

PESTICIDES *in* PERSPECTIVE

A Position Paper of the



**AMERICAN COUNCIL
ON SCIENCE AND HEALTH**

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Based on a paper by

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Executive Summary

Year in and year out, agricultural pesticides have been the subject of considerable fear-mongering, leaving the typical consumer with the impression that these chemicals taint much of our food supply and are harmful to human health. In fact, just the opposite is closer to the truth. The published scholarly literature has failed to turn up evidence of adverse human health effects from use of modern pesticides in the real world. Furthermore, in light of the current economic perturbations, as well as the progressive severity of worldwide food shortages and the resulting malnutrition and spiking prices of basic food commodities, the claims that these pesticides pose a threat to human health are false, misleading—and dangerously irresponsible.

Pesticides have boosted crop yields. They are also highly cost-effective. Pesticides are, in fact, our number one technology for protecting the nation's crops. And pesticide protection bears not only on farm output but also on public health, because pesticides are a buffer against naturally occurring toxins. For example, insect-induced damage to crops makes them more susceptible to actually dangerous fungal invasion.

The non-use of pesticides thus poses risks in and of itself—risks that extend to public health. Perhaps no better example of shortsighted regulation exists than the ban on DDT. Anopheles mosquitoes transmit well over 300 million cases of malaria a year, resulting in the deaths of approximately 800,000 children in the poorer countries of the world.

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Over the past 30 years, new chemistries have been introduced to narrow the spectrum of pesticide activity, making the chemicals more pest-specific and less toxic to mammals, birds, fish, and aquatic invertebrates. The vast majority of all foods, moreover, have no detectable pesticide residues.

So, why all the concern? The difficulty stems largely from a misinterpretation—to a large extent intentional by anti-chemical activists and sensation-hungry media—of various laboratory studies of toxicological mechanisms, as well as a corresponding failure to appreciate how agricultural chemicals are actually used.

Introduction

Most of us take our daily bread for granted, giving little thought to the multiple inputs that go into producing it. Farming has been able to keep pace with population growth over these many years, thanks largely to technological advances in agricultural chemicals, as well as patient hybrid cross-breeding. Innovations in fertilizers and pesticides, in particular, have enhanced food availability, with consequential benefits for nutrition and health.

Washington has, all the while, kept a watchful eye on the development and use of agricultural chemicals. Federal laws and regulations—especially those governing pesticides—have evolved over time and are now, arguably, the most precautionary of all congressional mandates involving technology. The three statutory pillars of agricultural chemical regulation are the Federal Food, Drug, and Cosmetic Act of 1938; the Federal Insecticide, Fungicide and Rodenticide Act of 1947; and the Food Quality Protection Act of 1996.

Despite such intense governmental oversight, agricultural pesticides have become subject to considerable fear-mongering. The many tirades against pesticides have left the perception that these chemicals now taint much of our food supply and are harmful to human health. In hopes of shedding new light on the topic, this report specifically examines the claims regarding four types of commonly used pesticides: atrazine, chlorpyrifos, pyrethroids, and glyphosate. In each case, we find that the published scholarly literature has failed to turn up evidence of adverse human health effects from use of modern

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pesticides in the real world. Furthermore, the claims that these pesticides pose a danger to human health are both false and irresponsible, and the public perception of health effects stemming from the use of these chemicals is also misguided and baseless.

The Benefits of Pesticide Use

Agricultural pesticides and fertilizers are nothing new. Farmers have been using them since ancient times. The reason is simple: Pesticides and fertilizers help to boost farm output. Pesticides, in particular, raise production efficiency and yields, reduce the cost of food, and increase both the quality and quantity of fruits and vegetables. In addition, pesticides tend to reduce losses during transport and storage, aid in soil conservation, and help to ensure a stable and predictable food supply.

While pesticide use in modern farming may be widespread, not all pesticides are the same, and the uses to which they are put are similarly varied. In 2002, for instance, over 300 million acres of crops were harvested in the United States, and roughly 95 percent of the acreage was treated with some type of pesticide. The most widely employed chemicals were herbicides used to control weeds; these covered 64 percent of total acreage. Insecticides ranked second, with treatment extending to 22 percent of U.S. acreage. Next came fungicides and nematicides, which were used on 6 percent of the nation's farmland to control fungal infection and nematodes (a type of crop worm). And, finally, plant-growth regulators were employed on an additional 4 percent of crop acreage for fruit thinning, growth control, or defoliation.

**Percentage Use of Pesticide Classes
on Major Crops During Crop Years 2003/2004**

Crop	Herbicide	Insecticide	Fungicide
Corn	95	29	<1
Soybean	97	4	1
Wheat	45	7	2
Cotton	98	64	7
Potato	91	84	91
Apple	42	94	90

Historical trend in U.S. corn production and approximate timeline for introduction of crop-production technologies.

These numbers speak to another important point—namely, that farmers do not use agricultural chemicals indiscriminately, as is sometimes charged. Rather, the data show that the use of crop protection chemicals with different activities and spectra of targets (i.e., herbicides, insecticides, fungicides, etc.) is tied to specific farming needs. Some crops require disproportionately more herbicide use, and some crops require more insecticide or fungicide use. Grains, for example, tend to be disproportionately treated with herbicides, while fruit and vegetables receive mostly insecticide and fungicide applications.

Crop productivity has improved immensely over time. Yet in trying to pinpoint the sources of the improvement, it is difficult to separate the benefits of crop-protection chemicals from the effects of hybrid-seed technology and other plant-breeding advances. Indeed, an examination of crop yields relative to land under production reveals that both of these technologies have made major contributions. Take corn, for example. The greatest proportion of U.S. farmland is devoted to corn production. A historical examination of acreage, yields, and the introduction of different technologies suggests that insect control (i.e., mainly of the corn rootworm complex) greatly enhanced the effectiveness of hybrid-seed technology. Additionally, the introduction of

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modern synthetic herbicides facilitated the widespread adoption of conservation tillage in the Corn Belt, and this, in turn, greatly reduced soil erosion and sedimentation in rivers—two major problems in agriculture.

Perhaps an even more compelling case for the role of crop-protection chemicals (especially fungicides and fumigants) in crop-production efficiency is suggested by the statistics for potato production. In 1900, nearly 3 million acres of potatoes were harvested in the U.S., yielding an average of 52 hundredweight per acre (cwt/acre). In 1950, average yields were 153 cwt/acre. In crop year 2004, 1.2 million acres of harvested potatoes yielded an average 752 cwt/acre. Surely, advances in plant breeding played an important role in the production increases. However, by the 1950s, fumigants for control of nematodes became widely available—virtually coinciding with the widespread adoption of mineralized fertilizers. The results have been startling. Yields have increased so vastly, in fact, that just 40 percent of the total potato acreage planted in 1900 could produce seven times more potatoes in 2004.

Pesticides are also highly cost-effective. One recent estimate (for crop year 2002) finds that pesticide purchases represent just 4.4 percent of total farm expenses. This compares with the 12.7 percent of expenses spent to hire outside labor.

Importantly, the pesticides used during the first half of the 20th century, as well as the first generation of synthesized pesticides after 1950, were generally broad spectrum but were not necessarily adequate for all cropping systems. Over the past 40 years, new chemistries have been introduced to narrow the spectrum of activity. The discoveries of chemicals with completely different modes of action resulted in the manufacture of new reduced-risk pesticides. Since the late 1980s, these reduced-risk chemicals have been used at rates lower than those of the pesticides they replaced, and this new generation of pesticides has also proved less toxic to mammals, birds, fish, and aquatic invertebrates. Similarly, insecticides introduced over the last 15 years are also much less toxic to the natural biocontrol organisms than the broad-spectrum synthetics introduced during the 1950s.

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Furthermore, modern pesticides are rapidly biodegradable in the environment and do not bioaccumulate in fatty tissues, as did the chlorinated hydrocarbons and some other types of pesticides that were heavily used prior to their ban in the early 1970s. In sum, along with new formulations and application methods, modern pesticides can be better tailored to specific crop pest problems.

The Risks of Forgoing Pesticides

Despite these technological improvements, public opinion surveys point to a pervasive misunderstanding about the nature of pesticides.

The misunderstanding starts with the fact that most consumers are not well-informed about the need for agricultural chemicals.

Pesticides are, in fact, our number one technology for protecting the nation's crops. If pesticides were not in use, the effects on crop production would be disruptive to food supplies in our country and Europe—but disastrous in areas of borderline or inadequate nutrition. According to one estimate, production losses here during the mid-1980s would have been as high as 37 percent of total output. Another economic analysis has projected the effect on vegetable and fruit yields if pesticide use were cut by half: subsequent reductions in vegetable and fruit yields were put at 40 percent. And yet another study has predicted substantial reductions in grain production under conditions of no herbicide use. Consequences of such a decline would include food shortages, starvation in the less-developed world, and spiking food prices everywhere.

Pesticide protection bears not only on farm output but also on public health, because pesticides are a buffer against naturally occurring toxins.

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Insect feeding, for example, causes damage that makes a food more susceptible to fungal invasion. Certain fungi commonly associated with crops produce mycotoxins that have well-documented physiological effects in mammals, including humans. The non-use of pesticides thus poses risks in and of itself—risks that extend to public health.

Perhaps no better example of shortsighted regulation exists than the ban on DDT.

Anopheles mosquitoes today still transmit over 300 million cases of malaria each year. Malaria causes daily paroxysms of an excruciating fever and, because infant and child nutrition is so deficient in the countries where malaria is endemic, a nearly unimaginable 800,000 children die from mosquito-transmitted malaria each year.

Many countries—mostly in the developed world—have malaria under control, thanks to the use of DDT to control mosquito populations between the Second World War and our EPA's 1972 ban. Data on malaria incidence before and after the use of DDT proved its effectiveness without causing harm—neither to people nor to the environment.

Nevertheless, DDT was essentially banned when the EPA decided to suspend its registration for any agricultural use. DDT, along with other pesticides, came under implacable attack in marine biologist Rachel Carson's influential book *Silent Spring*, first published in 1962. Most of her charges were unfounded—yet such is the myth of DDT that a number of countries continue to avoid the pesticide's use, with devastating consequences for the toll of malaria. To this day, unfortunately, much of the public does not understand how DDT is actually used, and thus visions of a "silent spring," in which birds no longer sing, dominate the conversation.

The Myth of “Organic Farming”

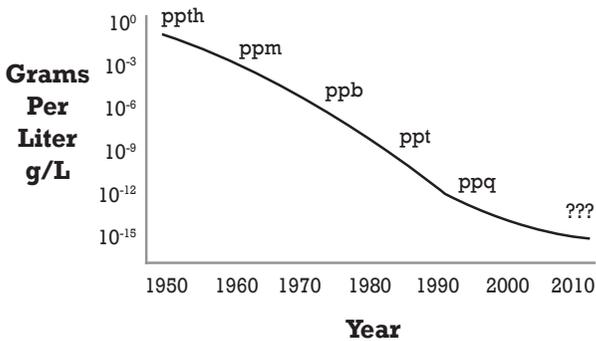
A corollary to the exaggerated fear of pesticides has been the rise in popularity of so-called organically grown produce. Perhaps the most popular belief among organic food adherents is that they lack pesticide residues and other additives. The basis for this belief is the often-repeated argument that organic agriculture distinguishes itself from conventional production methods because no synthetic pesticides are used. Prolonged pronouncements of “no synthetic pesticide use” easily evolve into a perception of “no pesticide use.”

The reality is considerably different, however. U.S. rules for certification of organic production allow for the use of approved crop protection products. Under the Federal Insecticide Fungicide and Rodenticide Act (FIFRA), many of these approved products are, in fact, pesticides and must be registered with the EPA. Organic growers, by rule, cannot use synthetic materials unless approved by the National Organic Standards Board (NOSB), and certified organic producers have an array of pesticides they can use under the rules of the NOSB.

NOSB contracts with the Organic Materials Research Institute (OMRI) to do a comprehensive hazard assessment of materials proposed for certified organic production. The EPA also conducts a comprehensive risk assessment

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Analytical Technology Has Advanced Faster than Biological Understanding



Depiction of change in detection limits ($1 \text{ ppm} = 10^3 \text{ g/L}$) over the last 60 years of contaminant monitoring. For year 2010, uncertainty is expressed owing to the unreliability of detections reported at parts per quadrillion (ppq) and lack of articles reporting detections lower than 1 part per trillion (ppt).

on all chemicals submitted for registration, using the raw data submitted by a prospective pesticide registrant. Indeed, no pesticide—be it NOSB-approved or otherwise—can be used unless vetted by the EPA first. The EPA and OMRI ask similar questions of a candidate pesticide, except the OMRI speculates as to whether the candidate product is really needed and therefore a credible substitute. Another criterion is whether the product is deemed to be “less hazardous.”

The truth is that some of the active ingredients used by conventional growers are the same as those used by certified organic growers. Take, for example, spinosad insecticide: it is used by today’s cherry growers regardless of their production philosophy. Ironically, the NOSB policy tends to eschew substances that are toxic, yet spinosad is definitively a neurotoxin, though not one as well characterized biochemically as the so-called conventional synthetic pesticides that have been on the market for decades.

Another reality of the purported “purity” of organic food is that analytical surveys of organic commodities reveal they contain residues of synthetic pesticides—both banned and currently registered—albeit at a much lesser frequency than conventional foods. The residues have not resulted from willful pesticide use but are more likely the inadvertent result of airborne transport and deposition, or are soil-borne from past use. Recognizing the ubiquity and mobility of environmental residues, current rules allow inadvertent pesticide residues of up

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to 5 percent of the established Federal tolerance level without a loss of organic certification.

Whether one likes or dislikes pesticide use, past practices continue to influence residues in food. Recent food-residue studies indicate, however, that the vast majority of conventional foods have no detectable pesticide residues.

Toxicity, Hazard, and Risk

Consumers ultimately want to know whether any given technology is safe. Among regulatory toxicologists, however, the concept of chemical safety is understood not as a hard-and-fast quantitative measure but rather as a probability—the probability of a reasonable certainty of no harm. The validity of the concept of a “reasonable certainty of no harm” depends on drawing a distinction among the terms *toxicity*, *hazard*, and *risk*. Distinguishing these terms is indeed crucial to any rational discussion of pesticide use in modern society.

Toxicity is an inherent property of both a particular molecule and any enzymes or receptors that it can react to or interact with. Such interaction results in a physiological reaction that could be harmful to an organism’s survival, and thus the substrate would be called a toxin. By definition, pesticides are harmful to the lives of pests; thus pesticides are toxins. The three dimensional structure of any toxin must be compatible with the three dimensional structure of an enzyme or receptor for any interaction to occur. Such interactions occur very readily if the structures of a toxin and its receptor are highly compatible. In such cases, the substance is considered highly potent. Interactions would occur with structures not as compatible only under conditions

of inordinately high toxin concentrations—typically those not likely to be found in the environment but certainly possible to create in the lab when animals are tested. A toxin requiring extremely high concentrations to interact with an enzyme or receptor, and thus cause an adverse physiological reaction, would be considered to have low potency. In summary, toxicity is the inherent property of any molecule to cause an adverse physiological response because a given organism has specific enzymes or receptors whose three-dimensional structures are compatible.

The nature of fundamental thermodynamic laws governing chemical interactions prescribes that any molecule could, theoretically, interact with any other molecule. However, the concentration of the two molecules must be sufficiently high for the interaction to have any reasonable probability of occurring. This latter concept leads to the definition of hazard. Hazard describes the potential of a chemical to cause harm under a particular set of conditions. In other words, toxins are not inherently hazardous unless the circumstances are sufficiently conducive to the interaction of the toxin with the particular enzymes or receptors with which it is compatible. To study how chemicals interact with organisms, scientists in the laboratory always create conditions in which the subject chemical will be hazardous. Often, the conditions are concentrations (or doses) of chemical sufficient to cause an observable response in a test population of organisms. If the conditions of the exposure change (for example, using a very low dose), then the hazard may change or simply disappear altogether.

Experiments repeatedly show that natural and synthetic substances at one dose may have no adverse effects on an organism, but at another, higher dose can cause harm. This concept, frequently called the “dose makes the poison,” is the fundamental principle guiding toxicological studies. The phrase, of course, belies more complex interactions between a substance and its effects on an organism. The dose required to cause deleterious effects within a population of organisms can vary, for instance, depending on the route of exposure to the substance (e.g., oral, dermal, or inhalational), the length of time over which it is administered (i.e., acute versus chronic), and the age, sex, and health of an organism. Nevertheless, the appearance or magnitude of an

effect of any substance is tied to its dose. Hazard, therefore, can be thought of as a substance's dose-related array of possible deleterious effects on an organism of a specific age, gender, and health status exposed via oral, dermal, and inhalational pathways.

This all gives rise to a fundamental question: should the knowledge that a substance is hazardous (i.e., potentially harmful under a specific set of circumstances) precipitate a corresponding—and urgent—reaction to do something about it?

Human beings are precautionary by nature. We thus want to be careful when we have knowledge of potential harm. The problem is knowing the appropriate amount of resources—such as time and money—to expend on potential harm. Furthermore, because the degree of hazard depends on variable circumstances, a toxic effect of a hazard may never be manifested under most conditions of chemical use.

To judge just how wary we should be about a toxin in the environment, and thus allocate the appropriate attention to its hazardousness, we have to understand the risk of adverse effect. A simple definition of risk is the probability, or likelihood, that an adverse effect would occur under a specific situation or set of conditions. Often, regulatory toxicologists will express risk as dependent on the product of toxicity (hazardousness) and exposure. Thus, risk is the probability, or likelihood, that the array of known hazards of a substance will actually occur if or when an organism is exposed. If exposure is nonexistent, then the likelihood of an effect is nil. If exposure does occur, then the likelihood of the substance being a hazard is conditioned not only on the dose but also on the age, gender, and health of that organism, as well as the specific route of exposure. The important point is that the risk of adverse effects after exposure to a substance may be low or high depending on all the factors affecting the hazards of that substance.

Low levels of exposure, even to a highly potent toxin, may have a low probability of causing an adverse effect. In any case of exposure, whether through skin contact, inhalation, or oral ingestion, a number of physiological processes occur to modulate the dose or concentration of toxin arriving at the level of the tissue cells and thus the probability of interaction with enzymes or

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receptors. All of these physiological processes that determine what amount of toxin arrives at the site of the cell enzymes or receptors is described by pharmacokinetics. Pharmacokinetic studies examine rate and extent of chemical uptake processes following dermal, oral, and inhalational exposures. The amount of toxin entering into the systemic circulation (i.e., blood) is studied and then followed to its subsequent distribution among all body regions down to the cellular level. The toxin amount changes as it is degraded by enzyme systems common to all cells, or is excreted, sometimes unaltered, from the body. Whatever toxin is left over from all these pharmacokinetic processes arrives at the site of the potential enzymes or receptors compatible enough in structure to have any probability of interaction. The specific interactions are called pharmacodynamics.

A threshold for a physiological or biochemical reaction is found by studying the interaction of toxins with whole animals (i.e., *in vivo* studies) or with tissues, cells, and large molecules (i.e., *in vitro* studies), using a sufficiently wide range of exposure doses or concentrations. The threshold is often reported as an observed dose level at which no adverse effect occurs. This is known as the no observable adverse effect level, or NOAEL. In determining the NOAEL, researchers must expose animals to sufficiently high concentrations to see what a typical response looks like. The lowest dose just causing a reaction is called the lowest observable adverse effects level, or LOAEL.

In summary, the toxicity of a chemical is an inherent property related to its structural ability to interact with some compatible enzyme or receptor in a cell. If the structures are so compatible such that there is a high probability of interaction, then the toxin is considered highly potent, but if there is no such structural alignment, the toxin has low potency. Regardless of potency, all toxins have thresholds for an effect. Pertinently, these interactions and thresholds apply to all chemicals, natural and synthetic, because all are under control of the fundamental laws of thermodynamics and kinetics.

Four Case Studies

Four cases studies of pesticides (including two herbicides)—atrazine, chlorpyrifos, pyrethroids, and glyphosate—that have come under criticism are worthy of consideration.

Atrazine

Atrazine inhibits plant physiology by disrupting photosynthesis, specifically one biochemical pathway called Photosystem II. Animals do not possess biochemical pathways that atrazine could affect at any possible environmental levels. According to the EPA, atrazine is considered to be of low toxicity

In separating the myths and realities of atrazine toxicity, hazards, and risks, knowing that it was first registered in 1958, and thereafter intensively used mainly in corn production throughout the Western world, should put some perspective on reports suggesting that the compound poses a risk to human health.

Atrazine is likely to have worked its way into shallow ground water of the Corn Belt after the first year of use. However, although atrazine residues are now increasingly detected in the environment, this is not because use is increasing: in fact, use has gone down. However, the frequency of reported atrazine detection is a classic story of detection sensitivity improvements, not actually related to an increase in residues entering the environment.

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Analytical technology to detect chemical residues in the environment, including our own bodies, has advanced by orders of magnitude over the last 60 years. Before the introduction of specialized instruments like GC-MS (gas chromatography-mass spectrometry) and LC-MS (liquid chromatography-mass spectrometry) became available to everyone's benchtop, identifying and quantifying environmental residues of chemicals was a slow and imprecise process that basically worked on one chemical entity at a time. Often concentrations of chemicals as large as parts per thousand, or perhaps parts per hundred thousand, could be detected. Today, routine measurements are finding anthropogenic (synthetic) chemicals in the environment at levels of parts per trillion and even less.

Thus, contemporary stories raising alarm about atrazine in water supplies are not plowing new ground but repeating information known for a very long time. The question about residues in water, especially drinking water, generally raises the question of whether humans are adversely affected. The answer to this question rests on understanding atrazine's toxicology and the potential for exposure based on residues found in environments relevant to drinking water.

Atrazine residues are not found in food, given that the chemical is applied directly to the soil of basically only one crop (i.e., corn), so the only pathway of exposure would be drinking water derived ultimately from field runoff or subsurface drainage in the Corn Belt. Thus, a very geographically limited population of consumers is going to be comparatively most exposed if atrazine residues survive water treatment and appear in finished drinking water supplies.

The ongoing, multiyear National Cancer Institute study, called the Agricultural Health Study (AHS), concluded in 2004: "Our analyses did not find any clear associations between atrazine exposure and any cancer analyzed." Significantly, the AHS study includes a cohort of over 70,000 pesticide workers and adult family members in Iowa and North Carolina. Thus, the pesticide users represent farms associated with grain production (i.e., corn and soybeans) and specialty crops (i.e., a diversity of fruit and vegetables grown in the mid Atlantic region).

Further concerns about atrazine adversely affecting human health from chronic exposure were most recently allayed by the World Health Organization's decision to raise the allowable "health protective" atrazine concentration in drinking water.

And even the EPA has again asserted: "Based on all the available test data, the Agency's evaluation, and scientific peer review, atrazine is not likely to be a human carcinogen."

Chlorpyrifos

First commercialized in 1965, chlorpyrifos is an organophosphorus (OP) ester insecticide, and until the FQPA of 1996, it was, arguably, the most widely used insecticide in the world. In 2002, however, its manufacturer, Dow Agro-Sciences, in an agreement with the EPA, voluntarily suspended the chemical's use on developing fruit, as a termiticide, and as a home and lawn insecticide.

Although chlorpyrifos is still registered for some vegetable crops, including corn, its use today is mainly restricted to dormancy-season sprays on pome fruits (e.g., apples and pears) and nuts. (Dormant spraying is conducted when no fruit is growing; thus, the opportunity for exposure through fruit and nut consumption is nil.)

Chlorpyrifos, like other organophosphorus esters, is an inhibitor of nerve transmission. However, chlorpyrifos has to be metabolized to an oxidized form—chlorpyrifos oxon—to have biological activity. Of public health interest, therefore, is whether chlorpyrifos oxon is detected in food. The USDA AMS reported no findings of oxon residues in food, thus confirming the very minimal exposure of human populations to chlorpyrifos. Furthermore, the latest data indicates that only 3 percent of more than 10,000 individual food items analyzed had detectable residues of chlorpyrifos. The levels found were typically 100 to 1,000 times less than the residue tolerance—that is, the legal concentration that has been set in part to protect human health.

Despite the low potential for current exposure to chlorpyrifos residues, the literature is replete with data showing widespread detection of an essentially nontoxic breakdown product called trichloropyridinol in human urine. Thus, the perception that people today are widely exposed to chlorpyrifos

residues is generated from these studies. However, these studies reflect the results from urine samples collected when chlorpyrifos was widely used and before the significant restrictions on its use.

The degree of hazard today is not reflected in the myriad studies putatively indicating widespread exposure because chlorpyrifos residues are only infrequently detected in food, and it is no longer used in urban environments or directly on fruit. Indeed, within two years after removal of chlorpyrifos from urban uses, researchers could detect lower exposures, concluding that aggregate potential doses of chlorpyrifos were well below published acceptable dose values.

Still, any compound that affects any of the nervous system biochemical parameters is considered automatically to be a neurotoxin. As is true for all biochemicals, the probability of causing observable neurotoxic responses to chlorpyrifos and similar chemical compounds depends on dose, or the degree of exposure. When there is proven exposure in a case-controlled epidemiological study, the results do not support a case for hazardousness. If chlorpyrifos were, in fact, a potent neurotoxin in adults, then one would predict severe neurotoxicology problems in those agricultural workers who apply the chemical in the field. Yet, an epidemiological study of 191 applicators whose urine proved they were over 100 times more exposed than a non-applicator control population concluded thus: “The exposed group did not differ significantly from the nonexposed group for any test in the clinical examination. Few significant differences were found in nerve conduction velocity, arm/hand tremor, vibrotactile sensitivity, vision, smell, visual/motor skills, or neurobehavioral skills.”

Pyrethroids

Another insecticide whose use has been called into question are pyrethroids. The term pyrethroid refers to all synthetic versions of insecticidal compounds based on the structure of components of the botanical extract called pyrethrum. *Chrysanthemum cinaerifolium*, one of about 30 species in the genus *Chrysanthemum*, is particularly useful as a source of pyrethrum extracts that become concentrated in the flowers. While gardeners still tell tales of

planting chrysanthemums to ward off insect pests around their gardens, in fact only *C. cinaerifolium* is a useful source of pyrethrum, and it is not commercially produced in the U.S. This particular species is still cultivated commercially for its trove of pyrethrum components, but the geographic foci includes Kenya, Rwanda, Ecuador, and Australia.

Pyrethrum and, more specifically, pyrethrins, which are components of the whole extract, are sold commercially, and one of the few neurotoxic insecticides approved for certified organic agricultural production under the USDA's National Organic Program and various state organic program rules. Pyrethrins can be rapidly acting and extraordinarily toxic to a number of insect pest species yet of extraordinarily low toxicity to mammals. Pyrethrins are quickly metabolized by birds and mammals to non-toxic compounds—and thus, when exposure to them is dermal, as would be typical of environmental or medicinal use, these components are extraordinarily safe yet effective at killing insect pests. Pyrethroids are typically hundreds to thousands of times less toxic to mammals than to insects. Such differences mean that very small amounts of pyrethroids can be deployed to control insects, and these amounts are vastly lower than the amounts that might cause harm to mammals.

Glyphosate

Marketed since about 1975, the herbicide glyphosate itself is best chemically described as a phosphonated amino acid. In fact, part of the structure is glycine, a non-essential amino acid. The only known mechanism of toxicity of glyphosate is through inhibition of the EPSPS enzyme, which is specific only to plant metabolism. Glyphosate has broad-spectrum activity against most plants, and thus it historically has had very limited uses in growing agricultural crops. Furthermore, only young weeds are susceptible to glyphosate at the levels that are allowed for use.

The World Health Organization, the Environmental Protection Agency, and the European Union have extensively reviewed the full range of toxicological information about glyphosate, pronouncing it of extremely low toxicity and thus nil risk when exposure is factored in. In addition to scrutiny by the various regulatory agencies, numerous risk assessments have been published

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in the scholarly literature. These latter publications pertinently consider all the toxicological endpoints that have been defined, and integrate the most sensitive effects with the likely exposures. None have suggested any environmental or human health hazard from the routine sanctioned uses of glyphosate. Indeed, pronouncements of adverse effects of glyphosate seem restricted solely to the laboratory. In the environment, by contrast, exposure is just too low for any measurable effects.

Conclusions

The prime objective of federal pesticide law is setting a tolerance—also known as a maximum residue limit, or MRL—for pesticide residues in foods.

In fact, the majority of all foods have no detectable pesticide residues. Of all foods, insecticide residues specifically are detected in less than half of commercial fruits. Few fungicides are detected, and almost no herbicide residues are detected.

So, why all the public and media concern about pesticides? The main source of the problem is the widespread misinterpretation of the results of various laboratory studies of toxicological mechanisms and the corresponding failure to consider how the chemicals are actually used in the real world. These misperceptions are aggressively encouraged by anti-chemical activist groups calling themselves “environmental” groups.

Missing from much of the public debate are the benefits of chemical technology, especially as applied to crop protection. This report has not engaged in defending old chemical technology because agriculture has moved far beyond it. Crop protection specialists themselves have long argued for judicious use of crop protection agents. And industry long ago began to examine the problems of the most persistent chemicals with broad spectrums of toxicity to nontarget organisms. Chemical manufacturers have since synthesized new compounds whose features include less persistence,

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less toxicity, and greater selectivity for specific pests versus nontarget organisms. Furthermore, the amounts of new chemicals needed to control pests are small fractions of what they were just 20 years ago.

The point is, chemical technology has continued and will continue to improve human health and nutrition, whether helping to make vegetables and fruits of high quality more abundant and cheaper or to preserve the health of individuals who can then help their society to progress.

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