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Concentrations of pesticide residues in baby foods:
understanding a common pathway of exposure for infants

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University of Maryland, College Park
2006

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Abstract

Concentrations of pesticide residues in baby foods:
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By Priya Esilda D'Souza

Background: The dietary pathway is the primary route of exposure to pesticides in the general population. An infant's diet is relatively restricted to breast milk or formula and baby food. Although baby food is an important part of an infant's diet, no studies have investigated pesticide residues in this commodity.
Objective: The aim of this study was to evaluate pesticide residues in baby foods to determine if pesticide residues were present and which brands and commodities contained the most residues.
Methods: A newly developed GC-MS/MS multi-residue method was used to evaluate a market basket survey of baby foods. The most commonly consumed baby food commodities and brands were included in this evaluation. Five brands of three different types of fruit baby foods were evaluated. Descriptive statistics were employed to determine the distribution of pesticide residues in baby foods. Fisher's Exact Tests of independence were undertaken to determine differences in the number of residues found in each product.
Results: The majority of the pesticides tested were not detected in any baby food samples. However, detectable levels of two isomers of dichlorodiphenyldichloroethylene (DDE), dicofol, fenobucarb, chlorpyrifos, and resmethrin were found in some samples. Among samples containing detectable residue levels, OC residues were found 23 times, carbamate residues 6 times, OP residues 5 times, and pyrethroid residues twice. Statistically significant differences were seen between some groups. Greater levels were seen in the conventional samples when compared to organic samples, apple samples when compared to pear samples, banana samples when compared to pear samples, Beech-Nut samples when compared to Gerber Organic samples, and Earth's Best samples when compared to Gerber Organic samples.
Conclusions: This study was the first to assess pesticide residues in baby foods in order to better understand a common pathway of exposure for infants. Characterizing dietary pesticide exposures for infants and children is an essential component of pesticide risk assessment. Several reports have demonstrated the significant contribution of dietary intake to overall pesticide exposure in children and highlighted the critical need to quantify the health risks associated with chronic low-level exposures to those pesticides.

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INTRODUCTION

The United States Environmental Protection Agency (EPA) defines a pesticide as, "any substance or mixture of substances that is used to prevent, destroy, repel, or mitigate any pest[1]." While pesticides serve an important public health role protecting fruits and vegetables and controlling insect disease vectors that affect both humans and livestock, many are known neurotoxicants with acutely toxic effects at high doses and subtle effects at lower doses. Pesticides can persist in the environment, accumulate in the food chain, and are regularly detected in humans[2]. Our dependence on these agents necessitates an in depth knowledge of their mechanism of action and potential effects on the human body, as well as the extent of their exposure to the human population.

The EPA estimated that world pesticide use exceeded 5 billion pounds (measured as pounds of active ingredients) in 2000 and 2001, with 1.2 billion pounds used in the United States alone[3]. Approximately 80 percent of this total was used for agricultural purposes[3]. Around 125 million pounds of organophosphorus (OP) insecticides were used for agricultural and residential pest control in 1999[3]. Agricultural use of OP insecticides persists although some use was restricted in 2001-2003[4]. Carbamate insecticides are widely used in agricultural applications[5]. Synthetic pyrethroids have mostly replaced residential uses of OP insecticides and are now the primary class of insecticides used in homes and gardens[5]. Based on Toxic Exposure Surveillance System[1a] statistics for the years 2001–2003 in the United States, the number of human exposures to OP insecticides decreased, while exposures to pyrethroid insecticides increased[6].

[1a] A national, real-time surveillance database that includes all human exposures reported to participating U.S. poison control centers.

Effects on Insects and Humans

Most insecticides rely on some form of neurotoxicity to exert their action. Most work on the neuronal axon or terminus to modulate nerve impulses, typically increasing these impulses or perturbing their frequency. Insecticides are not highly selective and consequently affect nontarget species as well as target organisms[1]. Acetylcholine (Ach) and gamma-aminobutyric acid (GABA), neurotransmitters shared by both insects and humans, are key targets of some insecticides[7]. Acetylcholine can excite or inhibit its target neurons and GABA is an inhibitory neurotransmitter[7]. Some insecticides interfere with the normal action of these neurotransmitters while others damage the nervous system by other means[7]. Potential sites of action of insecticides (see: Figure I) include interference with membrane transport of calcium, chloride, potassium, or sodium ions, disruption of enzymatic activity, and/or contribution to the release and/or persistence of neurotransmitters at nerve endings[1].

Currently, there are more than 200 OP insecticides (see: Figure II) and approximately 25 carbamate insecticides (see: Figure III) available, formulated into thousands of products, however, far fewer are currently registered for use in the United States[1]. All OP insecticides were derived from nerve agent chemistry such as that of soman, sarin, and tabun, but the insecticides used today are several generations of development away from those highly toxic chemicals[1].

OP and carbamate insecticides exert their toxicity through inhibition of acetylcholinesterase (AChE), the enzyme responsible for breaking down the neurotransmitter Ach, after it has carried its message across the synapse[7]. With the accumulation of free, unbound Ach at the nerve endings, there is continual stimulation of

electrical activity of the autonomic nervous system, the neuromuscular junction, and the central nervous system (CNS)[1]. This leads to overstimulation of the nervous system, and ultimately, death of an exposed organism[7].

The reaction between an OP insecticide and the active site in the AChE protein (a serine hydroxyl group) induces the formation of a transient, intermediate complex that partially hydrolyzes with the loss of a substituent group, leaving a stable, phosphorylated, and largely unreactive inhibited enzyme[1]. With many OP insecticides, an irreversibly inhibited enzyme is formed[1]. Without intervention, the toxicity persists until sufficient quantities of newly synthesized AChE are available 20 to 30 days later to catabolize excess Ach[1].

Alternatively, carbamate insecticides which attach to the reactive site of AChE undergo hydrolysis in two phases[1]. Phase one involves the removal of an aryl or alkyl group with the formation of a carbamylated enzyme and phase two is the decarbamylation of the inhibited enzyme with the generation of free, active enzyme[1]. The rate of dephosphorylation or decarbamylation is extremely slow for OP insecticides and very rapid for carbamate insecticides, which are reversible inhibitors[1]. OP insecticides bind to the active site of AChE and a neuronal, nonspecific carboxylesterase known as neuropathic target esterase (NTE) to produce an irreversibly inhibited enzyme through a mechanism known as "aging[1]." The aging process is caused by the dealkylation of the dialkylphosphorylated enzyme intermediates[1]. The aging process is thought to give an extra charge to the protein, agitating the active site and thus preventing dephosphorylation[1].

Repeated exposures to OP or carbamate insecticides cause the binding of more cholinesterase[7]. However, the signs and symptoms of acute intoxication by carbamate insecticides differ from those of OP compounds in regard to the duration and intensity of the toxicity[7]. While carbamate insecticides are reversible inhibitors of nervous tissue AChE and are biotransformed rapidly in vivo, OP insecticides will not release the bound cholinesterase once they have aged, which occurs fairly rapidly after binding[1]. The body does continue to produce cholinesterase, but it can take weeks or even months for circulating levels to stabilize[7]. There is little evidence of prolonged neurotoxicity, and carbamate ester insecticides do not inhibit NTE or elicit organophosphate-induced delayed neurotoxicity (OPIDN)[1].

Pseudocholinesterase or butyrylcholinesterase (BChE) is a toxicologically important enzyme that acts as an OP "sink" in humans, protecting the brain from OP and carbamate insults, and serving as a prophylactic agent against OP insecticide poisoning[8]. BChE detoxifies OP insecticides by forming a covalent bond with the OP, deactivating both the insecticide and the enzyme in the process[8]. While inactivation of BChE has no known adverse effects, inactivation of AChE in nerve synapses can be lethal[8]. OP-inhibited BChE and AChE can be reactivated with oximes, provided the OP insecticide has not yet aged[8]. Insects, by contrast, are more susceptible to the effects of all insecticide classes; as their enzyme system is less developed than that of humans.

Pyrethroids (see: Figure IV), synthetic versions of pyrethrins, are designed to be more stable in the environment and provide longer-lasting control[7]. Both type I and type II pyrethroid esters affect the activation (opening) and inactivation (closing) of sodium channels, resulting in hyperexitation[1]. Type I esters keep sodium channels open for

milliseconds, while type II esters keep sodium channels open for as long as several seconds[1]. Pyrethroid esters also inhibit calcium channels, calcium and magnesium ions, and adenosine triphosphatases (ATPase)[1].

There is little storage or accumulation of pyrethroid esters[1]. Although pyrethroid esters are susceptible to hydrolysis by nonspecific carboxylesterases, the microsomal monooxygenase system found in the tissues of almost all species is involved extensively in the detoxification of pyrethroid esters in mammals and of some of these agents in insect and fish species[1]. The importance of oxidative detoxification is demonstrated by the fact that the inclusion of piperonyl butoxide, a classic monooxygenase inhibitor, in preparations enhances the potency of pyrethroid esters 10- to 300-fold[1].

Organochlorine (OC) insecticides (see: Figure V), although banned for use in developed countries, are used throughout the developing world, particularly in tropical climates, because they are not only effective, but they are also inexpensive and therefore crucial tools in agriculture and public health. Low volatility, chemical stability, high lipid solubility, and slow rates of degradation contribute to their persistence in the environment, bioconcentration and biomagnification in the food chain, and the acquisition of biologically active body burdens at high trophic levels.

OC insecticides of the cyclodiene type affect chloride channels by inhibiting the GABA receptor. When a cyclodiene insecticide binds to the GABA molecule, the neurotransmitter is unable to close the chloride channel for which it acts as a gate. This allows electrical charges to continue down the neuron, resulting in overstimulation of the nervous system.

Dichlorodiphenyltrichloroethane, commonly known as DDT, is arguably the most well-known of the OC insecticides. Its mode of action (see: Figure VI) is nearly identical to the pyrethroid insecticides, except in its persistence. Biotransformation and degradation of DDT proceed exceptionally slowly. These highly lipophilic agents are stored in body tissue with high lipid content. In adipose tissue, DDT remains undisturbed, as only small amounts equilibrate with blood and are degraded and/or excreted.

Four potential mechanisms, possibly all functioning simultaneously, may be involved in the effects of DDT. DDT reduces potassium transport across the membrane and alters the porous channels through which sodium ions pass. These channels activate normally but are inactivated slowly, thus interfering with the active transport of sodium out of the nerve axon during repolarization. DDT inhibits neuronal ATPase, particularly calcium, potassium, and sodium ions, which play vital roles in neuronal repolarization. DDT also inhibits the ability of calmodulin, a calcium mediator in nerves, to transport calcium ions that are essential for the release of neurotransmitters. All these inhibited functions reduce the rate of depolarization and increase the sensitivity of neurons to small stimuli that would not elicit a response in a fully depolarized neuron.

The chlorinated cyclodiene-, benzene-, and cyclohexane-type insecticides differ from DDT, although both cause CNS stimulation. Figure VII shows how the cyclodiene compounds antagonize the action of GABA, acting at the GABA receptors, effectively blocking the GABA-induced uptake of chloride ions. The cyclodienes are also effective inhibitors of calcium, magnesium, potassium, and sodium ions, which are essential for the transport of calcium across membranes. Gamma-hexachlorocyclohexane

neurotoxicity is related primarily to the blockade of chloride ion fluctuation through the inotropic GABA receptors. The inhibition of calcium-magnesium ATPases in the synaptic membranes results in the accumulation of intracellular free calcium ions, which encourages the release of neurotransmitters, the subsequent depolarization of adjacent neurons, and the proliferation of stimuli throughout the CNS.

The Federal Insecticide, Fungicide, and Rodenticide Act of 1947 (FIFRA) required that all pesticides be under one law, administered by the United States Department of Agriculture (USDA)[1]. This authority, however, was passed to the EPA in 1972[1]. The new law and subsequent amendments defined, among other things, use restrictions, tolerances for pesticide residues on raw agricultural products, and responsibility for monitoring pesticide residue levels in food[1]. The United States Food and Drug Administration (FDA) is responsible for monitoring residue levels and for the seizure of foods that are not in compliance with regulations while the USDA monitors meat and poultry for pesticides and other chemicals[1]. The Food Quality Protection Act of 1996 (FQPA) gave special consideration to children[1]. When data on pesticides are not adequate, pesticide tolerances for children incorporate a 10-fold safety factor[1].

However, estimates of allowable daily exposure to the human population such as the minimal risk level (MRL), reference dose (RfD), and reference exposure level (REL)[9], are not available for all potentially harmful chemicals and are generally based on acute exposures leading to a specific endpoint and thus do not account for the often subtle effects of chronic, low-level exposures. Additionally, traditional risk assessment has used high-dose exposures to predict low-dose health outcomes but this may not be relevant to childhood exposures during periods of rapid development, and sensitive

population subgroups such as children may experience numerous routes of exposure to a variety of pesticide formulations[10]. For these reasons, existing regulations may not be sufficiently protective.

Numerous animal studies have demonstrated how pesticide exposure may adversely affect human brain development and alter neurological functions, and strong evidence suggests pesticide exposure predisposes to neurodegenerative diseases[2]. Research indicates that children are particularly vulnerable to these effects. Due to physiological and behavioral differences, chemical exposures among children are likely to be different, and often worse than exposures among adults. Children may be more exposed to environmental [dietary] contaminants because they consume more of certain foods and water per unit of body weight. Limited food choices lead to greater exposures to contaminants unique to certain foods that dominate their diets, such as: grains, fruit, and vegetables[11]. Rapid behavioral and physiological changes may also lead to differential exposures as a child develops;[12] affecting the absorption, distribution, metabolism, and excretion of chemicals[11]. Disruption of these processes or their coordination may lead to irreparable damage[11]. Rapidly dividing cells may be particularly vulnerable to carcinogens[11]. Children tend to have a higher metabolic rate, and in some instances, metabolic by-products are more toxic than their parent compound[11]. Even low level OC and OP pesticide exposures in infants and children have been shown to cause neurobehavioral outcomes[2].

Exposure to pesticides can occur via numerous exposure pathways, but according to the National Research Council's 1993 book, "Pesticides in the Diets of Infants and Children," "dietary intake represents the major source of pesticide exposure for infants

and children, and the dietary exposure may account for the increased pesticide-related health risks in children compared with adults[13]." The release of this book, along with the passage of the FQPA[14], demonstrated the United States' acknowledgment of the importance of reducing pesticide exposures in vulnerable populations, particularly infants and children. Monitoring and managing dietary pesticide exposures, however, remains a problem. The USDA found detectable levels of contemporary insecticides in approximately 47% of the fruit and vegetable samples tested as part of market-basket surveys in 2002[4].

Despite existing literature emphasizing the significance of the dietary pathway, it is puzzling that only limited data exists regarding pesticide residues in breast milk and residues have never been evaluated in solid baby foods or infant formula. The primary objective of this study was to measure pesticide residues in baby foods procured in the United States. The target pesticides are listed in Table I.

METHODS

Selection of baby foods

In order to determine the most widely consumed baby foods in the United States, a comprehensive literature search was conducted, reviewing all major scientific peer-reviewed journals and databases. Electronic databases were searched using the following key word strategy: popular or commonly consumed or most consumed and baby food(s) or packaged/prepared baby food(s) and United States or U.S. In addition, the works cited in the National Research Council's 1993 book, "Pesticides in the Diets of Infants and Children" and the United States Environmental Protection Agency's, "Child-Specific Exposure Factors Handbook" were manually searched, as well as websites for the United

States Department of Agriculture and the Food and Drug Administration, the National Institutes of Health, and the National Academies of Science. Emory University librarians at the Woodruff Health Sciences Center library and Goizueta Business library were also consulted for investigative strategies and in an attempt to access market research data.

Despite their everyday use, no pesticide residue data in baby food exist in the literature. The "Feeding Infants and Toddlers Study" (FITS) provided data on baby food consumption. The FITS was a national study conducted in 2002 and again in 2008, whose results were published in the Journal of the American Dietetic Association in 2004, 2006, and 2010. The study collected data on the eating habits and dietary intakes of more than 3,000 children 4 to 24 months of age from across America and included, among other components, a 24-hour dietary recall administered to parents or primary caregivers and a second dietary recall collected for a random subsample. The interviews were conducted by telephone, using a computerized dietary recall system from the University of Minnesota. This study provided data on the most consumed baby foods among infants in the United States. And according to Consumer Reports, "[t]he major brands of baby food are Beech-Nut and Gerber…[while] the major organic lines are Earth's Best and Gerber Organic."

Based on this information, a list of the top eleven baby food commodities within four major brands was compiled. These include orange/yellow vegetables (sweet potatoes, carrots, and squash), green vegetables (green beans and peas), fruit (apples, bananas, and pears), and meat (chicken, turkey, and beef) from Beech-Nut, Gerber, Gerber Organic, and Earth's Best. A market basket sample including 5 replicates (whenever possible) of each of these food commodities within each brand was purchased.

In order to get a complete, randomized sample, these samples were purchased at 3 grocery store chains (4 locations). Samples were logged, labeled, and stored in the laboratory until analyzed.

Sample Preparation

Prior to extraction and solvent preparation, all glassware and tools were washed, oven-dried, and solvent-rinsed according to Standard Operating Procedure (SOP) L01 Trace-cleaning of Glassware, Metalware, Teflon and Plastic Containers. A 3:1 toluene in acetonitrile extraction solvent was prepared by mixing 250 milliliters (mL) of American Chemical Society (ACS) reagent grade toluene with 750 mL of Chromasolv/HPLC grade or equivalent acetonitrile, measured with a 1000-mL graduated cylinder.

1.0 gram (g) of baby food matrix of interest, 5.0 mL of acetonitrile, and ~0.50 g of ACS reagent grade sodium chloride (NaCl) was added to a labeled, 15-mL, trace-cleaned, glass, disposable centrifuge tube. Acetonitrile was chosen because of its balanced polarity rather than using two solvents, one more polar and one less polar, to dissolve both the polar and non-polar insecticides of interest. Acetonitrile is miscible with water, except in the presence of NaCl. The addition of NaCl aids in the separation of the aqueous components from the solvent layer of this mixture. This mixture was then vortex mixed for 3 minutes and centrifuged for 5 minutes. Vortex mixing agitates the solvent in the matrix and extracts insecticide residues from the baby food, while centrifugation separates the solid from the liquid components.

With the sample in the centrifuge, a Supelclean™ ENVI-Carb-II/PSA (6 mL) Supelco cartridge was pre-conditioned with 5 mL of the 3:1 acetonitrile:toluene extraction solvent. This extraction solvent mixture was selected because this ratio has the

appropriate polarity for the best insecticide recovery. Next, 2.0 mL of the supernatant organic extract was loaded into the cartridge, and subsequently eluted with 10 mL of the 3:1 acetonitrile:toluene extraction solvent, collecting the eluate in a labeled 15-mL centrifuge tube. The insecticide is expected to adhere to the cartridge during the stationary phase. Rinsing with this solvent mixture encourages the release of these insecticide residues during the mobile phase, leaving behind impurities such as pigments.

The sample was then placed in a Turbovap concentrator (Zymark, Hopkinton, MA) for 15 minutes using nitrogen for evaporation at 5 psi and 50°C, followed by another 30 minutes of evaporation time, as needed. This step evaporates enough of the solution to allow for the next step. Collecting the eluate in the same labeled 15-mL tube, the cartridge was again eluted with 10 mL of the 3:1 acetonitrile:toluene solvent, bringing the total elution volume to 20 mL. The sample was placed in the Turbovap concentrator again and was evaporated to dryness for 45-60 minutes. Next the sample was reconstituted in 1 mL of acetonitrile, vortex mixed briefly (~10 seconds), and transferred to a gas chromatography (GC) vial for storage until analysis.

To ensure quality measurements, solvent spikes, matrix spikes, and all samples were extracted in triplicate.

Analysis

Extracted baby food samples were analyzed using a newly developed multi-analyte method for measuring 28 OP, OC, carbamate, and pyrethroid insecticides in baby food. This method consisted of GC separation using helium as a carrier gas followed by tandem mass spectrometry (MS/MS) detection. An Agielnt 7000 triple quadrupole mass spectrometer equipped with an Agilent 6890 GC was used. Splitless injection (2 µL) was

employed with a linear temperature gradient from 80-280º C using a DB5 column (5% diphenyl, 95% methylpolysiloxane; 32 mm ID x 30 m). The injection port and transfer line temperatures were 280º C. The mass spectrometer was operated in the negative ion electron impact ionization mode with precursor ions and product ions selected as shown in Table II. Quantification was achieved using an internal standard calibration plot with labeled internal standards for chlorpyrifos, cypermethrin, parathion, and p,p-DDE. The limits of detection ranged from 0.1-1.0 ng/mL (Table III) with relative standard deviations typically under 15% (Table IV). The relative recoveries were typically within 20% of the expected value (Table IV).

Statistical Evaluation

The frequency of detection and mean concentration was calculated for each analyte. Whenever a pesticide was detected at least once within a particular group, any remaining concentrations of zero within that group were imputed with the LOD of that pesticide divided by the square root of 2 before calculating the mean concentration for that group.

Differences between the frequencies of detection among groups based on growing convention, food commodity, and brand were determined using the Fisher's Exact Test of independence (Table VIII). Significance was set at $\alpha=0.05$ and marginal or nominal significance was set at $\alpha=0.1$. Fisher's Exact Tests were performed rather than Chi-square analyses due to the relatively low frequencies of detection in our sample data. The Fisher's Exact Test is significantly more accurate in evaluating the difference between groups when there are small numbers of observation. Table VIII provides the two-tailed p-values labeled for each comparison by pesticide.

RESULTS

We analyzed a total of 45 commonly-consumed fruit-based baby foods. These samples included conventional and organic apple-, banana-, and pear-based baby foods from three major brands: Beech-Nut, Gerber, and Earth's Best.

Table IV provides an overview of the recovery of all samples. According to standard analytical guidelines, recoveries should fall within ± 20% of the expected concentration for the measurements to be considered quantitative. Recoveries for fenobucarb, resmethrin, and two isomers of permethrin were outside of this acceptable range (37%, 216%, 200%, and 255% respectively). These altered recoveries only affected fenobucarb and resmethrin measurements in our samples as they were the only pesticides with poor recoveries that were detected. Fenobucarb recoveries were routinely ~30% suggesting a bias in the system that could be easily corrected. Prallethrin, piperonyl butoxide, one isomer of cyfluthrin, and one isomer of fenvalerate had recoveries that were slightly outside of the normally accepted range so these measurements should only be considered semi-quantitative.

Table III provides the limits of detection (LOD) and frequencies of detection (FOD), as well as the mean, median, and maximum concentrations for each analyte. The LODs for all pesticides were low (between 0.1 and 1.0 ng/g). The vast majority of the pesticides tested were not detected in any of the baby foods samples. However, we did find detectable levels of two isomers of dichlorodiphenyldichloroethylene (DDE), dicofol, fenobucarb, chlorpyrifos, and resmethrin in some samples. Pesticides were absent from all but two of the blank samples; for prallethrin and piperonyl butoxide, detectable levels were found in the blank samples. However, there were no detectable

levels of these two pesticides in the other samples themselves, so this did not affect our analysis. Among baby food samples containing detectable pesticide residue levels, OC residues were found 23 times, carbamate residues were found 6 times, OP residues were found 5 times, and pyrethroid residues were found twice.

General use of DDT, an organochlorine pesticide, was banned in the United States in 1972, ending nearly three decades of application. DDE, a metabolite and environmental degradation product of DDT, was the most frequently detected pesticide in our sample, indicating its environmental persistence and penetration of the food supply.

Samples were initially stratified by growing convention, with Beech-Nut and Gerber samples categorized as conventional and Gerber Organic and Earth's Best samples categorized as organic (Table V). Pesticide residues were detected 21 times in the conventional samples versus 15 times in the organic samples. The Fisher's Exact Test of independence revealed that there was a marginally significant difference between levels of chlorpyrifos in the conventional samples when compared to organic samples, with greater detectable levels found in the conventional samples. Significant differences were not seen for other pesticides of interest.

The following five pesticides were detected in conventional samples: p,p-DDE dicofol, fenobucarb, chlorpyrifos, and resmethrin. Among those detected in conventional samples, dicofol had the greatest frequency of detection at 29%, with a mean concentration of 0.08 ng/g and maximum concentration of 0.12 ng/g. Resmethrin had the greatest maximum concentration at 26.58 ng/g, with a mean concentration of 1.28 ng/g and a frequency of detection of 4%. The following five pesticides were detected in organic samples: p,p-DDE, dicofol, fenobucarb, resmethrin, and o,p-DDE. Among those

detected in organic samples, p,p-DDE had the greatest frequency of detection at 38%, with a mean concentration of 0.05 ng/g and maximum concentration of 0.03 ng/g. Resmethrin had the greatest maximum concentration at 9.74 ng/g, with a mean concentration of 0.63 ng/g and a frequency of detection of 5%.

Samples were then stratified by fruit type (Table VI). The following four pesticides were detected in apple samples: p,p-DDE, dicofol, fenobucarb, and resmethrin. Among those detected in apple samples, p,p-DDE had the greatest frequency of detection at 56%, with a mean concentration of 0.04 ng/g and maximum concentration of 0.04 ng/g. Resmethrin had the greatest maximum concentration at 29.59 ng/g, with a mean concentration of 1.64 ng/g and a frequency of detection of 6%. The Fisher's Exact Test of independence revealed that there was a significant difference between levels of p,p-DDE and o,p-DDE and a marginally significant difference between levels of fenobucarb in the apple samples when compared to pear samples, with greater detectable levels found in the apple samples. Significant differences were not seen for other pesticides of interest.

The following four pesticides were detected in banana samples: p,p-DDE, dicofol, chlorpyrifos, and o,p-DDE. Among those detected in banana samples, dicofol and chlorpyrifos had the greatest frequencies of detection at 33%, with dicofol having a mean concentration of 0.08 ng/g and maximum concentration of 0.12 ng/g, and chlorpyrifos having a mean concentration of 0.12 ng/g and maximum concentration of 0.33 ng/g. Chlorpyrifos had the greatest maximum concentration at 0.33 ng/g, with a mean concentration of 0.12 ng/g and a frequency of detection of 33%. The Fisher's Exact Test of independence revealed that there was a significant difference between levels of dicofol

and chlorpyrifos in the banana samples when compared to pear samples, with greater detectable levels found in the banana samples. Significant differences were not seen for other pesticides of interest.

The following two pesticides were detected in pear samples: p,p-DDE and resmethrin. Both pesticides detected in the pear samples had an 8% frequency of detection, with p,p,-DDE yielding a mean concentration of 0.07 ng/g and maximum concentration of 0.01 ng/g and resmethrin yielding a mean concentration of 0.97 ng/g and maximum concentration of 9.74 ng/g.

Samples were then stratified by brand (Table VII). Both conventional brands, Gerber and Beech-Nut, had detectable pesticide residue levels. Interestingly, while Gerber Organic only had one sample with one detectable pesticide residue (o,p-DDE), the other best-selling brand of organic baby foods, Earth's Best, had various detectable pesticide residues in numerous samples.

The following four pesticides were detected in Beech-Nut samples: p,p-DDE, dicofol, chlorpyrifos, and resmethrin. Among those detected in Beech-Nut samples, p,p-DDE, dicofol, and chlorpyrifos had the greatest frequencies of detection at 33%, with p,p-DDE having a mean concentration of 0.06 ng/g and maximum concentration of 0.04 ng/g, dicofol having a mean concentration of 0.08 ng/g and maximum concentration of 0.12 ng/g, chlorpyrifos having a mean concentration of 0.15 ng/g and maximum concentration of 0.33 ng/g, and resmethrin having a mean concentration of 3.11 ng/g and maximum concentration of 28 ng/g. Resmethrin had the greatest maximum concentration at 28 ng/g, with a mean concentration of 3.11 ng/g and a frequency of detection of 11%. The Fisher's Exact Test of independence did not detect a significant difference between

pesticide residue levels in the Beech-Nut samples when compared to Gerber Organic samples.

The following four pesticides were detected in Gerber samples: p,p-DDE, dicofol, fenobucarb, and chlorpyrifos. Among those detected in Gerber samples, dicofol had the greatest frequency of detection at 27% with a mean concentration of 0.08 ng/g and maximum concentration of 0.12 ng/g. Fenobucarb had the greatest maximum concentration at 11.78 ng/g, with a mean concentration of 1.14 ng/g and a frequency of detection of 20%. The Fisher's Exact Test for independence did not detect a significant difference between pesticide residue levels in the Gerber samples when compared to Gerber Organic samples.

The only pesticide detected in Gerber Organic samples was o,p-DDE. This analyte had a frequency of detection of 11% with a mean concentration of 0.58 ng/g and maximum concentration of 0.01 ng/g.

The following four pesticides were detected in Earth's Best samples: p,p-DDE, dicofol, fenobucarb, and resmethrin. Among those detected in Earth's Best samples, p,p-DDE had the greatest frequency of detection at 67% with a mean concentration of 0.03 ng/g and maximum concentration of 0.03 ng/g. Resmethrin had the greatest maximum concentration at 9.74 ng/g, with a mean concentration of 0.97 ng/g and a frequency of detection of 8%. The Fisher's Exact Test of independence revealed that there was a significant difference between levels of p,p-DDE and dicofol in the Earth's Best samples when compared to Gerber Organic samples, with greater detectable levels found in the Earth's Best samples. Significant differences were not seen for other pesticides of interest.

DISCUSSION

This study further demonstrates the challenges associated with monitoring and managing infant and child dietary pesticide exposures and risk. These data may serve as the impetus for future studies and new approaches to accurately capturing real-world exposures. However, there are many questions that remain unanswered: How exactly are these pesticides making their way into our food supply? Are these pesticides being applied appropriately? What happens to these chemicals during the manufacturing process? Are current regulations sufficiently protective of the human population and our environment?

Currently, baby food products sold in the United States that carry the "USDA certified organic" label must meet standards set by the USDA. Products labeled as, “100 percent organic” must contain only organically produced ingredients and processing aids (excluding water and salt). Products labeled, “organic” must consist of at least 95 percent organically produced ingredients (excluding water and salt). Any remaining product ingredients must consist of nonagricultural substances approved on the National List including specific non-organically produced agricultural products that are not commercially available in organic form. Foods labeled organic cannot be irradiated, genetically modified, or produced with hormones or antibiotics[15].

The USDA's National Organic Program (NOP) trains Accredited Certifying Agents to determine if a producer or handler is in compliance with the NOP regulations and is thus authorized to sell, label, or represent products as being “certified organic.” These agents certify that organic production and handling practices meet the national standards after seeing a history of substances applied to land for the previous 3 years, the

organic products being grown, raised, or processed, a plan describing practices and substances used in production and monitoring practices to be performed to verify that the plan is effectively implemented, a record-keeping system, and practices to prevent commingling of organic and nonorganic products and to prevent contact of products with prohibited substances[15]. While organic baby foods may contain lower pesticide levels than conventional baby foods, the "organic" label has more to do with growing patterns than with actual pesticide residue levels.

Given that the health risks associated with chronic, low-level pesticide exposures remain unclear, it may be appropriate to recommend a largely organic diet for infants and children, with special attention given to fruit, vegetable, and meat products that have been shown to contain the lowest pesticide residue levels. It may also be appropriate to recommend specific brand products that have been shown to contain significantly lower pesticide residue levels.

In conjunction with other relevant data, the findings from this study may help give parents and caregivers the information they need to make choices in order to reduce their children's intake of pesticide residues. These data may also contribute to awareness about organic labeling conventions and necessitate further studies evaluating the effects of chronic, low-level infant and child pesticide exposures via the dietary pathway.

Additionally, these findings may be used for the basis of future regulatory decisions in terms of implementing routine pesticide residue monitoring in finished food products, particularly for food items that are commonly consumed by infants and children and on pesticides routinely detected in those foods and may point to a need for stricter regulations of baby food products.

The US EPA's tolerances and regulations do not account for exposures that are compounded by frequent consumption of certain foods as well as exposures from multiple sources. The Agency for Toxic Disease Substances and Disease Registry (ATSDR) provides MRLs as an estimate of the daily human exposure to a hazardous substance that is likely to be without appreciable risk of adverse, non-cancer health effects over a specified duration of exposure[9]. These MRLs may help public health professionals decide where to look more closely to evaluate potential risk of adverse health effects from human exposure. Similarly, the US EPA provides RfD estimates of a daily oral exposure to the human population that is likely to be without an appreciable risk of deleterious effects during a lifetime[16].

Out of the six pesticides detected in our samples, ATSDR only provides an MRL for chlorpyrifos[17] while EPA provides an RfD for chlorpyrifos and resmethrin[16]. For chronic exposures, ATSDR quantifies the MRL for chlorpyrifos as 0.001 mg/kg/day[17] and EPA quantifies the RfD for chlorpyrifos as .003 mg/kg/day and 0.035 mg/kg/day for resmethrin[16]. Expanding these MRLs and RfDs to more chemicals may help determine if U.S. infants are being exposed to harmful insecticide residue levels. Pesticide residue data in baby foods from this study, in conjunction with food consumption information and information on the toxicological potency of individual pesticides can be used to estimate pesticide dietary exposures and risks.

The frequent consumption of food commodities with episodic presence of low-level pesticide residues that may cause developmental and neurological effects in young children supports the need for further research and mitigation.

Study Limitations

In interpreting the data obtained from these analyses, we must acknowledge the limitations of this study. As stated *vide supra*, according to standard analytical guidelines, our recoveries for fenobucarb, resmethrin, and two isomers of permethrin were outside of the acceptable range. These altered recoveries only affected fenobucarb and resmethrin measurements in our samples as they were the only pesticides with poor recoveries that were detected. We expect if the recoveries for fenobucarb and resmethrin were closer to 100%, they may have been detected more frequently in our samples. Fenobucarb recoveries were routinely ~30% suggesting a bias in the system that could be easily corrected. Prallethrin, piperonyl butoxide, one isomer of cyfluthrin, and one isomer of fenvalerate had recoveries that were slightly outside of the normally accepted range so these measurements should only be considered semi-quantitative. For some pesticides, we did not have the ability to see the lowest standard consistently.

Pesticides were absent from all but two of the blank samples; for prallethrin and piperonyl butoxide, detectable levels were found in the blank samples. However, there were no detectable levels of these two pesticides in the other samples themselves, so this did not affect our analysis.

All baby food samples were analyzed within a few months of their purchase. However, we do not have information as to when these foods were manufactured and packaged. There may have been varying degrees of degradation of pesticides in certain food samples that underestimated the true pesticide residues in some samples and/or overestimated pesticide residue levels in others. Variation in packaging materials (e.g. Gerber baby foods are packaged in glass jars while Gerber Organic baby foods are

packaged in plastic containers) may have also contributed to varying degrees of degradation of pesticides in certain food samples. Based on the information we have, it is impossible to quantify the magnitude of this potential degradation.

Additionally, the data reported here are solely based on 45 fruit-based baby food samples and therefore generalizing these results to all fruit-based baby food products may not be appropriate.

CONCLUSION

This study was the first to assess pesticide residues in baby foods in order to better understand a common pathway of exposure for infants. Characterizing dietary pesticide exposures for infants and children is an essential component of pesticide risk assessment. Several reports have demonstrated the significant contribution of dietary intake to overall pesticide exposure in children and highlighted the critical need to quantify the health risks associated with low-level chronic exposures to those pesticides.

TABLES AND FIGURES

Table I. Insecticides measured in this study are listed along with their class, toxicity classification, and mode of toxicity.

PESTICIDE	CLASS	TOXICITY CLASS */**	MODE OF TOXICITY ***
fenobucarb (fen)	carbamate	WHO: (a.i.) II; EPA: (formulation) II	1
propoxur (pro)	carbamate	WHO: (a.i.) II; EPA: (formulation) II	1
bendiocarb (ben)	carbamate	WHO: (a.i.) II; EPA: (formulation) II	1
hexachlorobenzene (hcb)	organochlorine	WHO: (a.i.) Ia; EPA: (formulation) IV	2
atrazine (atr)	organochlorine	WHO: (a.i.) III; EPA: (formulation) III	2
dicofol, p,p- (dic)	organochlorine	WHO: (a.i.) III; EPA: (formulation) II or III	2
heptachlor epoxide (hep)	organochlorine	WHO: (a.i.) II; EPA: (formulation) II	2
endosulfan-α (endoA)	organochlorine	WHO: (a.i.) II; EPA: (formulation) I (tech.)	2
endosulfan-β (endoB)	organochlorine	WHO: (a.i.) II; EPA: (formulation) I (tech.)	2
fonofos (fon)	organophosphate	WHO: (a.i.) Ia; EPA: (formulation) I or II	1
diazinon (dia)	organophosphate	WHO: (a.i.) II; EPA: (formulation) II or III	1
chlorpyrifos-methyl (chlm)	organophosphate	WHO: (a.i.) II; EPA: (formulation) II	1
chlorpyrifos (cpy)	organophosphate	WHO: (a.i.) II; EPA: (formulation) II	1
parathion (par)	organophosphate	WHO: (a.i.) Ia; EPA: (formulation) I	1
DDE, o,p- (ddeop)	organochlorine		1
DDE, p,p- (ddepp)	organochlorine		1
o,p-DDT (ddtop)	organophosphate	WHO: (a.i.) II; EPA: (formulation) II	1
azinphos-methyl (azm)	organophosphate	WHO: (a.i.) Ib; EPA: (formulation) I	1
piperonyl butoxide (pbo)	pesticide synergist	WHO: (a.i.) III (Table 5); EPA: (formulation) IV	
prallethrin (pral)	pyrethroid	WHO: (a.i.) II; EPA: (formulation) III	2
resmethrin (res)	pyrethroid	WHO: (a.i.) III; EPA: (formulation) III	2
permethrin (per)	pyrethroid	WHO: (a.i.) II; EPA: II (Ambush'); III ('Outflank')	2
cyfluthrin (cyf)	pyrethroid	WHO: (a.i.) II; EPA: (formulation) II	2
cypermethrin (cyp)	pyrethroid	WHO: (a.i.) II; EPA: (formulation) II	2
fenvalerate (fev)	pyrethroid	WHO: (a.i.) II; EPA: (formulation) II	2
deltamethrin (del)	pyrethroid	WHO: (a.i.) II; EPA: (formulation) II	2

Table I (continued). WHO and EPA toxicity classification scales are listed.

***WHO toxicity classes**
Class 1=extremely hazardous
Class 2=highly hazardous
Class 3=moderately hazardous
Class 4=slightly hazardous
****EPA toxicity classes**
I=most toxic (estimated to be fatal to an adult human at a dose of less than 5 grams)
II=moderately toxic (estimated to be fatal to an adult human at a dose of 5 to 30 grams)
III=slightly toxic (estimated to be fatal to an adult human at some dose in excess of 30 grams)
IV=practically non-toxic
***** modes of toxicity:** 1=cholinesterase inhibitor; 2=sodium channel modulator

Table II. Mass spectral parameters for analysis of pesticides in baby foods.

Pesticide	RT	MW	Quantitation Ion			Qualifier Ion		
			Q1	Q3	CE	Q1	Q3	CE
fenobucarb (fen)	10.51	207.3	121.2	103.1	20	121.2	51.2	40
hexachlorobenzene (hcb)	11.72	284.8	284.0	249.1	25	284.0	214.2	40
atrazine (atr)	12.09	215.7	200.3	122.2	10	200.3	104.1	20
propoxur (pro)	12.43	209.2	110.2	51.2	40	110.2	66.1	20
fonofos (fon)	12.44	246.3	109.1	63.1	15	246.2	137.2	5
bendiocarb (ben)	12.61	223.2	151.2	84.1	15	151.2	68.2	25
diazinon (dia)	12.61	304.3	304.3	179.3	15	179.3	121.0	40
chlorpyrifos-methyl (chlm)	13.57	322.6	286.2	93.2	26	288.2	93.0	20
chlorpyrifos (cpy)	14.53	350.6	314.2	286.1	5	314.2	258.0	25
parathion (par)	14.62	291.3	291.3	90.9	35	291.3	81.0	40
dicofol, p,p- (dic)	14.59	370.5	139.1	111.1	15	139.1	75.1	30
heptachlor epoxide (hep)	15.18	389.3	353.1	263.1	10	353.1	282.1	15
DDE, o,p- (ddeop)	15.73	318.0	248.2	176.3	30	246.2	176.2	35
prallethrin (pral)	15.73	300.4	123.2	87.1	15	123.2	105.2	20
endosulfan-α (endoA)	15.89	406.9	241.1	206.1	20	239.1	204.1	15
DDE, p,p- (ddepp)	16.32	318.0	246.2	176.2	35	248.2	176.2	30
endosulfan-β (endoB)	16.98	406.9	241.1	206.1	15	239.1	204.1	20
o,p-DDT (ddtop)	17.16	354.5	235.2	199.1	15	235.2	165.1	25
piperonyl butoxide (pbo)	18.17	338.4	176.2	103.1	30	176.2	91.1	40
resmethrin (res)	18.21	382.5	171.2	143.2	5	123.2	81.2	5
azinphos-methyl (azm)	19.43	317.3	160.2	77.2	20	132.2	77.1	15
permethrin (per)	20.42	391.3	183.2	77.0	40	183.2	168.2	20
cyfluthrin (cyf)	20.99	434.3	163.1	127.2	5	206.2	151.1	25
cypermethrin (cyp)	21.31	416.3	163.1	127.1	15	181.2	152.2	25
fenvalerate (fev)	22.21	419.9	167.0	125.0	10	125.2	89.0	20
deltamethrin (del)	22.72	505.2	253.1	93.2	20	253.0	174.0	15

RT = chromatographic retention time; MW = molecular weight (g/mL); Q1 = precursor ion (in mass/charge ratio units) selected in quadrupole 1 of mass spectrometer; Q2 = product ion (in mass/charge ratio units) selected in quadrupole 2 of mass spectrometer; CE = collision energy used for fragmentation in quadrupole 2 of mass spectrometer

Table III. Limits of detection, frequencies of detection, and mean, median, and maximum concentrations for each analyte are listed.

pesticide	limit of detection (ng/g)	# of times detected	total # of samples	frequency of detection (%)	mean concentration (ng/g)	median concentration (ng/g)	maximum concentration (ng/g)
p,p-dde	0.10	13	45	29	0.06	<LOD	0.04
dic	0.10	9	45	20	0.08	<LOD	0.12
fen	0.50	6	45	13	0.63	<LOD	11.78
cpy	0.10	5	45	11	0.09	<LOD	0.33
res	0.25	2	45	4	0.98	<LOD	26.58
o,p-dde	0.10	1	45	2	0.07	<LOD	0.01
ben	0.25	0	45	0	<LOD	<LOD	<LOD
chlm	0.10	0	45	0	<LOD	<LOD	<LOD
cyf_I	0.25	0	45	0	<LOD	<LOD	<LOD
cyf_II	0.25	0	45	0	<LOD	<LOD	<LOD
cyf_III	0.25	0	45	0	<LOD	<LOD	<LOD
cyp_I	0.25	0	45	0	<LOD	<LOD	<LOD
cyp_II	1.00	0	45	0	<LOD	<LOD	<LOD
cyp_III	1.00	0	45	0	<LOD	<LOD	<LOD
o,p-ddt	0.50	0	45	0	<LOD	<LOD	<LOD
del_I	1.00	0	45	0	<LOD	<LOD	<LOD
del_II	1.00	0	45	0	<LOD	<LOD	<LOD
dia	0.10	0	45	0	<LOD	<LOD	<LOD
endoA	0.10	0	45	0	<LOD	<LOD	<LOD
endoB	0.10	0	45	0	<LOD	<LOD	<LOD
fev_I	0.10	0	45	0	<LOD	<LOD	<LOD
fev_II	0.25	0	45	0	<LOD	<LOD	<LOD
hep	0.10	0	45	0	<LOD	<LOD	<LOD
pbo	0.10	0	45	0	<LOD	<LOD	<LOD
per_I	0.10	0	45	0	<LOD	<LOD	<LOD
per_II	0.10	0	45	0	<LOD	<LOD	<LOD
pral	0.50	0	45	0	<LOD	<LOD	<LOD

Table IV. Relative recoveries and standard deviations for each analyte are listed.

pesticide	average recovery	standard deviation
fen	37	11
dia	91	6
chlm	85	7
cpy	99	10
dic	85	10
ben	102	8
hep	93	8
o,p-dde	98	3
pral	132	12
endoA	107	10
p,p-dde	92	3
endoB	114	17
pbo	125	9
res	216	108
per_I	200	89
per_II	255	98
cyf_I	92	6
cyf_II	101	9
cyf_III	121	13
cyp_I	89	19
cyp_II	98	23
cyp_III	90	10
fev_I	110	10
fev_II	131	11
del_I	101	21
del_II	86	21

Table V. Pesticide residue distribution stratified by growing convention.

	conventional					
pesticide	**# of times detected**	**total # of samples**	**frequency of detection (%)**	**mean concentration (ng/g)**	**median concentration (ng/g)**	**maximum concentration (ng/g)**
p,p-dde	5	24	21	0.06	<LOD	0.04
dic	7	24	29	0.08	<LOD	0.12
fen	3	24	13	0.85	<LOD	11.78
cpy	5	24	21	0.10	<LOD	0.33
res	1	24	4	1.28	<LOD	26.58
o,p-dde	0	24	0	<LOD	<LOD	<LOD
	organic					
pesticide	**# of times detected**	**total # of samples**	**frequency of detection (%)**	**mean concentration (ng/g)**	**median concentration (ng/g)**	**maximum concentration (ng/g)**
p,p-dde	8	21	38	0.05	<LOD	0.03
dic	2	21	10	0.07	<LOD	0.11
fen	3	21	14	0.38	<LOD	0.66
cpy	0	21	0	<LOD	<LOD	<LOD
res	1	21	5	0.63	<LOD	9.74
o,p-dde	1	21	5	0.07	<LOD	0.01

Table VI. Pesticide residue distribution stratified by commodity type

	apples					
pesticide	**# of times detected**	**total # of samples**	**frequency of detection (%)**	**mean concentration (ng/g)**	**median concentration (ng/g)**	**maximum concentration (ng/g)**
p,p-dde	10	18	56	0.04	0.01	0.04
dic	4	18	22	0.08	<LOD	0.12
fen	6	18	33	1.05	<LOD	11.78
cpy	0	18	0	<LOD	<LOD	<LOD
res	1	18	6	1.64	<LOD	29.59
o,p-dde	0	18	0	<LOD	<LOD	<LOD

	bananas					
pesticide	**# of times detected**	**total # of samples**	**frequency of detection (%)**	**mean concentration (ng/g)**	**median concentration (ng/g)**	**maximum concentration (ng/g)**
p,p-dde	2	15	13	0.06	<LOD	0.03
dic	5	15	33	0.08	<LOD	0.12
fen	0	15	0	<LOD	<LOD	<LOD
cpy	5	15	33	0.12	<LOD	0.33
res	0	15	0	<LOD	<LOD	<LOD
o,p-dde	1	15	7	0.07	<LOD	0.01

	pears					
pesticide	**# of times detected**	**total # of samples**	**frequency of detection (%)**	**mean concentration (ng/g)**	**median concentration (ng/g)**	**maximum concentration (ng/g)**
p,p-dde	1	12	8	0.07	<LOD	0.01
dic	0	12	0	<LOD	<LOD	<LOD
fen	0	12	0	<LOD	<LOD	<LOD
cpy	0	12	0	<LOD	<LOD	<LOD
res	1	12	8	0.97	<LOD	9.74
o,p-dde	0	12	0	<LOD	<LOD	<LOD

Table VII. Pesticide residue distribution stratified by brand.

Beech-Nut

pesticide	# of times detected	total # of samples	frequency of detection (%)	mean concentration (ng/g)	median concentration (ng/g)	maximum concentration (ng/g)
p,p-dde	3	9	33	0.06	<LOD	0.04
dic	3	9	33	0.08	<LOD	0.12
fen	0	9	0	<LOD	<LOD	<LOD
cpy	3	9	33	0.15	<LOD	0.33
res	1	9	11	3.11	<LOD	28.00
o,p-dde	0	0	0	<LOD	<LOD	<LOD

Gerber

pesticide	# of times detected	total # of samples	frequency of detection (%)	mean concentration (ng/g)	median concentration (ng/g)	maximum concentration (ng/g)
p,p-dde	2	15	13	0.06	<LOD	0.03
dic	4	15	27	0.08	<LOD	0.12
fen	3	15	20	1.14	<LOD	11.78
cpy	2	15	13	0.08	<LOD	0.13
res	0	15	0	<LOD	<LOD	<LOD
o,p-dde	0	15	0	<LOD	<LOD	<LOD

Gerber Organic

pesticide	# of times detected	total # of samples	frequency of detection (%)	mean concentration (ng/g)	median concentration (ng/g)	maximum concentration (ng/g)
p,p-dde	0	9	0	<LOD	<LOD	<LOD
dic	0	9	0	<LOD	<LOD	<LOD
fen	0	9	0	<LOD	<LOD	<LOD
cpy	0	9	0	<LOD	<LOD	<LOD
res	0	9	0	<LOD	<LOD	<LOD
o,p-dde	1	9	11	0.58	<LOD	0.01

Earth's Best

pesticide	# of times detected	total # of samples	frequency of detection (%)	mean concentration (ng/g)	median concentration (ng/g)	maximum concentration (ng/g)
p,p-dde	8	12	67	0.03	0.01	0.03
dic	2	12	17	0.08	<LOD	0.11
fen	3	12	25	0.40	<LOD	0.66
cpy	0	12	0	<LOD	<LOD	<LOD
res	1	12	8	0.97	<LOD	9.74
o,p-dde	0	12	0	<LOD	<LOD	<LOD

Table VIII. P-values for Fisher's Exact Tests for independence by pesticide for each comparison group.

pesticide	conventional vs organic	apples vs pears	bananas vs pears	Beech-Nut vs Gerber Organic	Gerber vs Gerber Organic	Earth's Best vs Gerber Organic
p,p-dde	0.3234	0.0182	1.0000	0.2059	0.5109	0.0046
dic	0.1430	0.1297	0.0470	0.2059	0.2589	0.4857
fen	1.0000	0.0568	N/A	N/A	0.2663	0.2286
cpy	0.0514	N/A	0.0470	0.2059	0.5109	N/A
res	1.0000	1.0000	0.4444	1.0000	N/A	1.0000
o,p-dde	0.4667	0.0182	1.0000	1.0000	0.3750	0.4286

Figure I. Potential sites of action of classes of insecticides on the axon and terminal portions of the nerve[1].

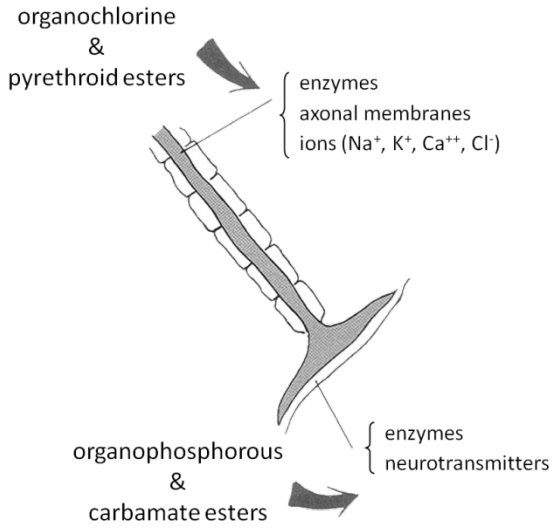


Figure II. The generic structure of the anticholinesterase-class organophosphorus insecticides is shown[1].

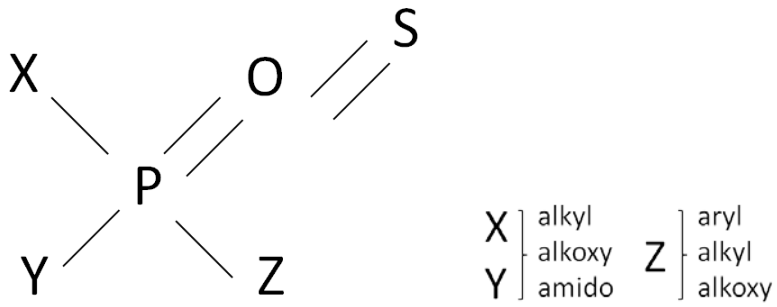


Figure III. The generic structure of the anticholinesterase-class carbamate insecticides is shown[1].

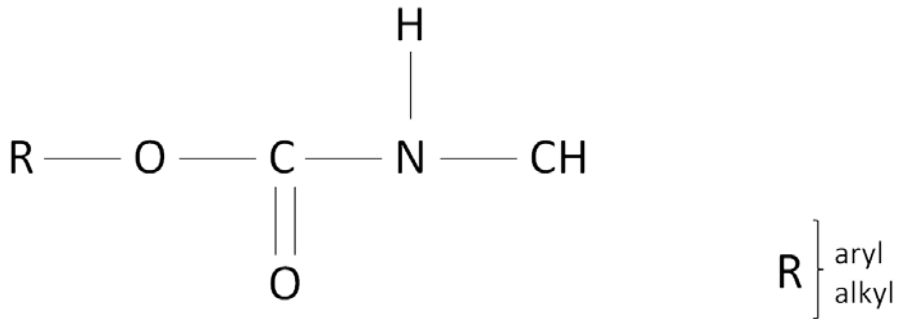


Figure IV. The basic structure of permethrin, a representative and most commonly used pyrethroid insecticide is shown[1].

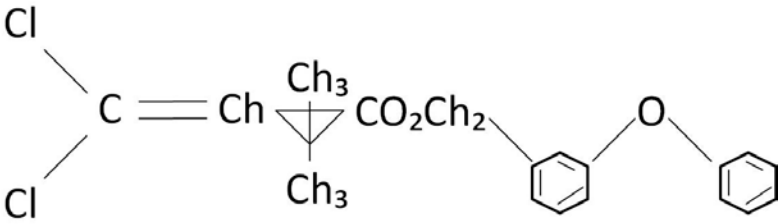


Figure V. The structure of p,p-DDT, a common organochlorine insecticide is shown[1].

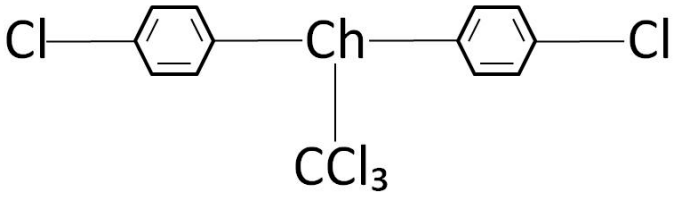


Figure VI. Potential sites of action of DDT[1].

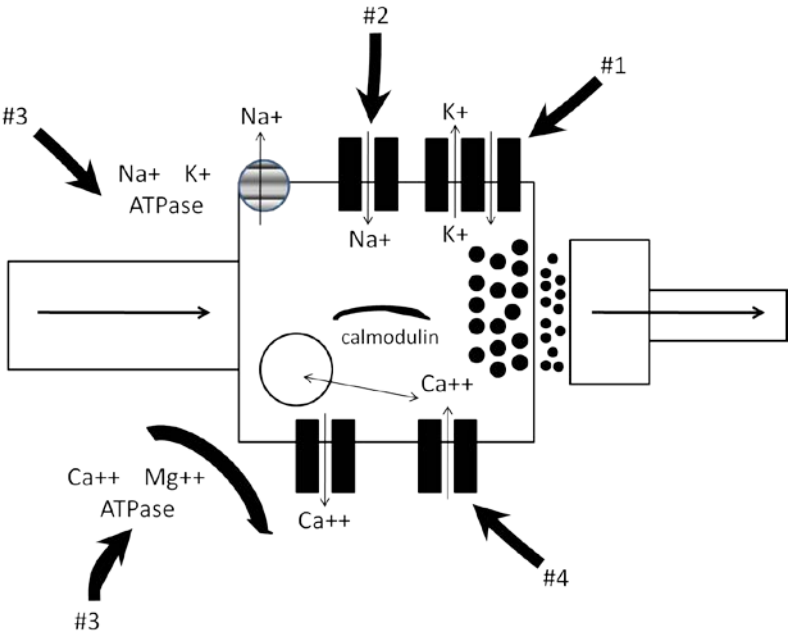
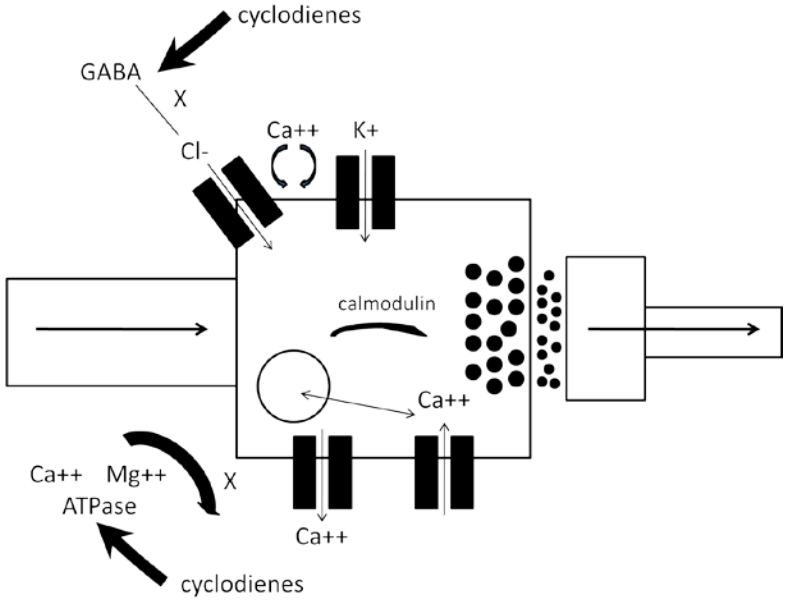


Figure VII. Proposed sites of action of cyclodiene-type organochlorine insecticides in chloride ion transport through inhibition of the GABA receptor channel as well as inhibition of calcium-magnesium ATPase[1].



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