UNIVERSITY OF CAPE COAST

PESTICIDE RESIDUES IN WATERMELON FRUITS AND SOILS OF

NSADWIR IN THE CENTRAL REGION OF GHANA

EMMANUEL AGYAPONG ASARE

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PESTICIDE RESIDUES IN WATERMELON FRUITS AND SOILS OF NSADWIR IN

THE CENTRAL REGION OF GHANA

BY

EMMANUEL AGYAPONG ASARE

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DECLARATION

Candidate's Declaration

I hereby declare that this thesis is the result of my own original research and that no part of it has been presented for another degree in this University or elsewhere.

Candidate: ....   Date: -------------------------

Emmanuel Agyapong Asare

Supervisors' Declaration

We hereby declare that the preparation and presentation of this thesis were supervised in accordance with the guidelines on supervision of thesis laid down by the University of Cape Coast.

Principal supervisor: --------------------------------   Date: ----------------------

Very Rev. Prof .D.K. Dodoo

Co-Supervisor: -------------------------------------   Date: ---------------------

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ABSTRACT

Watermelon fruits and soil samples (depth of $0-20$ cm) were analysed for pesticide residues. Residues were extracted from 10.0 g of watermelon fruit using a multi-residue method for extracting pesticides from non fatty foods with acetone as the extracting solvent. Soil and okro from a nearby okro farm which were used as control to study the effect of pesticides on non target crops were also analysed. Extract clean up was done using a 10 mm chromatographic column with a 1:1 solvent mixture of Cyclohexane and Dichloromethane as the eluting solvent. Organophosphate pesticides (OPPs), organochlorine pesticides (OCPs) and synthetic pyrethroid pesticides were detected in all the samples.

The OPPs levels ranged from 0.90 - 4383.20 μg/kg. OCPs residues occurred in the fruits at levels between 0.70 - 34.50 μg/kg. Synthetic pyrethroid pesticides fell in the range 0.10 - 6.40μg/kg. Non-target crop had residue levels ranging from 2.80 - 2016.80 μg/kg for OPPs, 1.20 - 15.83 μg/kg for OCPs and 3.10 - 7.60 μg/kg for synthetic pyrethroid pesticides. Soil residue levels were in the range 2.0 - 4121.70 μg/kg for OPPs, 1.10 - 12.90 μg/kg for OCPs and 1.10 - 8.20 μg/kg for synthetic pyrethroid pesticides. OPPs levels were appreciably high in all the samples followed by organochlorines and the synthetic pyrethroids. In general, organophosphate pesticides (OPPs) and the organochlorine pesticides (OCPs) levels were significantly higher than the WHO/FAO allowable levels. These show that watermelon fruits were contaminated to significant levels with organochlorine and organophosphate pesticides.

ACKNOWLEDGEMENTS

DEDICATION

To my mother for her love, Affection, Encouragement, Prayers and Passion

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<center>**CHAPTER ONE**</center>

<center>**INTRODUCTION**</center>

Background of watermelon

The world first recorded its watermelon harvest almost 5,000 years ago in Egypt. Hieroglyphics on walls in caves and tombs record the Egyptians first watermelon harvest. Historically, it is believed that watermelon has its origin from Egypt, and with the intentions of selling its seeds, some traders brought the watermelon from Egypt to Italy and Greece. Around the 10th century the melon was introduced to China and to the rest of Europe by the 13th century. It is believed that African slaves however, brought watermelon across the Atlantic Ocean into the United States.

Watermelon is botanically a fruit. It is similar to Cucumbers, Pumpkins, Squash and Gourds, all of which belong to the family Cucurbitaceae Citrullus and its specific scientific name is Citrullus lanatus (NationalWatermelonPromotionBoard, 2006a). The cultivation usually takes the right combination of water, weather and care. Watermelons are cultivated in rows of about twelve feet apart. Approximately 60 days after planting, the melon fruits nearest the root, called the "crown set" are produced. Within the following 30 days these fruits can be harvested and other melons further down the vine, which ripe after the crown set.

According to the (USNWPB, 2003d-b) watermelons are grown in more than 96 countries worldwide and are produced in 44 states in the United States, and presently, the U.S ranks fourth in production behind China, Turkey and Iran.

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As reported by the (USDepartmentofAgriculture, 2007), the country produced approximately 56.82 million kilograms of watermelons in 2004. Alabama was ranked fifteenth in national production in 2005 with 20.3 million kilograms melon produced, while Florida, Texas, Georgia and California top the list of production year after year.

Varieties of watermelon

Throughout the world, there are more than 1,200 varieties of watermelons with between 200 and 300 varieties cultivated in the United States (USNWPB, 2003d-a) .There are four basic groups of varieties of watermelons. These are: Picnic, Ice Box, Seedless, and Yellow-Flesh. The Picnic type melons are oblong, have dark green rind (with or without stripes), weigh 20- 25 kilograms on the average and have red flash (NWPB, 2003d-b). This group include varieties such as Sangria, Fiesta, and Regency. The Ice- Box group have varieties as Sugar baby, Petite Sweet, and Yellow Doll (NWPB, 2003d-a). Seedless watermelons weigh 10 - 25 kilograms averagely, they are oval to round in shape, having light green rind with dark green stripes, and can have either red or yellow flesh. The melons in the yellow – flesh variety have yellow to bright orange flesh, are oblong to long in shape, weighing 10 - 30 kilograms on the average, with light green rind and blotchy stripe (NWPB, 2003d-a).

According to , Desert King, Orangeglo, and Tender Sweet are all yellow – flesh type (NWPB, 2003d-a). The world record for the largest watermelon ever grown was set in 1999 with a watermelon that weigh 265 kilograms (NWPB, 2003d-a).

Nutritional value of watermelon

Watermelons have been found to contain no fat and have only 80 calories per serve. Watermelons also have no cholesterol or saturated fat per serve. Although watermelons are about 95 percent water, they contain many Vitamins such as A, B6, C, and other nutrients essential for good health. Vitamin A found in watermelons increase the number of lymphocytes (white blood cells) that help fight off infections and in turn improve the immune system, thus promoting good health. Vitamin B6 helps in the development of serotonin, dopamine and melatonin, all of which are neurotransmitters that help the body manage anxiety. Vitamin C helps prevent infections and viruses, and helps slow down the aging process and the development of cataracts. In addition, Vitamin C aids in strengthening blood vessels, bones and as well as help repair damaged tissues and healing of wounds (NWPB, 2003d-a). Small amount of potassium which can help alleviate muscle cramps, along with miniscule amount of calcium and iron are also found in watermelons (NWPB, 2003d-a).

Recent research has discovered yet another health benefit of watermelons: The carotenoid and Lycopene found in watermelons are powerful antioxidants that are thought to prevent diseases. This carotenoid gives fruits and vegetables their colour and is found in several other foods such as guava, tomatoes, and grapefruit. Lycopene which was once thought to be present only in tomatoes has recently been found to be present in watermelons in larger amounts than any other vegetable or fruit per serve. Watermelons have 9.09 mg of Lycopene, compared to the 4 mg found in one cup of tomatoes (NWPB, 2003d-b). Currently, pill or capsule forms of vitamins contain only 5-10 mg of lycopene which is the average

daily dose. Therefore, eating one serving of watermelon per day provide about the same health benefits as taking over – the – counter vitamins (NWPB, 2003d-b).

According to (Watson, 2000b), men who eat foods containing significant amount of lycopene were at lower risk for developing cancer, and in particular prostate cancer, and women who consumed high amounts of lycopene were five times less likely to develop precancerous indications of cervical cancer than those with low amounts of lycopene in their bodies. Lycopene is also thought to help battle cardiovascular disease by prohibiting the hardening of the arteries (Watson, 2000b).

Problems with watermelon production

Watermelons are susceptible to several kinds of insect infestations. Aphids, cabbage loopers, cucumber beetle, cutworms, thrips, leaf miners and spider mite are all known to infest watermelon crops. However, all of them can easily be controlled with pesticides or by biological means. Organisms such as lady beetles and lacewings, as well as food bran and molasses, can be used as alternative tools to manage pest (Kishi, Hirschhorn, Qjajadisastra, Sattlerlee, & Dilts, 1995).

Several diseases also threaten watermelon crops. Alternaria, leaf blight, anthracnose, bacterial rind necrosis, bacterial wilt, gummy stem blight, downy, mildew ,cercospora leaf spot, fusarium wilt, powdery mildew, pythium, southern blight and verticillium wilt are common diseases in watermelon crops (Kishi, et al., 1995). All diseases such as insect problems are controllable. Watermelons can also be plagued by a variety of viruses including watermelon mosaic virus -

2,Tobacco ring spot virus, papaya ring spot virus, squash mosaic virus, cucumber mosaic virus and zucchini yellow mosaic virus (Kishi, et al., 1995).

Weed control is also essential in successful watermelon production. Annual and perennial grasses along with broadleaf weeds commonly emerge throughout the watermelon growing season. Applying alanap or curbit to the soil surface after planting the watermelon crop according to helps control weed invasion (Kishi, et al., 1995). All these attempts to control diseases, pest, and weeds and to maximize crop yield have resulted in the use of toxic substances which affect man, animals, and the environment. As a result of these, the modern man is constantly exposed to a variety of toxic chemicals primarily due to changes in life style. The food we eat, the water we drink, the air we breathe, and the environments we live in are contaminated with toxic xenobiotics (Arthur & Cain, 1975). Humans are exposed to such chemicals while still in the womb of the mother. A number of studies have revealed the presence of pesticides in human milk, water, cow's milk and dairy products (Lederman, 1996) . The contamination of human milk by xenobiotics is a common problem worldwide; it is affected by geographical, climate, cultural and socio-economic variations in each individual location (Lederman, 1996).

In the last hundred years or so, human activities have been destroying the natural system upon which life depends. In fact after the advent of the Green Revolution, pesticide use was considered a sign of progress and modernization. The concepts of the Green revolution, excessive dependence on chemical fertilizers and synthetic insecticides, have proved to be a major cause of environmental concern. Rapid industrialization and ever increasing transport

emissions magnified the environmental problems several fold. Nevertheless, pesticide use has been a major concern, since the publication of *The Silent Spring* by the prophetic environmentalist, Carson in 1962. Substantial scientific evidence is now available since then, indicating the negative impact of inorganic pesticides viz neuro-toxicity, renal toxicity, respiratory toxicity, immuno-toxicity, reproductive toxicity, teratogenicity, genotoxicity and carcinogenicity (Jeraratnam, 1990). The Third World uses 80% of the world's pesticide and the World Health Organization estimates that all of the 220,000 annual pesticide related deaths occur in the third World (Wade, De Savelmers, Leingartner, & Foster, 1997) .

Pesticides

A pesticide has been defined as any chemical substance intended for preventing, destroying, repelling or mitigating any pest such as insects, bacteria, fungi, nematodes, weeds, rodents (Cox & Surgan, 2006). Globally, the use of synthetic pesticides has increased rapidly in the last fifty years due to intensification of farming in order to obtain higher yields. However, over dependence on chemicals not only resulted in a high cost of production but also irreparable damage to the environment and long term health problems to humans and other forms of life including marine organisms (Jeraratnam, 1990).

Currently, about 759 chemicals and biological pesticides are being used worldwide in the agriculture and health sectors. Of these, 33 pesticides have been classified by World Health Organization as extremely hazardous to human health (class Ia), 48 as highly hazardous (class Ib), 118 as moderately hazardous (class II) and 239 as slightly hazardous (class III) and 149 pesticides have been

considered as unlikely to cause acute hazard in normal use (class IV) (WHO, 1998). World pesticide consumption reached 2.6 million metric tons of active ingredients with a market value of USD 38 000 million and estimated 85% of this is used in the agriculture sector worldwide (WHO, 1998) . About three-quarters of pesticide use occurs in developed countries, mostly in North America, Western Europe and Japan. Although the volume of pesticide used in developing countries is small relative to the developed countries, it is nonetheless substantial and is growing rapidly. Furthermore, it is estimated that the pesticide market in developing countries is dominated by insecticides, which have higher acute toxicity than herbicides, the main pesticides used in the developed countries (WHO, 1998) . It has been reported that Pesticides may induce oxidative stress leading to the generation of free radicals and alteration in antioxidant or oxygen free radical scavenging enzymes such as superoxide dismutase, catalase, glutothione peroxidase, glutathione reductase and glutothione transferase (Ahmed , Vandana Seth Pasha , & Banerjee, 2000) .

The four main groups of pesticides viz the organochlorine, organophosphate, carbamates, and pyrethroid insecticides are of particular concern because of their toxicity and persistence in the environment (Ahmed , et al., 2000; Smith & Gangolli, 2002). However, several of the banned pesticides are still used on a large scale in developing countries and continue to pose severe health and environmental problems. It has been observed that farmers in developing regions seem to treat pesticides as substitutes for fertilizers (Rahman, 2003) and there is a need to create awareness among the farmers on Integrated Pest Management (IPM).

It has been observed (Smith & Gangolli, 2002) that technological development and cheap availability of raw materials have led to the production of a wide range of organochlorine insecticides, which unfortunately are non biodegradable, persistent and get accumulated in the environment and thus into the human food chain. Despite some regulatory measures, these compounds continue to be detected in measurable amounts in the ecosystem including marine life (Smith & Gangolli, 2002) . The pesticide DDT saved 25 million lives protecting them against the malarial mosquito and typhus fever. Simultaneously, the biological phenomenon of insect resistance to DDT was also developed in addition to its long standing persistence in the environment and its negative impact on marine life. Organochlorine pesticides DDT, DDD, DDE, aldrin, lindane and heptachlor residues, were detected in the muscle tissue of cat fish (Das, Khan, Das, & Shaheen, 2002). The presence of organochlorine pesticides in breast milk has been documented in many studies around the world (Hoyer , Jorgensen, Grandjean, & Hartvig, 2000)

Organophosphates are well known toxicants affecting the nervous system through the inhibition of acetyl cholinesterase (Karalliedde, Edwards, & Marrs, 2003). Most of the health problems due to acute poisoning of organophosphorus compounds with sensitive targets in the human body have been attributed to inhibition of enzyme acetyl cholinesterase in a range of nerve, neuromuscular and glandular tissues where this enzyme plays a key role in cell to cell communications (Karalliedde, et al., 2003).Organophosphate insecticides are metabolically activated to the corresponding oxon. The oxon selectively and strongly inhibits acetyl cholinesterase in cholinergic synapses resulting in

accumulation of acetylcholine and subsequently, cholinergic hyper excitation. The oxon is hydrolysed by A-esterases as a key detoxification step at high doses. The first line of defense is gut detoxification and p-glycoprotein exclusion of the oxon. The second line of defense is hepatic metabolism; the third line of defense is binding of oxon to B-esterases (butyl-acetyl-and carboxyl- esterase) when all these defenses are breached by high doses, then A-esterase becomes important. There is a marked interspecies difference in the enzymatic handling of paraoxon and humans may be less adept than rodents at detoxification (Kaliste & Korhonen, 1997) .

Over 300 chemicals including several known carcinogens have been identified in the adipose tissue and other organs including brain cells and the nervous system (Laessig, McCartty , & Silbergold, 1998). The brain and the endocrine (hormonal) glands are the target site for the fat-soluble toxins to accumulate (Laessig, et al., 1998). Continued exposure to these chemicals for a long period may result in symptoms of mild cognitive dysfunction (including problems in identifying words, colours or numbers, unable to speak fluently) and hormonal imbalances leading to infertility, breast pain, menstrual disturbances, adrenal gland exhaustions and early menopause. Eventually these toxins are stored in the fatty body tissues and in cells of the brain. These stored toxins may be slowly released and re circulated in the blood, contributing to many chronic illnesses. Whenever the body is under stress, the stored fat is released along with the toxins and circulates freely throughout the body. The resulting exposure can target various organs and body systems, contributing to many chronic illnesses (Sheehan, Sinks, & Tilson, 1996).

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Endocrine disruptors

Endocrine disruptors are exogenous chemical substances that cause adverse effects in the endocrine system. Pesticides can cause health problems thus acting as endocrine disruptors, resulting in reproductive defects, and immune dysfunction (Damstra, et al., 2000). These chemicals mimic the action of hormones and can damage or disrupt the normal functioning of an organism. In humans, the endocrine glands which include adrenal, thyroid, pancreas, ovary and testis produce hormones which are distributed to receptors through the blood stream. Many pesticides act as endocrine disruptors and affect sperm quality and reproductive development (Lintelmann, Katayama, Kurihara, Shore, & Wenzel, 2002). There is now considerable evidence that male reproductive function is declining in human and wildlife populations (Petrelli & Mantovani, 2002),that the mechanism of action may be disturbed testicular apoptosis and altered hepatic biotransformation of steroids. Animal studies have provided a range of effects that can be attributed to *in utero* endocrine disrupting chemicals (EDCs) exposure which include:

- A vast number of chemicals found to be EDC

- The ability of chemicals to bioaccumulate in the body lipid; and

- Metabolism of body lipid during pregnancy releasing the mother's life time EDC into circulation (Murray, Leam , Abramovich, Haites, & Fowler, 2001)

Sexual differentiation is regulated by reproductive hormones. Diethylstilbestrol is the best known endocrine disrupter and has caused abnormalities of sexual differentiation in both exposed female and male human fetuses (Toppari 2002).

According to (Smith & Gangolli, 2002) organochlorine contaminants in the human diets relate to the potential ability of many of these chemicals to act as endocrine disruptors even at low doses. Such chemicals are capable of disrupting the normal functioning of the endocrine system and may pose a growing threat to human and wildlife health. These compounds can modulate both the endocrine and immune systems resulting in alteration of homeostasis, reproduction, development and behavior. These chemicals in the environment cause endocrine disruption and result in pathological effects on the male and female reproductive systems, thyroid function, and the central nervous system (Amaral-Mendes, 2002)

Effect on reproductive system

There are reports of growing concern about environmental chemicals, both natural and man-made; having estrogenic property which may cause a variety of reproductive disorders in wildlife and human populations (Wade, De Savelmers , Leingartner, & Foster, 1997). Recent *in vitro* data suggest that the interaction between some weak estrogenic organochlorines, dieldrin, endosulfan, toxaphene, and chlordane cause a synergistic increase in their estrogenic potency, an effect due to joint action on estrogenic receptors (Wade, De Savelmers , et al., 1997).

It has been observed that exposure to environmental toxicants may play a role in adverse pregnancy outcomes (Fowler, 2001). It has been shown that there is detectable levels of ([4 chloro-2 methylphenoxy]) acetic acid in the semen of farmers who recently used the pesticides, and as these pesticides can be excreted in the semen, they could be toxic to sperm cells and be transported to the woman and developing embryo or fetus (Tye, et al., 1994) . Farm workers attending plant protection operations and persons working in the pesticide manufacturing units

are more prone to pesticide toxicity. In brief, exposure to pesticides with endocrine disrupting potential raises a particular concern for male fertility because of the possible occurrence of effects at low concentrations and additive interactions with other environmental risk factors. Epidemiological studies conducted by (Petrelli & Mantovani, 2002) have confirmed an increased risk of delayed conception associated with exposure to pesticides. Moreover, an increased risk of spontaneous abortion has been noted among wives of exposed workers (Petrelli & Mantovani, 2002) . Birth abnormalities have been reported in the offspring of registered users of pesticide as well as the general population living around agricultural areas (Garry 1996) . Studies have also shown a stronger association between foetal death due to elevated risk when the exposure occurred during the third – eighth week of pregnancy (Bell, Hertz-Piccotto, & Beaumont, 2001). In a study, umbilical cord blood was analyzed in a new-born, whose parents had been exposed to pesticides. The results indicated the presence of detectable dichlorodiphenyldichloroethylene (DDE), the main metabolite of DDT, and there was a positive correlation between maternal dichlorodiphenyldichloroethylene (DDE) and the consumption of fish (Sarcinelli, et al., 2003). It has been reported (Torres-Arreola, et al., 2003) that a cohort study of serum shortly after delivery indicated that DDE and other organochlorine pesticides may pose a risk to preterm birth in countries that continue to use such insecticides for malaria control .

Immune dysfunction

There is substantial experimental and epidemiological evidence that many pesticides in widespread use around the world are immunosuppressive. This

poses potentially serious health risks in populations highly exposed to infectious and parasitic diseases, subject to malnutrition and inadequately served by curative health programmes (Keifer , Rivas, Moon , & Checkoway 2001). Numerous animal studies show a variety of effects of pesticides on the immune system, including decreased antibody formation after exposure to pesticides such as captan, lindane and malathion and decreased cell mediated immunity has also been indicated**.** Frequent exposure to multiple toxins causes the detoxification system to be overloaded and inefficient. This leads to the accumulation of toxins and dead cells build up in the blood.

To combat these foreign bodies, the immune system produces excessive inflammatory chemicals. This may lead to symptoms of immune dysfunction such as allergies, inflammatory states, swollen glands, recurrent infections, chronic fatigue syndrome, and auto immune diseases. The immune system can be rejuvenated only by improving the liver function through proper dietary management (Johnson, 2002).

Parkinson's disease (PD) is the most common neuro degenerative disorder. It is now proposed that environmental factors in conjunction with genetic susceptibility may form the underlying molecular basis for idiopathic PD (Uversky , Li, Bower, & Fink 2002) . Epidemiological and experimental data suggest the potential involvement of specific agents as neuro toxicants such as pesticides (organochlorine and organophosphorus) in the pathogenesis of nigrostriatal degeneration, supporting a relationship between the environment and Parkinson's disease. According to Neuro degeneration results from multiple events and interactive mechanisms which include:

- The synergistic action of endogenous and exogenous toxins such as the ability of the pesticide diethyl dithiocarbamate to promote the toxicity of other compounds.

- The interaction of toxic agents with endogenous elements such as the protein alpha synuclein.

- Environmental factors that affect the background of genetic predisposition and aging (Di Monte, Lavasam, & Manning-Bog 2002)

Epidemiological studies provide evidence for an association between Parkinson's disease and past exposure to pesticides and other putative neurotoxins depending on variability in exposure to environmental agents including pesticides. Recent studies show clearly that genetic factors play a minor role in determining whether an individual develops this disease, rekindling an interest in the etiological significance of environmental factors (Lockwood 2000). *In vitro s*tudies have provided proof that several pesticides including rotenone stimulate the formation of alpha-synuclein fibrils. Moreover, a meta analysis of all case control studies so far showed a positive, statistically significant association between pesticide exposure and Parkinson's disease (Vanacore, et al., 2002).

Cancer

A number of studies have observed an association between brain cancers and pesticides as well as soft tissue sarcomas. Beginning in the late 1970s, there have been reports linking pesticides to leukemia in children. Case control studies have linked pesticide exposure to childhood cancer (Zahm , Ward, & Blair 1997). A number of studies have also demonstrated that maternal employment in agriculture has a link with leukemia. The most convincing evidence that

herbicides are human carcinogens comes from epidemiological studies(Hoar & Blair 1986). It is reported that the population living around the active agricultural regions are highly prone to cancer. Thyroid and bone cancers are prevalent in agricultural regions where fungicides are extensively used (Schreinemachers, Creason, & Garry, 1999). Recent studies have shown that the incidence of hormone related organ cancers or hormonal cancers have increased among farmers (Zahm , et al., 1997).

Exposure to endocrine disrupting pesticides, particularly DDT and phenoxy herbicides, is the suspected cause in some of these hormonal cancers (Buranatrevedh & Roy, 2001). The association between different types of pesticides and prostate cancer shows moderate risk among farmers exposed to organochlorine insecticides and acaricides specifically DDT and Dicofol (Settimi, Masina , Andrion, & Axelson 2003). Over the last 10 years, breast cancer in women has increased worldwide by 33 %. Various studies have linked our environment and the substances we are exposed to as prime suspects. There is growing evidence that the breast cancer epidemic is related to exposure to a wide range of environmental contaminants including DDT, other carcinogenic pesticides and oestrogenic stimulants. Organochlorine pesticides such as DDT and its metabolites DDD and DDE, dieldrin, heptachlor, HCH and its isomers were detected in the blood of breast cancer patients, irrespective of age, diet and geographic locations when compared to normal women (Mathur, Bhatnagar, Sharma, Acharya, & Sexana, 2002)

Cytotoxic defects

The potential genetic hazard of pesticides to human beings is of great concern. Results from the biological monitoring or cytogenetic methods for the detection of health risks to pesticides have showed DNA damage in peripheral lymphocytes among workers who are exposed to pesticides. The observed DNA damage was found to be significantly lower in workers taking some of the necessary safety precautions during their work (Undeger & Basaran, 2002). Malaoxon is the first and main metabolite that is more toxic than the parental compound, Malathion. Malaoxon can damage DNA in human lymphocyte, by various mechanisms including oxidative damage. Hydrogen peroxide and reactive oxygen species may be involved in the formation of DNA lesions induced by Malaoxon. Malaoxon can also methylate DNA bases.

Increased chromatid breaks and chromosomal aberrations in human lymphocytes were observed in workers occupationally exposed to pesticides. There has been an observed micronuclei frequency in peripheral blood lymphocytes among the farm workers, and was more evident among workers who avoided protective measures (Bolognesi, Perrone, & Landini 2002). Genetic damage, particularly gene loss, is a central cause of aging. Aging involves altered cellular and humoral immunological functions which include

- Decreased number of lymphoid precursor T- and B- cells;

- Reduced proliferative capacity of T-cells, loss of lymphocyte sub groups as a consequence of shortening of telomers;

- Qualitative deficiency of B-lymphocytes with reduced response to exogenous antigens;

- Compromised activity of the accessory cells, both by directly depressing the chemotactic and phagocytic responses and indirectly by increasing the prostaglandin production which inhibit the proliferation of T-cells; and finally,

- Alteration in the production and secretion of various cytokines (Malaguarnera , et al., 2001)

Factors influencing toxicity

A central tenet of the science of toxicology is that the toxic effect of any material monotonically increases with the amount of toxic material delivered to the target tissue. The factors to be considered include physicochemical properties, solvents, impurities of the pesticide, duration and route of exposure and also factors related to the individual exposed including variation in the metabolic, sequestration and excretory processes and health status, age, gender, and environmental factors (Karalliedde, et al., 2003). The effect of pesticides on human health depends on quantity of pesticide accumulated, the length and frequency of exposure and potential toxicity of the pesticide. Effects also depend on health of a person at the time of exposure. It is suggested that specific chronic pesticide effects may develop in elderly people because of the long latency period between exposure and disease. Organophosphate pesticides exert toxic effect on bone marrow and may be associated with hematopoetic cancer after a latency of 10–25 years (Hardell & Eriksson, 1999).

Infants and children are at great risk from the effects of pesticides. Children are exposed to potentially carcinogenic pesticides from use in school, home, other buildings, lawns and gardens, through food and contaminated

drinking water, from agricultural application drift, overspray or off-gassing and from carry home exposure of parents occupationally exposed to pesticides. Parental exposure during the child's gestation or even pre-conception may also be important. Malignancies linked to pesticides include leukemia, neuroblastoma, Wilm's tumor, soft-tissue sarcoma, Ewig's sarcoma, non-Hodgkin's lymphoma and cancer of brain and testes. Several studies suggest that children may be particularly sensitive to the carcinogenic effect of pesticides. There is a potential to prevent at least some childhood cancer by reducing or eliminating pesticide exposure (Zahm & Ward, 1998). Pesticides are effective in killing the pests through their neurotoxic effects. Infants appear to be particularly susceptible to the effect of these pesticides because they have an incompletely developed acetyl cholinesterase system and their immature liver cannot detoxify the compounds. Infants and children are especially sensitive to health risks posed by pesticides for several reasons. Their internal organs are still developing and maturing. Children eat and drink more than adults, in relation to their body weight, possibly increasing their exposure to pesticides in food and water (Hardell & Eriksson, 1999).

Experimental evidence suggests that specific chronic pesticide effects may develop in adults because of the long latency period between exposure and disease (Hardell & Eriksson, 1999). The use of animal data on toxicity is not sufficient for predicting human risk. The evidences suggest that human infants and children are much more susceptible, particularly to organo-phosphates and carbamates than animal species. The present assessment of the risk of pesticides is almost exclusively based on animal studies and this may greatly underestimate

the risk to humans, particularly infants and children. Risk assessment must be based on human physiology and metabolism. The proportionate organ development period and the impact of pesticides at different developmental stages vary widely between human children and test animals.

Human detoxification system

Inherently the human body is endowed with an efficient detoxifying system, which can handle minimum toxic exposure. The liver plays a pivotal role in breaking down the harmful substances that can be excreted by various means. The enzymes of the liver first deactivate the toxic substance and then convert the toxin into water soluble forms which are eliminated through urine, bile in the feces, sweat, lung vapour and sebum. At least two enzyme steps are involved in the processing of toxicants in the liver of humans; the first involves cytochrome p450 enzymes and the second involves glutathione S transferases (GST). Glyphosate, a systemic herbicide inhibits enzymes involved in the detoxification of chemicals in the body. Antioxidant enzymes (super oxide dismutase, catalase and glutathione peroxidase) are components of an organism's mechanisms for combating oxidative stress which is generated in normal metabolism and which may also be a reaction in response to external stimuli (Johnson, 2002). However, this function of the liver can be damaged by repeated exposure to chemicals and toxins in food, water and air. Another major threat is the excessive private and public use of volatile organic compounds (VOCs), which due to their high lipophilic nature, get stored in the brain and cell membranes. The detoxification process of VOCs largely depends on the activity of cytochrome oxidase p450 which converts the VOCs into more water soluble forms to be excreted through

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urine. However, if the conjugation process is not efficient, the hydrophilic form will prove to be more toxic than the parent chemical (Johnson, 2002).

Statement of the problem

Pesticides are not a problem of just the developing world. Highly industrialized countries still use large quantities of pesticides and these still cause numerous health and environmental injuries. Virtually, all countries need additional reforms to minimize and eliminate the harms caused by pesticide exposure. Exposure to pesticides is particularly a serious problem in much of the developing world for which Africa and for that matter, Ghana is no exception. Many active ingredients in pesticide are known or suspected to cause cancer. Individual pesticides have been linked, either by laboratory evidence or epidemiological studies, to a long list of cancers, including multiple myeloma, soft tissue sarcoma, Ewing's sarcoma, lymphoma, non-Hodgkin's lymphoma, leukemia, melanoma, neuroblastoma or Wilm's tumour, germ-cell tumours, retinoblastoma (eye tumour); and cancer of the oesophagus, stomach, prostate, testis, breast, ovary, cervix, bladder, thyroid, lung, brain, kidney, pancreas, liver, colon and rectum (UNEP, UNICEF, & WHO, 2002).

Pesticide exposure has been associated with impaired development of the nervous system which can result in lowered intelligence and behavioural abnormalities (UNEP, et al., 2002). There is evidence linking various pesticides to effect on the central nervous system, the peripheral nervous system and the pre-birth developing brain. These include: Inferior developmental skills and increased aggression in children, depressive effects that may lead to suicides, delayed neuropathy, involving degeneration of the peripheral nerves in the limbs

with muscular aches and pains and influenza-like symptoms, Personality change, impaired concentration and memory, language disorder, heightened sense of smell, deterioration of handwriting, impaired tolerance of exercise and neuromuscular deficits, Parkinson's disease and parkinsonism, a disorder with symptoms like Parkinson's disease, but which may be reversible (UNEP, et al., 2002) among other effects. In view of the above mentioned effects, it is clear that the population of Ghana where there is no proper monitoring of the kind(s) of pesticides applied for various purposes are at greatest risk from these chemicals. In recognition of the threat posed by these chemicals, it is evident that Ghana is faced with a very serious problem. There is, therefore, the need to investigate whether these pesticides are present in the foods particularly watermelon to ascertain if they are likely to pose a threat to the health of people who consume them.

Objectives of the research

The main objectives of this study are to:

- Know the types of pesticides which are being used among watermelon farmers in the country with particular attention to the Komenda-Edina – Eguafo –Abrem (K.E.E.A) district in the central region of Ghana.

- Determine the levels of pesticides in watermelons grown in Ghana.

- Determine the levels of the pesticides in the soils on which the fruits are grown.

- Determine some soil characteristics and how they affect pesticides accumulation in the fruits.

- Determine the effects of the applied pesticides on watermelons on non-target crops.

- Establish the levels of these pesticides in the watermelon fruits from these other selected regions.

- Ascertain the extent of contamination of watermelons by comparing the levels to those permitted by WHO/FAO and other countries and organisations.

Justification of study

It has been reported that humans, domestic and wildlife species have suffered adverse health consequences resulting from exposure to pesticides in the environment because of the ability of the pesticides to interact with the endocrine system (Fouler, 2001). These pesticides have the potential to interfere with the normal functioning of the endocrine system and therefore interfere with production, release, transport, metabolism, binding action or elimination of the natural hormones in the body responsible for the maintenance of homeostasis and regulation of developmental processes (Fouler, 2001).

In humans, the consequences of prenatal exposure to pesticides on the reproduction tract of both females and males are known, and developmental neurological problems have been identified in children exposed to pesticides such as DDT and its metabolite and / or endosulfan (Durham & Armstrong, 1965). Over the last four decades there have been reports of decline in the quality and quantity of sperm production in humans, and increases in certain cancers (such as prostate, testicular, breast etc) that have led to speculations about environmental etiologies (Fowler, 2001). Despite these health problems reported, little is known

about the distribution and the levels of pesticides that would induce these effects in human populations. It has been established that the normal functions of all organ systems are regulated by endocrine factors and small disturbance in the endocrine function, especially during certain stages of the life cycles such as development, pregnancy and lactation, can lead to profound and lasting effects (Garry, et al., 2002). Based upon the recognition of the scope of the potential problems, the possibility of serious effects on the health of human population and the persistence of some pesticides in the environment, this study would among other things help to:

- Establish data on pesticides that are used among watermelon famers in Ghana.

- Advise farmers on pesticides recommended for watermelon crops, the right period for application to reduce residue levels in the watermelon fruit to levels protective human health and the environment.

- Furnish the Environmental Protection Agency with the necessary data for the establishment and implementation of sustainable residue monitoring.

- Make significant contribution to the joint WHO/FAO pesticide residue programme, which recommend individual countries to provide information on all relevant pesticide data to help the joint WHO/FAO.

- With the goal of protecting human health, the study would finally serve as a blueprint for creating awareness of the existence of pesticides in our environment and foods. The consumer can then make informed decisions to mitigate any negative health impact which may result from exposure to pesticide

CHAPTER TWO

LITRATURE REVIEW

Pesticides in plant system

A number of chemicals of diverse characteristics have been classed together on the basis of their use and given the descriptive pesticides. An unfortunate aura of mystery has developed about these chemicals. However, there is nothing unique or mysterious about the chemicals referred to as pesticides. The chemical and physical properties of a pesticides and environmental factors determine the behaviour of pesticides. The behaviour in turn dictates the fate of the pesticide (Führ, 1991). To predict behaviour, it is necessary to measure the chemical and physical properties of the pesticides and the environment.

The behaviour of a pesticide is its characteristic movement, persistence, and fate in the environment and it determines its effectiveness as well as its residue levels. The absorption of pesticides by foliage for instance shows how pesticide behaviour can be understood by examining the interaction of physical and chemical properties with the environment.

All aerial portions of the plant are covered by cuticle which is continuous through stomata into the stomatal chambers, and chemicals entering the aerial portions of the plant pass through the cuticle. The cuticle is composed of plates and protuberances of wax imbedded in various layers of cutin which is a mixture of polymers of dicarboxylic and hydroxycarboxylicacid esters. The properties of the cuticle vary with environmental conditions and position on the plant (Kerle, Jenkins, & Vogue, 2007). In general, the polarity of the cuticle increases towards

the interior of the plant. The external cuticle contains much wax and is highly oxidized and polymerized. In the central portion of the cuticle, nearly continuous layers of wax plates may be imbedded in cutin. The interior cuticle has less wax and more pectinaceous material, and the epidermal cell walls are impregnated with a mixture of cutins and pectins. The cuticle inside the stomatal chamber is more polar than the cuticle on the leaf surface, and the under leaf surface cuticle is more polar than the upper surface cuticle. Plants raised under mesic conditions show a thinner and more polar cuticle than similar plants raised under xeric conditions. The cutin has some affinity for water and under conditions which favour hydration may swell and force plates of wax apart (Kerle, et al., 2007).

Pesticide entry into a plant requires contact with the surface and compatibility with the cuticle (Führ, 1991). Waxy projections and hairs may prevent good contact between leaf surfaces and spray solutions with high surface tension. Surfactants and emulsifiers may improve the leaf contact of aqueous spray solutions, and oil soluble pesticides are frequently applied in diesel oil, a carrier with good leaf contact properties. The compatibility of pesticide and leaf cuticle depends on the interaction of their respective chemical and physical properties. The polarity of the cuticle and the pesticide are of primary importance. The polarity of the cuticle increases from the waxy leaf surface toward pectins in cell walls and to the aqueous environment of the cell. The outer portion of the cuticle favours the entry of relatively nonpolar pesticides like 1, 1, l-trichloro- 2, 2-bis (p-chlorophenyl) ethane (DDT) or the long-chain alkyl esters of 2, 4-dichlorophenoxyacetic acid (2, 4-D). The inner portion of the cuticle favours

passage of more polar compounds like 2, 4-D acid but restricts the passage of lipophyllic compounds like DDT. Thus, DDT residues in plants are usually surface residues which can be removed with solvents which remove the outer cuticle. The systemic action of 2, 4-D requires a balance between absorption and translocation (Kerle, et al., 2007) . Extensive absorption with no translocation gives ineffective vegetation control. The penetration of surface cuticle by 2, 4-D increases with the size of the hydrocarbon portion of alkyl esters of 2, 4-D, but at the same time pesticide effectiveness reaches a maximum and then declines. The affinity of long-chain hydrocarbon esters for the lipid portions of the cuticle retards their entry into the aqueous portions of the plant and their subsequent transport to the site of action. However, long-chain glycol esters of 2, 4-D have good absorption characteristics and are compatible with the aqueous environment of the plant. These latter esters have a proper balance of hydrophilic and lipophillic properties which help make them effective systemic pesticides

The initial point of pesticide - plant contact depends on the method of application. Many pesticides are applied as aerial sprays, and the foliage and stems are primary intercepting organs. Some chemicals are injected into or directed on the stems of larger plants and roots are the principal absorbing organs for soil- applied pesticides.

Pesticides intercepted by aerial portions of plants may undergo several processes which include:

Uptake of pesticides into the plant is required for systemic chemical action, and the degree of absorption dictates the effectiveness of treatments with

systemic chemicals. The amount of absorption which is desirable, therefore, depends on the nature of the pesticide, its target, and its residue characteristics (Kerle, et al., 2007).

Surface adsorption of pesticides by plants

The physical or chemical binding of the chemical to the surface of the plant, is a form of storage, and the extent of adsorption depends on the physical and chemical properties of both the chemical and the leaf surface. Surface adsorption may inactivate pesticides since it prevents absorption of systemic chemicals and reduces pesticide contact action. It is important to realize, however, that surface adsorption is not final, but it is an equilibrium reaction. Environmental factors define the equilibrium between adsorbed and free chemical, and a change in environmental conditions will alter the point of equilibrium. Any reduction in the amount of free chemical leads to a release of adsorbed chemical until equilibrium is re estab1ished.

Volatilization of pesticides from plants

Vapourization of intercepted pesticides back into the atmosphere is not important for pesticides with a low vapour pressure or a high heat of vapourization (Kerle, et al., 2007). On the other hand, losses may be appreciable for compounds like ethyl N, N-dipropylthiolcarbate or the isopropyl ester of 2, 4-D. Volatilization moves the pesticides from the site of action, thereby reducing treatment effectiveness (Kerle, et al., 2007). Although volatilization reduces chemical residues on the plant, it adds to the total load of atmospheric pollutants.

Pesticides wash off from plants

Removal of surface residues by precipitation, the amount of pesticides not absorbed, adsorbed, degraded, or lost through volatilization may be subjected to wash off. Wash off may carry pesticides in solution or suspension depending on their water solubilities. This may reduce pesticide effectiveness and may as well lead to impact on nontarget species or water contamination (Dolgilevich, 2009). Pesticides washed to the soil may be leached to the root zone and absorbed by the plant.

Pesticides degradation in plants

Pesticide residues on the plant surface are important in determining both efficacy of treatment and impact on environment. Degradation of surface residues may reduce effectiveness by removing the pesticides from the site of action (Dolgilevich, 2009). On the other hand, degradation is the only mechanism which can reduce the total load of environmental pollutants, Absorption, adsorption, volatilization, and wash off only store or transport pesticides to other parts of the environment (Tiryaki & Aysal, 1999).

Pesticides in the root zone are subjected to the same processes as those intercepted by aerial portions of the plant. However, the degree to which a particular process operates may be quite different. Water-soluble pesticides are readily absorbed by the roots and may be transported to other parts of the plant (Tiryaki & Aysal, 1999) . Pesticide volatilization is relatively unimportant from root surfaces but may occur from the soil surface. Wash off does not occur, but leaching of chemicals from the root zone is an analogous process (Tiryaki &

Aysal, 1999). Photochemical degradation also does not occur on roots, but chemical and biological degradation in the root zone is important.

Behaviour of pesticides in plant is not important if absorption is limited. However If large amounts are absorbed internal behaviour determines both effectiveness of treatment and internal residue (Kerle, et al., 2007) . Pesticides inside plants may also undergo several processes such as:

Translocation to different parts of plant

Movement of pesticides in plants, may occur toward the top of the plant in both the xylem (water transport tissue) and phloem (photosynthetic transport tissue), but translocation toward the roots occurs only in the phloem and lateral transport is limited (Waite, Cessna, Grover, Kerr, & Snihura, 2002). Translocation is important because the fate of pesticides may vary in different plant parts. Those absorbed by foliage but not translocated to other plant parts may be lost in leaf fall, while pesticides transported to the roots may be exuded into the soil (Clay, DeSutter, & Clay, 2001) .

Pesticides storage in plants

Chemical or physical binding of pesticides to plant constituents, may occur in any part of the plant. Largest amounts are frequently found close to the point of absorption, in storage cells adjacent to the paths of translocation, and in areas of intense metabolic activity (Führ, 1991). Pesticide storage may be active or passive. The Active storage is pesticide accumulation against a concentration gradient and requires expenditure of metabolic energy. Pesticides may be passively adsorbed to structural components of plants. Both active and passive

storage are reversible, and pesticides may be released and translocated to other parts of the plant as conditions in the plant change (Larney, Cessna, & Bullock, 1999).

Pesticides metabolism in plants

This alters pesticide structure and may result in detoxication or activation and may occur anywhere in the plant. Metabolism of pesticides is nearly always a detoxication process for the plant, but the products may be biologically active in other systems and, therefore, still important as residues (Glotfelty, Wight, Leech, Jersey, & Taylor, 1989) .The phenoxybutyric pesticides are an exception (Glotfelty, et al., 1989). They are inactive as herbicides, but their active acetic acid derivatives are produced through betaoxidation of the butyric side chain. Insecticides are also metabolized in plants. Although they and their metabolites are generally not active in plants, they are frequently quite toxic to other organisms (Arias, et al., 2008).

Pesticides exudation from plants

Volatile pesticides and their metabolites may leave the plant as vapours through the stomata (pores in the leaves). Some pesticides like 2, 4-D, 2-methoxy-3, 6-dichlorobenzoic acid (dicamba) or 4-amino-3, 5, 6- trichloropicolinic acid (picloran) are exited from the roots (Briggs, Bromilow , & Evans, 1982). In contrast with animals, fish, and birds, however, exudation of pesticides from plants is not extensive (Briggs, et al., 1982).

Regulations in using pesticide and personal hygiene

The various items of protective clothing that may have to be used are described below with descriptions of their proper care.

Hats: These should be made of impervious material with a broad brim to protect the face and neck. Unless made from cheap material; they should be able to withstand regular cleaning.

Veil: A plastic mesh net will have an adequate protection of the face from the larger spray droplets and permit adequate visibility.

Capes: Short capes of light plastic may be suspended from the hat to protect the shoulders. Overalls: All of above should be made of light, durable cotton fabric and they must be washed regularly. The frequency depends on the pesticides being used. Washing with   soap, detergent, or soda is adequate in the case of organophosphorus and carbamate compounds and a rinse in light kerosene may be needed for compounds such as organochlorines and this should be followed by washing with soap, detergent or soda.   Rubber boots should be worn to protect the feet and legs.

Gloves: Polyvinyl chloride or rubber gloves or gauntlets should be used when handling concentrates with an organic solvent base. Cotton gloves offer some protection for hands when regularly washed. Impervious gloves must be cleaned regularly inside and out, but are unsuitable for continuous wear.

Face masks: Masks of gauze or similar material are capable of filtering the particles from water dispersible powder spray and may be worn to reduce inhalation of the spray and dermal exposure of the face, if such protection is

considered desirable. They must be washed regularly and, in some instances fresh masks may need to be used for the second half of the day's spraying, so that the face is not contaminated. Scrupulous attention to personal hygiene among spray operators is essential.

For professional spray men operating in the tropics, safety precautions may depend largely on personal hygiene, including washing and changing of clothes. A drill for carrying out and supervising personal hygiene, and the regular washing of protective clothes and cleaning of equipment should be organized along the following lines: (a) Spray men should be provided with at least two uniforms to allow for a change when required. (b) Washing facilities with sufficient water and soap should be made available in the field at appropriate locations. (c) All working clothes must be washed regularly with the frequency depending on the toxicity of the formulation. (d) Particular attention should be given to washing gloves as wearing of contaminated gloves may be more dangerous than not wearing gloves at all. (e) Spray operators must clean hand and take a shower themselves before eating. (f) Smoking during work should be forbidden. (g) When work involves insecticides of relatively high toxicity, the hours of work must be arranged so that exposure to the material used is not excessive; transport should be arranged so that a long delay between the end of the day's operations and the return to the base for washing. Personal protective equipment and pesticide safety training are among the standard required to reduce worker exposure and prevent transfer of workplace hazards to the home.

Furthermore , workers should be advised to wash work clothes separately from other clothes, and not to wear work clothes at home (Sumpter & Johnson, 2005).

Epidemiological studies of pesticides exposure

The first recorded use of chemicals to control pests comes from classical Greece where Homer mentioned the value of burning sulphur as a fumigant (Smith & Secoy, 1975). In Roman times, Pliny the Elder recommended the use of arsenic as an insecticide, as well as a mixture of soda and olive oil to treat legume seeds. In the sixteenth century, the Chinese appear to have independently discovered the value of arsenicals and soon afterwards tobacco extracts were used in Europe(Smith & Secoy, 1975).

The mid nineteenth century saw a systematic scientific approach applied to the development of possible pesticides. Pyrethrum, a natural insecticide derived from chrysanthemum flowers, and soap were widely used, as was a combined wash of tobacco, sulphur and lime (Tye, et al., 1994). Work on arsenicals saw the introduction of Paris green in 1867. This impure copper arsenite became so widely used to control the spread of Colorado beetle, which, by 1900, legislation had been enacted in the US to regulate its application. The early twentieth century saw the development of a number of other chemicals. Many, such as creosote, anthracene and naphtha, were based on tar oils. But it was the period during and following the Second World War that first saw the intensive development of synthetic insecticides. While 1, 1, 1-trichloro-2, 2'bis (*p-* chlorophenyl) ethane (DDT) was first synthesised in 1874, its action as an insecticide was not discovered until 1939. DDT was released commercially in August 1945, having

been used during the war for protection of military areas and personnel. The availability of such an effective and cheap insecticide heralded an agricultural revolution reflected in the phenomenal growth in pesticide use.

In 1944 total world DDT production was limited to 4366 tonnes and by 1963 production within the US alone had peaked at 81,154 tonnes (WHO, 1979). By the late 1960's, DDT was credited with the eradication of malaria from the United States and Italy (Attaran & Maharaj, 2000). However, following environmental concerns, by 1970 use in the US had dropped to 11,316 tonnes, although use in Australia remained high on a per capita basis at 1000 tonnes compared with Canada 287 tonnes and West Germany152 tonnes. The 1940's also saw the discovery of most of the major families of pesticides still in use at the end of the century- organophosphates, carbamates and other organochlorines such as chlordane. Until the 1960's however, DDT remained by far the most widely used pesticide throughout the world. During the 1970's and 1980's, many new pesticides were introduced, including the synthetic pyrethroids developed from naturally occurring pyrethrins. Increased knowledge about host -pest interactions also allowed for new formulations and methods of application, and a large number of new pesticides were developed within each family (Attaran & Maharaj, 2000).

By 1985 the explosion in pesticide use had created a total world pesticide market estimated to be worth over $US 4 billion. Most pesticide use is targeted at agricultural production, especially of pesticide dependant crops such as cotton (accounting for almost one quarter of world pesticide production), rice, maize,

soya beans, wheat and tobacco. Approximately 10 % of world pesticide use is aimed at public health programmes, largely relating to mosquito control in developing countries (WHO, 1979).

Unfortunately, further experience with DDT demonstrates that the widespread use of pesticides can have negative as well as positive effects (WHO, 1979). Part of the success of DDT, the first widely used synthetic pesticide, can be attributed to its persistence in the environment, thus reducing the need for frequent application. This chemical stability and an associated lipophilicity resulted in DDT and other organochlorines being readily stored in, and only slowly eliminated from, most living creatures. A large number of organisms, particularly marine filter feeders, can also act as bioconcentrators creating levels of DDT in their own flesh above ambient environmental concentrations. Organochlorine pesticides also have the capacity to accumulate through the food chain, particularly in predatory animals at the top of the ecological pyramid. In the late 1960's, dead Sea Eagles in the Baltic and North Sea areas were noted with up to 36,000 ppm of DDT in pectoral muscle (Jensen, 1969). Human biological sampling also showed increasing DDT levels in almost all human communities, mainly due to exposure to residues in food. By the mid 1950's DDT could be detected in most foods and daily exposure in the US had been estimated at 0.184 mg per man per day (Walker & Goette, 1954). Following restrictions on the application of DDT to livestock and forage crops, as well as on crops directly eaten by man, there was a gradual decrease in residues in animal and vegetable foods. By 1964, DDT intake in the US was estimated at 0.038 mg per person per

day (Durham & Armstrong, 1965). National surveys by the US Food and Drug Administration showed levels falling to 0.015 mg/man per day by 1970 (WHO, 1979). While contaminated food posed the greatest route of human exposure to DDT, the general community was also exposed through other media. In 1975, three years after DDT was withdrawn from use in the United States, it could still be widely detected in air in the Mississippi Delta (Arthur & Cain, 1975). Nor was agricultural use the only source of environmental contamination. Effluent from the Montrose chemical plant in California, near Los Angeles was discharged into the main city sewer, which, in turn, emptied into Santa Monica Bay.

From 1953 to 1971 an estimate of 270 kg per day of DDT was released into the marine environment, with resulting residues detectable in ocean creatures over an area of more than 10,000 km^2 (Garry, et al., 2002). Indeed, almost no part of the globe has escaped contamination with these persistent chemicals. While these substances have never been used in the Antarctic, ice cores taken from the Antarctic shelf show detectable levels of DDT and other organochlorines (WHO, 1979). The first studies of potential adverse effects of organochlorine pesticides were made in the mid-1950. In 1956, DDT added to the feed of pheasants was found to cause a decline in the number of eggs laid and the viability of chicks (Garry, et al., 2002).

In the mid-1960 the first suggestions were made that this might be having an impact in the broader environment. In comparative study of samples of eggs conserved in different British museums, it was found that the ratio of eggshell weight to the axial length of the egg in peregrine falcons decreased suddenly

between 1945 and 1946, years, which saw the introduction of DDT on a large scale (Ratcliffe, 1970). At the same time adverse effects were becoming obvious in wild animal populations. Examples include the virtual disappearance of the Coho and Kiyi salmon from Lake Michigan (the eggs and small fry of fish are particularly sensitive to organohalogen compounds, with a concentration of 5 ppm DDT in water resulting in 48.3 % mortality of carp embryos in the egg) (Garry, et al., 2002). Contamination of their prey with pesticide was also the main cause of a dramatic decline in numbers of fish eating birds including the peregrine falcon and osprey. The main impact of contamination appeared to be on reproductive success. For example, the Connecticut colony of osprey had fallen from 150 breeding pairs in 1952 to 5 pairs in 1970 with an average of 0.23 fertile young per nest, far below the number required to maintain the population. Perhaps the most spectacular case that can be directly ascribed to organochlorine pollution is that of colonies of the brown pelican (Blus & Cromartie, 1979). Colonies in islands off southern California dropped from 3000 breeding pairs in 1960 to only 300 in 1969. Among remaining pairs at that time, 1200 eggs were laid, but only 5 viable chicks were born.

The relationship between organochlorines and eggshell thinning and impaired fertility in birds was confirmed experimentally in ducks, and was thought to be due to changes in calcium metabolism (Lehner & Egbert, 1969 ; Peakall, 1969). A causal relationship is also supported by recent research in the US that indicates an increase in eggshell thickness of nearly 50 % in some bird species since the withdrawal of DDT (Burger & Viscido, 1995). However, the

devastating environmental impact of DDT remained mainly of academic interest until 1962, when public concern was aroused by the publication of "Silent Spring" which sold nearly a million copies in less than two years (Pranab & Shaheen, 2002). People were generally unaware of the toxicity of pesticides "until the publication of *Silent Spring* (Wicklund & Daling, 1988). The controversy arising from the ensuing public debate ultimately led to a raft of legislative initiatives. These included the withdrawal of DDT from use in the US, and the establishment of the United States Environmental Protection Agency in 1969. These concerns also led to a large number of studies investigating whether exposure to these substances might adversely affect human health (Wicklund & Daling, 1988).

It is widely reported that most pesticide related deaths involve acute poisonings rather than chronic exposure (WHO, 1990). As pesticide use has become more widespread, mathematical models have estimated increasing numbers of pesticide poisonings throughout the world, rising from 5×10^5 cases/yr in 1972 to 25×10^6 cases/yr in a 1990 estimate, though only a small percentage of deaths are identified and reported. 148 poisoning outbreaks were reported between 1951 and 1990, involving 24,731 cases and 1065 deaths, with food being the most common route of exposure. Two countries, the United States and Thailand, accounted for more than half of the reports, probably reflecting the quality of their notification systems. In the US, a descriptive analysis of national mortality data, National Hospital Discharge Survey data, and American Association of Poison Control Centers national data from 1985 to 1990 found 341

fatalities from agricultural and horticultural chemicals over the 6-year period, of which 64 % were suicides, 28 % were unintentional, and 8% were of undetermined intent (Klein-Schwartz & Smith 1997) . 25,418 hospitalizations were reported, 78 % identified as unintentional. Both deaths and hospitalizations occurred more frequently in males with rates higher in nonwhites than in whites. 338,170 poison exposures were reported to poison centers for fungicides, herbicides, insecticides, and rodenticides, with life-threatening manifestations or long-term sequelae occurring in 782 cases, and 97 deaths reported (Klein-Schwartz & Smith 1997).

Background to studies on chronic health effects

Epidemiologic research into the health effects of chronic pesticide exposure is plagued by many problems (Blondell, 1990). Foremost among these is the difficulty in getting accurate information on exposure. Exposures to pesticides in the general community are low and heterogeneous. To explore possible adverse impacts of pesticides, researchers have therefore frequently turned to occupational studies, where exposures are likely to be higher and more predictable. However, even in an occupational setting, pesticide exposure tends to be difficult to assess. Only workers involved in the production of pesticides operate in typical industrial settings where workers are indoors, the environment is relatively stable and environmental monitoring may allow an estimation of exposure. On the other hand, pesticide users rarely have standardized work practices (Alavanja , et al., 2004). Users are often either self-employed or members of family businesses, and generally work in environments that vary

markedly from minute to minute depending on the task at hand. Users may apply a range of pesticides for different purposes and may mix before application. Where biological monitoring has been undertaken in pesticide users, it has often been limited to monitoring of biological effect such as serum cholinesterase levels, rather than measuring internal exposure dose (Guillette, Meza, Aquilar, D, & Garcia, 1998). While biological monitoring for persistent pesticides such as the organochlorines can give a meaningful picture of total exposure over a number of years, metabolism and excretion of more modern pesticides is rapid and results of biological monitoring may only reflect exposure in the last few hours or days. In the absence of meaningful information on individual exposures, researchers have often turned to simple occupational categorisation. To be most effective, such an approach requires homogeneity in the exposures likely to be experienced by subjects identified in each category. Where, for example, categories such as "farm hand" are identified from census or other routinely collected document, heterogeneity of exposure lessens the ability of these studies to detect true associations. More accurate indications of occupational exposure can be developed when subjects are drawn from a single employer or setting, however, even these may be misleading. Some of the issues around exposure assessment were examined by a review of studies of the possible effects of dioxin contaminated "agent orange" used by military personnel during the Vietnam War (Wolfe & Michalek, 1995). A number of large studies have been unable to lead to agreement on a possible association between dioxin exposure and a range of reproductive outcomes including spontaneous abortion and birth defects.

Until 1992, exposure assessment was limited to military service in Vietnam as a surrogate for dioxin exposure. Sometimes this was supplemented by subjects' own estimate of exposure, or indices developed by researchers estimating the likelihood of exposure. However, in 1992, the air force released results of the first study to examine the relationship between individual serum dioxin contaminant and verified reproductive outcomes. Interpretation of the results of the study was complex, but personnel involved in the handling of dioxin were, indeed, shown to have significantly higher levels than controls (median12.8 pg/g and 4.2 pg/g respectively). However, these levels correlated poorly with both self reported exposure and exposure indices developed from military records. These findings confirm the potential bias in the results generated by research dependant on these surrogate measures, and the need for a degree of skepticism when interpreting their findings. Another problem for epidemiological studies exploring the health impact of pesticides relates to selection of controls.

A widespread problem in occupational health is the "healthy worker effect". This is characterised by the tendency for relatively healthy individuals to be more likely to gain employment and remain employed (McMichael & Andjelkovich, 1976). This may potentially bias the studies towards finding lower mortality rates in an occupational cohort when compared with the general community and thus mask true increases in mortality. The impact of the healthy worker effect can be seen in many of the occupational studies discussed below. The healthy worker effect may be exacerbated by the unique dietary and lifestyle factors associated with residence on a farm. For example, farmers are less likely

to smoke, more likely to eat local produce, more likely to be exposed to petrochemical products, exhaust fumes, mineral and organic dusts, and thus face biological exposure from animals and microbes. In general, residence on a farm is thus also associated with mortality and cancer rates below those in the broader community (Ritter & Wigle, 1990). A more general epidemiological problem faced by these studies is determining the outcome.

Most large studies have focused on defined endpoints such as mortality. In general, these have relied on death certificates for outcome data, causing a loss of study precision since death certificates are, in practice, poorly completed. This is exacerbated by difficulties matching subjects with deaths and cancer registries, particularly in developing countries where data collection may be less comprehensive (McMichael & Andjelkovich, 1976).

Finally, a range of other factors may confirm the relationship between pesticide exposure and cancer mortality. As observed by (Ritter & Wigle, 1990) these possibly include smoking, carcinogenic animal viruses (leukaemia), and the lymphoproliferative effect of prolonged antigenic stimulus (multiple myeloma and other haematological malignancies).

Descriptive and ecological studies

A number of studies have been undertaken using geographic indicators as surrogates for exposure. While useful for suggesting possible adverse outcomes and for generating hypotheses, these "ecological" studies suffer from a number of common weaknesses relating to exposure misclassification. A typical ecological study examined the relationships between mortality data for cancers of the brain,

lymphatic tissue and leukaemia, and the spatial distribution of agricultural

pesticide used for 34 drainage basins in Quebec, from 1976-1985 (Godon &

LaJoie, 1991). The basins were grouped into three categories (low, intermediate,

and high exposure) according to the level of sales of pesticides. For cancers of the

lymphatic tissues among women 35 to 64 years of age, a high relative risk (RR)

was observed (RR = 1.91, 95 % CI 1.14-3.18) in basins highly exposed to

pesticides compared to those with low exposure. Analysis of correlation for this

cancer at the 34 basins showed significant associations between geographical

distributions of the Standardized Mortality Ratio and those of numerous variables

indicative of pesticide use in agriculture. Another Canadian report highlights

some of the difficulties inherent in ecological analysis. This project investigated

the mortality of approximately 70,000 male Saskatchewan farm operators, a

subset of a larger group of 365,000 Canadian farm operators investigated

separately (Ritter & Wigle, 1990). Analysis indicated that during the study

period, the overall mortality among Saskatchewan farmers was 25 % lower than

that for all Saskatchewan men. During the same time interval, the risk of death

from all types of cancer was also about 25 % lower among Saskatchewan farmers

than for all Saskatchewan men. As discussed previously, these results restate

common findings in studies looking at the health of farmers. However, the study

did identify a relationship between non-Hodgkin's lymphoma mortality and acres

sprayed for weeds and concluded that the magnitude of risk for Saskatchewan

farmers was probably greater than that reflected in the estimates in the study, due

to the likelihood of misclassification of exposure. Non-Hodgkins Lymphoma has

been associated with herbicide use in a number of other studies, lending weight to these findings (Ritter & Wigle, 1990).

Mortality rates were investigated in three rural municipalities of population 96,000 in the Philippines before (1961-71) and after (1972-84) the widespread use of organophosphate and organochlorine pesticides (Loevinsohn, 1987). Deaths from pesticide poisonings increased significantly. For men aged 15-54 years, the mean age-standardised mortality rate for all cancers except brain increased from 21.1 to 25.9, although this was not significant. Mortality from leukaemia in men increased from 0.6 to 3.6 per 100,000 ($P < 0.05$). Leukaemia rates for women for the same periods were 0.6 and 0.7 per 100,000. Unfortunately, exposure classification in this study was crude, and the comparison between different time periods introduces the potential for confounding by other factors such as impoverishment, increasing use of tobacco and changing reporting habits. Computerized mortality listings for Wisconsin for 1968-1976 were used to compare death and cancer rates in residents with an occupation identified on death certificates of "farm owner", "tenant" or "labourer", with white, non-farming Wisconsin men (Blair & White, 1981; Saftlas & Blair, 1987).

Data from agricultural and population censuses were used to construct indicators of exposure. Among all Wisconsin farmers, significantly decreased proportional mortality ratios were seen for tobacco- and alcohol-related causes of death, while excesses occurred for accidental causes, asthma, and cancer of the stomach, prostate, eye, and lymphatic and haematopoietic systems. Elevated proportional cancer mortality ratios (PCMR's) for leukemia, all lymphopoietic

cancers and cancers of the stomach, rectum and eye occurred in farmers born 1905-1958, while deficits were observed for cancer of the pancreas and the category "all other cancers." Increases in PCMR's with level of various agricultural activities were largely associated with cancers of other lymphatic tissue, 2/3 of which were multiple myeloma and the rectum. No positive PCMR gradients were observed for leukemia and malignancies of the stomach and eye. In a separate, but related, study, 774 subjects identified from digitised Wisconsin mortality listings were compared with controls dying from other causes. Younger farmers in counties with high agricultural activity determined from agricultural census had an elevated risk for reticulum cell sarcoma (Cantor, 1982). One of the weaknesses of this and similar studies is reliance on information from death certificates. Even if the cause of death is accurately identified and the recorded occupation at the time of death is correct, no information is available on previous employment. In these studies, farm owners, tenants, foremen and labourers were all classified as farmers, although many of these may not have been actively engaged in farm work. Exposure to pesticides was estimated by the agricultural characteristics of the county and may not reflect individual exposure. Such problems can result in a bias toward the null. Another study assessed the contribution of vineyard pesticides to brain cancer mortality among agricultural workers (Challier & Viel, 1998). A pesticide exposure index (PEI) in vineyards was calculated for 89 French geographical departments. The authors identified male farmers and farm labourers aged 35-74 from national death data for the years 1984-1986. Brain cancer mortality among these subjects using census data

to identify total numbers of farmers and estimate rates was compared with that for the general population. Mortality from brain cancer among farmers was significantly higher than mortality for the overall population with standardized mortality ratio = 1.25, P < .001 and univariate and multivariate analysis accounting for economic status as a confounder revealed a significant link with pesticide exposure in vineyards (RR 1.11; 95 % CI 1.03-1.19). The accuracy of these findings is dependent on the "usual occupation' stated by relatives of the deceased. Since no cancer registry exists in France, the finding was limited to mortality which may also be influenced by other factors such as health service utilisation or access. The exposure index was estimated for 1970 and based largely on the proportion of usable agricultural land devoted to vineyards. Several case control studies have used ecological methods to examine possible associations between pesticides and specific cancers (Cocco & Benichou, 1998). One such study examined the relationship of prostate and testicular cancer mortality with environmental exposure to the DDT metabolite p, p'-dichlorodiphenyldichloroethylene (DDE), a known anti-androgen, during the period 1971-1994. Environmental DDE contamination by state was estimated by measuring DDE concentrations in the subcutaneous fat of population samples and by measurements of DDE in tree bark. However, sampling numbers were small, and representativeness of fat and tree samples was not determined. Neither prostate cancer mortality nor testicular cancer mortality showed a positive association with either indicator. The relative risk of prostate cancer in farmers was also estimated in a meta-analysis from articles published in peer-reviewed

journals between January 1983 and June 1994 (Keller-Byrne & Khuder, 1997).

Positive associations between prostate cancer and farming were found by analysis both for all studies and analysis limited to retrospective studies. No association was found with analysis that included only studies reporting a standardised mortality ratio. These findings may have been influenced by selection bias since negative studies may have been less likely to be reported in peer-reviewed publications. The analysis also did not distinguish between well-designed studies and those compromised by methodological flaws. However, many of the studies included were looking at multiple cancer sites and there was no temporal trend in risk estimates, which might be expected if publication bias were influencing the findings. One plausible explanation for the positive association between prostate cancer and farming is exposure to hormonally active agricultural chemicals. Another possible effect of hormonally active chemicals could be reproductive disturbances. A Norwegian study compared the perinatal health of 192,417 children born to parents identified by agricultural census as farm holders, with 61,351 births to non-farmers in agricultural municipalities (Kristensen & Irgens, 1997). Subjects were matched with the Medical Birth Registry of Norway, which comprises all births with more than 16 weeks of gestation. One of the strengths of this study is the completeness of these national records. Perinatal mortality between the two groups was similar, but the proportion of late-term abortions (gestational weeks 16-27) was higher among farmers' births (odds ratio (OR) = 1.9, 95 % confidence interval (CI) 1.6-2.3). The increase in late-term abortion among the farmers could to some extent be attributed to an excess of mid-

pregnancy deliveries among grain farmers. The authors concluded there was no convincing evidence that perinatal death was associated with use of pesticides. However, the results did support the hypothesis that occupational exposure to mycotoxins in grain induces labour at an early stage of pregnancy. A case-control study of multiple myeloma among males was conducted with the use of digitized mortality listings for 1968-76 from the State of Wisconsin (Cantor and Blair 1984). Age, year of death, race, county of usual residence, marital status, and usual occupation were available for the 411 male deaths due to multiple myeloma and for a matched series of deaths due to other causes. Controls dying from tobacco related causes were excluded to minimize bias created by differences in smoking patterns between farmers and other occupations. Farmers were at an elevated risk compared to non-farmers (odds ratio (OR) = 1.4), with decedents 65 years of age or older having a stronger association (OR = 1.5) than younger farmers (OR = 1.1). County levels of selected agricultural characteristics were used as surrogate measures of exposure. Significantly elevated odds ratios were observed for farmers residing in counties high in chicken inventory (OR = 1.6), fertilizer use (OR = 1.7), and acres treated with pesticides (OR = 1.9). As with similar studies, indirect exposure measures and reliance on death certificates for case identification potentially bias this study. On the other hand, excluding controls dying from tobacco related disease minimises problems arising from lower smoking rates in farmers. Other ecological studies have also suggested a link between predicted pesticide exposure and death rates from multiple myeloma and leukaemia. Census and deaths data was used in a study of the French farm

labourer population aged 35-74 in 89 departments for the period 1984-1986 (Viel & Richardson, 1993). SMRs were calculated using national rates. Rates were high for multiple myeloma (SMR 1.59; 95 % CI, 1.32-1.89) and leukaemia (SMR 1.33; 95 % CI 1.19-1.49), but not lymphoma. Leukaemia was also significantly associated with a calculated geographical pesticide exposure index. An early ecological study examined the risk of leukemia among farmers using records of death certificates from Nebraska, 1957-1974 (Blair & Thomas, 1979). Comparison of occupation, as recorded on the death certificate, for 1084 leukemia deaths and 2168 deaths from other causes, matched for age at death, year of death, county of residence, race, and sex, revealed an elevated risk of leukemia among farmers (OR, 1.64; 9 5% CI, 1.18-2.27). The risk was increased among farmers born after, but not before, 1900 suggesting a relationship with agricultural exposures of recent origin. Stratification by county of residence showed a significantly elevated risk for farmers from heavy corn producing counties.

In summary, results from ecological studies are inconsistent and subject to a number of potential biases. Associations identified in these studies that may be worth further investigation include leukaemia, prostate cancer, brain cancer, lymphopoietic cancers, multiple myeloma, and perinatal mortality.

CHAPTER THREE

METHODOLOGY

Description of study area

The study area, Nsadwir, is located at longitude $1^o \, 20^{'} \, 18^{''}$ W and latitude $5^o \, 03' \, 58''$ N within the Komenda – Edina – Eguafo – Abrem district of the central region of Ghana (Appendix A36). The area has dark brown soils rich in organic matter, have weak structure, and poor water retention. The soils also have high proportions of sand, low clay contents, and are highly sensitive to compaction. Internationally the soils are classified as histosols (FAO, 1988) and locally as savanna gleisols (Quiroga, Buschiazzo, & Peinemann, 1996). Comparison of various fractions of the soils to the textural triangle put the soil encountered into the sandy- loam textural class.

Several environmental factors play an influential role in the growth of watermelon crop, and include sun light, temperature, relative humidity, carbon dioxide and oxygen, and soil (Rivero, Sanchez, Ruiz, & Romero, 2003). For instance, adequate sun light is an important factor which influences the growth of watermelon crop. Blue light is essential for the growth of the leaves whereas a combination of red and blue light promotes flowering of crop (Drozdova & Bondar, 2001) . Temperature is also one of the pre-requisites for processes such as photosynthesis, respiration, germination, and flowering. The optimum temperature for germination of the seed ranges between 55-65 degrees Fahrenheit

whereas the optimum temperature for optimum photosynthesis and respiration ranges from 65 – 75 degrees Fahrenheit (Rivero, et al., 2003). Relative humidity plays important role in the growth of crop. It is defined as the ratio of water vapour in the air to the amount of water in the air, and is crucial for the transpiration of the plants. Manufacturing of sugar by watermelon crop requires the presence of carbon dioxide and hence it is one of the vital environmental factors affecting the growth of the watermelon crop and it is estimated that watermelon crops use approximately 1500 parts per million of carbon dioxide for sugar manufacturing (Farrar & Williams, 1991). Oxygen is also essential for crop respiration and utilization of the byproducts of photosynthesis (Farrar & Williams, 1991). Soil with proper humidity, right balance of minerals and nutrients, and the right pH balance is also essential environmental factor which influence the growth of the watermelon crop (Steiner, W.G, Lehmann, Nehls, & De Macedo, 2007).

Sampling and sample preparation

Watermelon fruit samples were collected from seven different locations. These locations were Nsadwir, in the Komenda-Edina-Eguafo-Abrem (KEEA) district in the Central Region of Ghana which is the study site, where watermelon is intensively cultivated. At Nsadwir, four different farms (labelled A, B, C and D) at entirely four different locations were visited. On these farms, watermelon fruit samples as well as soil samples were collected. To establish the effect of these pesticides on non-target species, Okro samples were collected from one of

the farms at Nsadwir and labelled B. To know the pesticides which are in circulation and those which are used most among farmers growing this crop in other regions of Ghana, other watermelon samples were randomly collected from the following areas for comparative studies: Ayensudo, Antado, and Enyenasi, all in the KEEA district of the Central Region of Ghana, Takoradi (Western), Kumasi (Ashanti), Cape Coast (Central), Bolgatanga (Upper East) and Accra (Greater Accra) regions of Ghana.

After the effective root length of the mature watermelon plant has been determined to be ranging from 13.80 to 14.67 cm per plant, soil samples were randomly collected at 0-20 cm depth from the surface by digging with a cutlass. The samples were stored in glass bottles, covered with lids and were transported to the laboratory for analysis. A total of 84 samples were used which included 16 soil samples, 5 Okro samples and 63 watermelons (rind and fruit).

Questionnaire development and administration

Prior to watermelon fruits sampling, a questionnaire was designed and administered to one hundred watermelon farmers at Nsadwir in the Komenda – Edina – Eguafo -Abrem district of the Central Region of Ghana, as away to seek information on the pesticides which are applied on watermelon crops in the locality, and included questions such as the type of pesticides apply on the watermelon plant, the period and frequency of application (Appendix A1).

Equipment

The Gas chromatographic (GC) instrument employed for the pesticide residue analysis was a Varian CP – 3800 series equipped with the [63] Ni selective Electron capture detector (ECD) for the analysis of organochlorines (OCs) and synthetic pyrethroids pesticides and a Pulsed Flame-Photometric Detector (PFPD) for organophosphorus (OPs) pesticides and capable of temperature programming. The GC column employed was a GS-Q (30 m x 0.53 mm i.d) supplied by J&W Scientific Inc, Folsom, California, USA. Also employed were high speed Binatone domestic blender (Model – BGL- 401), Stuart Scientific Flask Shaker (Model SF1), a Sartorius weighing balance (Model LE632P) with a weighing capacity of 620 g. Other equipment employed include: A rotary vacuum evaporator, BUCHI type (Model R-114), a Buchner Funnel fitted with a BUCHI B -169 vacuum system, a water bath (Model B- 840), a speed safe magnetic stirrer (Model HI 180F-2), a pH meter with glass – calomel combination electrode, PHYWE type and a 10mL safety pipette, a 50 mL burette, and 1L separating funnel (all of Silber Brand).

Reagents

Chemicals/reagents used were of analytical quality and were supplied by BDH chemical limited in the United Kingdom. The chemicals/reagents used include: anhydrous sodium sulphate, Dichlomethane, Acetone, Hexane, Cyclohexane and Florisil. Other chemicals/reagents used are N- Phenylanthranilic acid, ferrous sulphate, Potassium Dichromate, Magnesium Sulphate, Barium

Chloride, Sodium hydroxide Sodium hexametaphosphate, Sulphuric acid and Orthophosphoric acid.

Potassium dichromate solution (0.1667 M)

The Potassium Dichromate was dried at 105° C overnight in an oven before use. The potassium dichromate solution was prepared by weighing accurately 49.04 g of the dried analytical reagent crystals into a 100 mL beaker. A small volume of distilled water was added to the potassium dichromate in the 100 mL and was stirred with a clean stirring rod to dissolve it. The dissolved potassium dichromate was then transferred into a 1 L volumetric flask. The process was repeated until all the potassium dichromate in the 100 mL beaker had been quantitatively transferred into the 1 L volumetric flask. The volume of the solution was then made up to the 1 L mark with distilled water.

Ferrous sulphate solution (1 M)

In preparing the ferrous sulphate solution, 278.0 g of dried analytical reagent ferrous sulphate was carefully weighed into a 1 L beaker. About 750 mL of distilled water and a 15 mL of 96 % concentrated sulphuric acid were added. The mixture was then stirred continuously with a clean stirring rod to dissolve it. The mixture was then transferred quantitatively into a 1 L volumetric flask and made to the mark with distilled water.

Sodium hydroxide solution (0.1 M)

The 0.1 M sodium hydroxide solution was prepared by weighing 4.0 g of the analytical reagent sodium hydroxide pellets into a 250 mL beaker. 100 mL distilled water was added to the sodium hydroxide pellets in the beaker and was then stirred continuously with a clean stirring rod to dissolve it. The solution was then transferred into a 1L volumetric flask and made to the mark with distilled water.

N- Phenylanthranilic acid indicator solution

To prepare the N- Phenylanthranilic acid indicator solution, 0.1 g of the N- Phenylanthranilic acid powder was weighed and dissolved in 5 mL of 0.1 M sodium hydroxide solution and the mixture was diluted to 100 mL with distilled water.

Dispersing agent (Calgon type)

The chemicals used for preparing the dispersing agent were both dried at 105° C in an electric oven for 24 hours prior to their use. The dispersing agent which is a mixture of 4% sodium hexametaphosphate, and 1 % soda was prepared by dissolving a mixture of 40.0 g sodium hexametaphosphate and 10.0 g Soda in a 250 mL Erlenmeyer flask filled with 150 mL distilled water. The mixture was stirred continuously with a clean − dry stirring rod until all of the salts were dissolved. The mixture was then transferred quantitatively into a 1 L volumetric flask and made to the mark with distilled water.

Barium chloride extracting solution (0.1 M)

About 24.428 g of barium chloride was weighed accurately into a 250 mL beaker. A small volume of distilled water was added to the barium chloride in the 250 mL beaker and was stirred continuously with a clean stirring rod. The solution was then transferred into a 1 L volumetric flask containing 500 mL of distilled water. The flask was swirled until all the barium chloride had dissolved. The solution was diluted with more distilled water until it was at the 1 L mark.

Barium chloride equilibrating solution (2 mM)

About 20 mL portion of the 0.1 M barium chloride solution was measured into a 1 L volumetric flask. The solution was then diluted to 1 L by adding more distilled water.

Magnesium sulphate solution (0.1 M)

About 24.648 g of magnesium sulphate was weighed accurately into a 1 L volumetric flask. Five hundred millilitres of distilled water was then added to the content of the 1 L volumetric flask. The flask was then manually shaken until all the magnesium sulphate had dissolved. The content was then diluted to the 1L mark.

Magnesium sulphate solutions (1.5 mM and 5 mM)

About 15 mL and 50 mL of the 0.1 magnesium sulphate solution prepared as described above were measured separately into two different 1 L volumetric flasks. The content of each flask was then diluted with 500 mL of distilled water.

The contents of the two 1 L volumetric flasks were vigorously shaken by hand and were diluted to 1 L with distilled water.

Sulphuric acid solution (0.05 M)

About 2.8 mL of concentrated sulphuric acid was measured and transferred into a 1 L volumetric flask half-filled with distilled water. The mixture was vigorously shaken to ensure thorough mixing and then diluted with more distilled water and made to the mark.

Extraction of pesticides and determination

The watermelon fruit samples were divided into flesh and rind (i.e. 2 cm from rind). Each was homogenised separately using a high speed Binatone domestic blender (Model BLG-401) prior to sample extraction. The extraction method for obtaining multi-residue pesticides in non-fatty food crops was employed using acetone as the extracting solvent(Luke, Frodberg, Doose, & Mosumoto, 1981).

Ten gram portion of the homogenised watermelon fruit sample was weighed into a 500 mL flat bottom flask. One hundred millilitres of acetone was added. The mixture was then fitted onto a Stuart Scientific Flask Shaker (Model SF1) and shaken for 72 hours. The mixture was filtered using a Buchner funnel fitted with a BUHCI B- 169 vacuum system. The filtrate was quantitatively transferred into an acetone washed 1 L separating funnel for partitioning.

Figure 1: A laboratory picture showing pesticide extraction using a flask shaker

In partitioning the filtrate, a 40 mL portion of a 1:1 mixture of n- hexane and Dichloromethane was added to the filtrate. The funnel was corked and shaken vigorously for 3 minutes. Occasionally, the tap of the separating funnel was opened to expel pressure. The funnel was fitted onto a retort stand and its content allowed to separate for 30 minutes(Pang & Chao, 1995). The partitioning was repeated five times using 40 mL each of the 1:1 n- hexane- Dichloromethane mixture. The organic layer was drained into an acetone washed 500 mL flat bottom flask. Thirty gram portion of anhydrous sodium sulphate dried at 105° C for 24 hours (cooled in a dessicator) was added to the partitioned extract and allowed to stand for 48 hours to remove traces of water. The partitioned and dried

extract was filtered again using a Buchner funnel fitted with a BUCHI B -169 vacuum system. The filtrate was transferred quantitatively into another acetone washed 500 mL BUCHI round bottom flask, fitted onto a vacuum rotary evaporator and evaporated to dryness. The procedure was repeated for all other watermelon fruit samples. Each extract concentrate was dissolved in 5 mL 1:1 mixture of n- hexane and Dichloromethane for a clean- up. The procedure discussed above was followed for the rind of the watermelon samples, and the five Okro samples.

Figure 2: A laboratory picture showing separation of extract after partition

Procedure for a clean up

Prior to the Gas chromatographic (GC) analysis, the sample clean – up was done to remove co- extractives or extraneous materials from the extracts (Luke, et al., 1981). Ten millimetres chromatographic column was filled with 3.0 g of Florisil material activated at 130° C for 24 hours. This was topped up with 3.0 g of anhydrous sodium sulphate. Twenty millilitres portion of 1:1 solvent mixture of Cyclohexane and Dichloromethane mixture was added onto the column. The tap of the column was then opened to allow the solvent mixture through to wet and rinse the column.

The pesticide residue in 5 mL of 1:1 solvent mixture of n-hexane and Dichloromethane was quantitatively transferred onto the column and the extract vial was rinsed thrice with the 1:1 n- hexane - Dichloromethane mixture and added onto the column. The column was then eluted with a 100 mL portion of 1:1 Cyclohexane and Dichloromethane mixture at a rate of about 1 mL/min into a round bottom flask. The eluent was then evaporated to dryness using the vacuum rotary evaporator. The residue was then dissolved in 1mL solvent mixture of 1:1 acetone and Cyclohexane for gas chromatograph (GC) analysis.

Preparation of soil samples for pesticide residue extraction

The extraction was done with a flask shaker. Ten gram of air dried soil sample sieved through a 2 mm size mesh was weighed into a 500 mL flat bottom flask. One hundred millilitres of acetone - hexane mixture (3:2) was added. The

mixture was put into the flask shaker and shaken for 72 hours. After extraction, the soil samples were filtered using a Buchner funnel fitted with BUCHI B169 vacuum system. The extracts were quantitatively transferred into an acetone washed round bottom BUCHI-type flask and were evaporated to dryness. The residues were re dissolved in 5 mL of acetone and underwent clean – up as described above (Luke, et al., 1981) for Gas chromatographic (GC) analysis.

Spiked samples preparation

Spiked samples for both fruit and soil samples were prepared by adding standard pesticide spiking solution to 10.0 g of fruits and soil extracts respectively. The samples were spiked with a stock solution of the pesticides containing 1 μgmL^{-1} of the pesticides in ethyl acetate. The spiked samples were prepared at the time of extracting the pesticide residues and were subjected to the same extraction and clean – up procedures as described for each sample type. Samples were spiked with 0.1 mL of the standard as a way to establish the efficiency of the extraction procedure. The percentage recovery was then calculated by dividing the spike sample result by the expected result as follows:

Expected spike result = sample result + standard conc.

$$\text{Percent recovery} = \frac{\text{spike result}}{(\text{Expected result x spike volume})} \; x \; 100$$

Prior to pesticide residue extraction from the soil samples, certain parameters of the soils which determine the extent to which the soil can retain the pesticides were determined as a way of soil characterization. The parameters

determined included the soil moisture, soil pH, the organic matter, cation exchange capacity (CEC) and the soil texture.

Soil moisture content analysis (USDA, 1972,revised 1982)

Five grams of air dried fine soil samples were weighed into tarred moisture cans. The samples were then dried overnight at 105° C with the lid of the moisture can opened. After the drying period, the moisture cans were removed from the oven, covered with the lids, and cooled in a dessicator for 24 hours. After cooling, the moisture cans and their contents were weighed. The moisture content of the soil samples in percentage by weight (% Wt) was calculated using the empirical relationship below:

Moisture (% wt) = (A –B / B- tarred can) x 100,

Where A = weight of air dry soil and the moisture can

B = weight of oven dry soil and the moisture can

Soil particle size determination

The pipette method (Kilmer & Alexander, 1949) for determining soil particle size was employed for the determination of clay, sand and silt. To determine the weight of the dispersing agent in a 5 mL aliquot of soil sample suspension, a blank dispersing agent was prepared. Five millilitres of the blank was pipetted into a previously weighed porcelain evaporating dish. It was then evaporated to dryness in an oven at 105° C for 72 hours, cooled in a dessicator, and then reweighed. The weight of the salt was then calculated from the

difference in weight of the porcelain dish containing the dry salt residue and the weight of the empty evaporating dish.

The suspension of soil samples was prepared by weighing 10.0 g of an air-dried soil into a 250 mL beaker. Ten millilitres of 5 % the dispersing agent was added followed by a 100 mL distilled water. The mixture was then stirred for 20 minutes. The suspension was quantitatively transferred into a 500 mL measuring cylinder. Three hundred millilitres of distilled water was then added to bring the total water volume to 400 mL. The suspension was again stirred thoroughly for 10 minutes with a clean- dry stirring rod and placed in a water bath at 30° C. The suspension was then allowed to stand for 10 hours. The clay particles suspended in solution while sand and silt settled at the bottom of the measuring cylinder with the silt on top of the sand. Five millilitres aliquot of the suspension was collected at a depth of 5 cm from the top into a previously weighed porcelain evaporating dish using a pipette. The pipetted suspension was then evaporated to dryness in an electric oven at 105^{0} C for 72 hours. It was cooled in a dessicator and then weighed. This was used to determine the percentage clay of the soil sample. The remaining 395 mL of the suspension was quantitatively transferred onto a 63 µm size mesh sieve. The sand on the 63 µm size mesh sieve was then washed repeatedly with distilled water to remove any traces of silt and salt. It was then transferred quantitatively into a previously weighed porcelain evaporating dish and dried in the oven at 105-110 ^{o}C for 72 hours, cooled in a dessicator and sieved again to ensure that all silt particles were removed (Black, 1965). The sand was weighed.

The clay, sand and silt were expressed as percentages as illustrated below:

% Clay = Vsp (Wc-W) x100/VpWo

% Sand = Ws x 100/Wo

% Silt = 100 - (% Clay +**%** Sand)

Where Wo = Weight of soil used (10.0 g)

Ws = Weight of sand in 10.0 g of soil sample

W = Weight of salt in 5mL aliquot of soil suspension (0.001 g)

Wc = Weight of Clay residue in 5 mL aliquot of soil suspension

Vp = Pipette volume of soil suspension

Vsp = Total volume of soil suspension

Soil pH determination (ISO10390, 1994)

The pH of the soil samples was potentiometrically determined in a supernatant suspension of a 1:2.5 soil: water mixture. Twenty grams of soil samples was weighed into a 100 mL wide-mouth flat bottom flask and 50 mL of distilled water was added to each flask and the flasks were capped. The flasks and their contents were fitted into a flask shaker and were vigorously shaken for 2 hours. Prior to opening the flasks for measurement the flasks were manually shaken by hand thrice. The electrodes of the pH meter were then immersed in the upper part of the suspension. The pH readings were taken when reading had

stabilised. The readings were considered stable when they did not changed by more than 0.1 units per 30 seconds. The pH meter had been calibrated using buffer solutions of pH values 4.00 and 9.00.

Organic carbon determination (Walkley & Black, 1934)

In determining the organic carbon content of the soil samples, the (Walkley & Black, 1934) procedure which involves wet combustion of the organic matter with a mixture of potassium dichromate and sulphuric acid at 125° C was employed, and the residual dichromate was titrated against ferrous sulphate. To compensate for any incomplete destruction of the organic carbon, an empirical correction factor of 1.33 was employed in estimating the result to adjust the organic carbon recovery.

Five grams of the soil sample was ground to pass through a 0.25 mm sieve. One gram of this material was weighed into a 500 mL wide-mouth Erlenmeyer flask and 10 mL of the dichromate solution was added. Twenty millilitres of 96 % sulphuric acid was carefully added .The content of the flask was stirred continuously for 30 minutes and was allowed to cool for 30 minutes. Two hundred and fifty millilitres of distilled water was added to the mixture to quench the reaction. Prior to halting the reaction, interferences from the ferric ion that may be present in the sample was eliminated by adding 1 0mL of 80 % Orthophosphoric Acid. The chemistry of this extraction procedure is illustrated below:

$$2Cr_2O_7^{2-} + 3C^o + 16H^+ \rightarrow 4Cr^{3+} + 3CO_2 + 8H_2O$$
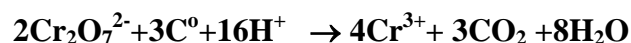
The excess $Cr_2O_7^{2-}$ in the digest was then titrated against ferrous sulphate solution with 1mL of N- Phenylanthranilic acid indicator solution, and the chemistry involve is : $K_2Cr_2O_7 + 6FeSO_4 + 7H_2SO_4 \rightarrow 3Fe_2(SO_4)_3 + K_2SO_4 +Cr_2(SO4)_3 +7H_2O$ At the endpoint, the dark violet − green colour disappeared and a new colour (light green) was observed. The volume of the ferrous sulphate solution which was required to completely react with all the excess $Cr_2O_7^{2-}$ was then noted. The procedure was repeated for all the other soil samples including a blank. The organic carbon content of the soil samples were obtained by the empirical relation: Percentage carbon (% C) = M x (V1-V2/S) x 0.39 x mcf

Where,

M = Molarity of ferrous sulphate solution from the blank titration.

V1= mL of ferrous sulphate solution required for blank.

V2= = mL of ferrous sulphate solution required for soil sample.

S= Weight of air- dry soil sample in gram.

mcf = Moisture correction factor = (100 + % moisture / 100).

1.3 = a compensation factor for any incomplete combustion of the organic matter in the procedure.

$0.39 = 3 \times 10^{-3} \times 100 \% \times 1.3$ (3 is the equivalent weight of carbon).Conversion of the percentage carbon (% C) to percentage organic matter was done by multiplying the percentage carbon by the empirical factor of 2.

Cation exchange capacity (CEC) determination

In the determination of the cation exchange capacity of the soil sample, the Barium Chloride Compulsive Exchange Method which involves the exchange of ions at the cationic sites on the soil surface with Barium ions and subsequent exchange of the barium ions with magnesium ions (Gillman & Sumpter, 1986) was employed . Two grams of the air dried soil sample was weighed into a funnel containing a medium grade filter paper and fitted onto a 250 mL measuring cylinder. To exchange the ions at the cationic site with barium ions, the soil sample was leached slowly with a 20 mL portion of 0.1 M barium chloride solution. The twenty millilitres portion of the 0.1 M barium chloride used was allowed to soak into the soil on each addition before more was added. To ensure a complete exchange of the cations with the barium ions, the soil sample was further leached with 60 mL portion of 2 mM barium chloride solution in six 10 mL aliquots. Again the solution was allowed to soak into the soil on each addition. The last 10 mL of the leachate was separated and its pH was determined separately.

After leaching, the soil sample was carefully transferred into a pre-weighed 250 mL flat bottom flask. Ten millilitres of 5 mM magnesium sulphate solution was added. The mixture was then fitted into a flask shaker and shaken for 1 hour after which the pH of the soil solution was determined using a pH meter. The conductivity of both the soil sample solutions and the 1.5 mM magnesium sulphate were determined using a conductivity meter. As a way to ensure total saturation of all cationic sites by the magnesium ions, the conductivity of the soil

sample solution was adjusted to that of the 1.5 mM magnesium sulphate by a careful addition of 1 mL increments of 1.5 mM magnesium sulphate solution .The volume of the 1.5 mM magnesium sulphate required to bring the conductivity of the soil sample to that of the 1.5 mM magnesium sulphate was noted .The equivalent conductance was determined by observation using a conductivity meter. Total saturation of all cationic sites was achieved when the conductivity of the soil solution was finally adjusted to 1.5 times the conductivity of the 1.5 mM magnesium sulphate solution by adding 0.1 mL increments of 0.1 M magnesium sulphate solution .The volume of the 0.1 M magnesium sulphate solution used was also noted.

The pH of the final soil solution was determined and compared with the pH of the last 10 mL leachate and the pH of the soil sample solution after the 1hour shaking. About 0.05 M sulphuric acid solution was added drop- wise until the pH of the final soil solution was within 0.1 unit of the previous measurements. The flat bottom flask was wiped clean outside and was weighed to determine the weight of the final solutions. The procedure was followed for all the soil samples and the cation exchange capacity of each soil sample was calculated from the relations CEC $(cmolckg^{-1}) = (C- B) \times 50$

Calculation of cation exchange capacity (CEC)

A. Total solution (mLs) = final weight of flask - the tare weight of flask – 2 g (weight of soil used).

B. Magnesium, Mg, in solution, not on CEC (cmolc) = Total solution (mLs) x 0.003 cmolc / mL of 1.5 mM magnesium sulphate solution.

C. Total magnesium, Mg, added (cmolc) = 0.1 cmolc in 10 mL of 5 mM magnesium sulphate solution + (mLs of 0.1 M magnesium sulphate solution added x 0.2 cmolc / mL of magnesium sulphate solution).

D. CEC (cmolckg^{-1}) = (C- B) x 50.

The factor 50 was used to convert the 2 g of soil to 100 g. The centimole of charge (cmolc) was calculated from the product of the concentrations per mL of the individual solutions and the charge on the magnesium ion.

Assumption: Density of water is 1gmL^{-1}

CHAPTER FOUR

RESULTS AND DISCUSSION

Questionnaire

Questionnaires administration proved that 45 % of farmers apply Confidor, 39 % use Dursban and karate, 13 % utilize Topson, Lambda, Supper and Akati master. About 3 % of farmers could not name the type of pesticides use because of their lack of ability to read, and farmers also gave reasons for using pesticides. About 83 % of farmers interviewed indicated they use the pesticides to control pests and diseases. However, they could not tell types of pests and diseases being controlled. Approximately 17 % of farmers indicated they use pesticides for their crops to grow well, and their choice of pesticides was based on recommendation by chemical sellers.

On effects of pesticides on human beings, the commonest effect indicated by 85 % of farmers was skin irritation. About 15 % also indicated that using pesticides cause their eyes to itch. On effects of pesticides on crops 92 % indicated pesticides do not affect the crops in any way apart from the purpose for which they were applied. However, 8 % of farmer disagreed as they indicated that the pesticides affect the crops by causing some of them to dry up.

On frequency of pesticide application, 70 % indicated applying the chemicals every 2 weeks, 20 % responded applying the pesticides every 10 days

and 10 % indicated applying pesticides when necessary from germination until maturity. None of these is approved for use on watermelon crops. The approved pesticides include Ridomil, Apron, Maxim, Busan, Naptalam or Alanap, Chlorothalomil, Captan, Thirim, Gavel, Mancozeb. This strongly indicates a large scale pesticides misuse among farmers in Ghana with particular reference to application on watermelon.

Quality control

Recoveries of various pesticides at 0.10 µg/kg fortification from watermelon fruit samples with florisil cleanup column are shown in the Tables 1, 2 and 3 below. The obtained recoveries were within the satisfactory range of 70.0 - 120.0 % (Kovacicova, Kovac, & Batora, 1975).

Table 1: Concentration of OP pesticides recovered from standard (µg/kg)

Pesticide	Mean (%)	Standard Deviation
Methamedophos	83.50	0.008
Enthoprophos	81.50	0.184
Phorate	84.00	0.09
Diazinon	84.50	0.006
Dimethoate	80.50	0.010
Pirimipphos	86.50	0.010
Chlorpyrifos	83.50	0.011
Fenitrothion	80.50	0.007
Parathion	84.00	0.007
Fonofos	79.50	0.007
Profenofos	79.00	0.023
Malathion	79.00	0.010
Chlorfenvinp	81.00	0.006

Table 2: Concentration of OC pesticides recovered from standard (µg/kg)

Pesticide	Mean (%)	Standard Deviation
Lindane	82.00	0.009
Heptachlor	78.00	0.009
Aldrin	79.00	0.006
Endosulfan	77.00	0.010
DDE	82.00	0.009
Dieldrin	76.50	0.009
DDD	87.50	0.008
DDT	82.00	0.008
Methoxychlor	78.00	0.010
Endrin	80.50	0.006

Table 3: Concentration of synthetic pyrethroids recovered from standard (µg/kg)

Pesticide	Mean (%)	Standard Deviation
Bifenthrin	83.50	0.009
Lambda-Cyhalothrin	86.50	0.007
Permethrin	83.00	0.006
Cyfluthrin	83.50	0.009
Cypermethrin	82.50	0.004
Fenvalerate	82.50	0.008
Deltamethrin	86.50	0.007

Particle size distribution and moisture content of the soil used

Particle size distribution analysis of soil taken from a depth of 0 –20 cm showed that soil contained high proportion of sand (52.35 – 57.59 %), followed by slit (37.31 - 42.82 %) and clay content (3.28 - 9.84%) for farm A. Particle size distribution of soil from farm B was sand (63.22 – 84.94 %), followed by silt (11.78 – 34.05 %) and clay (2.48 – 6.56 %). In farm C, the distribution was sand (59.23 – 75.99 %), silt (18.27 – 34.21 %) and clay (4.10 – 6.56 %). The proportion of particles in soil from farm D was sand (70.40 – 73.94 %), silt (17.11 – 23.86%) and clay (4.10 – 9.84 %) (Appendix A5). Mean percentage of

characteristic determined are represented in (Table 4) below. The low clay (< 0.002 mm) indicates that pesticides adsorption in the soil would be low, and would persist in the soil for a shorter period (Laabs, Amelung, Pinto, & Zech, 2002). Soils with low clay content have reduced reactive surface area for pesticide adsorption. Thus the capability of the soil to bind and retain pesticides molecules would be considerably reduced.

Table 4: Mean values of some chemical and physical properties of Nsadwir Soil

Soil Characteristics	Farm A Mean ± SD	Farm B Mean ± SD	Farm C Mean ± SD	Farm D Mean ± SD
Moisture (%)	3.00 ± 1.51	4.84 ± 1.64	3.70 ± 1.92	2.53 ± 0.39
Soil pH	6.30 ± 0.35	5.89 ± 0.47	6.49 ± 0.31	7.09 ± 0.24
Organic Matter (%)	8.30 ± 1.73	7.00 ± 2.61	9.90 ± 8.40	8.20 ± 1.34
CEC (cmolckg-^{1})	3.47 ± 0.73	3.80 ± 0.93	2.35 ± 1.14	2.82 ± 0.48
Clay %	5.74 ± 3.03	4.10 ± 1.81	5.33 ± 1.05	6.97 ± 2.54
Sand %	53.21 ± 2.43	68.89± 10.34	71.27 ±7.35	73.03 ± 1.52
Silt %	38.95 ± 2.51	25.50 ±9.98	24.21 ±6.90	20.37 ± 3.43
Texture	S.L	S.L	S.L	S.L

Mean soil moisture content ranged from 2.53 – 4.84 % (Table 4). This amount significantly favours the adsorption of pesticides. Low soils moisture renders many microorganisms in the soils inactive. This slows the breakdown rates of pesticides in the soils and consequently favours their absorption and persistence and the Water molecules which would compete with the pesticide molecules for adsorption sites on soil particles are reduced (Macalady & Wolfe 1985). This allows pesticide molecules to be firmly adsorbed onto soil particles, thus allowing high levels of accumulation (Broholm , Tuxen , Ru¨gge, & Bjerg 2001).

Some chemical properties of Nsadwir soil

The soils were also characterised by low cation exchange capacity. The pH ranged from weakly alkaline to neutral and the mean cation exchange capacity ranged from 2.35 - 3.80 cmolckg^{-1} (Table 4). Mean soils organic matter ranged from 7.0 – 9.90 % (Table 4). The organic matter fractions from all farms were high; some too high and ranged from 2.40 – 24.60 % (Appendix 5). The high organic matter could be attributed to high levels of inert organic matter or traces of charcoal derived from the burning of plant materials on individual farms (Quiroga, et al., 1996). This implies that pesticide adsorption and persistence in soil would be considerably reduced as pesticides would easily leached (Edwards, 1972).

The low cation exchange capacity could be attributed to the reduction of cation exchange sites on the soil as a result of low clay content of individual soil. This leads to a significant reduction of reactive sites on the soil particles. With such soils, pesticides adsorption and persistence would be significantly low since they disappear easily through leaching (Edwards, 1972).

Levels of pesticides in the soil and watermelon samples

The soil samples from four farms at Nsadwir were analysed for organophosphorus, organochlorine and synthetic pyrethroids pesticides. To ascertain their levels of contamination, concentrations of the different pesticide residues in the soils are presented in Tables 5, 6 and 7 below:

Table 5: Mean levels of OP pesticides in Nsadwir soils (µg/kg)

Pesticide	Mean Conc.	Maximum	Minimum	SD
Methamidophos	8.65	17.90	2.60	4.80
Enthoprophos	7.65	13.40	2.30	3.63
Phorate	76.75	176.60	2.00	55.26
Diazinon	5.10	9.50	2.30	2.03
Dimethoate	38.20	135.50	7.10	37.94
Pirimipphos	33.75	86.70	2.40	25 .38
Chlorpyrifos	3315.80	4121.70	1545.1	740.8
Fenitrothion	6.45	19.00	2.30	4.09
Parathion	16.25	35.40	4.70	9.08
Fonofos	6.75	12.70	2.80	2.77
Profenofos	72.80	170.10	10.50	43.32
Malathion	9.10	35.70	2.50	7.63
Chlorfenvinp	6.35	14.00	3.20	3.29

The concentration range and the mean levels of organophosphorus pesticides (OPs) are presented in Table 5. In general, the levels of Chlorpyrifos, Phorate, Dimethoate, Profenofos and Pirimipphos were high. This suggests that the pesticides viz Chlorpyrifos, Phorate, Dimethoate, Profenofos and Pirimipphos pesticides were able to bind more tightly to soil particles and had lower rate of leaching. Consequently, they persisted in the soil for longer period. The low levels of Metathamedophos, Enthoprophos, Diazinon, Fonofos, Malathion, and Chlorfenvinp could be attributed to fast disappearance rates from soil by leaching and surface runoff (Glotfelty, et al., 1989). Uptake by plants and migration of invertebrates or small mammals which incorporate the residues in their bodies could also have accounted for the low levels (Glotfelty, et al., 1989). The differences in the levels of the organophosphorus pesticides in the soil may perhaps also be ascribed to spontaneous entry of the pesticide into a dynamic ecosystem where they progress instantaneously from one part of the system to another and an *in situ* degradation or movement of these pesticides out of the soil system into other system as reported by (Edwards, 1972).

Table 6: Mean levels of OC pesticides in Nsadwir soil (µg/kg)

Pesticides	mean conc.	Min	Max	SD
Lindane	4.81	3.73	8.5	1.48
Heptachlor	5.15	2.10	12.90	2.52
Aldrin	5.05	2.00	12.40	2.52
P'P'- DDE	4.90	2.80	8.90	1.78
Dieldrin	3.90	2.20	9.20	1.69
P'P'-DDD	4.40	2.40	10.00	2.48
P'P'-DDT	7.05	1.20	12.20	3.78
Endosulfan	8.39	4.83	10.86	1.61
Methoxychlor	5.40	1.80	9.80	2.31
Endrin	3.45	1.10	9.70	2.88

Organochlorine pesticides in the soil had concentrations ranging 1.10 - 30.60 µg/kg. The Mean concentrations ranged from 3.45 ± 2.88 - 7.55±3.89 µg/kg. Endosulfan and DDT levels were high. This could be ascribed to repeated use of the pesticides, and/or wick effect which might have caused the pesticides to be accumulated in the soil surface. It could also be due to a significant decline in the population of soil organisms which are responsible for their degradation as a result of it long persistence in the soil (Glotfelty, et al., 1989). The decline in the population might have originated from application of mixtures of pesticides which have the ability to interfere with the activities of beneficial soil organisms other than their target species. The low levels of Endrin and Dieldrin could as well be ascribed to the uptake of the residues into the tissues of invertebrates that live in the soil, either through their body cells or in their food (Kookana, Baskara, & Naidu, 1998). Extensive breakdown of the pesticides by active soil enzymes which might have been released from dying organisms, soil micro-organisms, roots of plant or in excreta of soil animals could have also accounted for their low

levels (Kookana, et al., 1998). The levels of the organochlorine pesticides in the soil were within the 0.40 - 47.90 μg/kg detected in soils (Li & Lee, 1974).

Table 7: Mean levels of synthetic pyrethroids in Nsadwir soil (μg/kg)

Pesticide	Mean Conc.	Max. Conc.	Min. Conc.	SD
Bifenthrin	3.40	6.70	2.10	1.38
Lambda-Cyhal	3.85	5.30	2.10	1.04
Permethrin	2.65	4.80	1.20	0.93
Cyfluthrin	3.20	4.80	1.10	1.08
Cypermethrin	2.80	4.10	1.10	0.85
Fenvalerate	5.20	8.20	2.20	2.20
Deltamethrin	3.50	4.10	2.30	0.42

The extractable synthetic pyrethroid pesticides levels ranged from 1.10-8.20 μg/kg. The mean concentration of Fenvalerate, 5.20 ± 2.20 μg/kg was the highest level detected. In general, the synthetic pyrethroid pesticides detected in the soils were comparatively lower than the organophosphorus and organochlorine pesticides detected from the soils. The levels may result from an increased rate of leaching due to low soil organic matter and low clay contents (Harris 1966, 1967). The low levels may due also to effect of temperature gradient. A phenomenon which causes pesticide molecules to move from a higher to a lower temperature zones in the soil and thus reducing the concentration at depth 0-20 cm (Nielsen, Jackson, Carry, & Evans, 1972). Although there is presently no Ghanaian standard documenting the maximum permissible levels of pesticides in soils, their occurrence in the soils point to a possible environmental contamination though levels were all below the 50 - 10000 μg/kg detected in soils at depth of 0-30 cm by (McAllister, 1994).

Correlation analysis

To establish the factors accountable for the variations in the residue levels, and to substantiate importance of these factors, a correlation analysis was performed. Tables 8, 9 and 10 below show the results of the correlation of soil properties with the organophosphorus, organochlorine and synthetic pyrethroid pesticides in the soils.

The soil moisture and clay showed no significant correlation with the organophosphorus pesticides. However, the pH correlated significantly with Phorate and Diazinon ($P < 0.05$). The negative correlation observed for Phorate and Diazinon suggested that both pesticides degraded faster in the soil with increasing pH. The correlation also indicated a shorter period of persistence in the soil. This leads to corresponding low levels in soils. The faster degradation could also mean that majority of soil micro-organisms were active and might have fed on the pesticides (Mortland & Raman, 1967).

The clay content of the soil showed a significant correlation with Methamedophos and Pirimipphos (Table 8). The correlation was negative with Methamedophos ($r = 0.435$, $P < 0.05$, $n = 16$). The negative correlation means that either Methamedophos adsorption onto clay decreased with decreased clay content or its degradation was rapid as the clay content decreased. A positive correlation however existed between the clay content and Pirimipphos ($r = 0.499$, $n = 16$, $P \leq 0.05$). The sand content had a strong positive correlation with Methamedophos and Fonofos with ($r = 0.580$, $n = 16$, $P \leq 0.01$) for Methamedophos and ($r = 0.632$, $n = 16$, $P \leq 0.01$) for Fonofos. The correlation

was more positive with Fonofos showing a slower degradation of the pesticide in soil. It also proved its longer persistence period and the subsequent high levels. The Sand content correlated negatively with both Diazinon (r = - 0.533, n = 16, P $\leq$ 0.05) and Dimethoate (r = 0.597, n =16, P $\leq$ 0.05). The correlation coefficients also proposed that both pesticides have comparable degradation rates in sandy soils. The negative correlation coefficient for Diazinon suggested its rapid degradation in the soils. This implies shorter persistence and low levels in the soils. The correlations were significant (Table 8).

The silt content of the soils showed a significant positive correlation with Diazinon and Dimethoate. The correlations were significant (r = 0.561, n=16, P $\leq$ 0.05) for Diazinon and (r = 0.542, n = 16, P $\leq$ 0.05) for Dimethoate. The effect of silt on the degradation rates of both pesticides was similar as suggested by the correlation coefficients. Fonofos on the other hand correlated negatively and significantly with the silt content of the soils(r = 0.669, n= 16, P $\leq$ 0.01). Fonofos degraded at a faster rate in soils with small amounts of silt as predicted by the correlation coefficient.

Methamedophos correlated negatively with the cation exchange capacity. It was highly significant (r = 0.697, n = 16 P$\leq$ 0.01). This indicates an increased rate of adsorption and a decreased degradation rate with high CEC. The faster rate could be due to the absence or significantly reduced interaction between Methamedophos molecules and the cation exchange capacity (CEC) sites on the soil particles. For Diazinon, the correlation with CEC was significant (r = 0.509, n =16, P $\leq$ 0.05).

Table 8: Correlation of soil properties with levels of OP pesticides

Pesticide	Moisture	pH	OM	Clay	Sand	Silt	CEC
Methamedophos	-0.221	0.094	-0.395	-0.435*	0.580**	-0.490	-0.697**
Enthoprophos	0.309	-0.177	0.278	-0.284	0.162	-0.088	-0.173
Phorate	0.334	-0.586*	0.058	-0.466	-0.362	0.469	0.006
Diazinon	0.373	-0.595*	0.367	-0.074	-0.533*	0.561*	0.509*
Dimethoate	-0.020	-0.267	-0.060	0.286	-0.579*	0.524*	0.046
Pirimipphos	-0.145	0.173	0.151	0.499*	-0.248	0.138	0.082
Chlorpyrifos	-0.199	0.183	0.347	0.328	0.178	-0.264	0.038
Fenitrothion	-0.269	0.419	0.215	0.344	-0.013	-0.076	0.041
Parathion	-0.077	0.038	0.111	0.111	-0.125	0.088	0.144
Fonofos	0.034	0.405	0.120	0.108	0.632**	-0.669**	-0.159
Profenofos	0.143	0.154	0.421	0.374	0.292	-0.383	0.348
Malathion	-0.026	-0.053	0.105	-0.308	-0.007	0.084	-0.252
Chlorfenvinp	-0.126	0.131	0.322	-0.072	0.239	-0.226	-0.397

*Correlation is significant at the 0.05 level and **Correlation is significant at the 0.01 level

Table 9: Correlation of soil properties with levels of OC pesticides

Pesticide	Moisture	Soil pH	Organic Matter	CEC	Clay	Sand	Silt
Lindane	-0.056	-0.093	-0.288	0.171	0.165	0.008	-0.061
Heptachlor	-0.330	-0.207	0.077	0.103	0.141	-0.605*	0.566*
Aldrin	-0.133	0.024	-0.050	0.127	0.016	-0.359	0.357
Endosulfan	-0.331	0.153	0.019	-0.359	0.406	-0.114	0.016
P'P'-DDE	0.081	-0.498*	-0.102	0.493	-0.304	-0.496	0.567*
Dieldrin	0.081	-0.408	-0.059	0.219	-0.311	-0.324	0.385
P'P'-DDD	0.113	-0.533*	-0.308	0.245	-0.285	-0.003	0.057
Methoxychlor	0.197	-0.463	0.152	0.559*	-0.212	-0.736**	0.799**
Endrin	0.080	-0.467	-0.330	0.412	-0.340	0.371	0.572
P'P'-DDT	0.062	0.096	0.334	-0.442	-0.139	0.229	-0.188

The relation between the soil properties and the soil organochlorine pesticides (Table 9) did not show significant correlations between the soil properties and Lindane, Aldrin, Endosulfan, Dieldrin, Endrin and DDT.

Significant correlations were however observed between Heptachlor and sand (r = 0.605, n =16, P ≤ 0.05) and Heptachlor and silt (r = 0.566, n=16, P ≤ 0.05).

DDE correlated negatively with pH (r= -0.498, n = 16, P ≤ 0.05) and positively with silt (r = 0.567, n = 16, P ≤ 0.05). DDD correlated negatively with pH (r = -0.533, n = 16, P ≤ 0.05). The correlation was more negative with DDD than DDE. This shows that DDD persisted in the soils as pH increased. It also suggests that there was tight adsorption of DDD molecules onto soil particles which led to longer persistence in the soil and subsequently elevated levels.

Methoxychlor correlated positively with cation exchange capacity and silt but negatively with the sand (Table 9). While the silt enhanced adsorption, high sand content reduces adsorption of the pesticides and hence the high significant negative correlation (r = - 0.736, n = 16, P ≤ 0.01). Even though most of the soil properties had significant effects on the persistence of organochlorine pesticides, the effect of moisture and organic were not significant. This probably could be due to the very low levels of the pesticides in the soil which ranged from 1.10 - 8.50 µg/kg (Table 6).

Table 10: Correlation soil properties with levels of synthetic pyrethroids

Pesticides	%Moisture	Soil pH	%OM	CEC	%Clay	%Sand	%Silt
Bifenthrin	0.02	-0.04	0.35	-0.40	-0.05	0.26	-0.25
Lambda-cy	-0.11	0.38	-0.09	-0.40	-0.11	0.48	-0.46
Permethrin	-0.18	0.59*	-0.02	0.04	0.39	0.08	-0.16
Cyfluthrin	-0.41	0.65**	0.19	-0.39	0.39	0.06	-0.16
Cypermethrin	-0.34	0.64*	-0.17	-0.42	0.28	0.50**	0.57*
Fenvalerate	0.48	-0.29	0.25	-0.00	-0.43	0.47	-0.37
Deltamethrin	0.09	0.136	0.268	-0.24	0.17	0.03	-0.07

The soil properties did not significantly correlate with the synthetic pyrethroid pesticides Bifenthrin, Lambda-Cyhalothrin, Fenvalerate, and Deltamethrin (Table 10). Permethrin, Cyfluthrin and Cypermethrin however correlated positively with pH. For Permethrin ($P \leq 0.05$), Cypermethrin ($P \leq 0.05$) and for Cyfluthrin ($P \leq 0.01$). The coefficients of correlation between pH and extractable Permethrin, Cyfluthrin and Cypermethrin showed that the degradation of the pesticides decreased as the pH increased. However, highly significant correlation observed between Cyfluthrin and pH (Table 10) showed that degradation of Cyfluthrin was the slowest among them. Sand and silt contents showed a positive correlation with Cypermethrin ($P \leq 0.01$ and $P \leq 0.05$) respectively.

Levels of Pesticides in Watermelon Fruits from Various sampling sites

Watermelon fruits from Nsadwir, Ayensudo, Cape Coast, Sekondi – Takoradi, Kumasi, Accra and Bolgatanga were analysed for organophosphorus, organochlorine and synthetic pyrethroids pesticides. The average concentration of each pesticide was compared to the (WHO/FAO, 2006) permissible levels, (USA, 2009) allowable residue limits, (RussianHygieneAuthority, 2007) Standards, Thailand Agricultural Standard (TAS, 2008) maximum residue limits, (Japan, 2009) permissible levels and the (EU, 2006) maximum residue limits (MRLs).

Table 11: Mean levels OP pesticides in watermelon from Nsadwir (µg/kg)

Pesticides	Min. conc.	Max. Conc.	Mean conc.	SD	WHO/FAO Recommended
Methamedophos	3.20	10.10	4.98	2.06	4.00
Enthoprophos	2.30	9.00	3.95	1.98	0.40
Phorate	4.10	18.50	9.35	5.06	0.70
Diazinon	2.50	12.90	5.75	3.71	2.00
Dimethoate	4.70	63.20	21.90	19.07	2.00
Pirimipphos	4.90	31.00	8.70	7. 80	30.00
Chlorpyrifos	1069.40	4383.20	3049.30	1123.70	10.00
Fenitrothion	2.30	114.00	81.65	45.64	5.00
Parathion	3.20	17.30	8.80	5.070	3.00
Fonofos	2.10	8.50	5.65	1.97	10.00
Profenofos	2.70	15.00	6.55	4.13	10.00
Malathion	5.50	36.7	12.15	10.78	3.00
Chlorfenvinp	3.70	18.90	7.70	5.51	10.00

The results in Table 11 above show that the concentration of the Methamedophos ranged from 3.20 - 10.10 µg/kg. The mean concentration in the watermelon fruit was 4.98 ± 2.0 6µg/kg. Enthoprophos, Phorate, Diazinon, Dimethoate, Pirimipphos and Chlorpyrifos concentrations ranged from 2.30 - 4383.20 µg/kg (Table 11). These mean concentrations were higher than the WHO/FAO recommended maximum residue levels (MRLs) of 4.0 µg/kg, 0.4 µg/kg, 0.70 µg/kg, 2.0 µg/kg, 2.0 µg/kg and 10.0 µg/kg set for the pesticides. These levels could be due to the fact that the pesticides were applied a few days before the fruits were harvested at a time that no significant loses might have occurred through leaching and degradation. Pirimipphos mean levels detected were, however below the 30.0 µg/kg recommended level (WHO/FAO, 2006). Fenitrothion, Parathion, Fonofos, Profenofos Malathion and Chlorfenvinp were also present in the watermelon fruit with mean concentrations above the documented WHO/FAO permissible levels. For instance the mean levels of Profenofos, Parathion and Chlorpyrifos were respectively two, fourteen and two

hundred times higher than the permissible maximum residue levels jointly set by WHO/FAO (2006).

Table 12: Mean levels of OC pesticides in watermelon from Nsadwir (µg/kg)

Pesticides	Min. Conc.	Max. Conc.	Mean. Conc.	Standard Deviation	WHO/FAO Recommended
Lindane	5.06	8.56	7.13	1.43	10.0
Heptachlor	2.70	9.80	8.20	2.49	0.10
Aldrin	3.20	8.90	5.60	1.42	0.10
P'P'-DDE	2.80	9.10	4.90	1.63	10.0
Dieldrin	2.10	9.80	3.80	2.62	0.10
P'P'-DDD	2.40	6.50	3.70	1.25	10.0
P'P'-DDT	4.20	10.80	6.80	2.01	10.0
Endosulfan	6.23	17.9	8.08	3.21	6.00
Methoxychlor	3.10	13.90	6.45	3.27	50.00
Endrin	1.10	19.30	7.65	6.00	0.20

Concentrations of the organochlorine pesticides detected in the watermelon fruit ranged from 1.10 - 17.9 µg/kg (Table 12). The mean concentrations of Heptachlor, Aldrin, Dieldrin, Endosulfan (sum of alpha, beta and the sulphate) and Endrin were higher than the WHO/FAO acceptable values of 0.10 µg/kg, 6.0 µg/kg and 0.20 µg/kg. These high levels could be attributed to the failure of the crop to metabolize the pesticides much more rapidly, thus resulting in accumulation in the fruits (Glotfelty, et al., 1989). The mean concentrations of Lindane, DDT, DDE and DDD were lower than the documented WHO/FAO permissible levels (Table 12).

Table 13: Mean levels of synthetic pyrethroids in watermelon from Nsadwir

(µg/kg)

Pesticide	Min. conc.	Max. conc.	Mean conc.	Standard Deviation	WHO/FAO Recommended
Bifenthrin	2. 6 0	5.30	3.85	0.89	20.0
Lambda-Cyha	2.70	6.30	5.30	1.42	20.0
Permethrin	1.90	5.30	2.85	1.24	50.0
Cyfluthrin	1.90	6.40	4.50	1.45	20.0
Cypermethrin	2.20	4.30	3.55	0.62	50.0
Fenvalerate	1.60	5.50	3.20	1.28	20.0
Deltamethrin	2.40	5.20	3.35	0.82	10.0

As shown in Table 13 above, the mean concentrations of all residues detected in watermelon fruit from Nsadwir were below the WHO/FAO recommended maximum residue limit (MRLs). The residue levels were compared to Thailand and Russia permissible maximum residue limits and were found that Bifenthrin, Lambda – Cyhalothrin, Permethrin, Cyfluthrin, Cypermethrin, Fenvalerate and Deltamethrin levels were far below Thailand permissible levels of 50 µg/kg for Bifenthrin, Permethrin, Cypermethrin and Deltamethrin and Russia accepted 10 µg/kg for Bifenthrin, Permethrin, 20 µg/kg for Cypermethrin and 50 µg/kg for Lambda-Cyhalothrin. The low levels could be due to rapid metabolization of the pesticides in the crops or the ability of the crops to significantly dilute the residues to such low levels. It could also be due to reduced frequency of application.

Table 14: Mean levels of OP pesticides in watermelon from other sites (µg/kg)

Pesticide	E & F	G	H	I	J	K	WHO
Meth.	13.85±3	7.55±0.91	16.50±1	13.30±3	15.35±1	32.20±27.	4.0
Enth.	6.05±2.3	6±1.41	8.25±0.91	21.80±10.8	7.95±1.76	12.05±2.0	0.4
Phorate	5.85±4.28	8.55±4.73	3.55±0.91	8.60±5.51	6.85±6.43	9.90±3.67	0.7
Diaz.	7.30±2.39	7.20±0.28	3.85±2.75	13.40±13.0	6.80±2.68	16.60±6.7	2.0
Dim	26.40±13	89.95±74	84.40±71	61.60±13	37.55±10	34.80±7	2.0
Pir	3.40±3.00	6.35±6.29	9.30±1.97	19.10±15.4	16.40±2.1	16.85±5.4	30.0
Chp	1936±55	1405±26	314±267	207±126	249±26	176±94	10.0
Feni	8.05±3.56	13.45±12	5.50±0.84	11.20±4.3	5.45±0.3	14.80±3	10.0
Para	7.65±4.44	7.85±2.05	5.20±1.41	7.85±4.17	5.95±0.2	9.30±2.6	3.0
Fono	6.00±3.33	9.25±5.1	5.50±0.84	17.55±10	7.70±0.4	9.95±0.3	10.0
Prof	9.65±5.67	8.90±8.0	6.70±0.56	26.55±19	8.50±5.9	7.10±1.1	10.0
Mala	10.25±6.7	8.85±0.7	30.15±16	40.10±15	15.75±3	13.10±5	3.0
Chlf	6.95±3.84	5.60±2.5	6.20±0.14	5.55±0.35	10.40±2	5.95±0.4	10.0

Meth – Methamedophos, Enth – Enthoprophos, Diaz – Diazinon, Dim – Dimethoate, Pir – Pirimipphos, Chp – Chlorpyrifos, Feni – Fenitrothion ,Para–Parathion, Fono–Fonofos, Prof-Profenofos, Mala–Malathion ,Chlf–Chlorfenvinp

Watermelon fruits from Ayensudo (labelled E&F) had Methamedophos, Enthoprophos, Phorate, Diazinon, Dimethoate, Chlorpyrifos, Parathion and Malathion mean concentrations above 4.0 µg/kg for Methamedophos, 0.4 µg/kg for Enthoprophos, 2.0 µg/kg for Phorate, 10.0 µg/kg for, Diazinon and 3.0 µg/kg for Dimethoate threshold jointly established by WHO/FAO (Table14). The average concentrations of Pirimipphos, Fenitrothion, Fonofos, Profenofos and Chlorfenvinp were below the maximum residue limits of set by WHO/FAO (Table 14). The mean levels of the pesticides detected in Sekondi-Takoradi, Accra, Kumasi, Bolgatanga and Cape Coast labelled G, H, I, J and K (Table14) were similar to the levels detected in watermelon fruits from Ayensudo. The high levels of the pesticides (Table14) could be attributed to the frequency of their application, the stage of the crop development when the pesticides were applied, resistance of the pesticides to photolytic degradation. The inability of the watermelon crops to metabolize the pesticides could have also contributed significantly to the high levels detected.

Table 15: Mean levels OC pesticides in watermelon from other sites (µg/kg)

Pesticide	E& F	G	H	I	J	K	WHO levels
Lindane	6.88±2.93	4.77±0.89	4.91±0.96	4.84±0.82	8.04±0.54	9.91±0.35	10.0
Heptach	9.35±6.44	8.70±6.36	9.30±2.96	8.25±5.16	9.40±3.88	7.75±2.54	0.10
Aldrin	3.80±3.69	1.15±0.07	7.10±2.54	4.90±3.53	9.10±2.75	1.85±0.91	0.10
Endon	9.83±3.5	9.74±2.4	12.73±1.8	11.03±3.53	16.55±4.73	28.68±8.23	10.0
Dieldrin	4.40±4.1	4.85±3.3	3.75±0.77	6.10±4.66	3.90±1.62	3.80±1.83	0.10
DDE	3.55±3.4	3.50±0.7	4.10±0.28	3.05±0.21	7.10±3.39	5.70±2.12	10.0
DDD	5.45±2.8	3.40±3.1	2.65±2.19	7.30±7.07	3.20±1.13	2.65±2.05	10.0
DDT	4.70±2.8	2.90±0.8	3.25±2.75	6.75±3.60	11.10±4.5	4.15±3.32	6.0
Methox	11.70±9	5.60±4.9	15.45±8.6	8.55±7.42	14.30±1.3	14.60±2.9	50.0
Endrin	4.90±2.1	2.15±2.0	2.00±0.14	8.45±6.01	13.90±6.5	5.75±3.74	0.20

Heptach   - Heptachlor, Endon - Endosulfan, Methox – Methoxychlor , Ayensudo – E & F,

Sekondi-Takoradi – G, Accra – H, Kumasi – I, Bolgatanga- J, Cape Coast - K

The GC analysis of watermelon fruits for organochlorine pesticides in samples from Ayensudo (labelled E&F) recorded average concentrations of residues which were below the WHO/FAO recommended residue limits for Lindane, Endosulfan, DDE, DDD, DDT and Methoxychlor (Table 15). High residue levels exceeding WHO/FAO permissible limits were recorded for Heptachlor, Aldrin, Dieldrin and Endrin (Table 15). The high levels could be as a result of the frequency of pesticide application as 70 % and 20 % of farmers indicated applying pesticides every 2 weeks and every 10 days respectively from germination until the fruits were harvested. This might have resulted in high soil residue levels which intend made available sufficient amount to be uptaken by the crops. The concentration of the residues detected in the fruit flesh ranged from 0.20 - 34.30 µg/kg (Appendix A18).

The Sekondi – Takoradi watermelons (labelled G) results showed a similar pattern in the levels of residues detected in the fruit flesh from the other six

sampling sites. The mean levels detected for Lindane, Endosulfan, DDE, DDD, DDT and Methoxychlor were below WHO/FAO acceptable levels. The mean concentrations recorded for Heptachlor 8.70 ± 6.36 µg/kg, Aldrin 1.15 ± 0.07 µg/kg; Dieldrin 4.85 ± 3.32 µg/kg and Endosulfan 9.74 ± 2.42 µg/Kg were higher than WHO/FAO approved levels indicated (Table 15).

Comparatively, the residue levels Heptachlor, Aldrin, DDE, DDT, DDD, Methoxychlor and Endrin were high in fruits from Ayensudo than corresponding levels in fruits from the Sekondi – Takoradi Metropolis. Lindane, Endosulfan and Dieldrin levels however were high in fruits from the Sekondi – Takoradi Metropolis than the corresponding levels in fruits from Ayensudo.

From (Table 15), watermelons from Accra (Labelled H) were found to contain Heptachlor, Aldrin, Endosulfan and Dieldrin and Endrin as the main organochlorine pesticide contaminants since their mean concentrations were higher than the 0.10 µg/kg, 10.0 µg/kg and 0.20 µg/kg WHO/FAO recommended levels (Table 15) for individual pesticides, and their concentrations ranged from 0.7 - 13.20 µg/kg (Appendix A 18). The high levels might have originated from their ability to resist photolysis. This resulted in slower degradation, long persistence period and subsequently accumulated in the fruits (Armburst, 1992).

Lindane, DDE, DDD, DDT, and Methoxychlor concentrations ranged from 1.10 - 21.60 µg/kg. The mean concentrations detected for Lindane 4.91 ± 0.96 µg/kg, DDE 4.10 ± 0.28 µg/kg, DDD 2.65 ± 2.19 µg/kg, DDT 3.25 ± 2.75 µg/kg and Methoxychlor 15.45 ± 8.69 µg/kg were below permitted levels suggested by WHO/FAO (Table 15). The mean levels detected for the pesticides,

particularly DDT and its metabolites, DDD and DDE might have originated from remnants in soils which became available for absorption by the crops as a result of their long history of persistence in the environment years after applications had ceased.

The mean concentrations of Lindane, DDE, Methoxychlor, DDD and DDT levels recorded in watermelon fruits from Kumasi labelled I (Table 15) were below WHO/FAO recommended levels. Their concentrations ranged from 2.30 - 13.80 µg/kg (Appendix A19). Although the levels were low, their presence in the fruits presents some scope of contamination.

Heptachlor, Aldrin, Endosulfan, Dieldrin and Endrin concentrations were in the range of 2.40 -13.53 µg/kg. Their mean levels were above those authorized by WHO/FAO. These show a clear case of contamination of the fruits by these pesticides. The use of uncalibrated and faulty spraying equipment with enlarged nozzles, application of pesticides few weeks before crops were harvested and application of pesticides at high concentrations could have responsible for the contaminations observed in the fruits.

Most organochlorine pesticides detected in watermelon fruits from Bolgatanga (labelled J) had mean levels above WHO/FAO allowable levels. The levels by virtue of magnitude could be considered contaminants which could impact negatively on life. Lindane, Heptachlor, Aldrin, Endosulfan, Dieldrin, DDT and Endrin levels ranged from 2.40 - 13.35 µg/kg (Appendix A19) and the average residue levels were from 3.20 ± 1.13 - 23.03 ± 9.16 µg/kg (Table 15). DDD, DDE and Methoxychlor levels ranged from 3.20 ± 1.13 -14.30 ± 1.34

µg/kg and were below the levels considered safe by WHO/FAO. The concentrations ranged from 2.30 - 14.30 µg/kg. The levels of organochlorine pesticides in watermelon fruits from the Cape Coast Metropolis labelled K (Table 15) ranged from 1.20 - 34.50 µg/kg. The mean levels ranged from 2.65 - 28.68 µg /kg.

Table 16: Mean levels of synthetic pyrethroids in watermelon from other sites
(µg/kg)

Pesticide type	E&F	G	H	I	J	K	WHO/FAO
Bifenthrin	0.95±0	1.50±0.4	2.15±0.3	1.75±1.2	2.40±0.7	1.20±0.4	20.0
Lambda-c	1.45±0	2.20±0.9	2.40±1.8	2.40±0.9	1.55±0.0	1.65±0.3	20.0
Perm	2.00±0.8	2.05±0.0	2.35±0.0	3.30±1.9	2.35±1.7	1.70±0.5	50.0
Cyflu	2.35±0.9	1.25±0.0	2.05±1.0	2.55±0.4	2.60±0.7	1.85±0.0	20.0
Cyper	1.60±0.9	1.20±0.1	1.30±1.1	0.85±0.3	1.70±0.1	1.70±0.7	50.0
Fenv	1.70±0.8	2.15±0.3	1.80±0.5	1.55±0.2	3.05±0.9	2.00±0.5	20.0
Deltam	1.40±0	1.40±0	2.10±0	1.70±0.56	0.95±0.21	1.00±0.28	10.0

Lambda – C –Lambda – Cyhalothrin, Perm-Permethrin, Cyflu – Cyfluthrin, Cyper –Cypermethrin, Fenv –Fenvalerate, Deltam – Deltamethrin, Ayensudo –E & F, Sekondi-Takoradi – G, Accra – H, Kumasi – I, Bolgatanga - J, Cape Coast – K

The levels of synthetic pyrethroids ranged from 0.10 - 4.10 µg/kg (Appendices A21, A22& A23) and the mean levels were in the range 0.85 ± 0.35 - 3.30 ± 1.97 µg/kg (Table 16). All the levels were below the recommended levels by WHO/FAO. The low levels could be attributed to the ability of the watermelon crops to metabolize the pesticides, application of the pesticides several months before crop harvesting, application of the pesticides at reduced concentrations. Although the levels appeared to pose no threat to humans, a disturbing scenario identified is the gross misapplication of the pesticides since they are registered for use on cotton plants and for cocoa sacks.
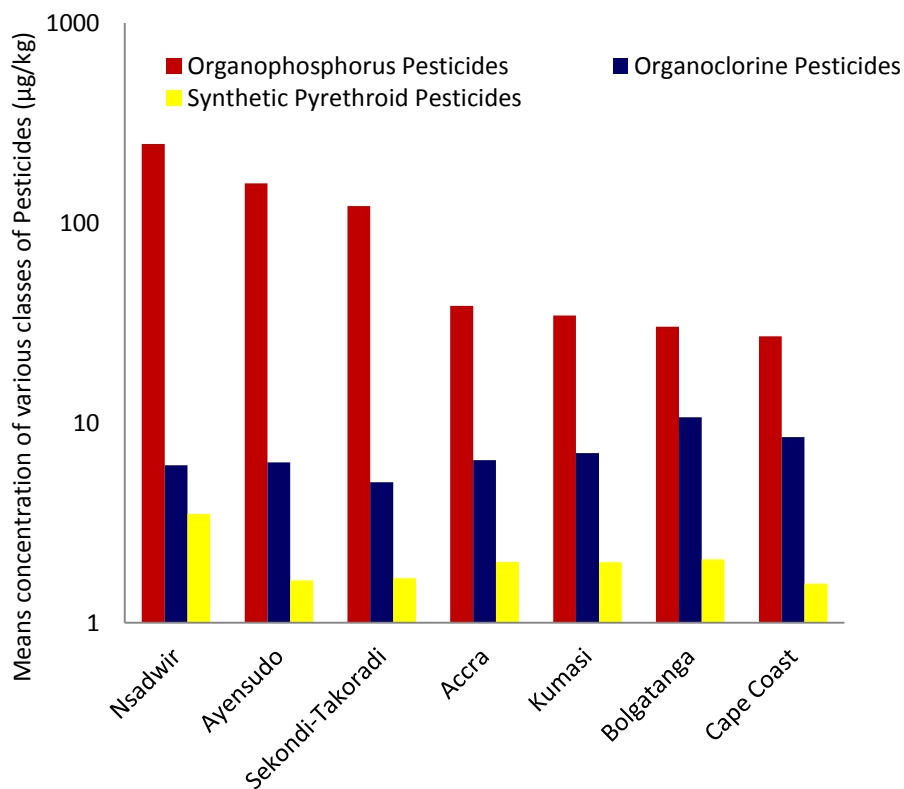
Figure 3 : A comparison of mean levels of OPs, OCs and synthetic
pyrethroids in watermelon from all sample sites

From Fig 3 above, organophosphorus and organochlorine pesticide residue were high in fruits collected from all seven sampling sites. In general the synthetic pyrethroid pesticides residues were low. These low observed levels could be due to the application of the pesticides at low dosages. The use of high pressure pumps which might have created a greater percentage of drift susceptible pesticide particles and non calibrated application equipment for lower boom height could have contributed to the low residue detected for the synthetic pyrethroid pesticides. The high levels of the organophosphorus and organochlorine pesticide residues particularly those from Nsadwir could be attributed to the application of pesticides more often than recommended by the manufacturers. The high organic matter content could also explain the high levels

of the organophosphorus levels detected in the fruits. This is because organic matter does not have anion exchange capacity and therefore not able to bind the phosphate anions. The phosphates were released easily into the soil solution and became available for the plant to absorb (Caro, Freeman , Clotfelty, Tunner, & Edwards, 1973). The high levels could also due to application of spray mixtures made more concentrated, and the use of spraying equipment with enlarged nozzle holes which discharges the pesticides at high dosage.
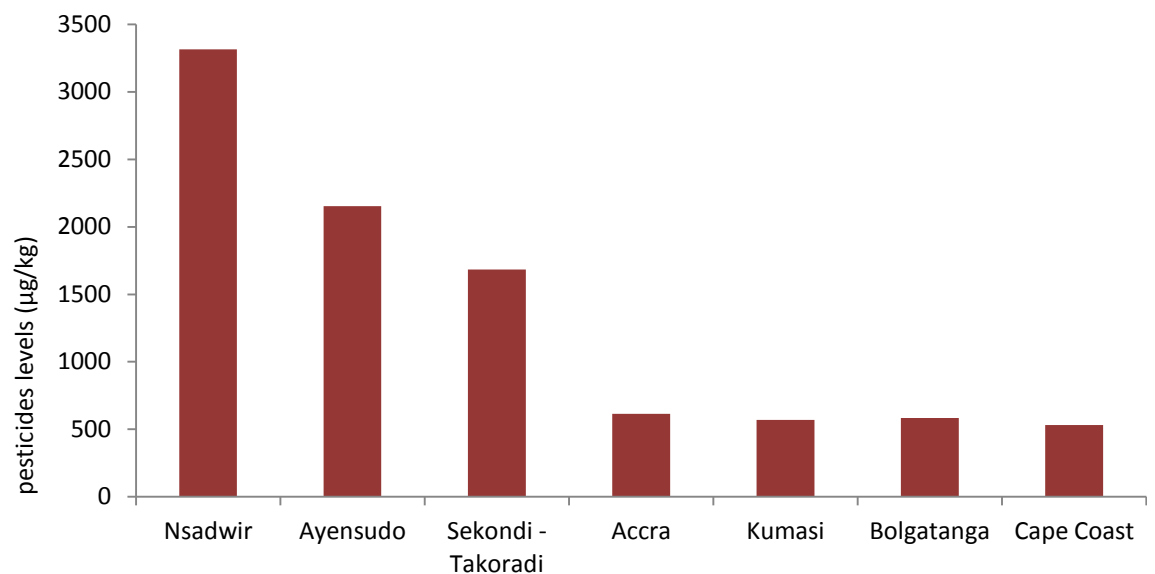
Figure 4: A comparison of total levels of pesticides in watermelon from all sample sites

A comparison of total pesticides in watermelon fruits from all sample sites showed that Nsadwir, Ayensudo and Sekondi- Takoradi had high levels of pesticides. Those in Accra, Kumasi, Bolgatanga and Cape Coast had levels relatively low (Figure 4). Based on the residue levels indicated (Figure 4), it appears that watermelon fruits from

Accra, Kumasi, Bolgatanga and Cape Coast may pose a little health threat to humans when consumed.
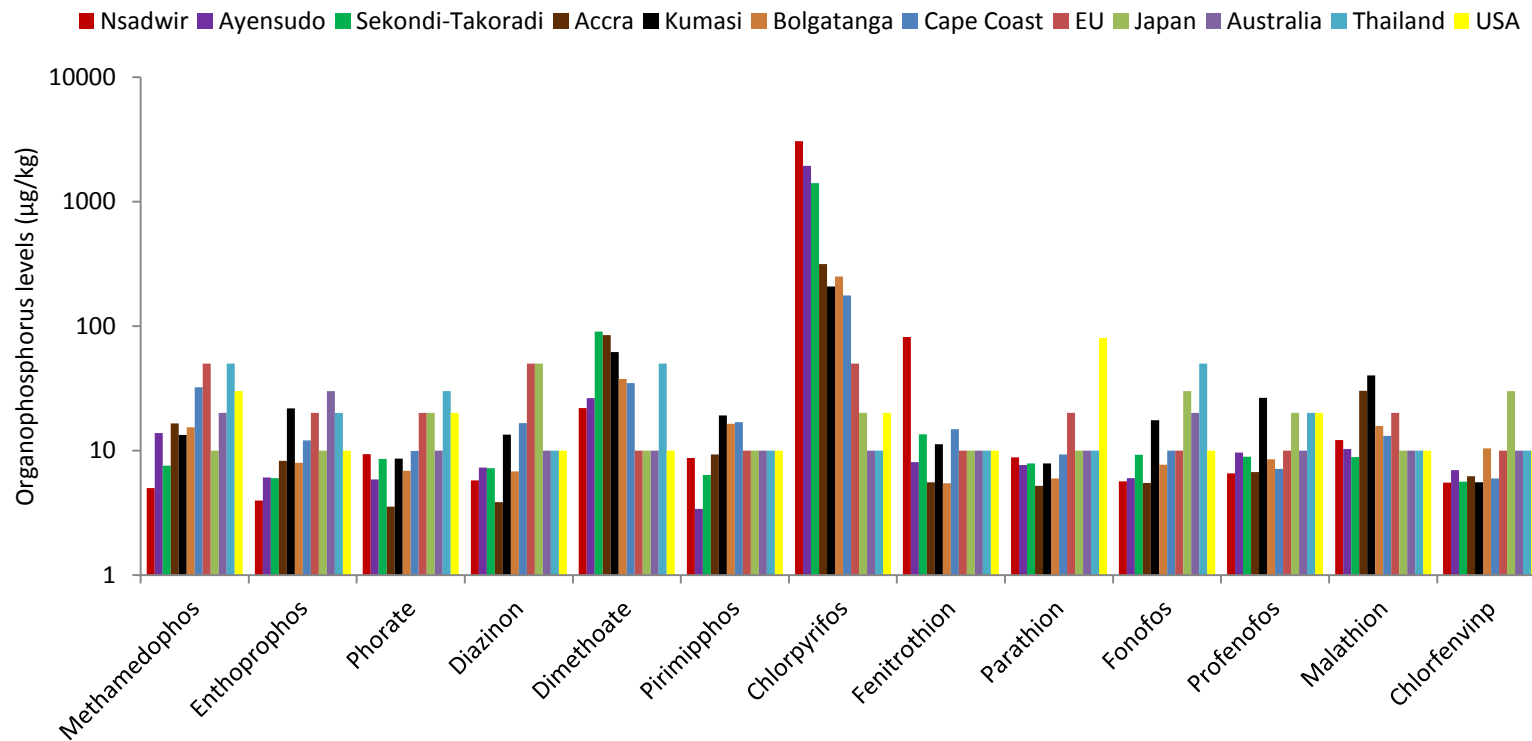
Figure 5 : A Comparison of OP pesticides levels in watermelon from study sites to the levels permitted by some countries and the E U
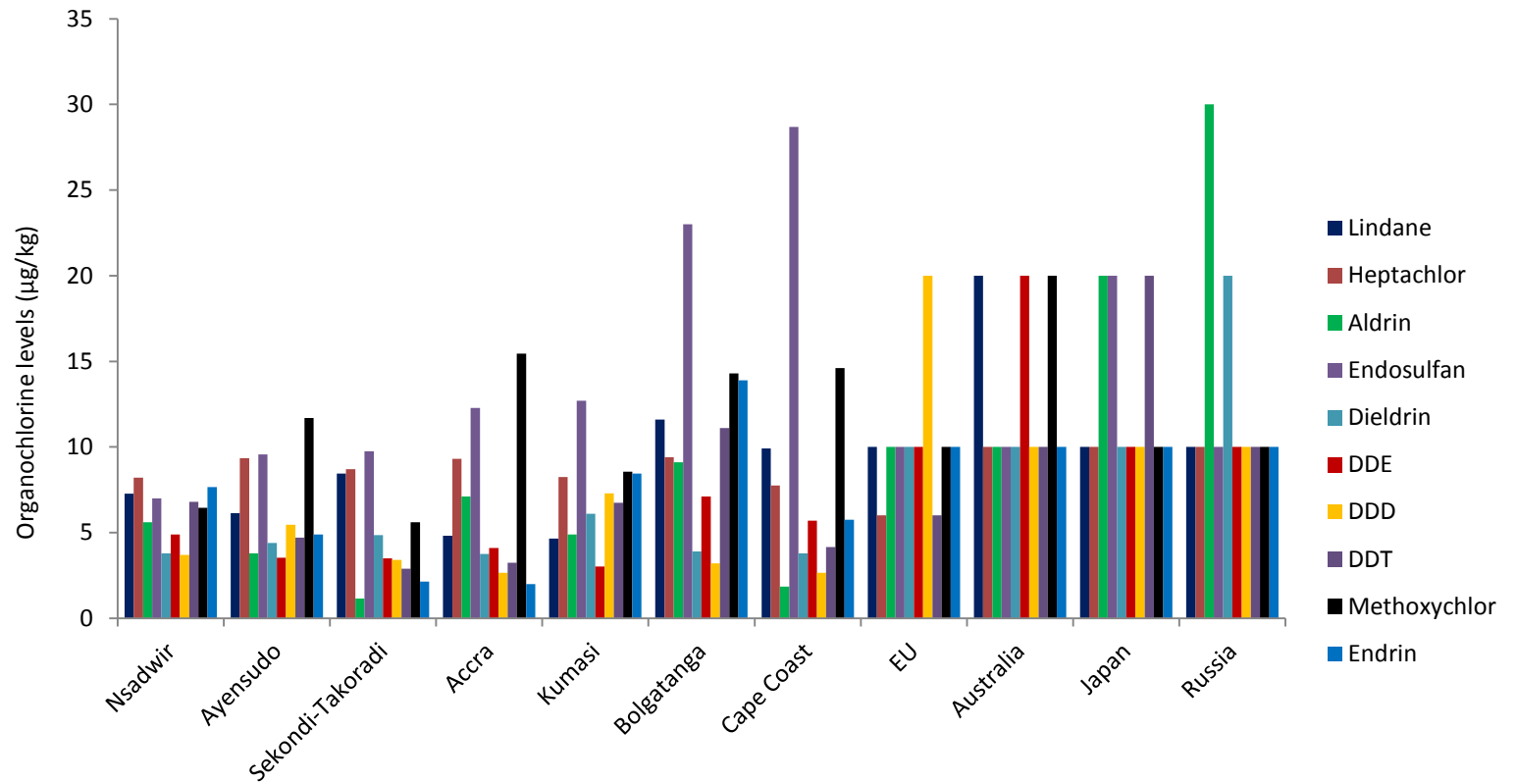
Figure 6 :   A Comparison of OC pesticides levels in watermelon from study sites to the levels permitted by some countries and the EU
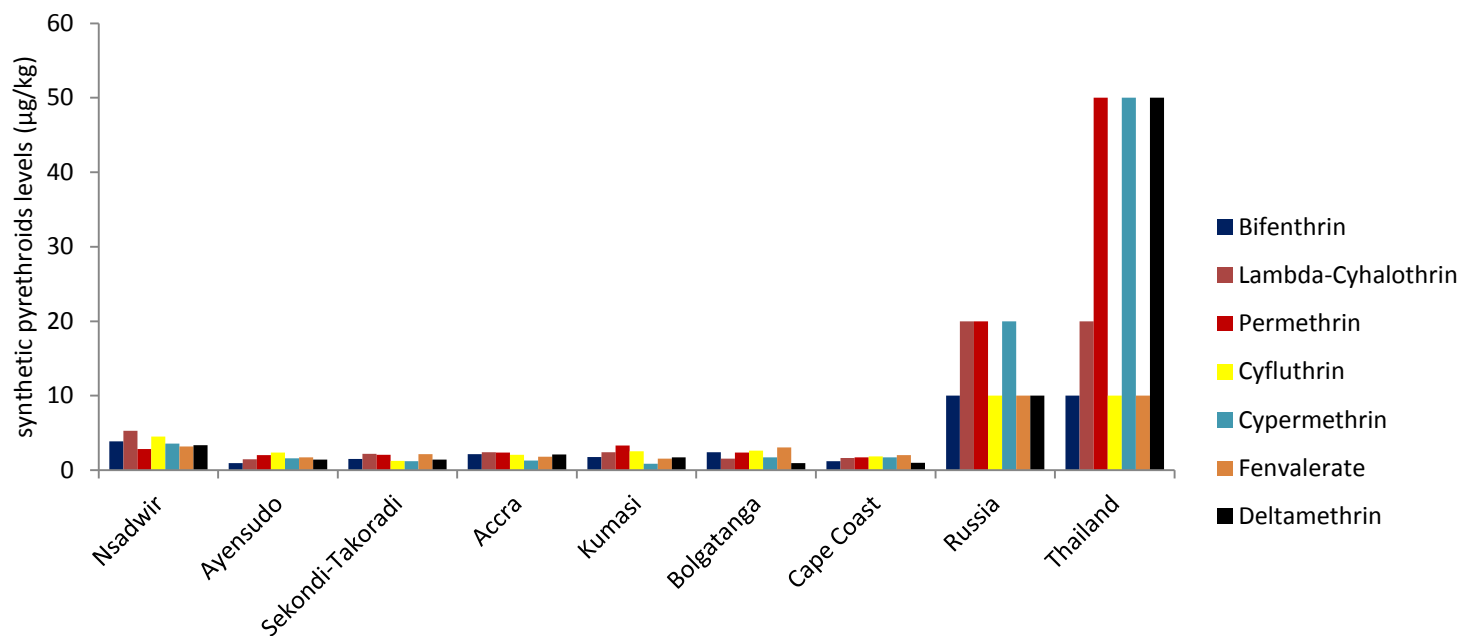
Figure 7:   A Comparison of synthetic pyrethroids levels in watermelon from study sites to the levels permitted by
Russia and Thailand

A comparison of the levels of various pesticides in watermelon fruits in Ghana to the levels permitted by the

European Union, Russia, Australia, Japan, Thailand and the Unites States (Figs 5, 6 and 7) showed that the levels of

synthetic pyrethroids in the fruits were generally low. However, this does not obliterate the fact that Ghana's

watermelons have high levels of pesticides particularly the organophosphorus and the organochlorine pesticides since

most of the levels were higher the recommended WHO/FAO (2006) levels which are considered safe.

Pesticides are manufactured taken into account the morphology of the crops for which they are developed. Failure on the part of farmers to seek expert advice in selecting the required pesticides, and wrong timing for application particularly when crops are nearing maturity could influence the behaviour of the chemicals towards the crops and in the soil.

Table 17: A comparison of mean levels of OPs, OCs and synthetic pyrethroids in watermelon fruit and fruit
rind from all sites

OPs Pesticides	Mean conc. in fruit(µg/kg)	Mean conc. in Rind(µg/kg)	OCs Pesticides	Mean conc. in fruit (µg/kg)	Mean conc. in Rind (µg/kg)	Synthetic Pyrethroid Pesticides	Mean conc. in fruit (µg/kg)	Mean conc. in Rind (µg/k)
Methamedophos	10.50±8.72	6.20±3.71	Lindane	6.58±4.07	7.60±5.00	Bifenthrin	2.25±1.43	2.45±1.04
Enthoprophos	5.95±5.08	6.50±8.04	Heptachlor	8.20±4.28	7.90±4.49	Lambda-Cy	2.70±1.79	3.50±1.29
Phorate	7.65±4.76	5.15±9.91	Aldrin	5.20±2.83	7.20±6.69	Permethrin	2.10±1.24	2.80±2.99
Diazinon	7.05±4.75	5.80±5.84	Endosulfan	8.71±8.84	9.13±9.95	Cyfluthrin	2.75±1.39	2.85±0.84
Dimethoate	29.60±31.11	18.55±16.41	DDE	4.20±2.31	4.80±4.79	Cypermethrin	2.10±1.19	3.10±1.44
Pirimipphos	8.35±7.69	6.90±5.33	Dieldrin	3.90±3.06	8.60±4.53	Fenvalerate	2.20±1.20	2.85±1.04
Chlorpyrifos	1662.75±1225.24	1842±1016.76	DDD	3.95±2.57	4.15±2.96	Deltamethrin	2.20±1.29	3.00±1.33
Fenitrothion	8.90±37.37	7.30±43.44	DDT	5.85±2.83	3.84±2.83			
Parathion	7.40±4.17	8.15±5.84	Methoxychlor	9.20±7.44	10.05±7.30			
Fonofos	6.25±4.28	5.70±7.03	Endrin	5.00±4.74	7.30±3.35			
Profenofos	8.05±7.22	7.80±68.97						
Malathion	13.65±11.58	9.56±11.94						
Chlorfenvinp	6.85±4.29	3.80±5.39						

OP = Organophosphorus Pesticide,   OC = Organochlorine Pesticide

Beside the juice or pulp for human consumption, watermelon rinds are used for Pickle, Jams, Preservers, Appetizers as well as for extraction of Pectin (Ahmed, 1961; Bawa & Bains, 1997; Hour, Ahmed , & Carter, 1980). Based upon this fact the rind was analysed to access the levels of pesticides and compare with the levels in the fruit flesh. Table 17 above shows the levels of the diverse pesticides detected in both fruit flesh the rind.

Analysis showed that the organophosphorus pesticides levels in the fruit flesh accumulated at higher levels than the rind (Table 17). The organochlorine and the synthetic pyrethroid pesticides on the other hand, were conversely higher in the rind. The high levels of the organophosphorus pesticides in the flesh may perhaps be due to a more rapid uptake rate of organophosphorus pesticides applied to the foliage, stem and the fruit and successive translocation into the watermelon fruit for storage. The low levels in the rind could be due to the fact that since the rind is in direct contact with the surroundings rainfall might perhaps have washed surface residue off the rind resulting in the low levels.

The work also assessed the extent of accumulation in a non-target crop (Okro) which was within approximately 80 meters of sampling site farm A. Concentrations of the various residues in the non-target crop were calculated in (µg/kg) and are presented in Table 18 below.

Table 18: Mean levels of OPs, OCs and synthetic pyrethroids in okro, a non-target species (µg/kg)

OP Pesticides	WHO	Mean±SD	OC Pesticides	WHO	Mean±SD	Syn Pyre	WHO	Mean±SD
Metha	4.0	6.05±2.57	Lindane	10.0	10.50±2.41	Bifenthrin	20.0	3.90±0.89
Entho	0.4	5.10±1.79	Heptachlor	0.10	4.00±1.87	Lambda-C	20.0	4.00±0.76
Phorate	0.7	19.40±12.64	Aldrin	0.10	4.90±1.71	Permethrin	50.0	4.80±0.75
Diazinon	2.0	6.10±1.73	Endosulfan	10.0	10.36±3.73	Cyfluthrin	20.0	5.80±0.80
Dimet	2.0	50.60±25.36	DDE	10.0	3.90±1.47	Cypermethrin	50.0	7.10±0.61
Pirim	30.0	9.80±3.86	Dieldrin	0.10	4.20±2.09	Fenvalerate	20.0	3.90±0.73
Chlorp	10.0	1321.10±340.22	DDD	10.0	3.10±0.90	Deltamethrin	10.0	5.80±0.88
Fenitr	10.0	36.50±21.19	DDT	6.0	6.20±3.58			
Parat	3.0	5.30±1.40	Methoxy	50.0	5.20±3.34			
Fono	10.0	3.90±1.60	Endrin	0.2	4.10±1.40			
Profen	10.0	13.80±2.72						
Malat	3.0	23.30±10.23						
Chlorfe	10.0	7.40±2.14						

Even though the okro crop was not directly treated with the pesticides, substantial levels were detected. Synthetic pyrethroids pesticides were found at moderately high levels in the okro with a mean of 5.04 µg/kg compared to 3.82 µg/kg in the target watermelon. The organophosphorus pesticides levels in the non-target crops (116.02 µg/kg) were approximately half the levels in the target crops (248.18 µg/kg). The organochlorine pesticides levels were somewhat low in the non- target crop. The differences in levels might have resulted from pesticide drift from the use of high pressure spraying equipment. This creates a greater percentage of minute particles which are predisposed to drift.

CHAPTER FIVE

SUMMARY, CONCLUSION AND RECOMMENDATIONS

Summary

Watermelons contain many Vitamins such as A, B6, C, and other nutrients critical for good health. Vitamin A boost the number of lymphocytes that help fight off infections by improving the immune system. Vitamin B6 facilitates the development of neurotransmitters such serotonin, dopamine and melatonin which help the body to deal with anxiety. Vitamin C helps thwart infections and helps delay the aging process, and the development of cataracts. It also toughens blood vessels, bones; help mend damaged tissues and promotes healing of wounds (United States Watermelon Promotion Board, 2003). Small amounts of potassium which can help alleviate muscle cramps, along with miniscule amount of calcium and iron are as well found in watermelons (United States Watermelon Promotion Board, 2003). Watermelons also contain considerable amounts of lycopene which lowers the risk for developing cancer, particularly prostate cancer in men and cervical cancer in women. The lycopene also help battle cardiovascular disease by prohibiting the hardening of the arteries (Watson, 2000).

Watermelons are vulnerable to several kinds of insect such Aphids, cabbage loopers, cucumber beetle, cutworms, thrips, leaf miners and spider mite are all known to infest watermelon crops. s infestations (Kishi, et al., 1995).

Diseases as Alternaria, leaf blight, anthracnose, bacterial rind necrosis, bacterial wilt, gummy stem blight, downy, mildew, cercospora leaf spot, fusarium

wilt, powdery mildew, pythium, southern blight and verticillium wilt also threaten watermelon crops (Kishi, et al., 1995). Watermelons can also be plagued by a variety of viruses including watermelon mosaic virus - 2, Tobacco ring spot virus, papaya ring spot virus, squash mosaic virus, cucumber mosaic virus and zucchini yellow mosaic virus (Kishi, et al., 1995).

Weed control is also essential in successful watermelon production, as annual and perennial grasses and broadleaf weeds emerge throughout the watermelon growing season (Kishi, et al., 1995). Attempts by farmers to control diseases, pest, and weed invasion and to maximize crop yield and profits have resulted in the use of pesticides which affect man, animals, and the environment.

Conclusion

The results of this investigation confirm that watermelon farmers in Ghana apply pesticides which are not recommended for use on the crop. The pesticides are catalogued by the Ghana Standards Board for use on cotton plants, cocoa plants and for treating cocoa sacks. This presents an obvious case of pesticides misapplication among watermelon farmers in the country. The quest to manage pests, and diseases, and to maximize crop yield have resulted in the application of pesticides on the watermelon. The work identified three categories of pesticides that are usually applied by the watermelon farmers viz the organophosphorus pesticides (OPs), the organochlorine pesticides (OCs), and the synthetic pyrethroid pesticides.

Considerable levels of each category of the pesticides were detected in the soil at a depth of 0 – 20 cm. The organophorus, the organochlorines and the

synthetic pyrethroid pesticides levels in the soil ranged from 2.0 - 4121.70 µg/kg 1.10 - 12.90 µg/kg and 1.10 - 8.20 µg/kg (Appendices A6, A7 and A8) respectively. This suggests that crops which take up nutrients at such depth stand the chance of being contaminated with the pesticides. The likelihood that populations of beneficial soil organisms that reside at such depth being affected by the pesticides could also not be ruled out.

The soil properties viz moisture 2.22 - 6.72 %, pH 5.34 - 7.53, cation exchange capacity 1.40 - 4.66 cmolckg^{-1}, clay 2.46 - 9.84 %, sand 52.35 - 84.94 % and silt 11.78 - 42.82 % and organic matter 4.20 − 24.60 % (Appendix A5) were found to have contributed significantly to the levels of some of the pesticides, for example Methamedophos, Diazinon, Pirimipphos, DDE, Heptachlor, Endosulfan, Permethrin and Cypermethrin in the soil. Even though some levels of pesticides were detected, the soil cannot be considered as contaminated because of lack of Ghanaian standards which suggest the permissible levels of pesticides in tropical soils. However, the presence of these pesticides point to a possible environmental contamination.

The results obtained from all sampling sites showed that watermelon farmers in the country apply almost the same types of pesticides on their crops. For example Methamedophos was present in all watermelons at concentrations ranging from 3.20 - 10.10 µg/kg for Nsadwir, 6.80 - 17.20 µg/kg for Ayensudo, 6.90 - 8.20 µg/kg for Sekondi − Takoradi, 15.20 - 17.80 µg/kg for Accra, 10.90 - 15.70 µg/kg for Kumasi, 14.20 - 16.50 µg/kg for Bolgatanga and 12.50 - 51.90 µg/kg for Cape Coast (Appendices A9, A15, A7 and A17).

A comparison of the residue levels in all watermelon fruits to the WHO/FAO recommended levels showed that the mean levels of most of the organophosphorus and the organochlorine pesticides were above the WHO/FAO permissible levels. Except for Chlorpyrifos, the lower limits of the levels under each category were in general below the WHO/FAO acceptable levels. The comparison also showed that even though significant levels of the OPs and the OCs were present in the fruits; the levels were lower than those permitted by countries such as the United States, Russia, Japan, Australia and Thailand. The levels were also below the European Union permissible levels.

The non − target crop okro (plants that were not directly sprayed but were about 80 meters away from the sprayed watermelon farms) was observed to suffer a similar fate as the target crop. The OPs and the OCs pesticides residues in the non − target specie were comparable to the corresponding levels in the target specie (Watermelon). For example the level of Methamedophos in the non − target specie ranged from 3.20 - 8.30 µg/kg (Appendix A12) as compared to 3.20 - 10.10 µg/kg (Appendix A9) for the same pesticide detected in the watermelon fruit from the locality (Nsadwir) where the non −target specie was grown.

The organochlorine pesticides (OCs), for example Lindane in the non-target crop ranged from 8.63 - 14.96 µg/kg (Appendix A13) as compared to 5.06 - 8.63 µg/kg (Appendix A10) in the target crop. The synthetic pyrethroid pesticide residues in both target and non − target crops were lower than the permissible levels set by the WHO/FAO, the United States, Russia, Australia, Japan and Thailand. The levels were also lower than those set by the European Union.

The findings verify that watermelon fruits grown in the country are contaminated with organophosphorus and the organochlorine pesticides. The findings also suggest that water bodies and other food crops, for example tuber crops in areas and communities where watermelons are produce on a large scale may have high levels of contaminations from these pesticides. The farmers who cultivate the crops are likely to have high levels of pesticides in their adipose tissues, blood and other body fluids. With the results above it is important that the watermelon farmer is educated on proper use and application of pesticides. If farmers are allowed to continue applying pesticides indiscriminately it is likely that consumers would not be exempted from the endocrine disrupting effects of some of these pesticides.

Recommendations

Based on the findings of this research, it is recommended that;

- Pesticide control act should be promulgated and implemented by government to prohibit the sale and distribution of pesticides without government approval.
- Plant protection centres should be established at places where there is heavy application of pesticides. The centres will be responsible for residue analysis, evaluate the safety of the pesticides and make necessary recommendations to government to either register or withdraw pesticides.

- Water bodies and other foodstuffs in areas of large scale watermelon productions should be analysed for residues to ascertain if levels could be injurious to life.

- Health surveillance should be carried out on those who are occupationally exposed to pesticides by analysing blood and other body fluids to know the extent of exposure.

- Government should develop regulations and implement licensing procedures to ensure that those involved in the sale of pesticides are able to provide buyers advice on risk reduction and effective use.

- Soil remediation strategies must be developed to remove those pesticides whose levels are high in the soils.

- Relevant authorities develop strategies to minimise exposure to pesticides in occupational settings and the broader environment.

- Further studies should be conducted in all areas in Ghana where watermelon is cultivated on a large scale, by taken fruits, soils and other foodstuffs, e.g. tuber crops to know the scope of contamination resulting from pesticide application.

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APPENDICES

APPENDIX A1

UNIVERSITY OF CAPE COAST

CHEMISTRY DEPARTMENT

QUESTIONNAIRE FOR WATERMELON FARMERS

Dear respondent,

The purpose of this questionnaire is to seek first-hand field information on the types of pesticides which are used on your farm for pest control. The information will form the basis of research into the effects of these pesticides on the soil, the crops and on humans. It will also help to educate you (the farmers) on choice of suitable pesticides for controlling these pests in order to maximize yield. Your response will be treated with utmost **confidentiality.** Therefore, please provide honest answers to the questions.

Instruction: Please complete or tick (√) as appropriate

1. Name :---
2. District:--
3. Sex: Male ( )   Female ( )
4. Age:--
5. Marital status: single ( )   Married ( )   Divorced ( ) Separated ( )

    Widowed ( )

6. Highest academic qualification:--

7. How many year have you been farming? -----------------------------------

8. Which of the following do you practice? Mixed farming ( ) Mono cropping ( )

9. What crops do you cultivate? Please list --

    -

10. How large is your farm? ---

    -

11. Do you use pesticides in farming? Yes ( )   No ( )

12. If yes, do you know the type?   Yes ( )   No( )

13. If yes, please list them

14. . Which of the listed pesticides do you often use? -----------------------------

    -

15. Which ones are you using currently? ---

16. Where do you obtain the pesticides? Licensed Agro-input shop ( )

    Open market ( )   A friend ( )   Extension agent ( )

17. When do you apply the pesticides? ---

18. Aside the purposes of pest control do the pesticides affect the crops in anyway?

    Yes ( )   No ( )

19. If yes please state the effect(s)

    ----

20. Do you know any effect(s) of the pesticides on humans? Yes ( )   No ( )

21. If yes please list the effect(s) --

APPENDIX A2

Quality control for organophosphorus pesticides at 0.1µg/Kg fortification

	Metha	Entho	Phorate	Diazin	Dimeth	Pirim	Chlor	Fenit	Parat	Fono	Prof	Mala	Chlorf
A	0.091	0.66	0.092	0.082	0.065	0.075	0.065	0.073	0.072	0.077	0.084	0.055	0.088
C	0.09	0.056	0.085	0.073	0.08	0.085	0.093	0.086	0.086	0.082	0.076	0.084	0.089
D	0.094	0.074	0.085	0.094	0.084	0.098	0.086	0.079	0.084	0.073	0.084	0.084	0.092
E	0.073	0.076	0.067	0.084	0.068	0.088	0.096	0.082	0.087	0.078	0.0066	0.073	0.083
F	0.076	0.087	0.082	0.092	0.082	0.076	0.071	0.09	0.073	0.085	0.076	0.083	0.079
G	0.072	0.063	0.07	0.076	0.081	0.065	0.066	0.078	0.071	0.081	0.086	0.077	0.073
H	0.085	0.089	0.092	0.085	0.061	0.094	0.092	0.089	0.091	0.072	0.082	0.066	0.084
I	0.082	0.095	0.096	0.086	0.081	0.098	0.069	0.078	0.076	0.082	0.074	0.081	0.076
J	0.085	0.092	0.076	0.087	0.076	0.094	0.081	0.088	0.086	0.076	0.083	0.087	0.075
K	0.071	0.061	0.083	0.081	0.097	0.084	0.088	0.069	0.084	0.097	0.075	0.068	0.073
Mean	**0.0835**	**0.0815**	**0.084**	**0.0845**	**0.0805**	**0.0865**	**0.0835**	**0.0805**	**0.084**	**0.0795**	**0.079**	**0.079**	**0.081**
%REC.	**83.5**	**81.5**	**84**	**84.5**	**80.5**	**86.5**	**83.5**	**80.5**	**84**	**79.5**	**79**	**79**	**81**
SD	**0.0084**	**0.1848**	**0.0095**	**0.0064**	**0.0105**	**0.0109**	**0.0119**	**0.0070**	**0.0072**	**0.00718**	**0.02362**	**0.01020**	**0.00698**

APPENDIX A3

Quality control for organochlorine pesticides at 0.1µg/Kg fortification

	Lindane	Heptac	Aldrin	Endosulfan	P'P'-DDE	Dieldrin	P'P'-DDD	P'P'-DDT	Methoxy	Endrin
A	0.084	0.072	0.081	0.076	0.094	0.07	0.09	0.063	0.096	0.087
C	0.086	0.095	0.085	0.079	0.082	0.067	0.087	0.086	0.061	0.081
D	0.079	0.063	0.075	0.076	0.086	0.092	0.088	0.09	0.075	0.086
E	0.093	0.086	0.077	0.08	0.082	0.075	0.091	0.077	0.086	0.082
F	0.061	0.073	0.09	0.064	0.081	0.077	0.09	0.091	0.077	0.081
G	0.08	0.075	0.086	0.062	0.095	0.086	0.065	0.073	0.084	0.071
H	0.07	0.085	0.071	0.083	0.064	0.076	0.084	0.081	0.073	0.069
I	0.089	0.079	0.074	0.061	0.071	0.094	0.078	0.083	0.073	0.072
J	0.081	0.085	0.072	0.097	0.085	0.089	0.084	0.081	0.094	0.074
K	0.083	0.077	0.088	0.078	0.082	0.069	0.091	0.083	0.079	0.08
Mean	**0.082**	**0.078**	**0.079**	**0.077**	**0.082**	**0.0765**	**0.0875**	**0.082**	**0.078**	**0.0805**
%RECOVERY	**82**	**78**	**79**	**77**	**82**	**76.5**	**87.5**	**82**	**78**	**80.5**
SD	**0.009252**	**0.009055**	**0.006999**	**0.010967**	**0.009331**	**0.009969**	**0.008066**	**0.008284**	**0.010528**	**0.00636**

APPENDIX A4

Quality control for synthetic pyrethroid pesticides at 0.1μg/Kg fortification

	Bifen	Lambda	Perm	Cyflu	Cyperm	Fenva	Deltam
A	0.098	0.093	0.084	0.081	0.088	0.091	0.096
C	0.084	0.079	0.09	0.086	0.082	0.079	0.081
D	0.073	0.086	0.079	0.081	0.078	0.084	0.077
E	0.086	0.071	0.074	0.092	0.081	0.09	0.089
F	0.071	0.082	0.079	0.098	0.083	0.095	0.091
G	0.083	0.095	0.082	0.083	0.081	0.073	0.084
H	0.074	0.087	0.091	0.072	0.082	0.096	0.093
I	0.092	0.091	0.076	0.084	0.075	0.081	0.072
J	0.09	0.081	0.089	0.072	0.09	0.073	0.08
K	0.07	0.091	0.093	0.097	0.086	0.08	0.091
Mean	**0.0835**	**0.0865**	**0.083**	**0.0835**	**0.082**	**0.0825**	**0.0865**
%RECOVERY	**83.5**	**86.5**	**83**	**83.5**	**82**	**82.5**	**86.5**
SD	**0.009723**	**0.007412**	**0.006734**	**0.009046**	**0.004477**	**0.008443**	**0.007792**

Some properties of Nsadwir soil

Farms	% Moisture	Soil pH	% OM	CEC	% Clay	% Sand	% Silt
A4	3.07	6.44	6.6	3.2	7.38	52.53	40.09
A3	2.94	6.32	8.2	3.74	9.84	52.35	37.81
A2	2.89	5.66	8.4	3.04	4.1	57.59	37.31
A1	6	6.29	10.8	4.66	3.28	53.9	42.82
Max	**6**	**6.44**	**10.8**	**4.66**	**9.84**	**57.59**	**42.82**
Min	**2.89**	**5.66**	**6.6**	**3.04**	**3.28**	**52.35**	**37.31**
Mean	**3.005**	**6.305**	**8.3**	**3.47**	**5.74**	**53.215**	**38.95**
SD	**1.518563**	**0.351034**	**1.732051**	**0.730844**	**3.031413**	**2.432233**	**2.518099**
B4	2.79	6.45	6.2	4.2	4.92	74.3	20.78
B3	4.41	6.07	7.8	3.4	6.56	63.22	30.22
B2	6.72	5.71	4.2	2.4	3.28	84.94	11.78
B1	5.28	5.34	10.4	4.5	2.48	63.49	34.05
Max	**6.72**	**6.45**	**10.4**	**4.5**	**6.56**	**84.94**	**34.05**
Min	**2.79**	**5.34**	**4.2**	**2.4**	**2.48**	**63.22**	**11.78**
Mean	**4.845**	**5.89**	**7**	**3.8**	**4.1**	**68.895**	**25.5**
SD	**1.64408**	**0.476401**	**2.619796**	**0.939415**	**1.811482**	**10.34717**	**9.986916**
C4	6.2	6.56	24.6	4.15	6.56	59.23	34.21
C3	2.24	5.97	9	2.3	5.74	75.99	18.27
C2	4.93	6.43	10.8	2.4	4.92	73.31	21.77
C1	2.48	6.7	5.4	1.4	4.1	69.24	26.66
Max	**6.2**	**6.7**	**24.6**	**4.15**	**6.56**	**75.99**	**34.21**
Min	**2.24**	**5.97**	**5.4**	**1.4**	**4.1**	**59.23**	**18.27**
Mean	**3.705**	**6.495**	**9.9**	**2.35**	**5.33**	**71.275**	**24.215**
SD	**1.924169**	**0.316491**	**8.405355**	**1.149909**	**1.058615**	**7.352176**	**6.906482**
D4	3.05	7.53	9.2	3.15	9.84	73.05	17.11
D3	2.22	6.97	7.2	2.5	4.1	73.02	22.88
D2	2.29	7.11	9.4	3.3	8.2	73.94	17.86
D1	2.77	7.07	6.8	2.3	5.74	70.4	23.86
Max	**3.05**	**7.53**	**9.4**	**3.3**	**9.84**	**73.94**	**23.86**
Min	**2.22**	**6.97**	**6.8**	**2.3**	**4.1**	**70.4**	**17.11**
Mean	**2.53**	**7.09**	**8.2**	**2.825**	**6.97**	**73.035**	**20.37**
SD	**0.396096**	**0.247117**	**1.340398**	**0.487126**	**2.549484**	**1.529104**	**3.434854**

APPENDIX A6

Organophosphorus pesticides (µg/Kg) in Nsadwir soils

	Metha	Entho	Phor	Diazin	Dimeth	Pirim	Chlor	Feni	Parat	Fono	Profe	Mala	Chlorf
A4	5.2	8.1	153.3	4.8	132.1	27.7	2942.4	4.3	19.1	2.8	41.4	6.9	4.8
A3	3.4	4.1	10.3	7.3	135.5	86.7	2484.9	6.7	13.3	4.1	43.8	11.4	5.2
A2	8.7	2.3	176.6	5.9	57.7	27.8	3405.6	11.4	35.4	4.8	42.6	8.5	6.4
A1	4.9	8.2	145.2	6.9	22.7	13.3	1545.1	7.4	15.8	3.2	56	9.3	6.3
B4	9.3	9.3	45.2	3.2	35.4	11.9	3273.4	3	28	6.7	99.2	16.8	7.4
B3	7.3	7.2	67.9	6.3	26.4	29.6	3088.5	2.9	11.2	3.2	85.6	7.3	4.1
B2	11.6	8.6	100.4	4.2	47	3.9	2194.9	3.8	11.9	9.1	68.9	6.8	5.7
B1	8.6	6.1	62.8	9.5	41	73.3	3533.7	2.3	13.4	7.4	106.3	13.9	3.2
C4	3.5	12.3	85.6	6.9	49.4	37.9	3778.8	8.2	20.5	7.9	124.2	17.9	11.9
C3	13.2	11.7	99.4	6.9	27.6	26.4	3358.2	4.9	16.7	4.5	124.9	10.3	12.9
C2	14.6	12.1	93.8	3.1	53.9	52.2	3790.3	6.4	19.2	8.9	76.7	15.7	10
C1	12.3	13.4	96	4.2	58.3	43.6	2222.4	4.2	4.7	6.8	10.5	35.7	14
D4	8.5	2.8	2	5.4	8.3	67.9	3528.2	7.5	28.5	9.4	170.1	2.5	5.5
D3	17	4.4	12.7	3.4	7.1	2.4	2286.7	6.5	6.5	5.6	20.2	7.4	6.9
D2	2.6	5.4	13.9	2.3	16.7	47.8	4121.7	19	7.4	12.7	116.7	7.5	8.2
D1	17.9	3.1	10.9	2.6	21.2	64.3	3886.7	8.5	30.8	8.7	60.7	8.9	3.8
Total	148.6	119.1	1176	82.9	740.3	616.7	49441.5	107	282.4	105.8	1247.8	186.8	116.3
Mean	8.65	7.65	76.75	5.1	38.2	33.75	3315.8	6.45	16.25	6.75	72.8	9.1	6.35
SD	4.8016	3.63335	55.2681	2.03771	37.9442	25.38309	740.8074	4.0929	9.08669	2.77893	43.3201	7.6347	3.2988
RSD	0.55510	0.47494	0.72010	0.39955	0.99330	0.75209	0.22341	0.6345	0.55918	0.41169	0.59505	0.83898	0.51950
Max	17.9	13.4	176.6	9.5	135.5	86.7	4121.7	19	35.4	12.7	170.1	35.7	14
Min	2.6	2.3	2	2.3	7.1	2.4	1545.1	2.3	4.7	2.8	10.5	2.5	3.2

APPENDIX A7

Organochlorine pesticides (µg/Kg) in Nsadwir soil

	Lindane	Hepta	Aldri	Endosul	P'P'-DDE	Dield	P'P'-DDD	P'P'-DDT	Methox	Endrin
A4	5.43	8.4	12.4	9.03	6.4	4.6	4.9	3.4	4.8	9.3
A3	4.76	7.8	3.9	10.86	6.7	4.9	5.4	2.6	8.9	5.8
A2	8.5	12.9	8.3	9.16	8.9	9.2	10	2.4	7.5	6.7
A1	5.43	4.8	6.9	6.63	8.1	5.8	4.5	3.1	9.8	4.1
B4	7.16	6	3.5	5.83	7.6	6.1	9.8	1.2	5.6	9.7
B3	4.73	5.3	2	7.66	3.4	2.2	4.3	9.9	7.4	4.1
B2	7.46	2.1	4.9	8.36	4.9	5	8.7	4.1	1.8	5.8
B1	4.13	3.6	7.2	4.83	6.4	3.9	5.1	8.5	9.4	6.6
C4	4.3	6.3	4.9	8.7	4.1	4.1	3.2	11.5	5.2	1.6
C3	4.36	5.1	4.3	9.06	4.2	3.2	3.1	10.2	4.2	1.2
C2	3.73	4.9	4.1	8.43	4.9	3.9	2.9	11.2	5.8	1.5
C1	4.06	5.2	4.5	8.06	4.2	3.9	3.2	12.2	5.6	2
D4	7.56	2.7	5.2	6.23	2.8	3.7	2.4	4.2	3.1	2.4
D3	4.86	4.1	5.3	9.4	3.8	3.9	2.4	7.8	4.2	1.2
D2	5.26	6.2	8.9	10.26	5.1	2.5	4.5	6.3	3.4	1.1
D1	4.03	5.1	6.6	7.83	4.2	2.7	3.6	10.2	4.7	2.8
Total	85.76	90.5	92.9	130.33	85.7	69.6	78	108.8	91.4	65.9
Mean	4.81	5.15	5.05	8.395	4.9	3.9	4.4	7.05	5.4	3.45
SD	1.486127	2.526122	2.524934	1.615718	1.788097	1.690365	2.485022	3.787171	2.319734	2.886802
RSD	0.308966	0.490509	0.499987	0.192462	0.364918	0.433427	0.564778	0.537187	0.42958	0.836754
Max	8.5	12.9	12.4	10.86	8.9	9.2	10	12.2	9.8	9.7
Min	3.73	2.1	2	4.83	2.8	2.2	2.4	1.2	1.8	1.1

APPENDIX A8

Synthetic pyrethroid pesticides (µg/Kg) in Nsadwir soils

	Bifen	Lambda	Perm	Cyflu	Cyperm	Fenva	Deltam
A4	2.1	2.7	3.2	3.9	1.1	2.4	3.1
A3	4	2.1	2.6	3.2	2.8	2.3	3.4
A2	3.4	2.3	1.2	2.8	2.2	2.2	3.6
A1	2.4	3.5	3.1	2.1	2.1	3.4	3.3
B4	2.3	3.4	2.3	1.9	2.8	7.7	2.3
B3	2.2	5.1	2.5	1.1	3.4	6.1	4.1
B2	3.4	3.2	2.7	1.7	2.4	7.6	3.4
B1	3.8	4.2	1.9	1.8	1.9	8.2	3.2
C4	5.2	3.3	2.8	3.6	2.2	8.1	3.7
C3	5.9	4.3	2.5	3.2	2.4	5.9	3.5
C2	6.7	4.8	1.2	4.8	3.7	7.3	3.9
C1	5.3	5.1	1.9	3.2	3.5	6.8	3.1
D4	2.9	5.3	3.2	3.9	3.9	3.9	3.5
D3	3.2	5.1	2.9	4.3	3.2	3.6	3.9
D2	4.2	3.5	4.1	4.1	3.8	3.8	3.8
D1	3.1	4.4	4.8	3.8	4.1	4.5	3.6
Total	**60.1**	**62.3**	**42.9**	**49.4**	**45.5**	**83.8**	**55.4**
Mean	**3.4**	**3.85**	**2.65**	**3.2**	**2.8**	**5.2**	**3.5**
SD	**1.386588**	**1.040813**	**0.934679**	**1.081896**	**0.853205**	**2.203898**	**0.425637**
RSD	**0.40782**	**0.270341**	**0.352709**	**0.338093**	**0.304716**	**0.423827**	**0.121611**
Max	**6.7**	**5.3**	**4.8**	**4.8**	**4.1**	**8.2**	**4.1**
Min	**2.1**	**2.1**	**1.2**	**1.1**	**1.1**	**2.2**	**2.3**

APPENDIX A9

Organophosphorus pesticides (µg/Kg) in watermelon flesh from Nsadwir

	Metha	Entho	Phorate	Diazin	Dimet	Pirim	Chlor	Fenit	Para	Fono	Prof	Mala	Chlorf
A4	10.1	4.6	8.9	2.8	22.1	6.8	1382.7	5.6	4.5	6.1	14.5	13.7	6.5
A3	3.2	3.6	5.1	3.8	4.7	6.5	3369.6	3.9	6.5	3.3	2.7	6.9	7.6
A2	4.8	4.2	7.6	2.5	11.7	8.5	4029.1	2.3	3.2	2.1	5.9	5.5	3.7
A1	5.06	5.1	4.4	3.3	22.6	7.3	4383.2	4.5	5.9	4.2	15	14.2	4.2
C4	4.3	2.3	9.8	8.5	63.2	13.4	2460.5	38.2	11.8	6.7	13.2	18.4	15.4
C3	7.8	3.1	16.3	5.6	21.7	4.9	1516.7	98.4	16.6	7.6	6.2	36.5	13.6
C2	4.9	4.7	13.7	7.8	62.1	13.7	1324.9	109.7	17.3	5.2	6.9	36.7	18.9
C1	7.3	3.4	16.8	5.1	41.3	5.8	1069.4	78.1	16.3	4.9	4.3	17.9	18.4
D4	3.3	3.7	18.5	5.9	22.9	31	2906.3	114	8.4	6.3	7.2	8.3	8.2
D3	4.1	7.5	8.9	12.6	18.5	22.4	3287.6	87.6	3.8	8.5	8.4	10.6	7.8
D2	7.2	9	14.2	12.9	17.5	15.9	3192.3	96.6	9.2	3.8	4.3	9.1	7.2
D1	5.3	2.5	4.1	10.8	10.4	8.9	3386.3	85.2	9.5	7.9	5.9	6.6	3.9
Total	67.36	53.7	128.3	81.6	318.7	145.1	32308.6	724.1	113	66.6	94.5	184.4	115.4
Mean	4.98	3.95	9.35	5.75	21.9	8.7	3049.3	81.65	8.8	5.65	6.55	12.15	7.7
SD	2.0679	1.9817	5.0647	3.7126	19.074	7.8578	1123.7	45.6414	5.07343	1.97737	4.13018	10.7842	5.51524
RSD	0.4152	0.5017	0.5416	0.6456	0.8709	0.9032	0.3685	0.55898	0.57652	0.34997	0.63056	0.887595	0.716265
Max	10.1	9	18.5	12.9	63.2	31	4383.2	114	17.3	8.5	15	36.7	18.9
Min	3.2	2.3	4.1	2.5	4.7	4.9	1069.4	2.3	3.2	2.1	2.7	5.5	3.7

APPENDIX A10

Organochlorine pesticides (µg/Kg) in watermelon flesh from Nsadwir

	Lindane	Hepta	Aldri	Endosul	P'P'-DDE	Dield	P'P'-DDD	P'P'-DDT	Methox	Endrin
A4	5.4	8.3	6.9	17.9	6.9	9	4.9	10.8	13.9	18.3
A3	5.13	8.4	5.2	8.93	4.9	9.8	5.4	5.5	11.2	5.6
A2	7.8	9.8	4.9	9.13	9.1	7.2	3.6	9.2	8.8	9.8
A1	6.7	4.2	3.2	8.33	4.9	6.7	6.5	6.5	9.1	19.3
C4	8.26	8.1	5.9	6.83	5.6	3.7	3.8	7.1	7.4	7.6
C3	8.33	9.5	5.3	6.83	3.9	2.1	3.3	6.2	6.8	8.1
C2	8.56	9.5	6.3	6.33	5.9	3.2	3.1	7.2	6.1	9.5
C1	8.56	9.1	7.2	6.23	4.8	3.9	5.1	5.2	5.4	7.7
D4	7.56	2.7	5.2	6.23	2.8	3.7	2.4	4.2	3.1	2.4
D3	5.06	4.1	5.3	9.4	3.8	3.9	2.4	7.8	4.2	1.2
D2	5.93	6.2	8.9	10.26	5.1	2.5	4.5	6.3	3.4	1.1
D1	5.36	5.1	6.6	7.83	4.2	2.7	3.6	10.2	4.7	2.8
Total	82.65	85	70.9	104.23	61.9	58.4	48.6	86.2	84.1	93.4
Mean	7.13	8.2	5.6	8.08	4.9	3.8	3.7	6.8	6.45	7.65
SD	1.438763	2.495754	1.424754	3.212268	1.637325	2.620664	1.251545	2.011708	3.272046	6.007394
RSD	0.20179	0.30436	0.25442	0.397558	0.334148	0.689648	0.338255	0.295839	0.507294	0.78528
Max	8.56	9.8	8.9	17.9	9.1	9.8	6.5	10.8	13.9	19.3
Min	5.06	2.7	3.2	6.23	2.8	2.1	2.4	4.2	3.1	1.1

APPENDIX A11

Synthetic pyrethroid pesticides (µg/Kg) in watermelon flesh from Nsadwir

	Bifen	Lambda	Perm	Cyflu	Cyperm	Fenva	Deltam
A4	3.1	3.4	2.5	2.8	2.9	2.5	2.9
A3	2.6	2.8	1.9	1.9	2.6	3.3	3.1
A2	3.8	2.7	2	2.2	3.8	3.3	2.4
A1	2.9	3.1	2.2	2.7	3.2	2.1	3.2
C4	3.1	5.2	4.6	5.4	3.2	3.1	4.7
C3	3.3	4.2	5.3	4.3	4.1	2.2	4.2
C2	4.8	5.4	5.2	3.1	4.3	2.6	3.2
C1	4.3	5.9	4.3	4.7	2.2	1.6	5.2
D4	4.9	5.9	2.8	5.2	3.8	4.7	3.5
D3	4.7	6.2	2.5	5.1	3.4	5.5	2.9
D2	3.9	6.1	2.9	4.9	3.7	4.8	3.5
D1	5.3	6.3	3.8	6.4	3.9	4.9	4.2
Total	46.7	57.2	40	48.7	41.1	40.6	43
Mean	3.85	5.3	2.85	4.5	3.55	3.2	3.35
SD	0.899958	1.428498	1.24632	1.45568	0.629755	1.287586	0.825539
RSD	0.23375	0.26952	0.43730	0.32348	0.177396	0.40237	0.24643
Max	5.3	6.3	5.3	6.4	4.3	5.5	5.2
Min	2.6	2.7	1.9	1.9	2.2	1.6	2.4

APPENDIX A12

Organophosphorus pesticides (µg/Kg) in okro

	Metha	Entho	Phor	Diazin	Dimet	Pirim	Chlor	Fenit	Para	Fono	Prof	Mala	Chlorf
B5	7.9	6.7	43.7	6.1	68	15.4	1143.8	36.4	5.3	2.8	16.1	20.6	9
B4	3,6	5.1	32.7	4.2	50.6	9.8	1271.6	36.5	4.2	6.9	18.9	40.8	5.9
B3	8.3	3.8	19.4	5.2	65.4	6.9	1321.1	73.2	4.5	3.2	12.4	23.3	7.4
B2	3.2	4.2	17.9	8.7	16.1	12.6	1426.7	26.6	7.6	4.1	12.7	37.7	4.6
B1	4.2	8.1	12.8	7.1	17.2	6.2	2016.8	68.9	6.4	3.9	13.8	18.8	9.8
Total	**23.6**	**27.9**	**126.5**	**31.3**	**217.3**	**50.9**	**7180**	**241.6**	**28**	**20.9**	**73.9**	**141.2**	**36.7**
Mean	**6.05**	**5.1**	**19.4**	**6.1**	**50.6**	**9.8**	**1321.1**	**36.5**	**5.3**	**3.9**	**13.8**	**23.3**	**7.4**
SD	**2.5781**	**1.7963**	**12.640**	**1.7358**	**25.360**	**3.86807**	**340.229**	**21.1902**	**1.4053**	**1.60841**	**2.72341**	**10.2363**	**2.1442**
RSD	**0.4261**	**0.3522**	**0.6515**	**0.2845**	**0.5012**	**0.39470**	**0.25753**	**0.58055**	**0.26516**	**0.41241**	**0.197349**	**0.439329**	**0.28977**
Max	**8.3**	**8.1**	**43.7**	**8.7**	**68**	**15.4**	**2016.8**	**73.2**	**7.6**	**6.9**	**18.9**	**40.8**	**9.8**
Min	**3.2**	**3.8**	**12.8**	**4.2**	**16.1**	**6.2**	**1143.8**	**26.6**	**4.2**	**2.8**	**12.4**	**18.8**	**4.6**

APPENDIX A13

Organochlorine pesticides (µg/Kg) in okro

	Lindane	Hepta	Aldri	Endosul	P'P'-DDE	Dield	P'P'-DDD	P'P'-DDT	Methox	Endrin
B5	8.63	2.1	4.3	15.83	2.2	1.2	3	13.2	12.4	4.9
B4	9.73	7.1	8.2	7.43	3.2	6.8	3.1	4.1	4.4	4.1
B3	11.33	4	5.9	10.36	3.9	5.6	3.2	5.2	7.6	2.5
B2	10.5	4.4	3.9	7.4	6.1	4.2	3	6.2	4.7	3.5
B1	14.96	3.1	4.9	13.53	4.6	4.1	5.1	6.2	5.2	6.2
Total	55.15	20.7	27.2	54.55	20	21.9	17.4	34.9	34.3	21.2
Mean	10.5	4	4.9	10.36	3.9	4.2	3.1	6.2	5.2	4.1
SD	2.411732	1.876966	1.716974	3.735097	1.471394	2.09571	0.909395	3.583574	3.343352	1.402854
RSD	0.229689	0.469241	0.350403	0.360531	0.37728	0.498979	0.293353	0.577996	0.642952	0.34216
Max	14.96	7.1	8.2	15.83	6.1	6.8	5.1	13.2	12.4	6.2
Min	8.63	2.1	3.9	7.4	2.2	1.2	3	4.1	4.4	2.5

APPENDIX A14

Synthetic pyrethroid pesticides (μg/Kg) in okro

	Bifen	Lambda	Perm	Cyflu	Cyperm	Fenva	Deltam
B5	3.9	5.6	4.8	5.1	7.6	3.2	5.8
B4	4.2	3.7	5.9	5.8	6.7	3.1	7.1
B3	5.5	4	4.1	4.1	7.1	4.8	6.7
B2	3.2	4.1	4.4	6.1	6.1	3.9	5.6
B1	3.5	3.9	5.5	5.8	7.5	4.4	4.9
Total	**20.3**	**21.3**	**24.7**	**26.9**	**35**	**19.4**	**30.1**
Mean	**3.9**	**4**	**4.8**	**5.8**	**7.1**	**3.9**	**5.8**
SD	**0.890505**	**0.763544**	**0.750333**	**0.804363**	**0.616441**	**0.739594**	**0.881476**
RSD	**0.228335**	**0.190886**	**0.156319**	**0.138683**	**0.086823**	**0.18964**	**0.151979**
Max	**5.5**	**5.6**	**5.9**	**6.1**	**7.6**	**4.8**	**7.1**
Min	**3.2**	**3.7**	**4.1**	**4.1**	**6.1**	**3.1**	**4.9**

APPENDIX A15

Organophosphorus pesticides (μg/Kg) in watermelon fruits from Ayensudo and Sekondi-Takoradi

	Metha	Entho	Phorate	Diazinon	Dimeth	Pirim	Chlor	Fenit	Parat	Fono	Prof	Mala	Chlorf
E5	15.8	6.9	12.6	3.7	51.7	3.8	2214.7	0.9	5.9	1.8	9.5	19.4	7.4
E4	13.9	3.5	8.2	9.5	14.7	1.5	3219.9	7.7	1.8	9.8	8.9	2.2	1.3
E3	9.9	4.4	5.7	7.5	40.6	1.5	2085.1	4	8.5	1.1	23.8	7.9	3.2
E2	17.2	9.4	2.8	8.9	33.7	3.3	1852.6	4.9	6.9	3.4	12.2	15	6.5
E1	14.5	3.1	3.6	6.3	13.2	6.4	1926.7	9.4	8.1	6.2	9.8	21.1	5.1
F5	16.8	9	2.9	7.1	23.7	8.9	2091.2	8.4	7.9	9	16.4	5.7	7.7
F4	12.9	6.9	7.7	10.5	17.2	3.5	1731.4	6.1	18.9	5.8	12	17.3	7.8
F3	12.2	3.9	2.9	2.8	15.8	9.4	1947.1	10.4	10.7	3.2	2.9	12.6	15.6
F2	6.8	8.2	15.2	7.6	29.1	1.5	1013.9	11.2	5.3	10.6	7.4	5.6	9.5
F1	13.8	5.2	6	7	38.2	2.2	1571.8	12.3	7.4	6.3	8.2	4.9	6.1
Total	**133.8**	**60.5**	**67.6**	**70.9**	**277.9**	**42**	**19654.4**	**75.3**	**81.4**	**57.2**	**111.1**	**111.7**	**70.2**
Mean	**13.85**	**6.05**	**5.85**	**7.3**	**26.4**	**3.4**	**1936.9**	**8.05**	**7.65**	**6**	**9.65**	**10.25**	**6.95**
SD	**3.17763**	**2.33963**	**4.28776**	**2.39603**	**13.0709**	**3.00555**	**557.340**	**3.56091**	**4.44577**	**3.33726**	**5.67478**	**6.76626**	**3.84557**
RSD	**0.22943**	**0.38671**	**0.73295**	**0.32822**	**0.49511**	**0.88398**	**0.28774**	**0.44235**	**0.58114**	**0.55621**	**0.58806**	**0.66012**	**0.55332**
Max	**17.2**	**9.4**	**15.2**	**10.5**	**51.7**	**9.4**	**3219.9**	**12.3**	**18.9**	**10.6**	**23.8**	**21.1**	**15.6**
Min	**6.8**	**3.1**	**2.8**	**2.8**	**13.2**	**1.5**	**1013.9**	**0.9**	**1.8**	**1.1**	**2.9**	**2.2**	**1.3**
G1	8.2	7	11.9	7.4	142.7	1.9	1217.8	4.8	9.3	12.9	14.6	9.4	3.8
G2	6.9	5	5.2	7	37.2	10.8	1594.1	22.1	6.4	5.6	3.2	8.3	7.4
Total	**15.1**	**12**	**17.1**	**14.4**	**179.9**	**12.7**	**2811.9**	**26.9**	**15.7**	**18.5**	**17.8**	**17.7**	**11.2**
Mean	**7.55**	**6**	**8.55**	**7.2**	**89.95**	**6.35**	**1405.95**	**13.45**	**7.85**	**9.25**	**8.9**	**8.85**	**5.6**
SD	**0.919**	**1.4142**	**4.7376**	**0.28284**	**74.599**	**6.2932**	**266.08**	**12.232**	**2.05061**	**5.16188**	**8.06101**	**0.77781**	**2.54558**
RSD	**0.1217**	**0.2357**	**0.5541**	**0.03928**	**0.8293**	**0.9910**	**0.1892**	**0.9095**	**0.26122**	**0.55804**	**0.90573**	**0.087889**	**0.454569**
Max	**8.2**	**7**	**11.9**	**7.4**	**142.7**	**10.8**	**1594.1**	**22.1**	**9.3**	**12.9**	**14.6**	**9.4**	**7.4**
Min	**6.9**	**5**	**5.2**	**7**	**37.2**	**1.9**	**1217.8**	**4.8**	**6.4**	**5.6**	**3.2**	**8.3**	**3.8**

APPENDIX A16

Organophosphorus pesticides (µg/Kg) in watermelon flesh from Accra, Kumasi and Bolgatanga

	Metha	Entho	Phorate	Diazinon	Dimeth	Pirim	Chlor	Fenit	Parat	Fono	Prof	Mala	Chlorf
H1	15.2	7.6	4.2	1.9	33.9	10.7	503.1	4.9	6.2	4.9	6.3	18.4	6.1
H2	17.8	8.9	2.9	5.8	134.9	7.9	125.3	6.2	4.2	6.1	7.1	41.9	6.3
Total	33	16.5	7.1	7.7	168.8	18.6	628.4	11.1	10.4	11	13.4	60.3	12.4
Mean	16.5	8.25	3.55	3.85	84.4	9.3	314.2	5.55	5.2	5.5	6.7	30.15	6.2
SD	1.83847	0.91923	0.91923	2.75771	71.4177	1.97989	267.144	0.91923	1.41421	0.84852	0.56568	16.6170	0.14142
RSD	0.11142	0.11142	0.25894	0.71629	0.84618	0.21289	0.85023	0.16562	0.27196	0.15427	0.08443	0.55114	0.02281
Max	17.8	8.9	4.2	5.8	134.9	10.7	503.1	6.2	6.2	6.1	7.1	41.9	6.3
Min	15.2	7.6	2.9	1.9	33.9	7.9	125.3	4.9	4.2	4.9	6.3	18.4	6.1
I1	10.9	14.1	4.7	4.2	70.9	8.2	296.4	8.1	4.9	10.4	12.8	29.4	5.3
I2	15.7	29.5	12.5	22.6	52.3	30	118.1	14.3	10.8	24.7	40.3	50.8	5.8
Total	26.6	43.6	17.2	26.8	123.2	38.2	414.5	22.4	15.7	35.1	53.1	80.2	11.1
Mean	13.3	21.8	8.6	13.4	61.6	19.1	207.25	11.2	7.85	17.55	26.55	40.1	5.55
SD	3.39411	10.8894	5.51543	13.0107	13.1521	15.4149	126.077	4.38406	4.17193	10.1116	19.4454	15.1320	0.35355
RSD	0.25519	0.49951	0.64132	0.97095	0.21351	0.80706	0.60833	0.39143	0.53145	0.57616	0.73240	0.37735	0.06370
Max	15.7	29.5	12.5	22.6	70.9	30	296.4	14.3	10.8	24.7	40.3	50.8	5.8
Min	10.9	14.1	4.7	4.2	52.3	8.2	118.1	8.1	4.9	10.4	12.8	29.4	5.3
J1	16.5	6.7	11.4	4.9	29.8	17.9	231.2	5.2	6.1	7.4	4.3	17.9	12.2
J2	14.2	9.2	2.3	8.7	45.3	14.9	268.1	5.7	5.8	8	12.7	13.6	8.6
Total	30.7	15.9	13.7	13.6	75.1	32.8	499.3	10.9	11.9	15.4	17	31.5	20.8
Mean	15.35	7.95	6.85	6.8	37.55	16.4	249.65	5.45	5.95	7.7	8.5	15.75	10.4
SD	1.6263	1.7677	6.4346	2.68700	10.960	2.1213	26.092	0.3535	0.21213	0.42426	5.93969	3.04055	2.54558
RSD	0.1059	0.2223	0.9393	0.39514	0.2918	0.1293	0.1045	0.0648	0.03565	0.05509	0.69878	0.193051	0.244768
Max	16.5	9.2	11.4	8.7	45.3	17.9	268.1	5.7	6.1	8	12.7	17.9	12.2
Min	14.2	6.7	2.3	4.9	29.8	14.9	231.2	5.2	5.8	7.4	4.3	13.6	8.6

APPENDIX A17

Organophosphorus pesticides (µg/Kg) in watermelon flesh from Cape Coast

	Metha	Entho	Phora	Diazin	Dimeth	Pirim	Chlor	Fenit	Parat	Fono	Prof	Mala	Chlorf
K1	12.5	10.6	7.3	11.8	40.2	13	242.9	12.5	11.2	9.7	7.9	17.3	5.6
K2	51.9	13.5	12.5	21.4	29.4	20.7	109.3	17.1	7.4	10.2	6.3	8.9	6.3
Total	**64.4**	**24.1**	**19.8**	**33.2**	**69.6**	**33.7**	**352.2**	**29.6**	**18.6**	**19.9**	**14.2**	**26.2**	**11.9**
Mean	**32.2**	**12.05**	**9.9**	**16.6**	**34.8**	**16.85**	**176.1**	**14.8**	**9.3**	**9.95**	**7.1**	**13.1**	**5.95**
SD	**27.860**	**2.0506**	**3.6769**	**6.7882**	**7.6367**	**5.4447**	**94.469**	**3.25269**	**2.68700**	**0.35355**	**1.13137**	**5.93969**	**0.49497**
RSD	**0.8652**	**0.1701**	**0.3714**	**0.4089**	**0.2194**	**0.3231**	**0.5364**	**0.21977**	**0.28892**	**0.03553**	**0.15934**	**0.453412**	**0.083189**
Max	**51.9**	**13.5**	**12.5**	**21.4**	**40.2**	**20.7**	**242.9**	**17.1**	**11.2**	**10.2**	**7.9**	**17.3**	**6.3**
Min	**12.5**	**10.6**	**7.3**	**11.8**	**29.4**	**13**	**109.3**	**12.5**	**7.4**	**9.7**	**6.3**	**8.9**	**5.6**

Organochlorine pesticides (µg/Kg) in watermelon flesh from Ayensudo and Sekondi-Takoradi

	Lindane	Hepta	Aldri	Endosul	P'P'-DDE	Dield	P'P'-DDD	P'P'-DDT	Methox	Endrin
E5	6.46	5.3	7.4	7.03	2.9	13.6	6.5	3.6	8.6	5.4
E4	6.9	3.1	6.1	7.6	4.2	3.1	2.2	6.4	11.1	7.4
E3	6.86	7.4	2.3	12.06	5.6	1.1	4.1	0.2	27.2	1.6
E2	10.73	11.6	3.7	10.4	1.9	9.9	1.4	7.8	23.9	3.9
E1	5.93	23.4	14.6	8.56	13	9.3	1.3	5.3	8.4	5.3
F5	3.6	1.9	2.1	3.1	1.9	1.4	8.2	3.1	12.3	5.6
F4	8.53	2.6	3.9	11.53	6.9	4.2	6.2	9.1	34.3	1.5
F3	5.1	11.7	3.4	9.26	1.8	7.2	9.9	1.9	17.7	1.3
F2	13.9	11.3	3.6	16.16	2.9	3.1	5.6	4.1	9.7	6.3
F1	7.76	11.3	4	11.76	5.9	4.6	5.3	8.1	3.1	4.5
Total	75.77	89.6	51.1	97.46	47	57.5	50.7	49.6	156.3	42.8
Mean	6.88	9.35	3.8	9.83	3.55	4.4	5.45	4.7	11.7	4.9
SD	2.939165	6.449841	3.698784	3.526954	3.444803	4.107784	2.860478	2.89298	9.891528	2.156025
RSD	0.427204	0.689823	0.973364	0.358795	0.970367	0.933587	0.524858	0.615528	0.84543	0.440005
Max	13.9	23.4	14.6	16.16	13	13.6	9.9	9.1	34.3	7.4
Min	3.6	1.9	2.1	3.1	1.8	1.1	1.3	0.2	3.1	1.3
G1	4.14	4.2	1.2	8.03	3	7.2	1.2	3.5	9.1	0.7
G2	5.4	13.2	1.1	11.46	4	2.5	5.6	2.3	2.1	3.6
Total	9.54	17.4	2.3	19.49	7	9.7	6.8	5.8	11.2	4.3
Mean	4.77	8.7	1.15	9.745	3.5	4.85	3.4	2.9	5.6	2.15
SD	0.890955	6.363961	0.070711	2.425376	0.707107	3.323402	3.11127	0.848528	4.949747	2.05061
RSD	0.186783	0.73149	0.061488	0.248884	0.202031	0.685237	0.915079	0.292596	0.883883	0.953772
Max	5.4	13.2	1.2	11.46	4	7.2	5.6	3.5	9.1	3.6
Min	4.14	4.2	1.1	8.03	3	2.5	1.2	2.3	2.1	0.7

APPENDIX A19

Organochlorine pesticides (µg/Kg) in watermelon flesh from Accra, Kumasi and Bolgatanga

	Lindane	Hepta	Aldri	Endosul	P'P'-DDE	Dield	P'P'-DDD	P'P'-DDT	Methox	Endrin
H1	4.23	7.2	5.3	14.03	3.9	4.3	1.1	1.3	21.6	2.1
H2	5.6	11.4	8.9	11.43	4.3	3.2	4.2	5.2	9.3	1.9
Total	9.83	18.6	14.2	25.46	8.2	7.5	5.3	6.5	30.9	4
Mean	4.915	9.3	7.1	12.73	4.1	3.75	2.65	3.25	15.45	2
SD	0.968736	2.969848	2.545584	1.838478	0.282843	0.777817	2.192031	2.757716	8.697413	0.141421
RSD	0.197098	0.319339	0.358533	0.144421	0.068986	0.207418	0.827182	0.848528	0.562939	0.070711
Max	5.6	11.4	8.9	14.03	4.3	4.3	4.2	5.2	21.6	2.1
Min	4.23	7.2	5.3	11.43	3.9	3.2	1.1	1.3	9.3	1.9
I1	4.26	11.9	2.4	8.53	2.9	2.8	12.3	9.3	13.8	4.2
I2	5.43	4.6	7.4	13.53	3.2	9.4	2.3	4.2	3.3	12.7
Total	9.69	16.5	9.8	22.06	6.1	12.2	14.6	13.5	17.1	16.9
Mean	4.845	8.25	4.9	11.03	3.05	6.1	7.3	6.75	8.55	8.45
SD	0.827315	5.16188	3.535534	3.535534	0.212132	4.666905	7.071068	3.606245	7.424621	6.010408
RSD	0.170756	0.625682	0.721538	0.320538	0.069551	0.765066	0.968639	0.534258	0.868377	0.711291
Max	5.43	11.9	7.4	13.53	3.2	9.4	12.3	9.3	13.8	12.7
Min	4.26	4.6	2.4	8.53	2.9	2.8	2.3	4.2	3.3	4.2
J1	7.66	3.9	5.2	19.9	7.1	1.6	1.6	4.6	12.4	13.9
J2	8.43	9.4	9.1	13.2	2.3	3.9	3.2	11.1	14.3	4.7
Total	16.09	13.3	14.3	33.1	9.4	5.5	4.8	15.7	26.7	18.6
Mean	8.045	6.65	7.15	16.55	4.7	2.75	2.4	7.85	13.35	9.3
SD	0.544472	3.889087	2.757716	4.737615	3.394113	1.626346	1.131371	4.596194	1.343503	6.505382
RSD	0.067678	0.584825	0.385695	0.286261	0.722152	0.591398	0.471405	0.585502	0.100637	0.699503
Max	8.43	9.4	9.1	19.9	7.1	3.9	3.2	11.1	14.3	13.9
Min	7.66	3.9	5.2	13.2	2.3	1.6	1.6	4.6	12.4	4.7

APPENDIX A20

Organochlorine pesticides (µg/Kg) in watermelon flesh from Cape Coast

	Lindane	Hepta	Aldri	Endosul	P'P'-DDE	Dield	P'P'-DDD	P'P'-DDT	Methox	Endrin
K1	9.66	5.9	2.5	22.86	4.2	2.5	1.2	1.8	16.7	3.1
K2	10.16	9.5	1.2	34.5	7.2	5.1	4.1	6.5	12.5	8.4
Total	19.82	15.4	3.7	57.36	11.4	7.6	5.3	8.3	29.2	11.5
Mean	9.91	7.7	1.85	28.68	5.7	3.8	2.65	4.15	14.6	5.75
SD	0.353553	2.545584	0.919239	8.230723	2.12132	1.838478	2.05061	3.323402	2.969848	3.747666
RSD	0.035676	0.330595	0.496886	0.286985	0.372161	0.48381	0.773815	0.80082	0.203414	0.651768
Max	10.16	9.5	2.5	34.5	7.2	5.1	4.1	6.5	16.7	8.4
Min	9.66	5.9	1.2	22.86	4.2	2.5	1.2	1.8	12.5	3.1

APPENDIX A21

Synthetic pyrethroid pesticides (µg/Kg) in watermelons fruits from Ayensudo and Sekondi - Takoradi

	Bifen	Lambda	Permet	Cyflu	Cyperm	Fenva	Deltam
E5	0.1	1.1	0.2	3.3	2.9	3.1	0.9
E4	1	0.5	2.1	4.1	0.7	1.2	3.1
E3	3.4	1.2	2.1	2.3	0.2	0.4	0.1
E2	0.8	2.2	1.2	2.9	2.1	1.7	2.3
E1	1.3	1.9	1.1	1.8	1.4	1.9	0.8
F5	0.9	1.1	2.1	1.2	0.6	1.2	1.8
F4	0.6	1.3	1.9	1.5	1.2	2.2	1
F3	1.9	1.6	2.1	2.4	1.9	1.2	2.8
F2	2.1	2.7	3.1	3.4	1.8	2.9	0.9
F1	0.9	3.5	1.1	1.8	3.1	1.7	1.8
Total	13	17.1	17	24.7	15.9	17.5	15.5
Mean	0.95	1.45	2	2.35	1.6	1.7	1.4
SD	0.942809	0.888757	0.806915	0.935771	0.959687	0.82361	0.969822
RSD	0.992431	0.612936	0.403457	0.3982	0.599805	0.484476	0.69273
Max	3.4	3.5	3.1	4.1	3.1	3.1	3.1
Min	0.1	0.5	0.2	1.2	0.2	0.4	0.1
G1	1.8	1.5	2	1.2	1.3	1.9	1.9
G2	1.2	2.9	2.1	1.3	1.1	2.4	0.9
Total	3	4.4	4.1	2.5	2.4	4.3	2.8
Mean	1.5	2.2	2.05	1.25	1.2	2.15	1.4
SD	0.424264	0.989949	0.070711	0.070711	0.141421	0.353553	0.707107
RSD	0.282843	0.449977	0.034493	0.056569	0.117851	0.164443	0.505076
Max	1.8	2.9	2.1	1.3	1.3	2.4	1.9
Min	1.2	1.5	2	1.2	1.1	1.9	0.9

APPENDIX A22

Synthetic pyrethroid pesticides (µg/Kg) in watermelons fruits from Accra and Kumasi

	Bifen	Lambda	Permet	Cyflu	Cyperm	Fenva	Deltam
H1	1.9	3.7	2.4	1.3	2.1	2.2	2.6
H2	2.4	1.1	2.3	2.8	0.5	1.4	1.6
Total	**4.3**	**4.8**	**4.7**	**4.1**	**2.6**	**3.6**	**4.2**
Mean	**2.15**	**2.4**	**2.35**	**2.05**	**1.3**	**1.8**	**2.1**
SD	**0.353553**	**1.838478**	**0.070711**	**1.06066**	**1.131371**	**0.565685**	**0.707107**
RSD	**0.164443**	**0.766032**	**0.03009**	**0.517395**	**0.870285**	**0.31427**	**0.336718**
Max	**2.4**	**3.7**	**2.4**	**2.8**	**2.1**	**2.2**	**2.6**
Min	**1.9**	**1.1**	**2.3**	**1.3**	**0.5**	**1.4**	**1.6**
I1	2.6	1.7	4.7	2.2	0.6	1.7	1.3
I2	0.9	3.1	1.9	2.9	1.1	1.4	2.1
Total	**3.5**	**4.8**	**6.6**	**5.1**	**1.7**	**3.1**	**3.4**
Mean	**1.75**	**2.4**	**3.3**	**2.55**	**0.85**	**1.55**	**1.7**
SD	**1.202082**	**0.989949**	**1.979899**	**0.494975**	**0.353553**	**0.212132**	**0.565685**
RSD	**0.686904**	**0.412479**	**0.599969**	**0.194108**	**0.415945**	**0.136859**	**0.332756**
Max	**2.6**	**3.1**	**4.7**	**2.9**	**1.1**	**1.7**	**2.1**
Min	**0.9**	**1.7**	**1.9**	**2.2**	**0.6**	**1.4**	**1.3**

APPENDIX A23

Synthetic pyrethroid pesticides (µg/Kg) in watermelons fruits from Bolgatanga and Cape Coast

	Bifen	Lambda	Permet	Cyflu	Cyperm	Fenva	Deltam
J1	2.9	1.6	1.1	2.1	1.8	2.4	1.1
J2	1.9	1.5	3.6	3.1	1.6	3.7	0.8
Total	**4.8**	**3.1**	**4.7**	**5.2**	**3.4**	**6.1**	**1.9**
Mean	**2.4**	**1.55**	**2.35**	**2.6**	**1.7**	**3.05**	**0.95**
SD	**0.707107**	**0.070711**	**1.767767**	**0.707107**	**0.141421**	**0.919239**	**0.212132**
RSD	**0.294628**	**0.04562**	**0.752241**	**0.271964**	**0.083189**	**0.30139**	**0.223297**
Max	**2.9**	**1.6**	**3.6**	**3.1**	**1.8**	**3.7**	**1.1**
Min	**1.9**	**1.5**	**1.1**	**2.1**	**1.6**	**2.4**	**0.8**
K1	0.9	1.9	1.3	1.9	2.2	2.4	0.8
K2	1.5	1.4	2.1	1.8	1.2	1.6	1.2
Total	**2.4**	**3.3**	**3.4**	**3.7**	**3.4**	**4**	**2**
Mean	**1.2**	**1.65**	**1.7**	**1.85**	**1.7**	**2**	**1**
SD	**0.424264**	**0.353553**	**0.565685**	**0.070711**	**0.707107**	**0.565685**	**0.282843**
RSD	**0.353553**	**0.214275**	**0.332756**	**0.038222**	**0.415945**	**0.282843**	**0.282843**
Max	**1.5**	**1.9**	**2.1**	**1.9**	**2.2**	**2.4**	**1.2**
Min	**0.9**	**1.4**	**1.3**	**1.8**	**1.2**	**1.6**	**0.8**

APPENDIX A24

Organophosphorus pesticides (µg/Kg) in watermelon rind from Nsadwir

	Metha	Entho	Phorate	Diazin	Dimeth	Pirim	Chlor	Feni	Parat	Fono	Profen	Mala	Chlorf
D4	6.8	2.5	15.2	5.8	35.2	4.8	2268.9	37.1	12.9	7	85.3	38.5	12.9
A3	7.2	5.9	19.8	9.8	60.4	6.5	932.8	89.4	6.2	6.7	90.9	31.8	4.2
A2	6.5	7.6	12.5	7.5	58.7	5.6	2129.7	65.4	6.2	8.5	59.5	44.9	4.3
A1	9.5	18.6	14.7	5.2	26.4	3.5	962.1	28.6	8.2	2.8	79.7	20.7	5.4
C4	16.8	23.3	32	14.1	19.1	13.3	1207.4	134.5	12	29	148.9	4.2	21.9
C3	16.76	22.1	8.3	16.8	18.9	10.3	1425.8	129.2	9.8	31	210.1	10.03	13.94
C2	8.54	16.98	25.9	26.9	12.1	7.2	1921.1	112.5	34	6.2	291.8	7.8	9.6
C1	4.19	32.8	39.1	12	4.5	13.6	2113.7	142.8	14.03	2.4	142.4	6	12.6
D4	6.8	22.5	5.2	19.8	35.2	4.8	2268.9	17.1	12.9	10	45.3	38.5	12.9
D3	8.2	2.1	23.2	2.5	15.5	7.3	3285.8	16.2	10.9	10.3	61.4	33.6	17.4
D2	9.4	2.7	27.4	1.4	12.9	5.2	2142.2	19.5	3.8	12	46.3	8.7	8.5
D1	5.3	13.2	17.9	11.9	19.3	6.5	2142.7	27.92	8	14.3	67.4	13.8	9.6
Total	105.99	170.28	241.2	133.7	318.2	88.6	22801.1	820.22	138.93	140.2	1329	258.53	133.24
Mean	7.7	15.09	18.85	10.85	19.2	6.5	2121.7	51.25	10.35	9.25	82.5	17.25	11.1
SD	4.02242	10.073	9.89150	7.47023	17.835	3.30477	669.378	50.4039	7.73779	9.22820	75.3725	14.9562	5.31486
RSD	0.5223	0.6675	0.52474	0.68850	0.9289	0.50842	0.31549	0.98349	0.74761	0.99764	0.91360	0.86703	0.47881
Max	16.8	32.8	39.1	26.9	60.4	13.6	3285.8	142.8	34	31	291.8	44.9	21.9
Min	4.19	2.1	5.2	1.4	4.5	3.5	932.8	16.2	3.8	2.4	45.3	4.2	4.2

Organochlorine pesticides (µg/Kg) in watermelon rind from Nsadwir

	Lindane	Heptachlor	Aldrin	Endosul	P'P'-DDE	Dieldrin	P'P'-DDD	P'P'-DDT	Methoxychlor	Endrin
A4	5.9	7.1	13.2	4.53	5.8	8.9	5.2	3.6	7.1	8.4
A3	9.9	8.1	5.6	5.76	4.9	14.3	9.4	7.9	7.1	3.8
A2	10.13	6.3	8.3	6.4	15.3	9	7.9	8.6	3.2	2.6
A1	8.33	5.9	5.6	7.43	5.3	9.6	4.2	4.9	7.9	9.5
C4	4.63	8.2	5.3	7.63	3.2	7.3	4.5	3.7	10.7	8.3
C3	5.3	7.9	4.8	8.63	3.9	8.5	3.5	3.9	10.2	7.6
C2	5	7.9	4.1	7.36	3.1	9.1	4	2.6	11.4	6.5
C1	3.36	6.2	5.2	9.06	2.2	8.7	4.9	1.9	9.9	8.4
D4	9.6	3.6	4.2	4.8	4.8	3.2	4.1	6.3	14.3	5.9
D3	6.33	4.8	6.1	2.8	2.4	12.8	9.9	3.2	15.9	2.9
D2	6.9	9.5	16.9	3.8	2.2	7	3.6	4.9	12.3	6.2
D1	3.9	12.4	20.4	3.4	4.5	15.9	12.7	10.4	3.1	3.2
Total	79.28	87.9	99.7	71.6	57.6	114.3	73.9	61.9	113.1	73.3
Mean	6.115	7.5	5.6	6.08	4.2	8.95	4.7	4.4	10.05	6.35
SD	2.372468	2.279603	5.469828	2.099287	3.530903	3.412311	3.044058	2.618278	3.956611	2.442599
RSD	0.387975	0.303947	0.976755	0.345277	0.840691	0.381264	0.647672	0.595063	0.393693	0.384661
Max	10.13	12.4	20.4	9.06	15.3	15.9	12.7	10.4	15.9	9.5
Min	3.36	3.6	4.1	2.8	2.2	3.2	3.5	1.9	3.1	2.6

APPENDIX A26

Synthetic pyrethroid pesticides (µg/Kg) in watermelon rind from Nsadwir

	Bifen	Lambda	Perm	Cyflu	Cyperm	Fenva	Deltam
A4	2.2	3.1	2.4	3.2	2.6	3.6	3.2
A3	2.1	4.1	3.2	2.8	3.1	2.9	3.1
A2	2.1	3.8	2.6	3.8	3.2	2.8	3.3
A1	2.5	2.2	3.2	2.9	2.9	2.2	2.9
C4	4.2	4.5	12.4	2.2	5.3	3.5	6.2
C3	5.3	5.2	11.9	4.2	6.4	4.2	4.5
C2	4.1	6.4	10.9	3.2	4.9	3.6	4.9
C1	3.2	4.2	10.3	3.9	4.1	3.4	5.3
D4	2.9	4.7	3.8	3.5	4.1	4.6	4.7
D3	2.8	4.8	3.9	3.9	4.9	5.1	5.3
D2	3.1	4.9	2.9	3.6	5.2	3.7	4.2
D1	3.3	4.1	4.3	3.4	6.4	3.9	3.9
Total	**37.8**	**52**	**71.8**	**40.6**	**53.1**	**43.5**	**51.5**
Mean	**3**	**4.35**	**3.85**	**3.45**	**4.5**	**3.6**	**4.35**
SD	**0.970941**	**1.053422**	**4.047633**	**0.562193**	**1.305321**	**0.788699**	**1.042251**
RSD	**0.323647**	**0.242166**	**1.051333**	**0.162954**	**0.290071**	**0.219083**	**0.239598**
Max	**5.3**	**6.4**	**12.4**	**4.2**	**6.4**	**5.1**	**6.2**
Min	**2.1**	**2.2**	**2.4**	**2.2**	**2.6**	**2.2**	**2.9**

Organophosphorus pesticides (µg/Kg) in watermelon rind from Ayensudo and Sekondi-Takoradi

	Meth	Enth	Phorate	Diazinon	Dimeth	Pirim	Chlor	Fen	Parat	Fono	Profen	Mala	Chlorf
E5	3.5	2.8	11.1	3.4	14.6	10.7	1768.5	2.2	7.8	2.1	7.7	12.3	0.6
E4	1.2	0.5	6.8	8.1	33.3	1.4	231.8	1.3	1.7	0.4	4.1	4.9	0.1
E3	6.4	0.6	2.1	1.5	12.9	3.6	1254.8	4.9	9.1	8.7	5.9	0.9	3.8
E2	1,5	3.2	2.9	4.9	19.6	4.5	2318.9	6.9	8.4	7.2	2.9	9.1	5.4
E1	7.4	5.5	12.1	2.8	18.2	1.4	2009.3	1.8	6.4	5.2	8.4	10.6	3.8
F5	4.4	0.9	2.9	7.4	7.9	4.7	2344.6	7.7	4.8	0.9	6.3	12.1	6.4
F4	1.5	0.5	2.9	2.8	6.8	5.9	613.9	1.3	8.1	0.7	7.9	13.3	0.8
F3	6.2	3.4	4.5	1.9	14.2	3.7	2212.1	7.9	9.2	5.4	3.7	7.3	1.9
F2	4.4	7.9	2.5	5.2	15.4	14.3	1235.8	6.5	2.1	6.8	8.1	1.9	0.1
F1	1.4	5.3	3.3	1.9	6.8	9.2	1917.3	1.2	8.4	1.1	0.9	7.3	5.4
Total	36.4	30.6	51.1	39.9	149.7	59.4	15907	41.7	66	38.5	55.9	79.7	28.3
Mean	4.4	3	3.1	3.1	14.4	4.6	1842.9	3.55	7.95	3.65	6.1	8.2	2.85
SD	2.33351	2.54785	3.6783	2.33164	7.86370	4.19661	733.167	2.87867	2.80317	3.14156	2.5735	4.34052	2.41984
RSD	0.53034	0.84928	1.18654	0.75214	0.54609	0.91230	0.39783	0.81089	0.3526	0.86070	0.42189	0.52933	0.84907
Max	7.4	7.9	12.1	8.1	33.3	14.3	2344.6	7.9	9.2	8.7	8.4	13.3	6.4
Min	1.2	0.5	2.1	1.5	6.8	1.4	231.8	1.2	1.7	0.4	0.9	0.9	0.1
G1	6.6	6.6	2.2	1.6	68.4	4.5	671.6	1.6	6.1	2.4	1.1	3.2	2.9
G2	2.8	2.9	4.2	2.8	16.5	12.8	215.7	4.9	3.8	0.5	0.9	15.7	2.2
Total	9.4	9.5	6.4	4.4	84.9	17.3	887.3	6.5	9.9	2.9	2	18.9	5.1
Mean	4.7	4.75	3.2	2.2	42.45	8.65	443.65	3.25	4.95	1.45	1	9.45	2.55
SD	2.68700	2.61629	1.41421	0.84852	36.6988	5.86898	322.37	2.33345	1.62634	1.34350	0.14142	8.83883	0.49497
RSD	0.5717	0.5507	0.4419	0.38569	0.8645	0.6784	0.7266	0.7179	0.32855	0.92655	0.14142	0.935326	0.194108
Max	6.6	6.6	4.2	2.8	68.4	12.8	671.6	4.9	6.1	2.4	1.1	15.7	2.9
Min	2.8	2.9	2.2	1.6	16.5	4.5	215.7	1.6	3.8	0.5	0.9	3.2	2.2

APPENDIX A28

Organophosphorus pesticides (µg/Kg) in watermelon rind from Accra and Kumasi

	Meth	Enth	Phorate	Diazinon	Dimeth	Pirim	Chlor	Fen	Parat	Fono	Profen	Mala	Chlorf
H1	4.5	8.2	3.1	5.8	57.9	13.2	5015	10.3	4.9	1	1.3	7.4	3.8
H2	3.6	11.9	5.1	6.2	13.2	7.2	1132.7	11.1	6.2	5.1	1.9	18.1	3.1
Total	8.1	20.1	8.2	12	71.1	20.4	6147.7	21.4	11.1	6.1	3.2	25.5	6.9
Mean	4.05	10.05	4.1	6	35.55	10.2	3073.85	10.7	5.55	3.05	1.6	12.75	3.45
SD	0.63639	2.61629	1.41421	0.28284	31.6076	4.24264	2745.20	0.56568	0.91923	2.89913	0.42426	7.56604	0.49497
RSD	0.15713	0.26032	0.34493	0.04714	0.88910	0.41594	0.89308	0.05286	0.16562	0.95053	0.26516	0.59341	0.14347
Max	4.5	11.9	5.1	6.2	57.9	13.2	5015	11.1	6.2	5.1	1.9	18.1	3.8
Min	3.6	8.2	3.1	5.8	13.2	7.2	1132.7	10.3	4.9	1	1.3	7.4	3.1
I1	6.2	2.2	6.8	3.1	25.8	6.6	22.7	6.8	9.4	6.5	1.4	6.2	1.8
I2	2.6	11	3.3	7.5	18.2	29.1	674.1	4.6	6.2	4	3.7	25.6	2.6
Total	8.8	13.2	10.1	10.6	44	35.7	696.8	11.4	15.6	10.5	5.1	31.8	4.4
Mean	4.4	6.6	5.05	5.3	22	17.85	348.4	5.7	7.8	5.25	2.55	15.9	2.2
SD	2.5455	6.2225	2.4748	3.11127	5.3740	15.909	460.60	1.55563	2.26274	1.76776	1.62634	13.7178	0.5656
RSD	0.5785	0.9428	0.4900	0.58703	0.2442	0.8913	1.3220	0.27291	0.29009	0.33671	0.637783	0.862759	0.25713
Max	6.2	11	6.8	7.5	25.8	29.1	674.1	6.8	9.4	6.5	3.7	25.6	2.6
Min	2.6	2.2	3.3	3.1	18.2	6.6	22.7	4.6	6.2	4	1.4	6.2	1.8

APPENDIX A29

Organophosphorus pesticides (μg/Kg) in watermelon rind from Bolgatanga and Cape Coast

	Meth	Enth	Phorate	Diazin	Dimeth	Pirim	Chlor	Fen	Parat	Fono	Profen	Mala	Chlorf
J1	3.5	11.2	2.4	10.4	18.9	12.4	232.4	4	18	2.2	4.1	4.7	1.1
J2	2.8	12.8	4.9	9.7	19.5	10.1	818.9	3.2	12.7	6	3.7	23.8	2.6
Total	6.3	24	7.3	20.1	38.4	22.5	1051.3	7.2	30.7	8.2	7.8	28.5	3.7
Mean	3.15	12	3.65	10.05	19.2	11.25	525.65	3.6	15.35	4.1	3.9	14.25	1.85
SD	0.49497	1.13137	1.76776	0.49497	0.42426	1.62634	414.718	0.56568	3.74766	2.68700	0.28284	13.5057	1.06066
RSD	0.15713	0.09428	0.48432	0.04925	0.02209	0.14456	0.78896	0.15713	0.24414	0.65536	0.07252	0.94777	0.57333
Max	3.5	12.8	4.9	10.4	19.5	12.4	818.9	4	18	6	4.1	23.8	2.6
Min	2.8	11.2	2.4	9.7	18.9	10.1	232.4	3.2	12.7	2.2	3.7	4.7	1.1
K1	3.7	14.1	4.9	9.2	17.3	9.7	2283.1	3.9	11.2	8.9	9.6	5.2	3.6
K2	9.3	6.4	2.2	3.4	24.4	12.9	2113.7	1.9	3.9	4.4	4.2	3.4	1.8
Total	13	20.5	7.1	12.6	41.7	22.6	4396.8	5.8	15.1	13.3	13.8	8.6	5.4
Mean	6.5	10.25	3.55	6.3	20.85	11.3	2198.4	2.9	7.55	6.65	6.9	4.3	2.7
SD	3.9597	5.4447	1.9091	4.1012	5.0204	2.2627	119.783	1.4142	5.16188	3.18198	3.81837	1.27279	1.27279
RSD	0.6092	0.5311	0.5378	0.6509	0.2407	0.2002	0.05448	0.48766	0.68369	0.47849	0.55338	0.295998	0.471405
Max	9.3	14.1	4.9	9.2	24.4	12.9	2283.1	3.9	11.2	8.9	9.6	5.2	3.6
Min	3.7	6.4	2.2	3.4	17.3	9.7	2113.7	1.9	3.9	4.4	4.2	3.4	1.8

APPENDIX A30

Organochlorine pesticides (µg/Kg) in watermelon rind from Ayensudo and Sekondi-Takoradi

	Lindane	Hepta	Aldri	Endosul	P'P'-DDE	Dield	P'P'-DDD	P'P'-DDT	Methox	Endrin
E5	3.73	15.9	14.9	8.86	1.4	12.7	0.3	0.2	4.2	9.2
E4	5.7	11.6	18.9	12.26	4.8	14.9	2.1	3.3	14.4	10.4
E3	14.73	18.2	12.9	10.16	7.9	10.8	1.3	7.2	12.6	14.5
E2	5.73	1.9	0.3	10.36	19.6	0.6	5.1	1.3	8.2	12.4
E1	3.63	17.1	17.4	8.66	2.5	10	1.2	3.8	24.3	8.9
F5	5.73	7.9	4.2	5.8	8.2	7.4	4.1	1.1	2.8	11.9
F4	6.33	1.9	4.8	10.86	11.5	8.1	2.3	4.2	8.9	6.3
F3	5.8	3	2.6	13.3	2.8	5.3	1.9	3.9	31.2	7.4
F2	2.03	4.7	12.8	7.4	9.3	4.9	3.1	1.2	12.6	6.1
F1	6.93	5.8	11.6	11.93	4.7	0.9	0.2	9.6	13.9	1.3
Total	60.34	88	100.4	99.59	72.7	75.6	21.6	35.8	133.1	88.4
Mean	5.73	6.85	12.2	10.26	6.35	7.75	2	3.55	12.6	9.05
SD	3.399046	6.42028	6.551539	2.311702	5.428331	4.738073	1.575648	2.951384	8.707525	3.79362
RSD	0.593202	0.937267	0.537011	0.225312	0.854855	0.611364	0.787824	0.831376	0.691073	0.419185
Max	14.73	18.2	18.9	13.3	19.6	14.9	5.1	9.6	31.2	14.5
Min	2.03	1.9	0.3	5.8	1.4	0.6	0.2	0.2	2.8	1.3
G1	6.56	3.5	18.4	23.8	4.6	7.8	3.2	2.7	29.7	6.2
G2	15.33	7.4	12.2	30.7	2.9	15.3	6.3	4.8	8.7	5.4
Total	21.89	10.9	30.6	54.5	7.5	23.1	9.5	7.5	38.4	11.6
Mean	10.945	5.45	15.3	27.25	3.75	11.55	4.75	3.75	19.2	5.8
SD	6.201326	2.757716	4.384062	4.879037	1.202082	5.303301	2.192031	1.484924	14.84924	0.565685
RSD	0.5	0.5	0.5	0.5	0.5	0.5	0.5	0.5	0.5	0.5
Max	15.33	7.4	18.4	30.7	4.6	15.3	6.3	4.8	29.7	6.2
Min	6.56	3.5	12.2	23.8	2.9	7.8	3.2	2.7	8.7	5.4

APPENDIX A31

Organochlorine pesticides (µg/Kg) in watermelon rind from Accra and Kumasi

	Lindane	Hepta	Aldri	Endosul	P'P'-DDE	Dield	P'P'-DDD	P'P'-DDT	Methox	Endrin
H1	10.93	2.5	2.4	17.7	4.6	1.7	4.9	1.7	3.8	4.3
H2	12.4	13.1	2.9	11.73	14.6	6.4	1.8	9.9	12.3	10.7
Total	23.33	15.6	5.3	29.43	19.2	8.1	6.7	11.6	16.1	15
Mean	11.665	7.8	2.65	14.715	9.6	4.05	3.35	5.8	8.05	7.5
SD	1.039447	7.495332	0.353553	4.221427	7.071068	3.323402	2.192031	5.798276	6.010408	4.525483
RSD	0.089108	0.96094	0.133416	0.286879	0.73657	0.820593	0.654338	0.999703	0.746634	0.603398
Max	12.4	13.1	2.9	17.7	14.6	6.4	4.9	9.9	12.3	10.7
Min	10.93	2.5	2.4	11.73	4.6	1.7	1.8	1.7	3.8	4.3
I1	7.1	10.5	11.6	15.13	1.9	9.8	7.3	4.2	3.3	6.8
I2	13.16	6.1	1.1	14.63	9.3	1.6	6.2	1.3	4.9	13.4
Total	20.26	16.6	12.7	29.76	11.2	11.4	13.5	5.5	8.2	20.2
Mean	10.13	8.3	6.35	14.88	5.6	5.7	6.75	2.75	4.1	10.1
SD	4.285067	3.11127	7.424621	0.353553	5.23259	5.798276	0.777817	2.05061	1.131371	4.666905
RSD	0.423008	0.374852	1.169232	0.02376	0.934391	1.017241	0.115232	0.745676	0.275944	0.46207
Max	13.16	10.5	11.6	15.13	9.3	9.8	7.3	4.2	4.9	13.4
Min	7.1	6.1	1.1	14.63	1.9	1.6	6.2	1.3	3.3	6.8

APPENDIX A32

Organochlorine pesticides (µg/Kg) in watermelon rind from Bolgatanga and Cape Coast

	Lindane	Heptac	Aldrin	Endosul	P'P'-DDE	Dieldrin	P'P'-DDD	P'P'-DDT	Methoxy	Endrin
J1	14.13	10.3	23.1	17.86	5.3	12.4	9.3	8.1	4.2	8.2
J2	8.06	17.3	19.6	20	15.3	17.3	7.3	1.7	23.5	7.2
Total	22.19	27.6	42.7	37.86	20.6	29.7	16.6	9.8	27.7	15.4
Mean	11.095	13.8	21.35	18.93	10.3	14.85	8.3	4.9	13.85	7.7
SD	4.292138	4.949747	2.474874	1.513209	7.071068	3.464823	1.414214	4.525483	13.64716	0.707107
RSD	0.386853	0.358677	0.115919	0.079937	0.686511	0.233321	0.170387	0.923568	0.985355	0.091832
Max	14.13	17.3	23.1	20	15.3	17.3	9.3	8.1	23.5	8.2
Min	8.06	10.3	19.6	17.86	5.3	12.4	7.3	1.7	4.2	7.2
K1	15.8	11.5	19.4	36	14.8	6.6	1.9	1.2	12.8	11.8
K2	9.4	7.9	5.2	22.73	9.3	1.9	4.9	3.9	8.3	1.8
Total	25.2	19.4	24.6	58.73	24.1	8.5	6.8	5.1	21.1	13.6
Mean	12.6	9.7	12.3	29.365	12.05	4.25	3.4	2.55	10.55	6.8
SD	4.525483	2.545584	10.04092	9.383307	3.889087	3.323402	2.12132	1.909188	3.181981	7.071068
RSD	0.359165	0.262431	0.816335	0.319541	0.322746	0.781977	0.623918	0.748701	0.30161	1.039863
Max	15.8	11.5	19.4	36	14.8	6.6	4.9	3.9	12.8	11.8
Min	9.4	7.9	5.2	22.73	9.3	1.9	1.9	1.2	8.3	1.8

APPENDIX A33

Synthetic pyrethroid pesticides (µg/Kg) in watermelon rind from Ayensudo and Sekondi-Takoradi

	Bifen	Lambda	Perm	Cyflu	Cyperm	Fenva	Deltam
E5	1.2	1.4	1.8	3.6	3.2	1.9	1.5
E4	2.2	1.9	3.6	1.9	3.1	1.6	2.5
E3	3.1	2.2	2.9	1.7	3.8	1.7	3.2
E2	4.2	1.6	2.2	3.9	1.6	3.8	1.2
E1	1.2	1.9	3.4	2.3	1.2	2.8	1.5
F5	2.9	4.2	2.1	1.6	3.4	2.5	2.7
F4	1.9	3.9	2.8	2.9	3.2	2.4	3.2
F3	4.3	3.6	2.3	2.7	2.2	1.6	1.6
F2	2.4	2.6	3.1	1.4	2.7	1.8	1.9
F1	2.2	1.9	2.3	3.3	2.3	2.1	3.1
Total	**25.6**	**25.2**	**26.5**	**25.3**	**26.7**	**22.2**	**22.4**
Mean	**2.3**	**2.05**	**2.55**	**2.5**	**2.9**	**2**	**2.2**
SD	**1.082384**	**1.014122**	**0.598609**	**0.885751**	**0.831398**	**0.689283**	**0.786271**
RSD	**0.470602**	**0.494694**	**0.234749**	**0.354301**	**0.286689**	**0.344642**	**0.357396**
Max	**4.3**	**4.2**	**3.6**	**3.9**	**3.8**	**3.8**	**3.2**
Min	**1.2**	**1.4**	**1.8**	**1.4**	**1.2**	**1.6**	**1.2**
G1	2.9	2.7	2.6	1.4	2.4	2.1	1.6
G2	1.8	3.8	2.3	2.3	1.4	4.5	1.8
Total	**4.7**	**6.5**	**4.9**	**3.7**	**3.8**	**6.6**	**3.4**
Mean	**2.35**	**3.25**	**2.45**	**1.85**	**1.9**	**3.3**	**1.7**
SD	**0.777817**	**0.777817**	**0.212132**	**0.636396**	**0.707107**	**1.697056**	**0.141421**
RSD	**0.330986**	**0.239328**	**0.086585**	**0.343998**	**0.372161**	**0.514259**	**0.083189**
Max	**2.9**	**3.8**	**2.6**	**2.3**	**2.4**	**4.5**	**1.8**
Min	**1.8**	**2.7**	**2.3**	**1.4**	**1.4**	**2.1**	**1.6**

APPENDIX A34

Synthetic pyrethroid pesticides (μg/Kg) in watermelon rind from Accra and Kumasi

	Bifen	Lambda	Perm	Cyflu	Cyperm	Fenva	Deltam
H1	2.6	1.2	2.4	3.6	2.5	3.7	2.8
H2	1.4	3.9	3.1	2.2	3.5	2.2	1.4
Total	**4**	**5.1**	**5.5**	**5.8**	**6**	**5.9**	**4.2**
Mean	**2**	**2.55**	**2.75**	**2.9**	**3**	**2.95**	**2.1**
SD	**0.848528**	**1.909188**	**0.494975**	**0.989949**	**0.707107**	**1.06066**	**0.989949**
RSD	**0.424264**	**0.748701**	**0.179991**	**0.341362**	**0.235702**	**0.359546**	**0.471405**
Max	**2.6**	**3.9**	**3.1**	**3.6**	**3.5**	**3.7**	**2.8**
Min	**1.4**	**1.2**	**2.4**	**2.2**	**2.5**	**2.2**	**1.4**
I1	1.7	3.8	2.3	3.1	2.5	2.9	2.4
I2	1.9	1.7	2.2	2.4	1.7	3.7	3.2
Total	**3.6**	**5.5**	**4.5**	**5.5**	**4.2**	**6.6**	**5.6**
Mean	**1.8**	**2.75**	**2.25**	**2.75**	**2.1**	**3.3**	**2.8**
SD	**0.141421**	**1.484924**	**0.070711**	**0.494975**	**0.565685**	**0.565685**	**0.565685**
RSD	**0.078567**	**0.539972**	**0.031427**	**0.179991**	**0.269374**	**0.17142**	**0.202031**
Max	**1.9**	**3.8**	**2.3**	**3.1**	**2.5**	**3.7**	**3.2**
Min	**1.7**	**1.7**	**2.2**	**2.4**	**1.7**	**2.9**	**2.4**

APPENDIX A35

Synthetic pyrethroid pesticides (µg/Kg) in watermelon rind from Bolgatanga and Cape Coast

	Bifen	Lambda	Perm	Cyflu	Cyperm	Fenva	Deltam
J1	1.2	1.7	2.1	1.4	3.3	1.2	3.4
J2	1.3	2.9	2.8	2.3	1.1	1.5	2.1
Total	**2.5**	**4.6**	**4.9**	**3.7**	**4.4**	**2.7**	**5.5**
Mean	**1.25**	**2.3**	**2.45**	**1.85**	**2.2**	**1.35**	**2.75**
SD	**0.070711**	**0.848528**	**0.494975**	**0.636396**	**1.555635**	**0.212132**	**0.919239**
RSD	**0.056569**	**0.368925**	**0.202031**	**0.343998**	**0.707107**	**0.157135**	**0.334269**
Max	**1.3**	**2.9**	**2.8**	**2.3**	**3.3**	**1.5**	**3.4**
Min	**1.2**	**1.7**	**2.1**	**1.4**	**1.1**	**1.2**	**2.1**
K1	2.9	2.3	2	1.7	1.2	3.3	2.6
K2	1.2	3.4	1.3	2.4	1.4	1.5	1.1
Total	**4.1**	**5.7**	**3.3**	**4.1**	**2.6**	**4.8**	**3.7**
Mean	**2.05**	**2.85**	**1.65**	**2.05**	**1.3**	**2.4**	**1.85**
SD	**1.202082**	**0.777817**	**0.494975**	**0.494975**	**0.141421**	**1.272792**	**1.06066**
RSD	**0.586381**	**0.272918**	**0.299985**	**0.241451**	**0.108786**	**0.53033**	**0.57333**
Max	**2.9**	**3.4**	**2**	**2.4**	**1.4**	**3.3**	**2.6**
Min	**1.2**	**2.3**	**1.3**	**1.7**	**1.2**	**1.5**	**1.1**
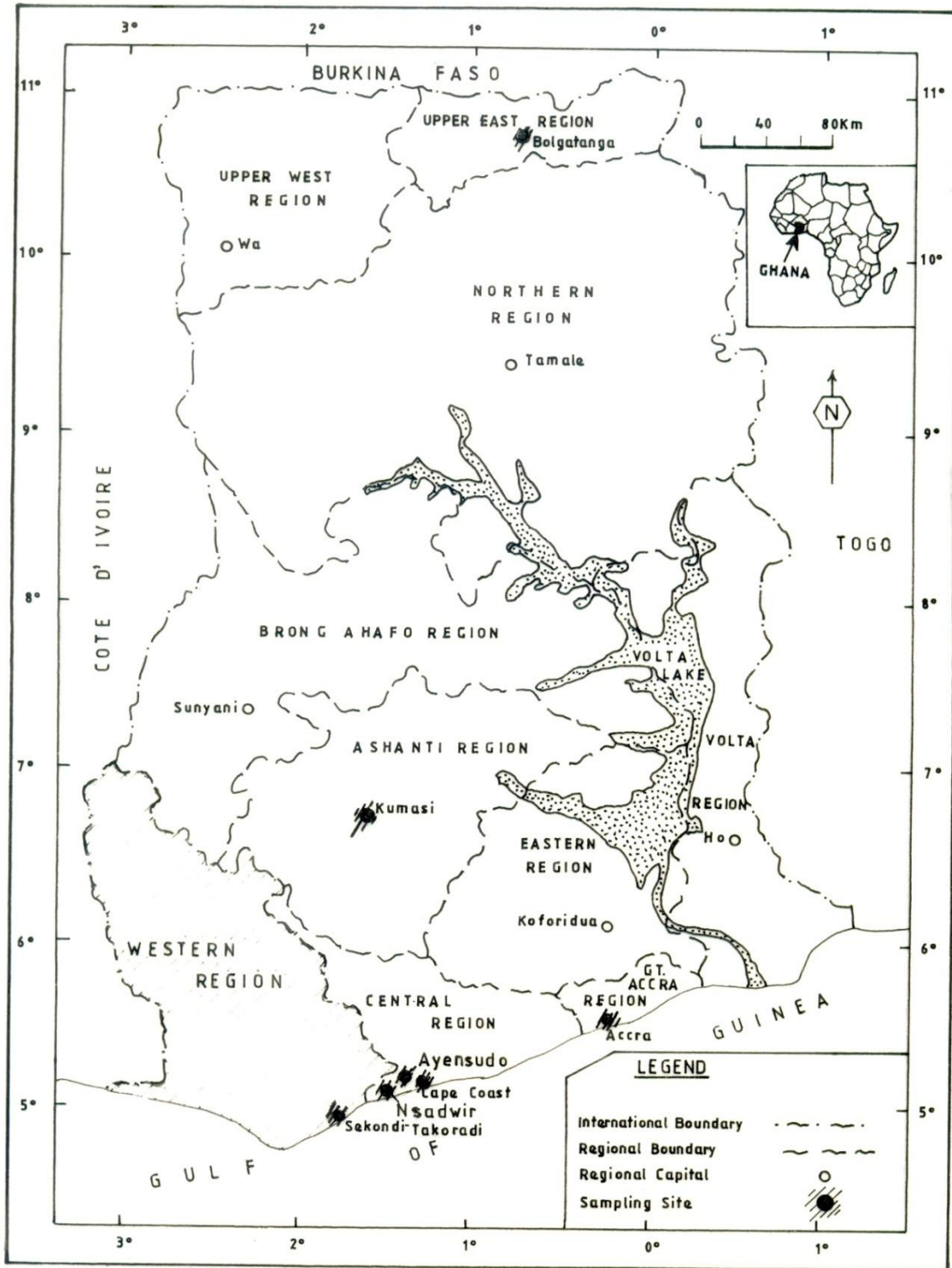
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