

Prevention, not profit, should drive pest management

Multi-nationals have driven pest management down a route of maximum corporate profit. In this year's Rachel Carson Memorial Lecture Chuck Benbrook questions the wisdom of their strategies and advocates more integrated approaches to pest management.

In 2006, Florida's 33,000 acres of sweet corn were sprayed on average 13 times with 2.3 different insecticides, amounting to 3.7 pounds of active ingredient per acre. Almost nine applications were made per acre with the carbamate methomyl. Few organisms would survive summer in such a corn field.

Just to the north in the State of Georgia, another leading sweet corn producer, the average acre was treated 14 times with methomyl. In its warm climate sweet corn grows rapidly and most varieties reach maturity in 80-110 days. In years with intense and early fall armyworm pressure, spraying starts about four weeks after planting. So, in Georgia in 2006, methomyl was applied every four to five days.

In south-central Florida in 2008, an experienced grower, producing several thousand acres of conventional vegetables, harvested 25 acres of organic sweet corn treated only with the natural insecticides *Bacillus thuringiensis* (*Bt*) and diatomaceous earth. This sweet corn suffered less pest damage than most conventional corn in the region. In the State of Oregon in 2006, about three-quarters of the sweet corn acres (mostly conventional) were not treated with insecticide.

Why are a dozen or more applications of relatively toxic, broad-spectrum insecticides required on some sweet corn but not others?

The answer lies in the differences between a 'systematic' or integrated approach to pest management, in contrast to management systems dependent on a few control tactics, especially those that are treatment oriented and 'systemic' in nature.

Prevention versus treatment

'Systematic' approaches to pest management integrate multiple tactics and practices to prevent pests from gaining a foothold in the field. Such approaches are typically referred to as Integrated Pest Management, or IPM. Unfortunately, an excessive reliance on pesticides in some IPM systems has sullied IPM's good name, leading many to place added emphasis on preventive practices within IPM systems. In the 1996 book published by Consumers Union called 'Pest Management at the Crossroads,' my co-authors and I coined the term 'biointensive IPM' to describe approaches which rely heavily on tactics known to avoid or prevent pest problems. Bio-IPM relies much less on treatment-based interventions, like spraying broad-spectrum insecticides.

In bio-IPM, a variety of tactics are used to suppress pest populations. The goal is to keep populations of pests below damage thresholds, so that more intrusive, costly, and often risky interventions are not necessary. When

pest avoidance and suppression prove inadequate, the hammer must fall, and pesticides (or other interventions) are deployed.

Long-term success with prevention-based bio-IPM depends on how effectively farmers and pest managers:

- Integrate multiple tactics with an emphasis on avoidance and suppression;
- Limit the need for fast-acting, hard-hitting toxin-based 'solutions'; and
- Avoid excessive reliance on any single tactic, practice, genetic trait, or pesticide.

By their very nature, bio-IPM systems are information and management intensive, and require a sophisticated understanding of real-time ecosystem dynamics. IPM practitioners must actively influence pest-plant interactions, and they must be willing to alter, in the name of safe and sustainable pest management, what they grow and how they grow it.

Some farmers in the US have done this and, in general, have thrived as a result. In many crops and regions, organic farmers are at the forefront of IPM-innovation. For example, a significant share of the tree-fruit industry in Washington State has converted, or is transitioning to organic production, in part because of the organic price premium, but also because prevention-based IPM is working. It has dramatically lowered risks to farm workers and non-target organisms, and costs no more, in most years, than chemical-intensive management.

Systemic pest management

'Systemic' approaches to pest management rest upon the incorporation in plants of chemicals or toxins which prevent pest damage. Taken to an extreme, the goal of systemic pest management is to eliminate any further need for the farmer to worry about pest management. Pardon the mixing of metaphors, but the Holy Grail of systemic pest management is the proverbial Silver Bullet – a plant variety or pesticide that frees the farmer from any further worry about pests (or at least one type).

Traditionally and with considerable success, plant breeders have applied their skills in pursuit of crop varieties with a high level of resistance to a pest. Such resistance is typically brought about by the production within the plant of secondary plant metabolites, or phytochemicals, that impact insects or plant pathogens attacking a plant. Some phytochemicals directly kill pests, others just ward them off by emitting offensive odours or tastes, and a few work indirectly, by attracting other organisms that eat, compete with, or control the reproduction of the target pest.

The pesticide industry has discovered and marketed over the years a number of pesticides that are systemic. This class of pesticides work, for the most part, by moving into a plant through its root system, where the chemicals are then spread by the plant's vascular system throughout the plant.

In the mid-1980s the seed and pesticide industries took advantage of the emerging tools of molecular biology to create transgenic plants expressing genes capable of the systemic production in plant cells of *Bacillus*



Scouting for pests and beneficials in a biointensive IPM orchard system

Photo: The Organic Center

thuringiensis (*Bt*) endotoxins. When ingested by Lepidopteron insects, *Bt* endotoxins result in ruptures of the gut, leading to leakage, dehydration, and death.

The first wave of *Bt*-transgenic crops was commercialized in the mid-1990s and today, millions of acres of *Bt* corn and cotton are grown around the world. Despite world-class hype and an enormous investment of research dollars spanning two decades, the second wave of *Bt* crops (beyond corn and cotton) is stuck in a holding pattern.

And then there is a little-known third rail of systemic toxins in the world of pest management. For decades farmers have relied on a series of seed treatments (a coating of pesticide(s) on a seed) to help assure healthy germination and robust early growth of young plants. The insecticides used for seed treatments have, until recent years, killed certain soil borne insects by contact, but since the late 1990s, systemic nicotinyl insecticides (also known as neonicotinoids) have taken over most of the seed treatment market around the world.

Nicotinyls kill sucking and chewing insects by disrupting the insect nervous system. In addition to moving to a new chemical family (the nicotinyls), seed treatment delivery has also dramatically changed. Today, treated seeds are coated with a material that encapsulates the seed treatment chemical(s), and releases them slowly. This extends the time period during which the root systems of newly germinated plants receive a measure of protection, but it also extends the time during which residues from seed treatments are likely to persist in treated plants.

Between their uses as seed treatments, granular formulations incorporated in the soil, and liquid sprays, the systemic nicotinyls have become, by some measures, the most important class of insecticides currently on the market. Resistance to organophosphate (OP) and synthetic pyrethroid insecticides, coupled with regulatory pressures on high-risk OP and carbamate insecticides, opened up major markets for the nicotinyls in the mid-1990s. At that time, there was near-universal agreement that the nicotinyls were the right chemicals at the right time.

In my work with the Wisconsin potato industry and World Wildlife Fund in the second half of the 1990s, we concluded that the adoption of the nicotinyl insecticide imidacloprid (Admire) for Colorado potato beetle control single-handedly reduced pesticide risk levels by about one-half, based on our ability to measure risks at the time. At about the same time in Florida, imidacloprid was providing tomato and pepper growers with a desperately needed alternative to deal with a variety of insects that were resistant to most OPs and pyrethroids, and were the primary vectors for plant viruses for which there were no economically acceptable solutions.

In the last decade, the seed and pesticide industry has made a major commitment to systemic-based technology. Systemic pesticides and genetically engineered plant varieties will remain a major focus of private sector innovation and marketing for at least the

next decade. This technological trajectory has advantages and disadvantages, and unique vulnerabilities that are, for the most part, out of sight and out of mind.

Understanding the fundamental differences between prevention-based bio-intensive IPM and treatment-oriented systemic approaches to pest management is a necessary first step to moving away from today's high-cost, high-risk, and unsustainable pest management technologies.

Unforeseen consequences

Well-meaning and long overdue changes in US pesticide regulatory law, the granting of intellectual property rights to transgenic plants, and the cash-driven takeover of the seed industry by the pesticide industry have, like three swollen rivers, combined to reshape the landscape of pest management in the US. Collectively, these forces have had a greater impact on pest management than advances in the biological sciences and 'green' chemistry, and for this a heavy price will be paid.

In the US, and to a lesser extent in Europe, public policies have created or reinforced incentives that are driving and/or luring agribusiness toward systemic approaches to pest management. Few farmers, scientists, and regulators understand the consequences of the shift that is occurring. The research and data collection required to serve as an early warning system, if major problems emerge, are not being made and as a result, we are flying full speed toward an uncertain location that may prove fundamentally inhospitable.

Our failure to ask ecologically-grounded questions, as Rachel Carson did so effectively, coupled with the economic power behind the private sector push toward high-cost systemic, genetic engineering and proprietary pest management technology, has set the stage for a series of train wrecks, large and small. Moreover, we are ill-equipped to deal with the unravelling of today's pest management technology because far too many eggs are in one technological basket, and investment in alternatives has waned. This is a sad state of affairs in an era of great scientific and technical progress, during which safe and sustainable pest management alternatives are emerging all over the world.

High dietary risk

So how important are systemic pesticides? There are more than three dozen systemic pesticides in current use around the world; most are insecticides. There are nearly a dozen systemic fungicides, but none are important, contemporary risk drivers. There are essentially no systemic herbicides.

The majority of the acres treated and pounds applied of systemic insecticides are organophosphates (OP) and nicotinyl insecticides that work both through systemic action and direct contact with insects.

OPs have been around since the 1960s and remain important in the US, although not nearly as dominant as 15 years ago. Nicotinyl insecticides are emerging as the most important class of systemic pesticides in terms of

the value of crops protected, since on conventional farms they are now the backbone of most insect pest management systems, especially in high-value fruit and vegetable crops.

In apples, 25% of the total crop acres in the US in 2007 were treated with imidacloprid, another 37% with acetamiprid, and 8% with thiamethoxam. These three nicotinyls were used on 70% of apple acres in 2007, 79% of pears in 2005, and in 2006, 54% of broccoli and 40% of cauliflower.

By far the most dangerous systemic pesticides ever used are the carbamates carbofuran (Furadan) and aldicarb (Temik). Both are among the most acutely toxic (to humans) pesticides ever registered. Because of their toxicity, these insecticides are typically only applied to, and incorporated into the soil at or near the time of planting. Both are broad spectrum and extremely hard on beneficial insects and birds and pose a wide range of other risks. Because of their systemic nature and placement in the root zone, the chemicals move up into the plant, and are then transported around by the plant's vascular system. As a result, residues are common in harvested crops, and are indeed almost inevitable.

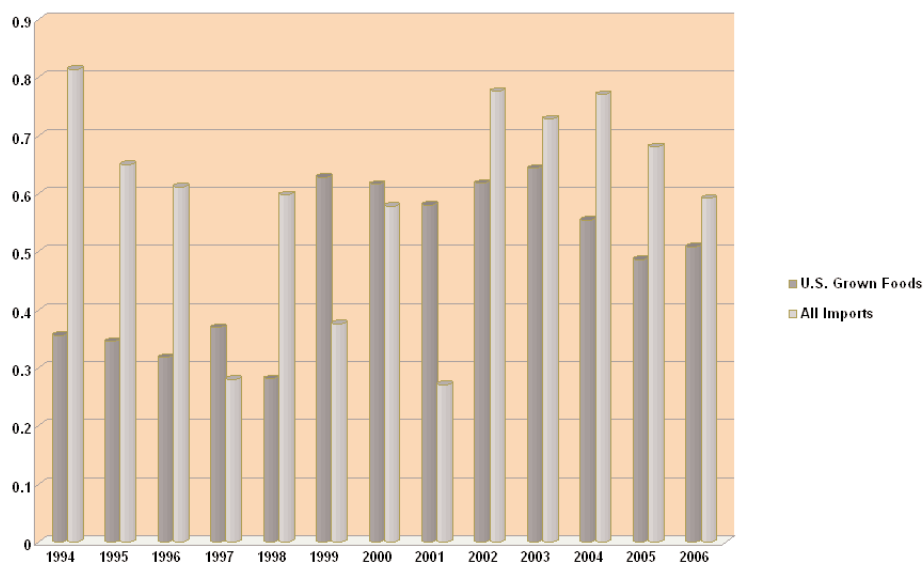
In the US both insecticides are on the way out. The EPA recently announced a decision to phase out all remaining uses of carbofuran, because of excessive risks to infants and children. These insecticides, in particular carbofuran, have killed millions of birds. Species at the top of the avian food chain, like the bald eagle, have been especially vulnerable. One of the regrettable ironies of the successful work of Rachel Carson and others that led to the cancellation of organochlorine insecticides like DDT in the 1970s is that many farmers switched to carbamates and OPs that have proven equally, if not more devastating to bird populations.

Regulators in most countries, and certainly those in the US EPA, have primarily focused on human risks from pesticide residues in food and beverages, as well as occupational exposures. With one minor exception (chlorfenapyr on cotton), the US EPA has never denied an application for a new pesticide, nor cancelled an existing registration solely or largely because of risks to birds, fish, or other non-target (not human) organisms.

In fact, the EPA's ecological risk assessments often show that legal applications of certain pesticides will almost certainly kill non-target species that happen to be nesting, feeding, flying or swimming in and around treated farm fields. Massive death of non-target species is, for example, virtually assured in a sweet corn field sprayed every four days with methomyl.

In general, the American public is unaware of the intensity of pesticide use in several important crops in the US and assumes that the EPA is taking whatever steps are needed to protect non-target organisms like birds, fish, and bees. But in the real world of pesticide regulation, birds, fish and bees are expendable. While the EPA strives to include label provisions that mitigate risks to non-target organisms, such provisions are, for

Figure 1. Share of DRI from systemic pesticides, 1994-2006



the most part, ineffective in the case of high risk chemistry. EPA scientists and decision-makers have known for years that hundreds of registered uses of certain pesticides kill non-target organisms that happen to be in harm's way.

One strategy to prevent harm to non-target organisms and farm workers has been to shift from pesticides sprayed as liquids on crop plants to systemic pesticides. Emphasis in the US on reducing pesticide dietary and occupational risks has clearly reinforced the recent shift toward systemic technologies.

Estimating dietary risk

We have developed a method to estimate the level of dietary risk associated with pesticide residues in food. The Dietary Risk Index (DRI) measures the relationship between residues found in a particular food, and the maximum amount of the pesticide that can be present in a typical serving of food without overexposing the individual consuming the food.

We use an estimate of the 95th percentile residue from all positive residues found in a given year by testing done through the USDA's Pesticide Data Program (PDP). The estimated 95th residue equals the mean of all positive values multiplied by seven. At the 95th percentile level in a distribution of residues, 5% of the values will be higher than the 95th residue level. The multiplier (seven) was empirically derived from in depth studies of residue distributions in several well-tested foods, and reflects the average difference between the mean of the positives and the 95th residue when residues are ranked in descending order.

The amount of pesticide that can be present in a given serving of food is calculated through a simple formula involving the weight of an individual, the size of a serving of food, and the pesticide's chronic Reference Dose (cRfD), also called the chronic Population Adjusted Dose (cPAD) by the EPA. A cPAD equals the chronic RfD divided by any additional safety factor imposed to

assure infants and children are adequately protected. The EPA has imposed added safety factors of three or 10 in several dozen risk assessments. Complex policies govern when a safety factor is imposed, and its value.

Because of the focus on pesticide dietary risks to infants and children in the 1996 Food Quality Protection Act (FQPA), the US EPA has conducted detailed risk assessments on children ages one to two years, three to five year olds, and those 5-13. Pesticides that exceed dietary exposure 'levels of concern' typically do so in one or more of these three age groups. In our work with the DRI, we mimic EPA dietary risk assessment policies and practices, and base the DRI on a 20 kilogramme child, and serving sizes at approximately the 95th level of the food consumption distribution, such as one large apple or one cup of fruit. The DRI measures risk from a single serving of food, it does not take into account the frequency of consumption of a food. An estimated 95th residue of 0.01 parts per million (ppm) in a serving of apples will have the same DRI score as 0.01 ppm residues in an equivalent serving of carrots.

Through the PDP, the USDA carries out annual residue testing of 12,000 to 16,000

samples of foods that play an important role in the diets of infants and children. Each year 10-15 fresh foods are included in the programme, along with 6 to 10 processed foods. Foods are brought into the programme for one to three years at a time. Major children's foods like apples have been tested multiple times since 1994, while foods that are less frequently consumed by children, like collard greens, have been tested only once or twice.

The selection of crops each year in the PDP has a big impact on overall, aggregate DRI values. In years with several foods that usually score high in terms of DRI values – like squash, wheat, greens, soft-skinned fruits, melons, and tomatoes – aggregate DRI scores can be twice or three times the level in a year with few of these high-scoring foods. For this reason, aggregate, all-food DRI scores must be interpreted with caution.

The PDP dataset does, however, provide an opportunity to assess trends in the importance of systemic pesticides as a percent of total DRI values. Figure 1 shows the result of such an analysis, based on all residues found in food grown in the US, as well as all food imported to the US. To my knowledge, this is the first empirically based assessment of systemic pesticides in terms of dietary risks.

It is clear from Figure 1 and the underlying analysis that:

- The systemic pesticide share of total DRI scores in domestically grown foods has risen from around one-third to over one-half in the last decade;
- In imported food, the systemic pesticide share of total DRI has fluctuated between one-third and three-quarters from year to year, but shows no clear trend up or down; and
- Systemic pesticides play a surprisingly major role in total dietary risk levels.

A half-dozen systemic insecticides account for the lion's share of total DRI values across all foods and pesticides in a given year (Table 1). The table reports the shares of total DRI accounted for by specific pesticides. In 2006, chlorpyrifos accounted for 32% (DRI total of 524) and was found in 15 of the foods tested for that year. The highest chlorpyrifos food-specific DRI scores were kale (81.2), peaches (70), orange juice (67.8), cranberries (67.2), and collards (58).

Table 1. Pesticides Accounting for Five Percent or More of Total Dietary Risk Index Values in Any Single Year: US Grown Foods, 2000-2004

	2000	2001	2002	2003	2004	2005	2006
Chlorpyrifos	31.2%	30.0%	32.3%	31.9%	21.4%	19.1%	31.8%
Heptachlor epoxide	3.2%	2.7%	6.9%		17.0%	5.9%	14.9%
Chlorpyrifos methyl	<0.01%	0.1%	0.4%			8.8%	9.8%
Endosulfan sulfate	1.7%	2.6%	3.0%	2.0%	2.1%	2.0%	8.3%
Dieldrin	4.3%	3.3%	5.0%	4.4%	6.2%	4.0%	5.1%
Dicofol p,p'	11.1%	12.3%	6.2%	9.4%	6.8%	15.7%	2.4%
Methamidophos	18.7%	7.6%	12.7%	9.7%	20.5%	12.7%	4.5%
Diazinon	2.2%	5.3%	5.0%	8.4%	0.4%	1.6%	2.1%
Parathion (ethyl or methyl)	1.8%	4.2%	0.1%	6.1%			
Dimethoate	1.4%	5.5%	3.3%	3.8%	2.2%	3.8%	1.2%

Table 2. Annual Total Dietary Risk Index (DRI) Shares for Foods Accounting for Eight Percent or More of Total DRI in Any One Year: US Grown Foods, 2000-2006

	2000	2001	2002	2003	2004	2005	2006
Apples	6.4%	19.2%	26.6%		8.2%	3.6%	
Broccoli		9.8%	4.8%				1%
Cantaloupe	7.2%			3.4%	10.7%	6.1%	
Collard greens							11.6%
Cucumbers	14.3%		11.4%	25.0%	29.6%		
Grapes	12.4%	7.5%			5.3%	7.9%	
Green Beans	7.1%	12.5%			7.9%	13.6%	
Greens, kale							8.2%
Lettuce	2.6%	8.8%			4.8%	3.9%	
Nectarines	3.8%	8.6%					
Pears				9.9%	5.6%	5.1%	
Potatoes	8.6%	7.9%	13.7%				
Spinach			2.5%	11.6%			2.8%
Strawberries	13.0%				5.1%	10.6%	
Summer squash							20.5%
Sweet Bell Peppers	9.4%		18.5%	13.7%	5.5%		
Tomatoes				18.5%	5.6%		
Wheat, grain						14.7%	14.3%
Winter Squash					6.1%	11.7%	6.8%

Chlorpyrifos methyl (used in stored grains) contributed another 10% to total all-food 2006 DRI score (1,647). In 2006 PDP testing:

- Five pesticides accounted for 5% or more of aggregate DRI;
- Twelve pesticides contributed aggregate DRI scores accounting for between 1% and 5% of total DRI; and
- One hundred and eight pesticides accounted for less than 1% shares.

Despite the changing food and crop mix in the PDP from year to year, two insights into trends in pesticide risk are evident in Table 1:

- Despite extensive focus on reducing OP risks and what the EPA billed as a 'historic' set of regulatory actions on chlorpyrifos in 2000, this systemic OP remains the major dietary risk-driver in the US food supply.
- Two organochlorine insecticides banned thirty years ago still account for 10% to 20% or more of overall dietary risk in recent years.

A relatively few foods also account for the majority of total DRI value in any given year, as shown in Table 2. Eighteen of the 19 high-risk foods are fresh fruits and vegetables. Wheat grain is the 19th food, and scores high on the DRI because of residues of the insecticides used to kill insects in stored grain (typically chlorpyrifos methyl, chlorpyrifos, pirimiphos methyl, and/or malathion).

In most foods at the top of the DRI ranking each year, several of the top five pesticides contributing to the foods' aggregate DRI scores are systemic insecticides. The contribution of systemic pesticides to overall dietary risk (now about 60% in food grown in the US) is remarkable given that systemic pesticides account for less than 10% of the total pounds of pesticides applied in the US.

High gear on the pesticide treadmill

The US corn industry stands alone on many counts. Corn is the backbone of the US food system, especially the animal products portion of our daily diet. It is planted on more acres than any other crop (around 85 million), has received more government subsidies than any other crop, requires more pounds of pesticides every year than any other crop, uses and wastes more nitrogen than any other crop, and is largely responsible for the steady growth in size of the Dead Zone in the Gulf of Mexico.

The big news in corn country in recent years has been the impact of ethanol production on crop prices and the economics of livestock production, and the dramatic upward trajectory in production costs. Prices rose sharply from \$2.25 a bushel, plus or minus \$0.50, in most years since 1990, to well over \$7.00 a bushel at the peak of craziness in global farm commodity markets this past summer. Prices are now falling rapidly, in step with other commodities and energy prices.

Below the radar

Historic and consequential changes in corn pest management are hidden in the shadow of ethanol, price volatility, and corn's changing role in the food industry. Since the 1970s, most corn acres have been treated with herbicides, and historically, about one-third of corn acres has been treated with insecticide. Roughly two-thirds of the corn insecticide acre-treatments targeted the corn rootworm, with the other third targeting the European

corn borer (ECB).

In addition, insecticide seed treatments have emerged in the last 20 years as a routine production input. Since 2000, virtually all conventional corn seed has been treated with one or more insecticide seed treatments, and in recent years, often one to three fungicides.

Evidence mounted in the 1980s that typical US corn production systems were wasting a significant share of the nitrogen (N) fertilizer applied each year, leading to serious surface and ground water quality problems. Between one-half and two-thirds of the total N available in corn cropping systems were being lost to the atmosphere or water resources. Moreover, entomologists had compiled convincing data that the corn-soybean rotation, common in the Midwest, was highly reliable in suppressing populations of corn insects and made it unnecessary to apply any insecticides on the majority of fields.

Peak adoption of proven IPM practices on corn farms occurred in the early 1990s. At that time, approximately 30% to 35% of corn acres were treated with insecticides, and about a third of acres were treated with a single insecticidal seed treatment (usually a synthetic pyrethroid). Accordingly, one-third to one-half of corn acres were not treated with any insecticide (since some acres planted with treated seed were also sprayed). In addition, for weed management, systems combining rotations, some cultivation, and targeted herbicide use were showing considerable promise in reducing reliance on herbicides, protecting ground water quality, and improving the efficiency of N use and nutrient cycling within farming systems.

The unravelling of corn IPM

Two factors began eroding the foundation of corn IPM in the US Corn Belt in the mid-1990s. A genetic variant of the western corn rootworm emerged early in the 1990s in one county in Illinois. This new subspecies had learned to leave the corn field in which it was borne and fed during the growing season, and move into a nearby soybean field to overwinter. When its new home was planted to corn the next spring, as is commonly the case, the insect was in the right place and ready to grow and reproduce. Populations of the variant corn rootworm grew and spread through the Midwestern US, increasing corn root damage and triggering a big jump in insecticide use from 1995 to 1996.

As the damage to corn root systems increased, farmers turned to corn insecticides and seed treatments to protect the integrity of young corn plants. In 1997, the first *Bt* corn was introduced, genetically engineered to express a *Bt* toxin active against the European corn borer. Most farmers were not treating fields for ECBs because populations rarely exceeded economic thresholds, and the available insecticides were costly, high-risk, and at most only 75% effective. But the early efficacy data on *Bt* corn for ECB control was breathtaking, and confirmed over 99% control in virtually all fields. Adoption reached about 30% of acres planted by 2003.

Today ECB varieties are planted on about two-thirds of conventional corn acres, a far higher percentage than ever treated with insecticides for ECB control. Entomologists across the Midwest recognize that a significant share of the acres planted to *Bt* corn for ECB control are planted to this GE trait prophylactically, in effect as an insurance policy against possible, but low-probability losses.

In 2003, as problems worsened with the variant corn rootworm, Monsanto introduced Cry 3bB *Bt* corn, engineered to express a *Bt* endotoxin active against corn rootworms and some other soil borne insects. In 2008 nearly one-third of corn acres were planted to *Bt* corn for corn rootworm control, and several million acres were planted to 'stacked' varieties expressing both *Bt* toxins, and conferring resistance to the herbicide glyphosate. All major corn seed companies are moving rapidly toward the day when most seed comes with at least a three-trait stack – both *Bt*s and resistance to glyphosate.

In addition, virtually all of this GE-corn seed will be treated with a systemic nicotinyl seed treatment, and some 30% of the acres will likely still be treated with an insecticide. Accordingly, in the next few years, the average acre of field corn in the US will contain 26,000 to 32,000 plants with over three insecticides moving through plant tissues – two *Bt* toxins manufactured in the plant, a nicotinyl seed treatment, and on perhaps 15% to 20% of the acres, a systemic insecticide applied at planting or during the growing season.

Contrast today's need for over three systemic insecticides to the ability of corn farmers in the early 1990s to get by with well less than 0.3 systemic insecticide applications per acre. The approximate ten-fold increase in reliance on systemic insecticidal toxins over about a decade marks a new high gear on the corn insect pest management treadmill. It also is imposing on several insect species tremendous selection pressure for resistance. We know from bitter experience that the corn rootworm is notorious for its genetic plasticity, and now, we are selecting for resistant variants to two *Bt* toxins and the whole class of nicotinyl insecticides, all at the same time.

New risks?

We are entering uncharted waters in the assessment of farm animal, human, and ecological impacts associated with the trend toward systemic solutions to corn insect management challenges. Millions of acres of corn silage are grown and harvested at a stage when there remain relatively high levels of *Bt* toxins, and perhaps even nicotinyls, through plant tissues. I know of no research exploring the impacts on animal health and reproduction of the toxin cocktail now in corn silage.

Corn enters the human diet in a myriad of ways. High fructose corn syrup (HFCS) is one of the most important in terms of public health. We know that both nutrients and chemical contaminants in grain crops like corn are sometimes concentrated in the developing kernel, as the plant redirects nutrients in leaves and stalks to its all-important seeds.

We also know that when raw commodities are processed into various fractions – corn to corn oil, HFCS, and corn meal – both nutrients and chemical contaminants sometimes concentrate in one fraction more so than others. Ten-fold concentration ratios are common. A few pesticides are known to concentrate 100-fold or more in a particular food fraction, especially when moisture is removed, dramatically reducing the weight of the remaining fraction of food.

Again, to my knowledge, no one in the US has carried out the studies necessary to determine whether *Bt* toxins or nicotinyl insecticides concentrate in corn kernels, or certain products made from corn. Proponents of these technologies argue that there is no evidence of novel food safety risks or ecological harm, which is not surprising, since no independent scientists have been given the resources to carry out the studies needed to detect such impacts.

It appears that once again, as in Rachel Carson's era when the damage caused by organochlorine insecticides was only recognized after years of use, we will learn about and confront unforeseen problems with today's systemic toxins after the fact.

Our ability to innovate in the discovery and delivery of insecticidal toxins continues to outpace our ability to understand the consequences. In the next section, one such new, unforeseen impact of the trend toward systemic pest management technologies is explored in more detail.

Honey bees in peril

In November UK beekeepers marched on Downing Street to call for more funding into the factors imperilling honey bees. Some analysts fear that the UK will run out of domestic honey by Christmas, 2008, as a result of the loss of billions of bees to varroa mite and other maladies of unknown etiology.

Because of the vital role played by bees in crop pollination, honey bee Colony Collapse Disorder (CCD) threatens the production of crops that produce about one-third of the UK and American diets, including nearly 100 fruits and vegetables. The value of crops pollinated by bees exceeds \$15 billion in the US alone. Populations of native pollinators are in decline worldwide, heightening the importance of reversing CCD. A survey of US beekeepers was conducted between September and March 2007 and reported an average bee loss of 37.6%, about triple the norm, with over half of respondents reporting 'abnormally heavy' losses¹. One in three bee colonies have been lost in the UK since autumn, 2007².

Progress has been made in identifying possible causes of CCD, which are likely to include complex combinations of pesticides, weakened immune systems, varroa mite, and viruses. Still, the epidemiology of CCD remains puzzling³. One team found an average of 3.7 pathogens in bees from CCD hives, compared to 2.1 pathogens in non-CCD hives.

Two possible CCD risk factors have received much attention – GE crops and nicotinyl insecticides (such as, imidacloprid,

thiamethoxam, clothianidin, acetamiprid). The Cry 1 and Cry 3 *Bacillus thuringiensis* endotoxins expressed in GE-corn varieties are not known to be active against hymenopteran insects, nor do CCD symptoms match the mode of action of *Bt* toxins. Several studies have found no adverse acute or subacute effects following the feeding of bees on pollen from *Bt* corn plants, nor pollen cakes spiked with purified *Bt* endotoxins⁴. Plus, the geographic distribution of *Bt* corn in the US is heavily concentrated in a few regions and clearly differs from the distribution of CCD.

Still, the potential for horizontal gene transfer from GE-crop pollen to microorganisms in the bee gut has been demonstrated⁵; pollination deficits have been recorded in canola fields planted to GE, herbicide-tolerant varieties, compared to 'moderate deficits' in fields planted to conventional seed and no deficit in fields under organic management⁶; and, behavioural effects linked to CCD have recently been demonstrated in bees exposed to high levels of *Bt* corn toxins.

Evidence in support of an impact on bee hive health by the systemic nicotinyl insecticides is far more convincing, and in my judgement well beyond the threshold needed to justify decisive action to prevent future losses. Nicotinyls are moderately persistent in the soil and are the most acutely toxic pesticides ever registered to bees. Major bee kills have occurred from foliar applications, leading to binding pesticide product label restrictions (such as, 'Do not spray when bees are actively foraging in the field').

These insecticides are also known to cause chronic and sublethal effects in bees at very low doses measured in parts per billion (microgrammes per kilogramme, or ug/kg), or even parts per trillion⁷. Such effects include impaired foraging ability, failure to return to the hive, and other neurobehavioural impacts⁸.

Concern over the extreme toxicity of nicotinyls to bees in Europe in the late 1990s led to questions about routes and levels of bee exposure, and possible chronic and subacute effects. While most of the initial focus was on standard agricultural applications of nicotinyls in the field, exposures linked to the seed treatments have emerged as possibly the missing piece in the CCD puzzle.

Focus on seed treatments

Most conventional corn seed, and virtually all *Bt* corn is now treated with a nicotinyl seed treatment to protect just-germinated corn plants from soil borne insects. Corn plants grown from seed treated with the typical commercial rate of 1 milligramme (mg) imidacloprid per seed produced pollen with an average level of 2.1 ug/kg (ppb) imidacloprid⁹. A bee ingesting just 6 mg of such pollen per day would have a PEC/PNEC (probable exposure concentration/predicted no effect concentration) ratio between 500 and 600 for chronic mortality after 10 days of exposure¹⁰, leading to the conclusion that imidacloprid 'is one of the major factors contributing to the weakening of bee colonies.'

Levels found in sunflower pollen and

flowers, from plants sown with imidacloprid-treated seed, were 3 ug/kg and 8 ug/kg, levels high enough to kill bees¹¹. Chronic and sub-lethal effects have been reported at levels between 0.1-10 ug/kg (ppb) in a bee food source. Neurobehavioural problems in bees have been reported from exposures to imidacloprid and other nicotinyls at levels routinely found in crops grown from seeds treated with nicotinyls. Plant tissues known to sometimes contain damaging levels of nicotinyls from seed treatments include corn foliage, silk, and pollen, and rapeseed and sunflower pollen and nectar¹².

The finding of neurobehavioural disruption is significant given that a hallmark of CCD is that foraging bees leave the hive but cannot find their way back. Still, if nicotinyl insecticides in silk, pollen or nectar are a major cause of CCD, the epicenters of CCD should include the American Midwest and the Canadian prairies where corn and canola seed treated with nicotinyls are widely planted. This does not appear to be the case.

In the US colony collapse disorder appears to disproportionately impact large, commercial bee keepers who often move their hives long distances in the late winter. Transporting hives is a source of stress, and also tends to bring multiple populations together, where pathogens from many areas and bee strains are readily exchanged. It is also known that CCD tends to happen early in the season during the first spring flights from a hive, suggesting that something in the winter feeding and management of the bees may be a CCD risk factor.

Other notable pieces of the CCD puzzle include the fact that a significant portion of commercial bee keepers leave inadequate stores of honey in their hives to sustain the bees through the winter, and instead feed bees a cheaper high fructose corn syrup (HFCS) supplement. Starting in about 2004, and roughly coinciding with the emergence of CCD, corn seed companies in the US began marketing seeds treated with a 5-X rate of nicotinyls (1.25 mg/seed, compared to the traditional 0.25 mg/seed). The rate was increased to expand the range of insects adequately controlled, and/or to control higher insect populations. For example, 80% of the corn seed sold in 2007 by corn seed market-leader Pioneer Hi-Breed Int. was treated with Poncho 250 or 1250 seed treatments containing clothianidin at 0.25 and 1.25 mg/seed respectively, plus two fungicides (the systemic azoxystrobin, and fludioxonil). Pioneer first sold seeds treated with the 5-X rate of clothianidin in 2004¹³.

Moreover, simultaneous bee exposure to nicotinyls and fungicides (triflumizole and propiconazole) can increase the potency of nicotinyls up to 1,141-fold¹⁴. Possible synergistic effects between nicotinyls and other fungicides have not been explored to date.

A growing body of evidence suggests that the most decisive and concrete action that can be taken worldwide to reduce the chances that honey bee CCD will persist, or grow worse, is to end seed treatments with any pesticide that is: (a) systemic, and (b) highly toxic to bees.

To my knowledge, the only class of contemporary seed treatments that meets both criteria is the nicotinyls.

Moreover, the impacts of nicotinyls on bees are likely just the first of many surprises in terms of the ecological impacts of systemic pesticides. Most regulatory programmes, and certainly those in the US, pay little attention to pesticide impacts on bees. In the face of clear evidence of substantial potential harm to bees, registrations are still granted, along with label language designed to limit bee exposures.

The US EPA has never denied an application for a new pesticide, nor banned a currently registered product because of adverse impacts on bees, nor is it likely to without new legislation and a push from the public and Congress. In the US, the focus of the EPA is on preventing adverse impacts of pesticides on human health, and secondarily on birds, fish, and endangered species.

The Agency does not assess, nor strive to control the adverse impacts of pesticides on agronomic crops, desirable vegetation near farm fields, or on 'personal property,' a category of assets that includes honey bees. Pesticide registrants are held accountable for such impacts and are encouraged by the EPA to include binding label restrictions to prevent damage to personal property. When such provisions on labels prove inadequate, and damages occur, victims must seek relief and compensation from the pesticide applicator or registrant. Many such cases lead to litigation, most of which is costly and protracted, and does little to resolve the circumstances that led to the damage in the first place.

Scientists in the US are close to proving that pesticides, and in particular the nicotinyls, play a critical role in triggering CCD. However, based on current law and EPA policy, little is likely to change as a result.

Lessons learned, new challenges

Like nearly all pest management tools, there are advantages and disadvantages to systemic pesticides. Systemic pesticides can limit exposures to just a few classes of organisms, in addition to target pests. Applying systemic pesticides via seed treatments or granular formulations incorporated in the soil dramatically reduces the risk of farm worker exposure, and spares individuals living near farm fields from some exposure pathways. Systemics can limit the impacts of insecticides on some beneficial insects and organisms, while increasing the risks to others, such as bees.

But there are also disadvantages. Residues of systemic pesticides cannot be washed off the surface of foods, because they are inside the food. This increases the frequency and potential risk associated with dietary residues. Organisms other than target pests that feed on plants treated with systemic pesticides are more likely to be exposed, since the residues of systemic pesticides, or their metabolic breakdown products, tend to be more persistent inside plant tissues than when lodged outside, on plant surfaces, where rainfall and

sunshine washes them off and breaks them down.

By incorporating systemic toxins into plants, pesticide and seed companies bear a more complex scientific burden, since the impacts of the toxins on the physiology of the plant should be explored with a high degree of sophistication. Today's cursory reviews and 'substantial equivalence' policies are grossly inadequate for this purpose and will not detect most subtle changes in gene expression and regulation brought about by the presence of systemic pesticidal toxins and, in the case of *Bt*-transgenic plants, the genes needed to produce *Bt* endotoxins within plant cells.

Moreover, the trend toward systemic pest management technologies is likely to alter how plants respond to unusual biotic or abiotic stresses. Such responses by plants trigger and control production of phytochemicals, and hence can lead to possibly significant nutritional and food safety consequences, some beneficial, others likely not.

The best way to minimize the chance that systemic pest management solutions trigger unforeseen problems is to rely on them sparingly and only when prevention-based bio-intensive IPM systems are overwhelmed. That is not the path we are now on. Our current path is leading inevitably to the need for more toxins, which will trigger more resistance, kill more beneficial organisms, narrow biodiversity and set the stage for higher costs and new and anticipated problems.

If we travel too far down our current path, we could create conditions in our food system much like those that brought down the financial system. That is an outcome we should all work tirelessly to avoid.

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