OVEREXPOZED

Organophosphate Insecticides in Children's Food

Environmental Working Group™

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Acknowledgments

Special thanks to Molly Evans who designed and produced the report, and to Allison Daly for coordinating the release of *Overexposed*. We are grateful to Ken Cook and Mark B. Childress for editing and insight.

Overexposed was made possible by grants from The Pew Charitable Trusts, the W. Alton Jones Foundation, the Joyce Foundation, and the Working Assets Funding Service. The opinions expressed in this report are those of the authors and do not necessarily reflect the views of The Pew Charitable Trusts or other supporters listed above. Environmental Working Group is responsible for any errors of fact or interpretation contained in this report.

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Foreword

With Oprah Winfrey's "food disparagement" trial underway as this study goes to print, we're not sure if we should send reporters a press release or read them their Miranda rights.

We all have the right to remain silent about risks in the food supply; silence would suit many in the agribusiness world just fine. In publishing *Overexposed*, EWG has emphatically chosen instead to exercise our First Amendment right to highlight concerns about food safety, specifically, that many different types of foods—peaches, apples, nectarines, and popcorn, to name the top of the list—are routinely contaminated with levels of organophosphate insecticides that are unsafe for infants and children under the age of 5 years old. Based on more than 80,000 government lab test results from recent years, and detailed data on children's food consumption habits, our analysis shows that the average 25–pound one-year-old could receive an unsafe dose of neurotoxic pesticides from eating just a few bites of some of the more contaminated foods.

It is possible, even likely, that exposure to organophosphate insecticides in the most contaminated food is even now producing immediate, flu-like symptoms of neurotoxic poisoning among some unlucky children: headaches, nausea, irritability. The more worrisome threat, of course, is the more subtle, long term damage that may be caused from combinations of 13 different organophosphate insecticides found by government lab tests in a wide array of foods. The risk is that harm may be done to many thousands of children over many years, starting in the womb. The organophosphates are related chemically to nerve gases like Sarin (used to lethal effect by terrorists in Tokyo's subway), and cause all manner of problems to the nervous system, up to and including brain damage and impairment of intelligence.

The EPA must act immediately to eliminate the threat these insecticides pose to children and the rest of us. The steps we outline—banning all use of these compounds for bug control in the home and on crops used to make baby food, for starters—are prudent and necessary measures in line with a new pesticide law Congress passed unanimously in 1996.

The EPA must act immediately to eliminate the threat these insecticides pose to children and the rest of us.

We plan to keep talking about these poisons and the foods they contaminate until EPA has done its job.

That law mended some major holes in the regulatory safety net for pesticides in food.

Now that the law is in effect, EPA has all the tools it needs to end the overexposure of children to organophosphate bug killers. We plan to keep talking about these poisons and the foods they contaminate until EPA has done its job.

Kenneth A. Cook
President
Environmental Working Group

Executive Summary

Every day, nine out of ten American children between the ages of 6 months and 5 years are exposed to combinations of 13 different neurotoxic insecticides in the foods they eat. While the amounts consumed rarely cause acute illness, these "organophosphate" insecticides (OPs) have the potential to cause long term damage to the brain and the nervous system, which are rapidly growing and extremely vulnerable to injury during fetal development, infancy and early childhood.

Based on the most recent government data available on children's eating patterns, pesticides in food, and the toxicity of organophosphate insecticides, we estimate that:

- Every day, more than one million children age 5 and under (1 out of 20) eat an unsafe dose of organophosphate insecticides. One hundred thousand of these children exceed the EPA safe dose, the so-called reference dose (see p. 3), by a factor of 10 or more.
- For infants six to twelve months of age, commercial baby food is the dominant source of unsafe levels of OP insecticides. OPs in baby food apple juice, pears, applesauce, and peaches expose about 77,000 infants each day, to unsafe levels of OP insecticides.

This estimate very likely understates the number of children at risk because our analysis does not include residential and other exposures to these compounds, which can be substantial, and because EPA's estimates of a safe daily dose (the so-called references dose or RfD) are based on studies on adult animals or adult humans, and almost never include additional protections to shelter the young from the toxic effects of OPs.

Our analysis also identified foods that expose young children to the most toxic doses of these pesticides. We found that:

- One out of every four times a child age five or under eats a peach, he or she is

"Organophosphate" insecticides have the potential to cause long term damage to the brain and the nervous system, which are rapidly growing and extremely vulnerable to injury during fetal development, infancy and early childhood.

Table 1. One out of every four times a child under 6 eats peaches, he or she is exposed to an unsafe dose of organophosphate insecticides.

Foods	Likelihood of being exposed to an unsafe dose of OPs
Peaches	**24.8%**
Apples	**12.9%**
Nectarines	**12.2%**
Popcorn	**8.5%**
Pears	**7.5%**
Cornbread	**5.6%**
Applesauce	**5.2%**
Grapes	**5.1%**
Corn Chips	**4.5%**
Pears (baby food)	**3.8%**
Raisins	**3.3%**
Cherries	**3.2%**
Kiwi	**2.9%**
Peaches (baby food)	**2.4%**
Apple Juice (baby food)	**2.0%**

Source: EWG, compiled from USDA food consumption data 1989-1995, USDA and FDA pesticide residue data 1991-1996 and reference doses (RfDs) obtained from EPA in January 1998.

exposed to an unsafe level of OP insecticides. Thirteen percent of the apples, 7.5 percent of the pears and 5 percent of the grapes in the U.S. food supply expose the average young child eating these fruits to unsafe levels of OP insecticides (Table 1).

- A small but worrisome percentage of these fruits — 1.5 to 2 percent of the apples, grapes, and pears, and 15 percent of the peaches — are so contaminated with OPs that the average 25 pound one year old eating just two grapes, or three bites of an apple, pear, or peach (10 grams of each fruit) will exceed the EPA (adult) safe daily dose of OPs.

- The foods that expose the most children age six months through five years to unsafe levels of OPs (because they are more heavily consumed) are apples, peaches, applesauce, popcorn, grapes, corn chips, and apple juice. Just over half of the children that eat an unsafe level of OPs each day, 575,000 children, receive this unsafe dose from apple products alone (Table 2).

- Many of these exposures exceed safe levels by wide margins. OPs on apples, peaches, grapes, pear baby food and pears cause 85,000 children each day to exceed the federal safety standard by a factor of ten or more (Table 2).

This Environmental Working Group study utilizes detailed government data on food consumption patterns and pesticide residues to conduct the first comprehensive analysis of the toxic dose that infants and children receive when the entire organophosphate family of insect killers is assessed in combinations, and at levels, that actually occur in the food supply.

Table 2. Apples and apple products account for over half of the unsafe organophosphate exposure for children under six.

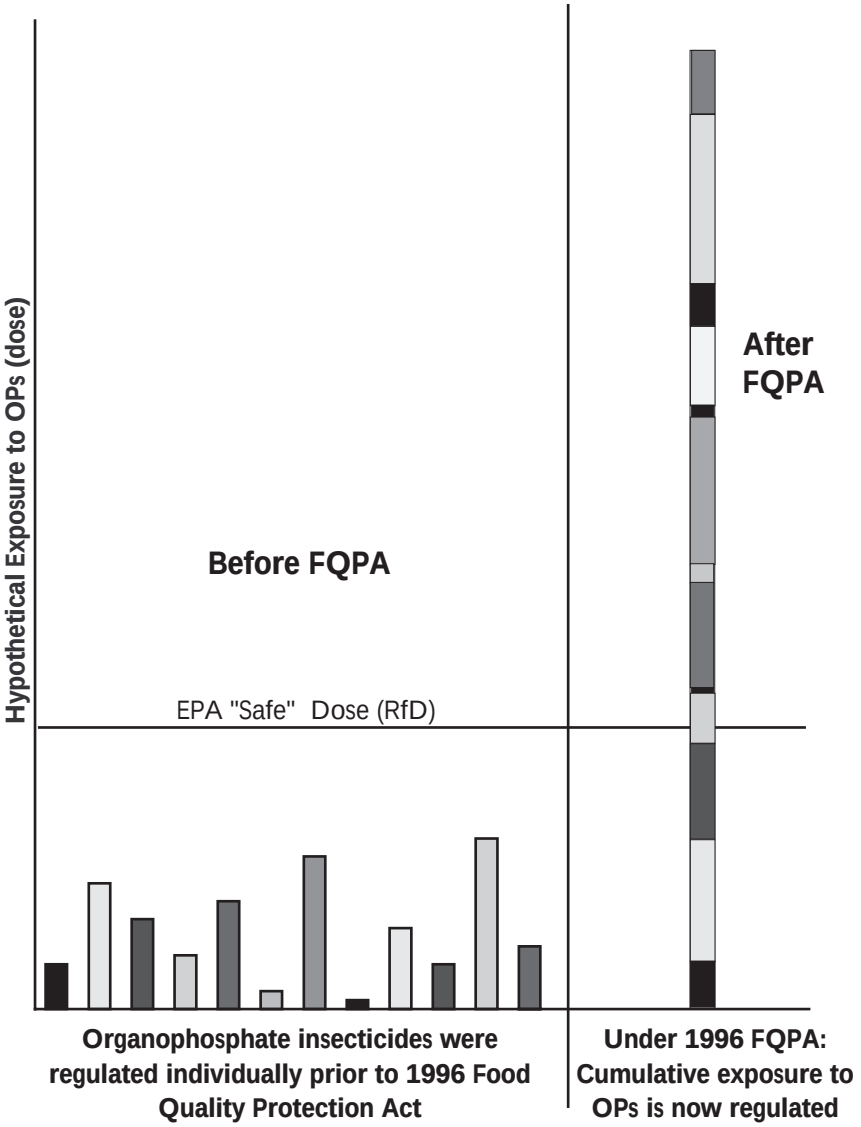
	Children exceeding "safe" dose/day	Children exceeding ten times the "safe" dose/day
Apples	408,680	34,600
Peaches	77,440	30,900
Applesauce	70,150	0
Popcorn, popped in oil	68,370	880
Grapes	54,630	12,560
Corn Chips	53,080	710
Apple Juice	46,060	180
Oat Ring Cereal	45,600	0
Apple Juice (baby food)	43,400	0
Pears (baby food)	33,060	4,610
Cornbread	27,810	3,700
Raisins	21,800	2,230
Pears	17,420	4,040
Applesauce (baby food)	10,980	0
Peaches (baby food)	10,970	0
Apple products total	**579,300**	**34,800**
Total (all foods)	**1,142,500**	**106,600**

Source: EWG, compiled from USDA food consumption data 1989-1995, USDA and FDA pesticide residue data 1991-1996 and reference doses (RfDs) obtained from EPA in January 1998.

EPA's Safety Standards: The Reference Dose

This report is based on the most recently calculated reference dose values used by EPA scientists, obtained directly from the EPA in January 1998. A reference dose is the agency's determination of a safe daily dose of a pesticide, expressed in milligrams of pesticide per kilogram of body weight per day (mg/kgbw/day). All of these reference doses represent final agency decisions, except that for chlorpyrifos. In early January 1998, the reference dose committee of the Office of Pesticide Programs recommended that the reference dose for chlorpyrifos have an additional ten-fold safety factor, per the requirements of the Food Quality Protection Act to protect infants and children from pesticides. The reference dose committee is the pesticide program committee of scientists charged with making recommendations for pesticide safety standards under the FQPA. Generally, recommendations from the reference dose committee have been adopted as agency health standards.

Figure 1. Under the 1996 Food Quality Protection Act, the EPA now must regulate cumulative exposure to pesticides with similar health effects.



Source: Environmental Working Group.

The study was prompted by the 1996 Food Quality Protection Act, which requires the government, for the first time, to consider the total risk posed to humans when they are exposed to any and all pesticides that have a common mode of toxic action and a similar type of effect. Prior to 1996 law, the government determined a separate, "safe" level of exposure for each of the dozens of registered pesticides found in food, but did not regulate as a group chemicals that produce the similar health problems. The new law further required specific protections for infants and children, who are more vulnerable to pesticides and other toxins.

Recently, the Environmental Protection Agency (EPA) concluded that the organophosphates have a common toxic mechanism, and that exposure to combinations of the chemicals should be considered in setting a "safe" dose (Figure 1).

FQPA Mandates Extra Protection for Kids

The Food Quality Protection Act (FQPA) requires EPA to *act* to protect infant and child health, even in the absence of total scientific certainty regarding the toxicity or exposure of pesticides to the fetus, infant or young child. This is a dramatic reversal of previous statutory requirements where EPA had no mandate, and arguably could not act to protect the public health, even child health, in the absence of complete data on the risk from a

pesticide. Now the law is clear. In the absence of complete and reliable data on pre- and postnatal toxicity and exposure to a pesticide, the EPA must err on the side of child safety and apply an additional ten-fold margin of safety to food tolerances for the pesticide (FQPA section 408(b)(2)(C)(ii)(II)).

Contrary to the clear requirements of the law, the EPA has devised and implemented an official policy in response to FQPA that disregards the requirement for a ten-fold safety factor (SAP 1997, EWG 1998, Cushman 1997). This policy plainly undermines protection of the nation's children from pesticides. If the new ten-fold safety factor were applied to all organophosphate insecticides found in food, we estimate that nearly 3.6 million children age 5 and under would be exposed to levels of these pesticides in food that would exceed the new standard.

High Risk Pesticides

Analysis of more than 80,000 samples of food inspected by the federal government for pesticide residues from 1991 through 1996, revealed that 13 organophosphate insecticides were found in or on food by the Food and Drug Administration and the U.S. Department of Agriculture.

The highest risk OP compounds are methyl parathion, dimethoate, chlorpyrifos, pirimiphos methyl, and azinphos methyl which account for more than 90 percent of the risk from OP insecticides in the infant and child diet.

Achieving a safe food supply for children, however, is not as simple as banning the five highest risk OPs. Home and other non-food uses must be considered, as well as the fact that other OPs will likely substitute for those that are banned in the first wave of standard setting. And most importantly, infant and child safety must be measured in terms of safety standards designed to protect these children, not in terms of the current adult-based standards.

Conclusions

American children are routinely exposed to unsafe levels of OP insecticides in the food they eat. On any given day we estimate that more than one million children under age six exceed federal safety standards for OPs. One hundred thousand of these children exceed these same standards by a factor of 10 or more. The potential public health impact of these exposures is substantial, but as yet is not precisely understood.

For perspective, it is helpful to view the situation with OPs through the lens of experience with lead. For years lead was known to be toxic, but its special hazards to children, while suspected, were difficult to confirm. Only recently has science been able to bring into focus the

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Given this overwhelming evidence of unsafe exposure to organophosphate insecticides in the diet, EPA has little choice but to act to protect infants and children.

subtle, yet profound learning deficits that result when infants and children are exposed to levels of lead that are perfectly safe for adults, and that were thought, until recently to be safe for children as well.

In some ways, the situation with OPs may be worse than lead because significant numbers of infants and children receive daily doses of multiple OPs that far exceed the safe dose for an adult. It is probable that these high OP exposures early in life are causing long term functional and learning deficits that scientists are just beginning to understand.

Given this overwhelming evidence of unsafe exposure to organophosphate insecticides in the diet, EPA has little choice but to act to protect infants and children. The solution to the problem of unsafe levels of OPs in food, however, is *not* for children to eat less fruits and vegetables. Infants, children and pregnant women should be able to eat a diet rich in fruits and vegetables without any concern about short term illness or long term brain and nervous system damage that may result from unsafe levels of OP pesticides on these foods. The solution is to rid these healthful foods of the most toxic pesticides.

Recommendations

To begin to meet the requirements of FQPA and retain the greatest number of safe pesticides for farmers, several decisive but reasonable steps must be made. These actions would reduce risk from OPs to a level deemed acceptable under current EPA policy. We must emphasize again, however, that current EPA safety standards do not yet incorporate explicit or adequate protections for infants and children. Until reliable data on fetal and infant toxicity are available for all OPs, the actions recommended here, while significant, must be viewed as first steps in an ongoing process of protecting infants and children from OP insecticides.

First, all home and other structural use of OP insecticides must be banned. These uses put a small but significant number of infants and toddlers at extremely high risk, and in doing so jeopardize current agricultural uses of these compounds. Indeed, if food uses of any OPs are to be retained, all non-food uses with potential to expose pregnant women, infants or toddlers must be banned.

Second, at least five high risk OPs, methyl parathion, dimethoate, chlorpyrifos, pirimiphos methyl, and azinphos methyl, must be banned immediately for all agricultural use.

Third, all OPs must be banned for use in food that ends up in commercial baby food.

Fourth, EPA must require developmental neurotoxicity studies for all the remaining OPs

found in the food supply. Prompt action can ensure that this critical information is available by the time EPA must take regulatory action on OPs in August 1999. At that time, the required additional ten-fold level of protection must be applied to any OP for which a developmental neurotoxicity study is not performed.

Fifth, food tolerances for all OPs must be lowered to levels that are safe for infants and children. To quote the National Research Council report, Pesticides in the Diets of Infants and Children, "Children should be able to eat a healthful diet containing legal residues without encroaching on safety margins" (NRC 1993, pp 8-9). That is to say, legal residues, or tolerances, must be safe for infants and children. There is simply no scientific justification for retaining legal limits for pesticides in food that allow hugely unsafe levels of exposure, just because most children do not receive this exposure. This nonsensical notion is like leaving the speed limit at 500 miles per hour just because most people would still drive at 65.

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Introduction

On August 3, 1996, President Clinton signed the Food Quality Protection Act (FQPA), new legislation governing the regulation and use of pesticides. This legislation enacts a strict, uniform, and unambiguous safety standard for allowable levels of pesticides in food and from all other routes of exposure. Under the law, a pesticide will not be allowed in the food supply if the total exposure to the pesticide (exposure from food, water, and home use) is not safe. Safe is defined as a "reasonable certainty" that "no harm" will come to exposed individuals, including infants and children. For carcinogens, reasonable certainty of no harm is defined as a one in one million risk, or less, of cancer (FQPA House Report 1996).

The organophosphate (OP) insecticides will be one of the first group of chemicals regulated under FQPA. This distinction is well deserved. Infants and children are exposed to many different OPs via many different routes, occasionally at relatively high levels. Animal studies, as well as evidence from human poisonings show that the fetus, infants and children are often more susceptible to OP toxicity than adults. And perhaps most important, organophosphate compounds are toxic to the nervous system, a critical, sensitive, and developing organ system in the very young.

A New Mandate

The sweeping new mandate of FQPA requires both a new assessment of health risks, and a new orientation to risk management.

First, the Act requires the EPA to protect infants and children from pesticides. In the past, regulations had been based on average exposures across the whole population and on studies primarily conducted on sexually mature (adult) animals. Now, before a pesticide can be allowed on food, it must be found to be safe for infants and children, based on reliable scientific data. Further, the finding that exposure to a pesticide is safe must include a thorough assessment of all routes of exposure to that pesticide, as well as exposure to all other compounds with a common toxic mechanism.

Second, after 25 years of risk-benefit balancing where the agency was required to weigh

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FQPA requires EPA to *act* in the absence of total scientific certainty regarding the toxicity or exposure of pesticides to the fetus, infant or young child. This is a dramatic reversal of previous statutory requirements.

farmer profits against the risks to the public health, FQPA *prohibits* the use of economic arguments as a rationale for exposing infants and children to risks that exceed the health standard of the Act.

Third, FQPA requires EPA to *act* in the absence of total scientific certainty regarding the toxicity or exposure of pesticides to the fetus, infant or young child. This is a dramatic reversal of previous statutory requirements. Prior to FQPA, EPA had no mandate, and arguably could not act to protect the public health, even child health, in the absence of complete data on the risk from a pesticide. Now the law is clear. In the absence of complete and reliable data on pre- and postnatal toxicity and exposure to a pesticide, the Admistrator must apply an additional a ten-fold margin of safety to food tolerances for the pesticide (FQPA section 408(b)(2)(C)(ii)(II)).

The OPs: A Critical Test Case

Every important new requirement of FQPA comes to bear in some important way on the regulation of the OPs.

- FQPA requires that pesticides be safe for infants and children, defined as a reasonable certainty that no harm will come to any exposed individual. Organphosphate compounds are typically more toxic to fetal and infant animals than to adult animals, both in terms of cholinesterase effects and other increasingly well documented brain and nervous system toxicity (see chapter one). There are exceptions to this rule, but in general because the OPs are toxic to the nervous system, the FQPA requirement to protect the young from pesticides is particularly relevant.

- All non-occupational routes of exposure to pesticides must be considered when food tolerances are set, including risks to infants from pesticide in drinking water, the home, school, and garden. Perhaps more than any other group of chemicals, infants and children are exposed to OPs via multiple routes, in food, around the house, at school or in the garden.

- Under FQPA, Exposure to all pesticides with a common mechanism of toxicity or similar toxic action on the body must be combined when establishing safe levels of those pesticides in food. OPs are all neurotoxins, acting on the same enzyme in the nervous system. Two separate scientific review panels agree that organophosphate insecticides share a common mechanism of toxicity (ILSI 1997, SAP 1997), and that, for purposes of protecting infants and children, exposure to

these pesticides must be considered in aggregate.

Roadblocks

Since the passage of FQPA in 1996, the application of the ten-fold safety factor has emerged as the key obstacle to successful implementation of the law. Other important issues relevant to OPs — whether they share a common mechanism of toxicity and the consideration of all routes of exposure — have been decided by the EPA in favor of children. Not so with the ten-fold safety factor. (Based on a reading of the record and EPA's policy positions before the Scientific Advisory Panel (SAP), it appears that OPs will be considered to share a common toxic mechanism and that all routes of exposure will be incorporated into risk assessments.)

FQPA's requirement for an additional ten-fold level of protection for infants and children is clear. To quote the law:

> In the case of threshold effects, for purposes of clause (ii) (I) an additional ten-fold margin of safety for the pesticide chemical residue *shall* be applied for infants and children to take into account potential pre- and post-natal toxicity and completeness of the data with respect to exposure and toxicity to infants and children. Notwithstanding such *requirements* for an additional margin of safety, the administrator may use a different margin of safety for the pesticide chemical *only if, on the basis of reliable data*, such margin will fully protect infants and children. [Emphasis added]

In spite of this unambiguous legal requirement, the EPA and its special pesticide Scientific Advisory Panel (SAP) have concluded that the agency need not apply a ten-fold safety factor to pesticide tolerances, even in the absence of reliable data on toxicity or exposure as the law so plainly sets forth. The SAP response to the EPA proposal — dated December, 1996 — plainly articulates the agency policy against implementation of the ten-fold safety factor. According to the SAP: "The Agency position that a ten-fold uncertainty factor (UF) should not be applied in every case is reasonable."

Justifying this position, the SAP invokes a logic that stands the mandate of FQPA on its head. The SAP does not challenge the fact that data on pre- and post-neonatal toxicity and exposure are unreliable; "This is a growing area of toxicology in which there are numerous data gaps and uncertainties" (SAP 1997) Instead, with total disregard for the letter of the law and the public health ethic of precaution on which it is based, they argue that the unreliability of existing data is the precise reason *not* to invoke the ten-fold safety factor required by law; "Without additional information or the use

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The decision by the Congress to require additional safety factors in the absence of reliable data was made with a full understanding of the paucity of the current database regarding pesticide toxicity and exposure to infants and children.

of additional testing, it would be difficult to assign a narrowly defined uncertainty factor (UF)." Or put another way, "The circumstances under which greater or lower UFs would be made are not clear and cogent" (SAP 1997). Indeed, it is exactly the recognized lack of information on the precise level of additional protection needed by infants and children that led the Congress to require a ten-fold safety factor until such time as reliable data become available.

FPQA requires protections unlike those in any other federal environmental law. The law mandates that the combined effects of all exposures to a pesticide must be safe for infants and children, including the effects that may result from in-utero exposure. Further, FQPA shifts the burden of proof. Under the law, pesticide manufacturers must prove that a pesticide is safe for infants and children, as opposed to prior law where EPA had to prove that the risks of a pesticide outweighed its benefits to farmers. And last, to ensure that these obligations are achieved, FQPA adds the requirement that EPA must err on the side of child health in the face of scientific uncertainty. If registrants do not provide reliable information to prove the safety of a pesticide, FQPA stipulates clearly that food tolerances for pesticides must include an additional ten-fold margin of safety, i.e. that infant and child exposure must be reduced by a factor of ten.

Congress did not enact this mandate unwittingly. During the three year period between the release of the National Academy of Sciences (NAS) study (*Pesticides in the Diets of Infants and Children*, NRC 1993) and the unanimous passage of FQPA, numerous hearings were held on the report, its findings and the recommendations of the committee for legislative reform. The Congress, and particularly the Commerce Committee of the House that produced the bill, was well aware of the state of the science at the time of enactment. The decision by the Congress to require additional safety factors in the absence of reliable data was made with a full understanding of the paucity of the current database regarding pesticide toxicity and exposure to infants and children.

When it comes to protecting children from pesticides, the law and the intent of the Congress are exceptionally clear. Under FQPA, pesticide manufacturers now have two choices: they can provide reliable data on pre- and post-neonatal toxicity and exposure to their pesticides, or they can accept a ten-fold reduction in the allowable levels of that pesticide in the food supply. Congress has taken a stand; now it is up to EPA to implement the law.

Organophosphate Toxicity

Organophosphate insecticides (OPs) share a common mechanism of toxicity, identified as inhibition of acetylcholinesterase (AChE) in the nervous system, leading to a spectrum of cholinergic symptoms (EPA 1997, ILSI 1997). The role of acetylcholine in nervous system function, however, is not completely understood. What is clear is that this ubiquitous enzyme is essential to smooth operation of both the central and peripheral nervous system. An EPA review of the science offered the following observations on the critical role of acetylcholine:

> "Most impressive is the singular fact that acetylcholine is the only substance that can influence every physiological or behavioral response thus far examined" (Myers 1974 in EPA 1997).
>
> Adding that:
>
> "From central to peripheral neurohormonal functions and from simple to complex behavioral acts the cholinergic system plays an essential role in the capability of living organisms to cope with the demands of constantly varying internal and external environments" (Russell 1981 in EPA 1997).

Forty years of animal studies have produced a wealth of peer-reviewed literature on OP toxicity. Much of this work focuses on observable cholinesterase related symptoms (nausea, vomiting, blurred vision, convulsions, irregular heart beat, and even death) but there are many aspects of OP toxicity reported in the literature that remain less well understood, and perhaps are of greater concern. Among these are the facts that:

- Fetal and neonatal animals are often more sensitive than adults to the toxic effects of OP exposure. This vulnerability includes increased sensitivity to cholinesterase effects and other potentially more serious brain and nervous system damage.
- OP exposure can produce long term behavioral and functional damage to the nervous system in the absence of observable signs of toxicity, and with little correlation with ChE levels.

"The data strongly suggest that exposure to neurotoxic compounds at levels believed to be safe for adults could result in permanent loss of brain function if it occurred during the prenatal or early childhood period of brain development."

—National Research Council, 1993

- OPs produce a range of toxic effects on different regions of the brain in the absence of overt effects (increased brain weight or size).

Infant Sensitivity

The nervous system in the developing human is incomplete and growing rapidly at birth. Cortical migration and neuron proliferation are complete at 5 and 12 months of age respectively, while mylination is only 50 percent complete at 18 months after birth. Seventy-five percent of brain growth occurs during the first two years, the remaining 25 percent is not completed until adulthood. Brain size in the newborn is proportionately greater than in adults. The newborn brain weighs one third of an adult brain, while the newborn weighs only 4 percent as much as the average adult (Snodgras 1992 in ILSI 1997).

The blood brain barrier, which restricts the penetration of toxicants to the brain is not fully developed in humans until about one year of age. It is not known when the barrier becomes fully functional. Connections in the visual system are not fully achieved until three or four years of age (Schlicter 1996).

Not surprisingly, experimental studies and clinical observations have demonstrated that the central nervous system of the human infant is more sensitive to the toxic effects of heavy metals, ethanol, retinoids, and neuroactive drugs (Schlicter 1996, NRC 1993, EWG 1993). Further, it is well documented that functional impairment of the nervous system can occur after exposures that produce no overt neurologic toxicity, no gross morphologic changes in the brain, and no overt toxicity to the mother (NRC 1991, NRC 1993). The best example of this is lead, which causes long term loss of intelligence when young children are exposed to levels that are non-toxic to the adult. In several cases for currently used pesticides, the doses subsequently found to affect functional development in test animals were lower than the doses identified as no-effect levels in long term animal studies used by the EPA for regulation (Schlicter 1996).

In 1993, the National Research Council described the situation this way:

> "The data strongly suggest that exposure to neurotoxic compounds at levels believed to be safe for adults could result in permanent loss of brain function if it occurred during the prenatal or early childhood period of brain development. This information is particularly relevant to dietary exposure to pesticides, since policies that established safe levels of exposure to neurotoxic pesticides for adults could not be assumed to ad-

equately protect a child less than four years of age." (NRC 1993 p. 61)

OPs are generally more acutely toxic to young animals. Weanling rats (23 days old) were more susceptible than adult rats to the acute toxicity of 14 out of 15 OPs tested (Brodeur and Dubois 1963 in NRC 1993). A study of rats at five different ages (1 day, 12 to 13 days, 23 to 24 days, 35 to 40 days and 63 days), showed a decreasing susceptibility to parathion and methyl parathion with increasing age (Benke and Murphy 1975 in NRC 1993). One-day-old rats are four times more sensitive to chlorpyrifos than week-old rats, and six times more sensitive than adult rats (Pope and Chakraborti 1992, Whitney 1995). And according to these authors, it is conceivable, given this steep dose-response curve, that "...even lower concentrations of chlorpyrifos may be toxic to the fetus" (Whitney 1995 p. 58).

Many other studies also show that organophosphates often have a greater toxic effect (a smaller dose is equally toxic) when exposure occurs before birth or neonatally as compared to adult exposure (Mendoza 1977, Gupta et al. 1985, Gaines et al. 1986, Pope et al. 1992, Pope & Chakraborti 1992, Campbell 1997, Song 1997, Schlicter 1996, Chakraborti et al. 1993, Chanda et al. 1996). Increased vulnerability is not limited to cholinesterase effects. One recent study has shown that behavioral abnormalities could be produced in juvenile rats (17 days old) with one fifth the dose of chlorpyrifos required to produce the same result in adult rats (Moser et al. 1995). Other work has shown that susceptibility to cell death in different regions of the brain from chlorpyrifos exposure was highly dependent on the day of gestation on which exposure occurs (Campbell et al. 1997, Whitney et al. 1995).

Studies have also shown that infant animals often have less developed OP detoxification mechanisms compared to older members of the same species (Green 1990, Murphy 1982). Tests conducted by chemical companies on human volunteers have shown that humans share susceptibility to OPs with other mammals, and human OP poisoning incidents have shown higher susceptibility by infants and young than adults (Diggory et al. 1977 in NRC 1993).

Long term functional damage and behavioral effects in the absence of overt toxicity

OP insecticides can produce behavioral and functional deficits at doses that cause no overt signs of toxicity and absent any correlation with ChE levels. A recent review of the scientific literature on OP toxicity provides many examples of this phenomenon (EPA 1997), including a paper by Wolthuis and Vanwersch (1984) which concluded that exposure to low doses of cholinesterase

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Recent lab work has revealed brain cell loss in young rats given doses of OPs that "did not affect growth or survival" of those individual rats.

inhibitors may cause acute behavioral effects without overt symptoms, and without disturbance of the physical fitness of the subject. A more recent paper by Wolthius et. al (1994) concluded that, "All four of the test materials affected performance on behavioral testing in the absence of clinical signs..." In addition, blood cholinesterase levels did not correlate with behavioral effects. The authors add that the doses administered "are so low and the CNS effects are so uncharacteristic of the classical intoxication picture that these subtle incapacitating effects may go undetected" (Wolthius et. al 1994 in EPA 1997 p. 34).

EPA analysis of a study by Desi et al. (1974) concludes that "Behavioral effects and brain cholinesterase inhibition were occurring in this study at doses which did not measurably inhibit plasma ChE or erythrocyte ChE..." (EPA 1997 p 54). Several studies by Kurtz (1976 and 1977) found behavioral effects in the absence of clinical signs after exposure to the widely used insecticide malathion. In the later study, test animals showed significantly impaired avoidance behavior while cholinesterase levels remained at 90 percent of baseline levels (EPA 1997).

Additional studies on malathion reached the same result. "It is concluded that the sensitive functional tests have supplied evidence that malathion may affect the more sophisticated functions of the body, or at least it exerts a load on and exhausts adaptability of the organisms. When the conventional toxicological examinations yielded negative results, the neurotoxicological tests demonstrated alterations in mammals" (Desi et al. 1976). A study by Nagymajtenyi et al. (1988) of dimethoate, dichlorvos and methyl parathion found that "administration of relatively small doses could produce the same kinds of effects as administration of large doses. In the case of EEG variables these data suggest CNS dysfunction from repeated low dosages of cholinesterase-inhibiting compounds." EPA staff found this study to be "...another example of study in which organophosphates caused functional effects in the nervous systems at doses which did not yield clinical signs" (EPA 1997 p. 86).

Recent lab work has revealed brain cell loss in young rats given doses of OPs that "did not affect growth or survival" of those individual rats: "Below the threshold for systemic toxicity, chlorpyrifos nevertheless damages the developing brain with little or no body or brain growth impairment" (Campbell et al. 1997). Previous work had suggested this effect, "At doses well below those that cause cell death or standard signs of systemic toxicity such as weight loss or brain weight deficits, the development of the brain may be compromised by chlorpyrifos exposure" (Whitney et al. 1995).

Fetal animals also can be harmed by OPs in the absence of any observable effect on the mother. Whitney et al. (1995) also found that "low doses of chlorpyrifos target the developing brain during the critical period when cell division is occurring." When pregnant rats were given low level repeated does of OPs, researchers have observed altered nerve development in the offspring causing neurochemical and neurobehavioral changes in developing rats, without visible signs of maternal toxicity (Gupta et al. 1985, Muto 1992, Chanda and Pope 1996). In one experiment, mother rats dosed with chlorpyrifos on gestational days 9 through 16 showed no ill effects while their offspring had behavioral problems including the inability to recognize the edge of a table before falling off and difficulty righting themselves when placed on their backs on postnatal day three (Chanda and Pope 1996). A report from Geller et al. (1985) revealed similar difficulties in avoidance behavior in rats exposed to 0.046 mg/kg soman every three days for three weeks.

Tolerance to OPs

It is generally accepted that animals build up a tolerance to the ChE inhibiting effects of OP's after repeated subacute exposures. It is not as well appreciated, however, that OP tolerance is a complex phenomenon that may have more to do with compensation behavior, than with the actual ability to detoxify or tolerate increasing doses of cholinesterase inhibitors (Bushnell 1991). In a study with the compound DFP, 21 days of exposure at 0.2 mg/kg/day produced no cholinergic symptoms or body weight changes. ChE depression and downregulation, an indicator of OP tolerance, was measured in the test animals. Notably, the tolerant animals were not able to fully regain the ability to perform normally during the repeated dosing with DFP in spite of evidence that the animals were compensating for normal cholinesterase effects (EPA 1997 p. 77).

Subsequent work by Bushnell et al. (1994) with the widely used OP chlorpyrifos (CPF) confirmed this effect. Here the authors concluded that: "Repeated dosing with CPF induced long-term inhibition of ChE activity; clinical, neurochemical, and pharmacological indications of tolerance to the effects of this inhibition; and persistent impairment of cognitive and motor function. The fact that the behavioral deficits developed slowly and did not fade as other signs of tolerance emerged indicates that tolerance to this OP did not extend to all functions of the nervous system, and may in fact exert a cost to some aspects of central nervous system function."

Indeed, according to the EPA, "cholinesterase inhibition at subclinical levels potentially alters a plethora of neurologic phenomena that may go unidentified until the individual is challenged in

Fetal animals also can be harmed by OPs in the absence of any observable effect on the mother.

Low doses of chlorpyrifos target the developing brain during the critical period when cell division is occurring.

some way" (EPA 1997 p. 76). And as concluded by Annau (1992) "It is still not clear what level of exposure (to OPs) does not result in latent toxicity" (Parenthesis added).

The Brain

The unpredictability of these latent behavioral and functional effects reflect the complexity of the central and peripheral nervous systems and the continuing evolution of scientific understanding about the workings and interactions of the brain and related organs. However, the varying effects that OPs exert on different regions of the brain do provide some insight into why the toxic effects of OPs are more complicated than simple cholinesterase depression.

Chlorpyrifos causes brain cell damage and interferes with DNA synthesis in young animals with no signs of cholinesterase depression or other toxicity. These effects would not be detected by the tests required by the EPA which only measure brain weight and observe only gross lesions.

Hammond et al. (1996) found substantially different levels of ChE inhibition in different regions of the brain after exposure to chlorpyrifos. According to the authors, "Local variations in acetylcholinesterase turnover could lead to differential inhibition in different parts of the brain, especially during chronic pesticide exposure or during recovery from acute exposure" (EPA 1997 p. 36). One consequence of this variable ChE depression is the inability of currently used tests to measure brain ChE levels (Desi et al. 1974). According the EPA scientists "The variable region by region cholinesterase inhibition in the brain suggests that more profound cholinesterase inhibition may be occurring in certain regions than would be apparent in a single brain cholinesterase assay" (EPA 1997 p. 54).

Studies have shown that low doses of chlorpyrifos target the developing brain but produce different effects in different brain regions (Campbell et al. 1997, Whitney et al. 1995). According to the authors, "low doses of chlorpyrifos target the developing brain during the critical period when cell division is occurring" (Whitney et al. 1995). The effect of chlorpyrifos in different regions of the brain, however, changes with the age of the animal and the corresponding developmental window of vulnerability. Compared to administration at 1 to 4 days of age, "When chlorpyrifos was administered at days 11-14 the major target for cell loss shifted from the brainstem to the forebrain, and in this case, effects (reduction in forebrain cell number) were seen at doses that did not compromise growth or survival" (Campbell et al. 1997 p. 179).

Chlorpyrifos causes brain cell damage and interferes with DNA synthesis in young animals with no signs of cholinesterase depression or other toxicity. These effects would not be detected by the tests required by the EPA which only measure brain weight and observe only gross lesions; in spite of the "severe brain cell loss in the brainstem, brainstem growth was maintained by enlargement of other cells" (Campbell 1997). Accord-

ing to the authors, these cellular deficits could lead to behavioral abnormalities later in life, a conclusion that may provide some insight to the behavioral effects that have been reported in the literature, in the absence of overt toxic signs. Further it appears that this type of cell damage in the developing animal is not limited to the central nervous system, but involves a more widespread disruption of normal cell development that includes the heart and perhaps other organs (Song et al. 1997).

Conclusions

There is consistent and compelling evidence in the peer-reviewed literature that the fetus, infant and young child are more vulnerable to a wide variety of toxic effects caused by organophosphate insecticides. These effects include increased sensitivity to cholinesterase effects as well as increased vulnerability to a variety of toxic effects on the nervous system that occur during critical periods of prenatal, neonatal, and postneonatal brain development.

None of the OPs widely found in the food supply have been tested for their developmental neurotoxicity (one OP, chlorpyrifos, is currently being tested), nor are there plans to test the OPs for their developmental neurotoxicity (SAP 1996, SAP 1997). This data gap is critical because the standard battery of toxicity tests does not reveal any significant age related neurotoxicity, nor are animals examined for long term behavioral or functional effects unless gross brain or nervous system abnormalities occur in the developmental and reproductive toxicity studies. Indeed, the standard battery of toxicity tests would not detect such well known developmental neurotoxins as lead, PCBs, and mercury. EPA Assistant Administrator for Prevention, Pesticides, and Toxic Substances, Dr. Lynn Goldman concurs: "We agree that the standard protocols for developmental toxicity studies and reproductive toxicity studies do not provide much information on the effect of neurotoxic pesticides on the performance of the nervous or immune systems in developing animals." (Goldman 1997).

There is consistent and compelling evidence in the peer-reviewed literature that the fetus, infant and young child are more vulnerable to a wide variety of toxic effects caused by organophosphate insecticides.

Unlike data generated under EPA protocols, the peer reviewed literature analyzed here shows a consistent and repeated pattern of behavioral and functional deficits from low level OP exposure in the absence of any overt toxic effects, usually with no correlation to ChE levels, and in some cases in the absence of significant cholinesterase depression.

Current regulation of OPs is based almost exclusively on ChE effects. The typically greater sensitivity of young animals to these effects alone argues for increased protection for children from OPs, and in and of itself supports the FQPA requirement for an additional ten-fold level of protection in the absence of reliable data to the contrary. There is mounting

Both current studies and the regulations they support do not account for these toxic effects.

evidence, however, that OPs are toxic to the developing brain and nervous system at very low levels of exposure. Again, both current studies and the regulations they support do not account for these toxic effects. Admittedly, many of the more subtle and long term neurodevelopmental effects emerging in the literature today may not be ripe for use as regulatory endpoints. This growing body of evidence, however, plainly supports the need for greater protection from OPs for infants and the developing fetus and embryo.

Data Sources and Methodology

Data Sources

All data used in this risk assessment are from federal government sources. Food consumption data are from the USDA, pesticide residue data are from both the USDA and the FDA, and toxicity data are from the EPA.

Food Consumption Data

The food consumption data used in this analysis are from the USDA Continuing Survey of Food Intakes by Individuals (CSFII) for the years, 1989, 1990, 1991, 1994 and 1995, the most recent years for which data are available. No data were collected in 1992 and 1993. The CSFII contains 3,695 coded foods and beverages reported as eaten by the survey population ranging from blueberry pie to scrambled eggs, potato chips to mint juleps. A total of 4,082 children between one month and five years of age were surveyed in the years 1989-91 and 1994-95 (Table 3). These 4,082 children provided one to three days of valid information each, for a total of 8,302 valid eating days.

Survey participants are asked to complete a diary containing the amount (by weight) of each food eaten at each meal during the three nonconsecutive days of the survey, and the weight, sex and date of birth of the person consuming that food. The information in the diaries was confirmed by telephone interview. These data allow age group analyses, as well as estimates of food consumption on a per kilogram of body weight per day basis, for each individual in the database.

The CSFII is a weighted, stratified sample of individuals that is designed to provide a representative picture of the dietary patterns of the U.S. population. Risk as-

Table 3. Children's food consumption data used in this analysis.

Age	Children Included In CSFII Survey	Valid Eating Days
6-12 mo.	274	572
1	842	1,732
2	868	1,768
3	726	1,448
4	701	1,410
5	671	1,372
TOTAL	**4,082**	**8,302**

Source: USDA Continuing Survey of Food Intake by Individuals. Survey Years: 1989-91, 1994-95.

Table 4. Pesticide residue data used in this analysis.

Database	Source	Dates	Number of foods used in analysis	Number of samples used in analysis	Attributes
Total Diet Study	FDA	1991-96	90	1,459	cooked and prepared food
Pesticide Data Program	USDA	1992-95	14	25,886	washed and peeled *
Pesticide Monitoring Database	FDA	1992-96	68	23,238	raw commodities/reduction factors applied (see table 5)
TOTAL			**172****	**50,583**	

**90% reduction factor was applied to green bean results from the PDP because they are tested raw but eaten cooked.*
*** These 172 foods were linked to 407 distinct foods in the USDA food consumption database.*

Source: EWG, compiled from USDA food consumption data 1989-1995, USDA and FDA pesticide residue data 1991-1996 and reference doses (RfDs) obtained from EPA in January 1998.

sessments and estimates of the number of children exceeding specific risk standards and safety margins are based on EWG's assessment of the significance of the sample weights supplied by USDA.

Pesticides in the Food Supply

Data on pesticides in the food supply are collected by the USDA and the FDA. There are three major programs: the USDA Pesticide Data Program, the FDA Pesticide Surveillance and Monitoring Program and the FDA Total Diet Study (Table 4). Each has a specific purpose, and its own strengths and weaknesses.

USDA/Pesticide Data Program. The USDA Pesticide Data Program (PDP) was started in 1991 specifically to monitor pesticide levels in fruits and vegetables most commonly consumed by children. The purpose of the program was to supplement the FDA surveillance data with more accurate and statistically representative information on pesticides residues on fruits and vegetables heavily consumed by infants and children. PDP typically samples twelve to fourteen foods, mostly fresh fruits and vegetables, per year. Samples are collected to accurately reflect the percent contribution to the national food supply for a given crop by growing region and season. Samples are then washed, peeled, and cored to reflect normal food preparation and consumption practice for that fruit or vegetable. PDP residue testing uses powerful analytical techniques that can detect trace residues in the 1 part per billion range or less, similar to the range of detection in the Total Diet Study (TDS). PDP takes 400-700 samples of each crop per year. More than 25,800 PDP samples from the years 1992 through 1995 were used in this analysis. These data were the data of first choice for fresh fruits and vegetables.

FDA Surveillance Data. The FDA Pesticide Surveillance and Monitoring Program enforces food tolerances established by the EPA. Because the monitoring is designed for regulatory enforcement purposes, as opposed to dietary exposure assessment, the data do not provide a strictly statistically representative picture of pesticides in the US food supply. This shortcoming, however, is largely offset by the sheer size of the database generated by the program and the fact that the program does sample food from all regions of the country at labs located in nine different metropolitan areas. Between 12,000 and 16,000 samples of food are tested for pesticides each year, about half of which are imports. We analyzed all records from the FDA surveillance database from the years 1992 through 1995, which contained residue findings for 51,280 food samples.

Our analysis used only random "surveillance" samples. "Compliance" samples, which are specifically aimed at crops or growers with a known problem or history of violations, are not included in the analysis. Surveillance samples are typically taken at packing sheds, warehouses, or other central distribution points. They are not taken at retail points of sale or from grocery store shelves. Further, the samples are not washed or peeled prior to testing — e.g. the melon is tested with the rind, the banana is tested with the peel — so that the residue levels found tend to overstate the amount of pesticides consumed when the fruit is eaten. Because of these biases built into the FDA surveillance protocols, data from this program were used only as a last resort. And as discussed below, when they were used, a reduction factor was applied to the residues found on each sample, to better estimate actual exposure.

FDA Total Diet Study. The second FDA pesticide residue monitoring program is the Total Diet Study (TDS). The TDS was started in the early 1960s to study the prevalence of radioactive fallout in the food supply as a result of atmospheric nuclear weapons testing. Today, the program tests 234 different foods four times a year for a host of contaminants. The 234 foods sampled are determined to be representative of the U.S. diet. The entire sample is purchased at grocery stores four times each year, one in each of four geographic region of the country. This "market basket" covers a broad range of both processed (bottled, canned, frozen) and fresh foods including fresh fruits and vegetables, as well as baby food, dairy products, frozen meals, fresh meats, cereals and peanut butter, and prepared foods like pizza.

Prior to testing, the foods are prepared as they normally would be in the home. Bananas are peeled, tuna casserole is baked, rice is boiled, and the hamburger is grilled. The prepared food is then analyzed for pesticides and other toxic contaminants.

We analyzed all records from the FDA surveillance database from the years 1992 through 1995, which contained residue findings for 51,280 food samples.

To our knowledge, the Total Diet Study data have never before been assembled in electronic form, nor have they ever been released to the public in their entirety in any form.

Unlike the FDA surveillance data, the TDS data are designed to provide a representative snapshot of contaminants in the U.S. diet. The biggest shortcoming of these data is that the sample sizes tend to small (4 samples of each food per year). The strength of the program is that it provides real world data that reflect pesticide residues very likely encountered by the average person. In addition, the TDS uses powerful analytical techniques that can detect low level residues of pesticides in the range of 1 part per billion or less, a significant advantage over the FDA surveillance program which does not employ such advanced technology.

After several years of repeated inquires, multiple Freedom of Information Act requests, and many rounds of discussions with FDA staff, Environmental Working Group received TDS data for the years 1991 through 1996 in electronic form. These six years of data contain 4,520 food samples that were analyzed for pesticides. To our knowledge, these data have never before been assembled in electronic form, nor have they ever been released to the public in their entirety in any form.

Toxicity Data

EPA Reference Doses. The combined toxicity of organophosphate insecticides is measured in terms of cholinesterase inhibition. The standard of safety used to protect the pol;ulation from these effects, is known as a reference dose or RfD. Specifically, the reference dose is the agency's determination of a safe daily dose of a pesticide, or in this case, the dose of OPs that will produce no adverse cholinesterase effects, expressed in milligrams of pesticide per kilogram of body weight per day (mg/kgbw/day). The reference doses (RfDs), used in this report are the most recently calculated reference dose values used by EPA scientists, obtained directly from the EPA in January 1998.

All of these reference doses represent final agency decisions, except that for chlorpyrifos. At a recent meeting, the reference dose committee of the Office of Pesticide Programs recommended that the reference dose for chlorpyrifos have an additional ten-fold safety factor, per the requirements of the Food Quality Protection Act to protect infants and children from pesticides. The reference dose committee is the pesticide program committee of scientists charged with making recommendations for pesticide safety standards under the FQPA. Generally, recommendations from the reference dose committee have been adopted as agency health standards.

The chronic RfD was chosen as the appropriate measure of toxicity in this study only after our initial analysis revealed that on average, 88 percent of all children 5 and under were exposed to at least one OP each

day. Use of an acute cholinesterase RfD in a situation where nearly 90 percent of the study population is exposed each day to the pesticides being studied, would be inappropriate.

The chronic reference dose is derived from any of a number of animal toxicity tests required by the EPA. These studies range from the 90-day rat study, to a two-year chronic feeding study. The test animals are usually rats or dogs, but, for example, the RfD for chlorpyrifos is based on data from a study conducted on 16 adult male Dow chemical employees in 1972. RfDs for pirimiphos-methyl, ethion and diazinon are also based on human data (Table 5).

Exposure Assessment

Food Consumption Data.

Each year of CSFII data contained from 150 to 200 individuals per age group (one-year-olds, two-year-olds etc.). Each individual reported from one to three eating days that were validated by USDA. An eating day can be thought of as all the food reported eaten by one individual on one day. Only eating days with complete information and positive validation by USDA were used. The five years of CSFII data used in the report contained a total of 8,302 valid eating days for children age six months through five years. Age group cohorts were constructed by combining individuals of the same age from the five years of CSFII data used in the analysis.

Table 5. EPA reference doses (RfD) used in this analysis.

Pesticide	Reference Dose RfD (mg/kg/day)	Uncertainty Factor	Critical Studies Used	Critical ChE Effect	NOEL Used (mg/kg bw)	Company that Submitted Study	Date of Study
acephate	0.0012	100	Rat - 90 day	plasma	0.12	Chevron Chemical	1987
azinphos methyl	0.0015	100	Dog - 1 year	RBC	0.15		
chlorpyriphos	0.0003	100*	Human - 28 day	brain	0.03	Dow Elanco	1972
diazinon	0.0007	30	Human	plasma	0.02		
dichlorvos	0.00017	300	Dog - 1 year	brain, RBC, plasma	0.05	AMVAC Chemical	1990
dimethoate	0.0005	100	Rat - 2 year	RBC	0.05	American Cyanamid	1986
ethion	0.0005	100	Human	plasma; brain	0.05	FMC	1970; 1988
malathion	0.04	100	Rat - 2 year	RBC, plasma	4.0	Moeller and Rifder	1962
methamidophos	0.001	100	Rat - 8 week	?	0.1		
methidathion	0.0015	100	Dog - 1 year	?	0.15		
methyl parathion	0.00002	1,000	Rat - 2 year	brain, RBC, plasma	0.02	Monsanto	1984
phosmet	0.003	300	Rat - 2 year	brain, RBC, plasma	1.1	Stauffer Chemical	1967
pirimphos methyl	0.00008	3,000	Human - 56 day	plasma	0.25	ICI Americas Corp.	1974, 1976

Source: EPA, Office of Pesticide Programs, January 1998.

RBC = red blood cell
ChE = cholinesterase inhibition
RfD or Reference Dose = derived from the NOEL in the "critical or most sensitive study which is then divided by a variable "uncertainty factor" and determined to be the daily dose or exposure at which no harm should occur over a lifetime.
A lower reference dose (RfD) means the pesticide is thought to be more potent.
NOEL = No Observed Adverse Effects Level

The goal of the exposure analysis was to produce the most accurate real world picture of pesticide exposure via the diet.

Survey participants through five years of age reported eating about 3,695 different foods. Many of these different foods, however, are nearly identical versions of the same food. For example, orange juice drinks that would be considered different foods in the CSFII include unsweetened orange juice, orange juice with sugar, orange juice with calcium, orange juice from concentrate and fresh orange juice. For purposes of linking food consumption data with residue data in this report, these similar foods are considered the same food. The federal pesticide residue databases used in this analysis contained residue results for 561 of the 3,695 foods reported eaten by children six months through five years of age. These 561 foods account for 68 percent of the diet of these children. Of these 561 foods, 407 were found to contain detectable levels of OP insecticides. Our method for matching the specific foods reported eaten, with residue testing results, is described below.

Residue Data. The goal of the exposure analysis was to produce the most accurate real world picture of pesticide exposure via the diet. To achieve that end the three residue databases described above were used in the analysis, in the following order of priority. For fruits and vegetables eaten raw, PDP data were used because the data represent residues after washing and peeling, and because samples are statistically reliable and representative of U.S. food consumption. For all other non-processed foods, FDA surveillance data were used. These data provide large sample sizes, but generally overstate residues at the time of consumption. To account for this, a residue reduction factor of from 25 to 90 percent was applied to all FDA surveillance data (Table 6). The reduction factors are based on actual reductions observed when PDP and FDA surveillance data for individual OP insecticides were compared on similar fruits and vegetables.

For processed and cooked foods, data from the FDA Total Diet Study were used. The small sample sizes in the TDS created some concern that TDS data might overstate exposure to some OPs. For example, the six years of TDS data provide to EWG contained only 16 samples of wheat bread, but all of them were positive for OP residues. Using these 16 samples to represent the entire U.S. wheat bread supply might overstate OP exposure via wheat bread. On the other hand the residues in these products, while ubiquitous, were generally at low levels, and not likely by themselves to present great risk to any consuming individual.

To test the validity of the bread product residue findings, we examined OP residue data in all of the more than 600 samples of processed wheat

Table 6. Pesticide residue reductions used in EWG analysis.

95% reduction
Heavy peeled fruits and vegetables

Corn
Peanuts, in shell
Lemon
Lime
Tangerine
Watermelon
Honeydew Melon
Cantaloupe
Plaintain
Kiwi Fruit
Avocado
Mango
Papaya
Pineapple

90% reduction
Spinach-like crops (raw to cooked)

Raw Collards
Mustard Greens
String Beans, cooked
Green Peas, cooked

85% reduction
Lettuce-like crops (washed w/ outer leaves removed)
Chicory Leaf
Lettuce, Loose Leaf
Boston Lettuce
Other Lettuce
Endive, Escarole
Radicchio
Green Cabbage
Chinese Cabbage
Red Cabbage
Cauliflower
Brussels Sprouts

75% reduction
Juice concentrate uncertainty
Apple Juice
Apple Cider
Orange Juice
Orange Juice (baby food)
Grape Juice
Grape Juice (baby food)

70% reduction
Carrot/root vegetable comparison
Onions
Sweet Potatoes
Sugar Beets
Red Beets
Turnips
Radishes

50% reduction
Core and pit fruit comparison (compared apples and peaches: FDA to PDP)
Apricots
Dried Apricots
Apricot Paste
Peach
Pear
Cherries
Nectarines
Pear
Plums
Grape comparison (FDA to PDP)
Blackberries
Blueberries
Cranberries
Raspberries
Strawberries

Celery-like crops comparison (FDA to PDP)
Asparagus
Leeks
Scallions

Others
Mushrooms
Olives, Green, Black, Stuffed

25% reduction
Green bean comparison (compared green beans - FDA to PDP)
Tomatoes
Green Tomatoes
Cucumbers
Eggplant
Hot Peppers
Poblano Peppers
Serrano Peppers
Green, Red or Sweet Peppers
Banana Peppers

Pesticide residues found in fresh produce by the FDA surveillance program and USDA Pesticide Data Program were reduced by 25 to 95 percent to more accurately reflect the levels likely to be found in cooked and prepared foods.

Reduction factors were derived by either comparing data from the FDA surveillance program with levels of OPs reported in Total Diet Study or the Pesticide Data program.

As a general rule foods were linked with residue values only when a direct match between the two foods was available.

products in the TDS. From pasta to pretzels, to wheat bread and wheat breakfast cereal, more than 99 percent of more than 600 samples tested for pesticides were positive for either chlorpyrifos, malathion, or both. This strongly suggests that the low level OP residues reported in the TDS for any single processed wheat product are very likely representative of the commodity as a whole. To increase the sample size for baby food, tests of baby food for pesticides commissioned by EWG in 1995 were added to TDS data from FDA. The results from both TDS and EWG were quite similar.

Of the 39 OP insecticides with a common mechanism of toxicity, only 13 were detected in the food supply. These 13 OP compounds, in turn, were found on 407 of the 3,695 foods reported eaten by children age five and under in the USDA survey.

Linking Food and Residue Data. More than 3,695 food items were reported eaten by children under age five in the CSFII. For purposes of predicting pesticide exposure, however, many of these 3,695 foods can be considered the same food. For example, it is reasonable to assume that cooked carrots with fat, cooked carrots without fat, and cooked carrots (fat unspecified), are the same in terms of pesticide residues. Many other decisions were not that straight forward. Links between foods reported eaten, and residue findings were made as described below. As a general rule foods were linked with residue values only when a direct match between the two foods was available. Any deviation from this rule is described below.

For fruits and vegetables eaten raw, food consumption values were matched first to data from PDP, when available, and then with FDA surveillance data with a residue reduction factor applied. Frozen fruits and vegetables (not canned) were assumed to have the same residue levels as fresh fruits and vegetables and the same residue values were applied. For fruits and vegetables eaten cooked, either from canned or fresh vegetables, residue values from the total diet study (TDS) were used.

For all other processed and cooked foods that were reported in the CSFII, TDS data were used when a direct match was available. For thousands of specific foods reported eaten by the population studied — cherries jubilee, pepperoni pizza, all soft drinks — no direct matches were available in the residue files. These foods were not used in the analysis. For example, we did not attempt to match the pepperoni pizza consumption data with OP residue data from cheese pizza, because of uncertainty about the exact weight ratio of the foods that constitute each respective pizza. Likewise we did not match cherry pie residues with cherries jubilee consumption data, and so on for thousands of foods

with no direct match. With sandwiches, consumption data was matched with residue data only when the sandwich consumption data was reported in its component parts that matched the residue data. For example, when a peanut butter and jelly sandwich was reported as X grams of bread, X grams of peanut butter and X grams of jelly, these consumption values were matched with corresponding test results from the TDS. When the sandwich was simply report as a peanut butter and jelly sandwich, it was not used.

The one technical exception to this rule was with wheat products in the form of pasta and bread. In this case residue data were available for white bread, wheat bread, macaroni and spaghetti. Children age five and under, however, reported eating many types of pasta (spaghetti, macaroni, lasagna noodles etc.) and many different types of bread (French bread, Italian bread, pita bread etc.). In this case, any wheat based bread or pasta was matched with the residue values from the most closely matched wheat based bread or pasta products in the TDS.

The Monte Carlo. The exposure assessment is a Monte Carlo style probability distribution analysis designed to simulate real world dietary exposure to OP pesticides using the best available data. The analysis was modeled after that used by the National Research Council Committee on Pesticides in the Diets of Infants and Children (NRC 1993 pp. 297 through 307).

Dietary exposure to OPs was analyzed in 24 hour units to match the toxicity of OPs which are active for a least 24 hours after they are consumed. For example, an OP eaten at breakfast, will remain active in the body in the afternoon and can be added, for purposes of risk assessment, to an OP eaten at dinner.

The program was run on a Power Mac 8100 using FoxPro software. A distribution of dietary OP exposure was simulated for each age group year (one-year-olds, two-year olds etc.) The distribution was created by instructing the computer to identify a valid individual eating day in the database (person one, day one) and to match each food eaten by that individual on that day with a randomly selected residue result from all the samples for that food in the residue database described above. Total daily exposure to each of the thirteen individual OPs in the residue files was then calculated and converted to a mg/kg exposure value for each OP consumed, depending on the amount of the food consumed, the residue(s) found on the sample that was selected (zeros were included as reported in the data), and the weight of the child.

For example, if a child ate 100 grams (a little under four ounces) of green beans and the green bean sample, randomly chosen from the residue database, had 1

The analysis was modeled after that used by the National Academy of Science Committee on Pesticides in the Diets of Infants and Children.

part per million (ppm = mg/kg) of acephate, the program would calculate that 1 mg/kg acephate x 100 grams of beans = 0.1 mg of acephate on those beans. If the child weighed 10 kg, the *dose* of acephate that child got from those green beans would be 0.1 mg/10 kg = 0.01 mg/kg body weight acephate.

For each of the valid eating days available for each age year, this process was repeated 2,000 times, to produce a distribution of three to four million individual exposure days, per age group, for each of the OP compounds.

Risk Assessment

Each of the three to four million individual exposure days for each age group contains a total mg/kg exposure value for each of the OPs for which residue data were available. For example, individual #2,789,450 might have eaten 0.3 mg of acephate, 0.04 mg of azinphos methyl and so on for all thirteen OPs found in food.

Conversion to Chlorpyrifos Equivalents. To assess the risk of this exposure, an individual's total OP exposure on any given day was then converted to chlorpyrifos equivalents. To do this, a chlorpyrifos toxic equivalency factor (TEF) was applied to convert the mg/kg dose of each OP to the appropriate dose of chlorpyrifos. This TEF accounts for the difference between the reference dose of chlorpyrifos and the reference dose of any other OP.

A conversion factor for pesticide X would be calculated by dividing the reference dose for chlorpyrifos by the reference dose for pesticide X. For example, the proposed reference dose for chlorpyrifos is 0.0003 mg/kg and the reference dose for pesticide X was 0.0001 mg/kg. The conversion factor for pesticide X would be 0.0003/0.0001, or 3, meaning that pesticide X is three time more toxic than chlorpyrifos. To express the dose of pesticide X in chlorpyrifos equivalents, one would simply multiply the mg/kg dose of pesticide X by 3, and so on for all of the OPs. Total daily exposure is then calculated as the sum of chlorpyrifos equivalents for each OP, on any given day. An individuals total daily OP exposure, expressed in chlorpyrifos equivalents, can then be compared to the chlorpyrifos reference dose. Using samples weights in the USDA food consumption, one can then estimate the number of children in the U.S. population that will exceed appropriate safety margins each day.

This procedure differs slightly from the method used by the National Research Council Committee on Pesticides in the Diets of Infants and Children. The committee conducted a similar Monte Carlo analysis and converted exposure to chlorpyrifos equivalents using "no observable effect levels" (NOELs) instead of reference doses (RfD). An RfD, which is the functional equiva-

lent of what EPA deems a "safe" daily dose of the pesticide, is derived by dividing the NOEL by a specified safety factor. Safety factors differ from pesticide to pesticide, depending on the quality of the data and the effects observed in critical studies.

Initially we employed a methodology similar to that used by the NRC committee. However, the results produced by this analysis (the number of children exposed to levels that exceed a specific safety margin) were entirely dependent on the safety factors applied to the various NOELs (Table 5). In essence, when NOELs are used to convert the toxicity of multiple pesticide exposures to a baseline compound, the number of children that exceed the reference dose changes, depending on the pesticide chosen as the baseline compound. For the results to be meaningful and unbiased, the estimated number of children exposed to OPs in food at levels that exceed a specific safety margin must be the same, regardless of the chemical chosen as the baseline pesticide. Basing the TEF on RfDs corrects this problem.

For example, the chlorpyrifos proposed RfD is based on a 100-fold uncertainty factor applied to a NOEL from a study on humans, whereas methyl parathion is based on a 1,000-fold uncertainty factor applied to a NOEL from a study on rats. When conversions were based on the NOELs, using methyl parathion as the baseline pesticide put 9.1 percent of all one-year-olds over the RfD, whereas using chlorpyrifos as the baseline chemical put only 3.2 percent of these same one-year-olds over the RfD. In contrast, when the conversions are based on the RfDs, the analysis yields the same percentage of one-year-olds exposed to an unsafe dose of OPs on any given day (5.2 percent) regardless of the pesticide chosen as the baseline compound.

Findings

After eighteen months of research and analysis of all relevant federal data on food consumption, pesticide residues, and OP toxicity, as well as a thorough literature review on the toxicity of the OPs a number of basic findings emerge.

First, the EPA has more than enough valid data at its disposal to conduct a similar analysis and to meet the deadline for regulation of the OPs imposed by FQPA. There are no limitations in the existing taxpayer-supported food consumption or pesticide residue monitoring programs that are so serious as to undercut the timely completion of the agency's regulatory charge. EWG is prepared to cooperate fully with the agency in analyzing existing data and in developing the capacity to conduct analyses in-house.

The analysis reveals several important facts about dietary exposure to OP pesticides. Every day, nine out of ten American children from ages six months through 5 years are exposed to OP insecticides in the food they eat. The foods with the highest percentage of contamination are the processed grain products, particularly wheat products such as breads and pasta. Virtually 100 percent of all processed wheat products are contaminated with low levels of chlorpyrifos, malathion, or both.

While almost always contaminated, the levels found on grain products are typically low. The foods with the most toxic combination of OPs are apples, peaches, grapes and pears. The OPs that placed children at the greatest risk, were methyl parathion, dimethoate, chlorpyrifos, pirimiphos methyl, and azinphos methyl.

Using reference doses (RfDs) updated by the EPA on January 14, 1998, we estimate that every day, more than 1.1 million children age six months through 5 years exceed the current safe daily dose of OPs, the so-called reference dose, set by the EPA. More than one hundred thousand of these children exceed the EPA safe dose by a factor of ten (Table 7). Only three of the safe daily doses, or reference doses, set by EPA for the 13 OP insecticides found in food, contain added protections to shelter infants and children from the toxic effects of OPs.

EPA has more than enough valid data at its disposal to conduct a similar analysis and to meet the deadline for regulation of the OPs imposed by FQPA.

Table 7. More than one million children under age 6 eat an unsafe dose of organophosphate insecticides each day.

Age	Estimated number of children exceeding "safe" dose per day	Percent of population	Children exceeding 10 times the "safe" dose per day	Children exceeding 100 times the "safe" dose per day
6-12 mo.	110,900	5.5%	6,400	100
1	206,200	5.2%	20,500	900
2	219,300	5.5%	21,400	1,000
3	222,200	5.6%	25,000	900
4	191,900	4.8%	16,300	500
5	192,000	4.8%	17,000	800
Total	**1,142,000**		**107,000**	**4,200**

(Figures rounded to nearest hundred)
Source: EWG, compiled from USDA food consumption data 1989-1995, USDA and FDA pesticide residue data 1991-1996 and reference doses (RfDs) obtained from EPA in January 1998.

For most of the children put at risk each day, the problem is caused by children doing what health experts want them to do, eating fruits or vegetables.

All the pesticide exposure levels used in the risk assessment represent actual residues found or estimated to be present after washing, cooking, processing or otherwise preparing the food for consumption. Data were acquired from the U.S. Food and Drug Administration and the Department of Agriculture. Food consumption amounts are based on more than 8,300 days of food consumption reported by more than 4,000 children surveyed by the USDA during the years 1989 through 1995. Toxicity data were provided by the EPA, Office of Pesticide Programs on January 14, 1998.

High risk foods

For most of the children put at risk each day, the problem is caused by children doing what health experts want them to do, eating fruits or vegetables.

The most contaminated foods. A significant percentage of several fresh fruits expose children to unsafe levels of OP insecticides. For example, our analysis indicates that one out of every four times (25 percent of the time) a child under six years of age eats a peach, he or she exceeds the EPA (adult) safe does of OPs. Thirteen (13) percent of the time a child of this age eats an apple, he or she exceeds the EPA (adult) safe dose of OPs (Table 8). Apples are followed by nectarines (12 percent), popcorn (8.5 percent), and pears at (7.5 percent).

Commercial Baby Food. OP compounds in commercial baby food present real risks to infants. We estimate that nearly ten percent of the time an infant between six and twelve months of age ate pear baby food, he or she exceeded the (adult) daily safe dose of OPs. Baby food apple juice and peaches caused this problem 5 and 2.4 percent of the time respectively.

Foods that put the greatest number of children at risk. The foods that put the greatest number of children at risk are not usually those with the highest percentage of unsafe OP residues, but instead are those that have moderate to significant residues of OP, *and* are widely consumed in significant amounts.

Infants Six to Twelve Months. For infants six to twelve months of age, commercial baby food is the dominant source of unsafe levels of OP insecticides. OPs in baby food apple juice, pears, applesauce, and peaches expose about 77,000 infants each day, to unsafe levels of OP insecticides (Table 9).

One through Five Year Olds. Apples, apple juice, and apple sauce expose the most children age one through five years of age to unsafe levels of OPs. In fact, just over half of the children that eat an unsafe level of OPs each day, receive this unsafe dose from apple products alone. We estimate that each day, fresh, raw, apples expose more than 400,000 children age one through five to unsafe levels of OP insecticides (Table 9). When all apple products are included, the number jumps to 575,000 children per day.

Peaches and grapes are also significant source of unsafe OP exposure, putting 77,000 and 54,000 young children over the safe dose each day.

Just over half of the children that eat an unsafe level of OPs each day, receive this unsafe dose from apple products alone.

Foods that expose the most children to the most toxic dose of OPs

High consumption food items expose many children to levels of OPs that exceed safe levels

Table 8. Infants often exceed safe levels of organophosphate intake when consuming one food item at one meal.

	Likelihood of being exposed to an unsafe dose of organophosphates						
Food	6-12 months	1 year olds	2 year olds	3 year olds	4 year olds	5 year olds	Average of Age Groups
Peaches	24.2%	25.7%	26.9%	25.6%	24.0%	22.4%	24.8%
Apples	12.0%	12.2%	14.1%	13.8%	13.1%	12.1%	12.9%
Nectarines	11.6%	14.1%	8.3%	12.1%	15.3%	11.8%	12.2%
Popcorn	0.0%	10.5%	10.2%	10.4%	10.0%	10.1%	8.5%
Pears	11.6%	6.9%	7.5%	6.1%	6.5%	6.1%	7.5%
Cornbread	5.6%	5.3%	6.1%	6.0%	5.7%	5.0%	5.6%
Applesauce	3.7%	5.7%	5.9%	5.8%	5.4%	4.7%	5.2%
Grapes	5.6%	4.9%	5.4%	5.3%	4.5%	4.9%	5.1%
Corn Chips	2.9%	3.8%	4.7%	5.6%	5.3%	4.8%	4.5%
Pears (baby food)	9.6%	13.4%	0.0%	0.0%	0.0%	0.0%	3.8%
Raisins	2.2%	2.9%	4.8%	3.4%	3.6%	2.7%	3.3%
Cherries	0.0%	3.3%	4.5%	3.7%	3.5%	4.3%	3.2%
Kiwi	0.0%	1.5%	3.7%	5.0%	4.2%	2.7%	2.9%
Peaches (baby food)	2.4%	4.6%	0.0%	7.1%	0.0%	0.0%	2.4%
Apple Juice (baby food)	5.0%	6.8%	0.0%	0.0%	0.0%	0.0%	2.0%

Source: EWG, compiled from USDA food consumption data 1989-1995, USDA and FDA pesticide residue data 1991-1996 and reference doses (RfDs) obtained from EPA in January 1998.

Table 9. More than half a million American infants get an unsafe dose of organophosphate insecticides from apples and apple products.

Estimated number of children exceeding safe dose/day from individual foods							
Food	**6-12 months**	**1 year olds**	**2 year olds**	**3 year olds**	**4 year olds**	**5 year olds**	**Total**
Apples	2,630	**53,940**	**87,100**	**95,160**	**84,190**	**85,670**	**408,700**
Peaches	3,520	19,710	12,060	20,300	8,610	13,250	77,400
Applesauce	5,390	16,190	11,590	13,320	9,900	13,750	70,200
Popcorn, popped in oil	0	10,340	16,110	12,600	17,060	12,270	68,400
Grapes	1,220	9,730	12,660	12,080	8,800	10,150	54,600
Corn Chips	210	4,740	10,240	11,600	11,650	14,640	53,100
Apple Juice	2,790	14,430	11,440	8,400	6,160	2,830	46,100
Oat Ring Cereal	490	12,390	12,610	9,250	5,930	4,930	45,600
Apple Juice (baby food)	**36,530**	6,870	0	0	0	0	43,400
Pears (baby food)	24,370	8,690	0	0	0	0	33,100
Cornbread	1,640	4,840	5,070	5,860	5,590	4,810	27,800
Raisins	80	4,890	7,040	4,300	3,900	1,590	21,800
Pears	850	4,320	4,750	2,150	3,090	2,260	17,400
Applesauce (baby food)	8,250	2,050	680	0	0	0	11,000
Peaches (baby food)	7,760	3,000	0	210	0	0	11,000
Tomatoes	0	3,460	1,490	2,040	1,450	1,800	10,200
Oatmeal	1,120	3,220	1,420	1,640	1,480	590	9,500
Nectarines	420	1,010	1,180	1,780	1,370	1,840	7,600
Green Beans	340	1,560	1,140	1,900	1,340	1,060	7,300
Lettuce	0	430	710	840	1,060	1,310	4,400
Peas	70	970	950	540	560	660	3,700
Strawberries	0	460	760	540	560	850	3,200
Baked Beans	0	480	540	610	530	810	3,000
Plums	0	540	570	670	830	340	2,900
Kiwi	0	180	430	1,170	500	660	2,900
Celery	0	330	400	450	980	600	2,800
Grapefruit	0	0	400	0	1,320	660	2,400
Green Peppers	0	0	730	380	300	950	2,400
Carrots	0	110	290	190	300	400	1,300
Oranges	10	120	230	140	130	130	800
Total for all foods	**110,920**	**206,150**	**219,320**	**222,230**	**191,870**	**191,970**	**1,142,500**

Source: EWG, compiled from USDA food consumption data 1989-1995, USDA and FDA pesticide residue data 1991-1996 and reference doses (RfDs) obtained from EPA in January 1998.

by wide margins. OPs on washed apples, peaches, grapes, pears and pear baby food cause 85,000 children each day to exceed federal safety standards by a factor of ten or more (Table 10).

A small but real percentage of some foods are so loaded with OP insecticides that virtually any consumption will put a child over the safe daily dose. For example, roughly 2 percent of the apples, grapes, raisins and pears have such a potent dose of OP insecticides that eating 10 grams of these fruits (the equivalent of two grapes) would cause the average 25 pound one-year-old to exceed the daily OP safety standard (Table 11). Peaches topped the list at 15 percent, meaning that 15 percent of 1,500 peaches tested had residues of OPs where 10 grams of consumption (about three bites) by the average sized one year old, would put that child over the safe daily exposure level for

OPs. (All pesticide residues in the analysis were measured after the produce samples were washed and otherwise prepared for normal consumption by USDA technicians).

A child who eats an average amount of these foods has an excellent chance of exceeding the (adult) safe daily dose of OPs. For example, 26 percent of the time an average amount of peaches was eaten by a one year old, that single dose put that child over the safe daily dose of OPs. Apples put the average one year apple eater over the safety standard for OPs 13 percent of the time, followed by pears and grapes at about 7 and 6 percent (Table 11).

A child who eats a lot of these foods is even more likely to exceed the (adult) safe dose of OPs. About 22 percent of the one-year-old apple eaters that eat at the 90th percentile of reported consumption (the equivalent of about one apple), exceed the (adult) safe daily dose of OP insecticides. Heavy (90th percentile) consumption of pears put 11 percent of the one-year-old pear eaters over the daily safety standard, followed grapes at 7.5 percent and raisins at 6.3 percent (Table 11).

Table 10. More than 100,000 American children under six are exposed to 10 times the safe daily dose of organophosphate insecticides in food each day.

Food	Estimated number of children exposed to 10 times the "safe" daily dose of OPs/day
Apples	34,600
Peaches	30,900
Grapes	12,560
Pears (baby food)	4,610
Pears	4,040
Cornbread	3,700
Baked Beans	2,640
Nectarines	2,420
Raisins	2,230
Popcorn, popped in oil	880
TOTAL for all foods	**106,600**

Source: EWG, compiled from USDA food consumption data 1989-1995, USDA and FDA pesticide residue data 1991-1996 and reference doses (RfDs) obtained from EPA in January 1998.

High Risk Pesticides

There are 39 OP insecticides registered for use on food crops in the United States. Only a subset, however, are found on food. Our analysis of more than 80,000 samples of food inspected by the federal government for pesticide residues from 1991 through 1996, revealed that only 13 organophosphate insecticides were found in or on food by the Food and Drug Administration and the U.S. Department of Agriculture.

26 percent of the time an average amount of peaches was eaten by a one year old, that single dose put that child over the safe daily dose of OPs.

The highest risk OP compounds are methyl parathion, dimethoate, chlorpyrifos, pirimiphos methyl, and azinphos methyl which account for about 90 percent of the risk from OP insecticides in the infant and child diet.

Achieving a safe food supply for children, however, is not as simple as banning the five highest risk OPs. Home and other

Table 11. Eating an average amount of some fruits can easily expose a child to an unsafe daily dose of organophosphate insecticides.

	Percent of children exposed to an unsafe dose of organophosphate insecticides by eating:					
	10 grams		100 grams		the average amount a child consumes	90th percentile consumption*
Apples	1.9%	3 bites or 1/14 of an apple	17.9%	~3/4 apple	13.7%	22.1%
Grapes	1.6%	2 grapes	7.5%	20 grapes	5.9%	7.5%
Peaches	15.3%	3 bites or 1/9 of an peach	28.4%	~1 peach	26.3%	28.4%
Raisins	2.1%	1 small box	12.5%	~3/4 cup	2.1%	6.3%
Pears	2.1%	3 bites or 1/15th of a pear	7.5%	~3/4 pear	6.8%	11.1%

* highest 10 % of consumption

Source: EWG, compiled from USDA food consumption data 1989-1995, USDA and FDA pesticide residue data 1991-1996 and reference doses (RfDs) obtained from EPA in January 1998.

Infant and child safety must be measured in terms of safety standards designed to protect these children, not in terms of the current adult-based standards.

non-food uses must be considered, as well as the fact that other OPs will likely substitute for those that are removed from use in the first wave of standard setting. And most importantly, infant and child safety must be measured in terms of safety standards designed to protect these children, not in terms of the current adult-based standards.

Regulatory Options. EPA policy is that no more than 0.1 percent (1 in 1,000) of the infant and child population should be exposed to levels of pesticides that exceed safety margins on any given day. Current levels of OPs in food fail this standard of safety miserably, and again, the youngest are at greatest risk. We estimate that more than 5.2 percent (52 in 1,000) of American children age 6 months through 5 years, or more than 1.1 million children, exceed EPA safety standards for OP compounds on any given day.

The following scenarios describe the outer bounds of the spectrum of regulatory options available to the agency. Scenario one represents the absolute minimum amount of regulatory action that the agency must take just to begin to protect infants and children from OPs. This scenario does not include the ten-fold safety factor required by FQPA to account for lack of data on OP toxicity and exposure to infants and children. Scenario two, describes the other extreme, where an additional ten-fold level of protection is applied and is ultimately supported for every OP currently in use.

Both scenarios assume that all non-food uses of OP compounds are banned, and that as the high risk OPs are banned on food, the remaining OPs will substitute for all of the uses of the OPs that are banned.

Scenario One. It is possible to achieve a 99.9 percent level of protection by:

- banning the five OPs that present the greatest risk — methyl parathion, dimethoate, pirimiphos methyl, chlorpyrifos, and azinphos methyl.
- assuming zero non-food exposure to OP insecticides,
- using current safety standards which do not incorporate needed protections for infants and children, and
- prohibiting all OP compounds in commercial baby food.

Until reliable data on fetal and infant toxicity of OPs are available, however, this scenario can only be said to achieve adequate protection for adults.

Scenario Two. When the required extra ten-fold safety margin is added to the 10 OPs left on food after scenario one, all of the remaining OPs in food must be banned to achieve a 99.9 percent level of protection for infants and children. It is possible that an additional ten-fold safety factor will not be required for all OPs when the relevant data are made available on the effects of OPs on the developing fetus and infant (although the data may dictate this outcome). However, if developmental neurotoxicity studies are not conducted on these compounds, FQPA requires an additional ten-fold level of protection for infants and children.

Federal law requires extra protections for children

The Food Quality Protection Act (FQPA) requires the EPA to *act* to protect infant and child health, even in the absence of total scientific certainty regarding the toxicity or exposure of pesticides to the fetus, infant or young child. This is a dramatic reversal of previous statutory requirements where EPA had no mandate, and arguably could not act to protect the public health, even child health, in the absence of complete data on the risk from a pesticide. Now the law is clear, when there is incomplete data the pesticide's toxicity (pre- and postnatal) *or* incomplete data on infant and child exposure to that same pesticide, the EPA must err on the side of child safety and apply an additional ten-fold margin of safety to food tolerances for that pesticide.

If EPA were to grant infants and children the protection from OPs required by law and apply additional safety factors, we estimate that an additional 3.6 million infants and children five and under exceed the new safety standard each day (16.3 percent of the population). More than one in five infants age six through 12 months (over 400 thousand or 20.8 percent of the population) would exceed the safe level of exposure dictated in the FQPA (Table 12).

Now the law is clear, the EPA must err on the side of child safety.

Table 12. Nearly 3.6 million children 5 and under are exposed to levels of organophosphate insecticides in food that exceed new FQPA safety standards.

Age	Percent and number of children exceeding new health standards	
6-12 mo	20.8%	416,000
1	17.3%	692,300
2	16.4%	655,700
3	16.4%	657,300
4	14.6%	584,200
5	14.8%	592,500
Total	**16.3%**	**3,594,400**

Source: EWG, compiled from USDA food consumption data 1989-1995, USDA and FDA pesticide residue data 1991-1996 and reference doses (RfDs) obtained from EPA in January 1998.

Our analysis of exposure to multiple OPs in the infant and child diet compares this total exposure to an adult safety standard.

Toxicity Data

The risk assessment portion of this analysis — the estimates of the numbers of children exceeding the EPA safe dose of OPs each day — is based on the most recent data available from the EPA. The central flaw with the estimates, is that they compare a child's total daily dose of OPs to a reference dose (a safe daily amount) that is derived from studies on adult animals, and in most cases (10 out of 13) the references doses contain no additional protection or "safety margins" to protect infants or children. The result is that our analysis of exposure to multiple OPs in the infant and child diet compares this total exposure to an adult safety standard. This leaves us in the awkward position of only being able to say that a one-year-old's dietary exposure to OPs is safe or unsafe for adults. This is very much like saying that as long as a one-year-old does not exceed that adult daily dose of acetaminophen, that this child is safe.

There is consistent and compelling evidence in the peer-reviewed literature that the fetus, infant and young child are more vulnerable to a wide variety of toxic effects caused by OP insecticides. These effects include increased sensitivity to cholinesterase effects as well as increased vulnerability to a variety of toxic effects on the nervous system that occur during critical periods of prenatal, neonatal, and post-neonatal brain development.

None of the OPs widely found in the food supply have been tested for their developmental neurotoxicity (one OP, chlorpyrifos, is currently being tested), nor are there plans to test the OPs for their developmental neurotoxicity (SAP 1996, SAP 1997). This data gap is critical because the standard battery of toxicity tests does not reveal any significant age-related neurotoxicity, nor are animals examined for long term behavioral or functional effects unless gross brain or nervous system abnormalities occur in the developmental and reproductive toxicity studies. Indeed, the standard battery of toxicity tests would not detect such well known developmental neurotoxicants as lead, PCB's, and mercury. EPA Assistant Administrator for Prevention, Pesticides, and Toxic Substances, Dr. Lynn Goldman concurs: "We agree that the stan-

dard protocols for developmental toxicity studies and reproductive toxicity studies do not provide much information on the effect of neurotoxic pesticides on the performance of the nervous or immune systems in developing animals" (Goldman 1997).

Unlike data generated under EPA protocols, the peer reviewed literature shows a consistent and repeated pattern of behavioral and functional deficits from low level OP exposure in the absence of any overt toxic effects, usually with no correlation to cholinesterase levels, and in some cases in the absence of significant ChE depression (See chapter 2).

Current regulation of OPs is based almost exclusively on ChE effects. The typically greater sensitivity of young animals to these effects alone argues for increased protection for children from OPs, and in and of itself supports the FQPA requirement for an additional ten-fold level of protection in the absence of reliable data to the contrary. Their is mounting evidence, however, that OPs are toxic to the developing brain and nervous system at very low levels of exposure. Again, both current studies and the regulations they support do not account for these toxic effects. Admittedly, many of the more subtle and long term neurodevelopmental effects emerging in the literature today may not be ripe for use as regulatory endpoints. This growing body of evidence, however, plainly supports the need for greater protection from OPs for infants and the developing fetus and embryo.

Conclusions

American children are routinely exposed to unsafe levels of OP insecticides in the food they eat. On any given day we estimate that more than one million children under age six exceed federal safety standards for OPs. One hundred thousand of these children exceed these same standards by a factor of 10 or more. The potential public health impact of these exposures is substantial, but as yet is not precisely understood.

For perspective, it is helpful to view the situation with OPs through the lens of experience with lead. For years lead has been known to be toxic, but its special hazards to children, while suspected, were difficult to confirm. Only recently has science been able to bring into focus the subtle, yet profound learning deficits that result when infants and children are exposed to levels of lead that are perfectly safe for adults, and that were thought, until recently to be safe for children as well.

In some ways, the situation with OPs may be worse than lead, because current exposure to OP insecticides in the infant and child diet are frequently at levels that EPA deems unsafe for an adult. It is probable, given this exposure, that long

Unlike data generated under EPA protocols, the peer reviewed literature shows a consistent and repeated pattern of behavioral and functional deficits from low level OP exposure in the absence of any overt toxic effects.

Until reliable data on fetal and infant toxicity are available for all OPs, the actions recommended here, while significant, must be viewed as first steps in an ongoing process of protecting infants and children from OP insecticides.

term functional and learning deficits are occurring that scientists are just beginning to understand.

Given this overwhelming evidence of unsafe exposure to organophosphate insecticides in the diet, EPA has little choice but to act to protect infants and children. The solution to the problem of unsafe levels of OPs in food, however, is *not* for children to eat less fruits and vegetables. Infants, children and pregnant women should be able to eat a diet rich in fruits and vegetables without any concern about short term illness or long term brain and nervous system damage that may result from unsafe levels of OP pesticides on these foods. The solution is to rid these healthful foods of the most toxic pesticides.

Recommendations

To begin to meet the requirements of FQPA and retain the greatest number of safe pesticides for farmers, several decisive but reasonable steps must be made. These actions would reduce risk from OPs to a level deemed acceptable under current EPA policy. We must emphasize again, however, that current EPA safety standards do not yet incorporate explicit or adequate protections for infants and children. Until reliable data on fetal and infant toxicity are available for all OPs, the actions recommended here, while significant, must be viewed as first steps in an ongoing process of protecting infants and children from OP insecticides.

First, all home and other structural use of OP insecticides must be banned. These uses put a small but significant number of infants and toddlers at extremely high risk, and in doing so jeopardize current agricultural uses of these compounds. Indeed, if food uses of any OPs are to be continued, all non-food uses with potential to expose pregnant women, infants or toddlers must be banned.

Second, at least five high risk OP's, methyl parathion, dimethoate, chlorpyrifos, pirimiphos methyl, and azinphos methyl, must be banned immediately for all agricultural use.

Third, all OP's must be banned for use in food that ends up in commercial baby food.

Fourth, EPA must require developmental neurotoxicity studies for all the remaining OPs found in the food supply. Prompt action can ensure that this critical information is available by the time EPA must take regulatory action on OPs in August 1999. At that time, the required additional ten-fold level of protection must be applied to any OP for which a developmental neurotoxicity study is not performed.

Fifth, food tolerances for all OPs must be lowered to levels that are safe for infants and children. To quote the National Research Council report, Pesticides in the Diets of Infants and

Children, “Children should be able to eat a healthful diet containing legal residues without encroaching on safety margins” (NRC 1993, pp 8-9). That is to say, legal residues, or tolerances, must be safe for infants and children. There is simply no scientific justification for retaining legal limits for pesticides in food that allow hugely unsafe levels of exposure, just because most children do not receive this exposure. This nonsensical notion is like leaving the speed limit at 500 miles per hour just because most people would still drive at 65.

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