or concentration, **α** alpha is a constant.

The constant α is the Langmuir adsorption constant and increases with an increase in the binding energy of adsorption and with a decrease in temperature.



The Langmuir equation is expressed here as:

$$\Gamma = \Gamma_{max} \frac{Kc}{1 + Kc}$$

where K = Langmuir equilibrium constant, c = aqueous concentration (or gaseous partial pressure), Γ = amount adsorbed, and Γ_{max} = maximum amount adsorbed as c increases.

The equilibrium constant is actually given by Γ_{max}:

$$\Gamma(c = K^{-1}) = \Gamma_{max} \frac{KK^{-1}}{1 + KK^{-1}} = \frac{\Gamma_{max}}{2}$$

The Langmuir equation can be fitted to data by linear regression and nonlinear regression methods. Commonly used linear regression methods are: Lineweaver-Burk, Eadie-Hofstee, Scatchard, and Langmuir

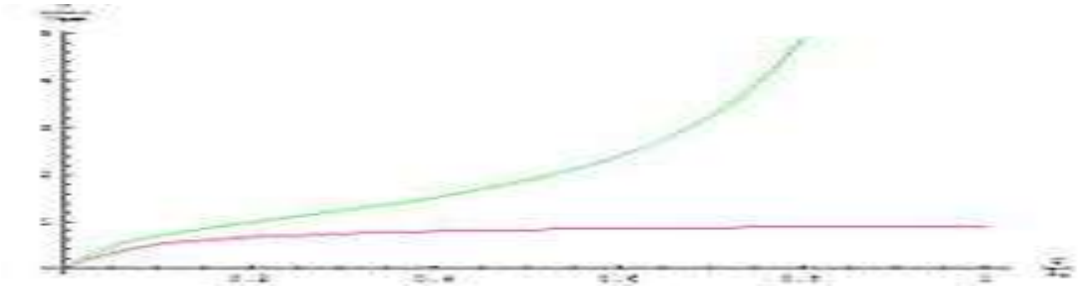
2.11.3.3 BET Theory

Often molecules do form multilayers, that is, some are adsorbed on already adsorbed molecules and the Langmuir isotherm is not valid. In 1938 Stephen Brunauer, Paul Emmett, and Edward Teller developed a model isotherm that takes that possibility into account. Their theory is called BET theory, after the initials in their last names. They modified Langmuir's mechanism as follows:

$A_{(g)} + S \rightleftharpoons AS$

$A_{(g)} + AS \rightleftharpoons A_2S$

$A_{(g)} + A_2S \rightleftharpoons A_3S$ and so on



Langmuir isotherm (red) and BET isotherm (green)

The derivation of the formula is more complicated than Langmuir's (see links for complete derivation). We obtain:

$$\frac{x}{v(1-x)} = \frac{1}{v_{\mathrm{mon}}c} + \frac{x(c-1)}{v_{\mathrm{mon}}c}.$$

x is the pressure divided by the vapor pressure for the adsorbate at that temperature (usually denoted P / P^0), *v* is the STP volume of adsorbed adsorbate, v_{mon} is the STP volume of the amount of adsorbate required to form a monolayer and *c* is the equilibrium constant *K* we used in Langmuir isotherm multiplied by the vapor pressure of the adsorbate. The key assumption used in deriving the BET equation that the successive heats of adsorption for all layers except the first are equal to the heat of condensation of the adsorbate. The Langmuir isotherm is usually better for chemisorption and the BET isotherm works better for physisorption for non-microporous surfaces

CHAPTER THREE
MATERAILS AND METHODS

3.1 Determination of Pesticide Urinary Metabolite in the Urine of Agrochemicals Retailers

3.1.1 Study Location and Area

The study was conducted in the North and Southern Senatorial districts of Taraba, Nigeria.Potential subjects were initially identified using a site-based sampling approach (Ohayo-mitoko 1999).This approach first identified locations where pesticide retail outlets were predominantly found in the two districts which comprise, Jalingo, Mutum Biyu, Garba Chede, Wukari, Ibi, Donga, Maraba, Takum, Bali and Lau. The research team visited these sites during the months preceding data collection to introduce them and to determine when and how many eligible retailers were expected to be enrolled. Fifty (50) retailers were recruited one each from 50 different retail outlets mapped out for the study across the two districts.

The researches returned to the sites in May and were introduced to the subjects by the support and partnership of the leadership of Agrochemical Dealers Association of Nigerian (ADAN), Taraba State chapter. The ADAN chapter did not provide any assistance in recruiting subjects except for granting permission for enrolment. Retailers who were eligible to participate in the study were those that had not worked in agriculture or involved in any fumigation exercise during the previous 12 months.

MAP OF TARABA STATE SHOWING SAMPLING LOCATION

3.1.2 Sampling Methodology

Spot urine samples for pesticide metabolic analysis were collected from the 50 subject recruited. Sterilized and residue free sampling bottles were given to all subjects on the day of urine collection. The urine samples were collected at the close of work in the evening. Samples were labeled, stored on ice and transported to the laboratory for pesticide residue analysis. Urine specimens were adjusted to pH3.0 and aliquated into test tubes, and stored at -20^{0}C until extraction and analysis.

3.1.3 Equipments

Gas Chromatographs used for pesticide residue analysis were Thermoquest-Trace GC Shimadzu- 17A series equipped with flame ionization detector) with advanced software and Nucon –GC- 5765 series equipped with Nitrogen Phosphorus Detector. GC columns employed were capillary column, DB- 1701 14 %-Cyanopropyl-phenyl-methylpolysiloxane (length 30m, ID 0.25 mm and film 0.25mm), DB- 17 50 % phenyl methylpolysiloxane (length 30m, ID 0.25 mm and film 0.25mm), and DB-5, coated with 5% diphenyl and 95 % dimethylpolysiloxane (length 30m, ID 0.25 mm and film 0.25mm). Rotatory evaporators (Buchi type) was employed.

3.1.4 Chemicals and Solvent

Pesticide reference standards of organophosphorus pesticides - dichlorvos, acephate, phorate, diazinon, monocrotophos, dimethoate,Pirimiphos –methyl, phosphamidon, chlorpyrifos, malathion, fenthion, quinalfos, phenthoate, profenofos and ethion were obtained .

3.1.5 Solvents and Glassware

Acetone, di ethyl ether and hexane (HPLC grade) were used. Organic solvents were glass distilled and checked for any pesticide contamination. All glassware were washed with detergent, rinsed with water, dipped in chromic acid for 24 hr and finally rinsed with distilled water and then hexane.

3.1.6 Sample Extraction and Clean-Up

The samples were analysed for organophosphorus compounds by using USEPA method 8141A by gas chromatograph -capillary column technique.

3.1.6.1 Extraction

Extraction was based on the method followed by Agarwal *et al*, 1976 with some modifications. Urine (5 ml) was diluted with 25 ml distilled water and 2 ml of saturated brine solution added and transferred to a 125 ml capacity separatory funnel and extracted with hexane: acetone (1:1) (20 ml) (thrice) by shaking the separatory funnel vigorously for 2-3 min, releasing the pressure intermittently. The layers were allowed to separate. The three combined extracts were passed through anhydrous sodium sulfate and concentrated to about 1-2 ml using rotary vacuum evaporator.

3.1.6.2 Sample Clean –Up

Clean up was done by USEPA Method 3620B- Florisil clean up by column chromatography. Florisil was activated at 130^0 C overnight and cooled in a dessicator before use. Weight of florisil taken was predetermined by calibration using lauric acid. 1g florisil was packed in the 20 cm length and 12 mm ID glass chromatographic column, anhydrous sodium sulfate was added to the top of the florisil column (0.5 cm) and the column was pre-eluted with hexane and discarded. Transferred the extract to the column and eluted with hexane (10 mL), 6 % diethyl ether in hexane (10 mL), 15 % diethyl ether in

hexane (10 mL), 50 % diethyl ether in hexane (10 mL) and finally with diethyl ether (10 mL). Eluent was collected and evaporated to dryness. Final samples were prepared in 2 mL hexane (HPLC grade) and analyzed by GC-ECD for organochlorines and GC equipped with NPD for organophosphorus pesticides.

3.1.7 Calibration of GC system*:*

GC system was calibrated using external standard technique. Stock standard solution (1000 mg/L): Individual stock solutions were prepared by weighing appropriate amounts of active ingredients in a brown bottle with a Teflon-lined screw cap and dissolving the weighed standard in HPLC grade hexane. The resulting concentration was corrected for the stated. Purity if purity was less than 96 %. Stock standard solution was used to prepare primary dilution standards. *Composite stock standard solution*: Appropriate volume of each individual stock solution was taken in a volumetric flask and mixed the solutions to obtain composite stock standard solution. *Calibration standard*: Calibration standard was prepared at different concentrations by dilution of the composite stock standard solution with hexane, corresponding to the expected range of concentrations found in samples and it was used to calibrate (retention time, area count) the instrument response with respect to analyte concentration.

3.1.8 Analysis of Organophosphorus Pesticide Residues

Organophosphorus pesticides were analysed by Gas Chromatograph (Nucon –5765 series equipped with Nitrogen Phosphorus detector). The capillary column used was another GLC capillary column – DB- 17. The carrier gas and the makeup gas was nitrogen with a 2.0 ml/min and 30 ml/min flow rate respectively, hydrogen at 5 ml/min and air at 80 ml/min respectively employing the split less mode. 2.0 ml of the final extract was injected at a temperature of 270 ^{0}C. The oven temperature was kept at 120 ^{0}C with a hold time of 1 minute, then from 120 ^{0}C to 205 ^{0}C at a rate of 25 ^{0}C/minute with a hold time of 1 minute then finally from 205 to 270 ^{0}C at a rate of 2 ^{0}C /

minute with a hold time of 1min. The total run length was 38.90 minutes. The detector was maintained at 290 ^{0}C. (USEPA, 1980).

3.1.9 Linearity checks

Gas chromatograph equipped with FID and NPD were checked for linearity. Instrumental limit of detection for GC- FID was 0.1 ng/mL for organophosphorus pesticides.

3.1.10 Laboratory Reagent Blank

An aliquot of reagent grade water was treated exactly as a sample including exposure to all glassware, equipments, solvents, and reagents used with the sample matrix. No analyte peak was detected in laboratory reagent blank.

3.1.11 Laboratory fortified blank

An aliquot of reagent grade water to which known amount of pesticides was added in the laboratory in ppb range was analyzed exactly like the sample. The recovery of the pesticides over the background values obtained from unfortified samples was more than 80 per cent for all the pesticides.

3.1.12 Laboratory fortified sample matrix

An aliquot of sample matrix (urine) was prepared to which known quantities of the pesticides were added in the laboratory in ppb range. This laboratory fortified matrix was analyzed exactly like the sample.15-25 % of the samples (minimum) were fortified with a known concentration of pesticides and percent recovery was calculated. Extraction and clean up was done as mentioned and the recovery of the pesticides over the background values obtained from unfortified samples were more than 80 percent. Standard deviation and coefficient of variation were less than 10 indicating repeatability of the method. All calculations were done as described in US EPA

8141A method and the amount of residues in samples was obtained in mg/L (ppm) of urine**.**

3.2 Analysis of Pesticide deposit on Worktables and Floors

3.2.1 Materials

Baby wipe, glass vial, cooler boxes, Ice pack, weighing balance

3.2.2 Selection

Retail outlets were initially identified using a site-based sampling approach (Arcury *et al*., 1999).This approach first identified locations where pesticide retail outlets were predominantly found in the two districts of Taraba State. Ten selected retail stores were adopted for the study.

3.2.3 Sampling methodology

Data collection took place in the participant's retail outlets. Standard wipe sampling technique was adopted following protocol described by Geno *et al*. (1996) for collecting wipe samples on surfaces. Baby wipe was used as the media for sampling collection. A measured surface area of 100 cm^2 was apportion and marked using masking tape on the retailers worktable; floor inside the store, and floors outside the store at distances of 5 m & 10 m (directly opposite) and 5 m & 10 m (adjacent) to the entrance doors. The sampling was duplicated in ten (10) selected retail outlets. The designated surfaces were wiped with media using firm pressure in S-strokes, edge to edge and in concentric direction of the entire surface area. A fresh pairs of nitrite gloves was used on each surface. The media for sample collection was folded inward and placed in a sampling vial, labeled, store in ice and send to the laboratory.

3.2.4 Sample Extraction

Each wipe sample was shake-extracted as described (Geno *et al*., 1996). The wipe samples were spiked with chlorfenvinphos as an extraction surrogate and then shake extracted, first with isopropanol, and then twice with 1:1 deithyl ether: hexanes. The combined extract was concentrated with an N-Evap evaporator to 20 mL. One milliliter was passed through a carbograph cartridge to remove interferences from co-eluting compounds and

concentrated to a final volume of 1.0 ml in 10 % diethyl ether in hexanes. Amounts of the target pesticide in samples were determined using a Shimadzu – 17A series Gas chromatography equipped with flame ionization. No target analytes were detected in the matrix blanks. Indicating that there was no laboratory introduced contamination.

3.2.7 Pesticide Analysis on surface Samples

Wipe extracts were analyzed for five dialkylphosphate (DAP) metabolites dimethylphosphate(DMP),diethylphosphate (DEP), dimethylthiophosphate (DMTP) diethylthrophosphate (DETP), and dimethyldithiophosphate (DMDYP) . Wipe samples were prepared for GC analysis based on the method followed by Moate *et al*. (1999). Each specimen underwent azeotropic distillation with methanol and evaporation under a nitrogen stream. Sample extracts were the derivatized with 2, 3, 4, 5, 6-pentafluorobenzylbromide to convert phosphate acids to esters.

3.2.8. Data analysis

All samples were successfully analyzed for pesticides. Pesticide amounts removed (micrograms) were measured separately on the three surfaces: work table, inside floor and outside floor. Pesticide loading (micrograms per square meter) were calculated by dividing amounts by corresponding surface areas.

3.3 Monitory of Cholinesterase Inhibition

3.3.1Questionnaire Administration

Structural survey questionnaires as contained in Occupational Safety and Health Administration (OSHA) website, www.osha_slc.gov/osh.std for medical evaluation questionnaire (Standard – 29CFR Regulation) were modified and administered to 50 exposed retailers of agrochemical and 50 non exposed retailers of animal feed as control mapped out for the study across the two Districts .These were retailers who were not occupationally exposed to pesticide. The group was as similar to the exposed group as possible for occupation, nutrition and socio economic status. The Sampled questionnaire is contained in Appendix.

3.3.2 Data Collection

Data collection took place in the participant's retail outlets. It consists of a structural questionnaire in English given to all subject, and control. The questions were sometimes translated orally into Hausa language by the interviewers as they were asked. The medical evaluation questionnaire obtained on OSHA's website, extracted and relevant medical symptoms were reviewed and modified by Physician Consultants from Federal Medical Centre Jalingo Nigeria. Demographic information was completed at the time of registration. The extracted information included the retailers name, sex, age, duration of trade, hygienic behaviour (eating, drinking, washing of hands / face and smoking at work place), protective clothing (overall, face & nose mask, hand gloves, boot, goggles). Questions were asked on the design of their retail outlet i.e. (suitability and cross ventilation), and symptoms experienced at the time of interview, with a checklist of 58 symptoms.

Symptoms reported during the high exposure period were clustered initially with reference to literature on health effects of pesticide (organophosphate and carbamate) that inhibit cholinesterase activity. This approach was used to support data reduction strategies (Ohayo-mitoko 1999).The study period was divided into periods of low exposure (dry season, Nov-April) and high exposure (wet season, May-Oct). During the low exposure period cholinesterase activity were measured in the subjects and controls to determine baselines for these people. A field – testing kit consisting of a portable spectrophotometer and pre-weighed reagents was used. Acetylcholinesterase activity was determined in the field using venous blood samples. Prior to obtaining blood, all donors washed their hands and arms with soap and water to remove any contamination. After drying with tissue paper, the skin was swabbed with an alcohol-based tissue swab before sampling.

3.3.3 Sample Analysis

Samples were collected at close of work; 10 ml blood was taken by venepuncture, and was analyzed immediately in the field. The acetylcholinesterase activity was measured with a World Health Organization (WHO) approved field spectrophotometric kit, from EQM Research, based on the method of Ellman *et al.*, (1961). During the period of high volume of sales (wet season for Agricultural activities), cholinesterase measurement were again performed on the exposed and the controls.

3.3.4 The variables used to describe change in Acetylcholinesterase.

The variables used to describe change in acetylcholinesterate activity (expressed as % inhibition) was derived as the ratio of acetylcholnesterase activity during high exposure divided by activity during low exposure. These variables are related to acetylcholinesterase activity determined in International Units (IU/mL) of substrate hydrolysed per ml blood at 25^0 C and corrected for hemoglobin concentration. The interpretation of cholinesterase test result was carried out by a consultant physician. A 10-20 % depression in cholinesterase means that slightly poisoning has taken place, A 20 to 30 % drop signal, moderate poisoning and a 30 to 50 percent decline in the cholinesterase readings indicates severe poisoning.

3.3.4 Data Analysis

Descriptive statistics (mean, standard deviation and percentage) were used to analyzed the data obtained from the structural questionnaires.

3.4 Evaluation of Hexachlorocyclohexane Isomers and DDT

3.4.1 Sampling Methodology

Venous blood (10 mL) of 50 agrochemical retailers selected for the study were collected with the technical assistance from the department of Haematology Federal medical centre Jalingo, Taraba State Nigeria .Prior to obtaining blood at close of work all donor washed their hands and arms with soap and water to remove any contamination. After drying with tissue paper, the skin was swabbed with an alcohol-based tissue swab before sampling. Blood samples were collected in residue free heparins 20 mL glass vials containing 200 USP units of heparin in 0.2 mL solution with the help of sterilized syringe. Blood samples were label appropriately and transported in dry ice to the laboratory and stored at-20^{o}C until analyzed.

3.4.4 Hexachlorocyclohexane Residues and DDT Metabolites Determination:

Weight of florisil taken was predetermined by calibration using lauric acid. 1g florisil was packed in the 20 cm length and 12 mm ID glass chromatographic column, anhydrous sodium sulfate was added to the top of the florisil column (0.5 cm) and the column was pre-eluted with hexane and discarded. The extract were transferred to the column and eluted with hexane (10mL), 6 % diethyl ether in hexane (10 mL). Eluent was collected and evaporated to dryness. Final samples were prepared in 2 mL hexane (HPLC grade). Quantification of HCH isomer residue and DDT levels were done by shimadzu – 17A series Gas chromatograph equipped with 63 Ni selective electron capture detector. (USEPA, 1980).

Quantitative analysis of HCH and DDT residues in each sample was done by comparing the peak heights with those obtained from a chromatogram of a mixed HCH Isomers and DDT standard of known concentration. The average recoveries of fortified samples were exceeding 95

%. An aliquot of reagent grade water was treated exactly as a sample including exposure to all glass ware, equipments, solvents and reagent used with the sample matrix. No analyte peak was detected in laboratory reagents blank. Blood samples were analyzed based on the categorization of the agrochemical retailers on their years of exposure duration.

3.5 Adsorption Efficiency of Activated Carbon for pesticide Vapour

3.5.1 Materials

Selected grades of cattle bones and coconut shell were locally sourced abattoir and Jalingo, washed with hot water and boiled for 10 minis to remove fat and oil .The shell and bones were further dried and heated in a furnace at a temperature of 400^{O}C -600^{O}C in oxygen -deficient atmosphere that cannot support combustion. The carbonized particles were made "active" by exposing them to steam at high temperature for an hour. The steam burns off the decomposition products from the carbonization phase to develop a porous to activate the pore sizes. The carbonized charcoal were separately grated and sieved to fine particle size on a U.S standard mesh size No. 40 sieve (0.42 mm).Corn cob, cotton seed, Kokoali seed, Murichi fibre, and saw dust were also used as adsorbents in this study. Granulated activated carbon (GAC) is designated by mesh sized; 4x6, 4x8 or 4x10 for vapour phase application

3.6.2 Surface Characterization measurements.

The surface characterization of carbon were carried out only on the adsorbent of carbon from coconut shell (CSA) and Borne Char (BCA) for they were the most effective of the adsorbents in this study. They were measured by using precise instrumental techniques for the particle size and true densities.

The particle density was measured by a mercury displacement method using a mercury porosimeter (Autopore II 9200; Micromeritics, Inc GA, USA). The true density was determined by a helium displacement method using a pycnometer (Accupyc 1330; micromeritics, Inc, GA USA).The BET surface

area, total pore volume and adsorption /desorption apparatus ASAP 200 (Micromeritics Inc GA USA) using a continuous flow method. The BET equation was used to calculate the surface area of GACs.

3.6.2.1 Procedure for Adsorption

Commercial pesticides (Chlorpyrifos and Dichlorvos) were obtained from agrochemical stores in Jalingo. Different percentages of concentrations were prepared i.e. 0.4, 0.6, 0.8 and 4.0 %. 20 mL of the already prepared concentration of each pesticide were measured separately and placed at the centre of an enclosed heating glass bowl. 10 g each of the selected adsorbents were measured and placed within 20 cm^2 radius of the measured 20 mL pesticide in the heating bowl. The vaporization of the pesticide occurred at a given temperature. The adsorption temperature was performed at 20,30 40 and 50 ^oC which were regulated and maintained by water constant bath the process was allowed to stay for 1-2 hours. The entire experiment was repeated for the pesticides and adsorbent at various concentrations and temperature as in above. The respective adsorbents were collected after the exposure duration of 2hours and placed in a sealed glass vial label properly and stored at 4^oC until analysis.

3.6.2.2 Sample extraction, clean up and analysis.

The adsorbent samples were analysed for organophosrus compound using USEPA method 8141A by Gas chromatography- capillary column technique. Extraction was based on the method followed by Agarwal *et al.* 1976 with some modification using hexane: acetone (1.1) 20 mL (thrice) as solvent for extraction. Clean up was done by USEPA method 3620B by column chromatography. The extract were transferred to the column and eluted with hexane (10 mL), 6 % diethyl ether in hexane (10 mL), 50 % diethyl ether in hexane (10 mL) and finally with diethyl ether (10 mL). Eluent were collected and evaporated to dryness. Final samples were prepared in 2 mL hexane (HPLC grade). (USEPA, 1980).

Quantification of organphosrus pesticide adsorbed by the adsorbent was done by Shimadzu – 17A series GAS chromotographly equipped with 63 NI selective electron capture detector. Quantitative analysis of OP residues in each sample was done by companying the peak height with those obtained from a chromatogram of a mixed O P standard of known concentration the average recoveries of fortified samples were exceeding 95 %. An aliquot of reagent water was treated exactly as a sample including exposure to all glass ware, equipments, solvents and reagent used with the sample matrix. No analyte peak was detected in laboratory regents blank.

CHAPTER FOUR

RESULTS AND DISCUSSION

4.1 Pesticide Metabolite in the Urine of Agrochemical Retailers

Figure 4.1 shows a graphic representation of distribution across the grouping among the retailers, with the duration of exposure per day and corresponding numbers of detects with pesticide in their urine. The grouping 1-3 years representing 40 % of the total numbers of participants had the highest average exposure duration of 9.5 hours per day, followed by the grouping 4-7 years representing 32 % of the subjects with 8 hours exposure per day. The grouping 8-11 years, 12-15 years and greater than 15years had the corresponding lower exposure duration per day of 7.5 hours, 6.5 hours and 6hours respectively.

The study revealed that the grouping 1-3 years ,4-7 years and 8-11 years had the highest number of pesticide metabolite detects of 100 %, 93.75 % and 87.5 % respectively, while the grouping 12-15 years and >15 years had lower values of pesticide detects of 50 % each. The findings revealed also that the retailers with 1-3 years and 4-7 years exposure were mostly youth and had the highest pesticide residue representing 40 % and 30 % respectively. The high number of detects may be attributed to lack of adequate safety practice, inadequate training and inexperience in handling pesticides, accelerated with the high duration of exposure per day ranging from 8.5 - 9.0 hours on daily bases. While the lower values may be attributed to the their low exposure period of 6 hours per day, adherence to safety measures , training and experience on the job as well as metabolite activities of the body detoxification mechanism over time.

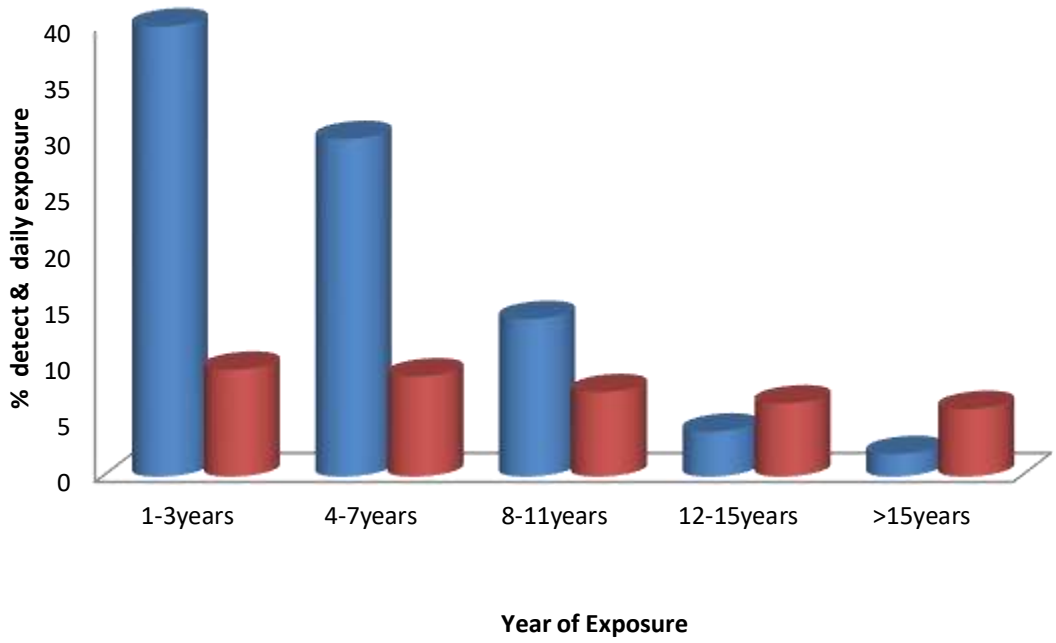


Figure 4.1 Grouping of retailers showing % of pesticide detect and daily Exposure

The high percentage of pesticide detects of 100 % across the grouping 1-3 years and 4-7 years which are youth, is of great concern. It worth noting that Rogan and Chen,(2005) reported decrease in semen quality among young men with high exposure, prolong menstruation and delay of lactation among young women.

4.1.1 Organophosphorus Metabolite Detects in the Urine Sample

Figure 4.2 provides descriptive statistics of the mean concentration and urinary metabolite detects based on their classification. The urinary pesticide metabolite were detected in 46 of the 50 subject sampled representing 92 %. Seven groups of the metabolites were identified. Out of the seven, four (4) were metabolite of organophosphorus pesticide belonging to the class of dialkylphosphate (DAP) which appeared as dimethylphosphate (DMP) for dichlorvos and monocrotophos, diethylthiophosphate (DETP) as Pirimiphos-methyl, dimethyldithiophosphate (DMTP) as chlorpyrifos-methyl and diethylphosphate (DEP) as chlorpyrifos. This agrees with the findings of Lu *et al.*, 2001 which states that the common urinary metabolite that are identified after exposure to OP pesticide are DAPs metabolite that are formed

when OP pesticide undergo cleavage of the leaving group with substitution for a hydrogen atom. When a person is exposed to pesticide, bodys detoxification mechanisms are activated, some pesticide like organophosphorus are metabolized into different chemicals and excreted while those of organochlorine are stored in fatty tissues in the body.

Body burden data from analysis of urine provides evidence of exposure to chemicals stored in the body. Scientific field investigations have focused on delineating the extent of exposure and potential health effects in agricultural and non-agricultural communities. Measurement of dialkyl phosphate (DAP) compounds in the urine from this study will be useful to assess exposure to organophosphate pesticide (Coronado *et al.*, 2006; Lu *et al.,* 2006; Lambertt *et al.*, 2005; Fenske *et al.,* 2005; Quandt *et al.,* 2006; Needham *et al.*, 2005b).

The findings from the study revealed the presence of two herbicides and one synthetic parathyroid. Figure 4.2 also shows the total number of pesticide detects, mean concentration and their corresponding amount in the urine analysis. 30 of the 50 retailers representing 60 % had residues of pirimiphos-methyl, 20 (50) representing 40 % had chlorpyrifos, 19(50) at 38 % had dichlorvos, 16(50) at 32 % had residues of monocrotophos at mean concentration of 0.018 mg/kg, 0.01 mg/kg, 0.0072 mg/kg and 0.0024 mg/kg respectively. Five retailers had residues of 2,4-D amine a herbicide representing 10 % of the subject. 4(50) had residues of Lambda-cyhalothrin a synthetic pyrathriod. These non DAPs appeared to have the lowest detects values and percentages at mean concentration of 0.0024 mg/kg, 0.012 mg/kg and 0.009 mg/kg respectively.

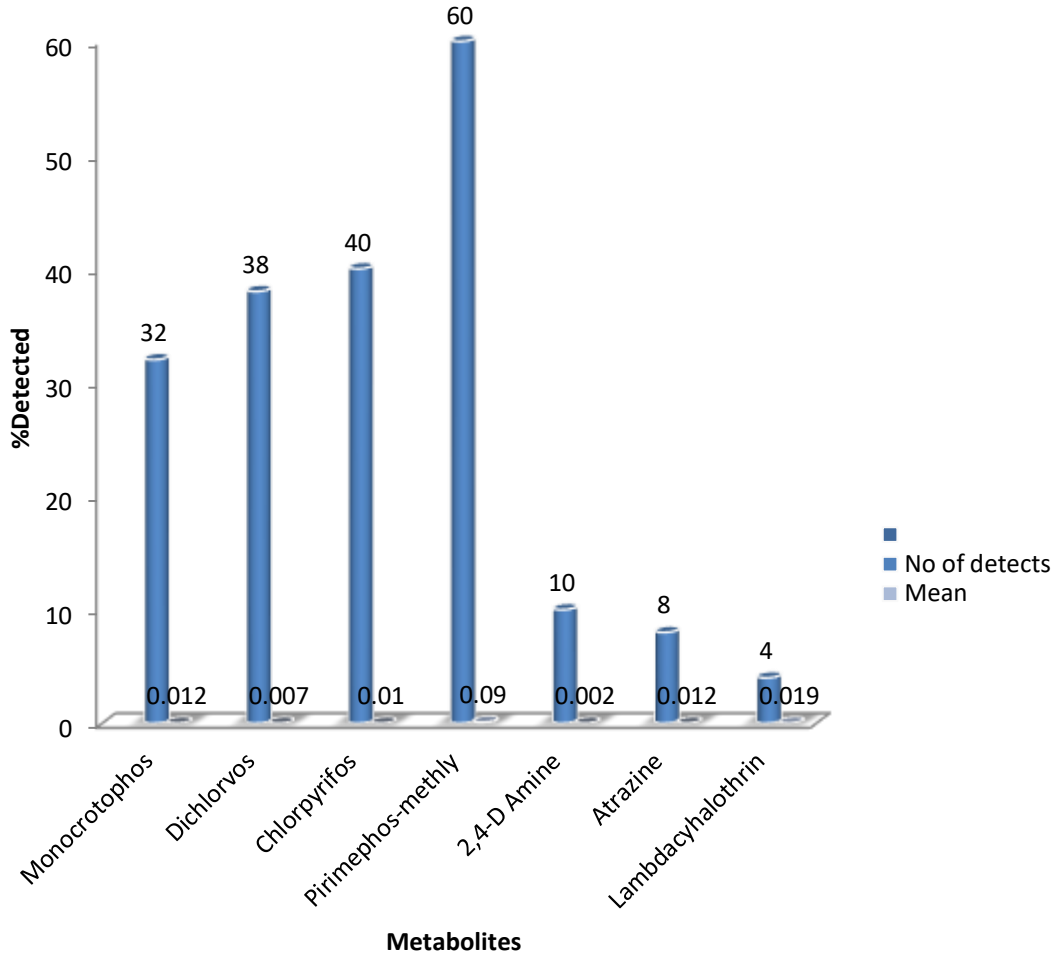


Figure 4. 2 % Pesticide Detects and metabolites

The major contributions to the total pesticide concentration in urine samples were of organophosphorus (OP) pestides. Common urinary metabolites that are identified after exposure to OP pesticide are DAPs metabolite that constitutes 75 % of the pesticide displayed for sales in their retail outlets. This is a pointer to occupational exposure.

Monocrotophos, an Op insecticide was detected in 32 % of the urine at mean level of 0.01 mg/kg. This insecticide was banned by National Agency for Food and Drug Administration and Control (NAFDAC) in Nigeria. It's classified as WHO 1b insecticide, highly hazardous and has been responsible for deaths resulting from accidental or intentional exposure, it is highly toxic orally, as well by inhalation or absorption through skin (WHO, 2006). It is capable of causing

acute as well as chronic intoxication. The joint FAO/WHO meeting on the join meeting on pesticide residue (JMPR) evaluated monocrotophos in 1972, 1975 and 1991.The acceptable daily intake (ADI) in man to be 0-0.00006mg/kg, comparing the residue level with the no observable adverse effect level (NOAEL). The residues of monocrotophos in the urine far exceeded NOEAL of 0.01mg/kg.

Chloripyfos was detected in 40 % of the urine sample. The mean concentration of the pesticide residues analysed exceeded the NOAEL of 0.01mg/kg. An earlier study demonstrated a correlation between prenatal chlorpyrifos exposure and children low weight and smaller head circumference at birth and also shows a strong correlation to chronic illness association with autoimmune disorders (Thrasher *et al.*, 2002). This is critical in that pregnant women and lactating mothers involved in the sales of agrochemical are at risk of this exposure.

Dichlorvos, a WHO class 1b OP insecticide, highly hazardous, LD_{50}-56 mg/kg body weight, with acceptable daily intake of 0.004 mg/kg body weight, found in 38 % of urine and was not detected in the wipe surfaces. This may be attributed to its volatility and its inability to withstand high temperature on surfaces.

It is worth noting that pirimiphos-methyl an OP insecticide had the highest level of detects 60 % in urine and at a mean level of 0.0118 mg/kg below the NOAEL of 0.03 mg/kg. lambda-cyhalothrin, atrazine and 2,4- D amine were among the detect analyzed, their respective mean levels were all below the NOAEL value, not withstanding, despite the low detects in urine, necessary steps need to be taken to prevent further accumulation of the pesticide in their bodies. Acute intoxication of DAPs is generally what is of immediate concern but this is also equally worry some as long term effect could results from the chronic accumulation.

4.2 Analysis of Pesticide Loading on Worktables and Floors in Retail Outlets

Figure 4.3 represent values of pesticide load on worktables and floors at various distances. Five major pesticide metabolites were identified, 60 % were detects of DAPs metabolites, 20 % herbicide and 20 % synthetic pyrathroids. The primary outcome measured were presence of at least one of DAP detected in wipes from all the surfaces analyzed, i.e. worktable, inside floors and outside floors. The floor inside the retail stores had the highest deposit of 41% pesticide load. The work table had 15 % deposit, the outside floor from the entrance door i.e. (5 m and 10 m) directly opposite and (5m and 10m) adjacent had deposit of 23 %, 13 % and 7 % 0.9 % respectively. The results revealed that floor samples analyzed at a distance of 5 m and 10 m directly opposite from the entrance door had higher percentages of pesticide levels of 22-9 % and 13-3 % respectively, while the floor surface at a distance of 5 m and 10 m directly adjacent to the entrance door had considerably lower and negligible pesticide load of 7.2 % and 0.9 % respectively. This also confirmed that the exposure pathway is minimal at a distance adjacent to the entrance door.

However, the study reveals that the retailers will be less exposed if their sales desk is position at a distance of 7-10 m adjacent to the entrance door rather than having it inside the retail store.

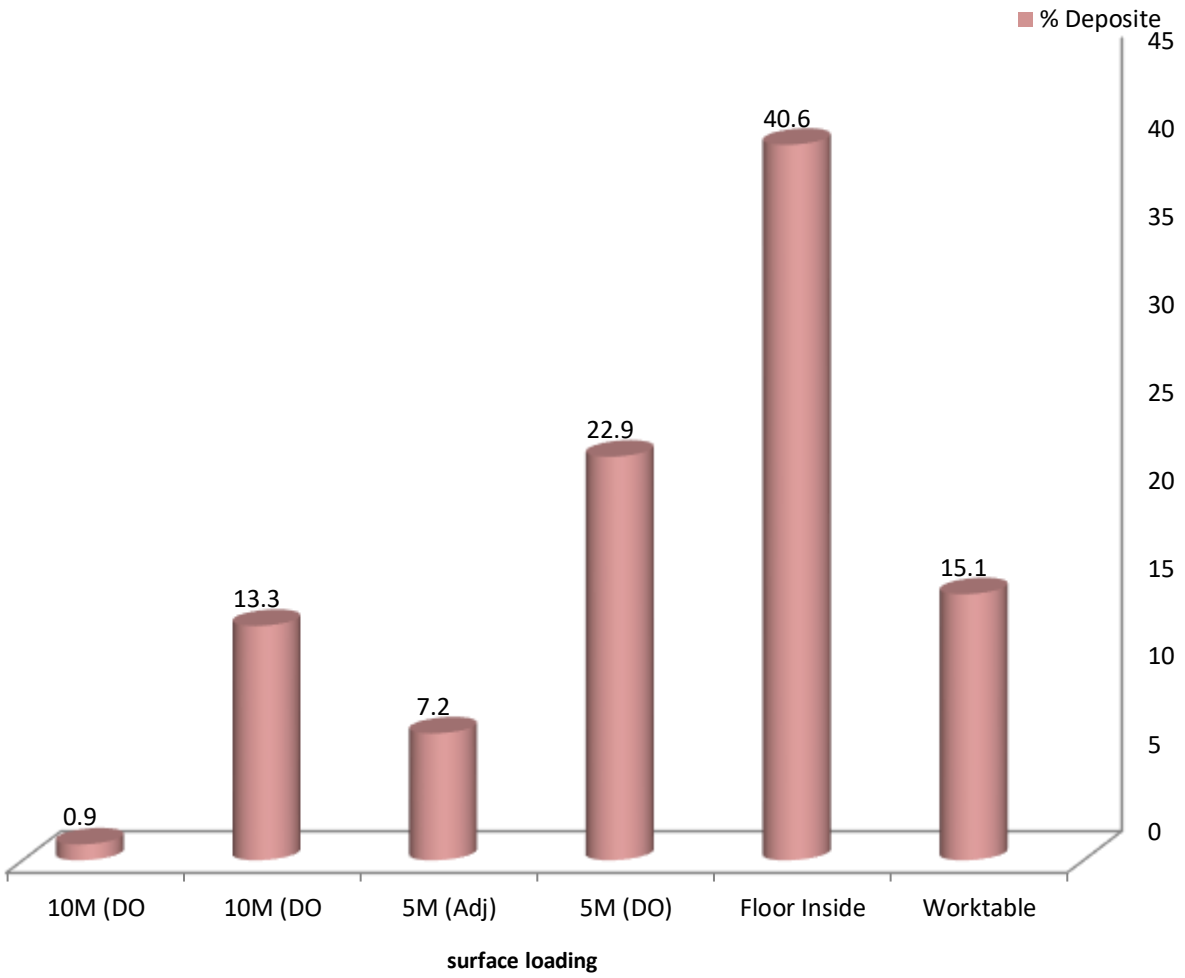


Figure 4.3 showing pesticide load on worktables and floors at various distance

Figure 4.4 shows the total metabolites deposits on surfaces in retail stores. Results from this study revealed that the total number of pesticide metabolites detected on surfaces of their worktables and floors by wipe sampling and indicating residues of monocrotophos, pirimiphos-methyl, chlorpyrifos, dichlorvos, 2, 4- D amine, atrazine and Lambdy-cyhalothrin; Primiphos-methyl and chlorpyrifos took the dominant lead with 32.6 % and 32 % respectively while monocrotophos (21 %), lambd-cyhalothin (13.3 %) and 2, 4 -D Amine with 1.4 %.It is interesting to note that the same metabolites that were evidence in the urine samples also find practical expression on the surface samples except for Dichlorvous and Atrazine that were not detected in any of the surface analysed. Pirimiphos-Methyl (40.6 %) and chlorpyrifos (15.1 %) had the highest percentage detects on all the surfaces analyzed by wipe.

The same OP metabolites also took the lead in the entire urine sample analyzed. This finding depicts strong positive association between pesticide load in work places and the amount of pesticide residue in body system. The highest level of detection was on the floor inside the store and considerably decreases as distance (directly opposite and adjacent) increase away from the entrance door. This also confirmed that there are some measures of pesticide drift emanating from the volatility of the pesticide constituent in the store due to high temperature profile of the region among other factors or the migration of the pesticide deposit by retailers on their clothing and shoes as they walk to and from the retail stores.

The findings from this study confirmed that the floors and worktable of the retail outlets are reservoirs of pesticide residues which indicate that each person might be exposed to and carries a body burden of multiple pesticides that might be due to a combination of direct and indirect exposure of these pesticides.To this end, critical measures should be made on how best to disposed the waste water and dust resulting from sweeping and mopping of the retail outlet daily.

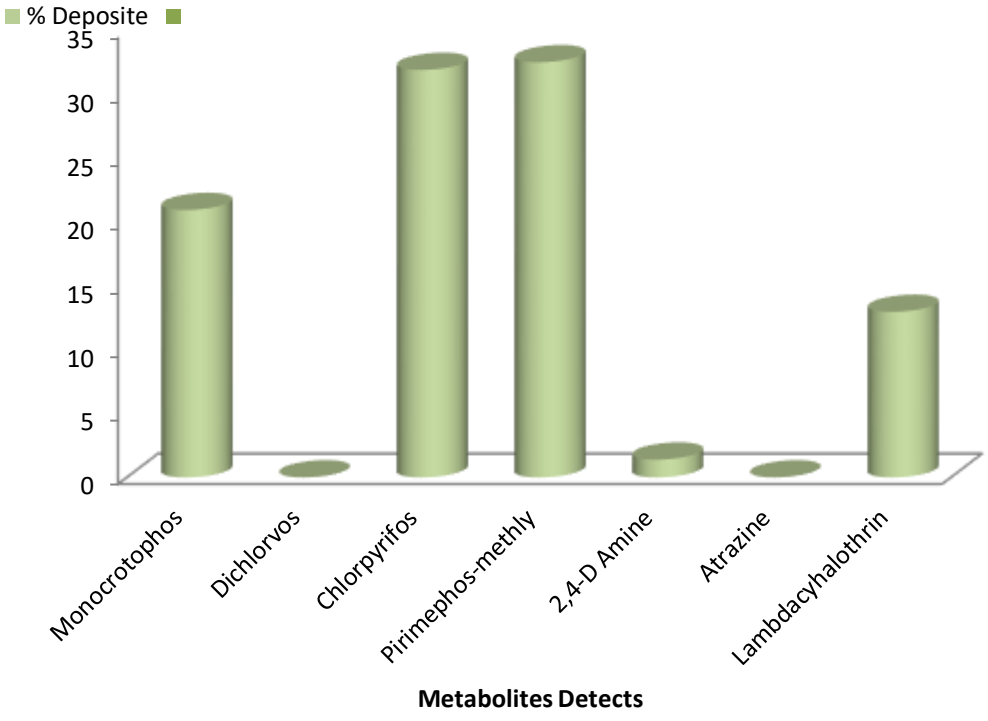


Figure 4.4 showing total metabolite deposits on surfaces in retail *stores*

4.3 Monitoring of Cholinesterase Inhibition with Symptom Prevalence

Figure 4.5 shows levels of educational background between the exposed and the control. The control groups for both sexes were older than the exposed. The control group have more education in the elementary, secondary and tertiary level ranging from 26 %, 52 % and 16 % as against the exposed group with 16 %, 31% and 3 % respectively as shown in Figure 4.5. The exposed group had the highest levels of uneducated participants (16 %) compared to the 6 % representing the control group.

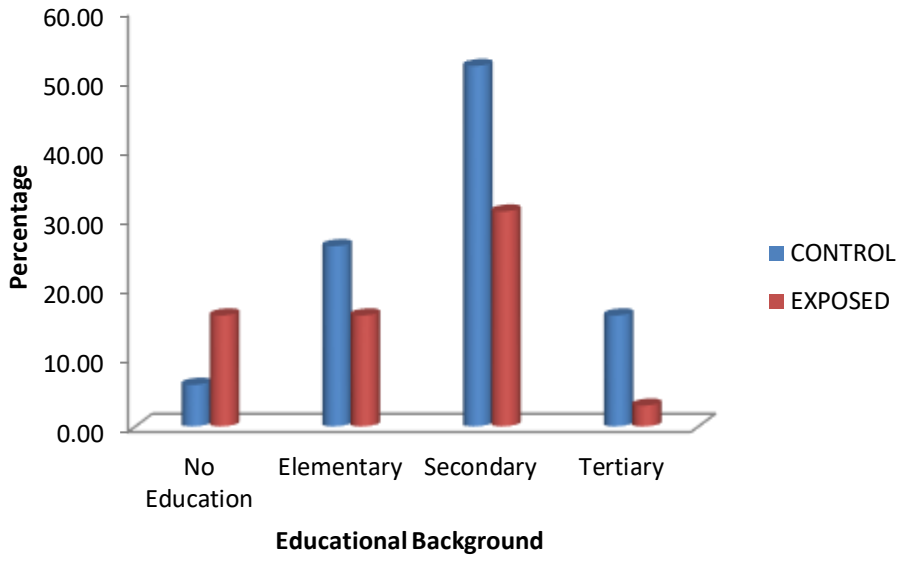


Figure 4.5 Showing levels of Educational Background between the Exposed and the Control

The overall statistics of the study group between the exposure and the control group shows that 19 out of the 50 of the exposed group and 5 of the 50 in the control where female in both categories with a mean age of 21 and 28 respectively.68 % of the exposed group eat and drink within the retail outlet as against 12 % recorded for the control. Only 8 %, 20 % and 10 % of the exposed group used overall, nose mask and hand gloves respectively when retailing pesticides as illustrated in Figure 4.6. This depicts the high percentages recorded in the control group having 36 %, 86 % and 44 % of the above protective apparel. A

cholinesterase monitoring is intended to prevent further exposure of workers with depressed cholinesterase activities, thereby preventing poisoning. The exposure to pesticide is widespread among retailers of agrochemical in North eastern Nigeria and the factors contributing to exposure such as the predominantly high temperature profile, humidity, volatility of the pesticide, design of the retail outlets, safety practice and hygienic behaviour among other factors may be different than report of exposure from farm workers, families and agricultural settings (Hotton *et al.,* 2010).

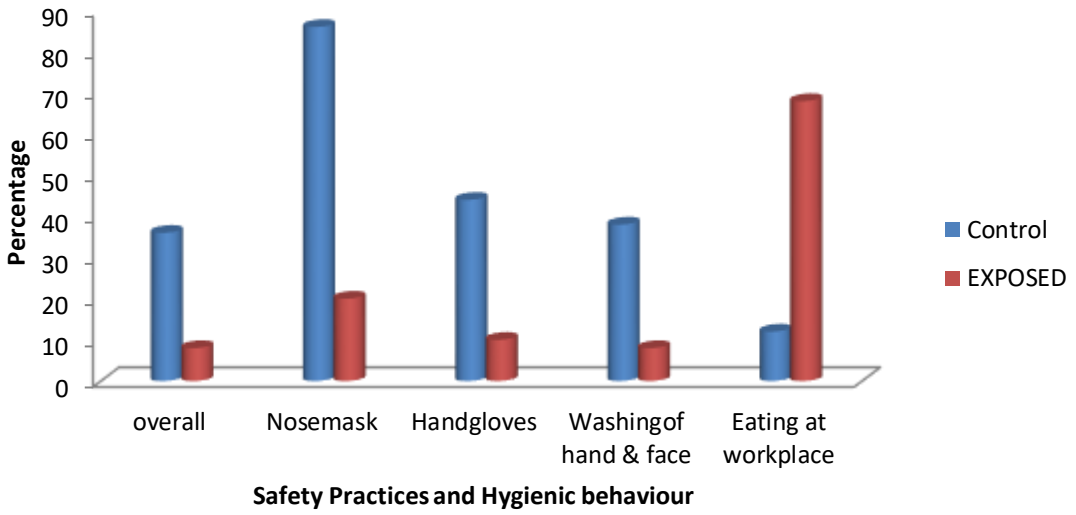


Figure 4.6: Showing % of Safety Practices and Hygienic behaviour for the Exposed and Control subjects.

Figure 4.7 shows the prevalence (%) of symptoms during high seasons for the exposed and control group. The first group comprised symptoms of the respiratory system included shortness of breath, running nose, irritation of the throat, chest pain, asthma, cough, thick sputum, wheezing, asbestosis, bronchilis, shortness of breath when washing or dressing, shortness of breath when walking fast, trouble smelling odour. Five out of the thirteen were positive representing 18.4 % of the exposed and 8 % of the control group.

Systemic symptoms included excessive salivation, poor appetite, vomiting, nausea, excessive sweating, diarrhea, burning on urination, abdominal pain. Three of the eight were positive representing 12.6 % exposed and 8 % control.

Cardiovascular or heart symptoms these include irregular heartbeat, high blood pressure, swelling in the legs and feet, frequent pain or tightness in the chest, heart skipping, heartburn; pain in the chest during physical activity.

Three out of the seven were positive representing 15 % and 5 % for both categories. The eye symptoms include irritation of eye, temporarily lost of vision, permanent loss of vision. One of the three was positive representing 15.2 % and 5 % for both categories. Symptoms of central nervous system (CNS) included restlessness, back pain, difficulty in moving arms and legs, weakness of arms, hands, legs, stiffeners of the waist, fatigue, difficulty in bending the knees, difficulty in moving the head up or down, difficulty squatting to the ground, difficulty in falling asleep, trembling of hands, difficulty in seeing .Five of the twelve were positive representing 16 % and 10 % for both categories.

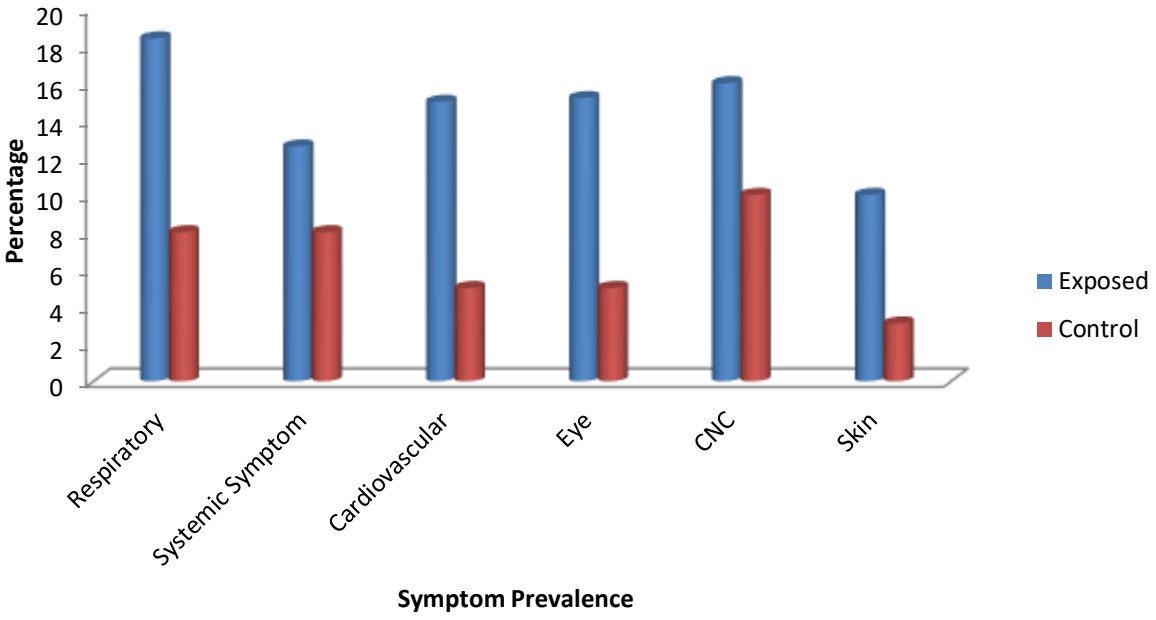


Figure 4.7 Illustrating Symptom Prevalence during the High Exposure period for the Control and Exposed subjects

Symptoms of the skin, included itching, numbness of hand, prickling of the skin, skin rash tingling of the face, cramps around the neck (Three of the six were positive)The Symptoms prevalence in all the categories were generally higher than in the control. The respiratory symptoms had the highest value of 18.4 % prevalence in the high exposure period and 9.6 % in the low exposure category. The symptoms were relatively lower in the low season for both categories as shown in Figure 4.8. CNS and cardiovascular symptoms in the exposed category had 20 % and 17.2 % respectively in the high exposure period with 10 % and 5 % for the control group.

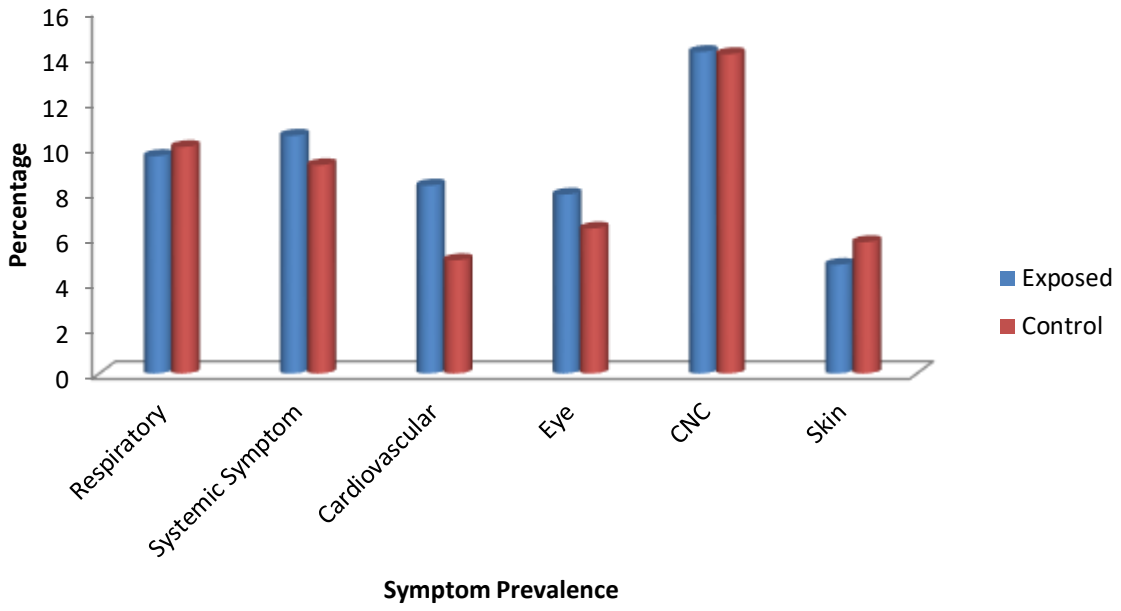


Figure 4.8: Illustrating Symptom Prevalence during the Low Exposure period for the Control and Exposed subject

The change in prevalence of symptoms in the controls from the high to the low exposure period is a key factor in explaining the relationship between activities during the high exposed period. During the high exposure period retailers spend more hours (8-9 hrs) daily retailing as a result of high volume of sales to farmers resulting to frequent contact of the hand with the pesticide container and also poor hygienic behavior could contribute to the change. Our findings reveals that

symptoms prevalence in the control during high exposure period were lower than those in the low exposure period, this however, pointed a significant difference in symptoms prevalence between exposed retailers of agrochemicals and the control during the high exposure period and also did not differ between these two categories during the low exposure periods. The low exposure period occurs during the harmattan and dry season of the year between November and March which is characterized by an extremely dry dusty wind that blows from the Sahara desert full of particulate and fug. The mist and high suspended particulate concentration might explain some of the respiratory and skin symptoms.

It could be argued that the increased symptom prevalence for some symptoms in both exposed retailers and control during the low exposure period is caused by unmeasured external non-occupational environmental factors such as climate (wind, humidity, temperature, fug, and dust). The high prevalence of symptoms especially for irritability, forgetfulness, fatigue, difficulties to fail asleep and restlessness could be explained by the high temperature during this period. Symptom in the control due to the high temperature and the environmental dust disappear during the high exposure period but remain in exposed retailers because of exposure to cholinesterase inhibiting pesticide.

It may be possible that some of the relations might have been confounded by some factors like the effect of smoking, residential pesticide for vector control. This however could have affected the relation found for respiratory symptoms and invariably the differences in smoking habit can account for the large differences in prevalence of respiratory symptom between the control and exposed retailers during the high exposure period.

Table 4.1 shows the variation of depression of cholinesterase level and rating of inhibition among exposed and control group. 42(50) exposed participants representing 84 % had change in acetylcholinesterase inhibition (≥30 %) while 5(50) representing 10 % in the control group had change in acetycholinesterase inhibition (≤ 30 %).The grouping on the basis of the year of involvement in trade, viz, 1-3 years,3-6 years,6-9 years,12-15 years,15-18 years and 18≥21years in the exposed categories had the following values of high inhibition (36.2 %,40.4 %,8.5 %,2.1 %,6.4 %,4.3 % and 2.1 %) respectively. The entire 19 participants in the

category 3-6years of the exposed group had the highest inhibition values of 40.4 %, while the grouping 9-12years and 18≥21years had the lowest inhibition values of 2.1 % inhibition each.

Table 4.1: Variation of the change of cholinesterase level and rating of inhibition among Agrochemicals retailers and the control group.

Depression of Cholinesterase (%)	Poisoning rating	No of Exposed participants' n (%)	No Control participants n (%)
1 – 10	Normal	NIL	45 (90)
11 – 20	Mild	8(16)	5(10)
21 – 30	Moderate	39 (78)	NIL
31 > 50	Severe	3 (6)	NIL

The present study found that agrochemical retailers in Northeastern Nigeria have considerable change in acetylcholinesterase inhibition and low acetylcholinesterase activities due to the handling and sales of pesticide during high exposure period in stores. 78 % of the exposed subjects during high exposure period had 20-30 % cholinesterase depression of baseline value while 3(50) retailers representing 6 % had 30-50 % depression. The results revealed that the agrochemicals retailers are occupationally exposed to cholinesterase inhibiting pesticide (Organophosphorus and carbamates) in their retailing activities.

This may be as a result of inhaling the pesticide vapour emanating from the containers due to the high temperature profile and humidity of the region, or dermal contact as a result of spill, accident or during the unethical dispensing of the pesticide from bigger containers into smaller aliquot as rightly observed during the

field survey among other factors. The control group had 90 % normal or no inhibition during the high exposed period with a cholinesterase depression level of 1-10 % of the baseline values, while 10 % had slight or mild inhibition with a depression of 10-20 %. This however, may be as a result of other factors order than direct exposure; it is unlikely that the dietary intake e.g. the eating of beans, dried fish and fruits preserved with pesticide or the use of residential insecticide for vector control may be responsible for such depression.

The finding of this study revealed also that the grouping based on the duration of years of involvement in trade shows the categories 1-3 years and 3-6 years had the highest percentages of inhibition with all the subjects having change in acetylcholinesterase activity. These group are mostly youth and younger than those in the other groups; the high inhibition rate may be as a result of their inexperience and lack of adequate safety measures in handling pesticide. This was obvious from our findings that only 8 % of the exposed retailers used overall, 20 % nose mask and 10 % hand gloves as personal protective devices as compared to the control group that had 36 %, 86 % and 44 % respectively. The hygienic behavior of the exposed retailers could be a contributing factor; it was revealed based on the findings that 68 % of the exposed retailers eat and drink within their retail outlet thereby making contamination via injection obvious. Most retail out lets had no facilities for washing in case of an emergency and the design of the stores does not allow for cross ventilation. Most retailers claimed that the design was adopted to ensure security of products.

The distribution of the exposed and control group on the basis of their levels of education shows that the control groups are more educated than the exposed; 16 % of the exposed had no formal education, all the uneducated retailers had high inhibition and a considerable drop of acetylcholinesterase activity in high exposure period. Their inability to read and interpret pesticide product labels on the handling, storage and hazard classification of the pesticide may be a factor that stimulated negligence to safety practices which might have resulted to their high inhibition compared to the 50 % with normal inhibition recorded for those with tertiary education.

The study also shows that relationship exist between change in cholinesterase inhibition and symptoms of respiratory, skin, central nervous system, eye and cardiovascular. Our findings supported the findings of previous study by Ohayo-mitoko (1999).The change in prevalence of symptoms in the controls from the high to the low exposure period is a key factor in explaining the relations between change in acetylcholinesterase activity and acetylcholinesterase activity during the high exposed period.During the high exposure period retailers spend more hours (8-9 hrs) daily retailing as a result of high volume of sales to farmers resulting to frequent contact of the hand with the pesticide container and also poor hygienic behavior could contribute to the change. Our findings reveals that symptoms prevalence in the control during high exposure period were lower than those in the low exposure period, this however, pointed a significant difference in symptoms prevalence between exposed retailers of agrochemicals and the control during the high exposure period and also did not differ between these two categories during the low exposure periods. The low exposure period occurs during the harmattan and dry season of the year between November and March which is characterized by an extremely dry dusty wind that blows from the Sahara desert full of particulate and fug. The mist and high suspended particulate concentration might explain some of the respiratory and skin symptoms.

It could be argued that the increased symptom prevalence for some symptoms in both exposed retailers and control during the low exposure period is caused by unmeasured external non-occupational environmental factors such as climate (wind, humidity, temperature, fug, and dust). The high prevalence of symptoms especially for irritability, forgetfulness, fatigue, difficulties to fail asleep and restlessness could be explained by the high temperature during this period. Symptom in the control due to the high temperature and the environmental dust disappear during the high exposure period but remain in exposed retailers because of exposure to cholinesterase inhibiting pesticide.

The study observed that percentage inhibition as well as acetyl cholinesterase activity was normal in the control during the high and low exposure period but change considerably over time in exposed workers. It may be possible that some of the relations might have been confounded by some factors like the

effect of smoking, residential pesticide for vector control. This however could have affected the relation found for respiratory symptoms and invariably the differences in smoking habit can account for the large differences in prevalence of respiratory symptom between the control and exposed retailers during the high exposure period. We deduced that increased symptom prevalence might occur at acetylcholinesterase activity that may not be considered adverse.

The level of cholinesterase inhibition and severity of behavioral symptoms among the retailers increases with increasing exposure time, poor hygiene practice and lack of protective clothing. The increased symptom prevalence was found at acetylcholinesterase activities which are considered to be no adverse compareD to the recommended WHO threshold of 70 % of baseline red blood cell. Retailers awareness of the hazard and route of entry of the pesticide should be increased by adequate enlightenment and training. Further research on the contribution of behavioral factors and exposure of agrochemicals retailers and families is needed in other to document the extent of the risk experience by the retailers.

4.4 Evaluation of Hexachlorocyclohexenane Isomers in the Blood of Retailers

The grouping of agrochemical retailers whose blood samples were taken for HCH isomers residues analysis is presented in Table 4.2.

The grouping shows the distribution of the retailers that had residues in their blood on the basis of their years of involvement in the sales of agrochemicals. Out of the 50 retailers assessed, 24 % of the retailers were within the 6-10 years and $\geq$ 21 years categories respectively, while 14 %, 18 % and 20 % were in the 16-20 years, 11-15 years and 1-5 years respectively. All the retailers in the grouping had residues of HCH isomers in their blood except for some few retailers. There were more male retailers 32 (64) in the study than the female 18 (36). Hexachlorocyclohexane (HCH) consists of eight isomers, only γ –HCH, α-HCH, ß–HCH, and δ- HCH are of commercial significant and considered in this profile as well as technical. Technical grade HCH is not an isomer of HCH but rather a mixture of several isomers. It constituted of approximately 60-70 % of α-HCH, 10-15 % of γ- HCH, 6 -10 % of

δ- HCH, 5-12 % ß –HCH, 3-4 % of ε – HCH and traces of λ-HCH. It has insecticide properties because of the γ- HCH Isomer.

Table 4.2: Number and presumed exposure period of agrochemical retailers in some retail Outlets with residues of HCH isomers in their blood

Duration of Exposure (Years)	No of Retailers Assessed n (%)	No. with Residue n (%)	No of Male with residue	No of female with residue
1 – 5	10 (20)	6 (60)	1 (16.7)	5 (83.3)
6 – 10	12 (24)	11 (91.7)	5 (45)	6 (55)
11 – 15	9 (18)	9 (100)	8 (89)	1 (11)
16 – 20	7 (14)	7 (100)	6 (86)	1 (14)
≥ 21	12 (24)	12 (100)	9 (75)	3 (25)

The high percentage of detect among the retailers could be linked to frequent exposure of the retailers to the pesticide during sales and unethical dispensing to smaller containers to peasants farmers who cannot afford to buy much. It may be attributed to the nature of HCH known for its bioaccumulation, long shelf life and persistence in fatty tissues and blood. The high percentage detects recorded calls for concern and steps need to be taken to prevent further residue accumulation in the body of the retailers. In developing counties agrochemical are carelessly handled and personal protective equipment as well as adequate clothing are seldom used by retailers and also lack of government and public concern, lack of stringent regulation, low standard of living, poor education and illiteracy in rural areas are factors that contributed to make occupational exposure to pesticide a major public health problem in less developed counties (Hotton *el al*., 2010)

The studies also reveals that all the female retailers representing 36 %, whose blood samples were measured in the present study had residues of HCH – isomers in their system, this however need to be caution in that, the storage of these toxins in fat is a problem of great concern in women because of their higher percentage of body fat, hormonal changes that occur during pregnancy, lactation and menopause result in mobilizing internal status of pollutants many years after exposure (Falcon *el al*., 2004). Torres *el al,* (2007) reported that newborns are exposed to pesticide through placenta transmission as well as breast feeding. HCH were progressively banished in most of the nations in the worlds, because of their persistence and their potential toxicity and carcinogenicity (Fabra *el al.,* 2005).

Farm works and retailers of Agrochemicals in the third world counties are at high risk for pesticide related illness. Generally, pesticide usage and handling is fraught with problem of undesirable side effect. Figure 4.9 shows the graphic mean concentration of HCH isomers detects in the blood samples of Agrochemicals retailers. Among the HCH isomers analyzed in whole blood samples, the mean levels of α, β γ and δ- isomers of HCH were 0.0087 mg/L, 0.0116 mg/L, 0.0180 mg/L and 0.0002 mg/L and ranged from n.d – 0.0511mg/L, n.d – 0.0411mg/L, n.d – 0.0901mg/L and n.d – 0.0500 mg/L respectively. The mean values of t – HCH, chlordane and Heptachlor were 0.0382 mg/L, 0.0017 mg/L and 0.0009 mg/L and ranged from 0.0100-0.1214 mg/L, n.d – 0.0017mg/L, and n.d-0.0059 mg/L

respectively.The residue analysis revealed that 45 of the 50 retailers representing 90 % had residues of HCH isomers in their blood. γ - HCH, commonly referred to as lindane, classified as WHO Class II (moderately hazardous) acute oral L $D_{50} \geq 50 \leq$ 250 mg/kg was detected in 74 % of the blood samples analyzed and invariably had the highest mean concentration of 0.0180 mg/L of all the isomers analyzed. This might be because γ - HCH is more resistant to biological and chemical degradation under aerobic conditions and is most commonly sold by the retailers. It appears in the list of pesticide under restricted (the use of lindane formulations generating smoke use is prohibited).

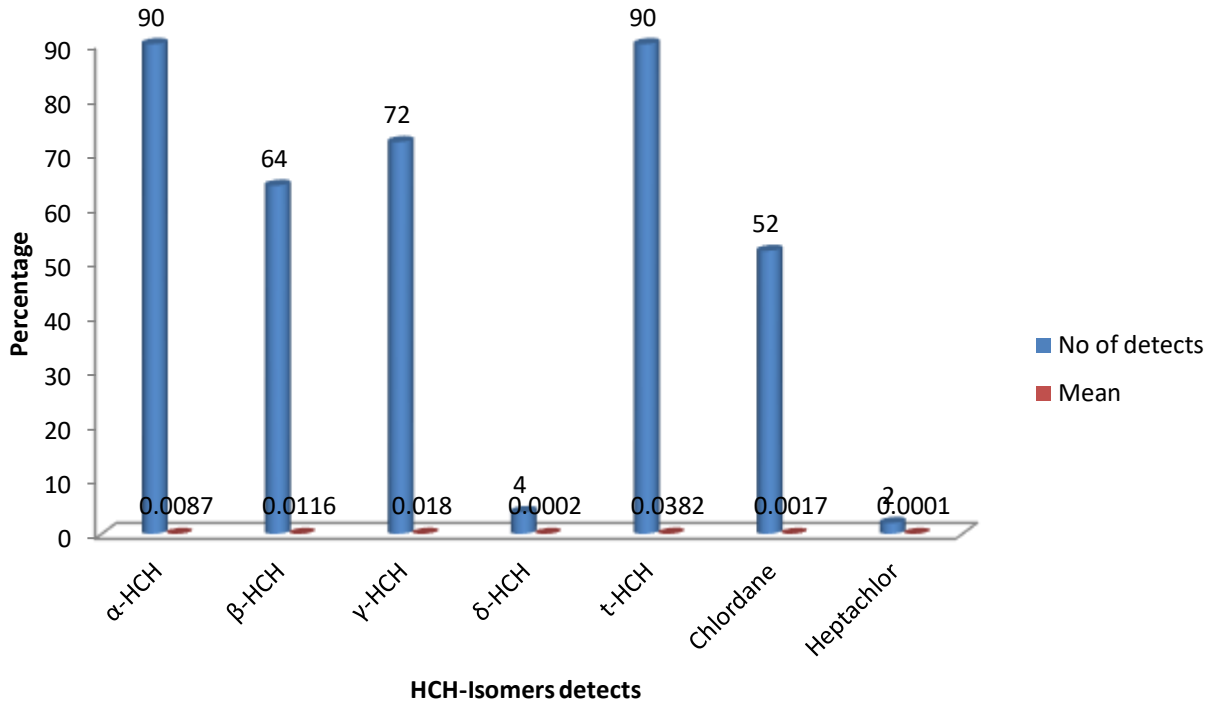


Figure 4.9 showing the % of HCH-Isomers detects and corresponding Mean

The overall variation of the mean concentration of hexachlorocyclohexane isomers detects (mg/L) among the grouping of agrochemicals retailers revealed the following mean concentration of α - HCH, β - HCH, γ - HCH, δ - HCH chlordane and Heptachlor residue for the grouping 1 – 5years, and 6 – 10 years were (0.0064 mg/L, 0.0034 mg/L, 0.0010 mg/L, n.d,0.0084 mg/L, 0.0020 mg/L and n.d) and (0.0072 mg/L, 0.0093 mg/L, 0.0110 mg/L, n.d, 0.0256 mg/L, 0.0003 mg/L,0.0001 mg/L and n.d) respectively. While the grouping 11 – 15 years, 16-20 years and $\geq$ 21 years had mean concentration of (0.0060 mg/L, 0.0132 mg/L, 0.00135 mg/L, 0.0001 mg/L, 0.00300 mg/L, 0.0032 mg/L and 0.0110 mg/L), (0.0113 mg/L, 0.0140 mg/L, 0.0118 mg/L, n.d, 0.0240 mg/L, 0.0014 mg/L and n.d) and (0.0148 mg/L, 0.015 mg/L, 0.0185 mg/L, 0.0010 mg/L, 0.0366 mg/L, 0.0104 mg/L and 0.0030 mg/L) respectively.

The results shows that the mean concentration of γ - HCH increases as the years of exposure increases across the distribution. The findings reveals also that there is a trend in insecticide accumulation in the blood of the retailers that seems dependent on the years of exposure which agrees with the report of previous research on cocoa farmers in West eastern Nigeria (Sosan *et al.,* 2008).Comparing the residue levels with the no observable adverse effect level (NOAEL) it can be deduced that the mean of γ - HCH in all the categories were far below the NOAEL. The codex /JMPR gave the Adverse daily intake (ADI) for humans estimated to be 0 – 0.008 mg/kg body weight (FAO, 1989). The mean values obtained ware lower than the levels previously reported in Delhi population which were 50 ng/ml (Nair *el al.,* 2006) and the studies of Pathak *el al.,* (2008) in North India population.

β - HCH was detected in 64 % of the blood samples analyzed at mean concentration of 0.0116 mg/L and ranged from n.d – 0.0411mg/L. β - HCH is only a minor component (5-6 %) of the technical HCH but it is the most persistent HCH isomer in the human body (WHO, 1992). It is eliminated more slowly from the body than the γ - HCH isomer. An MRL of 0.05 mg/kg / day has been derived for acute duration (14 days or less) oral exposure to β - HCH in rat. It accumulates 10 to 30 times more in fatty tissues than lindane. The high percentage of detect among the retailers could be linked to frequent exposure of the retailers to the pesticide during

sales and unethical dispensing to smaller containers to peasants farmers who cannot afford to buy much.

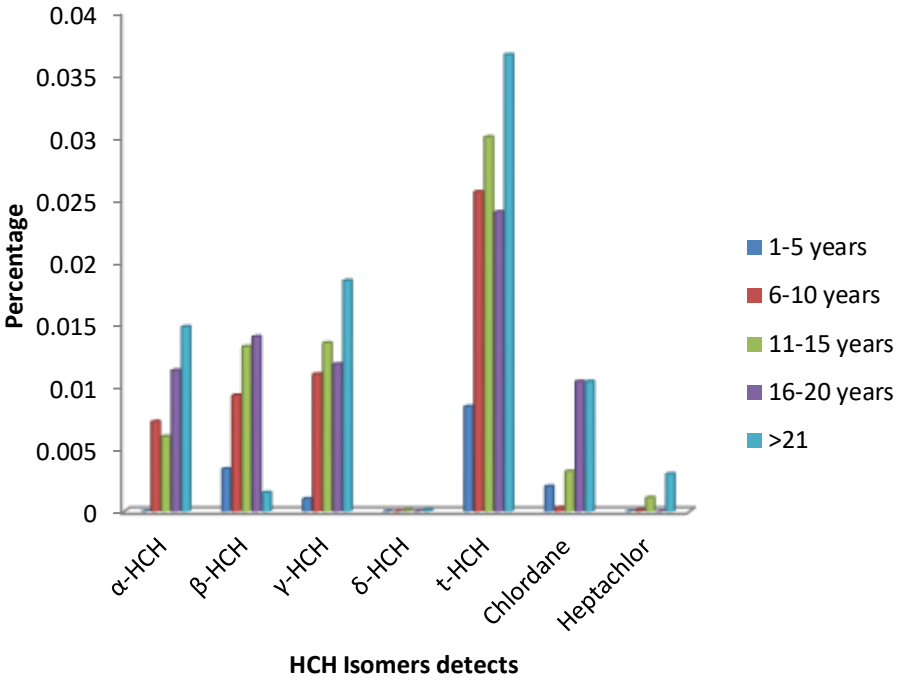


Figure 4.10 showing the distribution of grouping based on mean variation of HCH Isomers

The high percentage detects recorded calls for concern and steps need to be taken to prevent further residue accumulation in the body of the retailers. The values of β - HCH in the blood samples of agrochemicals retailers from Taraba were lower than values obtained from Punja as previously reported by Mathur *et al.,* (2005). The low mean values obtained may be a result of discontinuation of HCH use in public health programmers in Nigeria and restriction imposed on the use of these pesticides in agriculture. The studies also reveals that all the female retailers representing 36 %, whose blood concentration were measured in the present study had residues of HCH – isomers in their system, this however need to be caution in that, the storage of these toxins in fat is a problem of greater concern in women because of their higher percentage of body fat, hormonal changes that occur during pregnancy, lactation and menopause result in mobilizing internal status of pollutants many years after exposure (Falcon *el al.*, 2004). Torres *el al.,* (2006) reported that

newborns are exposed to pesticide through placenta transmission as well as breast feeding.

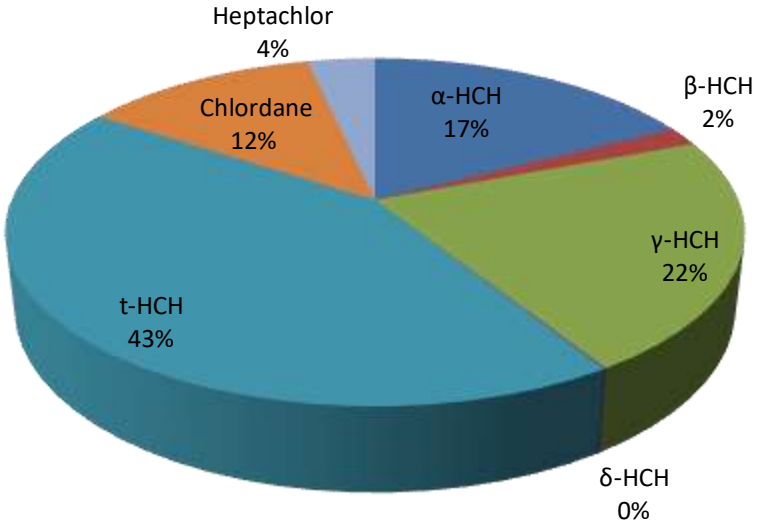


Figure 4.11 showing the percentages of mean HCH Isomers load among the retailers

α - HCH was detected in 90 % of the subject at mean level of 0.0087 mg/L and ranges from n.d-0.0511 mg/L. α - HCH and β- HCH are readily absorbed and may be toxic by mouth, by inhalation and by skin contact. They act primarily on the liver and the central nervous system. Symptoms of overexposure include decreased activity, trembling, dysphonic and convulsions (WHO, 2006). Interestingly the mean values in these studies were below NOAEL. The chronic oral Maximum Residue Limit (MRL) for α - HCH is based on a NOAEL of 0.8mg/kg/day and LOAEL of 3.5mg/kg/day for liver effect in rat. The 90% detects among the retailer is a pointer to occupational exposure in workplace.

This may be as a result of inhaling the pesticide vapour emanating from the contained due to high temperature profile and humidity of the region, or dermal contact as a result of spill, accident or during the unethical dispensing without protective clothing as rightly observed during the field survey among other factors. (Hotton *el al*., 2010).

Heptachlor is an organochlorine cyclodiene insecticide, a moderately toxic compound as classified under U.S EPA toxicity class II, used primarily by farmers to kill termites, ants and soil insects in seed grains and on crops. This was detected in the blood samples of one (1) retailer representing 2 % of the entire subjects, with a mean value of 0.0009mg/L and was categorized within the 11 – 15 year duration of exposure. It has an oral acute toxicity of LD_{50} 100 – 200 mg/kg in rat with an ADI value of 0.0001mg/kg / day. The mean values in this study were considerably low compare to the values obtained in Punja by Mathur *el al*. (2005). The low detects among the retailers may be attributed to its restriction in Nigeria. Health effect due to heptachlor exposure may include hyper excitation of the central nervous system, liver damage, and lethargy, in coordination, tremors, convulsion, stomach pain and coma.

Chlordane is an organochlorine pesticide. EPA has classified it as a Group B2, probable human carcinogen. Detected in blood samples of 26(50) retailers representing 54 %, at mean concentration of 0.0017 mg/L and ranged from n.d – 0.0017 mg/L. Despite the restriction of this pesticide .It worth nothing that its residues still reflect in the grouping 1 – 5years, 6 – 11years, 16 – 20 years and $\geq$ 21 years. The results revealed that this product is still in circulation. An occupational study reported an association between chlordane exposure and non-Hodgkins lymphoma, while animal studies indicate that single doses of chlordane administer orally are well absorbed extensively metabolized, distributed throughout the body, almost completely eliminated in 7 days and leave residue in several tissues predominately fat. Documented health problem can include child cancer, neuroblastoma, leukemia, chronic infection, bronchitis, asthma, infertility, neurological disorders, aggression and depression (CDC, 2005).

A survey conducted by U.S Food & Drug Administration (FDA) determined daily intake of chlordane form food to be 0.0013 µg/kg body weight for infant and 0.0005 – 0.0015 µg/kg body weight for teenagers and adult. The occupational safety & health Administration (OSHA) maximum allowable level of chlordane in workplace air is 0.5 mg/m^3 for a person who is exposed for 8 hours per workday.

Unfortunately, due to the lack of obvious odor of chlordane or easily administered test most retailers are unaware of it in the indoor air they breathe hour after hour (ATSDR 1994).δ - HCH was detected in two samples representing 4 % of the samples analyzed. The observed trends for t-HCH isomers are comparatively lower than the report from Punja (Mathur *et al.*, 2005)

The result of the present study confirmed, that the total number of hexachlorocyclohexane isomers detected in the blood samples of agrochemicals retailers in Taraba, Nigeria indicate that each retailer is exposed to and carries a body burden of pesticide which might be due to a combination of direct and indirect exposure to the sales, storage and handling of pesticide in stores. The need however for retailer awareness on the hazard and safety measure to be intensified via adequate enlightenment and training by regulatory bodies and private sector partnership in Nigeria.

4.5 Levels of DDT and its Metabolites in the Blood of Agrochemicals Retailers

All the blood samples analyzed, the presence of dieldrin was not detected in any of the samples from the exposed retailers and however, it was excluded from the table. The grouping of agrochemical retailers whose blood samples were taken for DDT and its metabolite residues analysis is presented in Table 4.3

Table 4.3:

Number and presumed exposure period of agrochemical retailers in some retail Outlets in Taraba with residues of DDT metabolite in their blood

Duration of Exposure (Years)	No of Retailers Assessed n (%)	No. with Residue n (%)	No of Male with residue	No of female with residue
1 – 5	10 (20)	6 (60)	2 (33.3)	4 (66.7)
6 – 10	12 (24)	9 (75)	5 (55.6)	4 (44.4)
11 – 15	9 (18)	7 (77.8)	7 (100)	-------
16 – 20	7 (14)	5 (71.4)	4 (80)	1 (20)
≥ 21	12 (24)	6 (50)	5 (83.3)	1 (16.7)

The grouping shows the distribution of the retailers that had residues in their blood on the basis of their years of involvement in the sales of agrochemicals. Out of the 50 retailers assessed 75 % of the retailers were within the 6-10 years and 50 % ≥ 21 years categories respectively, while 71.4 %, 77.8 % and 60 % were in the 16-20 years, 11-15 years and 1-5 years respectively.

All the retailers in the grouping had residues of DDT isomers in their blood except for some few retailers. There were more male retailers with residues 23 (46) in the study than the female 10 (20). Serum levels of 1, 1, 1-trichloro-2, 2-di (4-chlorophenyl) ethane (DDT) and its metabolite were measured among agrochemicals retailers. Persistent organochlorine pesticide such has DDT (dichlorodiphenyltrichloroethane) and its metabolites have provided great benefits to human since their introduction in public health to control mosquito-borne malaria and in agriculture. DDT is highly hydrophobic, colorless, and with a weak chemical odor. It has a good solubility in most organic solvents which is readily absorbed through the gastrointestinal tract with increase absorption in fat. Their intensive use throughout decades and their high persistence, accumulation in food chains and in the human body especially in lipid-rich tissues has raised the interest in knowing the extent of their spread leading to investigation of the magnitude of their residues in all compound of the human environment.

DDT is classified as moderately toxic by US National Toxicology Programmed (NTP) and moderately hazardous by the World Health Organization based on the rat oral LD_{50} of 113 mg/kg. DDT metabolite in the present study was detected in 58 % of the total number of blood samples analyzed and appeared more frequent within the grouping of 6-10years and 11-15 years. The high rate of DDT detects within these grouping may be attributed to their persistence nature due to their slow decomposition rate, long half life and high stability in the environment. Studies have shown that exposure to DDT at amount that would be needed in malaria control might cause preterm birth and early weaning associated with endocrine distrupting properties. Mean levels of DDE, DDD.DDT, t DDT and Aldrin were 0.0470 mg/L, 0.0101 mg/L, 0.0120 mg/L and0.0045 mg/L respectively which values ranges from (n.d-0.034 mg/L, n.d-0.1120 mg/L and 0.0100-0.0265 mg/L) respectively. The total content DDT (sum of DDE, DDD and DDT) in the whole blood samples was 0.062 mg/L and ranged from 0.0008-0.185 mg/L.

Our findings shows that values and mean concentrations of the DDTResidues among the retailers were relatively low but not withstanding otherStudies have found that even low levels of DDT in umbilical cord, serum atbirth are associated with decreased attention at infancy (Sagiv *et al.*,2008) anddecreased cognitive skill at 4 years of age (Philibert *et al.,* 2009) while highexposure decreases in semen quality among men (De Jagar *et al .,*2006).However, a human epidemiological studies suggest that exposure is a riskfactor for premature birth and low birth weight and may harm mother ability to breast feed (Rogan and Ragan 2003), there are some evidence that the daughter of highly exposed women may have more difficulty getting pregnant (Cohn *et al.,* 2003). It can be deduced that exposure at any level is a cause of concern and a threat to public health.

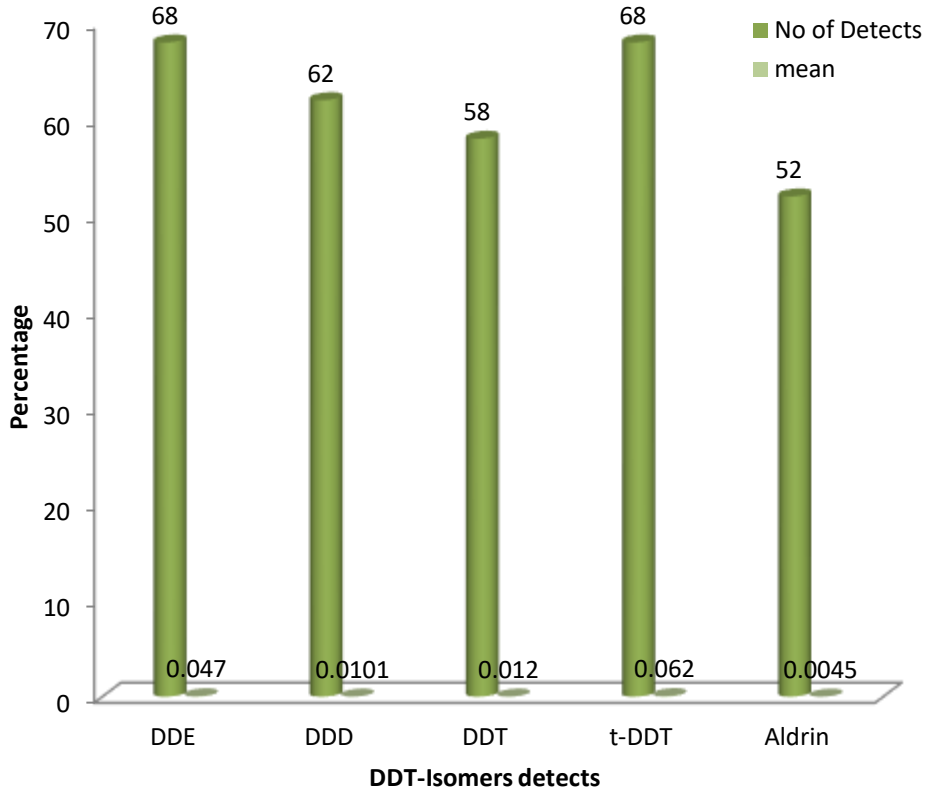


Figure 4.12 showing the % of DDT-Isomers detects and corresponding Mean

Potential mechanisms of action on humans are genotoxicity and endocrine disruption.DDT may be directly genotoxic but may also induce enzymes to produce other genotoxic intermediates and DNA adducts Coha *et al.,* (2007). DDT and DDE, specifically have been linked to diabetes. A number of studies from the US, Canada, and Sweden have found that the prevalence of thedisease in a population increase with Serum DDT or DDE levels (Jones *et al*., 2008; Turyk *et al* .,2009; Codru *et al*.,2007;Cox *et al*.,2007 and Philibert *et al*., 2009).

Toxicological evidence shows endocrine – disrupting properties; human data also indicates possible disruption in semen quality, menstruation, gestational length and duration of lactation (Rogan and Chen 2005). Several recent studies demonstrate a link between in utero exposure to DDT or DDE and development neurotoxicity in humans, a 2006 University of California, Berkeley study suggests that children exposed while in the womb have a greater chance of developmental problem and other studies document decreases in semen quality among men with high exposures (Jurewiez *et al*., 2010; Aneck *et al*., 2007; Dejar *et al.,* 206).
There is evidence of epidemiological studies that indicates that DDT causes cancrs of the liver, Pancreas and breast. There is mixed evidence that it contributes to leukemia, Lymphoma (Spinelli *et al*., 2007) and testicular cancer (McGlym *et al*., 2008).

Breast milk in regions where DDT is used against malaria greatly exceeds the allowable standards for breast feeding infants (Bovwman *et al*.,2006; Ntow *et al.,* 2008; Spicer and Kereu, 1993)It can be deduced that exposure at any level is a cause of concern and a threat to public health.DDE metabolite was detected in 68% of the blood samples analyzed at mean concentration of 0.0470 mg/L in the ranged n.d-0.462 mg/L classified by International Agency for Research on cancer as possible human carcinogen and also in the EPA class B2 probable carcinogens. We observed that the metabolite of DDE were the most predominant residue in the blood of the retailers. The high proportion and detect of DDE perhaps may be due to it more persistent nature than the DDT since DDT is known to undergo metabolic conversion and dehydrochlorination. The presence of metabolites of DDT i.e. DDE and DDD encountered in this study might be to such metabolic processed. The values obtained in this study were comparatively lower than the earlier reports from

India and Mexico on exposed population. Exposure to DDE were link between in utero exposure and developmental neurotoxocity in human studies demonstrated that children exposure while in the womb have a greater chance of developmental problem. Similarly, researchers have linked first trimester DDE exposure to retarded psychomotor development (Torres-Sanchez *et al.*2007).

Hence, the exposure of a pregnant retailer may invariably expose the baby in the womb to certain health hazard that may be harmful. The joint meeting on pesticide residues (JMPR) in 1984 gave an overall NOAEL for human as 0.25mg/kg body weight per day and the estimate of provisional tolerable daily intake for human as 0.01mg/kg body weight. The values obtained from this study were below the NOAEL.The values show that these pesticides are still in used.

The risk of bioaccumulation of these pesticides in the near future might be alarming. DDD was detected in 62 % of the blood samples analyzed at mean concentration of 0.0101mg/L. These low values may be attributed to the diminishing use of DDT in combating malaria vectors, thus a lower level of DDD was determined in the human body, which we considered an indicator of recent DDT exposure since DDD is determine in human body only for a short period immediately after DDT application or exposure. It can be deduced from this finding that the retailers were exposed recently to these banned pesticides in other wards they are still in circulation or may be attributed to dietary intake. Dieldrin a chlorinated hydrocarbon and extremely persistent organic pollutant, it metabolite was not detected in any of the samples analyzed.

The international Agency for research on cancer has stated that there is inadequate evidence of carcinogenicity in human beings and limited evidence of carcinogenicity in experimental animals. Both dieldrin and aldrin have been classified in Group 3; however the chemicals cannot be classified as to their carcinogenicity in human being but they are toxic and hazardous to humans. Aldrin was used to control soil pest namely termites and grasshoppers on corn and potatoes crop; we detected very low levels of aldrin in 52 % of the samples analyzed. Our data shows that the low values obtained by us (0.0042 mg/L) were lower than values previously reported in Punja which were 0.0062 mg/L.(Mathur *et al.*2005). Aldrin undergoes oxidation to give dieldrin. Over exposure symptoms may include

headache, dizziness, nausea, vomiting weakness in legs and stimulation of the central nervous system with jerks and convulsions sometimes leading to death.

Generally, the observed trend for t- DDT i.e. the summation of DDE, DDD and DDT were relatively lower than reports from Mexico and Ahmemdabed. (Bhatnagar *et al.,*2004). Exposure by the retailers may be via inhalation because of the volatility of the pesticide vapour within the indoor retail stores which are poorly ventilated, facilitated by the high temperature of the region and also via dermal contact as a result of frequent handling of the pesticide containers with hands or during the unethical dispensing of the pesticide from bigger containers to smaller ones. The general evaluation of the overall data strongly support the view that the body burdens resulting from the present level of exposure may constitute health risk for the agrochemical retailers and also the risk of bioaccumulation stand a point of concern. Despite the banned, the study shows that these pesticides are still in circulation among agrochemicals retailers in Nigeria. The need for stiff monitoring and enforcement by regulatory bodies is of paramount important.

4.6 Adsorption efficiency of Activated Carbon for Pesticide Vapour

Table 4.4 List the main physical properties and surface characterization of activated carbon used as adsorbents in this study. Physical properties of the adsorbents measured include micro pore surface area, total pore volume; micro pore volume, particle density and true density were measured. The surface area values of 1001 and 985 m^2g^{-1} were obtained by nitrogen adsorption using the BET equation for carbons prepared from coconut shell (CSA) and Borne char (BCA).The micropore surface area of carbons from coconut (CSA) shell was found to be 762 m^2g^{-1} larger than carbon from BCA (0.525 m^2g^{-1}).The micropore volume of carbon (CSA) appeared also to be

higher with 0.305 cm^3g^{-1} while the micropore volume of BCA yielded a lower value of 0.201 cm^3g .However, the values of total pore volume with the BCA have higher values of 0.5525 cm^3g^{-1} as against the 0.4271 cm^3g^{-1} CSA activated.

Table 4.4 Surface Characterization of Activated Carbons CSA and BCA used

Physical Properties	Activated Carbons CSA	Activated Carbons BCA
Total surface area (N_2 BET Method)(M^2g^{-1})	**1001**	**985**
Micropore surface area (M^2g^{-1})	**762**	**525**
Total Pore Volume (Cm^3g^{-1})	**0.4271**	**0.5525**
Micropore Volume (Cm^3g^{-1})	**0.305**	**0.201**
Particle density(gcm3)	**0.7672**	**0.6982**
True density (gcm3)	**1.9250**	**1.6947**

CSA=Coconut Shell 'Activated carbon BCA=Borne Charcoal Activated Carbon

Figure 4.13 Granulated Activated Carbon

Under an electron microscope in Figure 4.13, the high surface-area structures of activated carbon of CSA are revealed. Individual particles are intensely convoluted and display various kinds of porosity; there may be many areas where flat surfaces of graphite-like material run parallel to each other, separated by only a few nanometers or so. These micropores provide superb conditions for adsorption to occur, since adsorbing material can interact with many surfaces simultaneously.

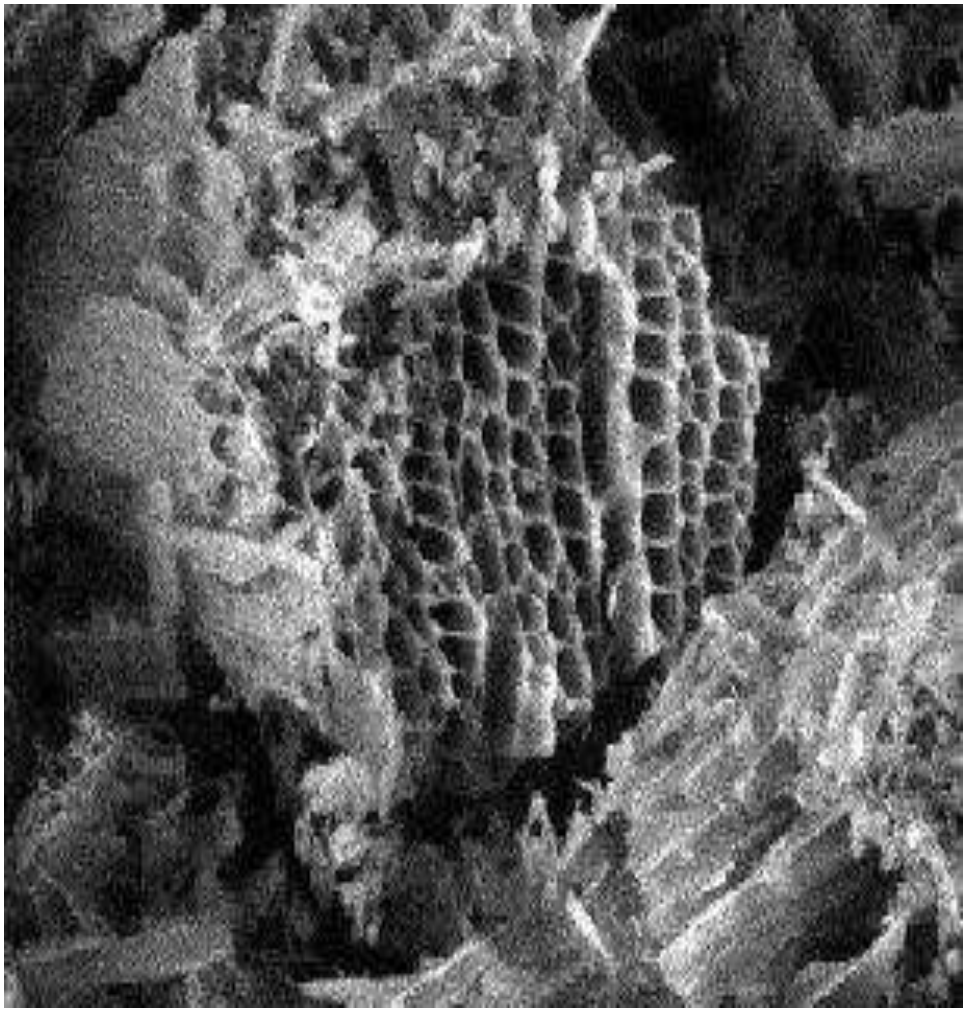


Figure 4.14 shows the electron micrograph of activated carbon of BCA.

The result obtained from the physical properties of the two adsorbent on the bases of surface characterization and the microscopic view shows that CSA has a large surface area properties over the BCA thereby stand to adsorbed more effectively than the BCA. However, the results indicate that the values of the surface areas, micropore surface area, micropore volume, particle density and true density support the fact that CSA in this studies exhibited a better heterogeneous distribution of surface energy for the adsorption of the adsorptive (pesticide vapour) and that the micropores are responsible for a large proportion of the surface area. The finer the particle sizes of an activated carbon, the better the access to the surface area and the faster the rate of adsorption kinetics..

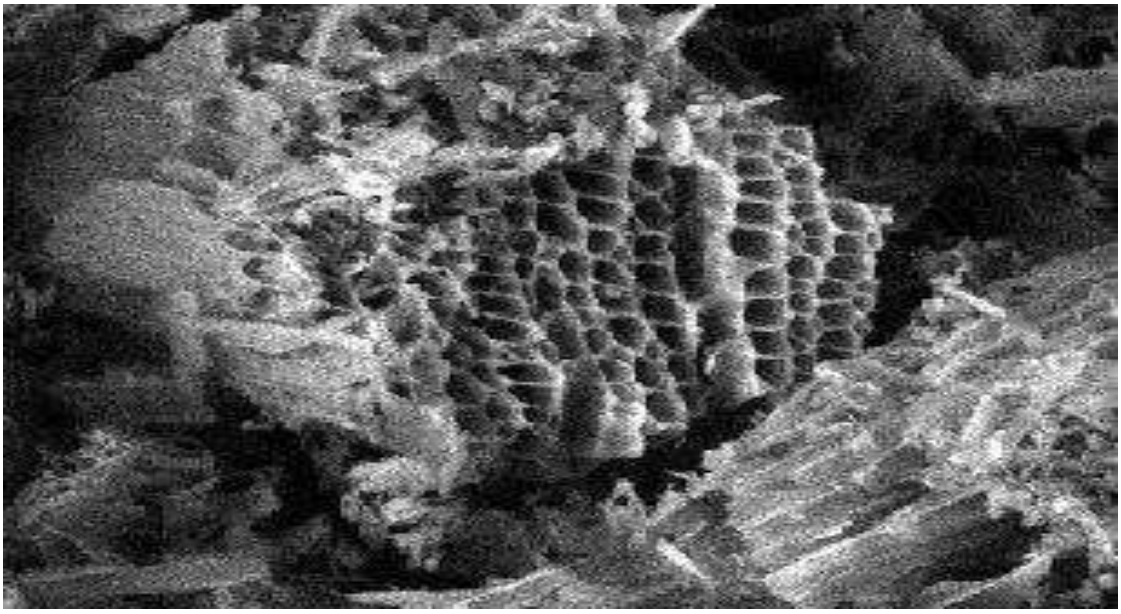


Figure 4.15 shows the electron micrograph of activated carbon of CSA.

According to the US Environmental protection Agency Toxic Release inventory (TRI). Under title III, that organic vapours are to be regulated, regulation requiring their emission sources to install maximum achievable control technology (MACTS). Current MACTS for organic vapors are condensation, incineration, carbon adsorption and liquid absorption. Of these technologies, carbon adsorption is a very common one because it offers some advantage over the others. The advantages include low cost, availability, the possibility of the recovery of raw materials or pure products for recycling the high removal efficiency (i.e.>95 %) at low inlet concentration, and the low fuel/energy costs (Ruddy and Carroll (1989); Spivey, 1988). Pesticides are semi-volatile organic compounds and include a variety of chemicals in various forms; exposure to it has been a concern to public health because of their persistence, toxicity and carcinogenicity (Febra *et al.,* 2005)

Table 4.5 shows the general trends of the adsorption capacities of the seven adsorbents at various concentrations and temperatures. Results show no peak detects on the chromatograms of the adsorbents of Corn cob, Kokoli, Saw dust, Murichi fiber and Cotton seed hence, the results were

presented as not detected (N.D) on the table. At a constant temperature of 20^{o}C upon various concentrations of 4 %, 0.8 %, 0.6 % and 0.4 % the adsorption capacity of CSA were 2.5 0 mg/L, 0.83 mg/L, 0.63 mg/L and 0.42 mg/L respectively while borne char (BCA) adsorption capacity at same concentrations and temperatures were relatively lower with 1.8 mg/L, 0.6 mg/L, 0.45 mg/L and 0.35 mg/L respectively. At 30^{o}C the adsorption capacity of CSA were 1.67 mg/L, 0.56 mg/L, 0.42 mg/L and 0.29 mg/L while that of BCA were 1.1 mg/L, 0.25 mg/L, 0.22 mg/L and 0.172 mg/L respectively. At 50 ^{o}C the adsorption capacity of CSA at the same condition were 1.0 mg/L, 0.36 mg/L 0.28 mg/L and 0.19 mg/L repressively, the corresponding value of BCA ranged from 0.75 mg/L 0.25 mg/L, 0.190 mg/L and 0.150 mg/L respectively. The result shows that the adsorptive capacity increases as the concentration increases at any given constant temperature for both adsorbents. On the same stratum, the adsorptive capacity increases as the temperature decreases and decreases as the temperature increases.

From the adsorption capacity of BCA and CSA as shown in Table 4.5 and the isotherm plots from figure 4.16-4.19, it can be deduced that the adsorption potential of CSA is of higher affinity than the BCA values. Higher density provides greater volume activity and normally indicates better adsorptive affinity of CSA over BCA. It was revealed also that the value of the adsorption capacity increases as the concentration increases at constant temperature for both adsorbent. However, the overall trend at various temperatures and concentration shows that the adsorption capacity decreases as the temperature increases. This also confirms the fact that the adsorption processes was of Vander Waals force of attraction, showing that physical adsorption has taking place on all surfaces.

4.6.1 Adsorption Isotherms

Since physical adsorption is non-specific every gas will adsorbed on the surface of active solid. How much gas adsorbed depends on the nature of the adsorptive, the surface area and temperature. Adsorption of pesticide vapour on CSA and BCA at various concentrations and temperature as contained in

fig 4.16 shows a linear relationship that fits both the theories of Freudlich adsorption isotherm and the langmuir equation.(Ching-Yuan,1994, Ruthven,1984 and Susaki 1991)

The Langmuir adsorption isotherm equation can be represented by

$$1/q = (1/q_m) + (1/q_m KC)$$

q = the adsorption capacity (amount adsorbed per Unit mass of adsorbent)
q_m = adsorption capacity at monolayer saturation
C = adsorbate equilibrium concentration
K = adsorption equilibrium constant

Table 4.5 Adsorptive Capacity of the Adsorbent Used

Temp (°C)	Conc (%)	CSA (mg/g)	BCA (mg/g)	Corn cob	Kokoli	Saw dust	Murichi fiber	Cotton seed
	0.4	0.420	0.350	N.D	N.D	N.D	N.D	N.D
20	0.6	0.63	0.450	N.D	N.D	N.D	N.D	N.D
	0.8	0.830	0.600	N.D	N.D	N.D	N.D	N.D
	4.0	2.500	1.800	N.D	N.D	N.D	N.D	N.D
	0.4	0.290	0.172	N.D	N.D	N.D	N.D	N.D
30	0.6	0.420	0.220	N.D	N.D	N.D	N.D	N.D
	0.8	0.560	0.250	N.D	N.D	N.D	N.D	N.D
	4.0	1.670	1.100	N.D	N.D	N.D	N.D	N.D
	0.4	0.250	0.180	N.D	N.D	N.D	N.D	N.D
40	0.6	0.360	0.250	N.D	N.D	N.D	N.D	N.D
	0.8	0.450	0.350	N.D	N.D	N.D	N.D	N.D
	4.0	1.250	1.200	N.D	N.D	N.D	N.D	N.D
50	0.4	0.190	0.150	N.D	N.D	N.D	N.D	N.D
	0.6	0.280	0.190	N.D	N.D	N.D	N.D	N.D
	0.8	0.360	0.250	N.D	N.D	N.D	N.D	N.D
	4.0	1.000	0.750	N.D	N.D	N.D	N.D	N.D

N.D = Not detected

Activated carbons made from coconut shell (CSA) and borne charcoal (BCA) were examined among other adsorbents for the adsorption of pesticide vapour conducted at adsorptive temperature of 20, 30, 40 and 50 ^{o}C.

Results from gas chromatograph and surface characterization revealed that the adsorptive capacities of carbon (CSA) at various temperatures were higher than that of carbon (BCA). Plots of Adsorptive Isotherm against Concentration at constant temperature for CSA and BCA as shown below.

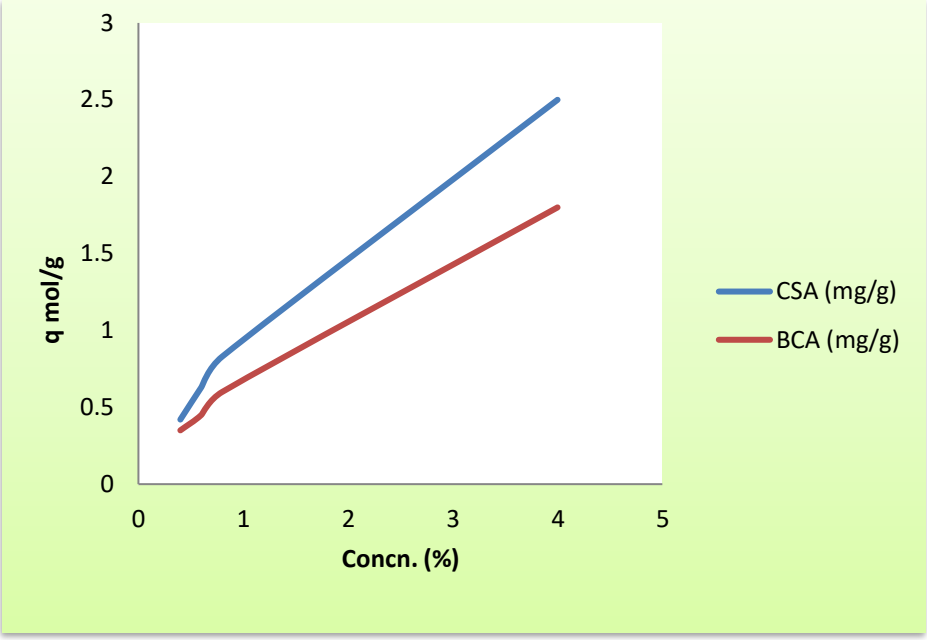


Figure 4.16 Adsorptive Isotherm for CSA and BCA at 20^{o}C

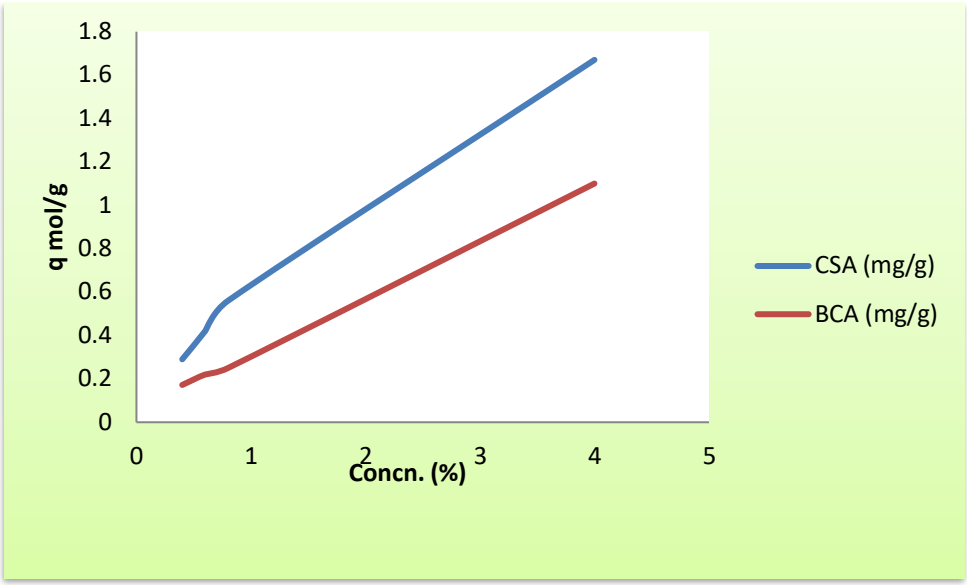


Figure 4.17 Adsorptive Isotherm for CSA and BCA at 30°C

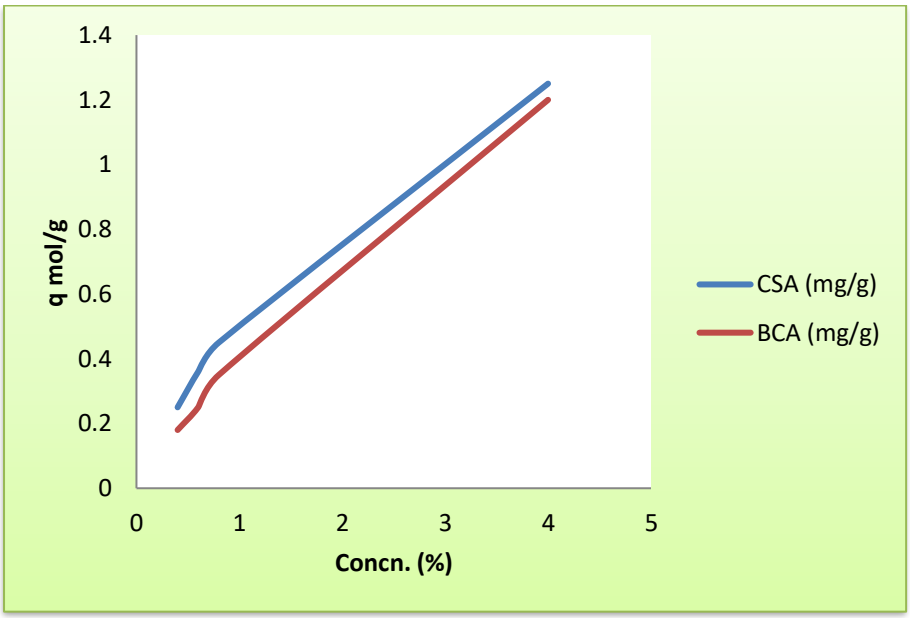


Figure 4.18 Adsorptive Isotherm for CSA and BCA at 40°C

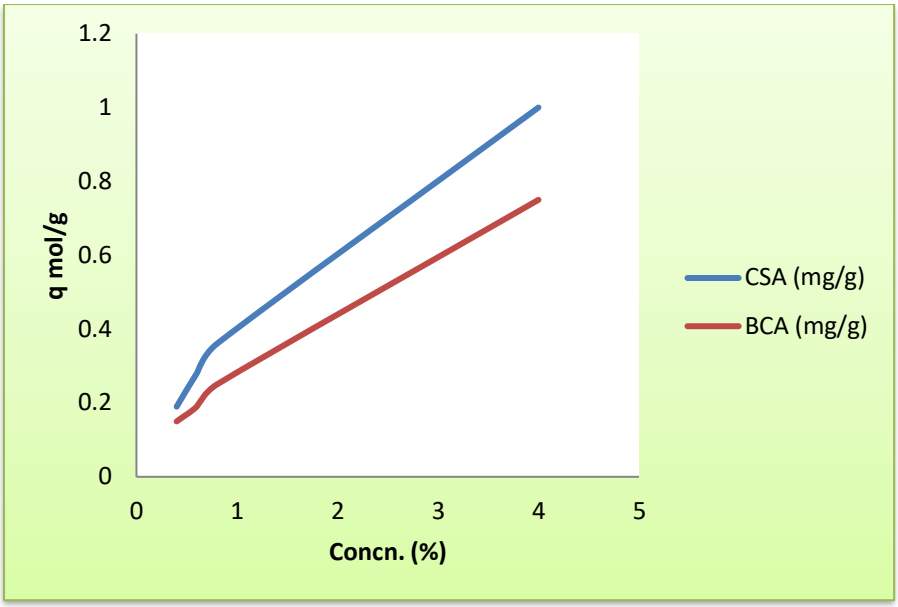


Figure 4.19 Adsorptive Isotherm for CSA and BCA at 50°C

A plot of (1/q) versus (1/c) for langmuir isotherm for both CAC and BCA yield

Slope = (1/q) k with $1/q_m$ = intercept

The plot of the Langmuir Isotherm shows a positive linear graph with the regression co-efficient justifying the fitness of the plotted experimental data which also agrees with the theory of adsorption as presented in figure4.20, 4.21, 4.22 and 4.23 for CSA and figure 4.24, 4.25, 4.26 and 4.27 for BCA .

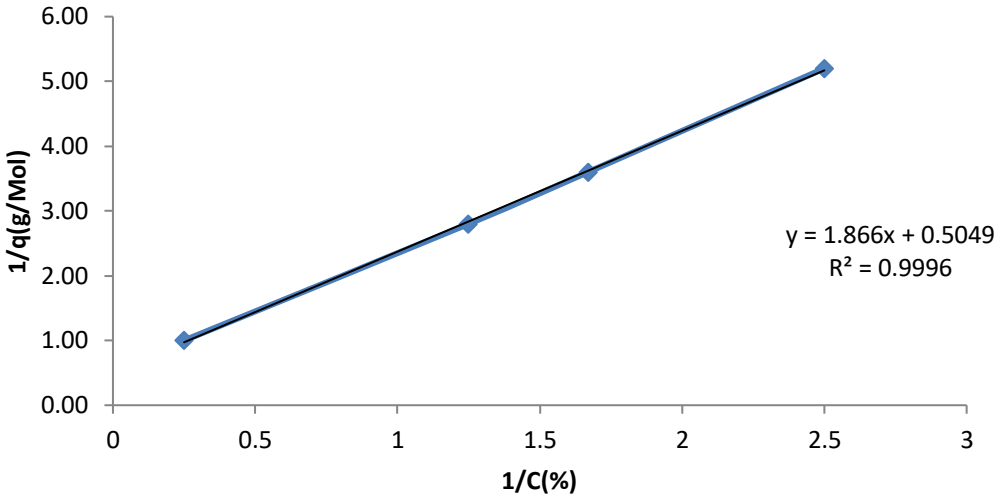


Figure4.20 Langmuir plots on carbon CSA at 50°C

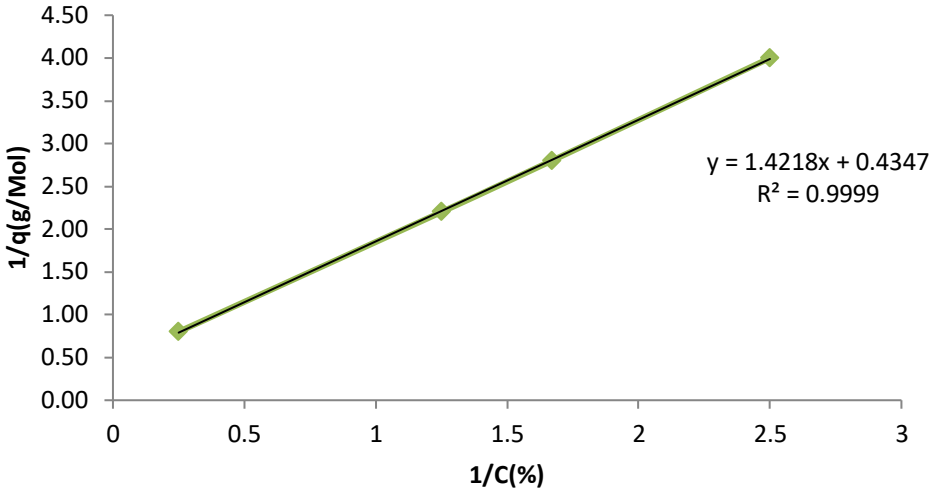


Figure4.21 Langmuir plots on carbon CSA at 40°C

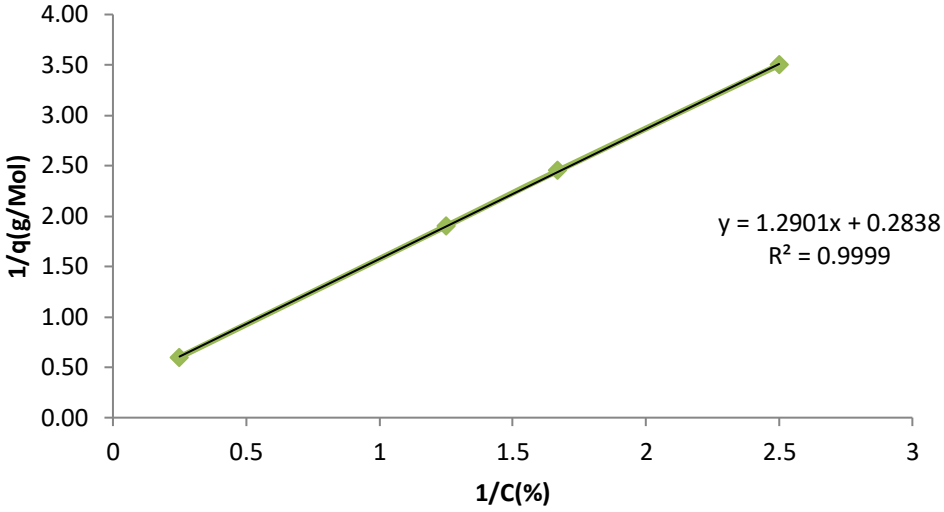


Figure 4.22 Langmuir plots on carbon CSA at 30°C

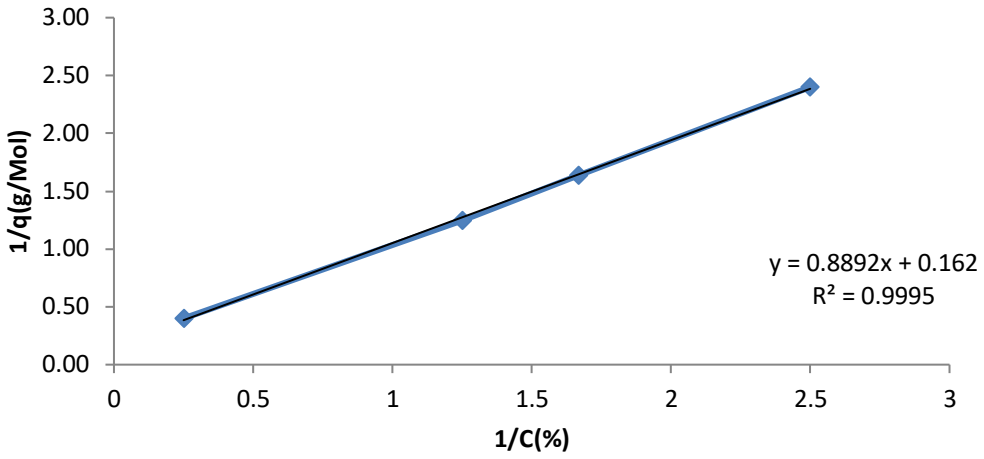


Figure4.23 Langmuir plots on carbon CSA at 20°C

Higher density and surface area provides greater volume activity and normally indicates better quality activated carbon .Physically, activated carbon binds materials by van der Waals force or London dispersion force. Micropores of CSA provide superb conditions for adsorption to occur, since adsorbing material can interact with many surfaces simultaneously.

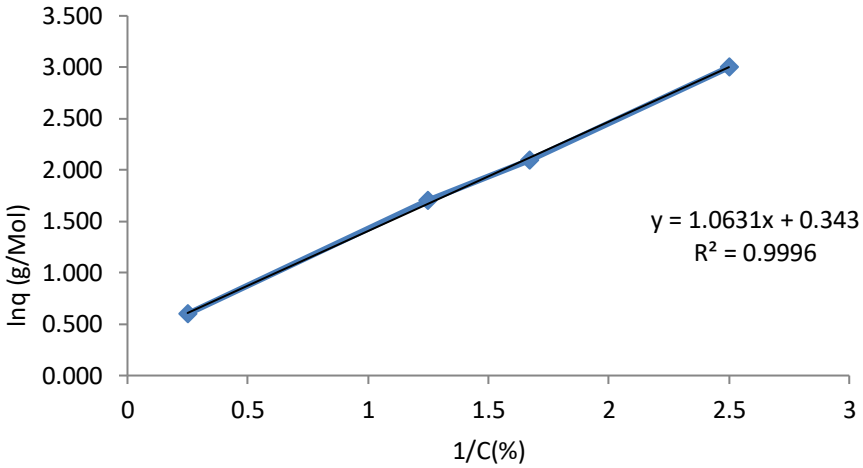


Figure4.24 Langmuir plots on carbon BCA at 20°C

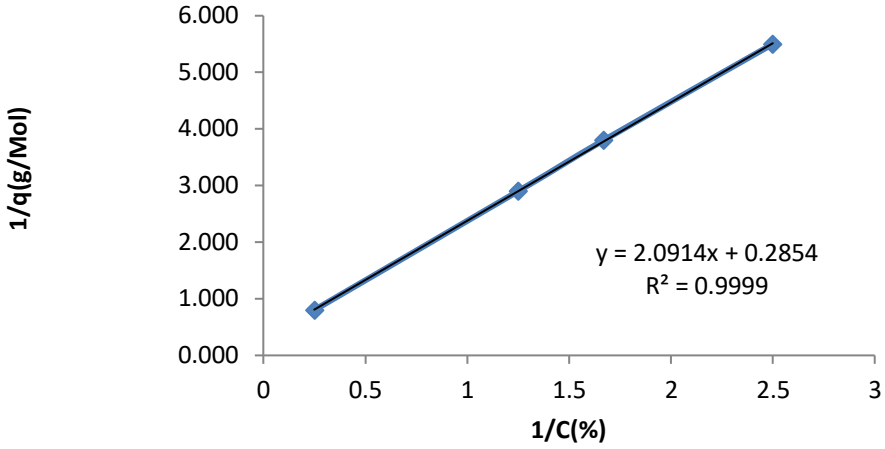


Figure 4.25 Langmuir plots on carbon BCA at 30°C

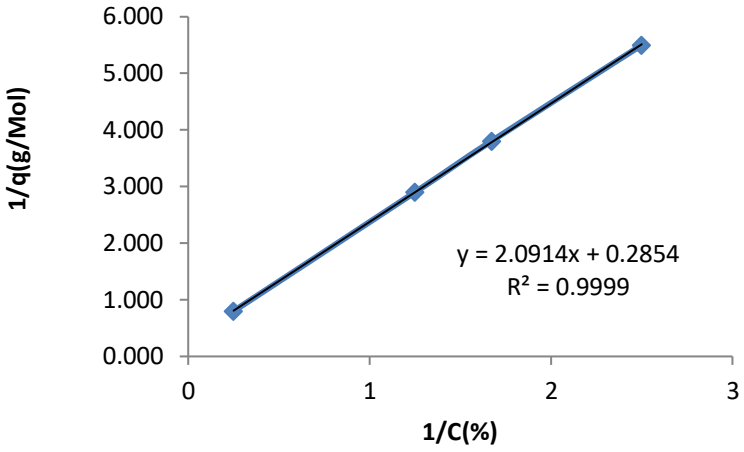


Figure4.26 Langmuir plots on carbon BCA at 40°C

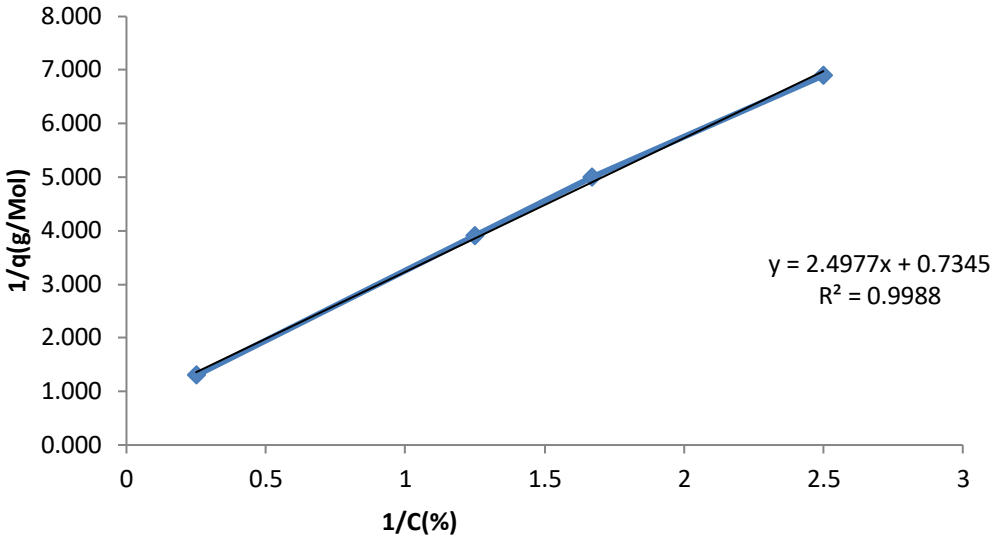


Figure4.27 Langmuir plots on carbon BCA at 50°C

The evaluation of the values of Langmuir adsorption isotherm parameter computed from the plot in figure4.16-4.19 and figure 4.20-4.33. It was glaring that the adsorption capacity of CAC based on the isotherm parameter buttressed the fact that the carbon of CAC adsorbed higher than the carbon of BCA in Table 4.6. This is also in agreement with values obtained in Table 4.5 that shows adsorption efficiency of CAC over BCA. The results from this study shows that the values of K decreases with increase temperature and K increases with decrease in temperature which confirms that the adsorbents displayed a higher adsorption affinity at lower temperatures.

Table 4.6: Langmuir Parameters for Adsorption of on Activated Carbon at Various Temperatures

Adsorbent	Adsorption Temperature (°C)	Langmuir Parameter q_m ($molg^{-1}$)	K	R^2
	50	11.85	3.10	0.999
	40	13.62	3.40	0.998
BCA	30	18.45	3.60	0.992
	20	20.6	3.80	0.996
CSA	50	19.8	3.27	0.999
	40	23	3.70	0.999
	30	35	4.55	0.999
	20	61.7	5.48	0.999

The Freundlich adsorption isotherm was also used whose equation was transformed into a linear by taking the logarithm on both sides.

Inq = Ink + (1/n) Inc Hence, using the values of q and c in Table 4.6 a plot of Inq versus Inc yielded a straight line graph as shown in Figure 4.28-4.35 respectively.

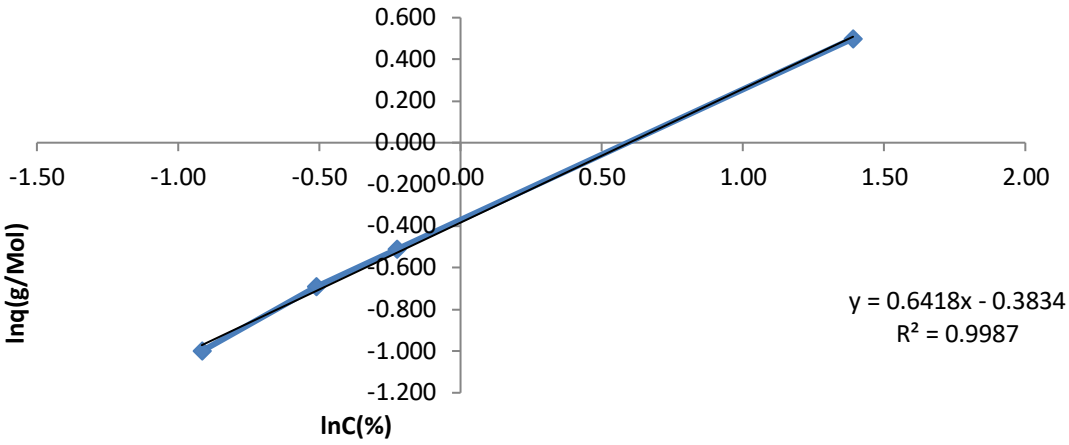


Figure 4.28 Freundlich plots of pesticide adsorption on carbon BCA at 50°C

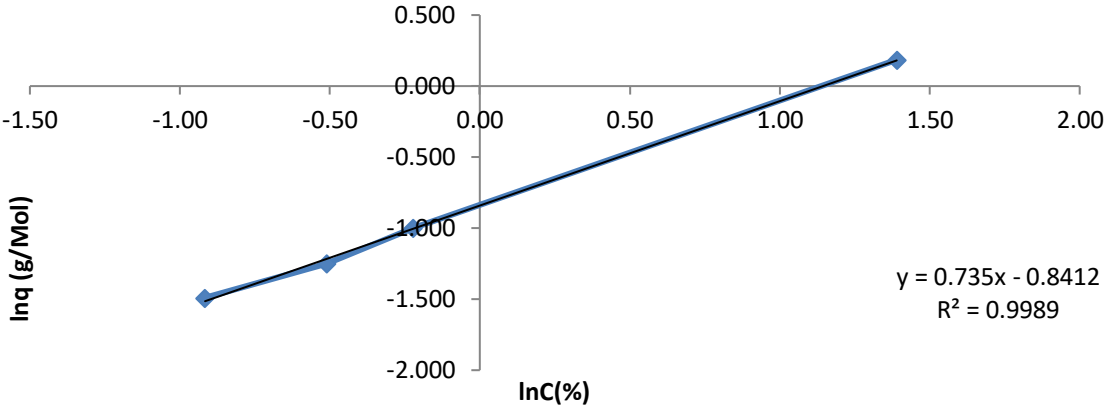


Figure 4.29 Freundlich plots of pesticide adsorption on carbon BCA at 40°C

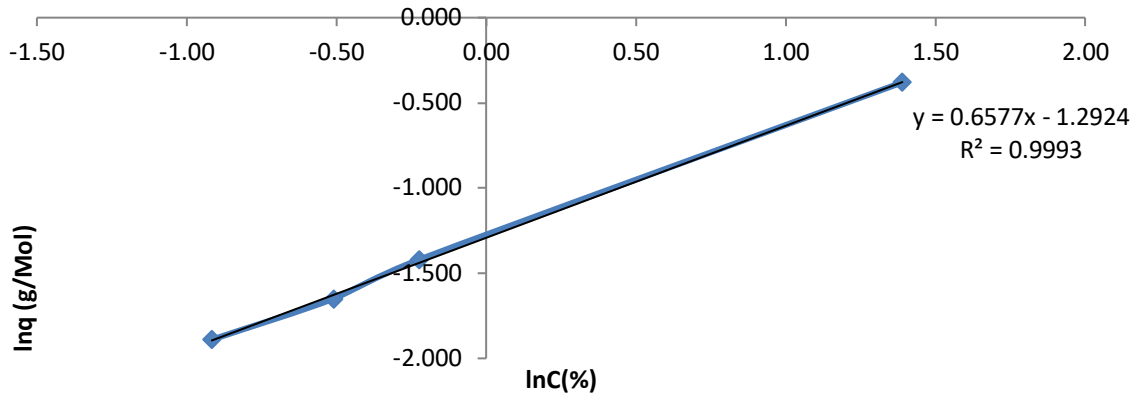


Figure 4.30 Freundlich plots of pesticide adsorption on carbon BCA at 30°C

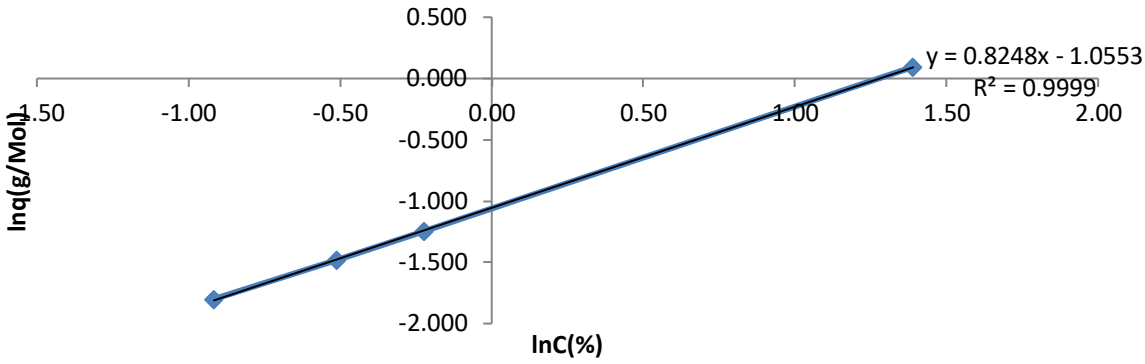


Figure 4.31 Freundlich plots of pesticide adsorption on carbon BCA at 20°C

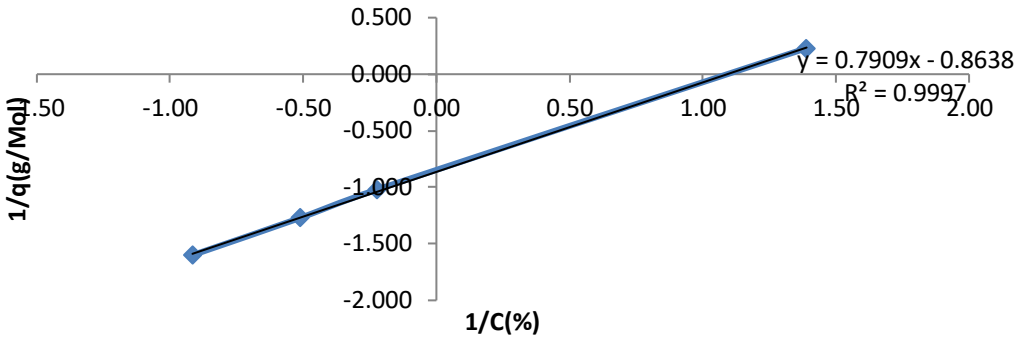


Figure 4.32 Freundlich plots of on carbon CSA at 20°C

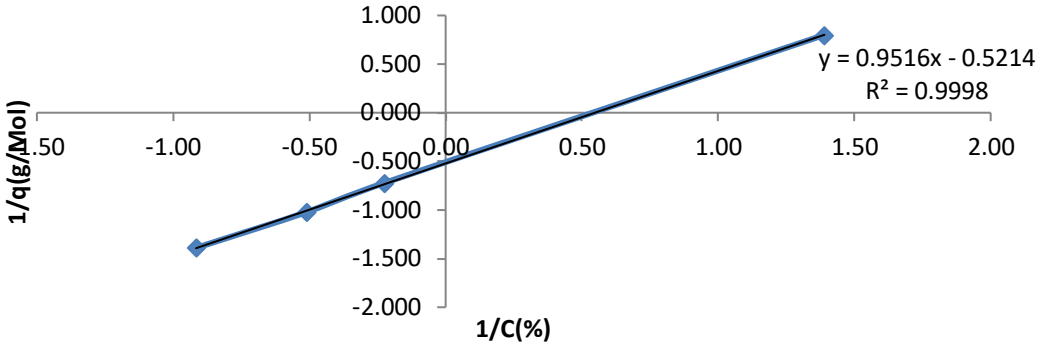


Figure 4.33 Freundlich plots of on carbon CSA at 30°C

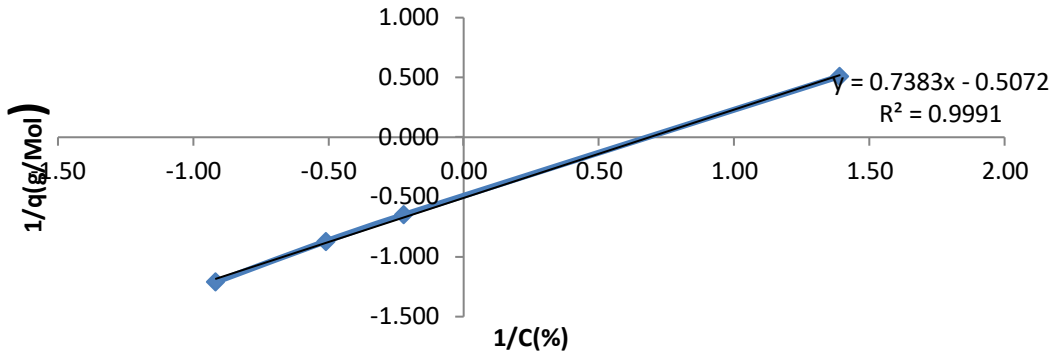


Figure 4.34 Freundlich plots of on carbon CSA at 40°C

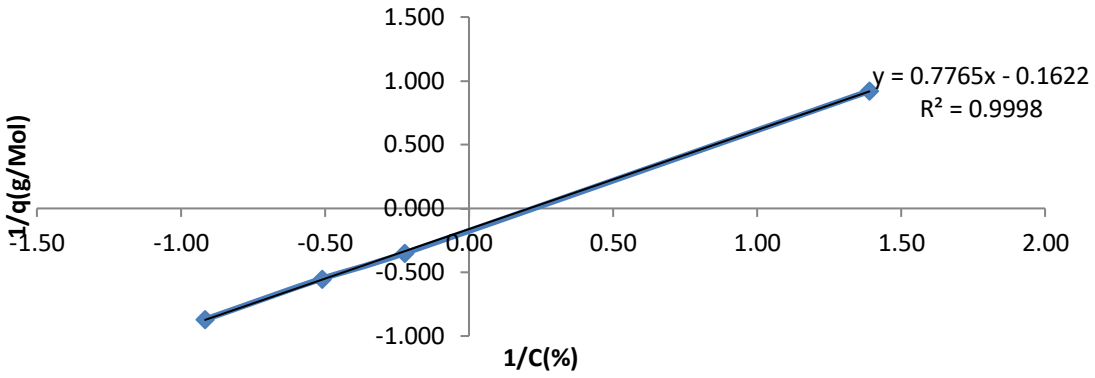


Figure 4.35 Freundlich plots of on carbon CSA at 50°C

This implies that the adsorption process conforms to Freundlich adsorption isotherm. The values of the parameter **K** and **n** obtained as shown in the Table 4.7 which shows that the values of the parameter n decreases with increasing temperature. A higher value of K increases the adsorption capacity of the adsorbent. The value of K also agrees that carbon CAC values were higher than carbon BCA at the same temperature.

Table 4.7 Freundlich Parameters for Adsorption of Pesticide on Activated Carbons at Various Temperatures

Adsorbent	Adsorption Temperature (°C)	Freundlich Parameter		
		K	n	R^2
	50	3.5	3.35	0.999
	40	4.31	3.89	0.998
BCA	30	2.7	4.57	0.996
	20	6.8	4.75	0.998
CSA	50	4.2	3.5	0.999
	40	5.9	2.9	0.999
	30	6.0	3.9	0.999
	20	8.5	3.6	0.999

The results confirmed and agreed also with our findings using the Langmuir adsorption isotherms, the Freundlich isotherm and the adsorption capacity. The overall results reveals that both adsorbent adsorbed better at low temperature of 20°C yielding high values of 61.7 mol/g at a monolayer saturation of CSA and 20.6 mol/g monolayer saturation for BCA as shown in Table 4.6. Relating the values of the monolayer saturation to the values obtained for the BET total surface area of CSA (1001) as shown in Table 4.4, it can be deduced that the surface area of CSA has the potential to absorb the pesticide vapour at an amount of 61,700 mol/g. This result is awesome, it implies that the adsorbent CSA will require longer duration of time for it to be saturated to that level if placed within the retail store or if in- cooperated into noise mask for the retailers to put on.

It is achievable to control and limits pesticide vapour to some considerable level in an indoor environment using carbon adsorption technology. Generally, from this study the adsorption capacity of carbon CSC is higher than that of carbon BCA which results agrees with the measured physical properties. The adsorption was more effective at low temperature and shows that the adsorption is of physical reaction that can be reversible.

CHAPTER FIVE
CONCLUSION AND RECOMMENDATION

5.1 CONCLUSION

This study has identified and monitored pesticide metabolites in the blood and urine of agrochemical retailers successfully in the Northern and Southern Senetorial districts of Taraba State Nigeria and also developed a novel (locally sourced adsorbent from coconut shell) to effectively adsorbed pesticide vapour in an indoor environment. Fifty (50) blood samples and fifty (50) spot urine samples from selected exposed Agrochemical retailers were analyzed for 15 organophosphorus and 14 organochlorine pesticides. The urinary pesticides metabolites were detected in 46 out of the 50 retailers selected representing 92 %. Among the fourteen OP analysed, four metabolites belonging to dialky phosphate (DAP) were commonly detected such as primiphos-methyl, chlorpyrifos, monocrotophos and dichlorvos. Pirimiphos – methyl had the highest detects amounting to 60% in the urine samples analyzed at mean level of 0.018mg/kg, which values were below the NOAEL of 0.03mg/kg and also had the highest deposit of 36.2 % on the retail surfaces analyzed. Chlorpyrifos was next with 40% in the urine sample ranged from 0.050-0.270 mg/kg, at a mean of 0.011mg/kg above the NOAEL values of 0.01mg/kg, and had 32 % on the surfaces. Dichlorvos and monocrotophos were detected in 38 % and 32 % at mean levels of 0.017 mg/kg and 0.012 mg/kg respectively whose mean values exceeded the NOAEL of 0.01mg/kg. 2,4. D- amine, atrazine and lambda-cyhalothrin were detected in (10 %), 8 % and 4 % respectively. Their mean values were found to be below the NOAEL.

The measures of pesticide loading on worktables and floors within and around the Agrochemical retail outlet were carried successfully. The floors inside the retail stores had the highest deposit of 40.6 %, worktable 15 %, floors surface at distances of 5m and 10m directly opposite and 5m adjacent to the entrance door had deposit of 22.9 %, 7.2 %, 13.3 % and 0.9 % respectively.Thus, results show that the pesticide load decreases as the distance increases away from the entrance door. Total pesticide load on surfaces by pirimiphos-methyl was 32.6 %, chlorpyrifos

(32 %), lambda-cyhalothrin (13.3 %), monocrotophos (21 %) and 2.4, D -amine (1.4 %). Dichlorvos was not detected many of the surface sample. The grouping of the agrochemical retailers for urine analysis shows that the grouping 1-3 years and 4-7 years had the highest number of detects in their urine amounting to 40 % and 30 % respectively.

The results of urine and findings on the deposition surfaces of the retail outlets show that agrochemical retailers in Taraba Nigeria are occupationally exposed due to the sales and handling of pesticide. These exposures might be minimal if their sales desk is position at a distance of 7-10 m adjacent to the entrance door. However, a better and safe retail outlet is advised. Results of the levels of organochlorine metabolites in blood samples of the exposed retailer reveals that total content of hexachlorocyclohexane (sum of α,β,γ and δ HCH) in the blood samples from the agrochemical retailers in Taraba was 0.0382 mg/L, γ isomer of HCH (lindane) was detected at a level of 0.0180 mg/L in blood samples and ranged from 0.0100-0.0382 mg/L .The results shows that 45 (50) retails had residue of α-HCH, β-HCH, γ -HCH and δ-HCH in their blood. Total content of DDT (sum of DDD, DDE and DDY) in blood sample from Agrochemical retailer was 0.0620 mg/L, mean levels of DDE, DDD and DDT in whole samples from Taraba was 0.0470 mg/L, 0.010/ mg/L and 0.0120 mg/L respectively. Detected in the blood of 34 retailer represented 68 % of the exposed subject. The total content of DDT (Sum of DDD, DDE and DDT) in blood samples from Taraba was 0.0620 mg/L which is comparatively lower than results in Punjab India of 0.065 mg/L.

The mean levels of DDE, DDD and DDT in the blood samples were 0.0470 mg/L, 0.010 mg/L and 0.0120 mg/L respectively. Aldrin, chlordane and heptachlor were detected in 63 %, 52 % and 2 % of the 50 blood samples analyzed from Taraba. Total number of pesticide detected in blood and urine samples from Taraba was 17 out of 30 pesticide analyzed which indicated that each person is exposed to and carries a body burden of multiple pesticide which might be due to direct and indirect exposure to these pesticides.

The major contribution to total pesticide load in the blood and urine from Taraba is of organophosphorus pesticide. The presence of cholinesterase inhibition in the blood means the presence of organophosphorus pesticide that they do persist

in the body for good amount of time. The research work show relationship between cholinesterase inhibition and organophosphorus pesticide, the findings indicate levels of cholinesterase inhibition with 78 % of the exposed participants having moderate poisoning rating of 20-30 % depression of control cholinesterase in their blood. The severity of behavioral symptoms increases with increase in the duration of exposure, poor hygiene practice and lack of protective clothing. The increased symptom prevalence was found at acetyl cholinesterase activities which are considered to be no adverse compare to the recommended WHO threshold of 70 % of baseline red blood cell.

The results of the present study shows levels of organochlorine metabolite in the blood of agrochemical retailers from Taraba, which reveals that 45 out of 50 exposed retailer have HCH in their blood. The total content of hexachlorocyclohezane (HCH) isomers (α sum of α, β, γ and δ - HCH) detected was 0.0382 mg/L. 72 % of the retailers have γ - isomer of HCH (lindane) which was detected at a mean level of 0.0180 mg/L and ranged from 0.0100 – 0.0382 mg/L. 90 % had α -HCH, 64 % β-HCH and 4 % δ-HCH.

This study examined various adsorbents for the adsorption of organophosphorus pesticide vapour using adsorbents.Some physical properties i.e. micro pore, surface areas, total pore volume, micro pore volume, particle density and true density were utilized. The activated carbon from coconut shell in this studies exhibited a better heterogeneous distributions of surface energy for the adsorption of the pesticide vapour and that the micropores are responsible for a large proportion of the surface area. The findings of the adsorption capacity via GC also confirmed and agreed also with the plots of Langmuir and Freundlich adsorption isotherm and their parameters.

It can be concluded from this study that it is achievable to control and limit pesticide vapour to some considerable level in an indoor environment using activated carbon made from coconut shell, which can be in cooperated into noise mask, or position in strategic areas within the retail store. This research work has identified and discovered some measures that will aid in the minimization and control of pesticide exposure for agrochemical retailers in an indoor retail store which include;

(a) The discovery of activated carbon made from coconut shell as a scientific control measure for the adsorption of pesticide vapour which is more effective at low temperature. This can be in cooperated into the noise mask or position in strategic areas within the retail store.

(b) Offers a structural control measures in the reduction of exposure pathways by repositioning the retailer's desk at a distance of 7 to 10 meters adjacent to the entrance door.

(c) Provision of some benchmark data on the intent and levels of body burden of organophosphorus and organochlorine pesticides and their metabolites among exposed retailers of agrochemical which may guide regulatory bodies and policy makers in evaluating risk assessment.

(d) Foster understanding on the nature and net impact of pesticide drift and distribution within retail outlets.

5.2 RECOMMENDATIONS

Pesticide storage, handling and usage are fraught with problems of undesirable side effects and food chain involvement. The need however for retailer awareness on the hazard and safety measures needed to be intensified via adequate enlightenment and training by regulatory bodies and private sector partnership in Nigeria.

(a) We recommend that further research should investigate the extend of saturation, re-generability and compatibility of in cooperating the activated carbon into face and noise mask

(b) Research on the contribution of behavioral factors and exposure of agrochemicals retailers and their families is needed in other to document the extent of the risk experience by the retailers

© We recommends also that for the health and welfare of retail workers and the general population, the handling and application of pesticide should be entrusted only to competently supervised and well-trained personnel, who must follow adequate safety measures and handle the chemical according to good application practices. Regular exposed workers should receive appropriate monitoring for cholinesterase test and health evaluation.

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