CAST® Issue Paper
Number 66
October 2019

Interpreting Pesticide Residues in Food

While many consumers are concerned with pesticide residues on produce, the significant health benefits from eating a diet rich in produce and whole grains dramatically outweigh the miniscule risks from pesticide residues. (Photo from Monkey Business Images/Shutterstock.)

ABSTRACT

Consumers in the United States are frequently exposed to residues of pesticides in their food. The existence of pesticide residues in food raises questions regarding what consumer health risks, if any, are posed by such chemical contaminants.

This report concludes that there is no direct scientific or medical evidence indicating that typical exposure of consumers to pesticide residues poses any health risk. Pesticide residue data and exposure estimates typically demonstrate that food consumers are exposed to levels of pesticide residues that are several orders of magnitude below those of potential health concern. Human epidemiological studies, often employing biomonitoring studies of pesticide metabolites as an indicator of pesticide exposure, have suggested correlations between pesticide exposure and specific types of disease, but such studies are limited in their ability to measure both disease and pesticide exposure and have been inconsistent in their findings. As an example, results of six epidemiological studies examining the relationship between exposure to the insecticide chlorpyrifos and childhood intelligence are discussed. Two of the six studies indicated a positive correlation between chlorpyrifos exposure and reduced childhood intelligence but both focused on exposure from non-food sources (indoor pesticide use and agricultural pesticide use). Another study looking at indoor chlorpyrifos use did not identify any correlation to childhood intelligence nor did three other epidemiological studies estimating chlorpyrifos food exposure.

The judicious use of pesticides in food production also provides numerous benefits to society. Such benefits include greater productivity, availability, and affordability of food; a reduction in pest damage, food loss, and waste; and public health benefits such as control of potentially dangerous mycotoxins or fungi in our food.

Consumers are frequently advised to avoid purchasing specific conventionally produced fruits and vegetables because of contamination concerns. Researchers have demonstrated that such advice lacks scientific justification and may result in some consumers reducing their consumption of fruits and vegetables, a practice strongly associated with adverse health effects. The best thing consumers can do is to eat a diet rich in fruits,

CAST Issue Paper 66 Task Force Members

Authors

Dr. Carl Winter (Chair), Department of Food Science and Technology, University of California at Davis, Davis, California

Dr. Carol Burns, Burns Epidemiology Consulting, LLC, Sanford, Michigan

Dr. Steve Savage, Independent Consultant, Vista, California

Reviewers

Kaci Buhl, MS, Environmental and Molecular Toxicology, Oregon State University, Corvalis, Oregon

Dr. Honglei Chen, Department of Epidemiology and Biostatistics, Michigan State University, East Lansing, Michigan

Dr. Franklin Mink, MAI, Corinth, Kentucky

CAST Liaison

Dr. Donald C. Beitz, Department of Biochemistry, Biophysics and Molecular Biology, Iowa State University, Ames, Iowa

vegetables, and whole grains, whether the foods are organic or conventional.

INTRODUCTION

There is a keen awareness among many consumers that pesticide chemicals frequently reach consumers in the form of food residues. This awareness can be troubling to many consumers since pesticides are typically used because of their ability to harm biological organisms such as insects, plant diseases, and weeds. Their presence in foods introduces the question as to what risks, if any, are consumers facing as they consume ubiquitous pesticide residues in their diet. Because of safety concerns, consumers frequently receive advice from advocates concerning whether one should eat conventional or organic foods, which conventional fruits and vegetables one should consider avoiding, which geographical sources of foods should be avoided, and what one could be doing to reduce exposures to pesticides by washing, peeling, cooking, or scrubbing foods before they eat them.

The scientific basis for such advice is frequently lacking. The simple presence of a pesticide residue on a food item is not sufficient evidence of harm since pesticides, like all chemicals, obey the principles of toxicology. It is the dose, or exposure to the chemical, that determines the potential risks. Consumers may be choosing to follow questionable advice to avoid specific foods because of potential pesticide residues, contradicting the sound advice from health care providers, requiring a balanced and varied diet rich in fruits, vegetables, and whole grains.

The pesticide regulatory system in the United States is simultaneously cited as an important safeguard of the U.S. food supply by some and as a cause for concern by others. As an example, the results of federal pesticide residue testing from foods often demonstrate that a majority of foods contain no detectable levels of pesticide residues and violation rates are low, yet the same studies indicate that residues are frequently detected and demonstrate that consumers are routinely being exposed to pesticides in their food. The relationship between allowable levels of pesticides on foods (tolerances) and safety is poorly understood and frequently implies that pesticide residue findings that violate U.S. laws may pose health risks to consumers. This is particularly true in the case of food imported into the United States from other countries, which frequently demonstrates much higher violation rates than food produced domestically.

In addition to pesticide residue regulatory programs, biomonitoring studies have frequently detected metabolites of pesticides in urine samples from consumers. Biomonitoring results may serve as an indicator of consumer exposure to pesticides and such results are often paired with epidemiological studies to study the potential links between pesticide exposures and diseases. A handful of recent epidemiological studies have suggested links between pesticide exposure and adverse male reproductive effects and developmental and behavioral effects in infants and children.

While such links between exposure and disease have received considerable media coverage and need to be taken seriously, it is also important to understand the strengths and limitations of epidemiological investigations for such results to be evaluated appropriately. The quality of data used and obtained in such studies is critical and findings from individual studies should be compared with those from other related studies to see if there are overlapping similarities. It is also critical to understand that such links between exposure and disease represent correlations or associations and not causes; additional plausible explanations for the links need to be considered as well.

The uses and benefits of pesticides in food production should also be recognized. Pesticides provide one tool to produce a safe, effective, and economically viable food supply, particularly when they are used in concert with a variety of other biological, genetic, cultural, and mechanical approaches to control pests. Pesticides can improve land- and water-use efficiencies that can minimize energy requirements and reduce greenhouse gas emissions. In addition, pesticides provide a powerful tool in reducing food waste as well as in increasing food safety.

This CAST Issue Paper focuses on pesticide residues in the food supply and describes several complex yet poorly understood aspects that are key to evaluating scientific papers, media food safety stories, and consumer advice regarding which foods consumers should (or should not) consume.

PESTICIDE RESIDUES IN FOODS: REGULATION AND RISK ASSESSMENT

The agricultural use of pesticides has enabled food producers to increase their crop yields significantly in the United States and throughout the world. Pesticides provide a useful tool to control insects (insecticides), weeds (herbicides), plant diseases (fungicides), as well as other agricultural pests.

One consequence of using pesticides in agriculture is that pesticide residues are often detected on our foods. The use of pesticides does not necessarily imply that residues will be encountered; many pesticide applications are made prior to the emergence of edible portions of plants while in other cases, sufficient time between pesticide application and crop harvest allows the pesticides to degrade below detectable levels. Nevertheless, residues of pesticides on food crops commonly occur and their existence is often cause for consumer concern.

Regulation of Pesticide Residues in the United States

In the United States, pesticides are primarily regulated by the United States Environmental Protection Agency (EPA). In cases where specific pesticides may pose the potential to expose consumers to pesticide residues from food, the EPA will not permit specific uses of pesticides unless it is determined that consumer exposure to the pesticide from all sources represents a "reasonable certainty of no harm" according to the 1996 Food Quality Protection Act (FQPA) (Winter 2005). The calculation of a "reasonable certainty of no harm" involves assessing realistic levels of human exposure through consideration of pesticide residue levels and food consumption patterns as well as comparisons of exposure estimates with toxicological criteria such as the reference or benchmark dose. In addition, the FQPA requires the EPA scientists to consider the special potential susceptibilities of infants and children to pesticide exposure, to consider "aggregate" exposure to the pesticide from food, drinking water, and residential environments, and to consider "cumulative" exposure to entire families of toxicologically related pesticides possessing similar mechanisms of biological action.

If, after investigating all of these issues, EPA scientists conclude consumer exposure to a pesticide represents a "reasonable certainty of no harm," the EPA will allow the pesticide to be legally used on specified food crops and will set the maximum allowable residue level, or tolerance, for the specific pesticide on the specific food crop. On the other hand, if consumer exposure to a specific pesticide exceeds a level of "reasonable certainty of no harm," the pesticide will not be registered for specific food uses (Winter 2005).

The processes used to establish pesticide residue tolerances are complicated and counterintuitive (Winter 1992a). Tolerances are not indicators of safety but serve instead as barometers to indicate if pesticide applications are performed according to good agricultural practices. Tolerances are typically set to be slightly higher than the maximum residue levels observed when the pesticide application is made according to legal directions (i.e., proper pesticide application rate, proper number of applications per growing season, application made to the proper crop, adherence to an established interval between final application and harvest). As such, anticipated pesticide residue levels following legal application of pesticides should fall below the tolerance level (usually far below the level) and only in the case of pesticide misuse would a food residue exceed the tolerance level.

Regulatory Monitoring of Pesticide Residues in the United States

The U.S. Food and Drug Administration (FDA) is the primary federal agency responsible for enforcing pesticide tolerances. In 2016, the FDA laboratories analyzed 2,670 domestic food samples and 4,276 imported food samples for pesticide residues in its "Regulatory Monitoring of Foods Program" using multiresidue techniques capable of detecting residues of more than 200 individual pesticides (USFDA 2018). Findings from the 2016 FDA program are shown in Figure 1.

The FDA found that a majority of samples from both domestic and imported foods contained no detectable pesticide residues while most of the detectable residues were within tolerance levels. A large difference in violation rates was seen with domestic samples showing a 0.9% violation rate while import sample violations were much higher (9.8%).

Pesticide residue violations can occur in two ways: (1) when residue levels exceed the tolerance established for the specific pesticide/food combination, and (2) when residue levels—at any level—are detected on foods for which a tolerance is not established (even if the pesticide is legally allowed on other foods). Violations in this category frequently occur through

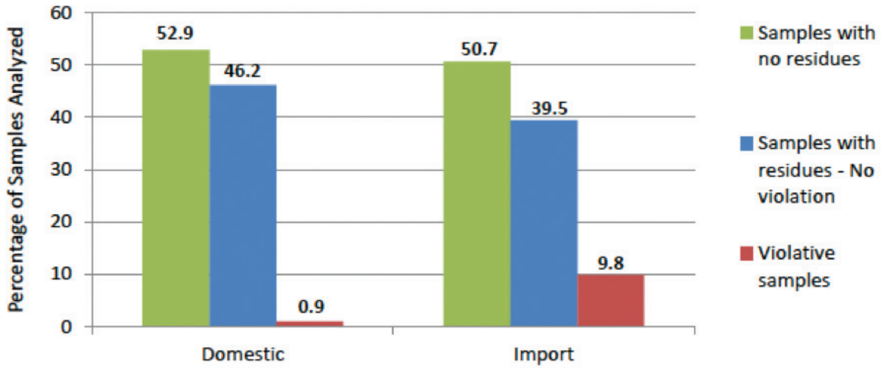


Figure 1. The percentage of food samples tested by the FDA in 2016 with pesticide residues. Pesticide residue violations can occur when residue levels exceed the tolerance established for the specific pesticide/food combination, and when residue levels are detected on foods for which a tolerance is not established. Source: FDA 2018

drift of a pesticide applied legally to one crop that may reach a different crop for which the pesticide is not registered, uptake from contaminated soil, or comingling of different agricultural products during transportation, packing, or distribution. The FDA identified 25 domestic food violations for pesticide residues in 2016; only four (16%) of the violations involved over-tolerance residues. Of the 418 imported food violations identified in 2016, 64 (15%) represented over-tolerance residues. Food identified as violative by the FDA is not allowed to enter the channels of commerce.

Interpreting Pesticide Residue Monitoring Results

The 2016 FDA residue findings suggest, particularly for domestically produced foods, that pesticide applications generally demonstrate compliance with legal and established agricultural practices. For both domestic and imported foods, the majority of samples contained no detectable pesticide residues while detected residues were typically present at levels far below the tolerance levels. Such findings are reassuring given that tolerance levels are set to represent the maximum residues anticipated following legal application of pesticides.

Much public focus concerns the violation rates (0.9% for domestic foods, 9.8% for imported foods) and the potential impact of violations on consumer safety. As discussed previously, most violations do not occur in cases where tolerance levels have been exceeded but rather in cases where the pesticide was detected on a commodity for which a tolerance was not established. Even in the infrequent cases where tolerances were exceeded, though, these instances very rarely constitute cases of food safety concern since tolerances are set on the basis of good agricultural practices and are not safety indicators (Winter 1992a). Tolerances are only established in cases where the EPA has already determined that the pesticide, through all its registered uses, poses a "reasonable certainty of no harm" to consumers. Monitoring of foods for pesticide residues therefore provides a valuable check to identify if pesticide applications are being made according to established legal requirements and as an economic disincentive to discourage food producers from using pesticides inappropriately since foods shown to contain violative residues are not permitted for sale in the United States.

Violative residues, however, should not be equated with "unsafe" residues. Tolerance levels could be raised significantly without anticipating adverse health effects. As an example, the current U.S. tolerance for captan on strawberries is 20 parts per million, which is 12.5 times lower than the safety level. Winter and Jara (2015) estimated that residues of the fungicide captan on strawberries would need to reach a level of at least 250 parts per million prior to eliciting any preliminary health concern.

Assessing Consumer Risks from Exposure to Pesticides in Food

The presence or absence of pesticide residues is not a valid indicator of health risk to the consumer. Pesticides, like all chemicals, obey the first principle of toxicology: "The dose makes the poison." Thus, it is the amount of exposure and not the presence or absence of a chemical that determines the potential for harm.

The calculation of consumer exposure to pesticides includes two components: (1) the level of pesticide residue on food, and (2) the amount of the food that is consumed. If a pesticide is found on multiple foods, the contributions of exposure from each food are combined to provide an estimation of the total dietary exposure to the food (Winter 1992b).

To determine the potential health risk from the estimated exposure, the exposure level is compared to a toxicological indicator of preliminary health concern. This indicator is commonly referred to as the reference dose (RfD) and represents the lowest level of exposure of health concern; exposures below the RfD are considered to be negligible in terms of health risks. RfDs may be calculated differently for acute (short term, aRfD) exposures or chronic (longer term, cRfD) exposure.

Since human toxicology data for pesticides are limited, most of the toxicology data used to calculate RfDs come from laboratory animal studies that examine a multitude of health and metobolic parameters in an attempt to determine the most sensitive effect (one that occurs at the lowest dose of the pesticide) in the most sensitive animal species. Once that effect has been established, the highest dose given to the most sensitive animal species that does not cause the most sensitive toxicological effect is identified as the no observable adverse effect level (NOAEL) (Winter and Francis 1997) or the comparable benchmark dose. Recognizing that humans could potentially be more sensitive to pesticides than the most sensitive animal species, prudent measures are taken into account when developing a RfD on the basis of the NOAEL. In practice, a 10-fold uncertainty factor is applied that assumes humans are 10 times more sensitive to the pesticide than the most sensitive animal species tested; this is multiplied by another 10-fold uncertainty factor that assumes some humans may be 10 times more sensitive than the average human. Using this approach, the NOAEL is divided by a factor of 100 (10 for animal/human extrapolation x 10 for human/sensitive human extrapolation) to yield the RfD (Winter and Francis 1995). In some cases, such as when it is determined that toxicology information is limited or when infants and children may be more susceptible to pesticides than adults, an additional 3- to 10-fold uncertainty factor may be included prior to calculation of the RfD.

Dietary pesticide exposure assessment often uses pesticide residue data collected from programs such as the FDA's "Regulatory Monitoring of Foods" program and the U.S. Department of Agriculture's Pesticide Data Program (PDP). The FDA's regulatory monitoring program does not sample foods randomly so data collected from this program are not considered to be representative of the food supply. The PDP samples only a few food commodities each year and thus its utility is also limited.

The FDA has conducted the Total Diet Study (TDS) annually since 1961. The TDS is a market basket study in which the FDA inspectors purchase food items from retail outlets in four separate locations each year and prepare 280 food items for consumption. At the time the food is ready to be eaten, it is analyzed for residues of hundreds of different pesticides as well as nutrients, metals, and other contaminants. Pesticide residue results are made available to the public and the FDA also provides consumption estimates for the 280 individual food items.

An analysis of consumer chronic exposure to pesticide residues, based upon 2004–2005 TDS results, was published by Winter (2015). Residues of 77 individual pesticides were detected and exposure estimates for each of these pesticides were compared to chronic RfD (cRfD) values. Exposures to only three pesticides exceeded 1% of the cRfD and included two pesticides no longer used in agriculture but persistent enough in the environment that residues are still common. Exposures between 0.1 to 1.0% of the cRfD were estimated for fourteen pesticides, while 19 pesticides had exposures between 0.01 to 0.1% of the chronic reference dose. Exposures to the remaining 41 pesticides that were detected in the 2004–2005 TDS were below 0.01% of the cRfD. Since an exposure of 0.01% of the cRfD represents an exposure one million times lower than the NOAEL (the highest dose that does not cause effects in the most susceptible animal species), it is reasonable to conclude based on the data that chronic exposure to pesticides from food in the United States is typically at levels far below those of health concern.

Risk assessment for acute (single day) exposures to pesticides is conducted differently. Since both pesticide residue levels and daily food consumption patterns vary significantly, probabilistic models have been developed to examine the distributions of both residues and food consumption. In the unlikely event of a very high pesticide residue occurring at the same time that a significant amount of the food containing the pesticide was consumed, it is possible that the daily exposure could approach the acute RfD (aRfD) level for the pesticide. The EPA, in determining what constitutes a "reasonable certainty of no harm" for acute exposures, calculates individual day estimates for a variety of population subgroups—including infants, children, and women of childbearing age. If exposure at the upper 99.9th percentile (the highest daily exposure for all population subgroups over 1,000 days) to the pesticide from all foods, drinking water, and residential use is below the aRfD level, exposure is considered to provide a "reasonable certainty of no harm" and the pesticide and all of its associated uses are considered safe for consumers.

Interpeting Consumer Advice Regarding Pesticides in Food

Consumer concern has been influenced by nontraditional and much less rigorous approaches regarding pesticide residues in the food supply. Among these is "Shopper's Guide to Pesticides," released annually by a Washington, D.C.-based environmental advocacy group and includes a list of the "Dirty Dozen" foods representing the 12 commodities most likely to contain pesticide residues. The authors of this guide urge consumers to avoid purchasing conventional (nonorganic) forms of these foods to avoid potential health effects from pesticide exposure (Environmental Working Group 2018).

The rankings of the "Dirty Dozen" and other fruits and vegetables are derived from PDP data collected by the U.S. Department of Agriculture. Specific fruits and vegetables are ranked on the basis of six criteria: (1) percentage of samples with detectable residues, (2) percentage of samples with two or more pesticides detected, (3) average number of pesticides found on a single sample, (4) average amount of all pesticides found, (5) maximum number of pesticides found on a single sample, and (6) total number of pesticides found on the commodity (EWG 2018).

A close look at the criteria used to rank pesticide "contamination" reveals that none of the metrics are useful in terms of traditional dietary pesticide risk assessment. Notably missing from the methodology are the three key pillars of risk assessment: levels of residues, food consumption estimates, and established toxicity indicators for individual pesticides.

Using a more traditional approach, Winter and Katz (2011) analyzed PDP data for 10 most frequently detected pesticides on each of the 2010 "Dirty Dozen" commodities and developed chronic dietary exposure estimates for 120 commodity/pesticide combinations. Winter and Katz indicated that estimated exposures were far below chronic RfD levels in all cases. Only one of the 120 commodity/pesticide combinations showed an exposure above one percent of the chronic RfD while only seven exposures were greater than 0.1% of the chronic RfD. Three-quarters of the exposure estimates were below 0.01% of the chronic RfD. The authors concluded that (1) consumer exposure to the most frequently detected pesticides on the "Dirty Dozen" commodities was negligible, (2) substituting organic forms of the "Dirty Dozen" commodities would not appreciably reduce risks, and (3) the methodology used to develop the "Dirty Dozen" list lacked scientific rigor or subsequent credibility.

The health benefits of eating a diet rich in fruits, vegetables, and whole grains have been well established and there is concern that attention given to inappropriate warnings of dire consequences resulting from pesticide residue exposure could encourage consumers to reduce their consumption of fruits and vegetables. A recent study was conducted of low-income shoppers who were provided a variety of statements regarding organic and conventional fruits and vegetables (Huang, Edirisinghe, and Burton-Freeman 2016). When responding to the statement: "An environmentalist group called the Environmental Working Group has developed a list of the 12 fresh fruits and vegetables they say have the highest pesticide levels on average: apples, bell peppers, carrots, celery, cherries, grapes, kale, lettuce, nectarines, peaches, pears, and strawberries. They suggest that it is best to buy these fruits and vegetables organically grown," a much greater number of low-income consumers indicated that they were less likely to purchase fruits and vegetables compared with their responses to the other statements.

Similar unfounded concerns regard the consumption of imported fruits and vegetables, which represent the primary source of fruits and vegetables for many U.S. consumers during the winter months. While pesticide residue violation rates tend to be greater for imported foods than for domestic foods, it is also clear that violative residues are rarely indicative of health consequences. Katz and Winter (2009) estimated dietary exposure to pesticides detected on domestic and imported produce during the FDA's 2003 Regulatory Monitoring Program. Even though violation rates were much higher for the imported produce, exposure estimates indicated that exposure to specific pesticides was more frequently lower from consuming imported produce than from consuming domestic produce. In the case of one pesticide that resulted in thirty six import violations and one domestic violation, overall exposure to the specific pesticide was still lower from consumption of imported produce while well below levels of safety concern for both domestic and imported produce.

PESTICIDE EPIDEMIOLOGY AND BIOMONITORING

Epidemiology studies look for patterns and potential causes of disease. In contrast to laboratory studies, because human subjects are observed, the concept of causality is softened with terms like risk factors, linkages, and associations. Regardless of the language, epidemiology investigations look for clues of health and exposure status between groups. Through careful observation, epidemiology research has identified many risk factors for diseases. Factors such as obesity, smoking, high cholesterol, hypertension, and physical inactivity have been determined to lead to increases in heart disease (Fryar, Chen, and Li 2012). Relatively uncommon diseases such as birth defects were found to be associated with too much of one thing (alcohol) and too little of another (folic acid) (Streissguth 1978; Viswanathan et al. 2017). The successes of the disease detectives who compared the noncases to the cases with toxic shock syndrome, for example, led scientists to better define the disease and determine how it was spread (Schuchat and Broome 1991).

Historically, the success of epidemiology as a science depends on two critical pieces of information: disease and exposure. To gain information for a study, investigators will often simply ask study participants about their medical conditions. The quality of the answers can vary with the seriousness of the disease; diagnoses of cancer are harder to forget (or mischaracterize on a questionnaire) than vague symptoms such as fatigue, headache, or being out of breath. The more robust epidemiology studies make the effort to confirm the sensitivity and specificity of the underlying data. For example, to study heart disease the best practice is to obtain medical records to confirm the diagnosis, date of onset, and treatment.

Similarly, with exposure, the quality of the information varies widely. Self-reported exposure(s) based on daily habits, like smoking or drinking coffee or one's job tend to be more accurate, even in the distant past. However, sometimes it is difficult to determine if people have been exposed to specific agents such as to a virus or a chemical. This is particularly true for pesticides. Whereas farmers and applicators may know what they have used, their internal exposure(s) is determined by a host of determinants such as what was applied, how it was applied, and the type, if any, of protective clothing worn.

For those not involved in agriculture and especially those living far from the source, we have little to no information about pesticide exposure. As noted previously, exposures to pesticides may occur from trace residues on fruits and vegetables. Random sampling programs in the United States and Canada have collected urine and blood from men, women, and children across the country to identify levels of all sorts of chemicals including metals, industrial contaminants, and pesticides (Health Canada 2010; CDC 2015). Researchers have shown that some pesticides and/or their metabolic breakdown products are detectable and may be found in large percentages of the population. The limit of detection, or the ability to identify a chemical through laboratory analysis, has gotten smaller over time. Scientists today can detect up to 100 times lower levels of the herbicide 2,4-D than just a few decades ago (LaKind et al. 2017). The U.S. Centers for Disease Control and Prevention (CDC) clearly states that detection alone is not an indication of health concern (CDC 2015). Further, this "snapshot" does not provide any information about when, how, or where the exposure occurred. Since most of the observations correspond to levels well below the doses administered to laboratory animals in toxicological studies, such findings would generally provide reassurance rather than alarm.

Source of Exposure

How do epidemiologists know that one person has more exposure than another? How do they know that it is from food residue?

Biomonitoring, from urine or blood, is both solution and problem. The advantage of one or more urine (or blood) samples is that they can be analyzed for traces of specific pesticides and other chemicals. The levels tell us "what" and "how much." The problem is that most pesticides used today break down to other components within days of application in the field as well as often within hours after ingestion. This means that when pesticide metabolites are found in human urine, it is impossible to tell whether the individual was exposed to the parent compound or the metabolite (Sudakin and Stone 2011). Futher, the biomonitoring information is only relevant for the previous day or two. Unless the exposure is from a food item that is eaten regularly, the investigator can't determine exposure last month (or year), which may be the important time period for disease onset.

Some studies of pesticide applicators and farmers can infer the *source* of exposure, especially if collection occurs soon after an application (Arbuckle et al. 2002; Mandel et al. 2005; Thomas et al. 2010). A study conducted in New York City assumed that the participants were exposed as the result of indoor pesticide treatments (Whyatt et al. 2005). Other studies with no other information simply assume the source is from food (Fortenberry et al. 2014; Imai et al. 2014; Ji et al. 2011). The lack of concrete data on the sources of pesticide exposure is concerning when associating diseases and pesticides levels from biomonitoring data.

Income, for example, is a common factor when evaluating health among populations that may also impact pesticide exposure. Table 1 illustrates how specific population groups may differ among each other with respect to pesticide exposure by income levels. It can be speculated that lower income participants eat fewer servings of fresh fruits and vegetables since fresh produce is relatively expensive, particularly in urban areas. If this is the case, it is reasonable to assume that the lower income group may demonstrate lower urinary levels of pesticide metabolites than higher income groups. Conversely, another rationale is that pesticide exposures may occur more frequently among lower income participants due to treatment of pests in their home or pesticide drift following pesticide applications from nearby fields in more rural environments. The opposite could be inferred of high-income participants who either eat organic produce (low pesticide exposure) or have high consumption of traditionally grown food (high pesticide exposure).

Table 2 similarly illustrates that people may change their behavior, and thereby exposure, based on disease. Imagine a family with a child on the autism spectrum. Their food choices and even residence may change because of the diagnosis. This is called reverse causality and makes interpretation of epidemiology studies difficult.

The problem is that speculation is poor science and is far removed from the scientific method of basing conclusions on strong data-driven evidence. As an example, a high-profile French study recently concluded that participants eating a high organic food diet experienced a significant reduction in the risk of cancer compared with those who did not eat a high organic food diet (Baudry et al. 2018). The women who reported a high frequency of organic food consumption were also more likely to eat a healthy diet and exercise frequently. Another limitation is that the authors of this study didn't actually test, even on a small sample, if their reported score for organic diet was correct.

Food Residue and Health

What does the epidemiology literature say about pesticides in general? Here are two examples.

Table 1. Theorized pesticide exposure by income.

	Low Pesticide Levels	High Pesticide Levels
Low Income	Cannot afford fruits and vegetables	Lives in pest-infested homes requiring treatments Lives near agricultural fields
High Income	Washes fruits and vegetables Buys organic	Consumes many fruits and vegetables

Table 2. Theorized pesticide exposure by disease.

	Low Pesticide Levels	High Pesticide Levels
No Disease	Doesn't follow any special diet Doesn't eat many fruits and vegetables	Doesn't worry about eating organic Eats traditionally grown produce
Disease	Washes fruits and vegetables Buys organic Keeps house very clean Moves to city to be near doctor(s)	Consumes many fruits and vegetables Moves to rural area for less pollutants

Example 1. Pyrethroid insecticides and sperm quality

Pyrethroids are a class of insecticides that are widely used in the home environment and in agriculture. In the body, pyrethroids break down quickly and several metabolize to the common metabolite 3-phenoxybenzoic acid (3-PBA) that is detectable in urine. Due to their widespread use, contact with pyrethroids can occur from residues on produce. It has been proposed that pyrethroids may affect the human reproductive system and several investigators have evaluated levels of urinary 3-PBA in men and semen quality (Imai et al. 2014; Ji et al. 2011; Meeker, Barr, and Hauser 2008; Perry et al. 2007; Radwan et al. 2014; Toshima et al. 2012; Xia et al. 2008). Each relied upon the same design, called a cross-sectional study. During a single visit, investigators collected urine and semen from the study participants. None of the participants were a pesticide applicator and the exposure was presumed to be from ingestion on food. Levels of 3-PBA and sperm quality were analyzed. When sperm quality was statistically significantly lower among the men with higher 3-PBA the association is considered to be inverse.

A summary of the results for sperm concentration and sperm motility from these studies is shown in Table 3. The collective evidence clearly shows that while some studies reported an inverse association for sperm concentration, others did not. If there were a positive, causal effect of pyrethroid exposure and decreased sperm quality, we would expect to see it in most investigations. Further, a different study observed an inverse association for sperm motility but found no adverse association with sperm concentration. Without going into the strengths and weaknesses of each study, a limitation common to all the studies is that the exposure (urinary 3-PBA) and outcome (sperm quality) were collected at the same time, preventing an evaluation of temporality. In other words, it is impossible to determine if the exposure preceded the outcome or vice versa, which is a hallmark of epidemiologic evaluation (Hill 1965). In summary, this example suggests that there is no effect of pyrethroid exposure (as measured using

Table 3. Associations of urinary 3-PBA for selected sperm characteristics.

Author, Year Published	Country	Sperm Concentration	Sperm Motility
Imai, 2014	Japan	No effect	No effect
Ji, 2011	China	Inverse	No effect
Radwan, 2014	Poland	No effect	No effect
Meeker, 2008	USA	No effect	No effect
Perry, 2007	China	Inverse*	Not reported
Toshima, 2012	Japan	No effect	Inverse
Xia, 2008	China	Inverse	No effect

*This was a pilot of 18 men, not statistically tested

Table 4. Association of estimated in utero chlorpyrifos levels and childhood intelligence.

Author, Year Published	Location	IQ Score	Exposure Source
Rauh, 2011	New York (Columbia)	Decreased	Indoor treatment
Engel, 2011	New York (Mt. Sinai)	No effect	Indoor treatment
Bouchard, 2011	California	Decreased	Agriculture
Donauer, 2016	Ohio	No effect	Food residue
Cartier, 2016	France	No effect	Food residue
Jusko, 2019	Netherlands	No effect	Food residue

urinary 3-PBA levels) and sperm quality. This is in line with results for laboratory animals which are conducted at a dose level many times higher than humans would encounter (USEPA 2011)

Example 2. Organophosphate insecticide and neurodevelopment

Like the pyrethroids, organophosphates are a broad class of several dozen insecticides. Each metabolizes within 24 hours in the body and can be detected as the generic dialkylphosphate. One organophosphate, chlorpyrifos, has been discussed prominently for a connection with brain development in growing children. Not unlike the example of pyrethroids, the evidence is mixed. Published reviews have highlighted the inconsistencies across studies and limitations in confirming in utero exposure such as relying on a single sample and using exposure estimates that are not specific to chlorpyrifos (Burns et al. 2013; Li et al. 2012; Mink, Kimmel, and Li 2012; Needham 2005; Prueitt et al. 2011).

So does chlorpyrifos reduce or impair brain development, particularly among children exposed to food residues containing chlorpyrifos? There are six epidemiology studies that evaluated chlorpyrifos (or a group of insecticides) and intelligence via an IQ test (Bouchard et al. 2011; Cartier et al. 2016; Donauer et al. 2016; Engel et al. 2011; Jusko et al. 2019; Rauh et al. 2011). Looking at the results across all the studies, the findings are mixed. Only two of the six studies reported a statistically significant decrease in IQ with increased estimates of exposure (Table 4). The exposures in all the studies were estimated from urine or blood collected when the child was born. Only two studies evaluated exposures to the growing child and found no link with development (Bouchard, et al. 2011; Cartier et al., 2016). It is important to understand that several studies are not about trace residues on purchased food. The source(s) of exposure to the New York study participants were presumed to be from indoor cockroach and ant treatments that are no longer legal in the United States and many other countries (Whyatt et al. 2005) while agricultural drift and/or track-in from farm workers was the alleged source for the California participants (Berkowitz et al. 2003; Castorina et al. 2010). The three studies that are relevant for general exposure via ingestion of residues on food found no link with impaired intelligence (Cartier et al. 2016; Donauer et al. 2016; Jusko et al. 2019).

What Does This Mean to Consumers?

There remains the concern among some consumers that detectable exposures to pesticides may lead to certain diseases. This is inferred to be from residues on foods, as well as from other sources. The types of diseases that have been reported range from reproductive effects in adult men, namely lower sperm counts and motility (Burns and Pastoor 2018; Koureas et al. 2012), to various developmental and behavioral outcomes infants and children (Burns et al. 2013; Muñoz-Quezada et al. 2013; Reiss et al. 2015). The studies vary in their approaches, tests, and conclusions. Even the reviewers vary in their approach to evaluating the studies. Some regard outcomes in more general categories and make conclusions that the evidence for some is indication of effects for all (González-Alzaga et al. 2014). Other reviewers take a more focused approach looking at evidence for specific pesticides and specific outcomes (Burns et al. 2013).

Consumers desire to make educated choices about their purchases. Scientists strive to allow people to also be educated consumers based on scientific evidence. In the event of the reporting of controversial new findings regarding pesticides and health outcomes, consumers should determine if the study adequately describes both exposure and health outcomes.

- If exposure was based on residence, did the authors provide evidence to support their assumptions?
- When relying on urine samples, did the authors collect more than one sample?
- Was information collected to reduce the speculation about how the study participants were exposed?
- Importantly, is the exposure setting based on what is known about agricultural practices?

It is critical that such questions be considered when interpreting the study and making personal, economic, and lifestyle decisions based upon study findings.

PESTICIDE BENEFITS

While much of the contemporary concern regarding pesticides involves pesticide residues in foods and their potential risks to consumers, it is important to understand how and why pesticides are used in the production of our crops and many of the benefits they provide.

How Pesticides Fit in Sustainable Pest Management

Plants provide the foundation of the food supply for all other living things. In many cases there are mutually beneficial relationships between plants and their consumers. However, even in undisturbed natural ecosystems, there are organisms that eat, damage, or infect plants in ways that effectively qualify them as "pests." Plants defend themselves from "pests" in various ways ranging from thorns to repellants to the production of pest-toxic chemicals (Osbourn 1996).

The crops that have been domesticated by humans are also subject to damage from pests such as insects, mites, nematodes, fungi, bacteria, and viruses. Crops also must compete for water, nutrients, and light with other plant species that could be viewed as weeds within the production system. While plants growing in natural settings can succeed despite a certain degree of pest damage, the threshold for acceptable pest damage in agriculture is lower because of the impact on financial risk for farmers, overall resource-use-efficiency, and also food safety and quality. It is not a viable option to simply tolerate pest damage, but pest management today involves much more than just "pesticides" (Savage 2013).

Since the 1970s, the trend in agriculture has been towards a more sustainable, integrated systems approach to the challenges of pest management. This is often described as integrated pest management (IPM) that combines diverse tactics in the context of in-depth knowledge of crop and pest biology with real-time data to implement the most economical, effective and minimally disruptive interventions (Tedford and Brown 1999). In the big picture, the goal is "sustainable pest management."

The sustainable, integrated programs involve chemical and biological crop protection products, genetic resistance within the crop itself, cultural practices that exclude or limit pests, and the encouragement of biological pest suppression through the use of beneficial organisms. Pesticide products are often a key part of the integrated system and this is true for both conventional and organic production. Even so, the goal is to use such products in a way that is safe, effective, and economically viable.

Often a pesticide is important in maintaining or enabling another strategy to work and to continue to work. As an example, if there is a genetic resistance trait in a crop for a major pest, exclusive dependence on that tool will enhance the likelihood that the pest will evolve resistance to the genetic trait so that benefit will be lost (Feng et al 2014).

Some insect pests can be defeated through a strategy of "pheromone confusion" in which non-toxic pheromone chemicals are deployed so that the male insect can't find a distinct scent trail to a female to allow mating. This approach can be quite effective, but only if the overall insect population is low. Chemical treatments targeting "hot spots" of the insect in question are needed for the pheromone strategy to succeed (Dorn et al 1999).

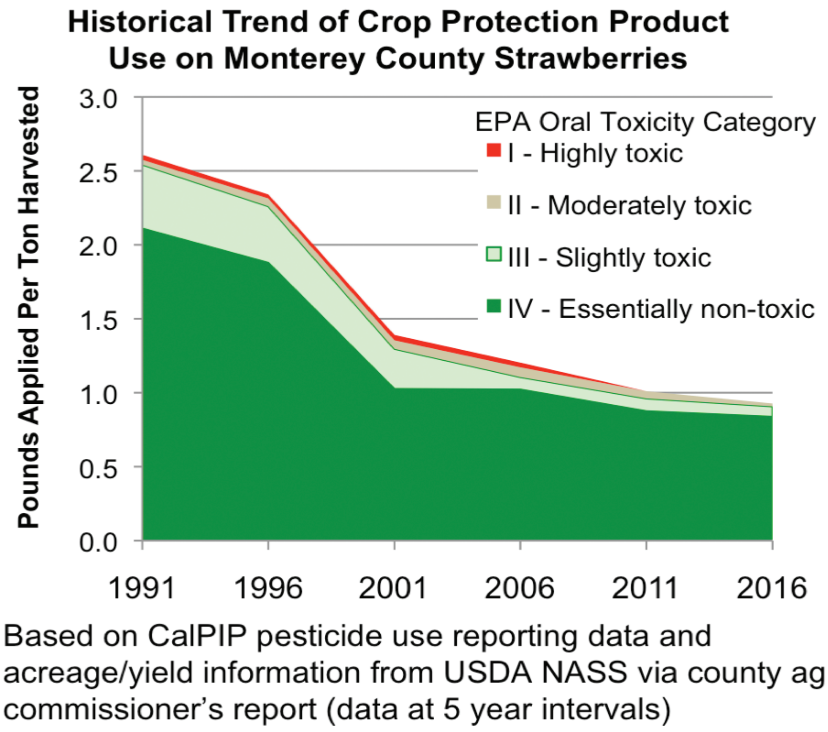
There are biological control products that can be applied to crops, but in general the best results are seen when those agents are combined or alternated with chemical control agents.

In summary, the goal of modern, sustainable pest management is not to simply rely on chemical controls for pests. Still, pesticides play an important role. Fortunately, the trend over time has been towards pesticides that are intrinsically much less toxic to humans or the environment than their predecessors (Savage 2014).

Historic trends in the quantity and oral toxicity of crop protection agents used for strawberries and apples are shown in Figures 2 and 3.

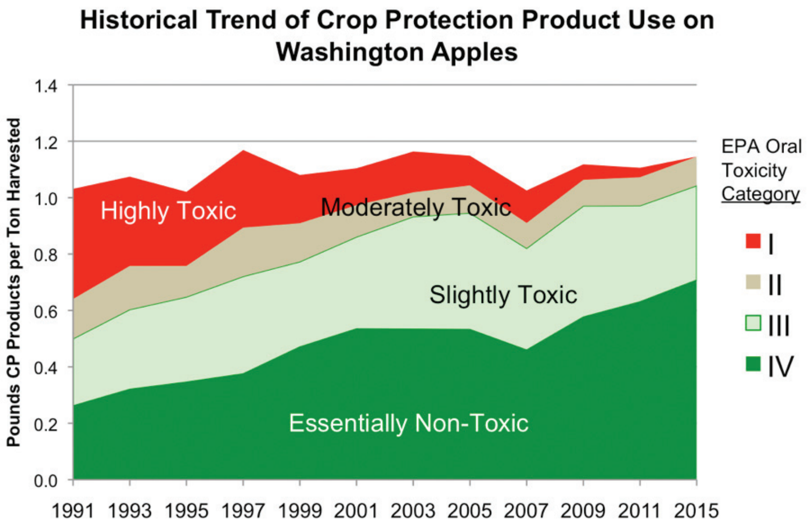
The Benefits of Crop Protection for Agricultural Communities

The humans most directly impacted by



Based on CalPIP pesticide use reporting data and acreage/yield information from USDA NASS via county ag commissioner's report (data at 5 year intervals)

Figure 2. The example of Strawberry pesticide use and relative toxicity in the key production area of Monterey County, California.



Data from USDA and Washington State Surveys

Figure 3. The example of Apple pesticide use and relative toxicity in the key production area in Washington State.

Loss Without Insect Control	Crops
90-100%	Hops, Apples, Olives
75-89%	Dates, Pears, Cherries, Oranges, Broccoli
60-74%	Blueberries, Asparagus, Nectarines, Pistachios, Peaches, Cabbage, Sunflowers
50-59%	Artichokes, Raspberries, Sweet Peppers, Pecans, Peanuts, Mint, Strawberries
40-49%	Green Beans, Lettuce, Cranberries, Celery, Avocados, Hazelnuts, Almonds, Sweet Corn
30-39%	Cucumbers, Grapes, Potatoes
20-30%	Cotton, Eggplant, Sugarbeets, Sugarcane, Onions
10-20%	Green Peas, Spinach, Rice
0-10%	Sorghum, Alfalfa, Carrots, Soybeans, Corn

Figure 4. Estimated economic loss ranges averaging data from 393 crop/state combinations: Adapted from Crop Life Foundation.

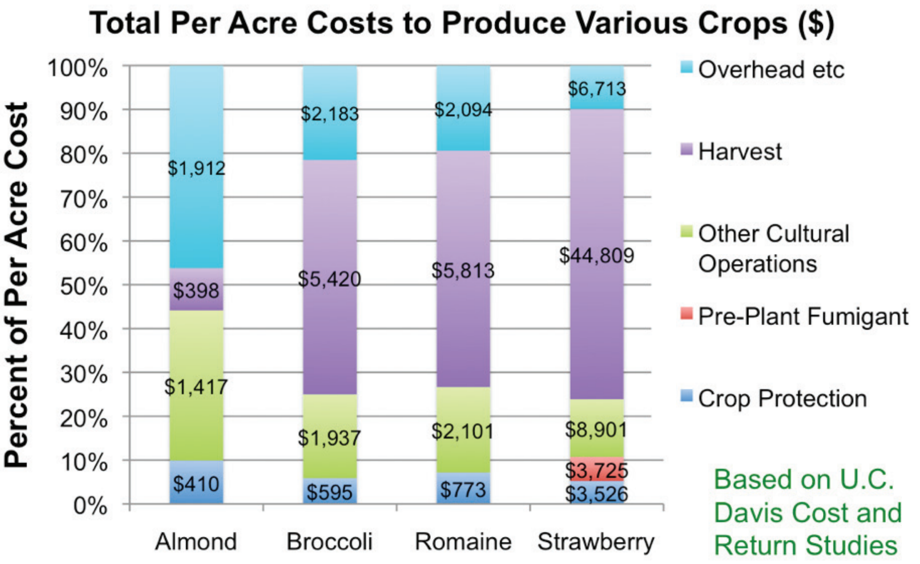
crop pests are farmers and the non-farming members of the agricultural communities whose livelihood is closely linked to the farm economy. Farming is an intrinsically risky enterprise as it seeks to manage a complex biological system that can be profoundly affected by variations in weather and by risks associated with plant pests. Farming generally involves the need to make a substantial financial investment well ahead of any income from the harvest crop (i.e., seed, fertilizer, fuel, labor, land rent). If the quantity and/or quality of the crop is compromised by serious pest damage, the farmer cannot repay those expenditures (Figure 4). While farming operations must survive bad seasons from time to time, without a reasonable degree of pest control, farming enterprises become non-viable, and those failures will also compromise the viability of other regional businesses.

An additional level of challenge occurs when new, invasive pests enter a growing region; control of this new pest may disrupt a previously well-developed IPM system. These events have occurred throughout agricultural history but are more recently increasing primarily because of increased world travel and climate change.

As an example, a new kind of fruit fly appeared in North American fruit growing regions around 2013 which can lay its eggs in fruits that are ripe, but not damaged. This is something that fruit flies were unable to do in the past. This means that fruit infested with maggots is much more likely to reach consumers. This infestation became a serious threat to the economic viability of various fruit and berry producers (Walsh et al. 2011).

In these cases it becomes a very high priority for the growers and supporting researchers to find new strategies to bring the new pests under control to prevent the financial viability of the region to be threatened.

Farm owners are not the only ones in agricultural communities who are impacted by pests, and likewise who benefit from using pesticides. For crops that are harvested by hand, pest damage can greatly reduce the picking efficiency, meaning the pounds that can be collected per hour of effort. Since pickers are typically paid by the pound, a pest-damaged crop is much less attractive from the worker's perspective. A farmer could pay by the hour in pest injury cases, but at a certain level of damage it becomes necessary to leave the crop unharvested because the cost of picking labor can be one of the single largest expenses for growers of fruit and vegetable crops. Figure 5 is based on the most recent University of California, Davis cost-return studies. For almonds, mechanical harvest represents 9.6% of the total cost to farm. But, for hand harvested crops like broccoli, romaine lettuce, and strawberries the percentage spent for harvest is 53.5%, 53.9% and 66.2% respectively.



Based on U.C. Davis Cost and Return Studies

Figure 5. Cost structure comparison for four important California crops demonstrating how much annual investment is being protected by minimizing pest-driven yield losses.

Modern pest control methods such as herbicides can also benefit workers by eliminating or greatly reducing the need for potentially injurious and difficult work such as hand weeding or weeding with a short handled hoe (Gianessi 2008).

Environmental Benefits

A sustainable pest management system is critical to the overall sustainability of any cropping system because if usable yields are significantly reduced by pest damage, land-use efficiency and water-use efficiency are greatly diminished. These key performance indicators of sustainability are expressed per unit of output (i.e., bushels/acre farmed, tons/gallon of water applied). Over the past several decades, food and fiber production have increased to meet rising demand, but most of the gains have been through increases in yield rather than through the addition of more farmed land. Pest control has been an important part of that success story. Ideally the further growth expected in food demand over the next half century will be met through increased yields and minimal land-use change, but this is only possible if pest damage is minimized (Tilman et al. 2011).

As water is likely to be a major challenge in an age of climate change (Wallace 2000), it will become increasingly irresponsible to allow excessive pest damage to undermine the efficient use of the supplies that are available.

Similarly, limiting pest damage is also critical in efforts to minimize other key “footprints” of agriculture such as energy and greenhouse gas emissions. Once again, these footprints are expressed per unit of output. Thus, pest impacts on yield effectively increases these footprints per bushel, ton, hundredweight or other measure of yield.

In recent years there has been increasing appreciation for the role of “soil health” both for ongoing agricultural productivity and for a variety of ecosystem services that a healthy soil provides (i.e., water capture and retention, carbon sequestration, nutrient buffering, surface water quality protection). The farming practices that are widely recognized as best for soil health are often best enabled by key pest management tools (e.g., reduced tillage and cover-cropping). Herbicides allow farmers to control weeds while still minimizing the soil-degrading effects of mechanical disturbance inherent in tillage (USDA 2017). While these “no-till” soils tend to be cooler and wetter, seed treatment fungicides and insecticides enable farmers to still successfully establish crops drilled into those soils (Hopkins 2017).

Cover crops are single or mixed species plantings grown between the main growing seasons of annual crops as a way to decrease soil erosion, enhance soil quality and nutritive value, and help improved air and water quality. They are sometimes grazed, but are not harvested for commercial sales. This practice can qualify for government conservation payments and is recognized as highly desirable from a sustainability perspective. However, it is critical that these crops are “terminated” prior to the next growing season so that they do not act as weeds and/or lead to cool and/or wet conditions for the germination of the crop seeds. Herbicides are an effective way to achieve timely and effective termination since tillage is counter-productive for the soil health benefits and “winter killing” is only effective for some cover crops in some regions (Legleiter et al. 2012).

Broad Societal Benefits

Food security and a diverse, affordable, healthy food supply are key societal benefits enjoyed in the developed world in the modern era. Throughout human history such abundance has not been something that can be taken for granted. There are still marginalized populations in developed countries and too many people in developing countries who do not have food security. Only by keeping pest damage to a minimum will it be possible to extend food security to all of humankind (Oerke and Dehne 2004).

The enjoyment and health aspects of a diverse food supply are enhanced by the farmer’s ability to grow many different crops in many different environments. This would not be possible without ways to deal with pest challenges that can vary enormously based on crop type and geography.

When insects damage some crops, the crops can then be subject to future damage by certain fungi that produce dangerous chemicals called mycotoxins. This serious threat to the safety of food and feed can be mitigated using an integrated system of cultural methods, genetic resistance, and chemical and biological pesticides. Few high-income country consumers even know that they are being protected from mycotoxin exposure, particularly when enjoying crops like tree nuts, peanuts, and grains.

In the low- and middle-income countries where there is not full access to the pest management "tool box," mycotoxins are a significant cause of illness and death (Appell, Kendra, and Trucksess 2010). Some of the tools for mycotoxin minimization are available to organic farmers such as biological control with strains of the same fungal species which lack the ability to make the mycotoxin. If large quantities of the spores of those strains are introduced into the field or orchard they can out-compete the dangerous strains for colonization of those insect-injury sites. Biotech crops such as corn that express genes for the highly specific protein toxins from the soil bacterum *Bacillus thuringiensis* prevent the insect damage insects thus greatly reducing the potential for contamination with aflatoxin or fumonisin.

Food System Benefits

In recent years there has been increased public awareness of the issue of food waste. Pest management is a very important component of mitigating food waste of several types. When pests damage crops in the field, that contributes to food waste to the extent that it renders the produce unharvestable or so undesirable so as to be avoided during harvest (Creamer and Johnson 2017). Another example of in-field food waste of grain crops is when damage by insects or fungi weakens the stalk so that the plant "lodges" or falls over and its grain will be missed by harvesting equipment (Nielsen and Colville 2014). In a handpicked, "field packed" crop like strawberries or grapes, rotting fruit is simply left on the plant or dropped to the ground by the pickers.

Many fruit and vegetable crops are delivered in large bins from the field to a "packing plant" where various steps like washing, sorting, packing, and chilling occur. There are generally detailed requirements in terms of size, shape, and color of the produce to meet industry standards or other customer specifications. Even relatively minor damage from insects and/or diseases can render the item to "cull status" (Baugher, Hogmire Jr., and Lightner 1990). Depending on the severity of the issue that piece may be able to go into a side-product stream (e.g., juicing, trimming, animal feed), but more severely pest damaged produce is unusable and therefore waste.

For crops like apples and potatoes that go into long term storage, very high pest control standards are needed in the field to sustain storage life. This is true both on the individual item level and to prevent major instances of waste where whole containers are destroyed by fungal infections spreading from item to item—hence the old expression: "One bad apple can spoil the whole bunch" (Ministry of Agriculture, Food and Rural Affairs 2016).

Pest-protected produce not only allows year-round access to these fresh products, it reduces the capital investment needed to have the capacity to fulfill processed markets like frozen fries. A potato processing plant using stored potatoes can handle the crop with a much smaller capacity facility working throughout the year.

"Latent" but non-visible infections of fruit in the field can cause subsequent decay and thus waste at any stage of the distribution chain from transport, to warehousing, to retail display, to the consumer's home. In some cases this kind of waste can be avoided by the use of a "post-harvest" fungicide treatment with a chemical and/or biological fungicide which is sufficiently low in toxicity so that it can be applied close to the time of consumption (Adaskaveg and Forster 2009).

In the international banana industry, there is a dramatic example of how pest control reduces food waste and enhances food availability for consumers. There is a banana disease called black Sigatoka, which only infects the leaves and not the fruit. However, if there is too much leaf infection, the bananas harvested from that tree will not survive the time it takes for the energy-efficient means of ocean shipping the fruit. Instead of a container of fresh, consumable bananas, the harvest from an infected tree will be a lake of decomposed mush. It is primarily because of an effective fungicide program during growth that bananas can be enjoyed as one of the most widely consumed fruits (Castelan et al. 2013).

Many consumers prefer to buy fresh and local food when it is available, and there are definitely some crops for which there are quality advantages of that status. Depending on the region and crop, there can be significantly more challenging pest issues in some locales. Areas with more rainfall tend to have more fungal pest issues. Warmer climate tends to have more insect challenges. Because of these challenges, pesticides provide significant benefits that allow certain local farming industries to succeed.

SUMMARY

While it is reasonable for consumers to be concerned about the presence of pesticide residues in their foods, there is an absence of direct scientific or medical evidence demonstrating that pesticide residues in the U.S. food supply pose a health threat to consumers. This conclusion is based upon findings from risk assessment studies identifying large differences between the estimated pesticide exposure levels of consumers with the levels required to be of toxicological concern. Epidemiological studies have suggested the potential for adverse effects but results from such studies are limited in their ability to measure both disease and pesticide exposure and have been inconsistent in their findings.

Before pesticides are allowed to be applied to crops that are produced domestically or imported from abroad, they must be registered for use by the EPA and all uses of pesticides are required to meet the "reasonable certainty of no harm" standard based upon anticipated consumer exposure and relation of such exposure to toxicologically significant levels. The "reasonable certainty of no harm" standard considers aggregate (i.e., food, water, residential) and cumulative (i.e., combining effects of toxicologically similar pesticides) exposure and also considers the potential increased sensitivity of population subgroups such as infants and children and pregnant women.

If it is anticipated that the agricultural use of a pesticide may result in the presence of a residue on a food crop, the EPA establishes a tolerance, representing the maximum level of the specific pesticide permitted on the specific crop. While confusing to many consumers, the tolerance level is not based upon safety, but represents the maximum residue anticipated provided the pesticide application was made following all legal requirements. Levels of pesticide residues detected in excess of the tolerance levels indicate product misuse but rarely indicate the potential for consumer health concerns. Levels of pesticide residues of health concern are typically far above tolerance levels and not commonly detected in the U.S. food supply chain.

Pesticide monitoring studies routinely demonstrate that most residues on foods are far below tolerance levels—if they are detected at all—and very few samples show residue levels in excess of tolerance levels. The majority of pesticide residue violations occur when residues of pesticides approved for use on other food crops are detected—at any level—on foods for which tolerances have not been established. As is the case with over-tolerance residues, such violations are rarely of health consequence.

Since pesticide tolerances are not safety standards, referencing results from pesticide monitoring programs that primarily focus upon enforcing tolerances is of little utility in evaluating consumer risks from exposure to pesticide residues in foods. Other studies, such as the FDA's Total Diet Study, provide evidence of the levels of pesticide residues existing on foods immediately prior to their consumption, and, through consideration of the residue levels as well as food consumption rates, allow estimates of consumer exposure from pesticide residues in foods to be calculated. Studies using this approach frequently demonstrate that such exposure is often more than one million times lower than levels that cause no effects in laboratory animals exposed to pesticides daily throughout their lifetimes. These levels are intentionally protective of public health and not predictive of human disease due to multiple levels of uncertainty factors employed by the regulatory agencies involved in food safety.

Consumers are frequently advised to seek out organic foods and/or to avoid purchasing specific conventionally-produced fruits and vegetables due to contamination concerns. Research has demonstrated that such advice lacks scientific justification and may result in some consumers reducing their consumption of fruits and vegetables, a practice strongly associated with adverse health effects.

Results from a handful of recent epidemiological studies have suggested links between pesticide exposure and disease, such as male sperm abnormalities and childhood neurodevelopment. It is critical to realize that such links represent correlations and that the effects could be caused by other factors or most likely due to chance. As an example, one study indicated that frequent consumers of organic produce have lower risks of particular diseases than do those who do not frequently consume organic produce. However, it was also the case that those frequently consuming organic foods also have greater income, have greater education, and exercise more frequently; these and other factors may be responsible for the lower incidence of disease in this cohort, rather than lower pesticide residue exposure.

It is extremely difficult to study the role of diet and health in an epidemiological study, let alone to study the role of trace levels of pesticides associated with our food. Consider trying to remember what you ate last week and how much! Unfortunately the quality of the epidemiology data for diet and the resulting pesticide residue exposure is often quite poor. Dietary exposure to pesticide residues is often estimated based on biomonitoring studies looking at the presence of pesticide metabolites in the urine but these cannot determine when, how, or where possible pesticide exposure occurred. Other studies have focused upon agricultural workers and have inferred that links between occupational exposure to pesticides and disease provide evidence that similar links might be evident from consumer exposure to food residues.

If a strong correlation between pesticide residue exposure and disease were to emerge, it is likely that results from a variety of related epidemiological studies would share similar findings, but evidence does not indicate that this is the case. For example, in studies of the relationship between organophosphate pesticide exposure and childhood intelligence, studies looking at general pesticide exposure from food residues found no consistent link. The only positive links were from studies looking at exposure from indoor pesticide use. Considering the conflicting evidence from these epidemiological studies as well as from classical toxicological risk assessments for the organophosphate pesticides examined, the overall weight-of-evidence would indicate that the link between food residue exposure to organophosphates and child neurodevelopment deficits is far from established.

Finally, it is important to recognize the benefits that pesticides provide for the agricultural sector and consumers. From a public health perspective, pesticides provide a tool that can help control fungi capable of producing mycotoxins of significant health concern.

It is critical that farmers continue to be able to maintain a sustainable pest management regime that includes the judicious use of pesticides. If growers did not prevent the damage the pests can cause, our food supply would be more expensive, less diverse, potentially dangerous, and of lower quality. Food waste is a complex issue and pest management importantly contributes to its management.

Perhaps most importantly, without effective pest control the small sector of our society that still engages in the high-risk business of growing our food would be unable to continue. The farm workers and rural businesses would also be hurt, as would "local growers" in pest-challenging regions.

Crop protection chemicals and biological agents are an important part of what is required to limit pest damage. The EPA and other agencies intensively scrutinize those products for human and environmental safety. That regulatory oversight covers the tools used in both organic and conventional farming. As long as the regulation continues to be science-based, farmers will continue to have the tools they need to provide the food and feed crops on which we all depend.

LITERATURE CITED

Adaskaveg, J. E. and H. Forster. 2009. New developments in postharvest fungicide registrations for edible horticultural crops and use strategies in the United States. Pp. 107–117. In D. Prusky and M. L. Gullino (eds.). *Postharvest Pathology*. Springer International Publishing, New York, New York, https://link.springer.com/chapter/10.1007%2F978-1-4020-8930-5_8 (21 February 2018)

Appell, M., D. Kendra, and M. Trucksess. 2010. *Mycotoxin Prevention and Control in Agriculture*. Oxford University Press, Oxford, England, UK, 336 pp., https://global.oup.com/academic/product/mycotoxin-prevention-and-control-in-agriculture-9780841269903?cc=us&lang=en& (21 February 2018)

Arbuckle, T. E., R. Burnett, D. Cole, K. Teschke, M. Dosemeci, C. Bancej, and J. Zhang. 2002. Predictors of herbicide exposure in farm applicators. *Int Arch Occ Env Hea* 75 (6): 406–414, doi:10.1007/s00420-002-0323-7.

Baudry, J., K. E. Assmann, M. Touvier, B. Allès, L. Seconda, P. Latino-Martel, K. Ezzedine, P. Galan, S. Hercberg, D. Lairon, and E. Kesse-Guyot. 2018. Association of frequency of organic food consumption with cancer risk: Findings from the nutrinet-santé prospective cohort study. *JAMA Intern Med* 178 (12): 1597–1606, doi: 10.1001/jamainternmed.2018.4357.

Baugher, T. A., H. W. Hogmire Jr., and G. W. Lightner. 1990. Determining apple packout losses and impact on profitability. *Appl Agr Res* 5 (4): 343–349, http://agris.fao.org/agris-search/search.do?recordID=US9107167 (21 February 2018)

Berkowitz, G. S., J. Obel, E. Deych, R. Lapinski, J. Godbold, Z. Liu, P. J. Landrigan, and M. S. Wolff. 2003. Exposure to indoor pesticides during pregnancy in a multiethnic, urban cohort. *Environ Health Persp* 111 (1): 79–84.

Bolda, M. P., L. Tourte, J. Murdock, and D. A. Sumner. 2016. *Sample Costs to Produce and Harvest Strawberries*. University of California Agriculture and Natural Resources, Cooperative Extension and Agricultural Issues Center, and UC–Davis Department of Agricultural and Resource Economics, https://coststudyfiles.ucdavis.edu/uploads/cs_public/e7/6d/e76dceb8-f0f5-4b60-bcb8-76b88d57e272/strawberrycentralcoast-2016-final2-5-1-2017.pdf (20 February 2018)

Bouchard, M. F., J. Chevrier, K. G. Harley, K. Kogut, M. Vedar, N. Calderon, C. Trujillo, C. Johnson, A. Bradman, D. B. Barr, and B. Eskenazi. 2011. Prenatal exposure to organophosphate pesticides and IQ in 7-year-old children. *Environ Health Persp* 119 (8): 1189–1195, doi:10.1289/ehp.1003185.

Burns, C. J. and T. P. Pastoor. 2018. Pyrethroid epidemiology: A quality-based review. *Crit Rev Toxicol* 48 (4): 297–311, doi:10.1080/10408444.2017.1423463.

Burns, C. J., L. J. McIntosh, P. J. Mink, A. M. Jurek, and A. A. Li. 2013. Pesticide exposure and neurodevelopmental outcomes: Review of the epidemiologic and animal studies. *J Toxicol Env Heal B* 16 (3–4): 127–283, doi:10.1080/10937404.2013.783383.

Cartier, C., C. Warembourg, G. Le Maner-Idrissi, A. Lacroix, F. Rouget, C. Monfort, G. Limon, G. Durand, D. Saint-Amour, S. Cordier, and C. Chevrier. 2016. Organophosphate insecticide metabolites in prenatal and childhood urine samples and intelligence scores at 6 years of age: Results from the Mother-Child PELAGIE Cohort (France). *Environ Health Persp* 124 (5): 674–680, doi:10.1289/ehp.1409472.

Castelan, F. P., C. Abadie, O. Hubert, Y. Chilin-Charles, L. de Lapeyre de Bellaire, and M. Chillet. 2013. Relation between the severity of Sigatoka disease and banana quality characterized by pomological traits and fruit green life. *Crop Prot* 50:61–65, http://www.sciencedirect.com/science/article/pii/S0261219413000665 (21 February 2018)

Castorina, R., A. Bradman, L. Fenster, D. B. Barr, R. Bravo, M. G. Vedar, M. E. Harnly, T. E. McKone, E. A. Eisen, and B. Eskenazi. 2010. Comparison of current-use pesticide and other toxicant urinary metabolite levels among pregnant women in the CHAMACOS cohort and NHANES. *Environ Health Persp* 118 (6): 856–863, doi:10.1289/ehp.0901568.

Centers for Disease Control and Prevention (CDC). 2015. *Fourth Report on Human Exposure to Environmental Chemicals, Updated Tables*. U.S. Department of Health and Human Services, Atlanta, Georgia.

Creamer, N., and L. K. Johnson. 2017. Introducing farm-level loss into the food waste discussion. The Blog, *HuffPost*, https://www.huffingtonpost.com/nancy-creamer/introducing-farm-level-lo_b_13941104.html (21 February 2018)

CropLife Foundation. 2009. *Executive Summary The Value Of Insecticides In U.S. Crop Production*https://croplifefoundation.org/wp-content/uploads/2016/11/insecticide-benefits-execsum.pdf. (12 June 2019.)

CropLife Foundation. 2016. *Insecticides*. Data Appendix. Appendix A-1: Insecticide Use, Cost and Impact Estimates by Crop and State, https://croplifefoundation.org/wp-content/uploads/2016/11/insecticides_data_appendix.pdf (21 February 2018)

Donauer, S., M. Altaye, Y. Xu, H. Sucharew, P. Succop, A. M. Calafat, J. C. Khoury, B. Lanphear, and K. Yolton. 2016. An observational study to evaluate associations between low-level gestational exposure to organophosphate pesticides and cognition during early childhood. *Am J Epidemiol* 184 (5): 410–418, doi:10.1093/aje/kwv447.

Dorn, S., P. Schumacher, C. Abivardi, and R. Meyhöfer. 1999. Global and regional pest insects and their antagonists in orchards: Spatial dynamics. *Agr Ecosyst Environ* 73 (2): 111–118, http://www.sciencedirect.com/science/article/pii/S0167880999000201 (20 February 2018)

Engel, S. M., J. Wetmur, J. Chen, C. Zhu, D. B. Barr, R. L. Canfield, and M. S. Wolff. 2011. Prenatal exposure to organophosphates, paraoxonase 1, and cognitive development in childhood. *Environ Health Persp* 119 (8): 1182–1188, doi:10.1289/ehp.1003183.

Environmental Working Group (EWG). (2018). *EWG's 2018 Shopper's Guide to Pesticides in Produce*. Environmental Working Group, Washington, D.C., https://www.ewg.org/foodnews/ (10 December 2018)

Feng, C., J. C. Correll, K. E. Kammeijer, and S. T. Koike. 2014. Identification of new races and deviating strains of the spinach downy mildew pathogen *Peronospora farinose f.sp. spinaciae*. *Plant Dis* 98 (1): 145–152, https://apsjournals.apsnet.org/doi/abs/10.1094/PDIS-04-13-0435-RE (20 February 2018)

Field to Market. 2016. *National Indicators Report*, https://fieldtomarket.org/national-indicators-report-2016/ (20 February 2018)

Fortenberry, G. Z., J. D. Meeker, B. N. Sánchez, D. B. Barr, P. Panuwet, D. Bellinger, L. Schnaas, M. Solano-González, A. S. Ettinger, M. Hernandez-Avila, H. Hu, and M. M. Tellez-Rojo. 2014. Urinary 3,5,6-trichloro-2-pyridinol (TCPY) in pregnant women from Mexico City: Distribution, temporal variability, and relationship with child attention and hyperactivity. *Int J Hyg Envir Heal* 217 (2–3): 405–412, doi:10.1016/j.ijheh.2013.07.018.

Fryar, C.D., T. Chen, and X. Li. 2012. Prevalence of Uncontrolled Risk Factors for Cardiovascular Disease: United States, 1999–2010. NCHS Data Brief, No. 103. National Center for Health Statistics, Centers for Disease Control and Prevention, U.S. Department of Health and Human Services, Hyattsville, Maryland.

Gianessi, L. P. 2008. The value of herbicides in U.S. crop protection. Crop Protection Research Institute, CropLife Foundation, Washington, D.C., http://www.cwss.org/uploaded/media_pdf/4512-24_2008.pdf (20 February 2018)

González-Alzaga, B., M. Lacasaña, C. Aguilar-Garduño, M. Rodríguez-Barranco, F. Ballester, M. Rebagliato, and A. F. Hernández. 2014. A systematic review of neurodevelopmental effects of prenatal and postnatal organophosphate pesticide exposure. *Toxicol Lett* 230 (2): 104–121, doi:10.1016/j.toxlet.2013.11.019.

Hammond, B. G, K. W. Campbell, C. D. Pilcher, T. A. Degooyer, A. E. Robinson, B. L. McMillen, S. M. Spangler, S. G. Riordan, L. G. Rice, and J. L. Richard. 2004. Lower Fumonisin Mycotoxin Levels in the Grain of Bt Corn Grown in the United States in 2000−2002. *J Agric Food Chem* 52(5): 1390–1397.

Health Canada. 2010. *Report on Human Biomonitoring of Environmental Chemicals in Canada*. Canadian Health Measures Survey, https://www.canada.ca/en/health-canada/services/environmental-workplace-health/reports-publications/environmental-contaminants/report-human-biomonitoring-environmental-chemicals-canada-health-canada-2010.html (13 March 2019).

Hell, K., C. Mutegi, and P. Fandohan. 2010. Aflatoxin control and prevention strategies in maize for Sub-Saharan Africa. Pp. 534–541. In *Proceedings of the 10th International Working Conference on Stored Product Protection*, 27 June–2 July, Estoril, Portugal.

Hill, A. B. 1965. The environment and disease: association or causation? *Proc R Soc Med* 58:295–300

Hopkins, M. 2017. Seed Treatment Q&A: Insight from Iowa State's Alison Robertson. CropLife, http://www.croplife.com/crop-inputs/seed-biotech/seed-treatment-qa-insight-from-iowa-states-alison-robertson/ (20 February 2018)

Huang, Y., I. Edirisinghe, and B. M. Burton-Freeman. 2016. Low income shoppers and fruits and vegetables: What do they think? *Nutr Today* 51 (5): 242–250.

Imai, K., J. Yoshinaga, M. Yoshikane, H. Shiraishi, M. N. Mieno, M. Yoshiike, S. Nozawa, and T. Iwamoto. 2014. Pyrethroid insecticide exposure and semen quality of young Japanese men. *Reprod Toxicol* 43:38–44, doi:10.1016/j.

reprotox.2013.10.010.

Ji, G., Y. Xia, A. Gu, X. Shi, Y. Long, L. Song, S. Wang, and X. Wang. 2011. Effects of non-occupational environmental exposure to pyrethroids on semen quality and sperm DNA integrity in Chinese men. *Reprod Toxicol* 31 (2): 171–176, doi:10.1016/j.reprotox.2010.10.005.

Jusko, T. A., M. A. van den Dries, A, Pronk, P. A. Shaw, M. Guxens, S. Spaan, V. W. Jaddoe, H. Tiemeier, and M. P. Longnecker. 2019. Organophosphate Pesticide Metabolite Concentrations in Urine during Pregnancy and Offspring Nonverbal IQ at Age 6 Years. *Environ Health Perspect* 127 (1): 17007.

Katz, J. M. and C. K. Winter. 2009. Comparison of pesticide exposure from consumption of domestic and imported fruits and vegetables. *Food Chem Toxicol* 47:335–338.

Koureas, M., A. Tsakalof, A. Tsatsakis, and C. Hadjichristodoulou. 2012. Systematic review of biomonitoring studies to determine the association between exposure to organophosphorus and pyrethroid insecticides and human health outcomes. *Toxicol Lett* 210 (2): 155–168, doi:10.1016/j.toxlet.2011.10.007.

LaKind, J. S., C. J. Burns, D. Q. Naiman, C. O'Mahony, G. Vilone, A. J. Burns, and J. S. Naiman. 2017. Critical and systematic evaluation of data for estimating human exposures to 2,4-dichlorophenoxyacetic acid (2,4-D)—Quality and generalizability. *J Toxicol Env Heal B* 20 (8): 423–446, doi:10.1080/10937404.2017.1396704.

Legleiter, T., B. Johnson, T. Jordan, and K. Gibson. 2012. *Terminating Cover Crops—Successful Cover Crop Termination with Herbicides*. Publication WS-50-W, Purdue Weed Science, Purdue Extension, Purdue University, Lafayette, Indiana. 6 pp., https://www.extension.purdue.edu/extmedia/ws/ws-50-w.pdf (21 February 2018)

Li, A. A., K. A. Lowe, L. J. McIntosh, and P. J. Mink. 2012. Evaluation of epidemiology and animal data for risk assessment: Chlorpyrifos developmental neurobehavioral outcomes. *J Toxicol Env Heal B* 15 (2): 109–184, doi:10.1080/10937404.2012.645142.

Mandel, J. S., B. H. Alexander, B. A. Baker, J. Acquavella, P. Chapman, and R. Honeycutt. 2005. Biomonitoring for farm families in the farm family exposure study. *Scand J Work Env Hea* 31:98–104.

Meeker, J. D., D. B. Barr, and R. Hauser. 2008. Human semen quality and sperm DNA damage in relation to urinary metabolites of pyrethroid insecticides. *Hum Reprod* 23 (8): 1932–1940, doi:10.1093/humrep/den242.

Ministry of Agriculture, Food, and Rural Affairs. 2016. Orchard network. *Ontario*, http://www.omafra.gov.on.ca/english/crops/hort/news/orchnews/2016/on-0816a5.htm (21 February 2018)

Mink, P. J., C. A. Kimmel, and A. A. Li. 2012. Potential effects of chlorpyrifos on fetal growth outcomes: Implications for risk assessment. *J Toxicol Env Heal B* 15 (4): 281–316, doi:10.1080/10937404.2012.672150.

Muñoz-Quezada, M. T., B. A. Lucero, D. B. Barr, K. Steenland, K. Levy, P. B. Ryan, V. Iglesias, S. Alvarado, C. Concha, E. Rojas, and C. Vega. 2013. Neurodevelopmental effects in children associated with exposure to organophosphate pesticides: A systematic review. *Neurotoxicology* 39:158–168, doi:10.1016/j.neuro.2013.09.003.

Needham, L. L. 2005. Assessing exposure to organophosphorus pesticides by biomonitoring in epidemiologic studies of birth outcomes. *Environ Health Persp* 113 (4): 494–498, doi:10.1289/ehp.7490.

Nielsen, B. and D. Colville. 2014. Stalk lodging in corn: Guidelines for preventive management. AY262. *Agron Guide*. Cooperative Extension Service, Purdue University, West Lafayette, Indiana, https://www.extension.purdue.edu/extmedia/AY/AY-262.html (21 February 2018)

Oerke, E.-C. and H.-W. Dehne. 2004. Safeguarding production—Losses in major crops and the role of crop protection. *Crop Prot* 23 (4): 275–285, http://www.sciencedirect.com/science/article/pii/S0261219403002540 (21 February 2018)

Osbourn, A. E. 1996. Preformed antimicrobial compounds and plant defense against fungal attack. *Plant Cell* 8 (10): 1821–1831, doi:10.1105/tpc.8.10.1821, https://www.ncbi.nlm.nih.gov/pmc/articles/PMC161317/ (20 February 2018)

Perera, F. P., S. M. Illman, P. L. Kinney, R. M. Whyatt, E. A. Kelvin, P. Shepard, D. Evans, M. Fullilove, J. Ford, R. L. Miller, I. H. Meyer, and V. A. Rauh. 2002. The challenge of preventing environmentally related disease in young children: Community-based research in New York City. *Environ Health Persp* 110 (2): 197–204.

Perry, M. J., S. A. Venners, D. B. Barr, and X. Xu. 2007. Environmental pyrethroid and organophosphorus insecticide exposures and sperm concentration. *Reprod Toxicol* 23 (1): 113–118, doi:10.1016/j.reprotox.2006.08.005.

Prueitt, R. L., J. E. Goodman, L. A. Bailey, and L. R. Rhomberg. 2011. Hypothesis-based weight-of-evidence evaluation of the neurodevelopmental effects of chlorpyrifos. *Crit Rev Toxicol* 41 (10): 822–903, doi:10.3109/10408444.2011.616877.

Radwan, M., J. Jurewicz, B. Wielgomas, W. Sobala, M. Piskunowicz, P. Radwan, and W. Hanke. 2014. Semen quality and the level of reproductive hormones after environmental exposure to pyrethroids. *J Occup Environ Med* 56 (11): 1113–1119, doi:10.1097/JOM.0000000000000297.

Rauh, V., S. Arunajadai, M. Horton, F. Perera, L. Hoepner, D. B. Barr, and R. Whyatt. 2011. Seven-year neurodevelopmental scores and prenatal exposure to chlorpyrifos, a common agricultural pesticide. *Environ Health Persp* 119 (8): 1196–1201, doi:10.1289/ehp.1003160.

Reiss, R., E. T. Chang, R. J. Richardson, and M. Goodman. 2015. A review of epidemiologic studies of low-level exposures to organophosphorus insecticides in non-occupational populations. *Crit Rev Toxicol* 45 (7): 531–641, doi:10.3109/10408444.2015.1043976.

Savage, S. 2013. Five ways farmers control pests other than with pesticides. *Applied Mythology* blog, http://appliedmythology.blogspot.com/2013/12/five-ways-farmers-control-pests-other.html (20 February 2018)

Savage, S. 2014. An example of how much pesticides have changed. *Applied Mythology* blog, http://appliedmythology.blogspot.com/2014/01/an-example-of-how-much-pesticides-have.html (20 February 2018)

Schuchat, A. and C. V. Broome. 1991. Toxic shock syndrome and tampons. *Epidemiol Rev* 13:99–112.

Streissguth, A. P. 1978. Fetal alcohol syndrome: An epidemiologic perspective. *Am J Epidemiol* 107 (6): 467–478.

Sudakin, D.L, and D. L. Stone. 2011 Dialkyl phosphates as biomarkers of organophosphates: the current divide between epidemiology and clinical toxicology. *Clin Toxicol (Phila)* 49(9):771-781.

Tedford, E. and R. A. Brown. 2000. Integrating new fungicide technologies in IPM. In G. G. Kennedy and T. B. Sutton (eds.). *Emerging Technologies for Integrated Pest Management: Concepts, Research, and Implementation*. The American Phytopathological Society, St. Paul, Minnesota. 526 pp.

Thomas, K. W., M. Dosemeci, J. A. Hoppin, L. S. Sheldon, C. W. Croghan, S. M. Gordon, M. L. Jones, S. J. Reynolds, J. H. Raymer, G. G. Akland, C. F. Lynch, C. E. Knott, D. P. Sandler, A. E. Blair, and M. C. Alavanja. 2010. Urinary biomarker, dermal, and air measurement results for 2,4-D and chlorpyrifos farm applicators in the Agricultural Health Study. *J Expo Sci Env Epid* 20 (2): 119–134, doi:10.1038/jes.2009.6.

Tilman, D., C. Balzer, J. Hill, and B. L. Befort. 2011. Global food demand and the sustainable intensification of agriculture. *P Natl Acad Sci USA* 108 (50): 20260–20264, doi:10.1073/pnas.1116437108, http://www.pnas.org/content/108/50/20260.short (20 February 2018)

Toshima, H., Y. Suzuki, K. Imai, J. Yoshinaga, H. Shiraishi, Y. Mizumoto, S. Hatakeyama, C. Onohara, and S. Tokuoka. 2012. Endocrine disrupting chemicals in urine of Japanese male partners of subfertile couples: A pilot study on exposure and semen quality. *Int J Hyg Envir Heal* 215 (5): 502–506, doi:10.1016/j.ijheh.2011.09.005.

University of California, Davis. 2019. Current Cost and Return Studies. https://coststudies.ucdavis.edu/en/current/ (12 June 2019).

United State Environmental Protection Agency (USEPA). 2011. Permethrin. https://iaspub.epa.gov/apex/pesticides/f?p=CHEMICALSEARCH:31:::NO:1,3,31,7,12,25:P3_XCHEMICAL_ID:3260. (Accessed 14 June 2019.)

U.S. Department of Agriculture (USDA). 2017. Saving money, time and soil: The economics of no-till farming, https://www.usda.gov/media/blog/2017/11/30/saving-money-time-and-soil-economics-no-till-farming (20 February 2018)

U.S. Food and Drug Administration (USFDA). 2018. *Pesticide Residue Monitoring Program Fiscal Year 2016 Pesticide Repor*t. U.S. Food and Drug Administration, https://www.fda.gov/downloads/Food/FoodborneIllnessContaminants/Pesticides/UCM618373.pdf (10 December 2018)

Viswanathan, M., K. A. Treiman, J. Kish-Doto, J. C. Middleton, E. L. Coker-Schwimmer, and W. K. Nicholson. 2017. Folic acid supplementation for the prevention of neural tube defects: An updated evidence report and systematic review for the US Preventive Services Task Force. *JAMA* 317 (2): 190–203, doi:10.1001/jama.2016.19193.

Wallace, J. S. 2000. Increasing agricultural water use efficiency to meet future food production. *Agr Ecosyst Environ* 82 (1–3):105–119, http://www.sciencedirect.com/science/article/pii/

S0167880900002206 (20 February 2018)

Walsh, D. B., M. P. Bolda, R. E. Goodhue, A. J. Dreves, J. Lee, D. J. Bruck, V. M. Walton, S. D. O'Neal, and F. G. Zalom. 2011. *Drosophila suzukii*: Invasive pest of ripening soft fruit expanding its geographic range and damage potential. *J IPM* 2 (1): G1–G7, https://doi.org/10.1603/IPM10010 (20 February 2018)

Whyatt, R. M., D. Camann, F. P. Perera, V. A. Rauh, D. Tang, P. L. Kinney, R. Garfinkel, H. Andrews, L. Hoepner, and D. B. Barr. 2005. Biomarkers in assessing residential insecticide exposures during pregnancy and effects on fetal growth. *Toxicol Appl Pharm* 206 (2): 246–254, doi:10.1016/j.taap.2004.11.027.

Winter, C. K. 1992a. Pesticide tolerances and their relevance as safety standards. *Regul Toxicol Pharm* 15:137–150.

Winter, C. K. 1992b. Dietary pesticide risk assessment. *Rev Environ Contam T* 127:23–67.

Winter, C. K. 2005. Pesticide residues in fruits and vegetables. Pp. 135–155. In W. Jongen (ed.). *Improving the Safety of Fresh Fruits and Vegetables*. Woodhead Publishing and CRC Press, London, England.

Winter, C. K. 2015. Chronic dietary exposure to pesticide residues in the United States. *Int J Food Contam*, doi:10.1186/s40550-150-0018y.

Winter, C. K. and F. J. Francis. 1997. Assessing, managing, and communicating chemical food risks. *Food Technol* 51 (5): 85–92.

Winter, C. K. and E. A. Jara. 2015. Pesticide food safety standards as companions to tolerances and maximum residue limits. *J Integr Agr* 14 (11): 2358–2364.

Winter, C. K. and J. M. Katz. 2011. Dietary exposure to pesticide residues from commodities alleged to contain the highest contamination levels. *J Toxicol*, doi:10.1155/2011/589674.

Xia, Y., Y. Han, B. Wu, S. Wang, A. Gu, N. Lu, J. Bo, L. Song, N. Jin, and X. Wang. 2008. The relation between urinary metabolite of pyrethroid insecticides and semen quality in humans. Fertil Steril 89 (6): 1743–1750, doi:10.1016/j.fertnstert.2007.05.049.

The mission of the Council for Agricultural Science and Technology (CAST): *CAST, through its network of experts, assembles, interprets, and communicates credible, balanced, science-based information to policymakers, the media, the private sector, and the public. The vision of CAST is a world where decision making related to agriculture and natural resources is based on credible information developed through reason, science, and consensus building. CAST is a nonprofit organization composed of scientific societies and many individual, student, company, nonprofit, and associate society members. CAST's Board is composed of representatives of the scientific societies, commercial companies, nonprofit or trade organizations, and a Board of Directors. CAST was established in 1972 as a result of a meeting sponsored in 1970 by the National Academy of Sciences, National Research Council.* ISSN 1070-0021

Additional copies of this Issue Paper are available from CAST, http://www.cast-science.org.

Citation: Council for Agricultural Science and Technology (CAST). 2019. *Interpreting Pesticide Residues in Food.* Issue Paper 66. CAST, Ames, Iowa

CAST®

The Science Source for Food,
Agricultural, and Environmental Issues

4420 West Lincoln Way
Ames, Iowa 50014-3447, USA
(515) 292-2125
E-mail: cast@cast-science.org
Web: www.cast-science.org