Intolerable Risk:
Pesticides in our Children's Food

Summary

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The Natural Resources Defense Council

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INTOLERABLE RISK:
PESTICIDES IN OUR CHILDREN'S FOOD

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Our nation's children are being harmed by the very fruits and vegetables we tell them will make them grow up healthy and strong. These staples of children's diets routinely, and lawfully, contain dangerous amounts of pesticides, which pose an increased risk of cancer, neurobehavioral damage, and other health problems. Although solutions are at hand, little is being done by the government to protect children from the intolerable risk to their health posed by pesticide residues in food.

In 1986, the Natural Resources Defense Council (NRDC) began a major study to determine whether levels of pesticide residues currently found in fruits and vegetables pose a health hazard to preschoolers. The potential effects of pesticide residues on children were examined for several reasons. First, the typical child consumes fruits and vegetables at a significantly greater rate than adults. With this increased intake comes greater relative exposure to pesticides present in food. Second, children may be more vulnerable to the effects of toxic chemicals, including pesticides. Experimental studies have found that the young are frequently more susceptible than adults to carcinogens and neurotoxins. Finally, although the Environmental Protection Agency (EPA) acknowledged in 1987 that children are invariably exposed to the highest levels of pesticides in food, neither the preschooler's dietary exposure to pesticides nor the resultant health risk has been previously quantified in a comprehensive manner. NRDC's report, therefore, represents the first detailed analysis of children's exposure to pesticides in food and a determination of the potential hazard that these residues pose to children.

Methodology

NRDC estimated the health risk to preschoolers during their first six years of life (0–5 years) by determining consumption rates for food items most frequently eaten by children. Data on the quantities of 23 pesticides known to have adverse health effects and commonly detected in these foods were obtained from federal government regulatory programs. Preschoolers' exposure to these 23 pesticides was determined by combining children's consumption rates for the food types with actual pesticide residue levels found in these foods. Pesticide exposure estimates were then assessed to determine the preschoolers' risk of developing cancer or experiencing a disruption in central nervous system function. These toxicological endpoints were selected because 20 of the 23 pesticides evaluated in

this report are either neurotoxic or carcinogenic. Furthermore, risk assessment procedures for these health effects are fairly well established.

To develop an adequate database of preschooler exposure topesticides, NRDC used consumption data from a nationwide food consumption survey conducted in 1985 by the U.S. Department of Agriculture (USDA) of children and adult women, and data on residue levels of 23 pesticides (and important metabolites) actually measured in types of fruits and vegetables. The data on pesticide residues in produce were derived from analyses of over 12,000 food samples conducted under regulatory programs of the Food and Drug Administration (FDA) and the EPA.

Principal Findings

Preschoolers are being exposed to hazardous levels of pesticides in fruits and vegetables. Between 5,500 and 6,200 (a risk range of 2.5×10^{-4} to 2.8×10^{-4}) of the current population of American preschoolers may eventually get cancer solely as a result of their exposure before six years of age to eight pesticides or metabolites commonly found in fruits and vegetables.[1] These estimates are based on scientifically conservative risk assessment procedures. They indicate that more than 50‰ of a person's lifetime cancer risk from exposure to carcinogenic pesticides used on fruit is typically incurred in the first six years of life.

The potent carcinogen, unsymmetrical dimethylhydrazine (UDMH), a breakdown product of the pesticide daminozide, is the greatest source of the cancer risk identified by NRDC. The average preschooler's UDMH exposure during the first six years of life alone is estimated to result in a cancer risk of approximately one case for every 4,200 preschoolers exposed. This is 240 times greater than the cancer risk considered acceptable by EPA following a full lifetime of exposure.[2] For children who are heavy consumers of the foods that may contain UDMH residues, NRDC predicts one additional case of cancer for approximately every 1,100 children, 910 times EPA's acceptable risk level.

The carcinogenic risk estimates for daminozide are based on results of a 1986 market basket survey that EPA required the manufacturers of daminozide to conduct. Although daminozide use may have decreased since 1986, there is no reliable information on whether—or to what degree—use has decreased. EPA has recently stated that approximately 5‰ of apples are treated with daminozide. However, this figure was based on informal conversations with growers, who may have a strong self–interest in portraying their products as daminozide–free. In contrast to EPA's figure, one Uniroyal manager privately stated that 10–11‰ of the nation's apple acreage was treated with daminozide in 1988. Further, an independent laboratory found in 1988 that 30‰ of apples tested from one large supermarket chain contained daminozide. More recently, a survey indicated that 23‰ of Vermont's apple acreage was treated with daminozide. These data were not considered when EPA developed its use estimate and raise serious questions about the accuracy of the Agency's figure. In the absence of government testing to verify grower claims about daminozide use, the manufacturer's 1986 market basket survey remains the only accurate indicator of actual residues in food.

Preschoolers also receive unacceptable exposure to the carcinogenic fungicides captan, chlorothalonil, folpet, and ethylene thiourea (ETU), the metabolite of the fungicide mancozeb. NRDC estimates that average exposure to these pesticides from consumption of fruits and vegetables from birth through age five may present a lifetime risk of one cancer case for every 33,000 to 160,000 children exposed. That means that out of the current preschool population, between 140 to 670 children may develop cancer sometime during their lifetime as a result of exposure to these fungicides. These risk estimates are approximately two to seven times what EPA considers acceptable following a full lifetime of exposure. These estimates are unchanged by EPA's recent decision to cancel certain minor food uses of captan since none of the food uses contributing to preschoolers' risk in our calculation were cancelled by EPA.

Of equal concern is NRDC's estimate that at least 17‰ of the preschool population, or three million children, receive exposure to neurotoxic organophosphate insecticides just from *raw* fruits and vegetables that are above levels the federal government considers safe. High level exposures to these insecticides can cause nausea, convulsions, coma and even death. Dietary exposures received by preschoolers may induce behavioral impairments and alter neurological function.

NRDC's analysis of exposure, based on studies of food consumption by children and women, determined that relative to their weight preschoolers receive much greater exposure than adults to the majority of the pesticides analyzed in this report. The average preschooler receives more than five times greater exposure to the fungicide mancozeb, nine times greater exposure to the neurotoxic organophosphate azinphos–methyl and 12 times greater exposure to UDMH, the carcinogenic metabolite of daminozide, than adults. The typical preschooler receives four times greater exposure, on average, than adults to the eight carcinogenic pesticides evaluated. The youngest children receive the greatest pesticide exposure. Relative to adult women, toddlers receive more than eight times the exposure to mancozeb, 15 times greater exposure to azinphos–methyl and 18 times greater exposure to UDMH, than women.

Preschoolers have greater exposure to pesticide residues than adults because they eat more food, relative to their weight, and consume much larger quantities of fruit, which has a high likelihood of being contaminated with pesticides. Fruit comprises 20‰ of the adult diet and 34‰ of the preschooler's diet. Preschoolers eat six times as much total fruit, seven times more grape products and seven times more apples and apple sauce, relative to their weight, than adults. Apple juice is a particular favorite of children. The typical preschool child consumes almost 18 times as much apple juice and the typical toddler more than 31 times as much apple juice, relative to their weight, than the average adult woman.

Fruit is highly likely to contain pesticide residues. The 1987 FDA's food monitoring program found that 50‰ of all fruit samples had detectable levels of pesticides. This contamination rate is higher than that of any other commodity and may significantly underestimate the full extent of contamination. Routine FDA monitoring methods cannot detect approximately 60‰ of the pesticides likely to leave residues on food, including many carcinogenic fungicides used widely on fruit.

Report Findings May Underestimate Preschooler Risk

The NRDC study may significantly underestimate the full extent of preschooler exposure and the subsequent health risk from pesticides in food for several reasons. First, this study assesses cancer risk that results from exposure only from birth through age five to pesticides in food. The total lifetime cancer risk will be greater since estimates do not include risk incurred from age six to 70+ years. Further, this study assesses the health risk from only 23 pesticides out of the 300 that can be legally used on food. Of the 66 pesticides EPA believes to be potentially carcinogenic and allows to be used on food, only eight were evaluated by NRDC. Routinely used FDA monitoring methods—from which much of the residue data used in the NRDC analysis were obtained—can detect only approximately 40‰ of the pesticides likely to leave residues on foods. Of all food use pesticides classified by the federal government as posing a moderate to high health hazard approximately 40‰ cannot be detected by FDA monitoring techniques.

NRDC has only assessed exposure from fruits and vegetables out of the many commodities that are consumed daily by preschoolers and that may contain pesticide residues. Milk products are perhaps the most conspicuous of the foods absent from the exposure estimates. The average preschooler has a milk intake that is almost five times higher than that of the typical woman. EPA estimates that 60‰ or more of the preschooler's exposure to the carcinogenic fungicide captan, for example, may come from residues in milk. EPA's recent cancellation of the minor food uses of captan does not appear to reduce this estimated exposure from milk. Pesticides get into animal products, including meat and eggs, as well as milk, via pesticide-contaminated feed. Drinking water may also be a significant source of pesticide exposure, especially in rural areas. EPA has reported that the normal agricultural use of pesticides has resulted in detectable pesticide concentrations in the groundwater of 26 states.

This report focuses primarily on the risk of developing cancer or the probability of disruption of normal nervous system function from dietary exposure to pesticides. However, many of the pesticides in the study cause additional adverse health effects, such as damage to the kidney or liver, effects on the immune system, or changes in reproductive capacity. Further, the full impact on preschooler health from exposure to pesticides in food is unknown since the majority of the 600 active pesticide ingredients (representing 50,000 pesticide products actually in use) have not been tested according to modern testing requirements, or the test data are unacceptable by today's standards. The National Academy of Sciences (NAS) concluded in 1984, based on an analysis of a representative sample of pesticides, that data needed to conduct a complete health hazard assessment were available for only 10‰ of the pesticide products on the market. Of the 23 pesticides evaluated for NRDC's study, 19 (83‰) were registered by USDA in the 1950s and 1960s before any comprehensive testing requirements were in place. EPA simply adopted their registrations later.

This study underestimates the risks to children for a number of other important reasons. Children are likely to be more susceptible to the effects of nervous-system toxins and cancer-causing chemicals than we have assumed in making

our estimates. However, data regarding the degree of enhanced sensitivity in preschoolers were not available for the specific pesticides evaluated in this report; therefore, susceptibility could not be factored into our health risk assessment.

The government does not require adequate testing for neurotoxic effects of pesticides. Long–term neurological testing for chronic effects of organophosphates and other neurotoxic pesticides is not required; the current tests assess only if the pesticide is capable of causing a specific delayed paralytic reaction following acute and subacute exposure.

Finally, "inert" ingredients, which act as the delivery vehicles for the active ingredients, are not regulated, even though many are known to cause cancer or other health hazards. Moreover, EPA has historically not required submission of health or safety information on "inerts". These compounds, labeled "inert" because they have no pest–killing action, have been exempted from federal requirements for setting permissible residue levels for pesticides in food.

Children's Physiological Vulnerability to Toxic Chemicals

Preschool children are receiving hazardous exposures to pesticides at the time when they are likely to be most susceptible to the toxic effects of these compounds. Experimental tests in laboratory animals have found the young to be more vulnerable than adults to the toxic effects of many chemicals, including a number of pesticides, due to their immature physiological systems. Studies have found that the young of various species retain a greater portion of a given dose of certain toxins than adults, because gastrointestinal absorption is increased and elimination is decreased. Further, the young are not capable of detoxifying many chemicals because detoxification enzymes are not fully functional. Young bodies are not capable of segregating toxins from the target organs.

Numerous studies have found that there is a greater risk of developing cancer if exposure to carcinogens begins during infancy rather than later in life. One reason that the young are more susceptible than adults to carcinogens is because cells are dividing rapidly during childhood. The cancer process is typically started when a carcinogen interacts with a cell's DNA, causing a mutation. If cells are dividing rapidly following exposure to a carcinogen capable of mutating DNA, there is a greater probability that the mutation of DNA will be fixed and the carcinogenic event initiated. In addition, the young may be at greater risk of developing cancer because they have a greater probability compared to adults of surviving the latency period prior to the manifestation of cancer.

The young have also been shown to be at greater risk from exposure to a number of neurotoxins, including neurotoxic pesticides. For instance, young rats are more susceptible than adults to the acute effects of 15 out of 16 organophosphate pesticides tested. In addition, experimental studies indicate that exposure to organophosphates and carbamate pesticides during the period of nervous system development surrounding birth may alter neurological function and may cause subtle and long–lasting neurobehavioral impairments.

Inadequate Government Programs

Current federal regulation of pesticides fails to protect the preschooler. EPA has virtually ignored infant and child food consumption patterns when regulating pesticides. Current legal limits for pesticides, or tolerances, in food are based on data collected over two decades ago on adult consumption levels. The consumption estimates that have been used by EPA in setting almost all current legal limits for pesticide residues on produce greatly underestimate preschooler intakes for most produce. Preschooler consumption of cranberries is 14 times greater than EPA's estimates; consumption of grapes is six times greater; apples and oranges, five times greater; apricots, almost four times greater; strawberries, almost three times greater; broccoli, two–and–a–half times greater; carrots, two times greater; and tomatoes, one–and–a–half times greater.

Because EPA has neglected preschooler consumption rates, the preschooler's maximum legally permissible exposure to many pesticides is hundreds of times higher than the level that EPA considers safe. The average preschooler exposure at legal limits to any one of the carcinogens captan, folpet and mancozeb, would present a risk of approximately one cancer case for every 2,000 to 3,000 children exposed simply during their first six years of life (340–460 times greater than EPA's "safe" standard of one cancer case per million following a full lifetime of exposure). Although EPA recently cancelled several minor food uses of captan, none of the commodities contributed significantly to preschooler exposure to captan. In other words, EPA has permitted the continuation of the captan food uses that present preschoolers with the greatest risk.

Legal exposures to neurotoxic pesticides also pose unacceptable risks. Preschooler exposure at the legal limit to demeton, a neurotoxic pesticide, would exceed the EPA–determined safe level by approximately 400 fold; exposure to another neurotoxin, disulfoton, by approximately 180 fold; and to another, diazinon, by approximately 160 fold.

Recommendations for Reform

Fundamental reforms in federal regulation are necessary if preschoolers are to be adequately protected from pesticides in food. Immediate action is necessary to close the loopholes in EPA's and FDA's regulatory programs. Further, Congress must act to assist growers in reducing their use of pesticides.

Congress must establish health–based standards for pesticide residues in food and require EPA to regulate pesticides so that the most exposed and most vulnerable members of society—infants and children—are adequately protected. EPA's current practice of basing risk assessment on the average adult diet does not provide this protection. Exposure at the legal maximum, or the tolerance level, should be assumed when EPA conducts risk assessments. EPA must ensure that consumption of food with residues at the legal maximum is safe for everyone, including children.

Congress must clarify EPA's authority to revoke or modify tolerances swiftly when dietary exposures to pesticides are found to present significant risk. It cur-

rently takes years to lower tolerances or remove hazardous pesticides from the market. In addition, EPA must consider risks from "inert" ingredients when regulating pesticides. Further, EPA should prohibit the use of dangerous "inerts." Congress should require that pesticide registrants develop practical analytical methods to detect pesticide residues, which can be effectively used by the government in enforcing tolerances. Finally, neurotoxicity testing should be required for all pesticides used on food and should evaluate both acute and long-term adverse effects on such processes as learning ability, memory, intelligence and behavior.

FDA must improve its methods for detecting pesticides in food. Accurate and detailed pesticide use information for both domestic and imported produce must be obtained to facilitate the choice of analytical method used in food samples. To do this, FDA's monitoring resources must be enhanced. Congress should require FDA to accelerate its analysis of food samples and give FDA the authority to detain domestic food shipments to insure that food with illegal residues can be removed from the market before it is sold or consumed. In the vast majority of cases, FDA currently fails to take action to prevent illegal food from reaching the market and being sold.

Congress must assist growers in reducing pesticide residues, by providing credit assistance, crop insurance and other financial protection for growers who are changing from conventional, high-chemical agriculture to innovative, low-input techniques. Congress should impose a tax on pesticide use to fund demonstration of farming techniques that will result in lower pesticide residues. Congress should establish national definitions of "integrated pest management" and "organic" farming techniques and develop a national certification process for commodities grown using these techniques. Congress should modify federal farm support programs to reward growers for using fewer chemicals and ensure that growers are permitted to use crop rotation and other pesticide-reducing techniques without jeopardizing their eligibility for commodity program benefits. Congress should legislatively modify agricultural supply-control systems to ensure that they do not create demand for cosmetically perfect produce which require excessive pesticide use.

Consumer Action

There are measures for limiting an individual's exposure to pesticides in food. However, specific advice is difficult to offer because data on this issue are generally scarce. The steps include: washing all produce, preferably with a diluted solution of dishwashing soap; buying domestically grown produce, preferably in season; purchasing organically grown fruits and vegetables; and being wary of perfect looking produce since it may contain higher pesticide residues. Ultimately, the best way to minimize the presence of pesticide residues in food is by reducing the widespread use of these chemicals in agriculture. Consumers can accelerate this transition in agriculture through their power in the market place. By demanding food without pesticide residues, consumers will deliver a clear message to our food producers and provide an incentive for farmers to decrease their use of pesticides.

Report Format

The NRDC study is arranged as follows: Chapter One examines food consumption differences between preschool children and adult women and quantifies the preschooler's exposure to 23 pesticides from consumption of different fruits and vegetables. Chapter Two estimates the potential health risk to preschoolers from exposure to these 23 pesticides, with emphasis on cancer risk and nervous system effects. Chapter Three examines the physiological immaturities of the young that make them more susceptible to the toxic effects of chemicals. Chapter Four describes the flaws in the government's regulation of pesticides that permit preschoolers to be exposed to significant health risks. Chapter Five recommends congressional reform measures necessary to remedy these regulatory programs and make the food supply safe from pesticides. Chapter Six offers advice on how to reduce an individual's exposure to pesticide residues.

There are three technical appendices. Appendix One contains a detailed description of the methodology used to estimate the preschooler's exposure to the 23 pesticides analyzed. Appendix Two explains the methodology used to conduct the health risk assessments for exposure to organophosphate insecticides. Appendix Three sets forth the methodology used to make the carcinogenic risk assessments.

Notes

1. These estimates are based on consumption statistics of preschoolers in the 1985 USDA survey who completed three or more days over the course of a year. These data were used to approximate average daily exposure over the year. However, cancer risk estimates were also made based on daily intake for all preschoolers in the survey and result in an estimated 5,700 to 6,400 additional cancer cases (2.6×10^{-4} to 2.9×10^{-4}) in the preschool population.

2. Using daily intake for all preschoolers in the survey (see footnote 1), UDMH still accounts for the majority of the cancer risk which is 250 times the level EPA considers acceptable following a full lifetime of exposure.

Monocrotophos (Sept., 1985)	oncogenicity - 1 species	Nov., 1989
	teratogenicity - 1 species	Dec., 1986
Omethoate	metabolite of Dimethoate, see Dimethoate	
Parathion (Dec., 1986)	neurotoxicity - 1 test	Sept., 1987
	oncogenicity - 1 species	Feb., 1990
	reproductive toxicity - 1 species	March, 1987
	mutagenicity - 3 tests	Sept., 1987
Permethrin	No Registration Standard on file	
UDMH	breakdown product of Daminozide, see Daminozide	

SOURCE: EPA, Guidance for the Reregistration of Manufacturing-Use and Certain End-Use Pesticide Products Containing Monocrotophos, 1985; EPA, Guidance for the Reregistration of Pesticide Products Containing Demeton, 1985; EPA, Daminozide Pesticide Registration Standard and Guidance Document, 1984; EPA, Daminozide Pesticide Registration Standard, 1983; EPA, Guidance for the Reregistration of Manufacturing Use and Certain End-Use Pesticide Products Containing Methamidiphos, 1982; data for all other pesticides were obtained from the EPA, Guidance for the Reregistration of Pesticide Products Containing (the pesticide) as the Active Ingredient, dates listed in the table.

[a]An additional 90-day subchronic neurotoxicity study is required if adverse effects are indicated (no date specified.)

[b]This data may be fulfilled by citing data on Disulfoton due to chemical structural similarities.

[c]An additional 90-day subchronic neurotoxicity test is required in December, 1985 if adverse effects are indicated.

[d]An additional "other mechanisms of mutagenicity" study is required contingent upon results of other tests.

[e]Unless results of a diet analysis and explanations of certain findings are found to be acceptable by this data, a second study will be required by July, 1991.

[f]Unless historical control data are submitted and found to be satisfactory (no date specified), a new study will be required by July, 1991.

[g]An additional 90-day neurotoxicity study is required (no date specified) if neurotoxicity is indicated.

[h]An additional subchronic 90-day neurotoxicity study is required in June, 1989 if neurotoxicity is indicated.

TABLE 3

YEAR OF REGISTRATION, TYPES OF PESTICIDE, VOLUME OF USE, AND MAJOR CROP USES FOR PESTICIDES INCLUDED IN NRDC STUDY

	YEAR 1ST REGSTRTN	TYPE	VOLUME OF U.S. USE:	MAJOR CROP USES LBS ACTIVE INGRED/YEAR
1. Acephate	1972	Insecticide	1,900,000	citrus
2. Azinphos-methyl (Guthion)	1956	Insecticide	2,500,000	peaches, pome fruits
3. Captan	1951	Fungicide	10,000,000	apples, peaches, almonds, seeds, other fruits & vegetables
4. Carbaryl (Sevin)	1958	Insecticide	10,005,100**	citrus, fruit, nuts, fodder
5. Chlorothalonil (Bravo)	1966	Fungicide	7,586,628**	fruits, vegetables, peanuts
6. Chlorpyrifos (Dursban/Lorsban)	1965	Insecticide	7,023,190*	citrus, corn, fruit, grain, nuts, vegetables
7. Daminozide[a]	1963	Growth Regulator	875,000	apples, peanuts
8. Demeton	1955	Insecticide	165,000*	vegetables and orchard crops
9. Diazinon (special review)	1952	Insecticide	2,125,274**	fruits, nuts, livestock, lawns & turf
10. Dicloran (DCNA)	1961	Fungicide	355,000	peaches, plums, cherries, grapes, other fruits and vegetables
11. Dimethoate	1963	Insecticide	1,453,000*	citrus, pome fruit, nuts, grapes, tomatoes & many vegetables

12.	Disulfoton	1958	Insecticide	2,111,200*	grains, strawberries & pineapples, vegetables
13.	Folpet	early 1950's	Fungicide	1,500,000	grapes, apples, melons
14.	Malathion	1956	Insecticide	15-20 million	many fruits & vegetables, tree nuts, grains, fodder
15.	Mancozeb[b]	1967	Fungicide	16,000,000	apples, onions potatoes, tomatoes, small grains
16.	Methamidophos	1972	Insecticide	1,258,947**	potatoes, cotton, cabbage & other crops
17.	Methyl Parathion	1954	Insecticide	8,934,119**	grains, peanuts berries, many fruits & vegetables
18.	Mevinphos	early 1950's	Insecticide	1,277,700*	many vegetables & fruits
19.	Monocrotophos	1965	Insecticide	760,000*	peanuts, sugarcane, tobacco, potatoes, tomatoes
20.	Omethoate (Folimat)	Not Registered in U.S.	Insecticide		fruit crops, vegetables, hops
21.	Parathion	1948	Insecticide	7,000,000	citrus, cotton, orchard crops, vegetables, fruits
22.	Permethrin (Ambush Pounce)	1978	Insecticide	1,475,000*	vegetables
23.	Quintozene, (PCNB[c])	1964	Fungicide	2,522,802**	vegetables, small grains

[a]Important Metabolite - Unsymmetrical Dimethylhydrazine (UDMH)
[b]Important Metabolite - Ethylenethiourea (ETU)
[c]Important Metabolite - Hexachlorobenzene (HCB)

SOURCE: *Gianessi, L., A National Pesticide Usage Data Base, Resources for the Future, Washington, D.C., 1986; **Gianessi, L., Use of Selected Pesticides in Agricultural Crop Production, Resources for the Future, Washington, D.C., 1988. Source for all others are from: NAS, Regulating Pesticides In Food: The Delaney Paradox, 1987, pp. 52-53.

TABLE 1

PESTICIDES AND METABOLITES EVALUATED IN STUDY AND THEIR POTENTIAL HEALTH EFFECTS

CHEMICAL	POTENTIAL HEALTH EFFECTS
Acephate	"Possible human carcinogen"; mutagen; mild eye irritant; causes nervous system effects (inhibits ChE); reproductive in animals.
Azinphos-methyl	Acutely toxic (nervous system toxin--inhibits ChE); severe eye and skin irritant; mutagen; causes cancer (liver tumors) in animals.
Daminozide	"Probable human carcinogen"; also contains and breaks down to UDMH (see below); causes multiple tumors at multiple organ sites (lung, liver, kidney, reproductive and vascular systems) in animals.
Disulfoton	Acute toxicity (inhibits ChE).
Captan	"Probable human carcinogen"; mutagen; causes reproductive effects in animals; possible teratogen.
Carbaryl	Mutagen; causes kidney effects in animals.
Chlorothalonil	"Probable human carcinogen"; mutagen; causes reproductive and kidney effects in animals.
Chlorpyrifos	Causes nervous system effects (inhibits ChE); mutagen; eye and skin irritant.
Demeton	Acute toxicity (nervous system toxin--inhibits ChE); mutagen.
Diazinon	Eye and skin irritant; causes nervous system effects (inhibits ChE).
Dicloran	Liver effects in animals.
Dimethoate	Nervous system effects (inhibits ChE); mutagen; causes reproductive effects in animals; some evidence of carcinogenicity in animals.
ETU	"Probable human carcinogen"; effects thyroid gland and causes other hormonal effects in animals; causes birth defects in animals.

Folpet	"Probable human carcinogen"; causes decreased weight gain and blood constituent level changes in animals.
Hexachlorobenzene	"Probable human carcinogen" (causes liver tumors in animals); possible developmental effects.
Mancozeb	"Probable human carcinogen"; also contains and breaks down to ETU (see below); mutagen; causes birth defects in experimental animals; effects on the kidney, thyroid and prostrate glands.
Malathion	Nervous system effects (inhibits ChE); reproductive effects in animals.
Methamidophos	Extremely toxic (nervous system toxin--inhibits ChE); causes reproductive effects in animals.
Methyl parathion	Acutely toxic (nervous system toxin--inhibits ChE); degenerative effects on nerve tissue; mutagen; causes birth defects and reproductive effects in animals; some evidence of carcinogenicity in animals; affects eyes (cataracts) in animal studies.
Mevinphos	Nervous system effects (potent ChE inhibitor); possible mutagen.
Monocrotophos	Extremely toxic (nervous system toxin--inhibits ChE); mutagen; possible reproductive effects; contaminated with mutagen and oncogen TMP.
Omethoate	Degradation product of dimethoate; more acutely toxic than dimethoate (nervous system toxin--inhibits ChE).
Parathion	"Possible human carcinogen"; mutagen; extremely toxic; causes nervous system effects (inhibits enzyme ChE, degenerative effect on nerve fibers); eye effects in animals.
Permethrin	Eye irritant; liver effects in animals; causes lung and liver tumors in animals.
UDMH	"Probable human carcinogen"; causes multiple tumors at multiple sites (lung, liver, pancreas, nasal tissue, vascular system) in animals; mutagen.

SOURCES: Based on EPA Registration Standards; EPA Tox One-Liners; EPA Pesticide Fact sheets; EPA Special Review Position Documents; California Department of Food and Agriculture (CDFA) SB950 Tox Summaries.

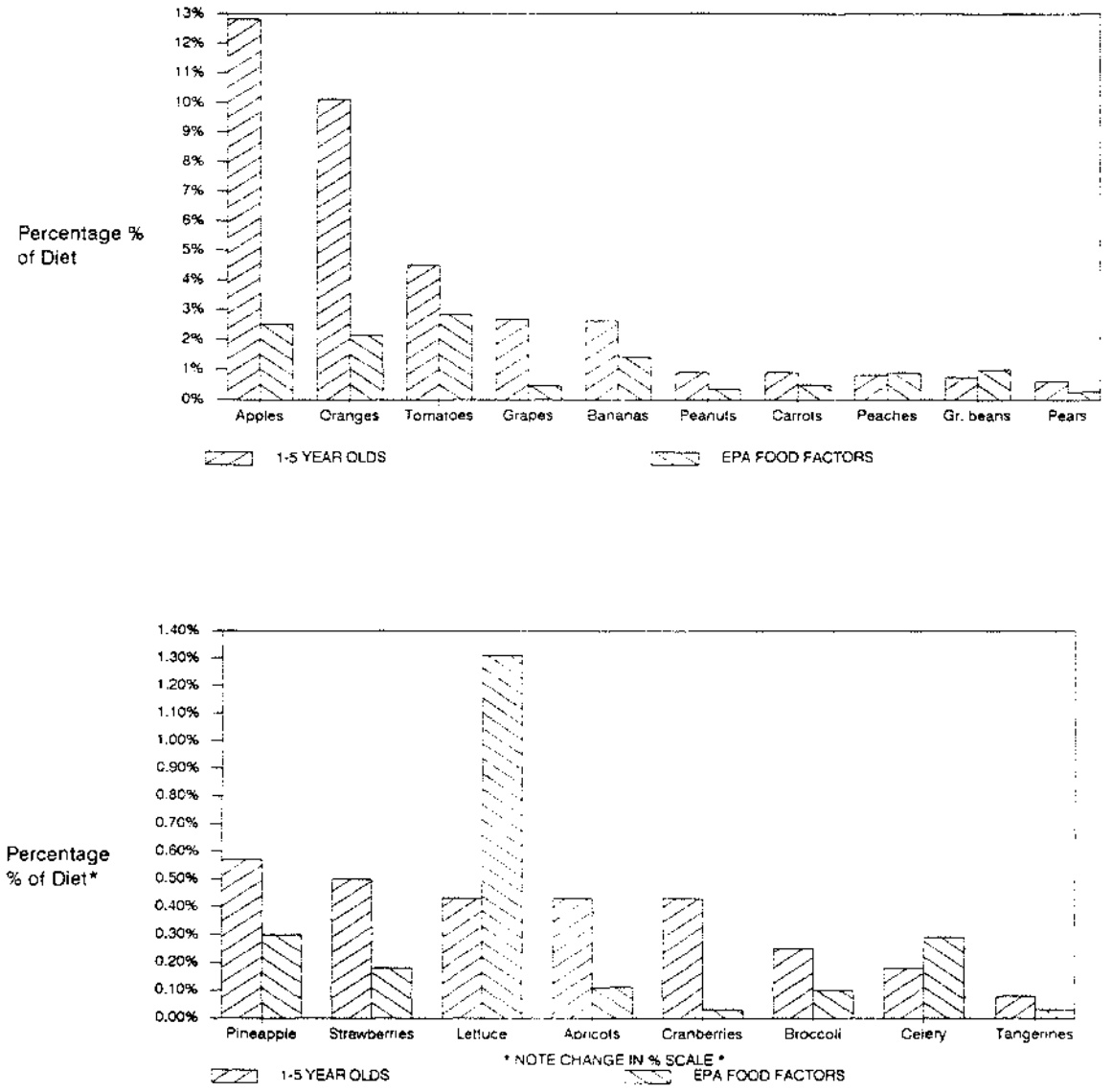
TABLE 2

HEALTH EFFECTS THAT HAVE NOT BEEN ADEQUATELY ASSESSED FOR THE PESTICIDES EVALUATED IN NRDC'S STUDY

Chemical (Date of RS)	Health Effects for Which Data Are Missing or Inadequate	Due Date
Acephate (Sept., 1987)	chronic toxicity - 1 species	March, 1988
	reproductive toxicity - 1 species	Dec., 1990
Azinphos-methyl (Sept., 1986)	neurotoxicity - 1 test	Sept., 1987
	chronic toxicity - 1 species	Aug., 1991
	oncogenicity - 1 species	Aug., 1991
	teratogenicity - 2 species	Sept., 1988
	reproductive toxicity - 1 species	Sept., 1990
	mutagenicity - 1 test	Sept., 1987
	" - 1 test	April, 1987
Captan (March, 1986)	chronic toxicity - 1 species	March, 1989
	neurotoxicity - requirements not specified	
Carbaryl (May, 1984)	chronic toxicity - 1 species	April, 1986
	teratogenicity - 1 species	April, 1985
Chlorothalonil (Sept., 1984)	oncogenicity - 1 species	July, 1985
	teratogenicity - 1 species	no date specified
Chlorpyrifos (Sept., 1984)	chronic toxicity - 2 species	Oct., 1988
	oncogenicity - 2 species	Oct., 1988
	mutagenicity - 3 tests	March, 1985
Daminozide (June, 1984)	chronic toxicity - 1 species	Oct., 1988
	oncogenicity (UDMH) - 2 species:	
	"low dose"	July, 1989
	"high dose"	Jan., 1990
	teratogenicity - 1 species	June, 1985
	reproductive toxicity - 1 species	June, 1986
	mutagenicity - 1 test	Dec., 1984
Demeton (Feb., 1985)	neurotoxicity - 1 test	no date specified[a]
	chronic toxicity[b]	
	oncogenicity[b]	
	teratogenicity - 2 species	no date specified
	reproductive toxicity[b]	
	mutagenicity - 2 tests	no date specified
Diazinon	No Registration Standard on file	
Dicloran	No Registration Standard on file	

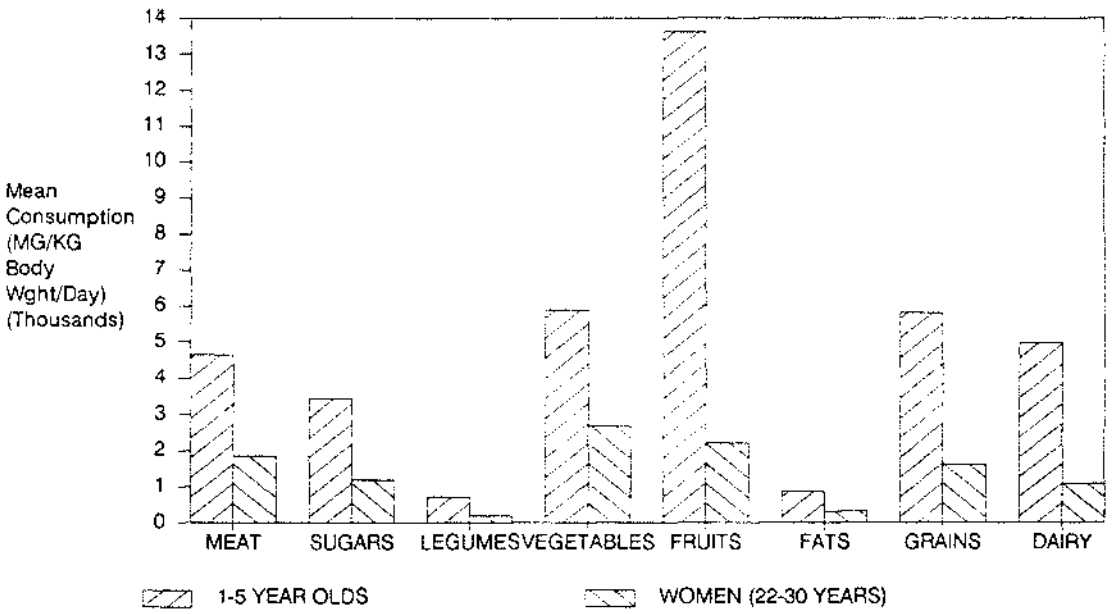
Dimethoate (March, 1983)	chronic toxicity - 1 species	Nov., 1985
	oncogenicity - 2 species	Nov., 1984
	teratogenicity - 2 species	May, 1983
Disulfoton (Dec., 1984)	neurotoxicity - 1 test	June, 1985[c]
	chronic toxicity - 1 species	Jan., 1985
	oncogenicity - 1 species	Jan., 1985
	teratogenicity - 1 species	Dec., 1987
	reproductive toxicity - 1 species	Dec., 1987
	mutagenicity - 2 tests	Dec., 1987[d]
ETU	breakdown product of EBDCs, see Mancozeb	
Folpet (June, 1987)	chronic toxicity - 1 species	Sept., 1987[e]
	reproductive toxicity - 2 species	Sept., 1987[f]
	mutagenicity - 1 test	June, 1988
Malathion (Feb., 1988)	neurotoxicity - 1 test	Nov., 1988[g]
	chronic toxicity - 2 species	April, 1992
	oncogenicity - 2 species	April, 1992
	teratogenicity - 1 species	May, 1989
	reproductive toxicity - 1 species	May, 1991
	mutagenicity - 1 test	Nov., 1988
	" - 2 tests	Feb., 1989
Mancozeb (April, 1987)	chronic toxicity - 2 species	June, 1991
	oncogenicity - 2 species	June, 1991
	teratogenicity - 1 species	July, 1988
	reproductive toxicity - 1 species	July, 1990
	mutagenicity - 1 test	April, 1988
Mevinphos (March, 1988)	neurotoxicity - 1 test	June, 1988[h]
	chronic toxicity - 2 species	May, 1992
	oncogenicity - 2 species	May, 1992
	teratogenicity - 1 species	June, 1989
	reproductive toxicity - 1 species	June, 1991
	mutagenicity - 1 test	Dec., 1988
	" - 2 tests	March, 1989
Methamidiphos (Sept., 1982)	chronic toxicity - 1 species	Aug., 1984
	oncogenicity - 1 species	Jan., 1983
	teratogenicity - 1 species	Oct., 1982
	reproductive toxicity - 1 species	June, 1984
	mutagenicity - 2 tests	Feb., 1983
Methyl Parathion (Dec., 1986)	neurotoxicity - "acute"	Sept., 1987
	" - "subchronic"	March, 1988
	chronic toxicity - 2 species	Dec., 1988
	oncogenicity - 2 species:	
	rat	Sept., 1987
	mouse (upgrade one)	Dec., 1987
	teratogenicity - 2 species	Dec., 1988
	" - additional study	Feb., 1991

Figure 3. Current Preschooler Intakes of Selected Produce Compared to the EPA Food Factor Estimates of Intake



NOTE: Current preschooler consumption estimates were derived from the *CSFII -- Nationwide Food Consumption Survey: Continuing Survey of Food Intakes by Individuals, Women 19-50 Years and Their Children 1-5 Years, 6 Waves*, 1985, and are average intakes of all forms (*i.e.* raw and processed) of each produce type for all children included in the 1985 Nationwide Food Consumption Survey. Food Factors were obtained from EPA, Toxicology Branch, *Revised Average Food Factors*, May 1, 1978.

Figure 2. Differences in Mean Food Intakes Between Children (Age 1-5) and Adult Women (Age 22-30) for Major Food Groups.



Children's food intakes exceed that of women for all food types, most notably fruit.

NOTE: Consumption estimates are derived from the USDA, Human Nutrition Information Service, *CSFII -- Nationwide Food Consumption Survey: Continuing Survey of Food Intakes by Individuals, Women 19-50 Years and Their Children 1-5 Years, 6 Waves,* 1985. Food group consumption estimates are based on mean intake for each age group (considering all respondents) of specific commodities.

Figure 1. Methodology of NRDC Study to Estimate Preschoolers' Health Risks From Pesticides in Foods

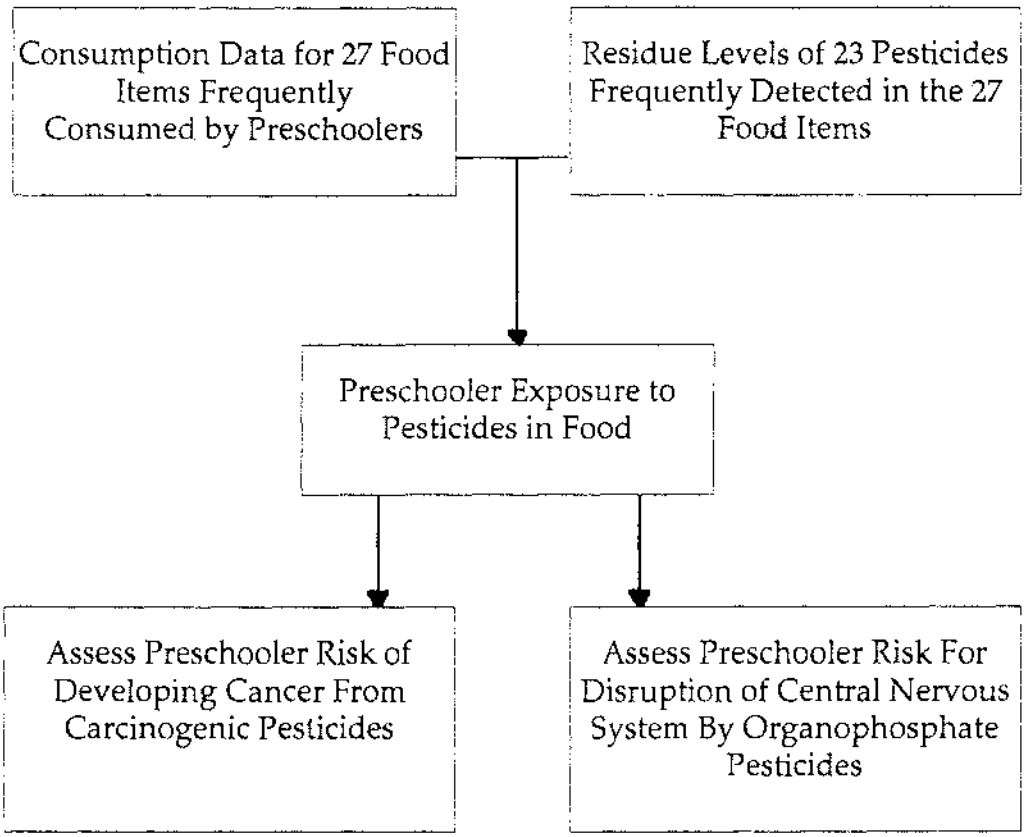


TABLE 4

TWENTY-SEVEN FRUITS AND VEGETABLES INCLUDED IN THE NRDC STUDY

Apple products
Apricot products
Blueberries
Broccoli
Cabbage
Cantaloupe
Carrots
Celery
Cherry products
Cranberry products
Cucumbers
Grape products
Grapefruit products
Green beans (including string)
Lettuce
Nectarines
Orange products
Peaches
Peanut products
Pear products
Peas, green
Pineapple products
Plum products
Potatoes
Spinach
Strawberries
Tomato products

TABLE 5

FREQUENCIES OF DETECTABLE PESTICIDE RESIDUES IN FRUITS AND VEGETABLES FROM FDA AND CALIFORNIA FOOD MONITORING PROGRAMS 1982-85

COMMODITY	PERCENT OF DOMESTIC AND IMPORTED SAMPLES WITH PESTICIDE RESIDUES
Strawberries	63%
Peaches	55%
Celery	53%
Cherries	52%
Cucumbers	51%
Bell Peppers	49%
Tomatoes	45%
Sweet Potatoes	37%
Cantaloupes	34%
Grapes	34%
Lettuce	32%
Apples	29%
Spinach	29%
Carrots	28%
Green Beans	27%
Pears	22%
Grapefruit	22%
Potatoes	22%
Oranges	22%
Cabbage	20%
Broccoli	13%
Onions	10%
Cauliflower	5%
Watermelon	4%
Bananas	1%
Corn	1%

NOTE: These data may not accurately reflect true contamination rates because many pesticides are undetectable by FDA methods, including widely used fungicides on fruits. Also, the FDA program lacks a sampling design to ensure representative sampling of all fruits and therefore certain fruits are sampled more rigorously than others.

SOURCE: Pesticide Alert: A Guide to Pesticides in Fruits and Vegetables, L. Mott and K. Snyder, Sierra Club Books, 1988. Reproduced with permission.

TABLE 6

ESTIMATED EXPOSURE OF TODDLERS (AGES 1-2) AND ALL PRESCHOOLERS (AGES 1-5) TO SELECTED PESTICIDES AS A MULTIPLE OF ADULT WOMEN (22-30) EXPOSURE

Chemical	All Children (1-5 Years)	Toddlers (1-2 Years)
UDMH	12.2	18.3
Azinphos-methyl	9.4	15.4
Daminozide	9.1	13.7
Parathion	5.6	6.4
Mancozeb	5.4	8.3
Methyl parathion	5.2	5.5
Captan	4.4	4.9
Carbaryl	4.4	5.3
ETU	4.2	5.2
Dicloran	4.2	6.8
Chlorpyrifos	3.7	4.6
Diazinon	3.3	6.3
Omethoate	3.1	3.9
Dimethoate	3.0	3.5
Folpet	2.9	3.4
HCB	2.0	3.0
Methamidophos	2.6	2.7
Acephate	1.8	2.5
Chlorothalonil	1.6	1.6
Demeton	1.0	1.0
Permethrin	0.7	0.8

NOTE: These estimates are based on lower-bound estimates of average exposure for each age group. Residue data was obtained from EPA and FDA. (See Appendix One.) Consumption estimates are derived from the CSFII -- Nationwide Food Consumption Survey: Continuing Survey of Food Intakes by Individuals, Women 19-50 Years and Their Children 1-5 Years, 6 Waves, 1985.

[a]The multiple is derived by dividing the toddler's and child's mean daily exposure in mg/kg by that of the adult woman.

TABLE 7

THE SEVENTEEN PESTICIDES AND TWO METABOLITES OF THOSE EVALUATED FOR THE NRDC REPORT THAT PRESENT THE GREATEST SOURCE OF EXPOSURE TO PRESCHOOLERS FROM CONSUMPTION OF 27 TYPES OF FRUITS AND VEGETABLES

CHEMICAL	PRESCHOOLER'S AVERAGE EXPOSURE (UG/KG.OF BODY WEIGHT)
Mancozeb	3.23-8.74
Daminozide	2.95[a]
Carbaryl	2.80-8.91
Captan	2.02-9.6
Dicloran	.44-4.2
Folpet	.12-6.19
ETU	.11- .31
Chlorothalonil	.1 -1.03
Parathion	.09-1.04
Methamidophos	.09- .44
UDMH	.082[a]
Chlorpyrifos	.07- .78
Azinphos-methyl	.066-1.3
Dimethoate	.07- .14
Omethoate	.06-1.14
Methyl Parathion	.05-1.24
Permethrin	.04- .37
Acephate	.02- .62
Diazinon	.015-.65

NOTE: The estimates are derived by combining average consumption estimates for each commodity by children age 1-5, with lower-bound and upper-bound estimates of average residue for each pesticide on each commodity. Pesticide

residue data were obtained from regulatory programs of EPA and FDA. Consumption estimates are derived from the U.S. Department of Agriculture, Human Nutrition Information Service, CSFII--Nationwide Food Consumption Survey, Continuing Food Intakes by Individual Women 19-50 Years and Their Children 1-5 Years, 6 Waves, 1985. Estimates are based on average exposure for all children participating in the dietary survey.

[a]No range is necessary for daminozide and UDMH because comprehensive residue data were available.

TABLE 8

LIFETIME CARCINOGENIC RISK FROM AVERAGE PRESCHOOLER EXPOSURE DURING AGES 0-5 TO EIGHT PESTICIDES IN 27 FRUITS AND VEGETABLES

CHEMICAL	ESTIMATED LIFETIME CARCINOGENIC RISK FROM AVERAGE CHILDHOOD EXPOSURE AGES 0-5	NUMBER OF ESTIMATED CANCER CASES PER PRESCHOOLER EXPOSED AGES 0-5
UDMH	2.4×10^{-4}	one out of every 4,200
ETU	4.1×10^{-6} - 1.2×10^{-5}	one out of every 83,000 to 244,000
Captan	1.8×10^{-6} - 8.4×10^{-6}	one out of every 119,000 to 556,000
Chlorothalonil	3.1×10^{-7} - 3.1×10^{-6}	one out of every 323,000 to 3,226,000
Folpet	1.3×10^{-7} - 6.4×10^{-6}	one out of every 156,000 to 7,692,000
Acephate	5.2×10^{-8} - 1.5×10^{-6}	one out of every 667,000 to 19,231,000
Parathion	6.3×10^{-8} - 7.6×10^{-7}	one out of every 1,316,000 to 15,873,000
HCB	1.7×10^{-7} - 9.0×10^{-6}	one out of every 111,000 to 5,882,000
Total	2.5×10^{-4} - 2.8×10^{-4}	one out of every 3,600 to 4,000

NOTE: Consumption estimates are derived from the USDA, CSFII -- Nationwide Food Consumption Survey: Continuing Survey of Food Intakes by Individuals, Women 19-50 Years and Their Children 1-5 Years, 6 Waves, 1985. Lower- and upper-bound average pesticide residues were derived from residue data obtained under the regulatory programs of the EPA and FDA. (See Appendix One.) Exposure estimates are based on the average exposure of the preschoolers who completed three or more days of dietary survey.

TABLE 9

LIFETIME CARCINOGENIC RISK FROM 95 PERCENTILE PRESCHOOLER EXPOSURE DURING AGES 0-5 TO EIGHT PESTICIDES IN 27 FRUITS AND VEGETABLES

CHEMICAL	ESTIMATED LIFETIME CANCER RISK FROM 95th PERCENTILE EXPOSURE AGES 0-5	NUMBER OF ESTIMATED LIFETIME CANCER CASES PER PRESCHOOLER EXPOSED AT THE 95 PERCENTILE EXPOSURE LEVEL
UDMH	9.1×10^{-4}	one out of every 1,100
ETU	1.1×10^{-5} - 3.4×10^{-5}	one out of every 29,000 to 91,000
Captan	5.4×10^{-6} - 2.9×10^{-5}	one out of every 34,000 to 185,000
Chlorothalonil	1.0×10^{-6} - 7.7×10^{-6}	one out of every 130,000 to 1,000,000
Folpet	3.9×10^{-7} - 2.0×10^{-5}	one out of every 50,000 to 2,560,000
Acephate	1.3×10^{-7} - 3.9×10^{-6}	one out of every 2,300,000 to 7,700,000
Parathion	1.8×10^{-7} - 1.8×10^{-6}	one out of every 556,000 to 5,556,000
HCB	5.0×10^{-7} - 2.6×10^{-5}	one out of every 38,000 to 2,000,000

NOTE: These risks are the result of changes in food consumption patterns only. Average pesticide residues were assumed in all calculations. Consumption estimates are derived from the USDA, CSFII -- Nationwide Food Consumption Survey: Continuing Survey of Food Intakes by Individuals, Women 19-50 Years and Their Children 1-5 Years, 6 Waves, 1985. Lower- and upper-bound average residues used in the calculations were derived from residue data obtained under the regulatory programs of the EPA and FDA. (See Appendix One.) Risk estimates are based on the 95 percentile chronic exposure for the preschoolers who completed three or more days of the 1985 dietary survey.

TABLE 10

THE POTENTIAL FOR NERVOUS SYSTEM EFFECTS: THE DEGREE TO WHICH PRESCHOOLER EXPOSURES EXCEED SAFE LEVELS FOR ORGANOPHOSPHATE INSECTICIDES FROM CONSUMPTION OF JUST RAW FORMS OF COMMODITIES

Chemical(s)	% of 1-5 Year-Olds Receiving Average Daily Exposures Above Acceptable Daily Intake (ADI)
All Organophosphates	16.8 - 58%
Parathion	0 - 12.6%
Methyl Parathion	0 - 24%
Methamidophos	12.3 - 71.5%
Diazinon	0 - 35.1%
Azinphos-methyl	0 - 2.1%
Omethoate	0 - 39.9%
Monocrotophos	0 - 58%
Acephate	0 - 29.4%

NOTE: Consumption estimates are derived from the CSFII -- Nationwide Food Consumption Survey: Continuing Survey of Food Intakes by Individuals, Women 19-50 Years and Their Children 1-5 Years, 6 Waves, 1985. Lower- and upper-bound average residues for each pesticide were derived from residue data obtained under the regulatory programs of the EPA and FDA. (See Appendix One.) Exposure estimates are based on the average exposure of the preschoolers who completed three or more days of dietary survey. The ADIs are for the cholinesterase-inhibiting effects of each pesticide and are presented in Appendix Two. The ADIs are from the EPA, Office of Pesticide Programs, PRODVOL, Computer File Containing Toxicological and Regulatory Status Summaries, 404 Pesticides, 1987.

TABLE 11

MEAN PRESCHOOLER (Age 1-5) EXPOSURE TO EACH ORGANOPHOSPHATE AND CUMULATIVE EXPOSURE TO ALL ORGANOPHOSPHATES FROM CONSUMPTION OF RAW COMMODITIES AND FROM ALL COMMODITIES, EXPRESSED AS A PERCENTAGE OF THE ADI.

	Mean Chronic Exposure to Pesticide(s), All Forms (raw or processed) of produce[a] (in ug/kg body weight)	As % of EPA's Acceptable Daily Intake (ADI)	Mean Chronic Exposure to Pesticide(s) Only Raw Forms of Commodity (in ug/kg body weight)	As % of EPA'S Acceptable Daily Intake (ADI)
Acephate	0.02 - 0.62	7 - 206%	0.011 - 0.26	4 - 87%
Azinphos-methyl	0.06 - 1.25	5 - 96%	0.014 - 0.27	1 - 21%
Diazinon	0.02 - 0.64	22 - 711%	0.002 - 0.09	2 - 100%
Methamidophos	0.09 - 0.44	180 - 880%	0.022 - 0.16	44 - 320%
Methyl Parathion	0.05 - 1.23	20 - 492%	0.005 - 0.16	2 - 64%
Monocrotophos	0.003 - 4.60	6 - 9200%	0.002 - 0.27	4 - 540%
Omethoate	0.06 - 1.11	20 - 370%	0.024 - 0.31	8 - 103%
Parathion	0.08 - 1.03	24 - 312%	0.007 - 0.14	2 - 42%
	TOTAL:	**284 - 12,267%**	TOTAL:	**67 - 1277%**

NOTE: Consumption estimates are derived from the USDA, CSFII -- Nationwide Food Consumption Survey: Continuing Survey of Food Intakes by Individuals, Women 19-50 Years and Their Children 1-5 Years, 6 Waves, 1985. Lower- and upper- bound average residues for each pesticide are derived from residue data obtained under the regulatory programs of the EPA and FDA. (See

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[a]Assumes same level of residue on processed forms of the commodity (including cooked) as on raw forms. The effects of processing on residue levels is generally poorly characterized. In most cases it tends to decrease residue levels. However, processing may leave a more toxic metabolite. Inadequate data exists regarding residue levels in processed foods themselves.

Appendix One.) Exposure estimates are based on the average exposure of the preschoolers who completed three or more days of dietary survey. The ADIs and NOELs are given in Appendix Two and are for the cholinestrase-inhibiting effects of each organophosphate. ADIs are from EPA, Office of Pesticide Programs, PRODVOL, Computer File Containing Toxicological and Regulatory Status Summaries, 404 Pesticides, 1987.

TABLE 12

THE POTENTIAL FOR NERVOUS SYSTEM EFFECTS: THE DEGREE TO WHICH PRESCHOOLER EXPOSURES EXCEED SAFE LEVELS FOR ORGANOPHOSPHATE INSECTICIDES FROM CONSUMPTION OF ALL COMMODITIES, RAW AND PROCESSED, ASSUMING THE SAME RESIDUE LEVEL ON ALL FORMS OF THE SAME COMMODITY

Chemical(s)	% of 1-5 Year-Olds Receiving Average Daily Exposures Above Acceptable Daily Intake (ADI)	% of 1-5 Year-Olds Exposed on Any Given Day Above The No-Observed Effect-Level (or NOEL)
All Organophosphates	83.8% - 98.5%	0 - 0.5%
Parathion	1.8-87.7%	0.0-2.2% > Lowest Effect Level (LEL)
Methyl Parathion	0.3-83.2%	0.0-0.21%
Methamidophos	63.0-97.6%	
Diazinon	0.3-94.6%	0.0-0.76%
Azinphos-methyl	0.0-34.2%	
Omethoate	0.0-91%	
Monocrotophos	0.0-87.1%	0.0-2.5%
Acephate	0.0-77.2%	

NOTE: Consumption estimates are derived from the USDA, CSFII -- Nationwide Food Consumption Survey: Continuing Survey of Food Intakes by Individuals, Women 19-50 Years and Their Children 1-5 Years, 6 Waves, 1985. Lower- and upper- bound average residues for each pesticide are derived from residue data obtained under the regulatory programs of the EPA and FDA. (See Appendix One.) Exposure estimates are based on the average exposure of the preschoolers who completed three or more days of dietary survey. The ADIs and NOELs are given in Appendix Two and are for the cholinestrase-inhibiting effects of each organophosphate. ADIs are from EPA, Office of Pesticide Programs, PRODVOL, Computer File Containing Toxicological and Regulatory Status Summaries, 404 Pesticides, 1987.

TABLE 13

THE EXTENT TO WHICH PRESCHOOLER'S MAXIMUM ALLOWABLE EXPOSURES (ALL CONSUMED FOODS WITH PESTICIDE RESIDUE AT TOLERANCE) EXCEED ACCEPTABLE LEVELS

PESTICIDE	AVERAGE INTAKE AS PERCENTAGE OF ACCEPTABLE DAILY INTAKE (ADI)
Acephate	2,210%
Azinphos-methyl	2,843%
Chlorpyrifos	753%
Demeton	41,395%
Diazinon	16,442%
Disulfoton	18,871%
Methamidophos	5,763%
Parathion	6,942%

NOTE: EPA has traditionally been concerned when allowable exposure exceeds 100%.

PESTICIDE	AVERAGE ALLOWABLE LIFETIME CARCINOGENIC RISK FROM PRESCHOOL EXPOSURES, AGES 0-5
Captan	4.6×10^{-4}
Chlorothalonil	2.6×10^{-5}
Folpet	3.4×10^{-4}
Mancozeb	3.5×10^{-4}[a]

NOTE: Consumption estimates are derived from the USDA, CSFII -- Nationwide Food Consumption Survey: Continuing Survey of Food Intakes by Individuals, Women 19-50 Years and Their Children 1-5 Years, 6 Waves, 1985. Exposure estimates are based on the average of daily intakes for all responders in the dietary survey. Residue values for each pesticide on each commodity are the tolerance limits. ADI values are presented in Appendix Two. Methodology for estimating carcinogenic risk is presented in Chapter Three.

[a]Assuming ETU residues, the proven carcinogenic component of mancozeb, are only 12% of mancozeb residue, the in vivo

conversion rate provided in EPA, Office of Pesticide Programs, Dietary and Worker Exposure and Risk Analyses for Mancozeb and Ethylene thiourea (ETU), April 1, 1987. EPA's estimate of in vivo conversion was recently increased to 20%, EPA, Ethylene Bisdithiocarbamate (EBDC) Pesticides, Proposed Regulatory Options for the EBDC, 1989.