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HEALTH RISK ASSESSMENT OF PESTICIDE RESIDUES IN PROCESSED CEREAL-BASED COMPLEMENTARY FOODS FOR INFANTS AND YOUNG CHILDREN

BY

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A Thesis submitted to the Department of Food Science and Technology in Partial fulfillment of the requirement for the degree of Master of Science in Food Quality Management

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DECLARATION

I hereby declare that, except for references to other people's work which have been duly acknowledged, this thesis, submitted to the School of Research and Graduate Studies, KNUST, Kumasi is the result of my own original research and that this thesis has not been presented for any degree elsewhere.

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ABSTRACT

Thirty six pesticides comprising 13 organophosphorous, 14 organochlorine and 9 synthetic pyrethroid pesticides were analyzed in 10 brands of processed cereal-based complementary foods for older infants and young children. The QuEChERS (quick, easy, cheap, effective, rugged and safe) method was used for extraction and clean-up. Subsequent detection and quantification was done using Gas Ghromatograph equipped with Electron Capture Detector and Pulse Flame Photometric Detector. Estimated Daily Intakes of pesticides were also determined. The results indicated that mean concentrations for all processed cereal-based complementary food ranged from 0.001 – 0.0126 $mgkg^{-1}$ for OPs, 0.002 – 0.022 $mgkg^{-1}$ for OCs and 0.002 – 0.017 $mgkg^{-1}$ for pyrethroids. The OP pesticide with the highest concentration was methamidophos; lindane (γ-HCH) recorded the highest concentration amongst the OCs detected whilst permethrin recorded the highest concentration amongst the pyrethroids detected. Maximum residue limit of 0.01 $mgkg^{-1}$ for baby food was exceeded for the following OP pesticides: pirimiphos-methyl in baby food I; fenitrothion in baby food E and I; chlorpyrifos in baby food C; and methamidophos in baby food F). For the OCs the following pesticides exceeded the MRL of 0.01 $mgkg^{-1}$: *p,p'*-DDE in baby food G; dieldrin in baby food G; β-endosulfan in baby food B; β-HCH in baby food C and I; and γ-HCH in baby food D and E. In the case of synthetic pyrethroid pesticides, cypermethrin in baby food J and permethrin in baby food D exceeded the MRL for baby foods. Results from exposure assessment indicated that EDI for OP and synthetic pyrethroid pesticides were below the acceptable daily intakes (ADI). The non-carcinogenic risk assessment conducted for these pesticides indicated Hazard Index (HI) of less than 1 for both OP and synthetic pyrethroid pesticides. The exposure levels of γ-HCH in baby food D and E, heptachlor in baby food E and dieldrin in baby food G and H were higher than their respective ADI. The three OC pesticides subsequently recorded HIs greater than 1 indicating the possibility of adverse health effect on consumers. The Hazard Ratio (HR) for carcinogenic risk for OC was greater than 1 for β-HCH in baby food A, C, D and I; dieldrin in baby food F, G and H; heptachlor in baby food E and F; γ-HCH in baby food B; and γ-chlordane in baby food E. This result raises serious concerns of possible carcinogenicity for infants and young children.

TABLE OF CONTENTS

DECLARATION i

ACKNOWLEDGEMENTS iii

ABSTRACT iv

TABLE OF CONTENTS v

LIST OF TABLES ix

LIST OF ABBREVIATIONS xi

CHAPTER ONE 1

1.0 INTRODUCTION 1

1.1 Background 1

1.2 Problem statement 4

1.3 Aim and objectives 5

1.3.1 Aim 5

1.3.2 Specific objectives 6

1.4 Justification 6

2.0 LITERATURE REVIEW 8

2.1 Classification of Pesticides 8

2.1.1 Organophosphorous pesticides 8

2.1.2 Organochlorine pesticides 9

2.1.3 Synthetic pyrethroid pesticides 9

2.2 Classification of Pesticides as Chemical Hazards 10

2.3 Pesticide use in Agriculture Production 11

2.4 Childhood Feeding 12

2.5 Exposure at Different Stages of Human Development 13

2.6 Risk Assessment 15

2.6.1 Hazard Identification 16

2.6.2 Hazard Characterization 16

KNUST

2.6.3 Exposure Assessment 17
2.6.3.1 Approaches for Measuring Exposure 18
2.6.3.2 Food Consumption Estimates 19
2.6.3.3 Prospective Method for Estimation of Food Consumption 20
2.6.3.4 Retrospective Method for Estimation of Food Consumption 21
2.6.3.5 Estimation of Dietary Exposure of Pesticide Residues 21
2.7 Risk Characterization 23
2.7.1 Approaches in Food Chemical Risk Characterization 24
2.7.1.1 Carcinogenic and Non-Carcinogenic Risk 24
2.7.1.2 Risk assessment for mixtures of compounds 25
2.8 Analytical Methods for Pesticide Residues in Cereal-based Complementary Foods 26
CHAPTER THREE 28
3.0 MATERIALS AND METHODS 28
3.1 MATERIALS 28
3.1.1 Certified Reference Pesticides Standards 28
3.1.1.1 Organochlorine (OC) Pesticides 28
3.1.1.2 Organophosphorous (OP) Pesticides 28
3.1.1.3 Synthetic pyrethroids 28
3.1.2 Other Reagents and Chemicals 29
3.1.3 Equipment 29
3.2 METHOD 29
3.2.1 Sampling 29
3.2.2 Preparation of Mixed Pesticide Reference Standards for Fortification and Instrument 30
3.2.3 Extraction and Clean-Up 30
3.2.4 Dispersive Solid Phase Extraction 31
3.2.5 Quality Control 32

3.2.6 Analysis of Pesticide Residue Content 32
3.2.7 Exposure Assessment 34
3.2.7.1 Food Consumption 34
3.2.7.2 Body Weight Estimation 34
3.2.7.3 Consumption Rate Estimation 37
3.6.3 Estimation of Dietary Exposure 37
3.7 Risk Characterization 37
3.8 Data Analysis 38
CHAPTER FOUR 39
4.0 RESULTS 39
4.1 Quality Control 39
4.2 Pesticide Residues in Processed Cereal-based Complementary Food Samples… 39
4.2.1 Organophosphorous Pesticide Residues in Processed Cereal-based Complementary 40
4.2.2 Organochlorine Pesticide Residues in Processed Cereal-based Complementary Food 44
4.2.3 Synthetic Pyrethroids Pesticide Residues in Processed Cereal-based Complementary 48
4.3 Health Risk Assessment of Pesticide Residues in Processed Cereal-based Complementary Food 52
4.3.2 Health Risk Assessment 64
4.3.2.1 Non-carcinogenic Risk 64
4.3.2.2 Carcinogenic Risk 65
CHAPTER FIVE 71
5.0 DISCUSSION 71
5.1 Concentration of Pesticide Residues in Processed Cereal-based Complementary Food 71
5.2 Risk Assessment 76

KNUST

5.2.1 Non-Carcinogenic Risk 76
5.2.2 Carcinogenic Risk 79
CHAPTER SIX 81
6.0 CONCLUSIONS AND RECOMMENDATIONS 81
6.1 CONCLUSIONS 81
6.2 RECOMMENDATIONS 82
REFERENCES 84

KNUST

LIST OF TABLES

Table 3.1: Average weight for infants aged 6 – 11 months……..…...35

Table 3.2: Average weight for young children aged 12 – 24 months……36

Table 4.2a: Range, mean and standard deviation of OP pesticide residues in processed cereal-based complementary food ……42

Table 4.2b: Range, mean and standard deviation of OP pesticide residues in processed cereal-based complementary food……..……43

Table 4.2c: Range, mean and standard deviation of OC pesticide residues in processed cereal-based complementary food……..……45

Table 4.2d: Range, mean and standard deviation of OC pesticide residues in processed cereal-based complementary food……..……47

Table 4.2e: Range, mean and standard deviation of synthetic pyrethroids pesticide residues in processed cereal-based complementary food……..……49

Table 4.2f: Range, mean and standard deviation of synthetic pyrethroids pesticide residues in processed cereal-based complementary food……..……51

Table 4.3: Consumption rate of processed cereal-based complementary food for infants and young children……..53

Table 4.3a: Health risk assessment for organophosphorous pesticide residues in processed cereal-based complementary food……..55

Table 4.3b: Health risk assessment for organophosphorous pesticide residues in processed cereal-based complementary food……..……56

Table 4.3c: Health risk assessment for organochlorine pesticide residues in processed cereal-based complementary food……..……57

Table 4.3d: Health risk assessment for organochlorine pesticide residues in processed cereal-based complementary food……..……58

Table 4.3e: Health risk assessment for organochlorine pesticide residues in processed cereal-based complementary food……..……59

Table 4.3f: Health risk assessment for synthetic pyrethroids pesticide residues in processed cereal-based complementary food……..…60

Table 4.3g - Health risk assessment for synthetic pyrethroids pesticide residues in processed cereal-based complementary food……..……61

Table 4.3h: Health risk assessment for synthetic pyrethroids pesticide residues in processed cereal-based complementary food……..……62

Table 4.3i – Health risk assessment for synthetic pyrethroids pesticide residues in processed cereal-based complementary food……..……63

Table 4.3j: Cancer Benchmark Concentrations for organochlorine pesticides in processed cereal-based complementary foods A – E……66

Table 4.3k: Hazard Ratios for organochlorine pesticides in processed cereal-based complementary foods A – E……..67

Table 4.3l: Cancer Benchmark Concentrations for organochlorine pesticides in processed cereal-based complementary foods E – I……...68

Table 4.3m: Hazard Ratios for organochlorine pesticides in processed cereal-based complementary foods E – I……69

KNUST

LIST OF ABBREVIATIONS

ADI	Acceptable Daily Intake
ARfD	Acute Reference Dose
BW	Body Weight
CAC	Codex Alimentarius Commission
CBC	Cancer Benchmark Concentration
EDI	Estimated Daily Intake
EFSA	European Food Safety Authority
FAO	Food and Agriculture Organization
FSA	Food Safety Agency of United Kingdom
GHS	Globally Harmonized System of Classification and Labelling of Chemicals
GSS	Ghana Statistical Service
HI	Hazard Index
HR	Hazard Ratio
IPCS	International Programme on Chemical Safety
MRL	Maximum Residue Limit
NRC	National Research Council
OC	Organochlorine
OP	Organophosphorous
OSF	Oral Slope Factor
POPs	Persistent Organic Pollutants
PSA	Primary Secondary Amine
QuEChERs	Quick Easy Cheap Effective Rugged and Safe
TMDI	Theoritical Maximum Daily Intake
UNECE	United Nations Economic Community for Europe
UNEP	United Nations Environmental Programme
US EPA	United States Environmental Protection Agency
WHO	World Health Organization

CHAPTER ONE

1.0 INTRODUCTION

1.1 Background

Agricultural production is often plagued by crop diseases which results in the reduction of production yield. These crop diseases are caused by pests such as insects, bacteria, fungi, and viruses. These pests interfere with production, processing, storage and transport of food. Substances or mixture of substances used for preventing, destroying, repelling, or mitigating pests are referred to as pesticides (Nollet and Rathore, 2010).

Despite the benefits associated with the use of pesticides in agricultural production (Warren, 1998; Webster *et al.,* 1999; Lewis *et al.,* 2005), global public health concerns persist on the utilization of pesticides because of the potential adverse health effects such as carcinogenesis, developmental and reproductive effects, immunological effects, neurotoxicity, cytogenetic damage, and endocrine disruption (FAO/WHO, 2007) as result of exposure to pesticides.

Evidence of pesticide contamination of raw agricultural products, processed foods, and environmental matrices such as water and soil is well documented. Carbamates, organophosphorous (OP) and pyrethroid pesticide residues have been detected in fresh water (Helisoma and Gabol, 2005), triazine and organochlorines (OC) in water (Yang *et al.,* 2005; Zhang *et al.,* 2005). Soils have also been found to be contaminated with various pesticides (Goa *et al.,* 2005; Saxton and Engel, 2005; Syversen and Haarstad, 2005).

There has also been widespread occurrence of different classes of pesticides in food in different regions of the world. Dithiocarbamate and organophosphorous pesticides in fruits and vegetables from Egypt (Dogheim *et al.*, 2002), organophosphorous and organochlorine pesticides have been detected in the Canadian diet (Rawn, 2003). In Cameroon organophosphorous, organochlorine and synthetic pyrethroid pesticides have been identified in various food products (Gimou *et al.*, 2008) whilst organophosphorous and dithiocarbamate have been detected in foods served at some restaurants (Caldas *et al.*, 2011) in Brazil.

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The global occurrence of pesticides in food necessitates the need for public health interventions to safeguard the health of consumers from these chemical hazards. One of the risk management approaches taken by the international community and national regulatory authorities to protect public health is the establishment of Acceptable Daily Intake (ADI) of pesticides which defines the toxicological limit for chronic exposure. In the case of short term exposure (acute toxicity), the Acute Reference Dose (ARfD) is used as the health-based reference limit. Thus, the regulatory goal is that the mean dietary intake of pesticide residue in food should not exceed the ADI over a considerable period of time, while short-term exposures should not exceed the ARfD.

The levels of pesticide residues in food are generally regulated to minimize the unnecessary exposure of the consumer to pesticides and ensure the proper use of pesticides in terms of application rates and pre-harvested intervals. Regulators use the concept of Maximum Residue Limits (MRL) for pesticides to protect the health of consumers. Unlike the ADI or ARfD, MRLs are not toxicological limits. If the

MRLs are exceeded, there is strong indication of violations of Good Agricultural Practices (GAPs). When the MRLs are exceeded the exposure is compared with ADIs and/or ARfDs to ascertain whether or not there is a possible chronic or acute health risk, respectively (Nasreddine and Parent-Massin, 2002).

The term 'risk' is defined as the probability of an adverse effect in an organism, system, or (sub) population caused under specified circumstances by exposure to an agent (IPCS, 2004). To determine whether pesticides posses risk to humans, risk assessment has to be conducted. Risk assessment refers to the process intended to calculate or estimate the risk to a given target organism, system, or (sub)population, including the identification of attendant uncertainties, following exposure to a particular agent, taking into account the inherent characteristics of the agent of concern as well as the characteristics of the specific target system (IPCS, 2004).

Dietary intake represents a major route of pesticides exposure to humans especially children (Fenske *et al*., 2002a; Clayton *et al*., 2003). Dietary exposure assessments are used to estimate level of exposure to a food chemical (e.g. pesticide) following ingestion (WHO, 2009). This is done by combining food consumption data with data on the concentration of chemicals in food. The process of risk assessment is completed by comparing the EDI of the food chemical e.g. pesticide with the relevant toxicological limit i.e. ADI or ARfD. If the EDI exceeds the ADI or ARfD, then there is the probability of an adverse effect.

Application of pesticides in agricultural production has the potential effect of leaving pesticide residues in raw agricultural produce and the subsequent carry-over into

final food products. For example processed cereal-based complementary foods formulated from raw agricultural produce such as wheat, maize, rice, millet etc. are very good examples. Thus the use of pesticides during agricultural production could result in the contamination of these produce which could eventually lead to the presence of pesticide residues in the processed product.

1.2 Problem statement

Given the global occurrence of pesticide residues in food, it is hypothesized that processed cereal-based complementary foods consumed in Ghana are contaminated with OP, OCs and synthetic pyrethroid pesticide residues. Thus infants and young children who consume complementary foods are exposed to pesticides residues above the health-based regulatory levels leading to a risk of adverse health effect.

Children utilize more oxygen and require higher food consumption rate per kilogram body weight than adults due to their higher basal metabolic rate and energy requirements (IPCS, 2004). This means that if foods fed to children are contaminated with toxic chemicals, children risk higher exposures than adults (NRC, 1993; Faustman *et al.*, 2000; Kroes *et al*, 2002; IPCS, 2004). The quality of food consumed by infants and young children is therefore critical in early childhood development.

Several adverse health outcomes have been linked with children's exposure to environmental toxicants. Exposure of children to pesticides may cause Parkinson-like declines in dopaminergic neurons in adulthood (Cory-Slechta *et al*., 2005). Studies also indicate that exposure to the herbicide, atrazine, could delay puberty (Laws *et al*., 2000; Ashby *et al*., 2002). Dietary exposure to pesticides is known to

result in several health effects in children. For example, childhood cancer, neurological and endocrine effects have been associated with the consumption of pesticide-contaminated foods (Garry, 2003).

Despite the peculiar vulnerabilities of children to pesticide exposure, studies on dietary exposure in children are generally limited (Flower *et al.*, 2004; Renwick *et al.*, 2005; Cohen, 2007), with most published studies focusing on chemical exposure among adults (Huybrechts *et al.*, 2011). It has however, been established that physiologic and behavioural patterns amongst adults and children result in different exposures for adults and children as well as children of different developmental stages (Hubal *et al.*, 2000). The paucity of exposure data for infants and young children may therefore present risk management challenges. Hence, monitoring of foods for infants and young children is crucial in assessing the dietary exposure to potentially harmful chemicals during the early years of life (Piccinelli *et al.*, 2010).

1.3 Aim and objectives

1.3.1 Aim

The aim of the study is to assess the risk posed to infants and young children in Ghana by OP, OC and synthetic pyrethroids pesticide residues following the consumption of locally produced and imported processed cereal-based complementary foods.

1.3.2 Specific objectives

The study is being conducted to:

i. Determine the types and levels of pesticide residues in different complementary foods consumed in Ghana.

ii. Perform dietary exposure assessment of processed cereal-based complementary foods.

iii. Characterize the carcinogenic and non-carcinogenic risks associated with the consumption of pesticide-contaminated processed cereal-based complementary foods.

1.4 Justification

In Ghana, studies on risk assessment of pesticide residues in food products are limited. In 2011, Bempah *et al.* reported the risk associated with the consumption of pesticide-contaminated fruits and vegetables in the Kumasi metropolis. Akoto *et al.* (2013) conducted health risk assessment of pesticide residues in maize and cowpea in Ejisu. These studies were limited to the general population and raw agricultural food products. Dietary exposure information on pesticide residues in processed cereal-based complementary foods for infants and young children is generally lacking.

From literature, there is no documented investigation of risk assessment of processed cereal-based complementary foods consumed by children in Ghana although some work has been done on raw cereals as reported by Akoto *et al.* (2013). Since infants and young children have greater intake of food than adults on a body weight basis and chemicals may have permanent effects on developing systems in children which

would transient in the adult (FSA, 2012), there is the urgent need to characterize the risk associated with dietary pesticide exposure in infants and young children. The outcome of this risk assessment work could provide valuable information to risk managers in Ghana on the best possible risk management options capable of protecting children against disease and injury caused by pesticides in cereal-based complementary foods consumed in Ghana.

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CHAPTER TWO

2.0 LITERATURE REVIEW

2.1 Classification of Pesticides

Several approaches exist for classifying pesticides. For example, pesticides can be classified based on the type of pest they control. Fungi, weeds, insects, nematodes etc. are all pests hence are controlled by pesticides class such as fungicides, herbicides, insecticides, nematicides respectively. Pesticides can also be classified based on their chemical nature, examples of such are organophosphorous, organochlorine, synthetic pyrethroid etc. Other categories of pesticides include biopesticides, antimicrobials, and pest control devices (US EPA, 2014). Amongst the classes of pesticides, the organophosphorous, synthetic pyrethroid and organochlorine pesticides have contributed immensely in controlling pest in agricultural production although pesticides in the later group have either been banned or restricted in use in most countries (Aktar *et al.*, 2009).

2.1.1 Organophosphorous pesticides

Organophosphate pesticides are synthetic in origin and are normally esters, amides, or thiol derivatives of phosphoric, phosphonic, phosphorothioic, or phosphonothioic acids. The OPs constitute one of the most widely used classes of pesticides for both agricultural and landscape pest control. Kamath and Rajini (2007), suggest that the use of OP pesticides has increased considerably due to their low toxicity and low persistence in the mammalian system compared to OC pesticides. Organophosphorus pesticides have therefore replaced OC pesticides in agricultural activities and are the most frequently detected pesticides in monitoring programs worldwide (Dogheim *et al*., 2002; Caldas *et al*., 2006a), including Ghana. Despite their advantages over OC

pesticides, OP pesticides are still recognized as potent neurotoxic chemicals at high doses (Abou-Donia, 2003; Costa *et al.*, 2005). The primary mode of mechanism for OP pesticides is by inhibition of the enzyme, acetylcholinesterase (AcHE), resulting in accumulation of acetylcholine at the nerve endings. This accumulation causes neurobehavioral dysfunction in the target pest (Fukuto, 1990; Ahlbom *et al*., 1995).

2.1.2 Organochlorine pesticides

Organochlorine pesticides are a group of synthetic compounds developed in the 1940s for use mainly as insecticides (BCPC, 1998). Organochlorine pesticides belong to the class of persistent organic pollutants (POP), thus they persist in the environment for long periods even after application. Many OC pesticides have been identified as hormone disrupters, exerting their toxic effects on the hormonal and reproductive system thus resulting in adverse health effects to man (Golden *et al.*, 1998 Hosie *et al.*, 2000). Despite the enormous restriction on the use of OCPs due to the entry into force of the Stockholm Convention on POPs (UNEP (2004)), there is still documented evidence of OC pesticides in food samples (Waliszewski *et al.*, 2003*;* Da Silva *et al.*, 2010; Dubois *et al.*, 2010; Fernandes *et al.*, 2011a; Akoto *et al.*, 2013). Organochlorine pesticides operate by alteration of ion channels (Karami-Mohajeri and Abdollahi, 2011) thus resulting in neurotoxicity.

2.1.3 Synthetic pyrethroid pesticides

Pyrethroids constitute the fourth major group of insecticides developed after OC, OP and carbamate pesticides. Synthetic pyrethroids are a group of pesticides widely used in agriculture and public health programs worldwide (Heudorf and Angerer, 2001; Lothrop *et al.*, 2007). They are non-systemic, contact and stomach poisons to many

insects and arachnids (Lopez-Lopez *et al.,* 2001). The synthetic pyrethroids mimic the insecticidal function of natural pyrethrins, which are extracts from the chrysanthemums plant. For purposes of insect control in agricultural production, the pyrethroids are preferred over their natural pyrethrin counterparts due to their resistance to environmental degradation, selective insecticidal properties and relative potency (Lopez-Lopez *et al*., 2001; Lu *et al.,* 2009). The pyrethroids differ from OP pesticides in terms of their lack of a common mechanism of action. Two types of pyrethroids exist. The pyrethroids lacking the α-cyano group cause tremors (Type I or T), whereas α-cyano pyrethroids produce a salivation/choreoathetosis syndrome (Type II or CS). Both types of pyrethroids primarily exert their effect by prolonging the opening of voltage-sensitive sodium channels (Soderlund *et al*., 2002; Shafer *et al.*, 2005) thereby the disrupting the normal functioning of the nervous system.

2.2 Classification of Pesticides as Chemical Hazards

Classification of pesticides with regards to their hazardous nature is based on the Globally Harmonized System of Classification and Labelling of Chemicals (GHS UNECE, 2009). This globally accepted system of classification has been used as the reference by many organizations including the World Health Organization (WHO) (WHO, 2009) and the US Environmental Protection Agency (US EPA, 2004) to classify pesticides according to their hazards. The classification system essentially differentiates between pesticides according to the acute risk to human health.

The WHO, for example, categorizes pesticides into five groups based on the GHS. The categories are: extremely hazardous (class Ia), highly hazardous (class Ib), moderately hazardous (class II), slightly hazardous (class III) and unlikely to present

acute hazard (Class U). Most of the pesticides in class I are banned or subject to strict regulations (WHO, 2009).

2.3 Pesticide use in Agriculture Production

Achieving food security is a major concern of governments in many countries; hence prevention of post-harvest losses is a key component of agricultural policies. In Ghana the agriculture sector is one of the most important sectors in the Ghanaian economy and is the third largest contributor (22%) to Ghana's GDP after the service (49.5%) and industry (28.6%) sectors (GSS, 2014). Pest and diseases posses serious threats to agricultural production and have the potential to reduce production if left uncontrolled, hence as part of integrated pest management strategies, pesticides have been used for plant protection (FAO, 2010).

However, the use of pesticides, presents food safety threats since pesticide residues have been detected in food from different regions of the world. The presence of pesticide residues in food could be due to their extensive use and/or misuse in agricultural production, non-compliance to pre-harvest intervals. Some banned pesticides may also be detected in food despite the fact that they are no longer in use, because of their persistent nature, e.g. Persistent Organic Pollutants such as organochlorine pesticides.

Available studies on pesticide residues in food indicate that contamination especially with OC, OP and synthetic pyrethroids pesticides is very common (Chen *et al.*, 2009a; Fenske *et al.*, 2002; Lu *et al.*, 2006; Tulve *et al.*, 2011; Bempa *et al.*, 2011; Akoto *et al.*, 2013). Toteja *et al.* (2003), detected HCH in rice, Bakore *et al.* (2004)

analyzed OC pesticides in wheat; Mawussi *et al.* (2008) detected various OCs in maize and cowpea. Organochlorine pesticides have also been detected in dairy products (Darko and Acquaah, 2008). Organophosphorus pesticides have been found to contaminate various food products including cereals (González-Curbelo *et al.*, 2012), wheat varieties (Škrbić, 2007), fruits and vegetables (Georgakopoulosw *et al.*, 2007). Studies also indicate that synthetic pyrethroid pesticides have been detected in fruits and fruit juices for preschool children (Chuang and Wilson, 2011). Some of pyrethroids pesticides detected in this work comprised permethrin, cyfluthrin, cypermethrin, fenvalerate etc.

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2.4 Childhood Feeding

Adequate nutrition is recognized as being fundamental to the development of infants and young children. The period from birth to two years is considered as the peak period for optimal growth, health and behavioral development. However, this period is also noted for retardation in growth, micronutrient deficiencies, and onset of childhood diseases (Martorell *et al.*, 1994, WHO, 2001).

The World Health Organization (WHO) recommends that infants should be exclusively breastfed for the first six months of life to achieve optimal growth, development and health. However, from the sixth month of life, breast milk is no longer adequate to meet the evolving energy and nutritional needs of infants due to the rapid developmental changes at infancy (WHO, 2002). Adequate and safe complementary foods should therefore be introduced at this stage to provide the additional energy and nutritional needs whilst breastfeeding continues to at least the twenty-fourth month. Hence the complementary feeding period (6 – 24 months) is a

transition from exclusive breastfeeding of milk and/or milk substitutes to eating the family diet (WHO, 2002).

Due to the peculiar role of complementary foods in the diet of infants and young children, complementary foods must contain all the essential nutrients needed for proper growth. Their major components therefore include legumes, pulses, animal source foods, cereals and fruits and vegetables (FAO/WHO, 2013) which are important sources of proteins, fats and oils, carbohydrates, vitamins and minerals. The important role of complementary foods during the critical stages of human development necessitates the need to safeguard the quality and safety of the ingredients for these foods in order to prevent the eventual carry-over of food chemicals into the final product.

2.5 Exposure at Different Stages of Human Development

Significant structural and functional differences exist between children and adults and this account for variations in susceptibility and toxicity to pesticides between them. A basic concept in pediatrics is that children are not "young adults". Children have distinct characteristics across life stages that account for their peculiar susceptibility with regards to exposure to pesticides (National Research Council, 1993; WHO, 2006).

The fact that organs are at immature stage in infants and children, predisposes them to the adverse effects of chemical exposures and their ability to cope with toxic injury. For example, the renal system plays a key role in detoxification; however, because the functions of the renal system are less developed in children than in adult

(Stewart & Hampton, 1987; Gomez *et al*., 1999), children are less capable of detoxifying harmful chemicals compared to adults.

Childhood is also marked by less developed nervous system thus rendering the brain more susceptible to the disrupting effects of toxic chemicals. Doses of toxic chemicals which may be toxicologically insignificant to the mature nervous system of adults may present serious toxicological implications to the developing nervous system in children (Faustman *et al*., 2000).

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Studies on metabolism at different stages of human development have suggested that differences in metabolic capacity may explain why children may be better or less able to deal with toxic chemicals than adults (Cresteil, 1998; Dorne *et al.*, 2005). Alcorn and McNamara, 2003, observed that the presence of alkaline pH (6 – 8) in neonatal for instance compared with the acidic pH (1 – 3) in adults has implications for the toxicokinetics of toxic compounds in neonatals. The alkaline pH promotes the bioavailability of weakly basic compounds but reduced bioavailability of weakly acidic compounds.

The age of humans can determine how compounds are physiologically distributed in the body (Kearns *et al*., 2003). Children are able to deal with water-soluble toxic compounds better than adults due to the presence of higher volumes of extracellular fluid (Alcorn and McNamara, 2003). However, in the case of fat soluble toxic compounds, children are less able to detoxify than adults. This is because serum lipoproteins which help in the elimination of fat soluble toxic compounds such as OC pesticides are less abundant in children than in adults. In general, it appears that the

toxicokinetics (absorption, distribution, biotransformation, and excretion) of toxic compounds occur a lower rate in children than in adults (Clewell *et al.*, 2002).

Data from experimental studies have suggested that exposure to organophosphorous pesticides could impair the developing brain during pregnancy and childhood (Eskenazi e*t al.*, 1999; Garcia *et al.* 2003). The peculiar nature of children to chemical exposure supports the need for the utilization of risk assessment paradigm which adequately addresses their special vulnerabilities (Daston *et al.*, 2004).

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2.6 Risk Assessment

Food safety risk assessment is the scientific evaluation of known or potential adverse health effects resulting from human exposure to foodborne hazards. The process is one of the components of risk analysis. Risk assessment consists of the following steps: hazard identification, hazard characterization, risk assessment, and risk characterization (WHO, 2014).

Food safety risk assessment is a very important component of risk analysis. This is because the outcomes from the risk assessment process, form the basis for making policy decisions used to protect consumers from foodborne hazards. Thus in order to arrive at practical risk management options, that adequately protects consumers, risk assessments must be based on realistic exposure scenarios. (FAO/WHO, 2014). Such scenarios must include consideration to the impact of foodborne hazards especially on susceptible and high-risk populations (e.g. children, pregnant women, and the elderly), taking into account acute, chronic, cumulative, and/or combined adverse health effects.

2.6.1 Hazard Identification

Hazard identification is defined as the identification of the type and nature of adverse effects that an agent has an inherent capacity to cause in an organism, system, or (sub) population. Hazard identification is the first stage in hazard assessment and the first of four steps in risk assessment (IPCS, 2004).

Food chemical hazard identification deals with identifying potential adverse health effects in humans associated with exposure to a chemical, the likelihood of such effects occurring and certainty or uncertainty associated with such effects and the circumstances under which the hazard may be expressed (WHO, 1995).

Hazard identification is conducted using the weight-of-evidence approach. This requires review of all available scientific information from epidemiological studies, animal toxicological studies, in vitro assays and, lastly, quantitative structure-activity relationships (WHO, 1995).

Pesticides are known chemical agents that can cause adverse health effect in humans and have been classified according to the level of toxicity of the technical compound and its formulation. The pesticide classes are: extremely hazardous pesticides, highly hazardous pesticides, moderately hazardous pesticides, slightly hazardous pesticides and pesticides which are unlikely to present acute hazard (WHO, 2009).

2.6.2 Hazard Characterization

Hazard characterization is defined as the qualitative and, wherever possible, quantitative description of the inherent properties of an agent or situation having the

potential to cause adverse effects. This should, where possible, include a dose–response assessment and its attendant uncertainties. Hazard characterization is the second stage in the process of hazard assessment and the second of four steps in risk assessment (IPCS, 2004). Exposure of humans to a food chemical or administration of dose of a chemical in the case of animal studies, may elicit onset of adverse health effect at a given dose. The primary purpose of risk characterization in food safety risk assessment is to establish the dose – response relationship.

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Exposure to pesticides generally results in adverse health effect, i.e. there exist a threshold concentration where effect is observed. In such situations, establishing health-based guidance values such as ADI or ARfD for the pesticide residues must be a key outcome of the risk characterization process (FAO/WHO, 2009).

2.6.3 Exposure Assessment

Exposure is the coming into contact (at visible external boundaries) of an individual with a pollutant for specific durations of time and it is expressed as mass per unit time (e.g. $mgkg^{-1}$ of body weight per day) (IPCS, 2004.2a). Exposure assessments should depict real-life situations, taking into consideration the identification of potentially exposed populations; identification of potential exposure pathways; and quantification of the magnitude, frequency, duration and time-pattern of contact with a chemical (potential doses) (Cohen *et al.*, 2000).

Exposure can occur through multiple pathways and routes (Clayton *et al.*, 2003). Depending on the life stage of the child, exposure media can include amniotic fluid, breast milk, air, water, soil/dust/sediments, food, and objects/surfaces. Dietary intake

accounts for the major source of pesticide exposure for infants and children (NRC, 1993; Clayton *et al.*, 2003). Exposure routes include transplacental transfer, inhalation, ingestion, dermal absorption, and indirect (non-dietary) ingestion (WHO, 2006).

2.6.3.1 Approaches for Measuring Exposure

Two approaches exist for conducting exposure assessment, namely direct (biologic) and indirect approach (environmental). The direct approach uses biological monitoring techniques to estimate a person's contact with a toxic compound in an exposure medium over a given duration and this reflects internal exposure (Cohen *et al.,* 2000). Various methodologies have been used for biomonitoring of pesticides. These include urinary monitoring (Barr *et al.,* 2005b; Kissel *et al.,* 2005), blood monitoring (Needham 2005) and other body fluids such as meconium (Whyatt and Barr, 2001), saliva (Lu *et al*., 1997), breast milk (Landrigan *et al.,* 2002; Sanghi *et al.,* 2003; Osei-Tutu *et al.,* 2013).

A major advantage of biologic monitoring over its environmental counterpart is that the concentration of measured toxicant is indicative of the cumulative effect of exposure from all exposure routes (WHO, 2006). The lack of validated biomarkers for most toxicants, the invasive nature of toxicant and the expensive nature of the direct approach however, limits its use.

The indirect approach for exposure measurement depends on the premise that toxic compounds are distributed in the environment and that humans are exposed to different environmental media contaminated with these toxicants. Studies exist on

monitoring toxic compounds in various environmental media. Pesticides have been measured in air samples (Clayton *et al.,* 2003), water (Rajendran and Subramanian, 1997., Albanis *et al*., 1998) and food (Kannan *et al.,* 1997, Bai *et al*., 2006; Adu-Kumi *et al.,* 2010; Bempa *et al.,* 2011; Akoto *et al.,* 2013).

Since the indirect approach uses environmental media, monitoring could be used to estimate concentrations of actual parent compound of toxicants and/or their metabolite depending on when sampling is done. Measurement of parent compound of toxicants is however, not the main focus of biologic monitoring since such compounds are mostly bio transformed into their metabolites (biomarkers) in the body. Coupled with the expensive nature of biologic monitoring (direct approach) the utility of environmental monitoring (indirect approach) for exposure measurement is the preferred approach for food safety regulatory purposes. The indirect approach has been used extensively for assessing the safety of food.

2.6.3.2 Food Consumption Estimates

Food consumption is an estimate of the daily average per capita quantity of a food or group of foods consumed by a specified population (WHO, 1997) and is used for estimating long term-hazards. Food consumption is expressed in grammes of food per person per day. Different population groups exhibit variable food consumption patterns thus different consumption data are required for assessing dietary exposure to food chemicals. Infants and young children have higher food consumption per kilogram body weight and as a result have higher estimated exposure levels, which in most cases, is higher than that estimated for all other age groups (EFSA, 2009). This makes infants and young children a prime focus for exposure assessment studies.

Two classes of method exist for assessing food consumption; the prospective methods which record data at the time of eating and the retrospective methods which use data about the food eaten over a specified duration of time (EFSA, 2009).

2.6.3.3 Prospective Method for Estimation of Food Consumption

The prospective methods comprise the dietary records and dietary recall techniques. In the dietary records technique, food must be measured before eating to enable quantification of actual amount consumed. This method requires that respondents report all food consumed during a specified period which is usually 7 days or less (IPCS, 2009). Although this method may provide exact amount of food consumed, weighing food for more than four consecutive days may elicit respondent fatigue which may lead to errors in quantification (Gersovitz *et al.,* 1978).

Portion sizes can be used in situations where weighing may interfere with eating habits such as eating in a restaurant (Van Staveren and Ocke, 2006). Based on the portion size suggested on commercial baby foods, and the recommended meal daily frequency by WHO, Piccinelli *et al*., 2013, estimated the daily food consumption for infants aged 1 – 9 months.

The dietary recall method or duplicate diet studies collects consumption data based on food intake for the immediate 24 or 48 hours, with the 24-hour dietary recall of duplicate diet samples being the most commonly used (EFSA, 2009). This method uses photographs, food models or household measure to quantify food intake. Duplicate diet studies are valid only for the period of study and may only be used in acute exposure estimations.

2.6.3.4 Retrospective Method for Estimation of Food Consumption

Retrospective methods which are commonly used to collect food consumption data are the diet history and food frequency questionnaire (FFQ) (IPCS, 2009). The dietary history approach involves interviewing subjects to obtain information on daily food consumed over a period of time which may vary from past month or six months to one year (EFSA 2009, IPCS, 2009). Data collected using this method reflects whole diet. On the other hand, FFQs are designed to collect dietary information based on a restricted list of food that respondents have to estimate the rate of consumption over a defined period of time (Haraldsdottir *et al*., 2001).

Thompson and Subar, 2008, have suggested that a limitation of both retrospective methods is that intake is often misreported because of difficulties involved in remembering what has been consumed due to the long study periods.

2.6.3.5 Estimation of Dietary Exposure of Pesticide Residues

Exposure to pesticide is estimated by combining the concentration of the pesticide residues in food (expressed in $mgkg^{-1}$) and the food consumption rate (expressed as kg/day/body weight). Dietary exposure can be estimated in terms of theoretical maximum daily intake (TMDI) or estimated daily intake (EDI). The TMDI uses internationally or nationally established MRLs of pesticides to estimate dietary exposure whilst the EDI approach uses actual residue concentration hence is recognized as more realistic.

Using MRLs to estimate the TMDI result in overestimation of actual pesticide residue intake. This is because the approach assumes that the pesticide is present at

its highest concentration at the time of consumption (WHO, 1997). Despite its limitation, the TMDI approach is a good screening tool for estimating dietary exposure. Theoretical Maximum Daily Intake has been used to assess the chronic dietary risk to 275 pesticides in Brazil. In this study, eight compounds had TMDI greater than the ADI, signifying the possibility for an adverse effect. Out of eight compounds whose TMDI exceeded the ADI five were organophosphorous insecticides namely prothiophos, ethion, fenitrothion, methidathion, and dimethoate. Citrus, tomato, rice, and beans were the commodities that most contributed to the intake (Caldas and Souza, 2004).

Chun and Kang, 2003, has evaluated TMDI and estimated daily intake (EDI) for Koreans by using MRLs, food factors, residue data, and correction factors, and compared with the ADI in order to estimate the health risk based on pesticide exposure. In a study by Fromberg *et al*., 2011, the dietary intake of PCB and organochlorine pesticides were estimated for children and adults. Children had a relatively higher intake from milk and milk products.

In Ghana dietary exposure to organochlorine, organophosphorous and synthetic pyrethroids pesticide residues in maize and cowpea have been estimated. The EDI for heptachlor, dieldrin, endrin, β-endosulfan, γ-chlordane and chlorfenvinphos in maize exceeded the ADI. Whilst the levels of heptachlor and *p,p*'-DDD found in cowpea also exceeded the ADI (Akoto *et al.,* 2013).

2.7 Risk Characterization

Risk characterization is the fourth and final step of the risk assessment process. According to the IPCS, risk characterization is the qualitative and, wherever possible, quantitative determination, including attendant uncertainties, of the probability of occurrence of known and potential adverse effects of an agent in a given organism, system, or (sub) population, under defined exposure conditions (IPCS, 2004).

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Risk characterization combines the results from the hazard characterization and the exposure assessment to estimate the potential risk to human health under different exposure scenarios including any assumptions (Renwick *et al*., 2005). The FAO/WHO recommends that as far as possible the risk characterization outcome should include qualitative and/or quantitative information to enable the formulation of risk management decisions (FAO/WHO, 2009).

a) Qualitative information may include:

i. A statement or evidence of the toxicological significance or otherwise of the chemical given the different scenarios of exposure;

ii. statements or evidence of the safety of the chemical within the framework of specified uses; and

iii. recommendations to avoid minimize or reduce exposure.

b) Quantitative information may include:

i. a comparison of dietary exposures with health-based guidance values;

ii. estimates of risks at different levels of dietary exposure;

iii. risks at minimum and maximum dietary intakes (e.g. nutrients); and

iv. margins of exposure (FAO/WHO, 2009).

The final risk characterization step involves comparison of the EDI or TMDI with the health-based toxicological value i.e. ADI, ARfD or Cancer Benchmark Concentration (CBC) in the case of chronic exposure, acute exposure or carcinogenic risk respectively. The concept of Hazard Index (HI) which is a measure of the ratio of the exposure to the health-based toxicological value (also referred to as hazard quotient) has been used in several studies to characterize risk (Chun and Kang, 2003; Fromberg *et al.*, 2011; Hong-Sheng *et al.*, 2011; Jang *et al.*, 2011; Akoto *et al.*, 2013). If the HI exceeds 1, the compound has exceeded the maximum acceptable level (e.g. ADI or ARfD) and there might be a risk (US EPA, 2000). The HI concept can be used for single compounds as well as mixtures of compounds. However, in the case of the later, the sum of hazard quotient (HQ) of the individual compounds in the mixture defines the HI.

2.7.1 Approaches in Food Chemical Risk Characterization

2.7.1.1 Carcinogenic and Non-Carcinogenic Risk

Risk can be characterized based on whether the pesticide being evaluated causes cancer (carcinogenic or oncogenic risk assessment) or does not cause cancer (non-carcinogenic risk assessment). Carcinogenic risk assessment is used for chemicals which are capable of producing toxicity at any level resulting in malignant or benign tumors. Non-carcinogenic risk is applied based on the assumption that, chemicals have toxicity threshold below which there is no significant health effect (WHO, 1995).

Wang *et al.*, 2011, estimated the human risk of OC pesticides using the concept of carcinogenic risk. According to this study, the carcinogenic hazard ratio for most investigated OC pesticides in vegetable and fish exceeded 1, signify the possibility of adverse health effect as a result of dietary exposure to OC pesticides. In another study, the dietary oncogenic risk of three pesticides (cypermethrin, chlorothalonil and parathion) was assessed from TMDI and EDI values. Although the level from TMDI exceeded the risk level ($1x10^{-6}$) of US EPA, the level from adjusted EDI, was below the risk level (Chun and Kang, 2003).

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2.7.1.2 Risk assessment for mixtures of compounds

Traditionally, risk assessment of chemicals in food has been based on toxicological evaluation of single compounds. However, in practical terms, humans are concurrently exposed to multiple chemicals through dietary and/or environmental exposures (FSA, 2012). Concerns have therefore been raised about the possible adverse health effect to humans if these chemicals interact simultaneously. When multiple chemicals act jointly in this way, the interaction could affect the overall toxicity which could lead to a lowering or increase in toxic effects (EC, 2012).

Current approaches and future strategies for risk assessment of mixtures of pesticides have been reviewed by Reffstrup *et al.* (2010). The Authors explain that two main possibilities may occur in mixtures of compounds. They are no interaction in the form of simple similar action (dose addition) or simple dissimilar action (response addition) or combined effect with interaction (antagonism, synergism).

A number of methods exist for estimating cumulative toxicity of chemicals in food. These methods are all inter-related, but some are mathematically more complex than others. The most useful methods, in increasing levels of complexity and refinement, are the hazard index, the reference point index, the relative potency factor method and physiologically based toxicokinetic modeling, although this last method would only be considered should a highly refined assessment be necessary (Boobis *et al.*, 2008).

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Several initiatives have been taken to develop principles that adequately address risk assessment of mixtures of compounds (Krishnan *et al.*, 1997; US EPA, 2000; EFSA, 2008). However, Reffstrup *et al.* (2010), argue that there appears to be uncertainties in the proposed approaches. They however suggest that where a common mechanism exists for a group of compounds and adequate data exist; cumulative risk assessment for the group can be performed assuming the compounds in the group operate by a simple similar action. In practice the HI based on the reference value i.e. ADI or ARfD would normally be sufficient (Reffstrup *et al.*, 2010). Among the classes of pesticides the OPs exhibit a common mechanism of action i.e. inhibition of acetylcholinesterase. Cumulative risk assessments have been conducted for OP pesticides (US EPA, 2002; Bosgra *et al.*, 2009).

2.8 Analytical Methods for Pesticide Residues in Cereal-based Complementary Foods

Considering the potential adverse health effects of pesticides exposure, it is important to regularly monitor the concentration of pesticide residues in food. Effective monitoring depends on the sensitivity of analytical method especially when

some residues occur in very low concentrations. The method used for such analysis must be adequately validated.

Different methods have been used for the extraction of pesticides in cereals. Schenck *et al.*, 2002, determined OP and OC pesticide residues in low moisture, non-fatty products using a solid phase extraction cleanup and gas chromatography. The main extraction solvent for this work was acetonitrile. Acetonitrile extraction followed by dispersive solid phase extraction employing bulk sorbent such as primary secondary amine (PSA) or graphitized carbon black (GCB), has been developed for pesticide residue determination in low fat produce (Anastassiades, 2003). This method is commonly referred to as the Quick, Easy, Cheap, Effective, Rugged, and Safe (QuEChERs) method.

Other studies have modified the QuEChERs method for determination of pesticide residues in food and feed including cereals and animal feed (Walorczyk, 2007) and wheat flour (Paya *et al.*, 2007). Currently, detection and quantification of pesticide residues in food for infant and young children is achieved using analytical instruments such as LC-MS/MS, GC-MS/MS. However, GC-MS and LC-MS can also be used.

CHAPTER THREE

3.0 MATERIALS AND METHODS

3.1 MATERIALS

3.1.1 Certified Reference Pesticides Standards

The following 36 certified pesticides, obtained from were studied.

3.1.1.1 Organochlorine (OC) Pesticides

Fourteen (14) certified OC pesticides used in this work were obtained from Dr. Ehrenstorfer Laboratories (GmbH Germany). They are: β-HCH, γ-HCH, δ-HCH, heptachlor, γ-chlordane, α-endosulfan, β-endosulfan, endosulfan sulfate, *p,p*'-DDE, *p,p'*-DDT, methoxychlor, aldrin, dieldrin and endrin,

3.1.1.2 Organophosphorous (OP) Pesticides

The following thirteen (13) certified OP pesticides were obtained from Dr Ehrenstorfer Laboratories (GmbH Germany): Methamidophos, ethoprophos, phorate, diazinon, fonofos, dimethoate, pirimiphos-methyl, chlorpyrifos, malathion, fenitrothion, parathion, chlorfenvinphos and profenofos.

3.1.1.3 Synthetic pyrethroids

The following nine (9) certified synthetic pyrethroids pesticides were obtained from Dr Ehrenstorfer Laboratories (GmbH Germany): Bifenthrin, allethrin, fenpropathrin, λ-cyhalothrin, permethrin, cyfluthrin, cypermethrin, fenvalerate and deltamethrin.

3.1.2 Other Reagents and Chemicals

Pesticide grade acetonitrile, analytical grade sodium chloride, disodium hydrogencitrate sesquihydrate, magnesium sulphate, trisodium citrate dehydrate, formic acid conc. (>95%) were obtained from BDH Laboratory Supplies, England. Sorbent Bondesil-PSA (Primary Secondary Amine) 40µm was obtained from Varian, USA.

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3.1.3 Equipment

The following are key equipment used in this work:

Centrifuge Cri multifunction (Thermo Electron Industries SAS, France), Metler Toledo PG 10035 weighing balance, rotary evaporator Buchi RE-200 equipped with Buchi B740 re-circulating water chiller and Buchi V700 vacuum pump (BÜchi Labortechnic AG Postfach Switzerland). A gas chromatograph, Varian CP-3800 (Varian Association Inc. USA) equipped with 63Ni electron capture detector (ECD), CTC Analytic Combi PAL autosampler, split-splitless injector, programmed pneumatic control (PPC) and a computer running star workstation data processor.

3.2 METHOD

3.2.1 Sampling

Ten brands of processed cereal-based complementary foods were sampled from local markets, shops and supermarkets in 4 districts in the Greater Accra region. The districts comprised the Accra Metropolitan District, Ga West Municipal District, La Nkwantanang Madina District and La Dade Kotopon Municipal District.

Metro, Ghana. A sample size of ten (10) different brands of processed cereal-based complementary foods was used for in this work. The ten brands comprised five (5)

locally produced and five (5) imported samples. For each brand, three (3) samples of the same batch were analyzed separately in triplicates. The mean concentration for each brand was done calculated.

The samples were stored at a temperature of -18 ^{o}C until analysis was performed. The processed cereal-based complementary foods analyzed herein after will be referred to as baby food to simplify presentation. The names of the ten baby foods were designated as baby food A, B, C, D, E, F, G, H, I and J.

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3.2.2 Preparation of Mixed Pesticide Reference Standards for Fortification and Instrument Calibration

Pesticides standard stock solutions (1000 µg/mL) of each certified OP, OC and synthetic pyrethroid pesticides were prepared. Mixed OP solutions and mixed OC and synthetic pyrethroid solutions were prepared from the stock solution by volumetric dilutions and used as fortification standards in the procedural recovery process, and as calibration standards in instrument calibration.

3.2.3 Extraction and Clean-Up

The method described by Anastassiades *et al.*, (2003) was used for extraction of the pesticides with slight modification. Triplicates of each brand of baby food having the same batch number were analyzed. For each batch of analysis, reagent blanks and spiked samples at fortification level of 0.05 mg kg^{-1} were used for quality control checks.

For each batch of analysis, 5 g analytical portion of the homogenous cereal-based baby food was weighed into 50 ml polypropylene (PP) centrifuge tube. Ten

milliliters (10 mL) of distilled water kept at 4 ^{o}C was added to the food sample. Addition of salts in the next stage of the method produces heat, hence the use of low temperature water compensates for the heat development. The mixture was shaken for 1 min. using a vortex mixer to disperse solvent and pesticides evenly throughout the sample. Ten milliliters (10 mL) of acetonitrile was then added to the content of the PP centrifuge. The mixture was shaken for 1 min.

To the acetonitrile-based mixture, a salt mixture containing 0.5 g disodium hydrogencitrate sesquihydrate, 1 g sodium chloride, 1 g trisodium citrate dihydrate and 4 g anhydrous magnesium sulphate was added and the mixture vigorously shaken for 1 min. The sample was then centrifuged at 3000 rpm for 5 min. After the centrifugation, an aliquot of 8 mL of the supernatant acetonitrile phase was taken and transferred into a PP centrifuge tube and stored for at least 1.5 hrs in the freezer. Freezing-out helped remove some additional co-extractives with limited solubility in acetonitrile, while the major part of fat and waxes solidify and precipitate.

3.2.4 Dispersive Solid Phase Extraction

Six milliliters (6 mL) of the cold acetonitrile phase (cleaned extract) was transferred into clean PP centrifuge tube containing 150 mg of Primary Secondary Amine (PSA) and 900 mg of $MgSO_4$ and the tube was shaken vigorously for 30 sec and centrifuged for 5 min at 3000 rpm. An aliquot of 4 mL of the cleaned extract was then transferred into a round bottom flask and the pH quickly adjusted to 5 by adding 40 µL of 5 % formic acid solution in acetonitrile (v/v). PSA treated extracts were acidified by adding a small amount of formic acid, to improve the storage stability of certain base-sensitive pesticides. The filtrate was concentrated below 40 ºC on the rotary

evaporator just to dryness. The concentrated extract was then re-dissolved in 1 mL ethyl acetate and the extract transferred into a 2 mL standard opening vial for quantitation by Gas Chromatograph Electron Capture Detector (GC-ECD) in the case of OC pesticides and synthetic pyrethroid pesticides and Gas Chromatograph-Pulsed Flame Photometric Detector (GC/PFPD) for OP pesticides.

3.2.5 Quality Control

All reagents used during the analysis were exposed to the same extraction procedures. Solvents used were run to verify for any interfering substances within the runtime. In all batches of pesticide residues analysis, reagent blanks and samples to be analyzed were fortified with mixed OP, OC and synthetic pyrethroid standards for quality control checks. All the samples were analyzed in triplicates. No pesticides were detected in the reagent blanks when analyzed on the GC. All extracts were kept frozen until quantification was achieved.

Procedural recoveries were analyzed concurrently with each batch of analytical extracts. Fortification level of 0.05 mg/kg was chosen based on the limit of determination of the pesticides being analyzed. Calibration curves were run with each batch of samples to check that the correlation coefficient was kept above r^2=0.99.

3.2.6 Analysis of Pesticide Residue Content

Gas chromatograph equipped with Electron Capture Detector (ECD) and Pulsed Flame Photometric Detector (PFPD) was checked for limit of detection. The limit of detection for OP, OC and synthetic pyrethroid pesticides was 0.001 $mgkg^{-1}$.

Organophosphorous pesticides were separated and quantified using Varian CP-3800 gas chromatograph with a CombiPAL Autosampler equipped with pulsed flame photometric detector (PFPD) on 30 m x 0.25 mm internal diameter fused silica capillary column coated with VF- 1701 (0.25 μm film). The column oven temperature was performed as follows: initial temperature was 70 ^{o}C, and increased to 200 ^{o}C at the rate of 25 ^{o}Cmin^{-1} then ramped to 250 ^{o}C at the rate of 20 ^{o}Cmin^{-1}, keeping the final temperature for 2 min. The carrier gas was nitrogen gas at the flow rate of 2 mLmin^{-1}. The injector and detector temperatures were maintained at 250 ^{o}C and 280 ^{o}C, respectively. The injector volume of the gas chromatograph was 2.0 μL.

Separation and Quantification of OC and synthetic pyrethroid pesticides were carried out using Varian CP-3800 gas chromatograph with a CombiPAL Autosampler equipped with an Electron Capture Detector (ECD, ^{63}Ni), on 30m+10mEZ Guard x 0.25 mm internal diameter fused silica capillary. Column coated with VF-5 ms (0.25 μm film). The column oven temperature was set from 70 ^{o}C, held for 2 min to 180 ^{o}C at a rate of 25 ^{o}Cmin^{-1}, and then from 180 ^{o}C to 300 ^{o}C at a rate of 5 ^{o}Cmin^{-1}. Purified nitrogen gas was used as carrier gas at the flow rate of 1.0 mLmin^{-1} and make up gas of 29 mLmin^{-1}. The injector and detector temperatures were maintained at 270 ^{o}C and 300 ^{o}C, respectively. The injector volume of the gas chromatograph was 1.0 μL.

Pesticide residues in the extracts were identified when the retention times matched those of the reference standards. Quantification was achieved by comparing sample peak areas with those of the reference standards under the same conditions. Each sample was analyzed three times and the mean values calculated.

3.2.7 Exposure Assessment

3.2.7.1 Food Consumption

The consumption rate for each brand of processed cereal-based complementary food was calculated using the recommended serving size according to the label on the sampled products (Picinelli *et al*., 2010).

The meal frequency was obtained from the WHO recommended meal frequency, assuming an average healthy breastfed infant (WHO, 2002). The appropriate number of feedings depends on the energy density of the local foods and the usual amounts consumed at each feeding. The WHO recommends a serving frequency of 2 – 3 serving per day for infants aged 6 – 8 months; and 3 – 4 servings per day for both infants aged 8 – 11 months and young children aged 12 – 24 months. Food products which were sampled were to be consumed from six (6) months onward with the exception of baby food G which was recommended only for young children. In the calculation of the consumption rate, serving frequency of four (4) depicting worst case scenario was used for all food products.

3.2.7.2 Body Weight Estimation

The average body weight of infants aged between 6 – 11 months and young children aged 12 – 24 months was estimated as shown in Table 3.1 and Table 3.2

Table 3.1: Average weight for infants aged 6 – 11 months

Age (months)	Average of the 50th percentile weight (kg)[a]		Monthly average weight (kg)
	Female	Male	
6	7.3	7.9	7.6
7	7.6	8.3	8.0
8	8.0	8.6	8.3
9	8.2	8.9	8.6
10	8.5	9.2	8.9
11	8.7	9.4	9.1
Average weight of infants			8.4

[a] Weights were obtained from the Multicentre Growth Reference Study Group, World Health Organization (2006).

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Table 3.2: Average weight for young children aged 12 – 24 months

Age (months)	Average of the 50th percentile weight (kg)[a]		Monthly average weight (kg)
	Female	Male	
12	8.9	9.7	9.3
13	9.2	9.9	9.6
14	9.4	10.1	9.8
15	9.6	10.3	10.0
16	9.8	10.5	10.2
17	10.0	10.7	10.4
18	10.2	11.0	10.6
19	10.4	11.2	10.8
20	10.6	11.4	11.0
21	10.9	11.6	11.2
22	11.1	11.8	11.4
23	11.3	12.0	11.7
24	11.5	12.1	11.8
Average weight of young children			10.6

[a] Weights were obtained from the Multicentre Growth Reference Study Group, World Health Organization (2006).

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3.2.7.3 Consumption Rate Estimation

The consumption of processed cereal-based complementary food per body weight for each category was calculated based on formula 1 below. The results of the estimation are presented in Table 4.3.

$$CR = W \times F \quad \ldots\ldots\ldots\ldots\ldots 1$$

Where CR is the consumption rate in (kgd^{-1}), W is the weight of food per serving and F is the meal frequency per day.

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3.6.3 Estimation of Dietary Exposure

The Estimated Daily Intake (EDI) also known as the exposure level for the pesticides detected in the various processed cereal-based complementary food was calculated for each age category based on the formula 2.

$$EDI = \frac{C \times CR}{BW} \quad \ldots\ldots\ldots 2$$

Where EDI is the estimated daily intake (mg kg^{-1} d^{-1}), C is the mean concentration of pesticide residues (mg kg^{-1}), CR is the consumption rate in (kg d^{-1}) and BW is the average body weight in (kg).

3.7 Risk Characterization

Risk of pesticides to infants and young children through daily food intakes were characterized based on the guidelines recommended by the US EPA (2005). For non-carcinogenic effects, EDI was compared with the ADI for each pesticide detected in the food samples. The comparison was done by finding the ratio of EDI to ADI. The results obtained were interpreted as follows: If the ratio is less than 1.0 it can be concluded with great certainty that there is essentially no probability of population or community level effect. However, the ratio exceeds 1.0 the potential for adverse

effects is indicated but not demonstrated. Within the context of risk assessment, a ratio which is greater than 1.0 indicates the need for further evaluation of specific issues surrounding chemical exposure and toxic potency (Giesy *et al*., 2000).

For the carcinogenic effects, the hazard ratios (HRs) were calculated by the formula (3) below (Dougherty *et al.*, 2000). Where EDI is the estimated daily intake and CBC is the Cancer Benchmark Concentration calculated using formula 3:

$$CBC = \frac{(RL/OSF) \times BW}{CR} \ldots\ldots\ldots 3$$

RL is the maximum acceptable risk level (1×10^{-6}, dimensionless), OSF is the Oral Slope Factor (mg kg^{-1} d^{-1}), BW is the body weight (kg) and CR is the consumption rate (kg d^{-1}). The CBC for carcinogenic effect is derived by setting the risk to one in one million due to lifetime exposure. The OSFs for the pesticides were obtained from US EPA, 2014. The average body weight used in the calculation is based on the WHO recommended average body weight for infants (8.4 kg) and young children (10.6 kg).

3.8 Data Analysis

Data was subjected to analysis using MS-Excel (2007). Descriptive statistics utilized included mean, range, minimum, maximum and standard deviation.

CHAPTER FOUR

4.0 RESULTS

4.1 Quality Control

Quality of analysis of pesticide residues was assured through the analysis of solvent blanks, spikes and triplicate samples. The solvent blanks were used to check for interferences from reagents, whilst the spike samples were used to determine recovery which is an indicator of methods performance and accuracy. Triplicate samples were used to confirm method precision. Recoveries ranged between 70 – 94 % for organophosphorous pesticide residues, organochlorine pesticide residues were recovered in the range of 69 – 119 % and 73 – 100 % for synthetic pyrethroid pesticide residues for all ten brands of processed cereal-based complementary foods. The results indicate acceptable method performance.

4.2 Pesticide Residues in Processed Cereal-based Complementary Food Samples

All ten brands of baby food samples were analyzed for a total of 36 pesticide comprising 13 organophoshorous pesticides (OPs) (methamidophos, ethoprophos, phorate, diazinon, fonofos, dimethoate, pirimiphos-methyl, chlorpyrifos, malathion, fenitrothion, parathion, chlorfenvinphos, profenofos), 14 organochlorine pesticides (OCs) (β-HCH, γ-HCH, δ-HCH, heptachlor, aldrin, γ-chlordane, *p,p*'-DDE, *p,p*'-DDT, dieldrin, endrin, α-endosulfan, β-endosulfan, endosulfan sulfate and methoxychlor), and 9 synthetic pyrethroid pesticides (allethrin, bifenthrin, fenpropathrin, λ-cyhalothrin, permethrin, cyfluthrin, cypermethrin, fenvalerate, deltamethrin).

4.2.1 Organophosphorous Pesticide Residues in Processed Cereal-based Complementary Food

Residues of nine (9) OP pesticides were detected in six (6) samples. The mean concentrations of OP pesticide residues in the ten brands of processed cereal-based complementary food are shown in Table 4.2a and 4.2b. The results indicate that mean concentration ranged from 0.001 $mgkg^{-1}$ for fenitrothion in baby food G to 0.026 $mgkg^{-1}$ for methamidophos in baby food F.

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Six out of 10 baby foods were contaminated with at least one OP pesticide. The implicated samples were baby food C, E, F, G, H and I. Two of the six contaminated baby foods (C and E) were locally produced in Ghana whilst the remaining four were imported products. No OP pesticide residues were detected in 3 of the locally produced baby foods (baby food A, B and D) and one of the imported baby food (baby food J).

The mean concentrations of methamidophos, chlorpyrifos and malathion in baby food C were 0.003±0.001 $mgkg^{-1}$, 0.014±0.002 $mgkg^{-1}$ and 0.003±0.001 $mgkg^{-1}$ respectively. Pirimiphos-methyl, chlorpyrifos 0.006±0.001 $mgkg^{-1}$ and fenitrothion were detected at the following respective concentrations in baby food E, 0.008±0.002 $mgkg^{-1}$, 0.018±0.002 $mgkg^{-1}$ as presented in Table 4.2a. Methamidophos was detected at mean level of 0.026±0.009 $mgkg^{-1}$ in baby food F. Six OPs were detected in baby food G, the mean levels were 0.005±0.003 $mgkg^{-1}$ for methamidophos, 0.003±0.001 $mgkg^{-1}$ for ethoprophos, 0.002±0.001 $mgkg^{-1}$ for diazinon, 0.003±0.001 $mgkg^{-1}$ for dimethoate, 0.007±0.001 $mgkg^{-1}$ for pirimiphos-methyl and 0.001±0.001 $mgkg^{-1}$ for fentrothion as presented in table 4.2b. Table 4.2b also shows that the only

OP pesticide detected in sample H was Chlorfenvinphos at mean concentrations of 0.005±0.001 $mgkg^{-1}$. Pirimiphos-methyl and fenitrothion were detected in sample J at mean concentration of 0.024±0.007 $mgkg^{-1}$ and 0.018±0.007 $mgkg^{-1}$ respectively as presented in Table 4.2b.

The number of individual OPs detected in baby food samples decreased in the following order Sample G (6), Sample C (3), Sample E (3), Sample I (2), Sample F (1) and Sample H (1). The remaining samples did not record detected amount of any of the OPs measured in this work.

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Table 4.2a: Range, mean and standard deviation of OP pesticide residues in processed cereal-based complementary food

Pesticides	Baby Food A		Baby Food B		Baby Food C		Baby Food D		Baby Food E	
	Range ($mgkg^{-1}$)	Mean±SD ($mgkg^{-1}$)	Range ($mgkg^{-1}$)	Mean±SD ($mgkg^{-1}$)	Range ($mgkg^{-1}$)	Mean±SD ($mgkg^{-1}$)	Range ($mgkg^{-1}$)	Mean±SD ($mgkg^{-1}$)	Range ($mgkg^{-1}$)	Mean±SD ($mgkg^{-1}$)
Methamidophos	ND	ND	ND	ND	0.001-0.006	0.003±0.001	ND	ND	ND	ND
Ethoprophos	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND
Phorate	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND
Diazinon	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND
Fonofos	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND
Dimethoate	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND
Pirimiphos-methyl	ND	ND	ND	ND	ND	ND	ND	ND	0.011-0.014	0.004 ±0.014
Chlorpyrifos	ND	ND	ND	ND	0.014-0.017	0.014±0.002	ND	ND	0.003-0.01	0.006 ±0.001
Malathion	ND	ND	ND	ND	0.002-0.005	0.003±0.001	ND	ND	ND	ND
Fenitrothion	ND	ND	ND	ND	ND	ND	ND	ND	0.005-0.033	0.018 ±0.002
Parathion	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND
Chlorfenvinphos	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND
Profenofos	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND

SD = standard deviation

ND = not detected.

Limit of detection for organophosphorous pesticides= 0.001 $mgkg^{-1}$

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Table 4.2b: Range, mean and standard deviation of OP pesticide residues in processed cereal-based complementary food

Pesticides	Baby Food F		Baby Food G		Baby Food H		Baby Food I		Baby Food J	
	Range ($mgkg^{-1}$)	Mean±SD ($mgkg^{-1}$)	Range ($mgkg^{-1}$)	Mean±SD ($mgkg^{-1}$)	Range ($mgkg^{-1}$)	Mean±SD ($mgkg^{-1}$)	Range ($mgkg^{-1}$)	Mean±SD ($mgkg^{-1}$)	Range ($mgkg^{-1}$)	Mean±SD ($mgkg^{-1}$)
Methamidophos	0.007-0.06	0.026±0.009	0.001-0.008	0.005±0.003	ND	ND	ND	ND	ND	ND
Ethoprophos	ND	ND	0.001-0.006	0.003±0.001	ND	ND	ND	ND	ND	ND
Phorate	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND
Diazinon	ND	ND	0.001-0.004	0.003±0.001	ND	ND	ND	ND	ND	ND
Fonofos	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND
Dimethoate	ND	ND	0.001-0.004	0.003±0.001	ND	ND	ND	ND	ND	ND
Pirimiphos-methyl	ND	ND	0.005-0.010	0.007±0.001	ND	ND	0.017-0.036	0.024±0.007	ND	ND
Chlorpyrifos	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND
Malathion	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND
Fenitrothion	ND	ND	ND-0.005	0.001±0.001	ND	ND	0.014-0.032	0.018±0.007	ND	ND
Parathion	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND
Chlorfenvinphos	ND	ND	ND	ND	0.002-0.008	0.005±0.001	ND	ND	ND	ND
Profenofos	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND

SD = standard deviation

ND = not detected

Limit of detection for organophosphorous pesticides = 0.001 $mgkg^{-1}$

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4.2.2 Organochlorine Pesticide Residues in Processed Cereal-based Complementary Food

Residues of eight (8) out of the 14 OC pesticides were detected in 9 samples. Table 4.2c and 4.2d show the range, mean and standard deviation of OC residues in the ten brands of baby food used in this work. The mean concentration ranged from 0.002±0.001 $mgkg^{-1}$ in baby food F to 0.022±0.007 $mgkg^{-1}$ in baby food E.

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Organochlorine pesticide residues were detected in 9 samples comprising 5 locally produced baby food and 4 imported baby foods. Baby food J did not record any OC residues, however, baby food A to I were all contaminated with OCs at varying degrees.

Mean concentrations of β–HCH , γ–HCH and heptachlor in baby food A were 0.005±0.001 $mgkg^{-1}$, 0.008±0.002 $mgkg^{-1}$ and 0.002±0.001 $mgkg^{-1}$ respectively, whilst baby food B recorded γ–HCH, α–endosulfan β–endosulfan at mean levels of 0.006±0.002 $mgkg^{-1}$, 0.008±0.003 $mgkg^{-1}$, 0.021±0.018 $mgkg^{-1}$ respectively as shown in Table 4.2c.

Table 4.2c also shows that β –HCH, δ–HCH and α–endosulfan were detected in baby food C at mean levels of 0.017±0.001 $mgkg^{-1}$, 0.007±0.003 $mgkg^{-1}$ and 0.006±0.001 $mgkg^{-1}$ respectively. δ–HCH and γ–HCH were detected at mean levels of 0.008±0.002 $mgkg^{-1}$ and 0.014±0.014 $mgkg^{-1}$ respectively in baby food D. In baby food E, γ–HCH, δ–HCH, heptachlor and γ–chlordane were detected at the following levels 0.022±0.007 $mgkg^{-1}$, 0.007±0.001 $mgkg^{-1}$, 0.006±0.001 $mgkg^{-1}$ and 0.013±0.002 $mgkg^{-1}$ respectively.

Table 4.2c: Range, mean and standard deviation of OC pesticide residues in processed cereal-based complementary food

	Baby Food A		Baby Food B		Baby Food C		Baby Food D		Baby Food E	
Pesticides	Range ($mgkg^{-1}$)	Mean±SD ($mgkg^{-1}$)	Range ($mgkg^{-1}$)	Mean±SD ($mgkg^{-1}$)	Range ($mgkg^{-1}$)	Mean±SD ($mgkg^{-1}$)	Range ($mgkg^{-1}$)	Mean±SD ($mgkg^{-1}$)	Range ($mgkg^{-1}$)	Mean±SD ($mgkg^{-1}$)
β-HCH	0.004-0.008	0.005±0.001	ND	ND	ND-0.024	0.017±0.001	ND	ND	ND	ND
γ –HCH	0.006-0.023	0.008±0.001	0.004-0.008	0.006±0.002	ND	ND	0.004-0.05	0.014±0.014	0.009-0.032	0.022±0.007
δ –HCH	ND	ND	ND	ND	ND-0.013	0.007±0.003	ND-0.014	0.008±0.002	0.007-0.007	0.007±0.001
Heptachlor	0.003-0.003	0.003±0.001	ND	ND	ND	ND	ND	ND	0.006-0.006	0.006±0.001
Aldrin	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND
γ –chlordane	ND	ND	ND	ND	ND	ND	ND	ND	0.005-0.016	0.013±0.002
α –endosulfan	ND	ND	0.006-0.012	0.008±0.003	ND-0.006	0.006±0.001	ND	ND	ND	ND
β –endosulfan	ND	ND	ND-0.04	0.021±0.018	ND	ND	ND	ND	ND	ND
Endosulfan sulphate	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND
p,p'-DDE	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND
p,p'-DDT	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND
Methoxychlor	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND
Endrin	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND
Dieldrin	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND

SD = standard deviation

ND = not detected

Limit of detection for organochlorine pesticides = 0.001 $mgkg^{-1}$

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The mean levels for baby foods F to J are presented in Table 4.2d. Baby food F was contaminated with γ–HCH, heptachlor, *p,p'*-DDE and dieldren at concentrations of 0.002±0.001 $mgkg^{-1}$, 0.006±0.001 $mgkg^{-1}$, 0.013±0.001 $mgkg^{-1}$ and 0.005±0.003 $mgkg^{-1}$ respectively β–HCH, *p,p'*–DDE, dieldren and β–endosulfan were detected at mean levels of 0.005±0.001 $mgkg^{-1}$, 0.013±0.001 $mgkg^{-1}$, 0.012±0.014 $mgkg^{-1}$, 0.006±0.001 $mgkg^{-1}$ respectively in baby food G. Endrin, was detected in baby food H at 0.006±0.002 $mgkg^{-1}$ whilst β–HCH was detected at mean concentration of 0.014±0.001 $mgkg^{-1}$ in baby food I.

The number of individual OCs detected in baby food samples decreased in the following order Sample G (4), Sample F (4), Sample E (4), Sample A (3), Sample B (3), Sample C (3), Sample D (2), Sample H (1) and Sample I (1).

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Table 4.2d: Range, mean and standard deviation of OC pesticide residues in processed cereal-based complementary food

Pesticides	Baby Food F		Baby Food G		Baby Food H		Baby Food I		Baby Food J	
	Range ($mgkg^{-1}$)	Mean±SD ($mgkg^{-1}$)	Range ($mgkg^{-1}$)	Mean±SD ($mgkg^{-1}$)	Range ($mgkg^{-1}$)	Mean±SD ($mgkg^{-1}$)	Range ($mgkg^{-1}$)	Mean±SD ($mgkg^{-1}$)	Range ($mgkg^{-1}$)	Mean±SD ($mgkg^{-1}$)
β-HCH	ND	ND	0.004-0.008	0.005±0.001	ND	ND	0.013-0.019	0.014 ±0.001	ND	ND
γ –HCH	0.002-0.004	0.002±0.001	ND	ND	ND	ND	ND	ND	ND	ND
δ –HCH	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND
Heptachlor	ND-0.006	0.006±0.001	ND	ND	ND	ND	ND	ND	ND	ND
Aldrin	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND
γ –chlordane	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND
α –endosulfan	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND
β –endosulfan	ND	ND	0.006-0.013	0.006±0.001	ND	ND	ND	ND	ND	ND
Endosulfan sulphate	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND
p,p'-DDE	ND-0.013	0.013±0.001	ND-0.019	0.013±0.001	ND	ND	ND	ND	ND	ND
p,p'-DDT	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND
Methoxychlor	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND
Endrin	ND	ND	ND	ND	0.003-0.009	0.003±0.009	ND	ND	ND	ND
Dieldrin	0.003-0.006	0.005±0.003	0.013-0.016	0.012±0.002	ND	ND	ND	ND	ND	ND

SD = standard deviation

ND = not detected

Limit of detection for organochlorine pesticides = 0.001 $mgkg^{-1}$

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4.2.3 Synthetic Pyrethroids Pesticide Residues in Processed Cereal-based Complementary Food

Synthetic pyrethroids pesticide residues were detected in all the ten brands of baby food analyzed. Results in Table 4.2e and 4.2f indicate that mean concentration ranged between 0.002 $mgkg^{-1}$±0.001 in baby food A recorded for fenpropathrin and 0.017±0.004 $mgkg^{-1}$ in baby food D recorded for permethrin.

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Cyfluthrin, cypermethrin, fenvalerate, deltamethrin and allethrin were detected at mean concentrations of 0.006±0.001 $mgkg^{-1}$, 0.006±0.003 $mgkg^{-1}$, 0.006±0.001 $mgkg^{-1}$, 0.011±0.003 $mgkg^{-1}$ and 0.004±0.001 $mgkg^{-1}$ respectively for baby food A. The only synthetic pyrethroids pesticide detected in baby for B was allethrin at mean level of 0.004±0.001 $mgkg^{-1}$. Baby food C was contaminated with five out of the nine synthetic pyrethroids screened. The detected pesticides included bifenthrin, lambda-cyhalothrin, cypermethrin, fenvalerate and deltamethrin at levels of 0.009±0.001 $mgkg^{-1}$, 0.008±0.001 $mgkg^{-1}$, 0.009±0.003 $mgkg^{-1}$, 0.006±0.001 $mgkg^{-1}$, 0.007±0.000 $mgkg^{-1}$ as shown in Table 4.2e.

The mean concentrations of synthetic pyrethroids pesticides in baby food D as shown in Table 4.2e were as follows: Permethrin (0.006±0.001 $mgkg^{-1}$), cyfluthrin (0.004±0.001 $mgkg^{-1}$), cypermethrin (0.006±0.002 $mgkg^{-1}$), deltamethrin (0.004±0.001 $mgkg^{-1}$) and allethrin (0.012±0.001$mgkg^{-1}$).

Table 4.2e: Range, mean and standard deviation of synthetic pyrethroids pesticide residues in processed cereal-based complementary food

Pesticides	Baby Food A		Baby Food B		Baby Food C		Baby Food D		Baby Food E	
	Range ($mgkg^{-1}$)	Mean±SD ($mgkg^{-1}$)	Range ($mgkg^{-1}$)	Mean±SD ($mgkg^{-1}$)	Range ($mgkg^{-1}$)	Mean±SD ($mgkg^{-1}$)	Range ($mgkg^{-1}$)	Mean±SD ($mgkg^{-1}$)	Range ($mgkg^{-1}$)	Mean±SD ($mgkg^{-1}$)
Bifenthrin	ND	ND	ND	ND	0.005-0.014	0.009±0.001	ND	ND	ND	ND
Fenpropathrin	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND
Lambda-cyhalothrin	ND	ND	ND	ND	0.004-0.013	0.008±0.001	ND	ND	ND	ND
Permethrin	ND	ND	ND	ND	ND	ND	0.006-0.033	0.017±0.004	ND	ND
Cyfluthrin	0.004-0.001	0.006±0.001	ND	ND	ND	ND	0.004-0.004	0.004±0.001	ND-0.004	0.004±0.001
Cypermethrin	0.004-0.005	0.006±0.003	ND	ND	0.005-0.024	0.009±0.003	0.004-0.013	0.006±0.002	0.004-0.008	0.005±0.001
Fenvalerate	0.003-0.009	0.006±0.001	ND	ND	0.003-0.010	0.006±0.001	ND	ND	0.004-0.004	0.004±0.001
Deltamethrin	0.004-0.019	0.011±0.003	ND	ND	0.004-0.007	0.007±0.001	ND	ND	0.003-0.009	0.004±0.002
Allethrin	0.005-0.010	0.004±0.001	0.004-0.004	0.004±0.001	ND	ND	0.004-0.016	0.012±0.001	0.004-0.004	0.004±0.001

SD = standard deviation

ND = not detected

Limit of detection for synthetic pyrethroids pesticides = 0.001 $mgkg^{-1}$

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Cyfluthrin, cypermethrin, fenvalerate, deltamethrin and allethrin were detected in baby food E at mean levels of 0.004±0.001 $mgkg^{-1}$, 0.005±0.001 $mgkg^{-1}$, 0.004±0.001 $mgkg^{-1}$, 0.004±0.002 $mgkg^{-1}$ and 0.004±0.001 $mgkg^{-1}$ respectively.

Table 4.2f shows the mean concentrations of synthetic pyrethroids pesticides in baby food F to J. Permethrin and cyfluthrin were detected in baby food F at mean levels of 0.009±0.003 $mgkg^{-1}$ and 0.004±0.001 $mgkg^{-1}$ respectively whilst the deltamethrin was detected in baby food G at 0.006±0.001 $mgkg^{-1}$. Mean levels for baby food H corresponding to the following pesticides were also recorded: cypermethrin, fenvalerate, deltamethrin and allethrin were 0.005±0.001 $mgkg^{-1}$, 0.003±0.001 $mgkg^{-1}$, 0.004±0.001 $mgkg^{-1}$, 0.004±0.001 $mgkg^{-1}$ respectively.

Cypermethrin had a concentration of 0.007±0.001 $mgkg^{-1}$ in baby food I and 0.016±0.002 $mgkg^{-1}$ in baby food J. Permethrin was detected at 0.008±0.001 $mgkg^{-1}$ in baby food I whilst fenvalerate recorded mean concentration of 0.007±0.001 $mgkg^{-1}$ in baby food J.

The number of individual synthetic pyrethroids detected in baby food samples decreased in the following order Sample A (5), Sample D (5), Sample C (5), Sample E (5), Sample H (4), Sample J (2), Sample B (1), Sample I (2), Sample F (2) and Sample G (1).

Table 4.2f: Range, mean and standard deviation of synthetic pyrethroids pesticide residues in processed cereal-based complementary food

Pesticides	Baby Food F		Baby Food G		Baby Food H		Baby Food I		Baby Food J	
	Range ($mgkg^{-1}$)	Mean±SD ($mgkg^{-1}$)	Range ($mgkg^{-1}$)	Mean±SD ($mgkg^{-1}$)	Range ($mgkg^{-1}$)	Mean±SD ($mgkg^{-1}$)	Range ($mgkg^{-1}$)	Mean±SD ($mgkg^{-1}$)	Range ($mgkg^{-1}$)	Mean±SD ($mgkg^{-1}$)
Bifenthrin	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND
Fenpropathrin	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND
Lambda-cyhalthrin	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND
Permethrin	0.005-0.016	0.009±0.003	ND	ND	ND	ND	ND	ND	ND	ND
Cyfluthrin	ND-0.004	0.004±0.001	ND	ND	ND	ND	ND-0.019	0.008±0.001	ND	ND
Cypermethrin	ND	ND	ND	ND	0.003-0.006	0.005±0.001	ND-0.014	0.007±0.001	0.009-0.028	0.016±0.002
Fenvalerate	ND	ND	ND	ND	ND-0.003	0.003±0.001	ND	ND	0.003-0.017	0.007±0.001
Deltamethrin	ND	ND	0.004-0.0023	0.006±0.001	0.004-0.004	0.004±0.001	ND	ND	ND	ND
Allethrin	ND	ND	ND	ND	0.003-0.008	0.004±0.001	ND	ND	ND	ND

SD = standard deviation

ND = not detected

Limit of detection for synthetic pyrethroids= 0.001 $mgkg^{-1}$

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4.3 Health Risk Assessment of Pesticide Residues in Processed Cereal-based Complementary Food

4.3.1 Estimation of Consumption and Daily Intakes

The estimated average food consumption rate for all processed cereal-based complementary foods was 0.2 kgd^{-1} (Table 4.3) for both infants and young children. The Calculated average body weight of 8.4 kg (Table 3.1) for infants and 10.6 kg (Table 3.2) for young children.

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Table 4.3: Consumption rate of processed cereal-based complementary food for infants and young children

Name of food product	Weight per serving (kg) for Infants (6 – 11 mo)	Weight per serving (kg) for Young children (12 – 24 mo)	Food Consumption rate (kgd^{-1})	
			Infant (6 – 11 mo)	Young children (12 – 24 mo)
Baby food A (Rice and Wheat base)	0.060	0.060	0.240	0.240
Baby food B (Rice base)	0.050	0.050	0.200	0.200
Baby food C (Maize base)	0.050	0.050	0.200	0.200
Baby food D (Maize and rice base)	0.083	0.083	0.332	0.332
Baby food E (Maize and rice base)	0.050	0.050	0.200	0.200
Baby food F (Maize base)	0.025	0.035	0.100	0.140
Baby food G (Maize and Wheat base)	NA	0.025	N/A	0.100
Baby food H (Rice base)	0.050	0.050	0.200	0.200
Baby food I Maize, Rice and (Wheat base)	0.050	0.050	0.200	0.200
Baby food J (Rice base)	0.050	0.050	0.200	0.200

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Results of estimated daily intake of OPs is summarized in Table 4.3a and 4.3b. The following four pesticides recorded the highest EDIs for infants and young children respectively: pirimiphos-methyl in baby food I ($5.7x10^{-4}$ $mgkg^{-1}d^{-1}$; $4.52x10^{-4}$ $mgkg^{-1}d^{-1}$), fenitrothion in baby food E ($4.29x10^{-4}$ $mgkg^{-1}d^{-1}$, $3.39\ x10^{-4}$ $mgkg^{-1}d^{-1}$), methamidophos in baby food F ($3.10\ x10^{-4}$ $mgkg^{-1}d^{-1}$; $3.43\ x10^{-4}$ $mgkg^{-1}d^{-1}$), and chlorpyrifis in baby food ($3.33\ x10^{-4}$ $mgkg^{-1}d^{-1}$; $2.64\ x10^{-4}$ $mgkg^{-1}d^{-1}$).

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Estimated daily intakes for organochlorine pesticide residues are presented in Table 4.3c, 4.3d, and 4.3e. The OC pesticide with the highest exposure level was γ-HCH. Its EDIs recorded were $5.53\ x10^{-4}$ $mgkg^{-1}d^{-1}$ and $4.25x10^{-4}$ $mgkg^{-1}d^{-1}$ for infants and young children respectively. Pesticides with high EDI after γ-HCH were β-endosulfan ($5.00x10^{-4}$ $mgkg^{-1}d^{-1}$; $3.90x10^{-4}$ $mgkg^{-1}d^{-1}$), β-HCH ($4.05x10^{-4}$ $mgkg^{-1}d^{-1}$; $3.21x10^{-4}$ $mgkg^{-1}d^{-1}$), and γ-chlordane ($3.10\ x10^{-4}$ $mgkg^{-1}d^{-1}$; $2.45\ x10^{-4}$ $mgkg^{-1}d^{-1}$) for infants and young children respectively.

Table 4.3f, 4.3g, and 4.3h show the intake levels for synthetic pyrethroids pesticides detected in baby food samples. The first four highest EDIs for pyrethroids pesticides were in the following respective order for infants and young children. Permethrin in baby food C ($6.72\ x10^{-4}$ $mgkg^{-1}d^{-1}$;$5.32\ x10^{-4}$ $mgkg^{-1}d^{-1}$), allethrin in baby food D ($4.28x10^{-4}$ $mgkg^{-1}d^{-1}$; $3.36x10^{-4}$ $mgkg^{-1}d^{-1}$), deltamethrin in baby food A ($3.14\ x10^{-4}$ $mgkg^{-1}d^{-1}$; $2.49\ x10^{-4}$ $mgkg^{-1}d^{-1}$), and bifenthrin in baby food C ($2.16\ x10^{-4}$ $mgkg^{-1}d^{-1}$; $1.71x10^{-4}$ $mgkg^{-1}d^{-1}$).

Table 4.3a: Health risk assessment for organophosphorous pesticide residues in processed cereal-based complementary food

Pesticides	ADI (mgkg^{-1}d^{-1})	Baby Food C					Baby Food E					Baby Food F				
		Infants		Young Children			Infants		Young Children			Infants		Young Children		
		EDI (mgkg^{-1}d^{-1})	Hazard Index	EDI (mgkg^{-1}d^{-1})	Hazard Index	Health Risk	EDI (mgkg^{-1}d^{-1})	Hazard Index	EDI (mgkg^{-1}d^{-1})	Hazard Index	Health Risk	EDI (mgkg^{-1}d^{-1})	Hazard Index	EDI (mgkg^{-1}d^{-1})	Hazard Index	Health Risk
Methamidophos	0.0010	$7.14x10^{-5}$	0.071	$5.66x10^{-5}$	0.057	No, No	-	-	-	-	No, No	$3.10x10^{-4}$	0.310	$3.43x10^{-4}$	0.343	No, No
Pirimiphos-methyl	0.03	-	-				$1.90x10^{-4}$	0.006	$1.50x10^{-4}$	0.005	No, No	-	-	-	-	-
Chlorpyrifos	0.01	$3.33x10^{-4}$	0.033	$2.64x10^{-4}$	0.026	No, No	$1.43x10^{-4}$	0.014	$1.13x10^{-4}$	0.011	No, No	-	-	-	-	-
Malathion	0.3	$7.14x10^{-5}$	0.000	$5.66x10^{-5}$	0.000	No, No					No, No	-	-	-	-	-
Fenitrothion	0.006	-	-	-	-	-	$4.29X10^{-4}$	0.072	$3.39x10^{-4}$	0.057	No, No	-	-	-	-	-
Cummulative risk (∑Hazard Index)	-	-	0.11	No, No	0.028	No, No	-	0.031	-	0.024	No, No	-	0.310	-	0.343	No, No

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Table 4.3b: Health risk assessment for organophosphorous pesticide residues in processed cereal-based complementary food

		Baby Food G					Baby Food H					Baby Food I				
		Infants		Young Children			Infants		Young Children			Infants		Young Children		
Pesticides	ADI ($mgkg^{-1}d^{-1}$)	EDI ($mgkg^{-1}d^{-1}$)	Hazard Index	EDI ($mgkg^{-1}d^{-1}$)	Hazard Index	Health Risk	EDI ($mgkg^{-1}d^{-1}$)	Hazard Index	EDI ($mgkg^{-1}d^{-1}$)	Hazard Index	Health Risk	EDI ($mgkg^{-1}d^{-1}$)	Hazard Index	EDI ($mgkg^{-1}d^{-1}$)	Hazard Index	Health Risk
Methamidophos	0.0010	-	-	4.72×10^{-5}	0.047	No, No	-	-	-	-	No, No	-	-	-	-	No, No
Ethoprophos	0.0004	-	-	5.66×10^{-5}	0.143	No, No	-	-	-	-	No, No	-	-	-	-	No, No
Diazinon	0.0050	-	-	1.89×10^{-5}	0.004	No, No	-	-	-	-	No, No	-	-	-	-	No, No
Dimethoate	0.002	-	-	2.83×10^{-5}	0.014	No, No	-	-	-	-	No, No	-	-	-	-	No, No
Pirimiphos-methyl	0.03	-	-	6.60×10^{-5}	0.002	No, No	-	-	-	-	No, No	5.71×10^{-4}	0.019	4.52×10^{-4}	0.015	No, No
Fenitrothion	0.006	-	-	9.43×10^{-6}	0.002	No, No	-	-	-	-	No, No	3.33×10^{-4}	0.056	2.64×10^{-4}	0.044	No, No
Chlorfenvinphos	0.0005	-	-	-	-	No, No	1.19×10^{-4}	0.236	9.43×10^{-5}	0.189	No, No	-	-	-	-	No, No
Cummulative risk *(∑Hazard Index)*					0.207	No, No	-	0.234	-	0.189	No, No	-	0.065	-	0.058	No, No

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Table 4.3c: Health risk assessment for organochlorine pesticide residues in processed cereal-based complementary food

		Baby Food A					Baby Food B					Baby Food C				
		Infants		Young Children			Infants		Young Children			Infants		Young Children		
Pesticides	ADI ($mgkg^{-1}d^{-1}$)	EDI ($mgkg^{-1}d^{-1}$)	Hazard Index	EDI ($mgkg^{-1}d^{-1}$)	Hazard Index	Health Risk	EDI ($mgkg^{-1}d^{-1}$)	Hazard Index	EDI ($mgkg^{-1}d^{-1}$)	Hazard Index	Health Risk	EDI ($mgkg^{-1}d^{-1}$)	Hazard Index	EDI ($mgkg^{-1}d^{-1}$)	Hazard Index	Health Risk
β –HCH	NA	1.42×10^{-4}	NA	1.13×10^{-4}	NA	NA, NA	-	-	-	-	NA, NA	4.05×10^{-4}	NA	3.21×10^{-4}	NA	NA, NA
γ-HCH	0.0003	2.22×10^{-4}	0.740	1.81×10^{-4}	0.603	No, No	1.43×10^{-4}	0.476	1.13×10^{-4}	0.477	No, No	-	-	-	-	No, No
δ-HCH	0.003	-	-	-	-	No, No	-	-	-	-	No, No	1.67×10^{-4}	0.056	1.32×10^{-4}	0.044	No, No
Heptachlor	0.0001	5.71×10^{-5}	0.570	4.53×10^{-5}	0.453	No, No	-	-	-	-	No, No	-	-	-	-	No, No
α-endosulfan	0.006	-	-	-	-	No, No	1.90×10^{-4}	0.032	1.51×10^{-4}	0.025	No, No	1.43×10^{-4}	0.024	1.13×10^{-4}	0.018	No, No
β-endosulfan	0.006	-	-	-	-	No, No	5.00×10^{-4}	0.083	3.96×10^{-4}	0.07	No, No	-	-	-	-	No, No

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Table 4.3d: Health risk assessment for organochlorine pesticide residues in processed cereal-based complementary food

		Baby Food D					Baby Food E					Baby Food F				
		Infants		Young Children			Infants		Young Children			Infants		Young Children		
Pesticides	ADI (mgkg-1d-1)	EDI ($mgkg^{-1}d^{-1}$)	Hazard Index	EDI ($mgkg^{-1}d^{-1}$)	Hazard Index	Health Risk	EDI ($mgkg^{-1}d^{-1}$)	Hazard Index	EDI ($mgkg^{-1}d^{-1}$)	Hazard Index	Health Risk	EDI ($mgkg^{-1}d^{-1}$)	Hazard Index	EDI ($mgkg^{-1}d^{-1}$)	Hazard Index	Health Risk
γ-HCH	0.0003	5.53×10^{-4}	**1.843**	4.25×10^{-4}	**1.417**	**Yes Yes**	5.24×10^{-4}	**1.747**	4.15×10^{-4}	**1.383**	**Yes, Yes**	2.38×10^{-5}	0.079	2.64×10^{-5}	0.088	No, No
δ-HCH	0.003	3.16×10^{-4}	0.105	2.51×10^{-4}	0.083	No, No	1.67×10^{-4}	0.056	1.32×10^{-4}	0.044	No, No	-	-	-	-	No, No
Heptachlor	0.0001	-	-	-	-	No, No	1.43×10^{-4}	**1.430**	1.13×10^{-4}	**1.130**	**Yes, Yes**	7.14×10^{-5}	0.714	7.92×10^{-5}	0.792	No, No
γ -Chlordane	0.0005	-	-	-	-	No, No	3.10×10^{-4}	0.62	2.45×10^{-4}	0.49	No, No	-	-	-	-	No, No
p,p'-DDE	0.02	-	-	-	-	No, No	-	-	-	-	No, No	1.55×10^{-4}	0.008	1.72×10^{-4}	0.009	No, No
Dieldrin	0.0001	-	-	-	-	No, No	-	-	-	-	No, No	5.95×10^{-5}	0.595	6.60×10^{-5}	0.660	No, No

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Table 4.3e: Health risk assessment for organochlorine pesticide residues in processed cereal-based complementary food

		Baby Food G					Baby Food H					Baby Food I				
		Infants		Young Children			Infants		Young Children			Infants		Young Children		
Pesticides	ADI ($mgkg^{-1}d^{-1}$)	EDI ($mgkg^{-1}d^{-1}$)	Hazard Index	EDI ($mgkg^{-1}d^{-1}$)	Hazard Index	Health Risk	EDI ($mgkg^{-1}d^{-1}$)	Hazard Index	EDI ($mgkg^{-1}d^{-1}$)	Hazard Index	Health Risk	EDI ($mgkg^{-1}d^{-1}$)	Hazard Index	EDI ($mgkg^{-1}d^{-1}$)	Hazard Index	Health Risk
β –HCH	NA	NA	NA	4.72×10^{-4}	NA	NA, NA	-	-	-	-	-	3.33×10^{-4}	NA	2.64×10^{-4}	NA	NA, NA
β-endosulfan	0.006	-	NA	5.66×10^{-5}	0.094	No, No	-	-	-	-	No, No	-	-	-	-	No, No
Endosulfan sulphate	NA	-	NA			No, No	-	-	-	-	No, No	-	-	-	-	No, No
p,p'-DDE	0.02	-	NA	1.22×10^{-4}	0.006	No, No	-	-	-	-	No, No	-	-	-	-	No, No
Dieldrin	0.0001	-	NA	1.13×10^{-4}	**1.130**	**NA, Yes**	2.86×10^{-4}	**2.860**	1.13×10^{-4}	**1.13**	**Yes, Yes**	-	-	-	-	No, No
Endrin	0.0002	-	NA	-	-	No, No	1.43×10^{-4}	0.714	1.13×10^{-4}	0.566	No, No	-	-	-	-	No, No

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Table 4.3f: Health risk assessment for synthetic pyrethroids pesticide residues in processed cereal-based complementary food

		Baby Food A					Baby Food B					Baby Food C				
		Infants		Young Children			Infants		Young Children			Infants		Young Children		
Pesticides	ADI (mgkg^{-1}d^{-1})	EDI (mgkg^{-1}d^{-1})	Hazard Index	EDI (mgkg^{-1}d^{-1})	Hazard Index	Health Risk	EDI (mgkg^{-1}d^{-1})	Hazard Index	EDI (mgkg^{-1}d^{-1})	Hazard Index	Health Risk	EDI (mgkg^{-1}d^{-1})	Hazard Index	EDI (mgkg^{-1}d^{-1})	Hazard Index	Health Risk
Bifenthrin	0.01	-	-	-	-	No, No	-	-	-	-	No, No	2.16×10^{-4}	0.022	1.71×10^{-4}	0.017	No, No
Permethrin	0.05	-	-	-	-	No, No	-	-	-	-	No, No	1.92×10^{-4}	0.004	1.52×10^{-4}	0.003	No, No
Cyfluthrin	0.04	1.71×10^{-4}	0.004	1.35×10^{-4}	0.003	No, No	-	-	-	-	No, No	-	-	-	-	No, No
Cypermethrin	0.05	1.71×10^{-4}	0.003	1.38×10^{-4}	0.003	No, No	-	-	-	-	No, No	2.14×10^{-4}	0.004	1.71×10^{-4}	0.003	No, No
Fenvalerate	0.02	1.71×10^{-4}	0.009	1.38×10^{-4}	0.007	No, No	-	-	-	-	No, No	1.43×10^{-4}	0.007	1.11×10^{-4}	0.006	No, No
Deltamethrin	0.01	3.14×10^{-4}	0.031	2.49×10^{-4}	0.025	No, No	-	-	-	-	No, No	1.67×10^{-5}	NA	1.32×10^{-4}	0.013	No, No
Allethrin	NA	1.14×10^{-4}	NA	9.06×10^{-5}	NA	NA, NA	9.52×10^{-5}	NA	7.55×10^{-5}	NA	NA, NA	-	-	-	-	No, N0

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Table 4.3g: Health risk assessment for synthetic pyrethroids pesticide residues in processed cereal-based complementary food

		Baby Food D					Baby Food E					Baby Food F				
		Infants		Young Children			Infants		Young Children			Infants		Young Children		
Pesticides	ADI ($mgkg^{-1}d^{-1}$)	EDI ($mgkg^{-1}d^{-1}$)	Hazard Index	EDI ($mgkg^{-1}d^{-1}$)	Hazard Index	Health Risk	EDI ($mgkg^{-1}d^{-1}$)	Hazard Index	EDI ($mgkg^{-1}d^{-1}$)	Hazard Index	Health Risk	EDI ($mgkg^{-1}d^{-1}$)	Hazard Index	EDI ($mgkg^{-1}d^{-1}$)	Hazard Index	Health Risk
Permethrin	0.05	6.72×10^{-4}	0.013	5.32×10^{-4}	0.011	No, No	-	-	-	-	No, No	1.07×10^{-4}	0.002	1.19×10^{-4}	0.002	No, No
Cyfluthrin	0.04	1.58×10^{-4}	0.004	1.25×10^{-4}	0.003	No, No	9.52×10^{-5}	0.002	7.55×10^{-5}	0.002	No, No	4.76×10^{-5}	0.001	5.28×10^{-5}	0.001	No, No
Cypermethrin	0.05	2.37×10^{-4}	0.005	1.88×10^{-4}	0.004	No, No	1.19×10^{-4}	0.002	9.43×10^{-5}	0.002	No, No	-	-	-	-	No, No
Fenvalerate	0.02	-	-	-	-	No, No	9.52×10^{-5}	0.005	7.55×10^{-5}	0.004	No, No	-	-	-	-	No, No
Deltamethrin	0.01	1.58×10^{-4}	0.016	1.25×10^{-4}	0.014	No, No	9.52×10^{-5}	0.01	7.55×10^{-5}	0.008	No, No	-	-	-	-	No, No
Allethrin	NA	4.74×10^{-4}	NA	3.76×10^{-4}	NA	NA, NA	9.52×10^{-5}	NA	7.55×10^{-5}	NA	NA, NA	-	NA	-	NA	NA, NA

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Table 4.3h: Health risk assessment for synthetic pyrethroids pesticide residues in processed cereal-based complementary food

		Baby Food G					Baby Food H					Baby Food I				
		Infants		Young Children			Infants		Young Children			Infants		Young Children		
Pesticides	ADI (mgkg^{-1}d^{-1})	EDI (mgkg^{-1}d^{-1})	Hazard Index	EDI (mgkg^{-1}d^{-1})	Hazard Index	Health Risk	EDI (mgkg^{-1}d^{-1})	Hazard Index	EDI (mgkg^{-1}d^{-1})	Hazard Index	Health Risk	EDI (mgkg^{-1}d^{-1})	Hazard Index	EDI (mgkg^{-1}d^{-1})	Hazard Index	Health Risk
Permethrin	0.05	NA	NA	-	-	No, No	-	-	-	-		1.90×10^{-4}	0.004	1.51×10^{-4}	0.003	No, No
Cyfluthrin	0.04	NA	NA	-	-	No, No	-	-	-	-						
Cypermethrin	0.05	NA	NA	-	-	No, No	1.19×10^{-4}	0.002	9.43×10^{-5}	0.002		1.67×10^{-4}	0.003	1.32×10^{-4}	0.003	No, No
Fenvalerate	0.02	NA	NA	-	-	No, No	7.14×10^{-5}	0.004	5.66×10^{-5}	0.003		-	-	-	-	No, No
Deltamethrin	0.01	NA	NA	5.66×10^{-5}	0.006	No, No	9.52×10^{-5}	0.010	7.55×10^{-5}	0.008		-	-	-	-	No, No
Allethrin	NA	NA	NA	-	NA	NA, NA	9.52×10^{-5}	NA	7.55×10^{-5}	NA	NA, NA	-	NA	-	NA	No, No

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Table 4.3i: Health risk assessment for synthetic pyrethroids pesticide residues in processed cereal-based complementary food

		Baby Food J				
		Infants		Young Children		
Pesticides	ADI (mgkg^{-1}d^{-1})	EDI (mgkg^{-1}d^{-1})	Hazard Index	EDI (mgkg^{-1}d^{-1})	Hazard Index	Health Risk
Cypermethrin	0.05	3.81 x10^{-4}	0.008	3.02 x10^{-4}	0.006	No, No
Fenvalerate	0.02	1.67 x10^{-4}	0.008	1.32 x10^{-4}	0.007	No, No

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4.3.2 Health Risk Assessment

4.3.2.1 Non-carcinogenic Risk

The estimated daily intake (EDI) for all detected pesticides were calculated and divided by the ADI for each pesticide to characterize the non-carcinogenic risk of dietary exposure using the concept of hazard index (HI).

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Hazard index for all OP pesticides and synthetic pyrethroids pesticides were below 1. The cumulative risk associated with mixtures of organophosphorous pesticides in each baby food was also estimated, since pesticides in this class are known to have a common mechanism of action (Fukuto, 1990, Cassee *et al*., 1998, Mileson *et al*., 1998). Hazard indices obtained from the cumulative risk estimations for OP pesticides were less than 1 for all baby foods. The results of risk assessment are presented from Table 4.3a and 4.3b for OPs; 4.3c, 4.3d and 4.3e for OCs; and 4.3f, 4.3g and 4.3h for synthetic pyrethroids.

Risk characterization of OC pesticides indicated that HIs for γ-HCH, heptachlor, and dieldrin were greater than 1 as shown in Table 4.3c-4.3f. For example the HI for γ-HCH in baby food D was 1.84 for infants and 1.42 for young children, whilst the HI for γ-HCH in baby food E was 1.75 and 1.38 for infants and young children respectively. Baby food E also contained δ-HCH and γ-chlordane, however, the HIs of the two pesticides were less than 1 when the risk was characterized. Heptachlor recorded HI of 1.43 and 1.13 in baby food E for infants and young children respectively. Baby food G which was contaminated with dieldren recorded the highest HI of 2.86 for infants and 1.13 in young children. Risk characterization of dieldren in baby food G also resulted in

an HI of 1.13 for young children; there was no HI value for infants since baby food G was recommended only for young children.

4.3.2.2 Carcinogenic Risk

Results of cancer benchmark concentrations (CBCs) and hazard ratios (HR) for organochlorine pesticides detected in the baby food samples have been summarized from Table 4.3j and 4.3l. The pesticides detected in all the locally produced baby food samples (baby food A-E) recorded HRs greater than 1 as shown in Table 4.3k. The HR for β-HCH for infants and young children were (7.35; 5.85) in baby food A, (17.35; 10.89) in baby food C and (28.43; 17.32) in baby food D. The HR derived for γ-HCH in baby food A was 8.49 for infants and 6.73 for young children, whilst HRs 4.42 and 2.78 were recorded for the same pesticide in baby food B for infants and young children respectively. The HR measured for heptachlor in baby food A was 7.35; 4.61, and 15.31; 9.61 in baby food E for infants and young children respectively. The hazard ratio for γ-chlordane in baby food E was determined to be 2.58 and 1.62 for infants and young children.

Hazard ratios were also derived for OC pesticides which were detected in four of the imported baby foods (F-I) as shown in Table 4.3m. γ-HCH and *p,p'*-DDE were both present in baby food F, however, their HRs were less than 1. Baby food F was contaminated with both heptachlor and dieldren. The HR calculated for both pesticides was greater than 1. The HR for heptachlor was 38.3; 4.4 for infants and young children. Dieldren also recorded a HR of 11.34 for infants and 13.96 for young children. F were greater than 1 for both infants and young children. The HR determined for β-HCH in

baby food I was 14.3 for infants and 8.97 for young children. Dieldrin which was detected in baby food G had an HR of 17.09 for young children. Among the OC pesticides for which carcinogenic risk was assessed, dieldrin recorded the highest HR, this was in baby food H. The HR of dieldrin for infants who consumed baby food H was 108.84 and 34.18 for young children.

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Table 4.3j: Cancer Benchmark Concentrations for organochlorine pesticides in processed cereal-based complementary food A - E

Pesticides	OSF[a]	CBCs ($ngkg^{-1}$ bwd^{-1})		CBCs ($ngkg^{-1}$ bwd^{-1})		CBCs ($ngkg^{-1}$ bwd^{-1})		CBCs ($ngkg^{-1}$ bwd^{-1})		CBCs ($ngkg^{-1}$ bwd^{-1})	
		Baby Food A		Baby Food B		Baby Food C		Baby Food D		Baby Food E	
		Infants	Young Children	Infants	Young Children	Infants	Young Children	Infants	Young Children	Infants	Young Children
β-HCH	1.80	19.4	24.5	-	-	23.3	29.4	19.5	24.6	-	-
γ-HCH	1.30	-		32.3	40.5	-	-	-	-	-	-
Heptachlor	4.50	-	-	-	-	-	-	-	-	9.33	11.8
γ –Chlordane	0.35	-	-	-	-	-	-	-	-	120	151.4

[a] Oral slop factors (OSF) in mg kg^{-1} d^{-1} were obtained from the USEPA's Integrated Risk Information System (IRIS)

Table 4.3k: Hazard Ratios for organochlorine pesticides in processed cereal-based complementary food A - E

Pesticides	OSF	Hazard Ratios		Hazard Ratios		Hazard Ratios		Hazard Ratios		Hazard Ratios	
		Baby Food A		Baby Food B		Baby Food C		Baby Food D		Baby Food E	
		Infants	Young Children	Infants	Young Children	Infants	Young Children	Infants	Young Children	Infants	Young Children
β-HCH	1.80	**7.35**[a]	**5.82**	-	-	**17.35**	**10.89**	**28.43**	**17.32**	-	-
γ-HCH	1.30	-	-	**4.42**	**2.78**	-	-	-	-	-	-
Heptachlor	4.50	-	-	-	-	-	-	-	-	**15.31**	**9.61**
γ –Chlordane	0.35	-	-	-	-	-	-	-	-	**2.58**	**1.62**

[a] Values in bold indicate that hazard ratio is greater than 1

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Table 4.3l: Cancer Benchmark Concentrations for organochlorine pesticides in processed cereal-based complementary food E - I

Pesticides	OSF	CBCs ($ngkg^{-1}$ bwd^{-1})		CBCs ($ngkg^{-1}$ bwd^{-1})		CBCs ($ngkg^{-1}$ bwd^{-1})		CBCs ($ngkg^{-1}$ bwd^{-1})	
		Baby Food F		Baby Food G		Baby Food H		Baby Food I	
		Infants	Young Children	Infants	Young Children	Infants	Young Children	Infants	Young Children
β-HCH	1.80	-	-	-	58.9	-	-	23.3	29.4
γ-HCH	1.30	64.6	58.2	-	-	-	-	-	-
Heptachlor	4.50	18.7	16.8	-	-	-	-	-	-
p, p'-DDE	0.34	247	233	-	-	-	-	-	-
Dieldrin	16	5.25	4.73	-	6.63	2.63	3.31	-	-

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Table 4.3m: Hazard Ratios for organochlorine pesticides in processed cereal-based complementary food E - I

Pesticides	OSF	Hazard Ratios		Hazard Ratios		Hazard Ratios		Hazard Ratios	
		Baby Food F		Baby Food G		Baby Food H		Baby Food I	
		Infants	Young Children	Infants	Young Children	Infants	Young Children	Infants	Young Children
β-HCH	1.80	-	-	-	0.80	-	-	**14.3**	**8.97**
γ-HCH	1.30	0.37	0.47	-	-	-	-	-	-
Heptachlor	4.50	**38.3**	**4.7**	-	-	-	-	-	-
p, p'-DDE	0.34	0.63	0.77	-	-	-	-	-	-
Dieldrin	16	**11.34**	**13.96**	-	**17.09**	**108.84**	**34.18**	-	-

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CHAPTER FIVE

5.0 DISCUSSION

5.1 Concentration of Pesticide Residues in Processed Cereal-based Complementary Food

Residues of 36 pesticides were analyzed in ten brands of processed cereal-based complementary foods for infants and young children. The cereals used in the formulation of the baby foods were rice, wheat and millet in combination with legumes such as soya beans, cowpea and peanut. The cereals were either uses as single ingredients or as cereal mix. Pesticide residues are likely to be detected in baby food if pesticide were applied on the ingredients before consumption. The presence of pesticides in the processed cereal-based complementary food is an indication of contamination of the raw cereals either on the farm or during storage.

The mean concentration of OPs in the food samples ranged from 0.001±0.001 $mgkg^{-1}$ for dimethoate in baby food G to 0.026±0.009 $mgkg^{-1}$for methamidophos in baby food F. The mean concentrations of chlorpyrifos (0.014±0.002 $mgkg^{-1}$) in baby food C, fenitrothion (0.018±0.002 $mgkg^{-1}$) in baby food E and I, methamidophos (0.026±0.009 $mgkg^{-1}$) in baby food F and pirimiphos-methyl (0.024±0.007 $mgkg^{-1}$) in baby food I exceeded the EC maximum residue level of 0.01 $mgkg^{-1}$ (EC, 2006) for pesticides in baby foods.

Metabolites of chlorpyrifos, diazinon and malathion have been detected in children's food in USA, with mean levels of metabolites ranging between 0.001 – 0.387 $mgkg^{-1}$

(Lu *et al.*, 2008). The samples used for the study were mostly of fruits and vegetable base.

Among the OP pesticides investigated, methamidophos had the highest concentration. Methamidophos and chlorpyrifos have been detected in maize samples from Ghana at concentrations of 0.003 $mgkg^{-1}$ and 0.013 $mgkg^{-1}$ respectively (Akoto *et al.*, 2013). The concentration of methamidophos recorded for baby food G was higher than the concentration reported by Akoto *et al.*, 2013 in raw maize. The higher levels of methamidophos recorded for baby food could have resulted from contamination of the different cereals used in the formulation of baby food G.

Another study conducted in China on rice samples reported the presence of methamidophos (0.0146 $mgkg^{-1}$) and chlorpyrifos (0.0065 $mgkg^{-1}$) in the samples (Chen, *et al.*, 2009). The imported baby food samples were contaminated with more OP pesticides (7) compared with 5 in the case of the locally produced baby foods as shown in Table 4.2a and 4.2b. Five out of the 13 OP pesticides analyzed were detected in two of the locally produced baby food samples. Methamidophos, pirimiphos-methyl and fenitrothion were detected in both locally produced and imported baby foods. The concentrations of methamidophos in imported baby foods were higher than in the locally produced baby foods. For example, levels of methamidophos in baby food F and G were (0.026±0.009 $mgkg^{-1}$) and (0.005±0.003 $mgkg^{-1}$) respectively whilst the concentration of methamidophos in baby food C was (0.003±0.009 $mgkg^{-1}$).

Similar trend was observed for pirimiphos-methyl in baby food G (0.005±0.001 $mgkg^{-1}$) and baby food I (0.024±0.007 $mgkg^{-1}$) in the case of imported products and (0.04±0.014 $mgkg^{-1}$) in baby food E in the case of locally produced baby foods. The mean concentration for fenitrothion (0.018 $mgkg^{-1}$) was the same for baby food E and baby food I (Table 4.2a and 4.2b). Phorate, fonofos, parathion, chlorfenvinfos and profenofos were not detected in any of the baby food samples.

KNUST

Organochlorine pesticides were detected in 90% of the baby food samples selected for this studies. All 5 locally produced baby foods (A, B, C, D and E) were contaminated with some OCs whilst 4 of the five imported baby foods (F-I) were also contaminated with some OC pesticide residues. Baby food I was the only product which did not contain OC pesticide residues. The highest concentration of OC pesticide was recorded for lindane (γ–HCH) at 0.022±0.007 $mgkg^{-1}$ (Table 4.2c) in locally produced baby food E. γ–HCH recorded the lowest mean concentration of 0.002±0.001 $mgkg^{-1}$ (Table 4.2d) in baby food F. Other OC pesticides which exceed their MRL were β-endosulfan (0.021±0.001 $mgkg^{-1}$) in baby food B, β-HCH (0.017±0.001 $mgkg^{-1}$) in baby food. Out of the fourteen OC pesticides screened 6 different OC pesticides were detected in the locally produced baby foods as shown in Table 4.2d.

The highest OC pesticide concentration recorded for an imported baby food was 0.014±0.001 $mgkg^{-1}$ for β-HCH in baby food I. This was followed by *p,p'*-DDE (0.013±0.001 $mgkg^{-1}$) which is a metabolite of DDT. Endrin and dieldrin were also detected in three imported products (baby food F, G, and H) (Table 4.2d). Four different types of OC pesticides were detected in both locally produced and imported baby foods.

The pesticides were β-HCH (baby food A, C, G, and I), β-endosulfan (baby food B, and G), γ-HCH (baby food A, B, D, E, and F) and heptachlor (baby food A, E, and F).

Results from this study are consistent with outcome of similar studies conducted for detection of OC pesticides residues in major ingredients used the formulation of cereal-based complementary foods such as wheat, rice and maize in different regions of the world. For example, Toteja *et al,* (2003), detected all four isomers of HCH (i.e. α-HCH, β-HCH, γ-HCH and δ-HCH) in rice samples from India. Wheat was found to have been contaminated with residues of DDT and its metabolites, HCH and its isomers, heptachlor and its expoxide and aldrin (Bakore *et al.,* 2004) in India. Mawussi *et al.* (2009) have detected γ-HCH, heptachlor, DDT and its metabolites, α and β-endosulfan in maize and cowpea in Togo. In a study conducted in Turkey, methoxychlor, DDT and its metabolites, aldrin, heptachlor β and γ-HCH were found to be the highest OC pesticide residues in wheat (Guler *et al.,* 2010). In Ghana all 13 OC pesticides screened in this present work were detected at various concentrations in maize and cowpea (Akoto *et al.,* 2013). The studies conducted by Akoto *et al* in Ghana recorded OC pesticides concentration range of 0.002-0.019 $mgkg^{-1}$ in maize samples. The concentration range for OC pesticide residues recorded in this work (0.003-0.022 $mgkg^{-1}$) was higher than the levels reported by Akoto *et al.* 2013. The higher concentrations of OC recorded in this work could have resulted from the processing of the raw produce since water and other components are removed during processing.

The OCs detected in the various baby food samples are persistent organic pollutants and have been banned in many countries (Aktar *et al.,* 2009) including Ghana due to their

toxic nature. The presence of these OC pesticides in food meant for vulnerable groups of the population such as children is a major food safety concern. This is because due to their persistent nature, OC pesticides can bioaccumulate in environmental matrix such as food over long period of time, with their eventual carry over to humans through dietary exposure.

Synthetic pyrethroids insecticides are generally used in public health and agriculture throughout the world and are considered to the safest class of pesticides available due to their lower toxicity and ability to rapidly biodegrade into inactive forms in mammals (Kolaczinski and Curtis, 2004; Lothrop *et al.,* 2007). In this study, fenvalerate recorded the lowest concentration (0.003±0.001 $mgkg^{-1}$) in baby food H whilst permethrin had the highest concentration of 0.017±0.001 $mgkg^{-1}$ in baby food A which was a locally produced food. The results in Table 4.2e indicate that the recommended MRL of 0.01 $mgkg^{-1}$ was exceeded in baby food A (0.011±0.003 $mgkg^{-1}$) for deltamethrin, in baby food D (0.017±0.001 $mgkg^{-1}$; 0.012±0.001 $mgkg^{-1}$) for permethrin and allethrin respectively. For the imported baby foods the MRL was exceeded for cypermethrin (0.016±0.002 $mgkg^{-1}$) in baby food J (Table 4.2f). All ten baby food samples were contaminated with pyrethroids pesticide residues. Cypermethrin was the most frequently occurring pyrethroids pesticide. It was detected in seven of the baby food samples. This was followed by allethrin which was detected in 5 samples. The locally produced baby foods contained more pyrethroids pesticide residues than the imported products.

5.2 Risk Assessment

5.2.1 Non-Carcinogenic Risk

Food consumption rates were estimated for infants and young children using their mean body weight (Table 3.1 and Table 3.2). The consumption rate for the baby foods ranged from 0.100 – 0.332 kgd^{-1} for both infants and young children as presented in Table 3.3. Although the estimated average consumption rate for the baby foods was 0.2 kgd^{-1}, the specific consumption rate for each baby food was used for the exposure assessment. Risk assessment was conducted only for pesticides which had been detected in baby food samples. Tables 4.3a to Table 4.3c summarize the outcome of the health risk assessment for OP pesticides. The highest EDI was recorded for pirimiphos-methyl in baby food I. Table 4.3c indicates that the EDI for infants ($5.7x10^{-4}$ $mgkg^{-1}d^{-1}$) was higher than the EDI for young children ($4.52x10^{-4}$ $mgkg^{-1}d^{-1}$). On the average, the intake levels of OP pesticides for infants were higher than that for young children. The EDIs of OP pesticides were more than 70% higher for infants with the exception of the EDI for methamidophos in baby food F which recorded lower EDI for infants ($3.10x10^{-4}$ $mgkg^{-1}d^{-1}$) than in young children ($3.43\ x10^{-4}$ $mgkg^{-1}d^{-1}$). All EDIs for OP pesticides were lower than their respective acceptable daily intakes (ADI). Subsequently, risk characterization resulted in hazard index (HI) of less than1 for all OP pesticides. For example, the highest HI recorded was 0.343 for methamidophos in baby food F (Table 4.3a). These results signify that there may not be an adverse health effect associated with dietary exposure to OP pesticides detected in the various baby foods.

In order to estimate the risk associated with exposure to mixtures of OP pesticides in baby food, cumulative risk was also assessed. Organophosphorous pesticides are known

to have common mechanism of VBaction (Fukuto, 1990, Cassee *et al*., 1998, Mileson *et al*., 1998); this makes it possible to assess their cumulative effect in food (Reffstrup *et al.,* 2010). Cummulative EDI for all OPs detected ranged from 2.4 to 33.8 % of ADI, which were still within acceptable health range. Although the outcome of the risk assessment for OP pesticides indicate that there is no risk of adverse effect, because children have longer years of exposure, lifetime accumulation of OP pesticide could be significant.

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The OC pesticide with the highest EDI was γ-HCH ($5.53x10^{-4}$ $mgkg^{-1}d^{-1}$; $4.25x10^{-4}$ $mgkg^{-1}d^{-1}$) in sample D for infants and young children respectively (Table 4.3d). The EDI recorded for both age categories were higher than the ADI for γ-HCH ($3x10^{-4}$ $mgkg^{-1}d^{-1}$), eventually, the hazard index calculated from these dietary exposure scenarios for γ-HCH was greater than 1 and also recorded HIs of 1.843 for infants and 1.417 for young children. The intake level for γ-HCH ($5.24\ x10^{-4}$ $mgkg^{-1}d^{-1}$; $4.15x10^{-4}$ $mgkg^{-1}d^{-1}$) also exceeded the ADI in baby food F, for infants and young children respectively. This led to the respective HIs exceeding 1, i.e. 1.747 and 1.383. Similar results obtained for heptachlor in baby food E whose EDI is $1.43x10^{-4}$ $mgkg^{-1}d^{-1}$ resulted in an HI of 1.43 for infants whilst an EDI of $1.13x10^{-4}$ $mgkg^{-1}d^{-1}$ resulted in an HI of 1.13 (Table 4.3d).

Table 4.3f also shows the health risk assessment for OC pesticides in baby food G, H and I. The EDI for young children exposed to dieldrin (ADI of $1x10^{-4}$ $mgkg^{-1}d^{-1}$) in baby food G was $1.13x10^{-4}$ $mgkg^{-1}d^{-1}$. The hazard index measured was 1.130. Table 4.3f further indicates exceedance of ADI for dieldrin in baby food H. Estimated daily intakes

and their corresponding HIs recorded were $2.86x10^{-4}$ $mgkg^{-1}d^{-1}$ and 2.860 for infants; and $1.13x10^{-4}$ $mgkg^{-1}d^{-1}$ and 1.130 were recorded for young children. A hazard index of 2.260 recorded for dieldrin in baby food H was the highest OC pesticide residue in this work. The ADI is a health-based regulatory value established to protect the health of consumers (FAO/WHO, 2009). Exceedance of the ADI for γ-HCH, heptachlor and dieldrin in this work demonstrates that there is the potential for adverse effect on infants and young children through dietary exposure of these OC pesticides. There is therefore the need for further evaluation of specific issues surrounding chemical exposure and toxic potency (Giesy *et al.*, 2000). The Results from the exposure assessment further indicates that estimated exposure levels are age-dependent and confirms the scientific opinion that infants and younger age groups have higher food consumption per kilogram body weight and as a result have higher estimated exposure levels, which in most cases, is higher than that estimated for all other age groups (NRC, 1993 and EFSA, 2009).

The risk assessment conducted for synthetic pyrethroid pesticides is summarized in Tables 4.3f, 4.3g, 4.3h, and 4.3i. Deltamethrin recorded the highest EDI of $3.14x10^{-4}$ $mgkg^{-1}d^{-1}$ for infants and $2.49x10^{-4}$ $mgkg^{-1}d^{-1}$ for young children in baby food A (Table 4.3f). The intake level calculated for all pyrethroids pesticides were lower than the corresponding ADIs of the pesticides. Hazard indices derived for the all synthetic pyrethroids pesticides were less than 1, thus indicating that there may not be adverse effect as a result of dietary exposure to these pesticides.

5.2.2 Carcinogenic Risk

Carcinogenic risk was calculated for the OC pesticides due to their potential to cause cancer. Table 4.3j and 4.3l summarize the cancer benchmark concentrations (CBC) derived using oral slope factors from US EPA (US EPA, 2014). Hazard Ratios (HR) were used to estimate potential carcinogenic risk to humans. Tables 4.3k and 4.3m show the HRs obtained for β-HCH, γ-HCH, heptachlor, γ-chlordane, *p,p'*-DDE and dieldrin. Hazard ratio values for β-HCH demonstrates that its contamination in baby food A, C, D and I could pose potential carcinogenic effect since the HRs were greater than 1. The HRs recorded for β-HCH with respect to infants and young children were (7.35, 5.82) for baby food A; (17.35, 10.89) for baby food C; (28.43, 17.32) for baby food D; and (14.3, 8.97) for baby food I.

The HR for γ-Chlordane in baby food E was 2.58 and 1.62 for infants and young children respectively. γ-HCH recorded hazard ratio of 4.42 for infants and 2.78 for young children in baby food B. The HR for γ-HCH in baby food F was however less than 1 for both infants and young children. Heptachlor also recorded hazard ratios of 15.31 and 9.61 in baby food E (Table 4.3k) for infants and young children respectively, and 38.3 and 4.7 in baby food F (Table 4.3m) for infants and young children respectively. Dieldren recorded the highest HR of 108.84 in the baby food H (Table 4.3m). The HR values obtained for dieldrin in baby food F and G were all greater than 1. These HR values indicate that the cancer benchmark concentrations exceeded the EDI for the respective OC pesticides, thus raising serious concerns for carcinogenicity. *p,p'-DDE* which is a metabolite of DDT was detected in baby food F. The calculated CBC for *p,p'-DDE* was less than the EDI for *p,p'-DDE* hence the HR was less than 1 (Table

4.3m), indicating that it was unlikely for infants and young children to experience carcinogenic effect from dietary exposure to *p,p'*-DDE.

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CHAPTER SIX

6.0 CONCLUSIONS AND RECOMMENDATIONS

6.1 CONCLUSIONS

This study provides information on the type, and levels of occurrence of pesticides in selected processed cereal-based complementary foods. From the study organophosphorous, organochlorine and synthetic pyrethroids pesticides were detected in at least one processed cereal-based complementary food. Mean concentration for OP pesticide residues ranged from 0.001±0.001 $mgkg^{-1}$ to 0.026±0.009 $mgkg^{-1.}$ Methamidophos had the highest OP concentration as well as the highest frequency of occurrence (40%). Detected organochlorine pesticides were in the range of 0.002 – 0.022 $mgkg^{-1}$. β – endosulfan recorded the highest OC concentration whilst γ – HCH had the highest frequency of occurrence (40%). Synthetic pyrethroids pesticides detected in the different food samples were in the range of 0.002 – 0.017$mgkg^{-1}$. Permethrin had the highest concentration with cypermethrin being the most frequently occurring (70%) pyrethroids pesticide in the baby food samples.

Health risk assessment conducted for detected OPs and synthetic pyrethroids indicated that these pesticides did not pose health threat to infants and young children since calculated EDI were below the ADI. Cumulative risk assessment of OP pesticide mixtures in sampled products did not indicate health threat.

Health risk assessment for detected OC pesticide indicated that the estimated daily intake for γ – HCH in baby food D and E; heptachlor in baby food E; and dieldren in baby food G and H were higher than their respective ADI's. Hazard indices for the three

OC pesticides were greater than one signifying that processed cereal-based complementary food containing these pesticides could pose adverse health effect to infants and young children. Carcinogenic risk assessed for β-HCH, γ-HCH, heptachlor, γ-chlordane and dieldrin indicated that their cancer benchmark concentrations exceeded the respective estimated daily intake subsequently the hazard ratios calculated for the five pesticides were greater than 1 with dieldrin recording the highest hazard ratio of 108.84. Hence there is the possibility for carcinogenicity.

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6.2 RECOMMENDATIONS

In this study only three groups of pesticides were assessed for their risk. However, since different classes of pesticides may be used during agricultural production, food products could be simultaneous contaminated with a cocktail of different classes of pesticides. A national pesticide residue monitoring programme covering all classes of pesticides registered by the Ghana Environmental Protection Agency (EPA), should be instituted.

The Ghana Standards Authority, which is responsible for destination inspection of imported products including food and the Food and Drugs Authority, which is responsible for regulating imported and locally produced food on the Ghanaian market must strictly enforce legislations and regulations on foods for infants and young children in order to avoid the unnecessary exposure to pesticides. Moreover, local manufacturers of food in Ghana must be educated to source their raw materials from only safe sources.

The Ministry of Food and Agriculture in collaboration with the EPA, must intensify its training and extension activities for farmers on Good Agricultural Practices with specific focus on pesticide use.

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