



FROM CROP TO TABLE
PESTICIDE REPORT



ConsumerReports[®]
FOOD SAFETY & SUSTAINABILITY CENTER

Pesticide Report

About Consumer Reports' Food Work and Its Food Safety and Sustainability Center

Consumer Reports has been concerned about the quality and safety of the food supply since its earliest years. It did pioneering research on the presence of nuclear fallout in the American diet (Strontium-90) in the 1950s and 1960s, which helped build support for the Test Ban Treaty of 1963. The magazine's 1974 landmark series on water pollution played a role in the Safe Drinking Water Act. The organization has been testing meat and poultry for pathogens and antibiotic resistance for more than 15 years and has used its research to successfully fight for reforms such as the 2010 campylobacter standard for chicken and turkey, the 2011 Food Safety Modernization Act, and improvements to the salmonella standards.

In 2012, Consumer Reports launched its Food Safety and Sustainability Center to fight for sweeping, systemic change and address the root causes of problems plaguing the food system. The Center's work focuses on issues including food-borne illness and antibiotic resistance; pesticide use; heavy metals (mercury, lead, arsenic); truth and transparency in labeling; and promoting more sustainable agricultural practices that advance the marketplace, such as animal welfare, organic farming, and fair trade. At the core of the Center's work is the principle that there is a clear intersection between how food is produced and the impact on public health.

About the Food Center

CR Scientists

Dr. Urvashi Rangan leads Consumer Reports' Consumer Safety and Sustainability Group and serves as the Executive Director of its Food Safety and Sustainability Center. Dr. Rangan directs all of the organization's food-safety testing and research in addition to the scientific risk assessments related to food and product safety, which she translates into actionable recommendations for lawmakers and consumers. She is an environmental health scientist and toxicologist and is a leading expert, watchdog, and spokesperson on food labeling and food safety. Dr. Rangan received her Ph.D. from the Johns Hopkins School of Public Health.

Charlotte Vallaeys is a senior policy analyst and writer for the Consumer Reports' Food Safety and Sustainability Center. She focuses on sustainability and works in the food system and works on a variety of food policy and food safety issues, including food labeling and organic policy. She regularly attends National Organic Standards Board meetings as a watchdog for the organic label and has done work for the National Organic Coalition. She previously worked as Policy Director at The Cornucopia Institute. She received her master's degree in theological studies from Harvard University, where she studied social and environmental ethics, and a master's of science in nutrition from the Friedman School of Nutrition Science and Policy at Tufts University.

Dr. Doris Sullivan is the Associate Director for Product Safety in Consumer Reports' Consumer Safety and Sustainability Group. She oversees product safety testing, research, and prioritization. She is also an expert in compiling and analyzing large datasets. She received her Ph.D. in

chemistry from Boston University and completed postdoctoral research at the Free University of Brussels and University of Pennsylvania.

Dr. Michael K. Hansen is a Senior Scientist with Consumers Union, the policy and advocacy arm of Consumer Reports. He works primarily on food safety issues, including pesticides, and has been largely responsible for developing the organization's positions on the safety, testing and labeling of genetically engineered food and mad cow disease. Dr. Hansen served on the Department of Agriculture's Advisory Committee on Agricultural Biotechnology from 1998 to 2002 and on the California Department of Food and Agriculture Food Biotechnology Advisory Committee from 2001 to 2002.

Dr. Keith Newsom-Stewart is a Statistical Program Leader at Consumer Reports. During his tenure, he has worked on a wide range of projects, including those related to meat, seafood, and poultry safety and food additives. He specializes in linear and nonlinear mixed models, experimental design, and analysis of complex surveys. Prior to coming to CR, he worked for the Cornell Biometrics Unit and College of Veterinary Medicine. His educational background is in statistics, general biology, and genetics. He is an adjunct math professor at Western Connecticut State University and a member of the American Statistical Association.

CR Communications

Jennifer Shecter is the Director of External Relations at Consumer Reports and the Senior Adviser to the Food Safety and Sustainability Center. In this capacity, she manages the center's partnerships and relationships, coordinates its

overall public service activities, and pursues strategic initiatives to build support for its mission. She has been with Consumer Reports for more than a decade, serving first in its Communications Department, promoting food and product safety issues, then working as the Senior Adviser to the President—writing speeches, op-eds, and briefing materials—and advising on key organizational issues.

CR Advisers

Dr. Charles Benbrook consulted to Consumer Reports on this pesticide report, including providing expert analysis of his Dietary Risk Index (DRI). Over a long career, Dr. Benbrook has developed a variety of analytical systems quantifying food quality and safety, and the impacts of agricultural technology and policy. Before moving west, Dr. Benbrook worked in Washington, D.C., for 18 years, including in the Executive Office of the President and on Capitol Hill. He collaborated with Consumer Reports on the organization's 1998 Worst First pesticides report. He currently serves as head of Benbrook Consulting Services.

Chantelle Norton is an artist and designer and is a lead designer of Consumer Reports' Food Safety and Sustainability Center reports. She has worked in many fields of design, from fashion to print to costume to graphic design. She lives in the Lower Hudson Valley with a medley of animals, including her pet chickens. Her latest paintings take the chicken as muse and feature portraits of her feathered friends in landscapes inspired by the Hudson Valley and Ireland.

We acknowledge the contribution from current adviser, Aimee Simpson, JD.

Contents

Introduction: Produce Matters	5
The Intersection of Safety and Sustainability: Why Pesticides Are a Bad Deal	6
Pesticides Used Before and After Cultivation	7
Human Health Concerns	9
Weighing Human Health in Pesticide Approvals	9
Pesticide Terminology	11
Risks to Young Children	12
Organophosphate Pesticides	13
Food Quality Protection Act	14
Risks to Farmers and Farmworkers	16
Environmental and Ecological Concerns	22
Honeybees and Pollination	22
The Impacts of Pesticide Use	24
Why Organic is the Right Way	25
Genetically Engineered Crops and Increased Pesticide Use	26
Reducing Exposure to Pesticides: What Consumers Can Do	27
A Consumer Reports Guide to Residue Risk	28
Calculation of the Dietary Risk Index DRI	29
Comparing Consumer Reports Guide with the Environmental Working Group's Shopper's Guide to Pesticides in Produce	31
Calculation of Cancer Risk in CR Guide	31
Fruit Risks Chart	34
Vegetables Risks Chart	35
High Risk Pesticides	37
Labels Guide Chart	38
Stretching Your Produce and Organic Dollar	43
Cleaning Produce	44
Conclusion	45
Consumer Reports' Food Safety and Sustainability Center's Pesticide Policy Recommendations	46
References	49



Introduction: Produce Matters

FRUITS AND VEGETABLES are a crucial part of a healthy diet. In 2015, a report on government dietary guidelines concluded that high levels of fruit and vegetable consumption are strongly or moderately associated with decreased risks of chronic diseases such as heart disease, high blood pressure, type 2 diabetes, obesity and cancer. The report also cites emerging evidence that suggests that dietary patterns with high fruit and vegetable consumption may decrease the likelihood of congenital anomalies as well as neurological and psychological diseases.

Unfortunately, Americans eat far too few fruits and vegetables. In fact, 80 percent of Americans fail to consume the daily recommended number of servings of fruit (1.5 to 2.5 cups for adults), and 90 percent fail to meet the daily recommended number of servings of vegetables (2 to 3.5 cups for adults). That means we are not even close to what some studies suggest may be the ideal number of servings of fruits and vegetables per day: 7 or more.¹ One simple thing you can do to be healthier and live longer is to eat a diet rich in fruits and vegetables.

Unfortunately, not all produce is created equal, and a key part of understanding the health of the produce we eat is to understand how it is produced. Since the industrial revolution, chemical-based pesticides have been used extensively in crop production. Farmers use nearly 700 million pounds of pesticides every year.²

We have come to learn that the widespread use of pesticides in crop production comes with a range of consequences that should affect our thinking on how crops *should* be produced. It also highlights the connections between practices on the farm and what ends up on our table. In an ideal world, pesticides sprayed on a farm field would kill only the targeted pests, then disappear. That, unfortunately, is not the case. Pesticides can harm their intended targets as well as nontargeted living organisms. Pesticides used in agriculture can contaminate not only our food but also the environment, and they're widely present in the air, rain, and rivers. Their use affects not only the consumers who eat the treated crops but also farmworkers, rural residents, wildlife, and pollinators that are exposed.

The good news is that over the past two

decades, there has been quite a bit of progress addressing the use of some of the most toxic pesticides we initially called out in our 1998 report, *Worst First*. That report identified 40 specific insecticide uses on nine fruit and vegetable crops that, together, accounted for a very large portion of children's overall dietary insecticide exposure and risk. But more work needs to be done so that we can maximize the benefits of eating produce, making our produce choices even healthier.

We also know from a Consumer Reports April 2014 national survey that consumers expect more when shopping for food. Eighty-nine percent of people think it is critical to protect the environment from chemicals. In addition, 86 percent think it is critical to reduce pesticide exposure and support fair working conditions.³ Those statistics indicate an overwhelming consumer demand for food produced in a way that is more sustainable and healthier for the environment, workers, and consumers.

This report discusses the

range of issues associated with chemicals used to grow and market the majority of fruits and vegetables in the U.S. It also takes a close look at the specific risks of individual crops, including where and how they are produced. We describe the effects of these chemicals on the environment and human health, and the importance of decreasing our use and exposure to these pesticides. We also discuss emerging science on the potential harms that can't be quantified at this time but, again, can guide us to make better decisions from crop to table.

Ideally *you* wouldn't have to worry about the potential hazards associated with pesticide use in fruit and vegetable production. We offer important recommendations for systemic policy changes that would better protect public health, but until that happens, we provide you with advice on how to reduce your exposure.

In our judgment, the best way to maximize the nutritional benefit from fruits and vegetables while minimizing the

negative effects of pesticides on the health of the environment, workers, and yourself is to buy organic. Federal law requires that food labeled as "organic" must be grown and handled without the use of synthetic chemicals, including synthetic pesticides, with very few exceptions.⁴ Studies have shown that people who eat organic fruits and vegetables reduce their exposure to pesticide residues.⁵⁻⁷

We do recognize that organic can cost more, and organic options may not always be available to all consumers, so we give you advice on how to find nonorganic produce items with the lowest levels of harmful pesticides. We also underscore that *even eating conventionally produced fruits and vegetables is always healthier than not eating any*. This report seeks to empower you, so you can make more informed decisions about the produce you eat and the policies that have an impact on their production. Together, we believe we can create a healthier food system. 🌱

The Intersection of Safety and Sustainability: Why Pesticides Are a Bad Deal

WHEN CHEMICAL PESTICIDES became widely available in the mid-20th century, the dominant approach to pest control in agriculture shifted. Farming practices that naturally prevent and control serious pest problems, such as rotating crops, planting cover crops, providing habitat for pest predators, maintaining diversity on the farm, selecting crops suited for particular

growing conditions and regions, scouting for pests, and labor-intensive weeding, fell out of favor. Farmers no longer needed to rely on those farming practices. They could plant vast fields of single crops—monoculture—and focus on exterminating pests through chemical means.

The chemical approach to pest control, however, has backfired. It has been shown

to be unsustainable because it ignores a basic reality that governs the natural world: adaptation. Within a decade, many pests will adapt and develop resistance to the pesticides designed to kill them.⁸ The National Academy of Sciences writes that "pesticide resistance is now universal across taxa."⁹ It typically takes less than a decade for insects to develop resistance to an

insecticide, sometimes as few as three to four years.¹⁰

The constant and inevitable evolution of resistance and the resulting need to develop and apply new chemicals has put farmers on what has been referred to as a "pesticide treadmill." That continued use and development of new pesticides comes at the expense of public health and the environment, which requires long-term testing and caution for maximum protection. Living organisms are complex, and it takes time to come to a thorough understanding of the often subtle ways that pesticides do harm. It was only after it was banned that the carcinogenic effects of DDT, and its effects on the endocrine system, became clear. As history shows, specific pesticides are approved, and millions of pounds are released into our environment and food supply, before scientists have thoroughly researched and understood their wide-ranging and long-term effects. We cannot assume that the safety tests required today will capture the full spectrum of negative effects and adequately protect us from those harmful effects.^{11,12}

Meanwhile, the long-term negative effects of pesticides on nontarget organisms—including humans—can linger longer than their effectiveness against the pests they are intended to kill. We are still living with the consequences of the pesticides that were used generations ago. Though inorganic arsenic-based pesticides were popular in the early 20th century and are no longer used (they were banned in the U.S. in the 1980s), arsenic continues to contaminate orchards and farm fields.^{13,14} Today arsenic is

considered a known carcinogen implicated as a risk factor for lung, bladder, and skin cancer, as well as a risk for other diseases such as cardiovascular disease and diabetes.¹⁵ In part because of its use as a pesticide in the past and the present, it is found in measurable amounts in highly consumed foods such as apple juice and rice.¹⁶

Likewise, the use of DDT in the mid-20th century continues to affect public health and the environment today. Though DDT was banned more than 40 years ago, its breakdown product continues to be found in certain foods, such as dairy, potatoes, and meat,¹⁷ and consumers are still at significant risk of exposure.¹⁸ Recent reports show that birds are still dying from DDT in towns where the pesticide was manufactured more than 50 years ago.¹⁹ Despite those clear lessons from history, the cycle continues.

Based on history, two things are certain: Pesticides eventually lose their effectiveness, and the full extent of negative effects on human health and the environment will eventually come to light—probably after the pesticide is no longer effective and has been replaced by something new and differently toxic. We believe it is important to step off the "pesticide treadmill" and break the cycle.

Consumers can play a key role in breaking this cycle by buying foods grown without the use of industrial and toxic pesticides.

Pesticides Used Before and After Plant Cultivation

When people think of pesticides applied to crops, they probably picture an airplane flying over a farm field, or a truck with a sprayer driving through an orchard, spraying pesticides on the crops while they are growing. But in the case of many pesticides, application is often applied to seeds, soil, and crops before and after the actual growing period.

For example, on some fruits and vegetables, one-third to one-half of the residues are from pesticides that were not applied in the fields or orchards but in storage.²⁰

Some pesticides applied in storage target insects, but others are used to lengthen the shelf life of the produce. For example, many types of fruit, such as oranges and peaches, are treated with a fungicide to inhibit mold.²¹ Chemicals can also be applied to vegetables after harvest to prevent sprouting.²²

In the case of seeds, fungicides may be applied during storage to prevent molding, or the seeds may be individually coated in pesticides as a prophylactic pest treatment.²³ Additionally, pesticides in the form of gas, known as fumigants, are often injected into the soil prior to planting to sterilize the fields from subterranean pests.²⁴



Human Health Concerns

PESTICIDES ARE UNIQUE among manufactured chemical products. Unlike other chemical products that are designed for a certain purpose and may have toxic properties as an unintended side effect, pesticides are intentionally toxic—*toxic by design*. They are made to interfere with biological functions in living organisms and are manufactured and released into our environment and food supply not in spite of their toxicity but *because* of their toxicity.

Fully understanding and documenting the full range of negative effects on nontargeted living organisms—including humans—requires long-term and in-depth study. Because of the inherent toxicity of pesticides, medical and public health experts have long raised concerns.

For example, the American

Academy of Pediatrics (AAP) points out that there is “a growing body of literature that suggests that pesticides may induce chronic health complications in children, including neurodevelopmental or behavioral problems, birth defects, asthma, and cancer.”³⁹

The President’s Cancer Panel of the National Institutes of Health writes that exposure to pesticides has been linked to brain/central nervous system, breast, colon, lung, ovarian, pancreatic, kidney, testicular, and stomach cancers, as well as Hodgkin and non-Hodgkin lymphoma, multiple myeloma, and soft tissue sarcoma.⁴⁰ Approximately 40 different EPA-registered pesticides that are currently on the market are classified as known, probable, or possible human carcinogens.⁴¹

Although 40 known,

probable, or possible human carcinogens may be a disconcerting number in and of itself, it occupies a small percentage of the approximately 900 registered active ingredients in use today.⁴² Unfortunately, many of these chemicals have not been proved noncarcinogenic but rather fall into the cancer classifications of “not likely to be carcinogenic to humans” and “not classifiable” (because of a lack of sufficient information on which to base an assessment).⁴³

The Environmental Protection Agency (EPA) acknowledges that the associations between pesticide exposure and certain cancer and noncancer chronic health effects are well documented in the peer-reviewed literature and sets tolerance levels for residues to try to protect the public and environment from adverse effects.⁴⁴

Weighing Human Health in Pesticide Approvals

THE EPA APPROVES and sets tolerances for pesticides.

Its regulatory authority comes from two federal laws: the Federal Insecticide, Fungicide, and Rodenticide Act (FIFRA) and the Federal Food, Drug, and Cosmetics Act (FFDCA).⁴⁵

FIFRA authorizes the EPA to approve or deny the registration and use of any pesticide.⁴⁶ While decisions to approve or prohibit a pesticide are based on a risk-benefit approach, FIFRA does not limit the decision to approve a pesticide solely on the human health or environmental *risks* of pesticides. Instead FIFRA also states that “*benefits* of the use of the

pesticide” must be considered.^{47, 48}

A problem with that approach is that the benefits are assessed under the assumption that farmers must have chemicals for pest control.⁴⁹ The availability and feasibility of “alternatives,” including alternative practices used in organic agriculture, do not appear to be given much weight when considering the benefits of the chemical.⁵⁰ And although the EPA’s mission is “to protect human health and the environment,”⁵¹ the law governing the EPA’s approach to pesticide regulation does not require that the risks to human health and

the environment be prioritized over “benefits” in the decision-making process.⁵² One of the ways EPA tries to balance the health risks and purported benefits is to set tolerance levels for most pesticides when they become registered. Tolerances are the maximum amount of the pesticide residue that is allowed to occur on a food, and that is an amount that the EPA expects with reasonable certainty to cause no harm. If levels above a tolerance are found, the government can take enforcement actions. Tolerances are based on a risk assessment conducted by the EPA that



Did You Know?

- The national Centers for Disease Control and Prevention (CDC) has found that 29 different pesticide metabolites—the components of pesticides that remain in the environment or body after they are broken down—are present in the bodies of most Americans.³⁶
- Approximately 40 different EPA-registered pesticides currently on the market are classified as known, probable, or possible human carcinogens.³⁷
- Pesticides may induce chronic health complications in children, including neurodevelopmental or behavioral problems, birth defects, asthma, and cancer.³⁸

takes into account a number of factors, including how much of the pesticide is used, how much is found on food, and the toxicity of the chemical.⁵³ The toxicity is determined by reviewing multiple toxicology studies conducted on animals. Typically the agency will determine what the lowest dose of the chemical is that can have an effect on animals and then apply additional safety or uncertainty factors. The factors are applied for many reasons, including the uncertainty inherent to extrapolate animal studies to humans.⁵⁴

Of course, those tolerance levels are only as good as the data on which they are based. Much of the scientific evidence and data that EPA reviews and relies upon in making its decision during the risk assessment are provided by the companies seeking pesticide registration.⁵⁵ And it is that initial submission of evidence and data that will continue to form the basis for re-evaluations and reviews in the future, despite the potential for the data to become outdated as scientific techniques and our understanding of pesticide toxicity evolves.⁵⁶

Another limitation is that for some effects, animal models may not be adequate for evaluating the effects on humans, such as the development of neurological diseases and the disruption of the human hormone system (endocrine disruption). There is widespread agreement in the scientific community that traditional safety testing does not adequately capture the effects of endocrine disrupting chemicals. A report by the World Health Organization and the United Nations Environmental Programme states:

“For a large range of human health effects, such as female reproductive disorders and hormonal cancers, there are no viable laboratory models. This seriously hampers progress in understanding the full scale of risks.”⁵⁷

The endocrine disruption effects of DDT are a good example of that issue. DDT was banned decades ago because it persists in the environment, accumulates in fatty tissues, and can cause adverse health effects on wildlife.⁵⁸ But it wasn't noted until well after its ban that it could have subtle but important effects on pregnancy, birth weight, and lactation.⁵⁹

Traditional safety testing also assumes that higher doses are more harmful than lower doses.⁶⁰ Yet in 2000, an independent panel of experts convened by the National Institute of Environmental Health Sciences and the National Toxicology Program found that some endocrine disrupting chemicals defy that convention and can have effects at *very low levels*, even below the “no effect” levels determined by traditional toxicology testing methods.⁶¹

In addition, while our ability to understand the true toxicity of individual pesticides is limited, mixtures of pesticides present an even greater challenge to toxicologists.⁶² Unfortunately, finding mixtures of multiple pesticide residues on one type of produce is the rule and not the exception. In addition, consumers probably consume multiple produce items with multiple different residues on them.

Numerous studies have reported that exposure to a combination of pesticides will have unique effects and that

mixtures of pesticides could have “greater than additive” effects.⁶³ Those combined effects are often termed “synergistic” effects. In addition, pesticides are formulated products, which means they are mixtures with an active ingredient and many “inert” ingredients.⁶⁴

Yet the effects of chemical mixtures are largely untested and unknown,⁶⁵ and “mixture assessment” is an evolving discipline within toxicology.⁶⁶ Testing is generally done on individual pesticides rather than on mixtures, and on the active ingredient alone, rather than on the final product with inert ingredients.⁶⁷

The active ingredients may be tested alone for toxicology studies, but the inert ingredients may also be toxic themselves.⁶⁸ And unlike the active ingredients, inert ingredient disclosure on pesticides labels is not required (because of confidential business information protections), making it almost impossible for the public or independent scientists to assess exposure and adverse impacts.⁶⁹

And finally, the EPA continues to make decisions based on incomplete safety information. For example, the agency approved the now widely used neonicotinoid pesticide imidacloprid, even though the EPA stated in a 2008 review that “the existing hazard data base for imidacloprid does not include any immunotoxicity data,” and “therefore, an immunotoxicity study will be required.”⁷⁰ Despite incomplete data, the EPA has approved the pesticide, and residues appear in and on the foods that Americans eat.⁷¹

PESTICIDE TERMINOLOGY

Types of Pesticides

PESTICIDE: A general term for products that control pests. Pesticides can target insects (“insecticides”), plants (“herbicides”), fungi (“fungicides”), or other pests.⁷²

FUMIGANT: A pesticide that produces gas or vapor intended to destroy pests.

HERBICIDE: These products kill weeds and other plants that grow where they are not wanted.

INSECTICIDE: These products kill insects and other arthropods.

FUNGICIDE: Kills fungi (includes blights, mildews, molds, rusts).

Classes of Insecticides

ORGANOCHLORINE pesticides: A class of insecticides commonly used in the mid-20th century. They're very harmful to wildlife, especially birds of prey. Almost all have been banned or phased out in the U.S.^{73, 74}

CARBAMATE pesticides: A class of pesticides that affects the nervous system of insects by disrupting an enzyme that regulates acetylcholine, a neurotransmitter also found in the nervous system of mammals (including humans).^{75, 76}

ORGANOPHOSPHATE pesticides: A class of pesticides that affect the nervous system by disrupting an enzyme that regulates acetylcholine, a neurotransmitter also found in the nervous system of mammals (including humans). They were developed prior to World War II as nerve gas, and they were developed as insecticides because of their similar toxic effects on insects.⁷⁷

PYRETHROID pesticides: A class of pesticides that is the synthetic version of a naturally occurring pesticide found in chrysanthemum flowers. The synthetic version resists degradation by sunlight and persists in the environment.⁷⁸

NEONICOTINOID pesticides: A class of pesticides with a common mode of action that affects the central nervous system of insects, causing paralysis and death. Neonicotinoids are a relatively new class of pesticides and are used heavily as a replacement for older pesticides that have lost their effectiveness. Neonicotinoids are implicated in honeybee deaths and colony collapse disorder.⁷⁹



Risks to Young Children

In a 1993 National Research Council report, public health experts articulated their concern that infants and children can be especially vulnerable to the effects of pesticides.⁸⁰ In 2012 the American Academy of Pediatrics reiterated that point, based on a growing body of scientific literature that links pesticide exposure to chronic health complications in children, including neurodevelopmental or behavioral problems, birth defects, asthma, and cancer.⁸¹

Most insecticides have neurotoxic potential, and children can be especially vulnerable because of their stage of development, differences in metabolism, and inability to detoxify compounds.⁸² Infants and children also eat more food per pound of body weight than adults. There are other, less obvious, reasons children may be more vulnerable to pesticide exposure. Children's bodies have much lower levels of detoxifying enzymes that deactivate widely used

pesticides.⁸³⁻⁸⁶ Children may also be vulnerable because their immune and nervous systems are still developing, in some cases through adolescence.⁸⁷

Some of the most widely used insecticides, such as chlorpyrifos,⁸⁸ belong to a class of pesticides called organophosphates.⁸⁹ Those pesticides are designed to interfere with nerve transmissions in insects—enough that it kills them. But the enzymes that those pesticides target in insects are also found in the nervous systems of mammals, including humans.⁹⁰ The Environmental Protection Agency readily acknowledges that organophosphates are neurological toxicants to mammals.⁹¹

The National Institutes of Health (NIH) has voiced concern that studies in animals show that “even a single, low-level exposure to certain organophosphates, during particular times of early brain development, can cause permanent changes in brain chemistry as well as changes in behavior, such as hyperactivity.”⁹² And the NIH

has pointed out that early childhood exposures to certain organophosphate pesticides, which can go undetected because of the lack of overt symptoms, can lead to lasting effects on learning, attention, and behavior.⁹³

Research is now beginning to show adverse effects on the neurological development of children who are exposed to organophosphate pesticides from the foods they eat. A 2010 study by researchers at the Harvard School of Public Health found that children with higher levels of organophosphate pesticide metabolites in their urine were more likely to be diagnosed with attention deficit hyperactivity disorder (ADHD). Their conclusion: “These findings support the hypothesis that organophosphate exposure, at levels common in U.S. children, may contribute to ADHD prevalence.”⁹⁴

Other types of pesticides, including those belonging to the neonicotinoid class, which are supposedly safer alternatives to organophosphates, also target the nervous system and are potentially harmful.⁹⁵ There are few studies on the precise effects of neonicotinoids on mammals, and emerging developmental neurotoxicity studies raise concerns that these pesticides may adversely affect human health, especially the developing brain.^{96, 97}

Organophosphate Pesticides

- ☞ Organophosphate pesticides are toxic to the neurological system.
- ☞ 33 million pounds of organophosphate pesticides were used in the U.S. in 2007.⁹⁸
- ☞ Organophosphate pesticides are prohibited on organic farms.
- ☞ Children who eat organic fruits and vegetables drastically reduce their exposure to organophosphate pesticides.
- ☞ Organophosphate pesticide metabolites are detected in the urine of children who eat conventional fruits and vegetables.
- ☞ Children with higher levels of organophosphate pesticide metabolites in their urine were more likely to be diagnosed with attention deficit hyperactivity disorder (ADHD) in a 2010 study.⁹⁹



Most insecticides have neurotoxic potential, and children can be especially vulnerable, because of their stage of development, differences in metabolism, and inability to detoxify compounds.



Food Quality Protection Act (1996): Focus on Protecting Children

In 1996, Congress unanimously passed the Food Quality Protection Act (FQPA), which amended two existing laws that address pesticides (the Federal Insecticide, Fungicide, and Rodenticide Act and the Federal Food, Drug, and Cosmetics Act).¹⁰⁰

The FQPA required the EPA to ensure that pesticide tolerances are safe for all vulnerable populations, including infants and children. When setting tolerance levels for residues on foods, the law requires that the EPA account for aggregate exposure from multiple sources of pesticide residues and apply an additional tenfold safety factor to the “reasonable certainty of no harm” tolerance-setting calculations, unless reliable and sufficient information exists for the agency to determine that another factor provides

adequate protection.¹⁰¹ That is to account for the unique risks faced by infants and children and to ensure that all sources of pesticide residue consumption and exposure (food, water, residential uses) are included in the calculations.¹⁰²

In 2001 Consumer Reports reported that in more than two-thirds of decisions on organophosphate pesticides, the EPA did not apply a tenfold safety factor. The EPA applied a tenfold safety factor in only 16 percent of decisions, and in another 16 percent of decisions, the EPA applied a threefold safety factor.¹⁰³

As of 2006, it appears that the FQPA has led to a reduction in pesticide dietary risks, especially from domestic produce.^{104, 105}

Q&A With Dr. Charles Benbrook

Q&A with Dr. Charles Benbrook on the progress since passage of legislation in 1996 mandating a reduction in pesticide use, with a special focus on crops affecting children and the challenges that still lie ahead.

Dr. Benbrook has been a collaborator on this project and is the co-author of Consumer Reports’ 1998 groundbreaking pesticide report, *Worst First*.

CR: What are the victories since the passage of FQPA almost 20 years ago? What progress have we seen?

Benbrook: Getting the EPA focused and moving to better protect vulnerable populations, especially pregnant women, infants, and children, was by far Consumer Reports’ most important accomplishment. In a 1998 report called *Worst First* (WF), Consumer Reports urged the EPA to act quickly and aggressively in reducing, or better yet eliminating, the major children’s food uses of the most toxic half-dozen organophosphate (OP) insecticides. The seminal 1993 NAS report “Pesticides in the Diets of Infants and Children” explained the compelling scientific case for action against the top OP risk drivers. The report named names and identified 40 pesticide-food combinations then accounting for disproportionately high risks to infants, children, and pregnant women. OPs accounted for 25 of those 40 pesticide-food combinations. Carbamate insecticides were the risk drivers in 13 more.

Since FQPA implementation began in 1996, U.S. growers have eliminated 99 to 100 percent of the risk stemming from 16 of the 40 Worst First pesticide uses. Overall among conventional farmers in the U.S., the cumulative risk accounted for by the 40 WF uses went down 85 percent. Consumer Reports obviously picked the 40 WF uses well, because they accounted for 66 percent of overall risk in 1996, across all 200-plus pesticides applied on 150-plus foods.

Taking into account both domestically grown and imported conventional foods, 40 WF risk fell 77 percent, and overall dietary risks were reduced 81 percent from 1996 to 2013.

But Department of Agriculture (USDA) pesticide residue data shows that there is unfinished business, still, on the 40 WF uses. Those uses accounted for 60 percent of overall risk in 1996, and though risks have gone down 81 percent since 1996, the 40 WF still account for 70 percent of total risk in 2013.

CR: What challenges still remain? What crop/pesticide combinations are still being used that are harmful to consumers, especially the most vulnerable populations?

Benbrook: Acephate, and its breakdown product methamidophos, on green beans was the No. 1 risk driver in 2013. That use accounted for around one-half of total risk across all pesticides and food. Residues and risk stemming from a half-dozen post-harvest fungicides applied in packing sheds and warehouses are a growing concern. Iprodione residues in fruits such as plums, nectarines, and peaches account for 10 percent or more of annual, overall risks. Fludioxonil, another packing-shed fungicide, is also often found in soft-skinned fruits, and a third fungicide, imazalil, is a problem in bananas, citrus, and some other fruits. The very high-risk insecticides chlorpyrifos and oxamyl still show up regularly in peppers, squash, and several other fruits and vegetables.

There are growing reasons for concern about residues of the widely used herbicide glyphosate as well, along with the half-dozen systemic insecticides in the nicotinyl family (the pesticides implicated in honeybee colony collapse disorder). While glyphosate and the nicotinyls are not nearly as toxic ounce for ounce as the 40 WF OPs, there are far more residues of both in the U.S. food supply and drinking water than of the OPs, even back in the mid-1990s. Risk, after all, is a function of dose, toxicity, and time of exposure (i.e., a person’s age and health status). The EPA needs to finish the job of getting the rest of the WF risk driver uses out of the food supply. Then it needs to sharpen its focus on the handful of pesticides used in conjunction with GE (genetically engineered) crops.

Risks to Farmers and Farmworkers

Compared with the general population, farmers, farmworkers, and even their children are at higher risk because they experience more direct exposure to pesticides, at higher doses, and through various routes (e.g., inhalation after spraying).¹⁰⁶

The Environmental Protection Agency (EPA) estimates that 10,000 to 20,000 physician-diagnosed pesticide poisonings occur each year among the approximately 2 million people who work in agriculture.¹⁰⁷ Those numbers, however, may actually underestimate the problem, because studies also estimate high rates of underreporting by workers.¹⁰⁸

Farmers and farmworkers are at higher risk not only for acute poisoning but also for illnesses associated with long-term exposure. The EPA has identified at least six chronic diseases that have a well-documented association with agricultural pesticide exposure: non-Hodgkin lymphoma, prostate cancer, Parkinson's disease, lung cancer, bronchitis, and asthma.¹⁰⁹

Laws and Regulations: Protecting Farmworkers

Most workers in the U.S. labor force are protected by standards of the Occupational Safety and Health Administration (OSHA) of the Department of Labor. Because they are places of employment, farms are required under the Occupational Safety and Health Act to provide safe and healthful working conditions to workers.¹³⁵ However, OSHA standards actually do very little to protect farmers and farmworkers from the harmful effects of pesticides.

The OSHA standards for agriculture that deal with pesticides are limited to "hazard communication"¹³⁶ and informing workers of the importance of "good hygiene practices" such as hand washing before eating to minimize exposure to agrochemical residues.¹³⁷ Farms with fewer than 10 employees are exempt from those OSHA regulations.¹³⁸

Through the Federal Insecticide, Fungicide, and Rodenticide Act (FIFRA), the responsibility to regulate pesticides, including to protect farmworkers from the hazards of pesticide exposure, has been assigned to the EPA. Specifically, in 1972 amendments to

FIFRA, Congress directed the EPA to take steps to protect humans and the environment from unreasonable adverse effects of pesticides, with the intent that farmers and farmworkers should be among those afforded protection.¹³⁹ As a result, the EPA issued its Worker Protection Standard (WPS) in 1992.¹⁴⁰

The EPA relies on risk-benefit analyses in its approach to protecting farmers and farmworkers from the toxic effects of pesticides. The EPA acknowledges that "the associations between pesticide exposure and certain cancer and non-cancer chronic health effects are well documented" and that reducing the risk of harm is an important goal.¹⁴¹ The agency's approach to protecting workers is not to prohibit the use of pesticides that are harmful to human health but rather to set requirements focused on reducing exposure to those hazardous pesticides. That includes requiring employers to provide personal protective equipment, clear instructions on pesticide labeling, and basic safety training as well as setting basic requirements for notification of treated areas and restricting entry by farmworkers after pesticides have been applied.¹⁴²

Those protections are important but have been inadequate.¹⁴³ For example, the EPA has found that risks from certain organophosphate exposures to farmworkers still exceed the agency's level of concern, even when all provisions in the current WPS are followed.¹⁴⁴ After years of pressure from various farmworker advocacy groups to afford farmworkers better protections from pesticides, the EPA is in the process of revising its standards (as of date of publication).¹⁴⁵ While the agency proposes to strengthen the protections from exposure, the basic risk-benefit approach remains the same and harmful pesticides will probably continue to be used.¹⁴⁶

Risks to Rural Residents

Pesticides contaminate the air, water, and even rain, with levels of contamination higher in rural areas near farms. That contamination affects those living near farms, who have been shown to have higher

levels of pesticide metabolites in their urine than those living farther away.¹⁴⁷ This puts rural residents at potentially higher risk.

Childhood exposure to pesticide residue is especially concerning. The neurological effects from pesticide exposure on children living near farm fields were first documented in the late 1990s. A study in Mexico compared children living in an agricultural valley where pesticide use is common with children living in the foothills of the same region, where pesticide use is less common. The children in the valley, who had been exposed to multiple pesticides in pregnancy and childhood, had more decreases in stamina, eye-hand coordination, memory, and figure-drawing skills than the children living in the foothills.¹⁴⁸

Research in the agricultural Salinas Valley in California (the CHAMACOS study) has linked organophosphate pesticide exposure in the womb and early childhood to lower birth

weight,¹⁴⁹ neurodevelopmental delays and problems with attention,¹⁵⁰ and reduced IQ.¹⁵¹ Those deficits in IQ are similar to those documented from exposure to lead.^{152, 153}

And a 2014 study found higher rates of autism in California children who were exposed to organophosphates and other types of pesticides when their mothers were pregnant with them. Children of mothers who were living within a mile of a farm field that was treated with a pesticide during their pregnancy were found to be at 60 percent increased risk for autism spectrum disorder.¹⁵⁴

The effects on the neurological system of rural residents starts in the womb and continues throughout life. Studies have suggested that people living in regions with greater pesticide use may be more likely to develop Alzheimer's disease, Parkinson's disease, and multiple sclerosis as compared with people living in regions with lower pesticide use.¹⁵⁵⁻¹⁵⁸



CHAMACOS Farmworker Family Health Study

Organophosphate pesticides are toxic to the neurological system at high doses, but much remains unknown about how long-term, low-level pesticide exposure in the womb, during infancy, and during early childhood affects health and development.

To shed more light on questions of pesticide exposure and growth, health, and development, researchers involved with the CHAMACOS (Center for the Health Assessment of Mothers and Children of Salinas) study in California have followed hundreds of children living in the agricultural Salinas Valley in California from birth to age 12. The study began in 1998 and is funded by the National Institute for Environmental Health Sciences, the Environmental Protection Agency, the National Institute for Occupational Safety and Health (2001 to 2006), and private foundations.

CHAMACOS study researchers have published studies linking pesticide exposure to lower birth weight, neurodevelopmental delays, lower IQ, and other adverse effects:

Neurodevelopmental development & attention (2010):

Exposure to organophosphate pesticides in the womb, as measured by the levels of the pesticide's breakdown products in the pregnant mother's urine, is associated adversely with attention, especially when measured at age 5, and especially in boys.¹⁵⁹

Reduced IQ (2011):

Prenatal exposure to organophosphate pesticides is associated with poorer intellectual development in 7-year old children. Children whose mothers had the highest levels of pesticide residues in their urine when they were pregnant have an average deficit of 7 IQ points compared with the children whose mother's urine had the lowest pesticide levels while pregnant.¹⁶⁰

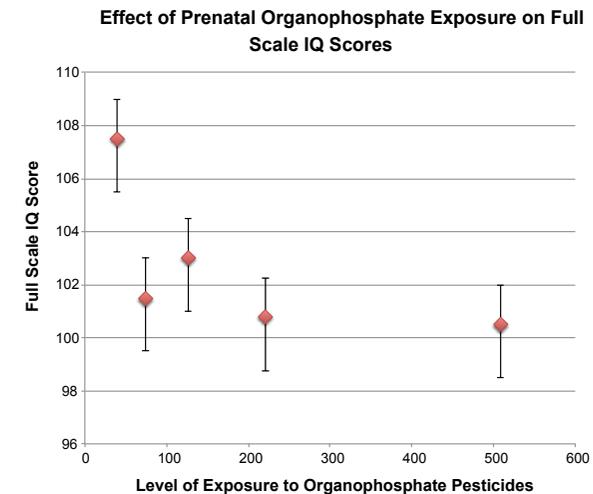
Birth outcomes (2013):

Living near farms where methyl bromide was used during pregnancy is associated with lower birth weight and shorter birth length.¹⁶¹

Jeff Greenberg / Getty Images

Higher prenatal exposure to organophosphate pesticides, as measured by levels of organophosphate breakdown products in the mother's urine during pregnancy, are associated with lower scores on their children's cognitive tests when performed at age 7.

The figure from a study of the CHAMACOS cohort shows how scores on a Full Scale IQ test are highest for those in the group with the lowest average levels of prenatal exposure compared to those in groups with higher levels of exposure.



Source: Bouchard et al, 2011





Strawberries: A Case Study in Industrial Agriculture

Conventional strawberry production, with its heavy reliance on chemicals, comes at a high cost to the environment and public health.

Conventional strawberry farmers often fumigate the soil to essentially sterilize it, killing most living organisms in the soil.¹¹⁰ Conventional strawberry growers have relied for years on the fumigant methyl bromide.¹¹¹ Methyl bromide is banned by international treaty because it depletes the ozone layer.¹¹² The EPA has granted “critical use exemptions” that have allowed California strawberry growers to continue using it.¹¹³

In an estimated 30 to 50 percent of agricultural applications, methyl bromide is released into the air when applied to the soil, even if soil covers are used.¹¹⁴

Methyl bromide is a “multisystem toxicant,” producing severe and sometimes permanent nervous system effects.¹¹⁵ Methyl bromide is also considered a potential occupational carcinogen¹¹⁶ and has been linked to an increase in stomach cancer rates among farmers.¹¹⁷ Methyl bromide poses risks not only to farmworkers but also to people living nearby.¹¹⁸ Studies of men who were not necessarily farming themselves but who were exposed to “ambient pesticides” from nearby farms found evidence of a strong association between exposure to methyl bromide and

prostate cancer risk.^{119, 120}

In addition, high methyl bromide use within close proximity of the home during the second trimester of pregnancy adversely affects babies (lower birth weight and shorter birth length).¹²¹

Methyl bromide is just one of many toxic pesticides used by conventional strawberry growers. Other types of pesticides, such as neonicotinoids (see “Honeybees and Pollination” section), are also used.



Agricultural Population Health Study

The Agricultural Health Study (AHS) is a prospective cohort study that is investigating the effects of environmental, occupational, dietary, and genetic factors on the health of the agricultural population. The first interview was conducted in 1993, and within four years nearly 90,000 participants had been enrolled.

The research project is sponsored by the National Institutes of Health, the Environmental Protection Agency, and the National Institute for Occupational Safety and Health. A major focus of the study is looking at the health effects of pesticides.

Some of the study's findings to date:

• Acute poisoning

People who work in agriculture are at risk for acute poisoning by pesticides, which can be fatal. Almost a quarter (23 percent) of pesticide applicators have reported at least one “high pesticide exposure event” in their lifetime. Episodes of high exposure are associated with changes in health, including subtle changes in memory and attention as well as increased respiratory disease.^{122, 123}

• Cancer

In general, farmers have lower levels of cancer than the rest of the population; however, rates of certain types of cancer are higher among farmers, including prostate cancer and ovarian cancer.¹²⁴ AHS researchers identified three organophosphate pesticides and one organochlorine pesticide that are significantly associated with aggressive prostate cancers.¹²⁵

Interestingly, participants in the AHS study have lower rates of lung cancer than the general population, probably because of low levels of smoking among farmers. But when researchers examined the relationship between pesticides and lung cancer incidence, they found that two widely used herbicides and two widely used insecticides—including the widely used organophosphate chlorpyrifos—increased the odds of being diagnosed with lung cancer in a dose-dependent fashion.¹²⁶ AHS researchers have also observed a significant increase in stomach cancer risk in those with high exposure to the soil fumigant methyl bromide,¹²⁷ which is the third most widely used soil fumigant.¹²⁸

AHS researchers have also found a potential for increased risk of colorectal cancer in farmers with high levels of exposure to two insecticides: chlorpyrifos and aldicarb.¹²⁹⁻¹³¹

• Thyroid disease

Surprisingly little is known about the causes of thyroid disease or abnormal thyroid hormone levels, conditions that affect between 1 and 9 percent of American adults.¹³² Hypothyroidism is the most common type of thyroid condition, which can cause weight gain, excessive tiredness, and sensitivity to cold.¹³³

In a 2013 study involving more than 22,000 male pesticide applicators, AHS researchers found a link between the use of many kinds of pesticides and hypothyroidism. There were increased odds of hypothyroidism with the use of six herbicides and eight insecticides. The analysis shows increasing odds of developing hypothyroidism with increasing level of exposure to two of the herbicides and five of the insecticides.¹³⁴

Environmental and Ecological Concerns

ONE OF THE inherent drawbacks of pesticides is that it is not possible to design the chemicals to kill only plants and animals that farmers consider “pests” while sparing all others. Many of the most commonly used classes of pesticides are very toxic to other living organisms, including beneficial insects and wildlife.¹⁶⁶

For example, the most widely used soil fumigant,¹⁶⁷ metam sodium, is toxic to turtles. When researchers applied this pesticide to a farm field, then deposited snapping turtle eggs in the treated soil, they found that even at the lowest rates of metam sodium application, all of the turtle eggs died.¹⁶⁸

Pesticides can also be harmful to birds. It has been estimated that 72 million birds die each year from pesticide poisoning, but the U.S. Fish and Wildlife Service states that this is an underestimate.^{169, 170}

The difficulty of controlling where pesticides and their metabolites end up makes it even more challenging to avoid unintended impacts to wildlife and the environment. Pesticide drift, runoff, leaching, and persistence all factor into a pesticide’s ability to migrate into the environment and pose threats to the health of people and ecosystems beyond the boundaries of the fields where a chemical is applied.¹⁷¹

Atrazine, one of the most broadly used herbicides in the United States, is a prime example.¹⁷² After scientific research revealed atrazine’s pervasive presence in water supplies, detrimental impacts

to aquatic life, and potential human health threats, the European Union banned atrazine in 2004.¹⁷³ Despite similar evidence available in the U.S., however, the EPA concluded after a review of atrazine in 2003 to allow the chemical’s continued use with some additional restrictions.^{174, 175}

Of course, atrazine is only one of many pesticides finding its way into surface and drinking water, soil, and air. And although monitoring requirements and regulatory safety standards exist to some degree for atrazine and other pesticides, many remain unmonitored and lacking in adequate safety standards.¹⁷⁶

Honeybees and Pollination

The EPA acknowledges that most insecticides are toxic to bees.¹⁷⁷ Healthy populations of bees are critical to maintaining a healthy food supply. Almonds, apples, cherries,

blueberries, squash, and pumpkins are just a handful of the 90 commercially grown crops—mostly fruits and vegetables—that depend on honeybees for pollination.¹⁷⁸

In southwest China, where wild bees have been eradicated by excessive pesticide use and a lack of natural habitat, farmers have been forced to painstakingly pollinate their apple and pear trees by hand.¹⁷⁹

Beekeepers in the U.S. have noticed devastating declines of honeybees in recent years: losses of entire beehives, known as colony collapse disorder (CCD). According to a survey funded by the USDA, 31.1 percent of managed honeybee colonies were lost nationwide during the winter of 2012 to 2013.¹⁸⁰

The EPA and USDA claim that CCD cannot be explained by a single factor. However, the National Research Council notes that pesticides can have negative effects on bee behaviors,¹⁸¹ and independent research increasingly points to a specific class of pesticides, neonicotinoids, as a major cause of CCD.¹⁸²⁻¹⁸⁸ The first of the neonicotinoid pesticides, imidacloprid, was registered with the EPA in 1994.¹⁸⁹

According to the EPA, neonicotinoids are highly toxic to aquatic organisms, honeybees, and other beneficial insects on an acute basis.¹⁹⁰ Neonicotinoids disrupt the central nervous system of insects, causing paralysis and death from acute exposure.¹⁹¹

The EPA generally attempts to protect honeybees from the acute toxic effects of pesticides,

including neonicotinoids,¹⁹² by urging farmers not to apply pesticides while bees are foraging. But neonicotinoids are unique in several ways, which has an impact on how they affect honeybees.

First, neonicotinoids are systemic pesticides, which means they are absorbed in every tissue of the plant. As the plant grows, it continues to release small doses of the insecticide through every tissue, including nectar and pollen, thereby continuing to expose beneficial insects such as honeybees that forage on those crops.¹⁹³

Second, neonicotinoids probably have harmful effects even at low doses that are not acutely toxic but ultimately affect long-term honeybee survival. Recent research increasingly links low-level exposure to neonicotinoid pesticides with effects on the memory¹⁹⁴ and navigational skills of bees, and survival of hives.¹⁹⁵⁻¹⁹⁸ A growing number of independent scientists are concluding that *sublethal* exposure to neonicotinoids is probably the main culprit for the

occurrence of CCD.¹⁹⁹

The European Food Safety Authority (EFSA) reviewed recent science,²⁰⁰⁻²⁰² and based on the findings, the European Commission announced in 2013 that it would impose a two-year restriction on the use of three commonly used neonicotinoid pesticides.²⁰³

Laws and Regulations: Protecting Wildlife and Pollinators

The Federal Insecticide, Fungicide, and Rodenticide Act (FIFRA) requires the EPA to ensure that a pesticide will not pose unreasonable harm to the environment, and defines “environment” as “water, air, land, and all plants and man and other animals living therein, and the interrelationships that exist among these.”²⁰⁴

However, it is very uncommon for the EPA to either deny or cancel an existing registration for a pesticide solely or largely because of risks to birds, fish, and other nontarget (non-human) organisms.²⁰⁵

Despite the mounting evidence implicating neonicotinoids in honeybee deaths, and pressure from environmental and public interest groups, the EPA continues to allow the use of neonicotinoids in agriculture.



The Impacts of Pesticide Use

Pesticides are designed to be toxic to living organisms.

Rural residents. 2,4-D and other chlorophenoxy herbicides are listed as 2B carcinogens, “possibly carcinogenic to humans.”²⁰⁶ Studies show an association between cancer mortality and living near farm fields treated with the herbicide 2,4-D.²⁰⁷

Honeybees. Neonicotinoids are linked to honeybee colony collapse disorder.²⁰⁸ Nearly one-third of managed honeybee colonies were lost during the winter of 2012 to 2013.²⁰⁹

Monarch butterflies. Glyphosate kills the milkweed plant, the primary food of monarch butterflies. Monarch butterfly populations have declined drastically over the past decade.²¹⁰

Children. Organophosphate pesticide exposure has been linked to deficits in IQ,²¹¹ attention,²¹² and behavioral development in children.

Rural residents. Atrazine is a suspected endocrine disruptor, which may affect reproductive health and children’s sexual development.²¹³

Turtles. The eggs of snapping turtles die when they are laid in a field treated with the common soil fumigant metam sodium.²¹⁴

Farmers. High pesticide exposure among farmers has been linked to prostate and ovarian cancer,²¹⁵ lung cancer,²¹⁶ stomach cancer,²¹⁷ hypothyroidism,²¹⁸ changes in memory and attention,²¹⁹ and increased respiratory disease.²²⁰

Birds. An estimated 72 million wild birds die each year from pesticide exposure.²²¹

Farmworkers. 10,000 to 20,000 physician-diagnosed pesticide poisonings occur each year among farmers and farmworkers.²²²



Why Organic is the Right Way

Federal law prohibits the use of almost all synthetic pesticides on organic farms.



- There are currently no synthetic herbicides approved for use on organic food crops.²²³
- All neonicotinoids are prohibited on organic farms.
- The herbicide glyphosate is prohibited on organic farms.
- All organophosphate pesticides are prohibited on organic farms.
- Only 10 synthetic insecticides are approved for use on organic farms. Some can be used only if they do not come into direct contact with soil or crops (e.g., as bait traps).²²⁴
- The herbicide atrazine is prohibited on organic farms.
- Synthetic soil fumigants are prohibited on organic farms.
- Biodiversity is richer in organic than conventional farm fields.²²⁵
- Children who eat organic fruits and vegetables have fewer pesticide residues in their bodies compared with children who eat conventional fruits and vegetables.²²⁶

Genetically Engineered Crops and Increased Herbicide Use

The same chemical companies that manufacture herbicides have genetically engineered crops such as corn, cotton, and soybeans to resist the application of their herbicides. Herbicides that would damage or kill the crops can be applied directly to the genetically engineered crop, and the resistant crop survives the herbicide application. Genetic engineering of crops is different from traditional plant breeding because it requires intensive genetic overwriting to allow for genetic changes that cannot occur in nature.

And some crops are genetically engineered to produce their own pesticides instead of having the pesticides sprayed onto crops or into the soil where they are planted. This type of genetic engineering incorporates a “natural” pesticide from bacteria into the genetic makeup of a crop. And because it is secreted by the plant, it cannot be washed off.²³⁰

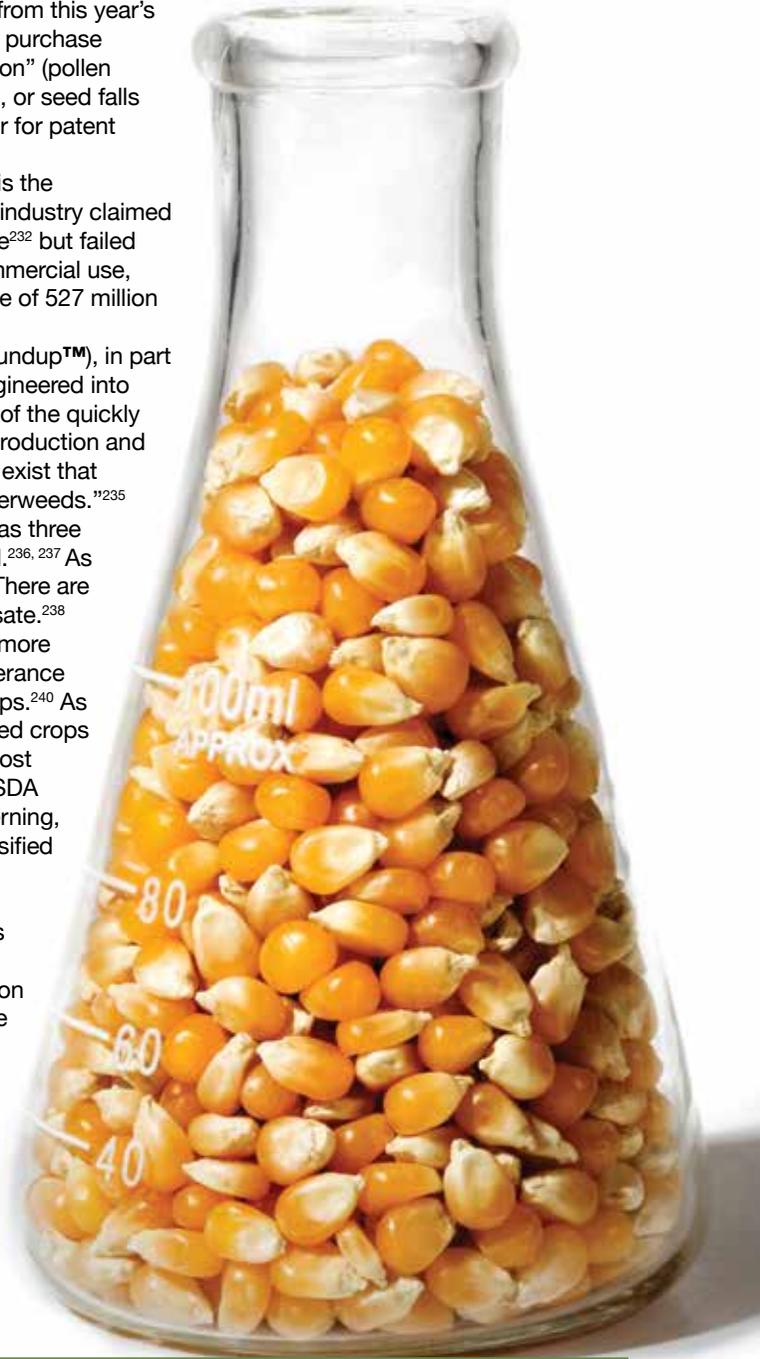
There are many ethical concerns around the genetic engineering of plant seeds and crops. Biotech corporations can patent genetically engineered seed and animals. That means corporations can claim ownership of the genetic code, and most important, ownership of the seed. That has eliminated the possibility of continuing the age-old practice of saving seed from this year’s crop for next year’s planting; instead, farmers are required to purchase new seed annually. If a farmer is the victim of “genetic pollution” (pollen from a genetically engineered plant drifts onto his or her field, or seed falls off a truck onto a farm field), the company can sue the farmer for patent infringement.²³¹

Another major concern with genetically engineered crops is the accompanying increase in pesticide use. The biotechnology industry claimed that genetically engineered crops would reduce herbicide use²³² but failed to deliver on those promises. During the first 15 years of commercial use, genetically engineered crops were responsible for an increase of 527 million pounds of herbicide use.²³³

One of the most widely used herbicides is glyphosate (Roundup™), in part because resistance to this chemical has been genetically engineered into popular commodity crops such as corn and soy.²³⁴ Because of the quickly expanding use of genetically engineered crops since their introduction and the corresponding expansive use of glyphosate, weeds now exist that are resistant to glyphosate, earning them the nickname “superweeds.”²³⁵ Resistance can develop relatively fast; in fact, within as little as three years, weed species may develop resistance to the chemical.^{236, 237} As a result, glyphosate may quickly be losing its effectiveness. There are currently at least 28 weeds with known resistance to glyphosate.²³⁸

With reduced effectiveness, farmers have used more and more glyphosate.²³⁹ For that and other reasons, the EPA raised tolerance levels for glyphosate to allow for expanded uses on food crops.²⁴⁰ As a result, recent studies have shown that genetically engineered crops often retain higher levels of glyphosate residues.²⁴¹ Yet the most recent pesticide-tolerance-testing reports released by the USDA failed to test for glyphosate residues.²⁴² Perhaps most concerning, the International Agency for Research on Cancer (IARC) classified glyphosate as a probable human carcinogen.

Rather than acknowledge the value in diversified crop practices and moderated pesticide use, chemical companies have come up with a new wave of genetically engineered common crops for resistance to the herbicide 2,4-D in addition to resistance to glyphosate. Much like the case of glyphosate and the first round of glyphosate-tolerant crops, the USDA estimates that approval of these new genetically engineered crops would lead to an increase in the use of 2,4-D on crops from 77.8 million to 176 million pounds.²⁴³ The USDA approved these 2,4-D-resistant varieties in six states in September 2014 and as of April 2015 is currently considering whether to approve expanded uses in 10 additional states.^{244, 245} In June 2015, IARC classified 2,4-D as a possible human carcinogen.



Reducing Exposure to Pesticides: What Consumers Can Do

It is important to remember that fruits and vegetables are a crucial part of a healthy diet. People who eat plenty of fruits and vegetables tend to be healthier and live longer, even when the produce they eat comes from conventional production systems. But there are many benefits to making choices that avoid pesticide residues and support farmers who are committed to reducing their dependence on pesticides. To help consumers make such informed decisions, we created two guides.

The first is our guide to residue risk, which is based on a comprehensive analysis of government data to generate a produce Dietary Risk Index (DRI). This index can help consumers minimize the risk from exposure to pesticide residues. The methodology for creating the index and our findings is explained in detail below. This index can help consumers minimize their risk from exposure to pesticide residues in and on the foods they eat.

The second is our guide to labels. Labels on foods can help consumers understand the way the food was produced, and better understand which pesticides were used even when there are low or no residues on the fruits and vegetables. For example, soil fumigants may not show up as residues on fruits and vegetables but their use may have negative impacts on farmworkers, rural residents and wildlife. Moreover, some pesticides that have low known risks to consumers (and therefore do not contribute significantly to DRI) may have unknown risks or known risks to the environment, wildlife and pollinators. Labels can vary widely in how meaningful they are, what the standards require, and how they are verified. Understanding labels is therefore critical for consumers who wish to support farmers following strict and verified standards that reduce the use of pesticides.

Did You Know?

A 2015 study found that people who ate conventionally grown produce had high concentrations of organophosphate pesticide metabolites in their urine, and people who reported eating organic produce had significantly lower levels.²²⁷

A 2006 study found that levels of two organophosphate pesticide metabolites in the urine of children fell to undetectable levels when the children were switched to an organic diet.²²⁸

A 2010 study suggests that children with higher levels of organophosphate pesticide metabolites in their urine are more likely to be diagnosed with attention deficit hyperactivity disorder (ADHD).²²⁹ All organophosphate pesticides are prohibited in organic agriculture.



4 digit: XXXX
Conventionally Grown

5 digit: 9-XXXX
Certified Organic

Tip for navigating the produce aisle:

Unpackaged, fresh fruits and vegetables are sold with a code, often found on stickers or rubber bands. If the code has four digits, the produce is conventional. If the code has 5 digits and starts with a “9,” that signifies that the produce is certified organic.

A Consumer Reports Guide to Residue Risk

Creating the Dietary Risk Index (DRI)

THE DRI IS a scoring system that compares the relative noncancer “risks” of pesticide exposures from different food sources, risk trends over time, and differences in relative risk levels in domestically grown vs. imported foods. It depends on EPA-recommended pesticide dietary risk assessment methods and takes into account:

- The amount and frequency of **residues** on a given food, as reported in annual Department of Agriculture pesticide residue-testing results.
- The typical **serving size** of that food.
- The **weight** of the person consuming that food.
- The **toxicity** of the pesticide as determined by the Environmental Protection Agency.

Below we explain in detail where the above data is obtained and how it is used to calculate the DRI score.

Data Sources

Residue Data: USDA PDP

The Department of Agriculture (USDA) monitors the presence of pesticide residues on foods through its Pesticide Data Program (PDP). In operation since 1991, the PDP tests a variety of food commodities from many different states, and some countries abroad, throughout a given year for residues of hundreds of pesticides. Congress designed the program to collect pesticide residue data in food “as eaten” to help the EPA sharpen the accuracy of its dietary risk assessments. Since the beginning, the PDP has focused mostly on foods that play an important role in the diets of infants and children.

The program focuses on testing a different set of approximately 12 to 15 commodities each year (though some items are tested for two to three years in a row). According to the USDA, commodities are purchased in bulk at terminal markets and large chain-store distribution centers as close as possible to the point

of consumption. The samples are chosen at random based on availability without regard for the origin of the item, though origin data is recorded. In many cases, large samples of products from both the U.S. and multiple other countries are collected, as well as smaller samples of imported and domestic organic products for some commodities.

After collection, samples are shipped to one of seven state laboratories, where they are prepared for residue testing by first washing, then removing inedible portions to mimic consumer experience. About 5 pounds of each food are then blended together into a composite sample. The USDA publishes an annual report that summarizes the results by food and pesticide. Detailed, raw data files are also made available; information in them includes commodity name, purchase state (e.g., California), purchase date, country of origin, production claims (such as organic), and pesticide residue level for each sample. Using that data, the frequency of finding a given pesticide on each food can be calculated, as can average residue levels in positive samples (called “mean of the positives”). In our analysis, we used only the most recent year(s) that data was available for a given produce-country-production method combination. Data was derived from the 2002 to 2013 testing years.

Serving Size: USDA

The serving size for a given food tested by PDP is obtained from the USDA recommendations for fruit and vegetable consumption, or other standard references addressing typical, average single-serving sizes.



Weight

Our calculations for the DRI are based on the diet of a 3½-year-old child estimated to weigh 16 kilograms (35.2 pounds). We chose to base our calculation on the diet of a child because children are especially vulnerable to the dietary risks from pesticides and because they have a relatively substantial amount of intake of a variety of foods relative to their body size (large dose). In addition, as part of its responsibilities set forth in the Food Quality Protection Act (FQPA), the EPA is required to consider the unique effects of pesticides on infants and children as their brains and bodies mature.

Toxicity Data: EPA Risk Assessment

The Environmental Protection Agency (EPA) has developed chronic reference doses (RfDs) for pesticides. RfD values are used by regulatory agencies to set limits for human exposure to chemicals via the diet and drinking water.

RfDs are the amount of a substance that people can consume on a daily basis, consistent with “a reasonable certainty of no harm,” both in the short run and over a lifetime. However, RfDs do not take the risk of cancer into consideration, nor has the agency developed test methods to take into account epigenetic or endocrine-system-driven impacts. RfDs are expressed as milligrams of a pesticide’s active ingredient per kilogram of body weight per day.

RfDs are calculated by evaluating multiple toxicology studies. The EPA first identifies the study producing a statistically significant,

adverse impact at the lowest dose level, compared with all other studies. It determines the lowest dose level producing the adverse impact (the Lowest Observed Adverse Effect Level, or LOAEL).

Then the agency identifies the next-lowest dose level (the No Observable Adverse Effect Level, or NOAEL). Because the toxicology studies are typically conducted on mammals other than humans, the study NOAEL is then typically divided by a hundredfold safety, or uncertainty, factor. A safety factor of 10 is thought to be warranted to account for the uncertainty in extrapolating from animals to humans, and another tenfold safety factor is applied to account for differences in the vulnerability of different population groups.

Since its passage in 1996, the FQPA mandates that the EPA apply an additional tenfold safety factor to better protect children, unless the agency determines that it has adequate data to fully account for the unique sensitivity on infants, children, and pregnant women. When this additional safety factor is applied, the new RfD value is called the chronic Population Adjusted Dose (cPAD). As a result of the FQPA, the total safety factor applied to a pesticide will typically be 1,000, although the EPA has dropped the added tenfold safety factor in many cases.

Ideally, on any given day, a person is not exposed to a substance at a level greater than his or her personal RfD. Amounts of a pesticide that lead to exposure greater than the RfD (or cPAD) can be referred to as exceeding the EPA’s “level of concern.” That puts those exposed to them at increased risk by eroding the safety factor embedded in cRfDs or cPADs.

Calculation of the Dietary Risk Index (DRI)

TO CALCULATE THE DRI for each food item addressed in this report, we divided the average residue level found in a set of samples, measured as milligrams of pesticide per kilogram of food (also called parts per million abbreviated as ppm), by what is called the chronic Reference Concentration (cRfC).

Just like average residue levels, the cRfC is measured as milligrams per kilogram body weight per day. It is the concentration

level in a single serving of a specific food that delivers the full amount of the pesticide that a child of known size could consume per day, without exceeding the EPA’s level of concern (or, put otherwise, retaining a “reasonable certainty of no harm”). A pesticide’s cRfD or cPAD drives cRfC levels; the more toxic the pesticide, the lower the cRfD or cPAD, and hence the lower the cRfC.

Based on our DRI scoring system, a value of 1 would indicate that the average residue level in a given food delivers to a consumer the

maximum allowed amount of a pesticide in a given day, in one serving of the food. DRIs that exceed 1 mean that the hundredfold, or thousandfold margin of safety is being cut into.

Our calculations for DRI are based on the diet of a 3½-year-old child weighing 16 kilograms (35.2 pounds). We chose to base our calculation on the diet of a child because

children are especially vulnerable to the dietary risks from pesticides. The index score would be lower for an adult than for a child because of an adult's greater weight (140 lb). Calculating the risk index for an adult diet would result in an index score that is 4 times lower than that of the 35 lb child, but relative risks across foods and pesticides would not change, nor would trends over time.

The Food-Supply Risk

THE DRI IS based on the mean level of all samples testing positive in a given year. Suppose PDP tests 500 samples of apples in a given year, and 100 of them tested positive for pesticide X, with a cRfC of 0.025 ppm. Also suppose the mean residue level across the 100 positive samples was 0.05 ppm.

Accordingly, the mean-of-residue DRI would be 2—well over the EPA level of concern. For consumers unlucky enough to buy only apples with residues of pesticide X, the DRI value of 2 would be accurate. But what about consumers choosing apples at random? On average, three out of four samples would have no residues, hence the DRI-mean metric could overestimate risk. In fact, the lower the frequency of positives among any set of samples, the bigger the difference between DRI-mean risk levels, and actual risk levels.

Therefore we took our DRI calculations one step further and created another metric—the Food Supply-DRI (FS-DRI), to take into

account the frequency of encountering the risk. The FS-DRI is simply the percent of samples testing positive multiplied by the DRI-mean. In the very unusual case where 100 percent of the samples tested are positive, the FS-DRI = DRI-mean. And in any single sample, compared with another single sample, the FS-DRI again equals the DRI-mean.

Because there may be aggregate risk from the combination of individual pesticides present on food items, and because the interaction of pesticides is not well understood, the FS-DRI for each pesticide-food combination can be added together to create a composite index (the aggregate FS-DRI). That sum can be used to compare the *relative “risk”* of pesticide residues on different foods. That value can also be summed across all foods eaten to determine an overall daily risk index. We recommend that individuals do not exceed a DRI of 1 based on all produce consumed in a day.

From DRI to Risk Categories

BASED ON OUR calculation of an FS-DRI for each produce item-country-production method, we placed items into one of five categories based on their FS-DRI score. These categories represent a range of FS-DRI scores that we have characterized as “very high” (one serving of an individual food with a FS-DRI of 1 or more), “high” (between one and five servings would lead to a FS-DRI score of 1 or more, which corresponds to a FS-DRI score of 0.2 to 1), “moderate” (five to 10 servings would lead to a FS-DRI score of 1 or more, which corresponds to a FS-DRI score of 0.1 to 0.2), “low” (10 to 100 servings would lead to a FS-DRI score of 1 or more, which corresponds to a FS-DRI score of 0.01 to 0.1), and “very low” (more than 100 servings would lead to a

FS-DRI score of 1 or more, which corresponds to a FS-DRI score of less than 0.01).

Risk Category	Daily Servings Needed to Exceed 1*	FS-DRI Range
Very High	1	1 or greater
High	2 - 5	0.2 - 1
Moderate	5 - 10	0.1 to 0.2
Low	10 - 100	0.01 to 0.1
Very Low	More than 100	Less than .01

* for a 35 lb child

Comparing Consumer Reports Guide with the Environmental Working Group's (EWG) Shopper's Guide to Pesticides in Produce™

THE CONSUMER REPORTS guide is significantly different from the Environmental Working Group's (EWG) Shopper's Guide to Pesticides in Produce™ (which includes the EWG's Dirty Dozen and Clean Fifteen lists). Both the Consumer Reports and EWG guides utilize the USDA PDP database, but the advice in the EWG guide is mainly driven by the number of pesticide residues on a produce item.

CR's advice and guide differ from the EWG's in the following ways:

- We take into account the number of residues per sample, the average level of

each residue, the frequency of finding it, the serving size, and the toxicity of each pesticide.

- We adhere closely to recommended EPA dietary risk assessment policies.
- We provide more complete information to consumers about differences in the risks associated with pesticides in domestically grown food, compared with imported food.
- We integrate noncancer as well as cancer risks.

Calculation of Cancer Risk in CR Guide

ALTHOUGH THE DRI score is driven by the chronic toxicity of pesticides, it does not take into account cancer risk. We did, however, perform a separate cancer risk analysis.

In order to calculate lifetime cancer risk for a given produce-country combination, we used the cancer slope factor for each pesticide (as available from the EPA) and assumed a lifetime of exposure of one serving per day for a 70-year lifetime. We multiplied that risk by the probability of the pesticide being on the item (i.e., percent of total samples testing positive). Then, for each pesticide-produce-country combination, we added up the cancer risk for all of the pesticides present in

the food to get a combined cancer risk score. Ideally, the population should be exposed to no excess cases of cancer from exposure to a substance.

Typically, from a regulatory perspective, an ideal cancer risk is considered to be no more than one excess case of cancer in a population of 1 million people exposed to the pesticide over a lifetime. For this study, we adopted a less strict public-health protection goal—a more lenient risk tolerance of no more than one excess case of cancer in 100,000 people over a lifetime. Produce-country combinations with a combined cancer risk from pesticide residues of more than one in 100,000 are denoted on our figures.

Produce DRIs: The Results

WE USED ALL available USDA PDP residue data to calculate the FS-DRI for each produce item-country-production method combination, including data from crop year 2013 that was released in December 2014. For all foods, we based our rankings on the “Most Recent Year” for which of PDP data are available.

How to Read and Understand Consumer Reports’ Residue Guide

We categorized each produce-country-production type combination into one of five categories discussed above. *The average FS-DRI score (referred from here on as DRI) for all organic produce item-country combinations fell into the “low” or “very low” category. As a result of this finding and because of the strict standards required to earn, and retain, organic certification, and well-documented and significant environmental benefits, we always recommend organic produce as the best choice.*

Fortunately, the majority of residues found in conventional produce items also fell into the “low” or “very low” categories as well. Conventional items that fall into the very low or low categories are roughly equivalent to organic produce when comparing residue related risk. For consumers who are mainly worried about exposure to residues, these conventional products represent good options, especially when available at a markedly lower price. Still, we recommend organic as the best choice.

Relatively smaller proportions of produce items fall into the “very high,” “high,” and “medium” categories. Items in the “very high” category have a DRI score of greater than 1. From a single serving of such items, children would ingest pesticides associated with a DRI risk level of 1 or higher (our “level of concern” that takes into consideration EPA reference doses). Items in the “high” risk category have a DRI score between 0.2 and 1. For those items, children would reach a DRI of 1 with two to five servings. Five servings of fruits and vegetables is the typical amount recommended by the USDA. Items in the moderate risk category have a score between 0.2 and 0.1. Children would reach a DRI of 1 after five to 10 servings.

Even for produce items that fall into one of the three highest risk categories (“very high,” “high,” or “medium”), there is often a lower risk (“low” or “very low”) conventional produce option.

Limits to Choosing Produce According Only to Pesticide Residues and Risk

ALTHOUGH THE GUIDE we created takes into account the chronic toxicity and the cancer risk of the residues, our guide does not consider many other important harmful effects of pesticides that consumers may want to consider (nor does the EWG guide). For example, our analysis does not take into account the well-known negative effects of the use of pesticides on farmworker health; beneficial insects such as honeybees and other pollinators; adverse impacts on birds, fish, and other wildlife; or the potential for low levels of pesticides to be endocrine disruptors, triggering subtle but sometimes heritable genetic mutations.

Given the pesticide-related as well as other environmental and soil health benefits of organic farming, *we always recommend certified organic food as the best choice.* Future iterations of this guide will attempt take these additional concerns into consideration as well.

Using Our Charts That Follow

The figures display the risk categories (“very low” to “very high”) into which different conventionally grown produce item-country combinations fall. The risk categories are calculated for a 16-kilogram (35.2-pound) child eating one serving of the item per day. For a 35.2-pound child, eating one serving of a given very high-risk produce-country item per day would lead them to their maximum daily limit based on our level of concern (that takes into consideration EPA reference doses). Though the absolute risk would be lower for a consumer who weighed more, the relative risk of each produce item and country combination would be unchanged.

We have created two tables, one for fruits and one for vegetables.

If the excess cancer risk for a produce item-country combination was greater than one in 100,000 exposed people, that item was marked with footnote. Even though all of the footnoted items were in the “high” category for DRI score, because of the cancer risk consumers should treat them as if they were in the “very high” category.

FRUIT

FRUIT	ORGANIC	CONVENTIONAL PRODUCE				
		VERY LOW	LOW	MEDIUM	HIGH	VERY HIGH
Peaches	👍				Chile, USA	
Tangerines	👍				Chile ¹ , South Africa ¹ , USA, (Australia, Spain)	
Plums	•		USA		Chile ¹	
Nectarines	👍			USA	Chile ¹	
Apples	•		New Zealand		USA	
Strawberries	👍				USA, (Mexico)	
Cantaloupe	•	Honduras, Mexico	Costa Rica, Guatemala		USA	
Cranberries	👍			USA		
Mangoes	•	Mexico	Guatemala	Brazil		
Pears	•		Argentina, USA			
Oranges	•		Chile, South Africa, USA			
Cherries	•		USA			
Grapefruit	•		USA			
Watermelon	•	Guatemala	Honduras, Mexico, USA			
Blueberries	•	Uruguay	Argentina, Canada, Chile, USA			
Grapes	•		Chile, Mexico, Peru, USA			
Raspberries	•		Mexico, USA			
Apple Sauce	•		Canada, USA			
Bananas	•		Columbia, Costa Rica, Ecuador, Guatemala, Honduras, Mexico			
Raisins	•		USA			
Papaya	•	Belize, Brazil, Guatemala, Jamaica, Mexico, USA	Columbia, Costa Rica, Ecuador, Guatemala, Honduras, Mexico			
Peaches, Canned	•	Greece, South Africa, USA				
Pineapples	•	Costa Rica, Ecuador, Mexico, USA				
Plums, Dried (Prunes)	•	USA				

👍 ALWAYS BUY • RECOMMENDED BUY

¹ Cancer risk greater than 1/100,000
For countries in parenthesis, average score placed country in displayed category, but sample size was small and there is less certainty.

VEGETABLES

COMMODITY	ORGANIC	CONVENTIONAL PRODUCE				
		VERY LOW	LOW	MEDIUM	HIGH	VERY HIGH
Green Beans	👍			Guatemala	Mexico	USA
Sweet Bell Peppers	👍				USA	Mexico
Hot Peppers	👍				USA	Mexico
Winter Squash	•	Guatemala	Honduras, Mexico		USA	
Cucumbers	•		Canada		Mexico, USA	
Summer Squash	•		Mexico		USA	
Snap Peas	•		Mexico, USA		Guatemala, Peru	
Tomatoes	•		Canada	USA	Mexico	
Sweet Potatoes	👍				USA	
Cherry Tomatoes	•		USA		Mexico	
Celery	•	Mexico		USA		
Carrots	👍			Canada, Mexico, USA		
Greens, Kale	•		Mexico	USA		
Potatoes	•		Canada	USA		
Asparagus	•	Mexico	USA	Peru		
Eggplant	•	Honduras	USA	Mexico		
Lettuce	•		Mexico, USA			
Spinach	•	Mexico	Mexico, USA			
Greens, Collard	•		USA			
Cauliflower	•		Mexico, USA			
Cilantro	•	USA	Mexico			
Green Onions	•	Mexico	USA			
Broccoli	•	USA	Mexico			
Mushrooms	•	Canada	USA			
Cabbage	•	Canada, Mexico, USA				
Sweet Corn	•	Mexico, USA				
Avocado	•	Chile, Mexico, Peru				
Onion	•	Peru, USA				

👍 ALWAYS BUY • RECOMMENDED BUY

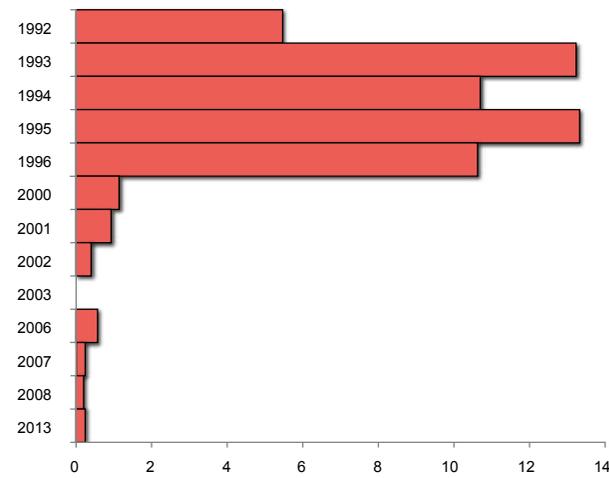
DRI Scores Have Decreased Over Time

DRI SCORES ARE a measure of both the amount of pesticide residues found on produce and their toxicity. Fortunately, the trend in total DRI score for all conventional domestic produce items over time shows that DRI scores have decreased dramatically over the past 20 years, especially for domestically grown produce. As discussed previously, much of this reduction in U.S.-grown produce can be attributed to the FQPA.

Looking at individual produce items over time provides interesting insights into the use of pesticides. Some produce items' FS-DRI scores have not changed much over time. For example, domestic green beans have remained in the very high-risk category almost every year since testing began in 1992.

Other produce DRIs have changed significantly since passage of the FQPA. Peaches are the most dramatic. Although conventional peaches from all origins are still in our high-risk category, their total DRI score has dropped significantly. Up until the testing for the year 2000, the main pesticide residue contributing to the DRI score for peaches was methyl parathion. That pesticide is one of the most potent in the organophosphate family. The EPA accepted the voluntary cancellation of the chemical for use on many crops, including peaches, in 1999. Today the

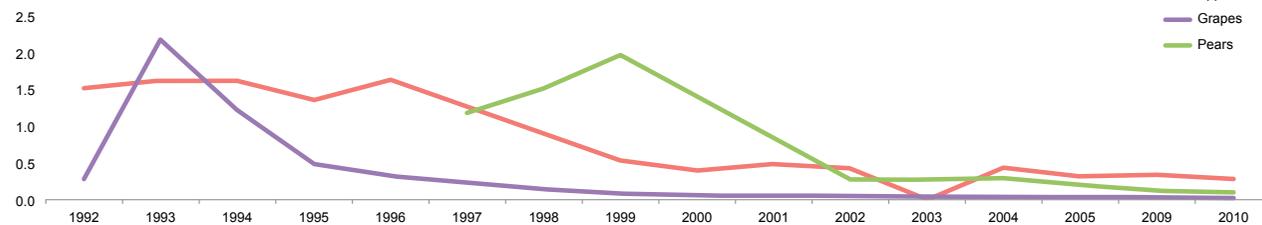
FS-DRI Domestic Peaches 1992-2013



majority of the peach DRI score is caused by residues of fludioxonil, a fungicide often used post-harvest.

Other commonly consumed crops that have also seen relatively large decreases in DRI score over the years (though not as dramatic as peaches) include pears and apples, on which methyl parathion was also commonly used. Grape DRI scores have dropped steadily from around 2.2 in 1993 to less than 0.02 in recent years.

FsDRI for Apples, Grapes, and Pears 1992-2010



High-Risk Pesticides

THE MAJORITY OF the known risk from pesticide-residue exposure is the result of only a few commonly found pesticides. There have been significant decreases in DRI scores over time, but there is still room for significant improvement, and today's relatively high-risk chemicals represent important targets for reduction.

The single most important contributor to toxic pesticide exposure and FS-DRI risk is an organophosphate chemical called methamidophos. Methamidophos residues also appear in food after applications of another organophosphate insecticide called acephate (methamidophos is a major breakdown product of acephate). Over the past 20 years the total DRI score for that chemical has remained relatively constant (the overall trend is important, and year-to-year variations may be the result of the differing mix of produce items tested each year).

Interestingly, all methamidophos uses were cancelled by the EPA in 2009 after registrants voluntarily requested an end to all food uses. Despite that, residues of methamidophos remain a concern. Acephate is another pesticide whose residues have been a relatively large contributor to the total DRI score each year over the past decade. Methamidophos

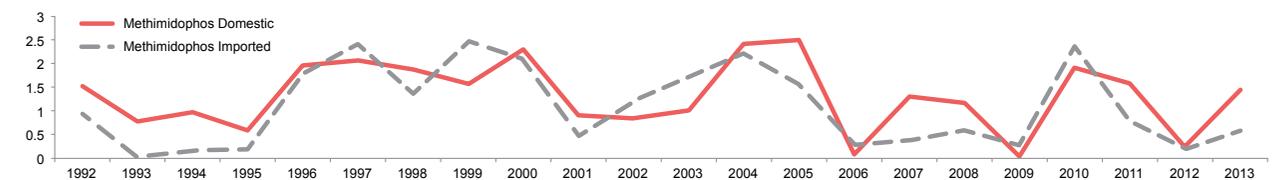
residues are a major contributor to the high DRI scores for green beans, sweet bell peppers, and hot peppers, and are also commonly found on other vegetables.

The carbamate insecticide oxamyl is another large contributor to total DRI scores during recent years. Oxamyl is used on a variety of vegetables and is one of the top contributors to the DRI for sweet bell peppers and summer squash.

Fungicides are another important class of chemicals that are significant contributors to total FS-DRI scores. Those pesticides are often applied after harvest to tree fruits, especially stone fruits such as peaches, tangerines, nectarines, and plums, as well as citrus fruits. The most common ones found are fludioxonil, iprodione, and imazalil.

Although not permitted in organic production, post-harvest fungicides leave residues that are sometimes detected on some organic produce. Those residues may be the result of cross-contamination in packing plants in which both organic and conventional produce is cleaned, packed, and shipped. Fungicide residue levels on organic produce are much lower, and present lower risk, compared with conventional produce.

Total Methamidophos DRI For All Produce 1992 to 2013



Behind the Label

EVEN WHEN A fruit or vegetable carries a low residue risk to consumers, pesticides could have been used to produce it, with potential negative impacts on farmworkers, rural residents, wildlife, pollinators, air, soil and water. But there are many labels that can help consumers make better choices when it comes to how farms and pesticide use are managed. The table that follows shows which labels are verified, which labels are backed by standards that prohibit or limit the number of toxic pesticides and require meaningful Integrated Pest Management (IPM) practices. The table also shows whether the standards behind the label allow, prohibit, or restrict the use of 18 specific pesticides of concern.

✓+ Yes, standards have a very comprehensive list of prohibited pesticides

✓ Yes, independent verification with on-site inspection

✓ Yes, standards have a comprehensive list of prohibited pesticides

✓- Verification, but not independent

✓- Some pesticides are prohibited

✗ No verification

✗ No list of prohibited pesticides, or list is minimal

LABELS GUIDE

✓ Yes

✓- Sometimes, or encouraged but not required

✗ No

LABELS	Is it verified?	Do standards prohibit toxic pesticides?	Do standards require nonchemical pest prevention & management?	Do standards require least-toxic options be used first?	Do standards require testing foods for pesticide residues?
--------	-----------------	---	--	---	--

Do standards prohibit the use of the following pesticide?

	GLYPHOSATE	ATRAZINE	2,4-D	CHLORPYRIFOS	ALDICARB	ACEPHATE	IMIDACLOPRID	ACETAMIPRID	OXAMYL	PERMETHRIN	PHOSMET	METAM SODIUM	1,3-DICHLOROPROPENE	METHYL BROMIDE	FLUDIOXONIL	IPRODIONE	IMAZALIL	THIABENDAZOLE
--	------------	----------	-------	--------------	----------	----------	--------------	-------------	--------	------------	---------	--------------	---------------------	----------------	-------------	-----------	----------	---------------

General Claims

Conventional /No label	✗	✗	✗	✗	✗	●	●	●	●	●	●	●	●	●	●	●	●	●	●
Natural	✗	✗	✗	✗	✗	●	●	●	●	●	●	●	●	●	●	●	●	●	●
Pesticide Free	✗	✗	✗	✗	✗	●	●	●	●	●	●	●	●	●	●	●	●	●	●

Environmental Sustainability Labels

USDA Organic	✓	✓+	✓	✓	✓-	●	●	●	●	●	●	●	●	●	●	●	●	●	●
Demeter Biodynamic	✓	✓+	✓	✓	✓-	●	●	●	●	●	●	●	●	●	●	●	●	●	●
Rainforest Alliance	✓	✓-	✓-	✓-	✗	●	●	●	●	●	●	●	●	●	●	●	●	●	●
Organic + Whole Foods Responsibly Grown - Good, Better, Best	✓	✓+	✓	✓	✓-	●	●	●	●	●	●	●	●	●	●	●	●	●	●
Conventional + Whole Foods Responsibly Grown - Unrated	✗	✗	✗	✗	✗	●	●	●	●	●	●	●	●	●	●	●	●	●	●
Conventional + Whole Foods Responsibly Grown - Good	✓-	✗	✓-	✗	✗	●	●	●	●	●	●	●	●	●	●	●	●	●	●
Conventional + Whole Foods Responsibly Grown - Better	✓-	✓-	✓	✓-	✗	●	●	●	●	●	●	●	●	●	●	●	●	●	●
Conventional + Whole Foods Responsibly Grown - Best	✓-	✓-	✓	✓-	✗	●	●	●	●	●	●	●	●	●	●	●	●	●	●
Certified Naturally Grown	✓-	✓+	✓	✓	✗	●	●	●	●	●	●	●	●	●	●	●	●	●	●
Eco Apple - certified by the IPM Institute	✓	✓	✓	✓	✗	●	●	●	●	●	●	●	●	●	●	●	●	●	●
Eco Stone Fruit - certified by the IPM Institute	✓	✓	✓	✓	✗	●	●	●	●	●	●	●	●	●	●	●	●	●	●

✓+ Yes, standards have a very comprehensive list of prohibited pesticides

✓ Yes, standards have a comprehensive list of prohibited pesticides

✓- Some pesticides are prohibited

✗ No list of prohibited pesticides, or list is minimal

LABELS GUIDE

✓ Yes

✓- Sometimes, or encouraged but not required

✗ No

Do standards prohibit the use of the following pesticide?

● YES ● NO ● RESTRICTED USE

LABELS	Is it verified?	Do standards prohibit toxic pesticides?	Do standards require nonchemical pest prevention & management?	Do standards require least-toxic options be used first?	Do standards require testing foods for pesticide residues?
--------	-----------------	---	--	---	--

GLYPHOSATE	ATRAZINE	2,4-D	CHLORPYRIFOS	ALDICARB	ACEPHATE	IMIDACLOPRID	ACETAMIPRID	OXAMYL	PERMETHRIN	PHOSMET	METAM SODIUM	1,3-DICHLOROPROPENE	METHYL BROMIDE	FLUDIOXONIL	IPRODIONE	IMAZALIL	THIABENDAZOLE
------------	----------	-------	--------------	----------	----------	--------------	-------------	--------	------------	---------	--------------	---------------------	----------------	-------------	-----------	----------	---------------

Food Alliance	✓	✗	✓-	✓-	✗
Stemilt Responsible Choice	✗	✗	✗	✗	✗
SCS Pesticide Residue Free	✓	✗	✗	✗	✓
Salmon Safe	✓	✗	✓	✓	✗
SCS Sustainably Grown	✓	✗	✓	✓	✓-

●	●	●	●	●	●	●	●	●	●	●	●	●	●	●	●	●	●
●	●	●	●	●	●	●	●	●	●	●	●	●	●	●	●	●	●
●	●	●	●	●	●	●	●	●	●	●	●	●	●	●	●	●	●
●	●	●	●	●	●	●	●	●	●	●	●	●	●	●	●	●	●
●	●	●	●	●	●	●	●	●	●	●	●	●	●	●	●	●	●

Non GMO Labels

Non-GMO Project Verified	✓	✗	✗	✗	✗
--------------------------	---	---	---	---	---

●	●	●	●	●	●	●	●	●	●	●	●	●	●	●	●	●	●
---	---	---	---	---	---	---	---	---	---	---	---	---	---	---	---	---	---

Social Responsibility Labels

Fair Trade Certified	✓	✗	✓-	✓-	✗
Fairtrade International	✓	✗	✓-	✗	✗
Fair for Life	✓	✓-	✓-	✓-	✗
Food Justice Certified	✓	✓	✓	✓	✗
Fair Food Program	✓	✗	✗	✗	✗
Responsibly Grown. Farmworker Assured	✓	✗	✓	✓	✓-
Whole Foods - Whole Trade Guarantee	✓	✗	✓-	✗	✗

●	●	●	●	●	●	●	●	●	●	●	●	●	●	●	●	●	●
●	●	●	●	●	●	●	●	●	●	●	●	●	●	●	●	●	●
●	●	●	●	●	●	●	●	●	●	●	●	●	●	●	●	●	●
●	●	●	●	●	●	●	●	●	●	●	●	●	●	●	●	●	●
●	●	●	●	●	●	●	●	●	●	●	●	●	●	●	●	●	●
●	●	●	●	●	●	●	●	●	●	●	●	●	●	●	●	●	●
●	●	●	●	●	●	●	●	●	●	●	●	●	●	●	●	●	●



A note about pest control in organic production. Some pesticides are derived from plants, bacteria, or other natural sources. Those pesticides are allowed in organic production, unless they pose potential harm to human health or the environment.²⁴⁶ For example, even though arsenic and lead are “natural” substances, pesticides containing those substances are prohibited.²⁴⁷ Natural pesticides that are permitted, such as those derived from the bacterium *bacillus thuringiensis* and the botanical extract pyrethrum, can be used only as a last resort after other methods of pest control have failed.²⁴⁸

Integrated Pest Management. Integrated Pest Management (IPM) is an approach to managing pests that uses a variety of methods, including cultural and biological methods. However, the use of chemical pesticides is also a component of IPM. IPM’s acceptance of chemical pesticides means it is significantly different from organic management, which prohibits nearly every chemical pesticide. IPM is a step in the right direction, though. It aims to solve pest problems while minimizing risks to people and the environment, using chemical pesticides only when needed. By scouting for pests, monitoring and keeping records of pests, growers can choose to use pesticides only when pest numbers are determined to exceed acceptable levels. IPM also aims to use pesticides with lower toxicity and risk before turning to pesticides with higher known toxicity. Unfortunately, there is no one way to practice IPM and some producers are more rigorous in its application than others.

Stretching Your Produce and Organic Dollar

ORGANIC FOOD — produced on organic farms without the use of most pesticides — is better for your health and for the environment. Organic produce also generally costs more than conventional, so here are some tips on how to fit organic in your budget.

Buy whole foods and process at home. Whole and unprocessed organic foods are often less expensive than their processed non-organic counterparts. Carrots are a perfect example: “Baby carrot” is just a fancy name for a carrot that has been peeled and cut into pieces for you. But if you don’t mind doing the peeling and cutting yourself, organic whole carrots will often cost less than the nonorganic “baby” variety. The same is often true for organic whole heads of lettuce vs. the bagged nonorganic kind, whole organic apples vs. sliced nonorganic, etc.

Buy in bulk. It’s often thought of as a cost-saving measure when buying grains, dried beans, nuts, and other grocery items, but buying in bulk can also save you money on fresh or dried fruits and vegetables. Look for dried fruit in the bulk section of your store. Buying fresh produce in bulk can save money as well. Many stores offer organic apples, oranges, carrots, avocados, and other organic produce in bags, which can cost less on a per-pound basis than buying loose produce.

Buy in season. Fruits and vegetables are much less expensive when you buy them in season. Stock up during the summer and fall months, when produce is abundant and prices are lower, and preserve them for the winter and early spring months. Canning, freezing, and drying are good options.

- **Find a farmers market.** They’re a great way to buy in season, directly from local farmers.
- **Or join an organic CSA.** CSA stands for “community supported agriculture.” When you join a CSA farm, you will receive a weekly share of the harvest, and you’re likely to spend much less money on fruits and vegetables than if you bought those items at the store. You’ll be supporting local farmers, and you’ll probably end up with more vegetables

and fruits than you know what to do with. Consider preserving them for the winter months by canning, drying, or freezing.

Buy frozen. If you didn’t have the chance to freeze produce at home during the summer, look for frozen options in the store. Many organic fruits and vegetables are available in the freezer section of most stores year-round and can cost less than the fresh nonorganic options. Because they are frozen immediately after harvest, nutrient levels in frozen organic fruits and vegetables are comparable to, or even better than, those of fresh produce that has been shipped many miles and arrives less than fresh at the store.

Or buy dried. In the winter months, when certain kinds of organic fruits and vegetables are either unavailable or too expensive, look for their dried versions instead. An additional benefit of buying organic dried fruit is that sulfite preservatives—often used in conventional dried fruits—are prohibited in organic foods.

Replace processed snack foods with organic fruits and vegetables. USDA researchers have found that some healthy foods, including fresh produce, can often cost less than unhealthy foods such as sweet and salty snacks.²⁴⁹ To fit organic fruits and vegetables in your food budget, consider cutting out unhealthy sweet and salty snack foods.



Cleaning Produce

WHEN ORGANIC OPTIONS are not available or affordable, consumers can take certain actions to lower their exposure to pesticide residues.

- Wash fruits and vegetables thoroughly under running water.²⁵⁰ Make sure you wash for 30 seconds to 1 minute, and gently rub the produce to dislodge the residues.²⁵¹
- Produce with firm edible skin can be scrubbed with a clean produce brush.²⁵² Wash produce brushes regularly with hot soapy water or in a dishwasher and dry completely. And consider not peeling since nutrients are in the peel too.
- Wash produce before removing inedible peels to prevent pesticide residue contamination on your hands and on the edible parts.²⁵³
- Remove the outer leaves of heads of lettuce or cabbages, because the outer leaves are likely to have higher levels of pesticide residues.^{254, 255}
- If you use citrus peel (such as for zesting), consider buying organic.
- Wash organic produce as well. Organic regulations prohibit the use of synthetic pesticides, but synthetic pesticides used on neighboring conventional farms could contaminate organic crops. Washing eliminates more than just pesticide residues; it also removes dirt and potentially harmful microorganisms that might contaminate.



Conclusion

THE RELIANCE ON toxic pesticides to produce food is neither safe nor sustainable. Since living organisms adapt, toxic pesticides eventually lose their effectiveness. This leads chemical companies to develop different pesticides to attack resistant weeds and pests — pesticides that are perhaps more toxic, or perhaps toxic simply in a different way. But while new pesticides can control pests for a while, it is never a long-term solution. Rather, pesticides — the chemicals that are supposed to be a solution — turn into problems themselves, as their toxic effects on non-target populations, including humans, become apparent.

Given the growing body of scientific evidence pointing to harm, we believe that the costs are too high and do not justify the short-term benefits of controlling pests with toxic chemicals.

We urge people to eat several servings of fruits and vegetables daily. Whenever available and affordable, we urge people to choose the most safe and sustainably farmed options, using our DRI/residues guide and labels guide. By using our guides and making informed purchases, consumers can protect themselves and support farmers who are reducing their pesticide use, which is better for human health and the environment. 🌱

Consumer Reports' Food Safety and Sustainability Center's Pesticide Policy Recommendations

Recommendations for the Environmental Protection Agency (EPA)

EPA should ban or take immediate action on the riskiest pesticides.

We agree with the canceling of methamidophos in 2009. However, methamidophos is also the breakdown product of acephate, and combined, these two chemicals make up the largest contribution to dietary risk. Acephate, along with other major pesticide contributors to risk such as iprodione, fludioxonil, imazalil, and oxamyl should be fully banned. Other pesticides we would like discontinued include methyl bromide, chlorpyrifos and other organophosphates.

EPA should take immediate action on neonicotinoids.

Many crops, especially fruits and vegetables, face an uncertain future with the severe decline of pollinators. While many factors are at play, mounting evidence implicates neonicotinoid pesticides in their decline. Thus far, the EPA has delayed meaningful action in the name of more research. We believe there is enough information to take action now. Research should continue, but in the meantime, the EPA should proceed immediately with cancellation or reclassification proceedings, utilizing the “imminent hazard” provision, and suspend the use of these pesticides while proceedings take place.

EPA should complete the delisting of arsenical pesticides.

Pesticides containing organic forms of arsenic are still permitted for use on golf courses, rights of way, sod farms and cotton crops. The use of these pesticides contributes to the contamination of our environment with this dangerous heavy metal. The EPA should deregister the use of these pesticides immediately.

EPA should improve the science behind tolerance limits.

Tolerance levels should incorporate the best available science, including potential toxicological and health endpoints that are not included today. Those include stricter tests for more immunologic and neurobehavioral endpoints. Further, the EPA's program for incorporating endocrine-disrupting effects into tolerances and pesticide approvals should be fully implemented as the FQPA directed in 1996. Epigenetic effects should also be incorporated when it becomes feasible.

EPA should rein in emergency exemptions and conditional registrations.

EPA “allows the use of a pesticide for an unregistered use for a limited time if EPA determines that emergency conditions exist.” The EPA can currently grant exemptions with or without public comment or granting public access to supporting data. Emergency exemptions should only be granted for a finite period of time and after three years, should not be allowed to continue, especially when there are alternatives, including integrated pest management, crop diversification, and organic production practices.

Conditional registration means that the use of the pesticide can be allowed while the EPA waits for additional data to be submitted by the registrant. This raises concerns about using materials where there is insufficient safety information. Many pesticides were first brought on the market through this pathway, including the neonicotinoid pesticide, imidacloprid. While the EPA has already started to close this loophole, there is still room for continued improvement.

EPA should require public access to all ingredients in pesticides and easy access to current registration status.

While active ingredients in pesticides must be disclosed, this requirement does not apply to “inert” ingredients. Only full disclosure of all ingredients on pesticide labels will enable the public and independent researchers to begin evaluating the full range of synergistic effects. The EPA should issue new rules concerning disclosure of inert ingredients and should do so immediately. In addition, the EPA should provide an easy-to-search database of current pesticide registration status on its website.

Recommendations for the US Department of Agriculture (USDA):

USDA should expand pesticide residue testing in the PDP.

The USDA Pesticide Data Program (PDP) is an important tool for informing the public about pesticide residues on produce and is used by the EPA for understanding pesticide exposure and setting tolerances. Currently, the USDA does not test for some widely used pesticides such as glyphosate and should.

USDA should protect and promote organic standards and meaningful integrated pest management (IPM).

Conservation incentives should promote organic production methods and meaningful IPM—where chemical pest controls are utilized as last resorts and require review and approval. The USDA should ensure the highest integrity in organics including minimizing the approval of exempted materials, such as pesticides.

Recommendations for the Food and Drug Administration (FDA):

FDA should expand and improve pesticide residue testing and enforcement.

The FDA is charged with enforcing pesticide tolerance levels on fruits and vegetables. According to a 2014 report by the U.S. Government Accountability Office (GAO), the FDA does not regularly test for some of the most common pesticides such as glyphosate, 2,4-D and methyl bromide. In addition, the FDA tests only a very small percentage of both imported and domestic fruits and vegetables. The FDA should begin regularly testing for those chemicals identified by the GAO, and should increase its sampling of both domestic and imported produce.

**This project was made possible in part
by the generosity
of the following donors to Consumer Reports'
Food Safety and Sustainability Center:**

Diane Archer & Stephen Presser
Joe and Barbara Ellis
Jessica Guff
Christopher Rothko & Lori Cohen
Ben Serebin

To contribute to the Food Safety & Sustainability Center
and support projects like these, please visit
www.greenerchoices.org/getengaged

References

- ¹Oyebode, O., Gordon-Dseagu, V., Walker, A. and Mindell, J.S. (2014) Fruit and vegetable consumption and all-cause, cancer and CVD mortality: analysis of Health Survey for England data. *Journal of Epidemiology and Community Health*. Published online first: March 31, 2014. doi:10.1136/jech-2013-203500
- ²Environmental Protection Agency, Office of Pesticide Programs. (2011). Pesticide industry sales and usage, 2006-2007 market estimates. Retrieved from <http://www.epa.gov/opp00001/pestsales/>. Page 12.
- ³Consumer Reports National Research Center. (2014) Food labels survey, 2014 nationally-representative phone survey. Retrieved from <http://www.greenerchoices.org/pdf/ConsumerReportsFoodLabelingSurveyJune2014.pdf>. Last accessed on March 4, 2015.
- ⁴The Organic Foods Production Act of 1990. 7 USC 6504(1)
- ⁵Lu, C., Toepel, K., Irish, R., Fenske, R.A., Barr, D.B. and R. Bravo. (2006). Organic diets significantly lower children's dietary exposure to organophosphorus pesticides. *Environmental Health Perspectives* 114(2): 260-263.
- Oates, L., Cohen, M., Braun, L., Schembri, A. and R. Taskova. (2014). Reduction in urinary organophosphate pesticide metabolites in adults after a week-long organic diet. *Environmental Research* 132: 105-111.
- ⁷Curl, C.L., Beresford, S.A.A., Fenske, R.A., Fitzpatrick, A.L., Chensheng, L., Nettleton, J.A. and Kaufman, J.D. (2015). Estimating Pesticide Exposure from Dietary Intake and Organic Food Choices: The Multi-Ethnic Study of Atherosclerosis (MESA). *Environmental Health Perspectives*. Retrieved from <http://ehp.niehs.nih.gov/1408197/>. doi:10.1289/ehp.1408197.
- ⁸Palumbi, S.R. (2001). Humans as the world's greatest evolutionary force. *Science* 293(5536): 1786-1790.
- ⁹Committee on the Future Role of Pesticides in US Agriculture, Board on Agriculture and Natural Resources, Board on Environmental Studies and Toxicology, National Research Council (2000). *The future role of pesticides in US agriculture*. Washington, D.C.: National Academy Press. Page 6.
- ¹⁰Committee on the Future Role of Pesticides in US Agriculture, Board on Agriculture and Natural Resources, Board on Environmental Studies and Toxicology, National Research Council (2000). *The future role of pesticides in US agriculture*. Washington, D.C.: National Academy Press. Pages 56-57.
- ¹¹Vandenberg, L.N., Colborn, T., Hayes, T.B., Heindel, J.J., Jacobs Jr., D.R., Lee, D-H., Shioda, T., Soto, A.M., vom Saal, F.S., Welshons, W.V., Zoeller, R.T. and Myers, J.P. (2012). Hormones and endocrine-disrupting chemicals: low-dose effects and nonmonotonic dose responses. *Endocrine Reviews* 33(3): 378-455.
- ¹²Grandjean, P., and Landrigan, P. J. (2014). Neurobehavioural effects of developmental toxicity. *The Lancet Neurology* 13(3): 330-338.
- ¹³Consumer Reports (November 2012). Arsenic in your food: our findings show a real need for federal standards for this toxin. Retrieved from <http://consumerreports.org/cro/magazine/2012/11/arsenic-in-your-food/index.htm>.
- ¹⁴Peryea, F.J. and Kammereck, R. (1997). Phosphate-enhanced movement of arsenic out of lead arsenate-contaminated topsoil and through uncontaminated subsoil. *Water Air and Soil Pollution* 93(1): 243-254.
- ¹⁵Hong, Y. S., Song, K. H., and Chung, J. Y. (2014). Health Effects of Chronic Arsenic Exposure. *Journal of Preventive Medicine and Public Health* 47(5): 245.
- ¹⁶Consumer Reports (November 2012). Arsenic in your food: our findings show a real need for federal standards for this toxin. Retrieved from <http://consumerreports.org/cro/magazine/2012/11/arsenic-in-your-food/index.htm>.
- ¹⁷Vogt, R., Bennett, D., Cassady, D., Frost, J., Ritz, B. and Hertz-Picciotto, I. (2012). Cancer and non-cancer health effects from food contaminant exposures for children and adults in California: a risk assessment. *Environmental Health* 11: 83-97.
- ¹⁸Vogt, R., Bennett, D., Cassady, D., Frost, J., Ritz, B. and Hertz-Picciotto, I. (2012). Cancer and non-cancer health effects from food contaminant exposures for children and adults in California: a risk assessment. *Environmental Health* 11: 83-97.
- ¹⁹Bienkowski, B. (2014, July 28). Songbirds dying from DDT in Michigan yards; Superfund site blamed. *Environmental Health News*. Retrieved from <http://www.environmentalhealthnews.org/ehs/news/2014/jul/dead-robins>.
- ²⁰Benbrook, C. (2011). The Organic Center's "Dietary Risk Index," tracking relative pesticide risks in foods and beverages. The Organic Center. Retrieved from http://organic-center.org/reportfiles/DRIfinal_09-10-2011.pdf.
- ²¹Sanzani, S. M., Schena, L. and Ippolito, A. (2014). Effectiveness of phenolic compounds against citrus green mould. *Molecules* 19(8): 12500-12508.
- ²²Environmental Protection Agency (EPA). (1996, October) Chlorpropham: RED Facts. EPA-738-F-96—O23.
- ²³Paulsrud, B.E., et al. (2001). Seed Treatment: Oregon Pesticide Applicator Training Manual. p. 1. Retrieved from <http://www.oregon.gov/oda/shared/documents/publications/pesticidesparc/pesticideapplicatorseedtreatmenttrainingmanual.pdf>.
- ²⁴U.S. Environmental Protection Agency. (2014, September). What are Soil Fumigants? [Webpage] Available online at <http://www2.epa.gov/soil-fumigants/what-are-soil-fumigants>.
- ²⁵Environmental Protection Agency, Office of Pesticide Programs. (2011). Pesticide industry sales and usage, 2006-2007 market estimates. Retrieved from <http://www.epa.gov/opp00001/pestsales/>. Page 12.
- ²⁶Majewski, M.S., Coupe, R.H., Foreman, W.T. and P.D. Capel. (2014). Pesticides in Mississippi air and rain: a comparison between 1995 and 2007. *Environmental Toxicology and Chemistry* 33(6): 1283-93.
- ²⁷Ward, M.H., Lubin, J., Giglierano, J., Colt, J.S., Wolter, C., Bekiroglu, N., Camann, D., Hartge, P. and J.R. Nuckols. (2006). Proximity to crops and residential exposure to agricultural herbicides in Iowa. *Environmental Health Perspectives* 114(6): 893-897.
- ²⁸Hladik, M.L., Kolpin, D.W. and K.M. Kuivila. (2014). Widespread occurrence of neonicotinoid insecticides in streams in a high corn and soybean producing region, USA. *Environmental Pollution* 193C: 189-196.
- ²⁹Chang, F.C., Simcik, M.F. and P.D. Capel. (2011). Occurrence and fate of the herbicide glyphosate and its degradate amino methylphosphonic acid in the atmosphere. *Environmental Toxicology and Chemistry* 30(3): 548-55.
- ³⁰Bouchard, M. F., Chevrier, J., Harley, K. G., Kogut, K., Vedar, M., Calderon, N., Trujillo, C., Johnson, C., Bradman, A., Barr, D.B. and Eskenazi, B. (2011). Prenatal Exposure to Organophosphate Pesticides and IQ in 7-Year-Old Children. *Environmental Health Perspectives*, 119(8), 1189-1195. doi:10.1289/ehp.1003185.
- ³¹Curl, C.L., Beresford, S.A.A., Fenske, R.A., Fitzpatrick, A.L., Chensheng, L., Nettleton, J.A. and Kaufman, J.D. (2015). Estimating

Pesticide Exposure from Dietary Intake and Organic Food Choices: The Multi-Ethnic Study of Atherosclerosis (MESA). *Environmental Health Perspectives*. Retrieved from <http://ehp.niehs.nih.gov/1408197/>. doi:10.1289/ehp.1408197.

32 Roberts, J.R., Karr, C.J.; Council on Environmental Health. (2012). Technical Report: Pesticide exposure in children. *Pediatrics* 130(6): e1765-e1788.

33 U.S. Department of Agriculture. (2014, February). Pesticide Data Program. Annual Summary, Calendar Year 2012 [Data file]. Retrieved from www.ams.usda.gov/pdp.

34 Gilliom, R.J., Barbash, J.E., Crawford, C.G., Hamilton, P.A., Martin, J.D., Nakagaki, N., Nowell, L.H., Scott, J.C., Stackelberg, P.E., Thelin, G.P. and Wolock, D.M. (2006). Pesticides in the nation's streams and ground water, 1992-2001. U.S. Department of the Interior, U.S. Geological Survey. Retrieved from <http://pubs.usgs.gov/circ/2005/1291/pdf/circ1291.pdf>.

35 Smalling, K.L., Reeves, R., Muths, E., Vandever, M., Battaglin, W.A., Hladik, M.L. and Pierce, C.L. (2015) Pesticide concentrations in frog tissue and wetland habitats in a landscape dominated by agriculture. *Science of the Total Environment* 502: 80-90.

36 Roberts, J.R., Karr, C.J.; Council on Environmental Health. (2012). Technical Report: Pesticide exposure in children. *Pediatrics* 130(6): e1765-e1788.

37 Leffall, L.D., Jr., and Kripke, M.L. (2010). 2008-2009 Annual Report: President's Cancer Panel: Reducing Environmental Cancer Risk: What we can do now. U.S. Department of Health and Human Services, National Institutes of Health, National Cancer Institute. Retrieved from http://deainfo.nci.nih.gov/advisory/pcp/annualReports/pcp08-09rpt/PCP_Report_08-09_508.pdf.

38 Roberts, J.R., Karr, C.J.; Council on Environmental Health. (2012). Technical Report: Pesticide exposure in children. *Pediatrics* 130(6): e1765-e1788.

39 Roberts, J.R., Karr, C.J.; Council on Environmental Health. (2012). Technical Report: Pesticide exposure in children. *Pediatrics* 130(6): e1765-e1788.

40 Leffall, L.D., Jr., and Kripke, M.L. (2010). 2008-2009 Annual Report: President's Cancer Panel: Reducing Environmental Cancer Risk: What we can do now. U.S. Department of Health and Human Services, National Institutes of Health, National Cancer Institute. Retrieved from http://deainfo.nci.nih.gov/advisory/pcp/annualReports/pcp08-09rpt/PCP_Report_08-09_508.pdf.

41 Leffall, L.D., Jr., and Kripke, M.L. (2010). 2008-2009 Annual Report: President's Cancer Panel: Reducing Environmental Cancer Risk: What we can do now. U.S. Department of Health and Human Services, National Institutes of Health, National Cancer Institute. Retrieved from http://deainfo.nci.nih.gov/advisory/pcp/annualReports/pcp08-09rpt/PCP_Report_08-09_508.pdf.

42 Leffall, L.D., Jr., and Kripke, M.L. (2010). 2008-2009 Annual Report: President's Cancer Panel: Reducing Environmental Cancer Risk: What we can do now. U.S. Department of Health and Human Services, National Institutes of Health, National Cancer Institute. Retrieved from http://deainfo.nci.nih.gov/advisory/pcp/annualReports/pcp08-09rpt/PCP_Report_08-09_508.pdf. Last accessed on February 11, 2015. Page 45.

43 U.S. Environmental Protection Agency. (2014, October 2). Office of Pesticide Programs. Chemicals Evaluated for Carcinogenic Potential. Retrieved from http://npic.orst.edu/chemicals_evaluated.pdf. Last accessed on February 27, 2015.

44 Environmental Protection Agency. (2014) 40 CFR Part 170. Pesticides; Agricultural Worker Protection Standards Revisions. Federal Register 79(53): 15444-15531. Page 15447.

45 Environmental Protection Agency. (2015, January 30). Laws and Regulations [Webpage]. Retrieved from <http://www.epa.gov/pesticides/regulating/laws.htm>.

46 Environmental Protection Agency. (2015, January 30). Laws and Regulations [Webpage]. Retrieved from <http://www.epa.gov/pesticides/regulating/laws.htm>.

47 21 USC 346a

48 Environmental Protection Agency. (2014) 40 CFR Part 170. Pesticides; Agricultural Worker Protection Standards Revisions. Federal Register 79(53): 15444-15531. Page 15451.

49 Environmental Protection Agency. Office of Pesticide Programs. (2006). Reregistration Eligibility Decision for Chlorpyrifos. Page 96.

50 Environmental Protection Agency. Office of Pesticide Programs. (2006). Reregistration Eligibility Decision for Chlorpyrifos. Page 96.

51 Environmental Protection Agency (n.d.). About EPA: Our mission and what we do [Webpage]. Retrieved from <http://www2.epa.gov/aboutepa/our-mission-and-what-we-do>. Last accessed on February 13, 2015.

52 Environmental Protection Agency. (2014) 40 CFR Part 170. Pesticides; Agricultural Worker Protection Standards Revisions. Federal Register 79(53): 15444-15531. Page 15451.

53 U.S. Environmental Protection Agency. (2012, May 9) Setting tolerances for pesticide residues in foods [Webpage] Retrieved from <http://www.epa.gov/opp00001/factsheets/stprf.htm>. Last accessed on March 4, 2015.

54 U.S. Environmental Protection Agency. (2007, April 5) Assessing health risks from pesticides [Webpage] Retrieved from <http://www.epa.gov/pesticides/factsheets/riskassess.htm>. Last accessed on March 4, 2015.

55 U.S. Environmental Protection Agency. (2015, February). Pesticide Registration: Data Requirements. [Webpage]. Retrieved from <http://www2.epa.gov/pesticide-registration/data-requirements>. Last accessed on February 27, 2015.

56 U.S. Environmental Protection Agency. (2015, February). Pesticide Registration: Data Requirements. [Webpage]. Retrieved from <http://www2.epa.gov/pesticide-registration/data-requirements>. Last accessed on February 27, 2015.

57 Bergman, A., Heindel, J.J., Jobling, S., Kidd, S.A. and Zoeller, R.T. (2012) State of the science of endocrine disrupting chemicals - 2012. World Health Organization and United Nations Environment Programme. Retrieved from <http://www.who.int/ceh/publications/endocrine/en/>. Page ix.

58 National Pesticide Information Center. Oregon State University and Environmental Protection Agency. (1999). DDT - General Fact Sheet. Retrieved from <http://npic.orst.edu/factsheets/ddtgen.pdf>.

59 Colborn, T. and L.E. Carroll. (2007). Scholarly Review - Pesticides, Sexual Development, Reproduction, and Fertility: Current Perspective and Future Direction. *Human and Ecological Risk Assessment* 13: 1078-1110. Page 1104.

60 andenberg, L.N., Colborn, T., Hayes, T.B., Heindel, J.J., Jacobs Jr., D.R., Lee, D-H., Shioda, T., Soto, A.M., vom Saal, F.S., Welshons, W.V., Zoeller, R.T. and J.P. Myers. (2012). Hormones and endocrine-disrupting chemicals: low-dose effects and nonmonotonic dose responses. *Endocrine Reviews* 33(3): 378-455.

61 National Institute of Environmental Health Sciences. U.S. Department of Health and Human Services. (2010, May). Fact Sheet: Endocrine Disruptors [Webpage]. Retrieved from https://www.niehs.nih.gov/health/materials/endocrine_disruptors_508.pdf.

62 Monosson, E. (2005). Chemical mixtures: considering the evolution of toxicology and chemical assessment. *Environmental Health Perspectives* 113(4): 383-390.

63 Cole, T.B., Jansen, K., Park, S., Li, W-F, Furlong, C.E. and Costa, L.G. (2010). The toxicity of mixtures of specific organophosphorus compounds is modulated by Paraoxonase 1 status. *Advances in Experimental Medicine and Biology* 660: 47-60.

64 Mesnage, R., Defarge, N., de Vendomois, J.S. and Seralini, G-E. (2014). Major pesticides are more toxic to human cells than their declared active principles. *BioMed Research International* 2014. Retrieved from <http://dx.doi.org/10.1155/2014/179691>.

65 Monosson, E. (2005). Chemical mixtures: considering the evolution of toxicology and chemical assessment. *Environmental Health Perspectives* 113(4): 383-390. Page 384.

66 Monosson, E. (2005). Chemical mixtures: considering the evolution of toxicology and chemical assessment. *Environmental Health Perspectives* 113(4): 383-390. Page 383.

67 Mesnage, R., Defarge, N., de Vendomois, J.S. and Seralini, G-E. (2014). Major pesticides are more toxic to human cells than their declared active principles. *BioMed Research International* 2014. Retrieved from <http://dx.doi.org/10.1155/2014/179691>.

68 Mesnage, R., Defarge, N., de Vendomois, J.S. and Seralini, G-E. (2014). Major pesticides are more toxic to human cells than their declared active principles. *BioMed Research International* 2014. Retrieved from <http://dx.doi.org/10.1155/2014/179691>.

69 U.S. Environmental Protection Agency. (2012). Pesticides: Regulating Pesticides: Rulemaking Underway Related to Disclosure of All Pesticide Ingredients. [Webpage.] Available at <http://www.epa.gov/opp001/inerts/inertdisclosure.html>; but see Rodriguez, J.C. (2014, October). EPA Wants 72 Inert Chemicals Out As Pesticide Ingredients. *Law 360*. Retrieved from <http://www.law360.com/articles/589944/epa-wants-72-inert-chemicals-out-as-pesticide-ingredients>.

70 Environmental Protection Agency. (2008). Imidacloprid Summary Document Registration Review: Initial Docket December 2008. Docket number EPA-HQ-OPP-2008-0844. Retrieved from www.regulations.gov. Page 5 of 13.

71 U.S. Department of Agriculture (2014, February). Pesticide Data Program. Annual Summary, Calendar Year 2012 [Data file]. Retrieved from www.ams.usda.gov/pdp. Appendix I. Page 2 of 4.

72 Environmental Protection Agency. (2012, May 9). Types of Pesticides [Webpage]. Retrieved from <http://www.epa.gov/pesticides/about/types.htm>.

73 Environmental Protection Agency (EPA). (2011, June 22) Requests to voluntarily cancel certain pesticide registrations: Dicofol. EPA-HQ-OPP-2005-0220-0017.

74 U.S. Fish and Wildlife Service. (2014, January 15). DDT and other organochlorine insecticides. Retrieved from <http://www.fws.gov/contaminants/info/ddt.html>.

75 Environmental Protection Agency. (2012, May 9). Types of Pesticides [Webpage]. Retrieved from <http://www.epa.gov/pesticides/about/types.htm>.

76 Fukuto, T.R. (1990) Mechanism of action of organophosphorus and carbamate insecticides. *Environmental Health Perspectives* 87: 245-254.

77 Environmental Protection Agency. (2012, May 9). Types of Pesticides [Webpage]. Retrieved from <http://www.epa.gov/pesticides/about/types.htm>.

78 Environmental Protection Agency. (2012, May 9). Types of Pesticides [Webpage]. Retrieved from <http://www.epa.gov/pesticides/about/types.htm>.

79 Henry, M., Beguin, M., Requier, F., Rollin, O., Odoux, J. F., Aupinel, P., ... and Decourtye, A. (2012). A common pesticide decreases foraging success and survival in honey bees. *Science*, 336(6079), 348-350.

80 Committee on Pesticides in the Diets of Infants and Children, Board on Agriculture and Board on Environmental Studies and Toxicology, Commission on Life Sciences, National Research Council (1993). *Pesticides in the diets of infants and children*. Washington, D.C.: National Academy Press. Page 3.

81 Roberts, J.R., Karr, C.J.; Council on Environmental Health (2012). Technical Report: Pesticide exposure in children. *Pediatrics* 130(6): e1765-e1788. Page e1781.

82 Committee on Pesticides in the Diets of Infants and Children, Board on Agriculture and Board on Environmental Studies and Toxicology, Commission on Life Sciences, National Research Council (1993). *Pesticides in the diets of infants and children*. Washington, D.C.: National Academy Press.

83 Costa, L.G., Giordano, G., Cole, T.B., Marsillach, J and Furlong, C.E. (2013). Paraoxonase 1 (PON1) as a genetic determinant of susceptibility to organophosphate toxicity. *Toxicology* 10(307): 115-122.

84 Furlong, C.E., Holland, N., Richter, R.J., Bradman, A., Ho, A. and Eskenazi, B. (2006). PON1 status of farmworker mothers and children as a predictor of organophosphate sensitivity. *Pharmacogenetics and Genomics* 16(3): 183-190.

85 Eskenazi, B., Marks, A.R., Bradman, A., Harley, K., Barr, D.B., Johnson, C., Morga, N., and Jewell, N.P. (2007). Organophosphate pesticide exposure and neurodevelopment in young Mexican-American children. *Environmental Health Perspectives* 115(5): 792-798. Page 792.

86 Cole, T.B., Jansen, K., Park, S., Li, W-F, Furlong, C.E. and Costa, L.G. (2010). The toxicity of mixtures of specific organophosphorus compounds is modulated by Paraoxonase 1 status. *Advances in Experimental Medicine and Biology* 660: 47-60.

87 Committee on Pesticides in the Diets of Infants and Children, Board on Agriculture and Board on Environmental Studies and Toxicology, Commission on Life Sciences, National Research Council (1993). *Pesticides in the diets of infants and children*. Washington, D.C.: National Academy Press. Pages 65, 66 and 68.

88 Environmental Protection Agency, Office of Pesticide Programs (2011). Pesticide industry sales and usage, 2006-2007 market estimates. Retrieved from <http://www.epa.gov/opp00001/pestsales/>. Page 14.

89 Environmental Protection Agency. Office of Pesticide Programs (2006). Reregistration Eligibility Decision for Chlorpyrifos.

90 Eskenazi, B., Bradman, A. and Castorina, R. (1999). Exposures of children to organophosphate pesticides and their potential adverse health effects. *Environmental Health Perspectives* 107(suppl. 3): 409-419.

91 Reigart, J.R. and Roberts, J.R. (1999). Recognition and management of pesticide poisonings. Environmental Protection Agency, Washington, D.C. Retrieved from <http://www.epa.gov/oppfead1/safety/healthcare/handbook/handbook.htm>. Page 34.

92 National Institute of Environmental Health Sciences. Organophosphates [Webpage]. Retrieved from http://tools.niehs.nih.gov/srp/research/research4_s3_s5.cfm.

⁹³ National Institute of Environmental Health Sciences. Organophosphates [Webpage]. Retrieved from http://tools.niehs.nih.gov/srp/research/research4_s3_s5.cfm.

⁹⁴ Bouchard, M. F., Chevrier, J., Harley, K. G., Kogut, K., Vedar, M., Calderon, N., Trujillo, C., Johnson, C., Bradman, A., Barr, D.B. and Eskenazi, B. (2011). Prenatal Exposure to Organophosphate Pesticides and IQ in 7-Year-Old Children. *Environmental Health Perspectives*, 119(8), 1189–1195. doi:10.1289/ehp.1003185.

⁹⁵ Kimura-Kuroda, J., Komuta, Y., Kuroda, Y., Hayashi, M. and Kawano, H. (2012). Nicotine-like effects of the neonicotinoid insecticides acetamiprid and imidacloprid on cerebellar neurons from neonatal rats. *PLoS ONE* 7(2): e32432. doi:10.1371/journal.pone.0032432.

⁹⁶ Kimura-Kuroda, J., Komuta, Y., Kuroda, Y., Hayashi, M. and Kawano, H. (2012). Nicotine-like effects of the neonicotinoid insecticides acetamiprid and imidacloprid on cerebellar neurons from neonatal rats. *PLoS ONE* 7(2): e32432. doi:10.1371/journal.pone.0032432.

⁹⁷ EFSA PPR Panel (EFSA Panel on Plant Protection Products and their Residues). (2013). Scientific Opinion on the developmental neurotoxicity potential of acetamiprid and imidacloprid. *EFSA Journal* 11(12):3471-3518. doi:10.2903/j.efsa.2013.3471.

⁹⁸ Environmental Protection Agency, Office of Pesticide Programs. (2011). Pesticide industry sales and usage, 2006-2007 market estimates. Retrieved from <http://www.epa.gov/opp00001/pestsales/>. Page 16.

⁹⁹ Bouchard, M. F., Chevrier, J., Harley, K. G., Kogut, K., Vedar, M., Calderon, N., Trujillo, C., Johnson, C., Bradman, A., Barr, D.B. and Eskenazi, B. (2011). Prenatal Exposure to Organophosphate Pesticides and IQ in 7-Year-Old Children. *Environmental Health Perspectives*, 119(8), 1189–1195. doi:10.1289/ehp.1003185.

¹⁰⁰ Environmental Protection Agency. (February 4, 2014) Pesticides: Regulating Pesticides, Food Quality Protection Act (FQPA) of 1996 [Webpage] Available online at <http://www.epa.gov/opp00001/regulating/laws/fqpa/>.

¹⁰¹ 21 U.S.C. § 346a(b)(2)(A)(i).

¹⁰² Food Quality Protection Act of 1996. Public Law 104-170.

¹⁰³ Groth III, E., Benbrook, C., Benbrook, K. and Goldberg, A.J. (2001, February). A report card for the EPA: Success and failures in implementing the Food Quality Protection Act. Consumers Union of the United States. Retrieved from http://consumersunion.org/pdf/fqpa/ReportCard_final.pdf. Page 6.

¹⁰⁴ Groth III, E., Benbrook, C., Benbrook, K. and Goldberg, A.J. (2001, February). A report card for the EPA: Success and failures in implementing the Food Quality Protection Act. Consumers Union of the United States. Retrieved from http://consumersunion.org/pdf/fqpa/ReportCard_final.pdf.

¹⁰⁵ Landrigan, P.J. and Benbrook, C.M. (2006). Impacts of the FQPA on children’s exposures to pesticides. Paper presented at AAAS 2006 Annual Meeting: Symposium on Opportunities and Initiatives to Minimize Children’s Exposure to Pesticides. St. Louis, MO. Pages 14-15.

¹⁰⁶ Calvert, G.M., Karnik, J., Mehler, L., Beckman, J., Morrissey, B., Sievert, J., Barrett, R., Lackovic, M., Mabee, L, Schwartz, A., Mitchell, Y. and S. Moraga-McHaley. (2008). Acute pesticide poisoning among agricultural workers in the United States, 1998-2005. *American Journal of Industrial Medicine* 51(12): 883-98. doi:10.1002/ajim.20623.

¹⁰⁷ Centers for Disease Control and Prevention (2013). Pesticide illness & injury surveillance - NIOSH workplace safety and health topic. Retrieved from <http://www.cdc.gov/niosh/topics/pesticides/>.

¹⁰⁸ Environmental Protection Agency (2014) 40 CFR Part 170. Pesticides; Agricultural Worker Protection Standards Revisions. Federal Register 79(53): 15444-15531. Pages 15450-15451.

¹⁰⁹ Environmental Protection Agency (2014) 40 CFR Part 170. Pesticides; Agricultural Worker Protection Standards Revisions. Federal Register 79(53): 15444-15531. Page 15450.

¹¹⁰ Environmental Protection Agency (2011, January 8) Methyl Bromide Questions & Answers [Webpage] Available online at <http://www.epa.gov/ozone/mbr/qa.html>.

¹¹¹ U.S. Department of Agriculture. Agricultural Research Service. (2014, March 13) Strawberry growers test methyl bromide alternatives [Webpage] Available online at <http://www.ars.usda.gov/is/ar/archive/jan01/straw0101.htm?pf=1>.

¹¹² Environmental Protection Agency (2014, August 5) The phaseout of methyl bromide [Webpage] Available online at <http://www.epa.gov/ozone/mbr/>.

¹¹³ Environmental Protection Agency (July 31, 2014) Final Rule. Protection of stratospheric ozone: a 2014 and 2015 critical use exemption from the phaseout of methyl bromide. Docket ID No. EPA-HQ-OAR-2014-0065.

¹¹⁴ Gemmill, A., Gunier, R.B., Bradman, A., Eskenazi, B. and K.G. Harley. (2013). Residential proximity to methyl bromide use and birth outcomes in an agricultural population in California. *Environmental Health Perspectives* 121(6): 737-743. <http://dx.doi.org/10.1289/ehp.1205682>.

¹¹⁵ Centers for Disease Control and Prevention (2011, July 15) Illness associated with exposure to methyl bromide — fumigated produce — California 2010. *Morbidity and Mortality Weekly Report* 60(27): 923-926.

¹¹⁶ Centers for Disease Control and Prevention. (n.d.) Documentation for immediately dangerous to life or health concentrations, Methyl Bromide [Webpage] Available online at <http://www.cdc.gov/niosh/idlh/74839.html>.

¹¹⁷ Barry, K. H., Koutros, S., Lubin, J. H., Coble, J. B., Barone-Adesi, F., Freeman, L. E. B., ... & Alavanja, M. C. (2012). Methyl bromide exposure and cancer risk in the Agricultural Health Study. *Cancer Causes & Control* 23(6): 807-818.

¹¹⁸ Budnik, L.T., Kloth, S., Velasco-Garrido, M. and Baur, X. (2012) Prostate cancer and toxicity from critical use exemptions of methyl bromide: Environmental protection helps protect against human health risks. *Environmental Health* 11:5. doi:10.1186/1476-069X-11-5.

¹¹⁹ Cockburn, M., Mills, P., Zhang, X., Zadnick, J., Goldberg, D., & Ritz, B. (2011). Prostate cancer and ambient pesticide exposure in agriculturally intensive areas in California. *American journal of epidemiology*, 173(11): 1280-1288.

¹²⁰ Budnik, L.T., Kloth, S., Velasco-Garrido, M. and Baur, X. (2012) Prostate cancer and toxicity from critical use exemptions of methyl bromide: Environmental protection helps protect against human health risks. *Environmental Health* 11:5. doi:10.1186/1476-069X-11-5.

¹²¹ Gemmill, A., Gunier, R.B., Bradman, A., Eskenazi, B. and K.G. Harley. (2013). Residential proximity to methyl bromide use and birth outcomes in an agricultural population in California. *Environmental Health Perspectives* 121(6): 737-743. <http://dx.doi.org/10.1289/ehp.1205682>.

¹²² Starks, S.E., Gerr, F., Kamel, F., Lynch, C.F., Alavanja, M.C., Sandler, D.P. and Hoppin, J.A. (2012) High pesticide exposure events and central nervous system function among pesticide applicators in the Agricultural Health Study. *International Archives of Occupational and Environmental Health* 85(5): 505-515.

¹²³ Payne, K., Andreotti, G., Bell, E., Blair, A., Coble, J. and Alavanja, M. (2012). Determinants of high pesticide exposure events in the agricultural health cohort study from enrollment (1993-1997) through phase II (1999-2003). *Journal of Agricultural Safety and Health* 18(3):167-179.

¹²⁴ Alavanja, M. C., Sandler, D. P., Lynch, C. F., Knott, C., Lubin, J. H., Tarone, R., ... & Blair, A. (2005). Cancer incidence in the agricultural health study. *Scandinavian Journal of Work, Environment & Health* 31(suppl. 1): 39-45.

¹²⁵ Koutros, S., Beane Freeman, L.E., Lubin, J.H., Heltshe, S.L., Andreotti, G., Barry, K.H., DellaValle, C.T., Hoppin, J.A., Sandler, D.P., Lynch, C.F., Blair, A. and Alavanja, M.C. (2013). Risk of total and aggressive prostate cancer and pesticide use in the Agricultural Health Study. *American Journal of Epidemiology* 177(1): 59-74. doi:10.1093/aje/kws225.

¹²⁶ Alavanja, M. C., Dosemeci, M., Samanic, C., Lubin, J., Lynch, C. F., Knott, C., ... & Blair, A. (2004). Pesticides and lung cancer risk in the agricultural health study cohort. *American Journal of Epidemiology*: 160(9), 876-885.

¹²⁷ Barry, K. H., Koutros, S., Lubin, J. H., Coble, J. B., Barone-Adesi, F., Freeman, L. E. B., ... & Alavanja, M. C. (2012). Methyl bromide exposure and cancer risk in the Agricultural Health Study. *Cancer Causes & Control*: 23(6), 807-818.

¹²⁸ Environmental Protection Agency, Office of Pesticide Programs. (2011). Pesticide industry sales and usage, 2006-2007 market estimates. Retrieved from <http://www.epa.gov/opp00001/pestsales/>. Page 14.

¹²⁹ Lee, W. J., Sandler, D. P., Blair, A., Samanic, C., Cross, A. J., & Alavanja, M. C. (2007). Pesticide use and colorectal cancer risk in the Agricultural Health Study. *International journal of cancer*: 121(2), 339-346.

¹³⁰ Environmental Protection Agency, Office of Pesticide Programs. (2011). Pesticide industry sales and usage, 2006-2007 market estimates. Retrieved from <http://www.epa.gov/opp00001/pestsales/>. Page 14.

¹³¹ Environmental Protection Agency (2007, September). Reregistration Eligibility Decision for Aldicarb. Retrieved from http://www.epa.gov/pesticides/reregistration/REDS/aldicarb_red.pdf.

¹³² Goldner, W. S., Sandler, D. P., Yu, F., Hoppin, J. A., Kamel, F., & LeVan, T. D. (2010). Pesticide use and thyroid disease among women in the Agricultural Health Study. *American Journal of Epidemiology*: 171(4), 455-464.

¹³³ Goldner, W. S., Sandler, D. P., Yu, F., Hoppin, J. A., Kamel, F., & LeVan, T. D. (2010). Pesticide use and thyroid disease among women in the Agricultural Health Study. *American Journal of Epidemiology*: 171(4), 455-464.

¹³⁴ Goldner, W. S., Sandler, D. P., Yu, F., Shostrom, V., Hoppin, J. A., Kamel, F., and LeVan, T. D. (2013). Hypothyroidism and pesticide use among male private pesticide applicators in the agricultural health study. *Journal of Occupational and Environmental Medicine* 55(10): 1171-8.

¹³⁵ U.S. Department of Labor (n.d.). OSHA law & regulations [Webpage]. Retrieved from <https://www.osha.gov/law-regs.html>. Last accessed on February 13, 2015.

¹³⁶ Code of Federal Regulations. Title 29, Part 1910 - Occupational Safety and Health Standards.

¹³⁷ Code of Federal Regulations. Title 29, Part 1928 - Occupations Safety and Health Standards for Agriculture. 29 CFR 1928.110(c)(4)(v).

¹³⁸ U.S. Department of Labor (2007, July 16). Letter from Richard Fairfax, Director, Directorate of Enforcement Programs at OSHA to Mr. Michael J. Frenzel, Associated Safety Consultants, Inc. Retrieved from https://www.osha.gov/pls/oshaweb/owadisp.show_document?p_table=INTERPRETATIONS&p_id=25803. Last accessed on February 13, 2015.

¹³⁹ Environmental Protection Agency (2014). 40 CFR Part 170. Pesticides; Agricultural Worker Protection Standards Revisions. Federal Register 79(53): 15444-15531. Page 15451.

¹⁴⁰ Environmental Protection Agency (2014). 40 CFR Part 170. Pesticides; Agricultural Worker Protection Standards Revisions. Federal Register 79(53): 15444-15531. Page 15446.

¹⁴¹ Environmental Protection Agency (2014) 40 CFR Part 170. Pesticides; Agricultural Worker Protection Standards Revisions. Federal Register 79(53): 15444-15531. Page 15447.

¹⁴² Environmental Protection Agency (2014) 40 CFR Part 170. Pesticides; Agricultural Worker Protection Standards Revisions. Federal Register 79(53): 15444-15531. Page 15451.

¹⁴³ Farmworker Justice. (2013) Exposed and ignored: How pesticides are endangering our nation’s farmworkers. Available online at <http://www.farmworkerjustice.org/sites/default/files/aExposed%20and%20Ignored%20by%20Farmworker%20Justice%20ingles%20compressed.pdf>. Last accessed on February 10, 2015.

¹⁴⁴ U.S. Environmental Protection Agency (2000, September 29) Pesticide registration notice 2000-9, Notice to manufacturers, producers, formulators and pesticide registrants of pesticide products. Retrieved from http://www.epa.gov/PR_Notices/pr2000-9.pdf. Last accessed on February 28, 2015.

¹⁴⁵ Environmental Protection Agency (2014) 40 CFR Part 170. Pesticides; Agricultural Worker Protection Standards Revisions. Federal Register 79(53): 15444-15531.

¹⁴⁶ Environmental Protection Agency (2014) 40 CFR Part 170. Pesticides; Agricultural Worker Protection Standards Revisions. Federal Register 79(53): 15444-15531. Page 15449.

¹⁴⁷ Coronado, G.D., Holte, S., Vigoren, E., Griffith, W.C., Faustman, E., and B. Thompson. (2011). Organophosphate pesticide exposure and residential proximity to nearby fields: evidence of the drift pathway. *Journal of Occupational and Environmental Medicine* 53(8): 884-891. doi:10.1097/JOM.0b013e318222f03a.

¹⁴⁸ Guillette, E.A., Meza, M.M., Aquilar, M.G., Soto, A.D. and I.E. Garcia. (1998). An anthropological approach to the evaluation of preschool children exposed to pesticides in Mexico. *Environmental Health Perspectives* 106(6): 347-353.

¹⁴⁹ Gemmill, A., Gunier, R.B., Bradman, A., Eskenazi, B. and K.G. Harley. (2013). Residential proximity to methyl bromide use and birth outcomes in an agricultural population in California. *Environmental Health Perspectives* 121(6): 737-743. <http://dx.doi.org/10.1289/ehp.1205682>.

¹⁵⁰ Marks, A.R., Harley, K., Bradman, A., Kogut, K., Barr, D.B., Johnson, C., Calderon, N. and B. Eskenazi. (2010). Organophosphate Pesticide Exposure and Attention in Young Mexican-American Children. *Environmental Health Perspectives* 118(12):1768-1774. doi:10.1289/ehp.1002056.

¹⁵¹ Bouchard, M. F., Chevrier, J., Harley, K. G., Kogut, K., Vedar, M., Calderon, N., Trujillo, C., Johnson, C., Bradman, A., Barr, D.B. and B. Eskenazi. (2011). Prenatal Exposure to Organophosphate Pesticides and IQ in 7-Year-Old Children. *Environmental Health Perspectives*, 119(8), 1189–1195. doi:10.1289/ehp.1003185.

¹⁵² Bouchard, M. F., Chevrier, J., Harley, K. G., Kogut, K., Vedar, M., Calderon, N., Trujillo, C., Johnson, C., Bradman, A., Barr, D.B. and B. Eskenazi. (2011). Prenatal Exposure to Organophosphate Pesticides and IQ in 7-Year-Old Children. *Environmental Health Perspectives*, 119(8), 1189–1195. doi:10.1289/ehp.1003185.

¹⁵³ Lanphear, B. P., Hornung, R., Khoury, J., Yolton, K., Baghurst, P., Bellinger, D. C., ... Roberts, R. (2005). Low-Level Environmental Lead Exposure and Children's Intellectual Function: An International Pooled Analysis. *Environmental Health Perspectives*, 113(7), 894–899. doi:10.1289/ehp.7688.

¹⁵⁴ Shelton, J.F., Geraghty, E.M., Tancredi, D.J., Delwiche, L.D., Schmidt, R.J., Ritz, B., Hansen, R.L. and I. Hertz-Picciotto. (2014). Neurodevelopmental disorders and prenatal residential proximity to agricultural pesticides: the CHARGE study. *Environmental Health Perspectives* 122(10): 1103-9. doi:10.1289/ehp.1307044.

¹⁵⁵ Parron, T., Requena, M., Hernandez, A.F. and R. Alarcon. (2011). Association between environmental exposure to pesticide and neurodegenerative diseases. *Toxicology and Applied Pharmacology* 256(3): 379-85. doi:10.1016/j.taap.2011.05.006.

¹⁵⁶ Baltazar, M.T., Dinis-Oliviera, R.J., de Lourdes Bastos, M., Tsatsakis, A.M., Duarte, J.A. and F. Carvalho. (2014). Pesticides exposure as etiological factors of Parkinson's disease and other neurodegenerative diseases-A mechanistic approach. *Toxicology Letters* S0378-4274(14): 00059-9. doi:10.1016/j.toxlet.2014.01.039.

¹⁵⁷ London, L., Beseler, C., Bouchard, M. F., Bellinger, D. C., Colosio, C., Grandjean, P., ... Stallones, L. (2012). Neurobehavioural and neurodevelopmental effects of pesticide exposures. *Neurotoxicology*, 33(4), 887–896. doi:10.1016/j.neuro.2012.01.004.

¹⁵⁸ Hayden, K.M., Norton, M.C., Darcey, D., Osbye, T., Zandi, P.P., Breitner, J.C.S. and K.A. Welsh-Bohmer. (2010). Occupational exposure to pesticides increases the risk of incident AD, The Cache County Study. *Neurology* 74: 1524-1530.

¹⁵⁹ Marks, A.R., Harley, K., Bradman, A., Kogut, K., Barr, D.B., Johnson, C., Calderon, N. and B. Eskenazi. (2010). Organophosphate Pesticide Exposure and Attention in Young Mexican-American Children. *Environmental Health Perspectives* 118(12):1768-1774. doi:10.1289/ehp.1002056.

¹⁶⁰ Bouchard, M. F., Chevrier, J., Harley, K. G., Kogut, K., Vedar, M., Calderon, N., Trujillo, C., Johnson, C., Bradman, A., Barr, D.B. and B. Eskenazi. (2011). Prenatal Exposure to Organophosphate Pesticides and IQ in 7-Year-Old Children. *Environmental Health Perspectives*, 119(8), 1189–1195. doi:10.1289/ehp.1003185.

¹⁶¹ Gemmill, A., Gunier, R.B., Bradman, A., Eskenazi, B. and K.G. Harley. (2013). Residential proximity to methyl bromide use and birth outcomes in an agricultural population in California. *Environmental Health Perspectives* 121(6): 737-743. <http://dx.doi.org/10.1289/ehp.1205682>.

¹⁶² Gemmill, A., Gunier, R.B., Bradman, A., Eskenazi, B. and K.G. Harley. (2013). Residential proximity to methyl bromide use and birth outcomes in an agricultural population in California. *Environmental Health Perspectives* 121(6): 737-743. <http://dx.doi.org/10.1289/ehp.1205682>.

¹⁶³ Bouchard, M. F., Chevrier, J., Harley, K. G., Kogut, K., Vedar, M., Calderon, N., Trujillo, C., Johnson, C., Bradman, A., Barr, D.B. and B. Eskenazi. (2011). Prenatal Exposure to Organophosphate Pesticides and IQ in 7-Year-Old Children. *Environmental Health Perspectives*, 119(8), 1189–1195. doi:10.1289/ehp.1003185.

¹⁶⁴ Eskenazi, B., Marks, A.R., Bradman, A., Harley, K., Barr, D.B., Johnson, C., Morga, N. and Jewell, N.P. (2007). Organophosphate pesticide exposure and neurodevelopment in young Mexican-American children. *Environmental Health Perspectives* 115(5): 792-798.

¹⁶⁵ Marks, A.R., Harley, K., Bradman, A., Kogut, K., Barr, D.B., Johnson, C., Calderon, N. and B. Eskenazi. (2010). Organophosphate Pesticide Exposure and Attention in Young Mexican-American Children. *Environmental Health Perspectives* 118(12):1768-1774. doi:10.1289/ehp.1002056.

¹⁶⁶ Environmental Protection Agency (2012, July 27). Clothianidin - registration status and related information [Webpage]. Retrieved from <http://www.epa.gov/pesticides/about/intheworks/clothianidin-registration-status.html>.

¹⁶⁷ Environmental Protection Agency, Office of Pesticide Programs. (2011). Pesticide industry sales and usage, 2006-2007 market estimates. Retrieved from <http://www.epa.gov/opp00001/pestsales/>. Page 14.

¹⁶⁸ de Solla, S.R., Palonen, K.E. and P.A. Martin. (2014). Toxicity of pesticides associated with potato production, including soil fumigants, to snapping turtle eggs (*Chelydra serpentina*). *Environmental Toxicology* 33(1): 102-6. doi:10.1002/etc.2393.

¹⁶⁹ U.S. Fish and Wildlife Service, Division of Migratory Bird Management (January 2002). Migratory bird mortality: many human-caused threats afflict our bird populations [Fact sheet].

¹⁷⁰ Pimentel, D. (2005). Environmental and economic costs of the application of pesticides primarily in the United States. *Environment, Development and Sustainability* 7: 229-252. doi:10.1007/s10668-005-7314-2.

¹⁷¹ Davis, J. U.S. Fish & Wildlife Service. (2011, July). Pesticide Fate and Transport (a.k.a. Where do pesticides go in the environment?). Retrieved from <http://nctc.fws.gov/resources/course-resources/pesticides/2011Presentations/Tab%20%20Pesticide%20Fate%20&%20Transport.pdf>. Last accessed on February 27, 2015.

¹⁷² U.S. Geological Survey. National Water-Quality Assessment Program. Pesticide National Synthesis Project. Pesticide Use Maps—Atrazine. [Webpage]. Retrieved from http://water.usgs.gov/nawqa/pnsp/usage/maps/show_map.php?year=2011&map=ATRAZINE&hilo=L&disp=Atrazine. Last accessed on February 27, 2015.

¹⁷³ Sass, J.B. and Colangelo, A. (2006). European Union bans atrazine, while the United States negotiates continued use. *International Journal of Occupational and Environmental Health* 12(3):260-7.

¹⁷⁴ U.S. Environmental Protection Agency. (2015, January). Pesticides: Reregistration. Atrazine Updates. [Webpage archive.] Retrieved from http://www.epa.gov/opprrd1/reregistration/atrazine/atrazine_update.htm. Last accessed on February 27, 2015.

¹⁷⁵ U.S. Geological Survey. (2006, March). Pesticides in the Nation's Streams and Ground Water, 1992–2001—A Summary. Retrieved from <http://pubs.usgs.gov/fs/2006/3028/>. Last accessed on February 27, 2015.

¹⁷⁶ See, e.g., U.S. Environmental Protection Agency (2009, May). National Primary Drinking Water Regulations. Retrieved from <http://water.epa.gov/drink/contaminants/upload/mcl-2.pdf>.

¹⁷⁷ Environmental Protection Agency (2012, July 27). Clothianidin - registration status and related information [Webpage]. Retrieved from <http://www.epa.gov/pesticides/about/intheworks/clothianidin-registration-status.html>.

¹⁷⁸ Committee on the Status of Pollinators in North America. Board on Life Sciences. Board on Agriculture and Natural Resources. Division of Earth and Life Sciences. National Research Council (2007). *Status of Pollinators in North America*. Washington, D.C.: National Academies Press.

¹⁷⁹ Goulson, D. (2012, February 10). Decline of bees forces China's apple farmers to pollinate by hand. *China Dialogue*. Retrieved from <https://www.chinadialogue.net/article/show/single/en/5193>.

¹⁸⁰ U.S. Department of Agriculture, Agricultural Research Service (2013, May 6). Survey of bee losses during winter of 2012/2013 [Webpage]. Retrieved from <http://www.ars.usda.gov/is/br/beelosses/index.htm>.

¹⁸¹ Committee on the Status of Pollinators in North America. Board on Life Sciences. Board on Agriculture and Natural Resources. Division of Earth and Life Sciences. National Research Council (2007). *Status of Pollinators in North America*. Washington, D.C.: National Academies Press. Page 80.

¹⁸² Chensheng, L. U., Warchol, K. M. and Callahan, R. A. (2014). Sub-lethal exposure to neonicotinoids impaired honey bees winterization before proceeding to colony collapse disorder. *Bulletin of Insectology* 67(1): 125-130.

¹⁸³ Henry, M., Beguin, M., Requier, F., Rollin, O., Odoux, J. F., Aupinel, P., ... and Decourtye, A. (2012). A common pesticide decreases foraging success and survival in honey bees. *Science* 336(6079): 348-350.

¹⁸⁴ Williamson, S. M. and Wright, G. A. (2013). Exposure to multiple cholinergic pesticides impairs olfactory learning and memory in honeybees. *The Journal of Experimental Biology*, 216(10): 1799-1807.

¹⁸⁵ Palmer, M. J., Moffat, C., Saranzewa, N., Harvey, J., Wright, G. A. and Connolly, C. N. (2013). Cholinergic pesticides cause mushroom body neuronal inactivation in honeybees. *Nature Communications* 4: 1634. doi:10.1038/ncomms2648.

¹⁸⁶ Medrzycki, P., Montanari, R., Bortolotti, L., Sabatini, A.G., Maini, S. and Porrini, C. (2003). Effects of imidacloprid administered in sub-lethal doses on honey bee behaviour. Laboratory tests. *Bulletin of Insectology* 56(1): 59-62.

¹⁸⁷ Whitehorn, P. R., O'Connor, S., Wackers, F. L., and Goulson, D. (2012). Neonicotinoid pesticide reduces bumble bee colony growth and queen production. *Science* 336(6079): 351-352.

¹⁸⁸ Henry, M., Beguin, M., Requier, F., Rollin, O., Odoux, J. F., Aupinel, P., ... and Decourtye, A. (2012). A common pesticide decreases foraging success and survival in honey bees. *Science*, 336(6079), 348-350.

¹⁸⁹ U.S. Environmental Protection Agency (2008). Imidacloprid Summary Document Registration Review: Initial Docket December 2008. Docket number EPA-HQ-OPP-2008-0844. Retrieved from <http://www.regulations.gov/#!docketDetail;D=EPA-HQ-OPP-2008-0844>.

¹⁹⁰ U.S. Environmental Protection Agency (2008). Imidacloprid Summary Document Registration Review: Initial Docket December 2008. Docket number EPA-HQ-OPP-2008-0844. Retrieved from <http://www.regulations.gov/#!docketDetail;D=EPA-HQ-OPP-2008-0844>.

¹⁹¹ U.S. Environmental Protection Agency (2008). Imidacloprid Summary Document Registration Review: Initial Docket December 2008. Docket number EPA-HQ-OPP-2008-0844. Retrieved from <http://www.regulations.gov/#!docketDetail;D=EPA-HQ-OPP-2008-0844>. Page 3.

¹⁹² Environmental Protection Agency (2014, October 16). EPA actions to protect pollinators [Webpage]. Retrieved from <http://www2.epa.gov/pollinator-protection/epa-actions-protect-pollinators>.

¹⁹³ Henry, M., Beguin, M., Requier, F., Rollin, O., Odoux, J. F., Aupinel, P., ... & Decourtye, A. (2012). A common pesticide decreases foraging success and survival in honey bees. *Science* 336(6079): 348-350.

¹⁹⁴ Williamson, S. M. and Wright, G. A. (2013). Exposure to multiple cholinergic pesticides impairs olfactory learning and memory in honeybees. *The Journal of Experimental Biology*, 216(10): 1799-1807.

¹⁹⁵ Palmer, M. J., Moffat, C., Saranzewa, N., Harvey, J., Wright, G. A., & Connolly, C. N. (2013). Cholinergic pesticides cause mushroom body neuronal inactivation in honeybees. *Nature Communications*, 4: 1634.

¹⁹⁶ Medrzycki, P., Montanari, R., Bortolotti, L., Sabatini, A.G., Maini, S. and C. Porrini. (2003) Effects of imidacloprid administered in sub-lethal doses on honey bee behaviour. Laboratory tests. *Bulletin of Insectology* 56(1): 59-62.

¹⁹⁷ Whitehorn, P. R., O'Connor, S., Wackers, F. L., & Goulson, D. (2012). Neonicotinoid pesticide reduces bumble bee colony growth and queen production. *Science*, 336(6079), 351-352.

¹⁹⁸ Henry, M., Beguin, M., Requier, F., Rollin, O., Odoux, J. F., Aupinel, P., ... & Decourtye, A. (2012). A common pesticide decreases foraging success and survival in honey bees. *Science* 336(6079), 348-350.

¹⁹⁹ Chensheng, L. U., Warchol, K. M. and Callahan, R. A. (2014). Sub-lethal exposure to neonicotinoids impaired honey bees winterization before proceeding to colony collapse disorder. *Bulletin of Insectology* 67(1): 125-130.

²⁰⁰ European Food Safety Authority. (2013). Conclusion on the peer review of the pesticide risk assessment for bees for the active substance clothianidin. *EFSA Journal* 11(1):3066-3124. doi:10.2903/j.efsa.2013.3066.

²⁰¹ European Food Safety Authority. (2013). Conclusion on the peer review of the pesticide risk assessment for bees for the active substance imidacloprid. *EFSA Journal* 11(1):3068-3123. doi:10.2903/j.efsa.2013.3068.

²⁰² European Food Safety Authority. (2013). Conclusion on the peer review of the pesticide risk assessment for bees for the active substance thiamethoxam. *EFSA Journal* 11(1):3067-3135. doi:10.2903/j.efsa.2013.3067.

²⁰³ European Commission. Regulation (EU) No. 485/2013.

²⁰⁴ Federal Insecticide, Fungicide, and Rodenticide Act. Title 7 U.S.C. Section 136.

²⁰⁵ Benbrook, C. (December 2008). Prevention, not profit, should drive pest management (Rachel Carson Memorial Lecture). *Pesticides News* 82: 12-17.

²⁰⁶ International Agency for Research on Cancer. (2015) Agents classified by the IARC Monographs, Volumes 1-111. Retrieved from <http://monographs.iarc.fr/ENG/Classification/ClassificationsAlphaOrder.pdf>. Last accessed on March 4, 2015. Page 8.

²⁰⁷ Schreinemachers, D.M. (2000) Cancer mortality in four northern wheat-producing states. *Environmental Health Perspectives* 108(9): 873-881.

²⁰⁸ Chensheng, L. U., Warchol, K. M. and Callahan, R. A. (2014). Sub-lethal exposure to neonicotinoids impaired honey bees winterization before proceeding to colony collapse disorder. *Bulletin of Insectology* 67(1): 125-130.

²⁰⁹ U.S. Department of Agriculture, Agricultural Research Service (2013, May 6). Survey of bee losses during winter of 2012/2013 [Webpage]. Retrieved from <http://www.ars.usda.gov/is/br/beelosses/index.htm>.

²¹⁰ Pleasants, J.M. and Oberhauser, K.S. (2012) Milkweed loss in agricultural fields because of herbicide use: effect on the monarch butterfly population. *Insect Conservation and Diversity*. doi:10.1111/j.1752-4598.2012.00196.x.

²¹¹ Bouchard, M. F., Chevrier, J., Harley, K. G., Kogut, K., Vedar, M., Calderon, N., Trujillo, C., Johnson, C., Bradman, A., Barr, D.B. and B. Eskenazi. (2011). Prenatal Exposure to Organophosphate Pesticides and IQ in 7-Year-Old Children. *Environmental Health Perspectives*, 119(8), 1189–1195. doi:10.1289/ehp.1003185.

²¹² Marks, A.R., Harley, K., Bradman, A., Kogut, K., Barr, D.B., Johnson, C., Calderon, N. and B. Eskenazi. (2010). Organophosphate Pesticide Exposure and Attention in Young Mexican-American Children. *Environmental Health Perspectives* 118(12):1768-1774. doi:10.1289/ehp.1002056.

²¹³ Environmental Protection Agency (2007, April 24) Atrazine, Chemical Summary [Webpage] Retrieved from http://www.epa.gov/teach/chem_summ/Atrazine_summary.pdf. Last accessed on February 18, 2015.

²¹⁴ de Solla, S.R., Palonen, K.E. and P.A. Martin. (2014). Toxicity of pesticides associated with potato production, including soil fumigants, to snapping turtle eggs (*Chelydra serpentina*). *Environmental Toxicology* 33(1): 102-6. doi:10.1002/etc.2393.

²¹⁵ Alavanja, M. C., Sandler, D. P., Lynch, C. F., Knott, C., Lubin, J. H., Tarone, R., ... & Blair, A. (2005). Cancer incidence in the agricultural health study. *Scandinavian Journal of Work, Environment & Health* 31(suppl. 1): 39-45.

²¹⁶ Alavanja, M. C., Dosemeci, M., Samanic, C., Lubin, J., Lynch, C. F., Knott, C., ... & Blair, A. (2004). Pesticides and lung cancer risk in the agricultural health study cohort. *American Journal of Epidemiology*: 160(9), 876-885.

²¹⁷ Barry, K. H., Koutros, S., Lubin, J. H., Coble, J. B., Barone-Adesi, F., Freeman, L. E. B., ... & Alavanja, M. C. (2012). Methyl bromide exposure and cancer risk in the Agricultural Health Study. *Cancer Causes & Control*: 23(6), 807-818.

²¹⁸ Goldner, W. S., Sandler, D. P., Yu, F., Shostrom, V., Hoppin, J. A., Kamel, F., and LeVan, T. D. (2013). Hypothyroidism and pesticide use among male private pesticide applicators in the agricultural health study. *Journal of Occupational and Environmental Medicine* 55(10): 1171-8.

²¹⁹ Starks, S.E., Gerr, F., Kamel, F., Lynch, C.F., Alavanja, M.C., Sandler, D.P. and Hoppin, J.A. (2012) High pesticide exposure events and central nervous system function among pesticide applicators in the Agricultural Health Study. *International Archives of Occupational and Environmental Health* 85(5): 505-515.

²²⁰ Payne, K., Andreotti, G., Bell, E., Blair, A., Coble, J. and Alavanja, M. (2012). Determinants of high pesticide exposure events in the agricultural health cohort study from enrollment (1993-1997) through phase II (1999-2003). *Journal of Agricultural Safety and Health* 18(3):167-179.

²²¹ U.S. Fish and Wildlife Service, Division of Migratory Bird Management (January 2002). Migratory bird mortality: many human-caused threats afflict our bird populations [Fact sheet].

²²² Centers for Disease Control and Prevention (2013). Pesticide illness & injury surveillance - NIOSH workplace safety and health topic. Retrieved from <http://www.cdc.gov/niosh/topics/pesticides/>.

²²³ Code of Federal Regulations. Title 7, Part 205. 7 CFR 205.601(b).

²²⁴ Code of Federal Regulations. Title 7, Part 205. 7 CFR 205.601(e).

²²⁵ Schneider, M.K., Luscher, M., Jeanneret, P., Arndofer, M., Ammari, Y., Bailey, D., Balazs, K., ... and Herzog, F. (2014) Gains to species diversity in organically farmed fields are not propagated at the farm level. *Nature Communications* 5. doi:10.1038/ncomms5151.

²²⁶ Lu, C., Toepel, K., Irish, R., Fenske, R.A., Barr, D.B. and R. Bravo. (2006). Organic diets significantly lower children’s dietary exposure to organophosphorus pesticides. *Environmental Health Perspectives* 114(2): 260-263.

²²⁷ Curl, C.L., Beresford, S.A.A., Fenske, R.A., Fitzpatrick, A.L., Chensheng, L., Nettleton, J.A. and Kaufman, J.D. (2015). Estimating Pesticide Exposure from Dietary Intake and Organic Food Choices: The Multi-Ethnic Study of Atherosclerosis (MESA). *Environmental Health Perspectives*. Retrieved from <http://ehp.niehs.nih.gov/1408197/>. doi: 10.1289/ehp.1408197.

²²⁸ Lu, C., Toepel, K., Irish, R., Fenske, R.A., Barr, D.B. and R. Bravo. (2006). Organic diets significantly lower children’s dietary exposure to organophosphorus pesticides. *Environmental Health Perspectives* 114(2): 260-263.

²²⁹ Bouchard, M.F., Bellinger, D.C., Wright, R.O. and M.G. Weisskopf. (2010). Attention deficit/hyperactivity disorder and urinary metabolites of organophosphate pesticides in U.S. children 8-15 years. *Pediatrics* 125(6): e1270-1277. doi:10.1542/peds.2009-3058.

²³⁰ Environmental Protection Agency. (n.d.). EPA’s regulation of *Bacillus thuringiensis* (Bt) Crops [Webpage]. Retrieved from <http://www.epa.gov/pesticides/biopesticides/pips/regofbt crops.htm>.

²³¹ Freese, B., Kimbrell, G., Cohen, S., Kingston, H., Perrone, S., Seiler, A., Stella, C. and Tomaselli, P. (2013). Seed giants vs. US farmers. Center for Food Safety & Save Our Seeds. Retrieved from http://www.centerforfoodsafety.org/files/seed-giants_final_04424.pdf.

²³² Monsanto. (n.d). Roundup Ready Plus [Webpage]. Retrieved from <http://www.monsanto.com/products/pages/roundup-ready-plus.aspx>.

²³³ Benbrook, C. M. (2012). Impacts of genetically engineered crops on pesticide use in the US—the first sixteen years. *Environmental Sciences Europe*, 24(1): 1-13.

²³⁴ Environmental Protection Agency, Office of Pesticide Programs. (2011). Pesticide industry sales and usage, 2006-2007 market estimates. Retrieved from <http://www.epa.gov/opp00001/pestsales/>. Page 14.

²³⁵ Union of Concerned Scientists (December 2013). The rise of superweeds, and what to do about it. Retrieved from www.ucsusa.org/superweeds.

²³⁶ VanGessel, M.J. (2001) Glyphosate-resistant horseweed from Delaware. *Weed Science* 49: 703-705.

²³⁷ International Survey of Herbicide Resistant Weeds. (n.d.). Weeds resistant to the herbicide glyphosate [Data file]. Retrieved from <http://www.weedscience.org/summary/home.aspx>.

²³⁸ International Survey of Herbicide Resistant Weeds. (n.d.). Weeds resistant to the herbicide glyphosate [Data file]. Retrieved from

<http://www.weedscience.org/summary/home.aspx>.



For more information, please contact:
Jen Shecter
Director, External Relations,
(914) 378-2402, jshecter@consumer.org
greenerchoices.org