

Maine Board of Pesticides Control

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February–May 2012**

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'Yardscaping' demo to be unveiled at Flower Show

Written by David Carkhuff



A pedestrian strolls through the Yardscaping Gardens at Back Cove Tuesday, where signs explain the benefits of "low maintenance plants." An exhibit at this year

A low-impact approach to landscaping that could help

protect Casco Bay from pesticide and fertilizer runoff is receiving its first formal public exhibition this week at the Portland Flower Show.

An exhibit at this year's Portland Flower Show, which starts tonight and runs through Sunday at the Portland Company complex, will offer a "sneak preview" of the newly completed Yardscaping Gardens at Back Cove.

"We've been working on this for a long time trying to encourage people to reduce their use of pesticides and fertilizers," said Mary Cerullo, associate director of Friends of Casco Bay, a marine stewardship organization.

For more than a decade, Friends of Casco Bay tackled "Bayscaping," an education effort aimed at convincing landowners to reduce pesticide and fertilizer use.

Today, "Yardscaping" is the term coined to describe ecologically sensitive landscaping that minimizes

reliance on water, fertilizer and pesticides.

The Back Cove demonstration gardens have been in the works for about six years, said Gary Fish, Yardscaping coordinator and manager of pesticides programs for the Maine Board of Pesticides Control.

"Yardscaping is a partnership of public and private entities that are trying to encourage Maine gardeners to minimize reliance on pesticides, fertilizers and irrigation water, primarily because of some of the monitoring we've done where we've found pesticides and fertilizers in both the sediments and the water of Casco Bay," Fish said.

The goal of the demonstration project is to educate the public about the availability of locally sold plants and grasses that don't require as much fertilizing or spraying, he said.

The forward-looking Yardscaping approach has won adherents in the landscaping industry, although homeowners have been slower to embrace the concept, Fish said.

"We've been doing this kind of promotion since about 1999, and over the years we've certainly changed the perspective of landscape practitioners," Fish said.

A new sustainable landscaping training manual and sustainable landscaping certification are among advances within the industry, he said.

"I think at the homeowner level it's slower to be adopted. We certainly have a lot of people interested in it," Fish said.

But public interest was heightened over the past six years as the demonstration gardens took shape on the Back Cove, near Preble Street Extension, Fish said. The demonstration project was completed last year. A grand opening, originally scheduled for last fall, had to be postponed to this spring, on a date to be announced.

Still, during its emergence, the demonstration site generated word-of-mouth interest, Fish said.

"Hundreds of people go by, they ask questions all the time. We've had a number of landscape architects and others interested in it," he said.

The city provided the two-and-a-half-acre unused field now planted with more than 2,000 specially selected trees, shrubs and perennials, including six different grass types and two wildflower fields. A kiosk with information accompanies a set of four signs describing Yardscaping. This summer, there's hope of establishing a YouTube tour explaining the plants, Fish said.

"We plan to have training programs there for local landscape practitioners and municipal folks," he said.

Fish's office already has scheduled a class tour with Waynflete School in the spring, he said.

Master Gardener volunteers from University of Maine Cooperative Extension tend the site, but the demonstration site still needs volunteers for weeding and mulching, Fish said.

The YardScaping booth, No. 33 on the ground floor at the Portland Flower Show, will provide additional information. The public also can go to www.yardscaping.org.

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Articles

Casco Bay testers: 'Green slime' one threat from fertilizer runoff

Written by David Carkhuff

"Green slime" isn't something out of a science fiction movie, but a real environmental threat that can strangle waterways, environmental officials say.

One exhibit at the Portland Flower Show this week aims to keep the expanding algae at bay, at least over the long term, by educating the public about more environmentally sensitive landscaping approaches that curb the use of fertilizers, a key source of slime-inducing nitrogen.

A gardening and landscaping exhibit on the Back Cove, the Yardscaping Gardens, could over time help reduce this form of algae growth, organizers of the exhibit said. At this week's Portland Flower Show, creators of the gardens will staff a booth to talk about low-intensity landscaping.

Nitrogen runoff — a common result when landowners use fertilizers that leak into storm drains — can create algae blooms, which threaten to choke out clams and other marine life, said Mary Cerullo, associate director of Friends of Casco Bay, a marine stewardship organization.

Slime-covered coves and low dissolved oxygen can result from nitrogen runoff, said Cerullo. "Green slime algae can smother mudflats," and when the algae decays, bacteria can consume the water's oxygen, she noted.

In 2007, Friends of Casco Bay helped persuade the Maine Legislature to pass a law requiring the Maine Department of Environmental Protection to set a limit on nitrogen discharges into coastal waters.

The group acknowledges that excess nitrogen comes from more than just stormwater runoff carrying fertilizers from lawns — the city's sewage overflows, a problem being tackled through sewer system upgrades; and air pollution from tailpipes and smokestacks are also listed as sources by Friends of Casco Bay.

The group also concedes that there is a host of factors, not just one, that contribute to marine degradation. Yet, the Friends group keeps a close eye on Casco Bay, "monitoring all year round" for nitrogen, fluctuating temperatures and other data.

"The more kinds of stressors you put on marine life, it's one more factor that they have to deal with," said Cerullo.

The Yardscaping project, which ultimately could attack the problem of nitrogen runoff and pesticide pollution at the source, was funded in part by a \$35,000 grant from the Environmental Protection Agency and a \$10,000 grant from the Davis Conservation Foundation.

"One of the things that we have found that's the driver of pesticide use on people's home properties is their lawns," said Gary Fish, YardScaping coordinator and manager of pesticides programs for the Maine Board of Pesticides Control.

"The products that we're finding are lawn products," he said.

These discoveries are based on Casco Bay water monitoring, conducted since 2002, Fish said. Last year was the first year that the Maine Board of Pesticides Control did not do monitoring due to budget restraints, he said. He said he's not sure about monitoring this year.

Water sampling was done in cooperation with Friends of Casco Bay in cooperation with the cities of South Portland and Portland, Fish said.

"The federal grant has stayed at the same level for about 18 years now, and it's getting to the point now where additional requirements that we have from federal laws that have come around in the past few years have forced us to divert that money into other areas," he said.

Still, as Fish tries to channel money toward water monitoring, Friends of Casco Bay continues working with the state, communities, residents and sewage treatment plant operators to reduce the amount of nitrogen flowing into Casco Bay.

"It's a fallacy if you think your storm drain goes to the waste-treatment plant," Fish said.

For more information on the Yardscaping demonstration project, visit www.yardscaping.org.

For more about Friends of Casco Bay, visit <http://friendsofcascobay.org>.



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'Green slime' one threat from



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April 1

State wants help fighting potato blight

By [Mechele Coopermcooper@centralmaine.com](mailto:Mechele.Coopermcooper@centralmaine.com)
Staff Writer

AUGUSTA -- Much of the state's seed potato supply for this year is infected with blight and the state has asked the federal government for an emergency exemption to make an effective but expensive toxic seed treatment available to farmers.

"The time is pretty germane to have it now," said Steve Johnson, a crops specialist with the University of Maine cooperative extension. "The pathogen has been found in seeds, and so the last thing we want to do is start our own epidemic by planting these seeds."

Extreme wet weather and infected seed potatoes that were imported during the 2011 growing season resulted in a severe outbreak of late blight on Maine's potato crop. Saturated soil late in the season transferred it to the tubers in the ground, Johnson said.

To ward against another severe outbreak of late blight on Maine's potato crop, the Board of Pesticides Control has asked the federal government for an emergency exemption registration for Revus fungicide, an expensive pesticide that's mildly to highly toxic to different species.

Revus is new on the market and is registered for blight control in the U.S. for grapes and vegetables, including potatoes, but not for seed potato pieces, according to the U.S. Environmental Protection Agency.

Johnson said Revus is an effective plant health medicine on the path of becoming a fully registered material, but not in time for this year's potato growing season.

If the exemption is granted, farmers will weigh the high cost of the fungicide -- \$350 a gallon -- against the potential money they'd lose if blight ruined their crop.

Bruce Flewelling, a potato grower in Easton, said it will cost about \$20,000 to treat seeds with revus on his 1,000-acre farm.

"I'm looking at using it. I'm excited to use it, but then I looked at the price tag," Flewelling said. "There again, if we do get blight, everything goes out the window. If we can keep it out (of our crop) it's better for me and my budget. Last year it was a nightmare. We had a rough time with the wet weather."

Flewelling said revus is a good chemical that he has sprayed over the top of his potato plants over the past four years, but never on seed pieces.

"It's a big area, so we would use it on all the seed we got," he said.

Paul Schlein, spokesman for the Maine Board of Pesticides Control, said Maine has about 56,000 acres of potatoes. An average farm is about 190 acres. He said it would cost a potato grower using Revus between \$15 and \$30 an acre depending on the potato variety.

Johnson said it could cost farmers \$3,000 per acre if blight hits their crops. He said potato farmers suffered tremendous losses last year because of the blight. On top of the unprecedented rain, Tropical

Storm Irene spread the epidemic to other fields.

The state board unanimously approved the request and sent it along to the U.S. Environmental Protection Agency for consideration, which has 50 days to make a decision, Johnson said.

"They approved this material in Montana, so it isn't unprecedented," he said. "They will let us know within 50 days, which is a little bit too long." He said the treatment has to be done before planting and ideally, the seed would be treated in April for May planting. He added the federal government is aware of the time crunch, so may act fast.

"That's a serious loss," he said. "This is trying to control and manage the disease."

Rob Johanson, a certified organic grower in Dresden, said he will treat his potato seeds this year with biological inoculates to protect them from blight.

"It's the only thing we got going to protect the plants in an organic system. We don't have chemicals like the other guys," he said.

Revus contains the active ingredient mandipropamid. According to a 2008 California Department of Pesticide Regulation Public Report, mandipropamid is slightly toxic to birds and honeybees, moderately toxic to fish and some shrimp, and highly toxic to eastern oyster.

Denis Thoet, who grows potatoes on his small West Gardiner farm each year for his community-supported agriculture customers, said he would never pre-treat anything with chemicals.

He said the use of a fungicide like Revus only benefits large-scale potato farms in Maine.

"It's not good for you and probably not good for the plant," Thoet said. "There's other ways to control blight. Our crop was affected in 2009. That's the first year blight was a factor in small farms; it's always a factor in large farms.

"They treat it on a large scale and have a large-scale problem with seed. They're putting (chemicals) in the ground and the consequences are worse than they think."

Growers who sell more than \$1,000 of plant products intended for human consumption and use over-the-counter pesticides must get an applicator license, which is good for three years and requires passing an examination.

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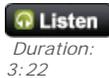
Lawmakers Hear Testimony on Proposed Merger of Maine Ag and Conservation Depts

03/13/2012 Reported By: [Patty B. Wight](#)

In an ongoing effort to increase efficiencies and boost Maine's economy, the LePage administration wants to merge the Departments of Agriculture and Conservation. The two agencies share similar interests, and the thought is that combining them will create a more powerful, unified voice in Washington. While the commissioners from both departments support the merger, some farmers and environmental groups say both sides could lose in the deal.

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If you ask both Commissioner Bill Beardsley of the Department of Conservation and Walt Whitcomb of the Department of Agriculture, they'll tell you that merging their agencies is just plain common sense. Here's Whitcomb:

"These are the entities that really I think nurture the land," he says. "These are the people who are the forest interests or recreational interests or interests as diverse as the maple industry and cross country skiing and our farming community. They all have this one common denominator - the land."

And, says Whitcomb, they often have their hands in the same pot. Take the maple industry. Whitcomb says the Department of Agriculture markets and encourages maple syrup production. The Department of Forestry provides technical advice, and the Conservation Department provides thousands of acres of leases for people who harvest maple syrup.

"Perfect example of where you go across the boundaries, where we really shouldn't be trying to make up our minds in different buildings," Whitcomb says.

While the proposed merger isn't being pitched as a cost-cutting measure, Whitcomb says down the road, there likely will be efficiencies and savings. What's more, he says, the two agencies will have a united vision that can only help when they seek federal support.

But some, like former Maine Lawmaker Wendy Pieh, a farmer from Bremen who once chaired the Agriculture Conservation and Forestry Committee, say at their core, each agency's mission is too different to consolidate.

"If you were to ask these two commissioners to outline for you what they do over a week, I don't know how you're going to cover the different needs that are needed by the people of Maine with one commissioner, regardless of knowledge base," Pieh says."

This is one of the main concerns of farmers opposed to the bill: making sure the new commissioner has experience in agriculture. Agriculture Commissioner Walt Whitcomb is a dairy farmer, and many say that fact alone goes a long way towards ensuring the agency has a positive approach towards helping farmers comply with regulations.

The fear is if a new commissioner didn't have that same background, the approach may be more "gotcha." Some conservationists share similar fears that their interests could get lost if the proposed merger becomes a reality.

"We don't think that the natural resources that the Department of Conservation is managing should be treated like a crop," says Cathy Johnson, the North Woods Project Director for the Resources Council of Maine. She says the language of the bill shifts the focus from conservation towards economic development.

"Even the forests, which some people analogize to be like crops, really are much more," she says. "Yes, trees are harvested like a crop, but they also provide important ecosystem values, wildlife habitat, clean water and so forth."

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Stakeholders split on LePage department merger plan



[Steve Mistler](#), Staff Writer

[Maine](#) | Tuesday, March 13, 2012 at 4:04 pm

AUGUSTA — Stakeholders appear divided over Gov. Paul LePage's proposed merger of the Agriculture and Conservation departments.

LePage announced the consolidation plan last year. On Tuesday, lawmakers on the Legislature's Agriculture Committee began hearing public comments on the actual legislation.

The state's influential forest products lobby stood firmly behind the proposal, arguing that it could lead to further promotion and development of the state's timber industry.

LePage has championed the merger as bolstering the state's forest and farming economies. On Tuesday, the Maine Farm Bureau also testified in favor of the plan, LD 1830, but acknowledged that some of its membership did not support it.

Several farmers told lawmakers they were concerned the bill's plan to have one commissioner split duties between two very different agencies may hurt the responsiveness of the new department.

Conservation groups are unanimously opposed to the plan. Several groups said the mission of the new agency focused too much on the extraction of natural resources and too little on protection. That, combined with the fact that the merger isn't expected to produce any cost savings, had several opponents wondering why the administration was proceeding with the plan.

Some who testified wondered if the merger would cost money.

George Smith, former head of Sportman's Alliance of Maine, urged lawmakers to "insist on an honest and accurate fiscal note." Smith warned that merging two completely different departments could mean combining computer systems.

Smith and conservation groups noted that given the stark mission differences between Agriculture and Conservation, combining the two could create a larger, inefficient bureaucracy.

"It's a fact that small, mission-focused agencies work better than large departments bound up in bureaucracy and strangled by conflicts of competing interests," Smith said.

Patrick Strauch, head of the Maine Forest Products Council, lauded the plan. Strauch said his industry, which is currently overseen by the Conservation Department, had long been envious of the Agriculture Department's promotion and advocacy of farming. Currently, he said, the goals of the forest products industry were a distant second to the Conservation Department's culture of natural resources protection.

The administration says the consolidation would help Maine align its agencies with its counterparts at the federal level. Conservation Commissioner William Beardsley told the committee the new department will feature the same structural and administrative composition, the same budgets and basically the same staffing levels.

Some farmers challenged the administration's claim that the new agency would further the goals of the state's farmers.

Former Democratic Rep. Wendy Pieh said one commissioner may not be able to respond to the state's more than 7,000 farms. The Maine Organic Farmers Association also opposed the bill.

The Maine Farm Bureau supports the consolidation, saying it would create a more streamlined agency.

Cathy Johnson of the Natural Resources Council of Maine said the merger would remove any "high level focus" on conservation.

"We support natural-resources-based economic development, but we don't want to see economic development become the exclusive role of the Department of Conservation," Johnson recently told the Sun Journal. She said the guiding principles described in the legislation appear to exclude goals designed to protect and preserve the state's natural resources.

LePage was explicit in outlining his goals for the new agency. In a written statement last month he mentioned "economy" several times, saying the merger was not just a cost-saving proposal, but one that would create jobs.

"Farming and forestry are an important part of Maine's heritage, and can play a significant role in our economic engine," LePage said. "These industries are important to Maine's future, and it is important we maximize the potential of our natural-resource-based economy to provide jobs and economic prosperity to Maine people."

If it passes, the plan will save \$139,980 in fiscal year 2013 — the cost of one commissioner position. That appears to be the only savings and the only job cut.

The commissioner of the new agency will appoint two deputy commissioners whose duties will be to assist the commissioner "with agriculture, forestry and natural-resources-based economic development."

The fact that upper-level staff will have no conservation directive underscored Johnson's concerns about the mission change. She said the plan appeared to be a strategy to bury the Conservation Department's mission to conserve Maine's natural resources "deep within another bureaucracy."

Johnson and others also question whether the Agriculture Committee would have enough time to adequately address the proposal.

Rep. Kenneth Fredette, R-Newport, speaking on behalf of farmers in his district, said the merger plan lacked details. Fredette recommended forming a stakeholders group to hammer out the specifics.

Former Gov. John Baldacci twice tried — and twice failed — to merge the state's natural resource agencies. LePage's plan may stand a better chance because unlike previous merger proposals, he'll likely only face opposition from environmental groups.

Baldacci's consolidation plan included Marine Resources and Inland Fisheries and Wildlife, which have vocal and effective lobbying organizations.

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Repeal of anti-pesticides policy leads to acrimony, accusations in Scarborough

By [Mario Moretto](#)

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Apr 19, 2012 12:10 pm

SCARBOROUGH — Amid finger-pointing, gavel-pounding and bickering that led to one councilor angrily leaving Wednesday's meeting, the Town Council replaced a 7-month-old policy promoting the use of organic pest management methods with one that allows chemical pesticides on town-owned property.

The new Integrated Pest Management Policy calls for the town to use the least-harmful product available, rather than always using organics, as long as it still manages pests.

The policy also reduces the new Pest Management Advisory Committee from seven members to five, which would effectively remove two pro-organics members. A decision about who to appoint to the committee was tabled until the next Town Council meeting.

The new policy was approved 3-0. Councilor Richard Sullivan recused himself and Councilors Karen D'Andrea and Carol Rancourt abstained, saying they believed the vote was out of order.

"I don't think we should be voting on something that is in violation of our policy rules," D'Andrea said. She scolded councilors during the vote, which led to Vice Chairwoman Judy Roy telling her she was out of order.

"You're all out of order!" D'Andrea shouted back before leaving the meeting.

Adoption of the new policy is opposed by some residents. They are angry at the town for replacing the organic policy that took a year to develop before even implementing the policy or filling the advisory board it created [when it was approved in September](#).

"Let this, at least for one season, play out with the recommendations of the organic policy and go from there," said Loan Lorie, one of about a dozen residents who spoke against the new policy. "I don't understand why something that was decided in September after such a long policy would have to be reconsidered."

Sullivan, who was the lone dissenter in the 4-1 decision to pass the organic approach last year, proposed the replacement policy, which adopts the "Best Management and Practices for Athletic Fields and School Grounds" approved by the Maine Board of Pesticide Control in February.

He first [proposed a replacement policy in March](#), but it was removed from the agenda and not discussed.

The first goal of the Maine board policy is to minimize the human exposure to pesticides. It creates a ranked system, with Level 1 fields getting the most attention, and probably application of pesticides, and Level 4 fields getting little more than mowing and water.

D'Andrea, Rancourt and Elizabeth Peoples – a lawyer working with Citizens for a Green Scarborough, which worked for a year in the Ordinance Committee to craft the organic policy – believe Sullivan had no standing to propose the new policy because he voted in the minority in September.

They cite a Town Council rule about reconsideration, which states that "only those Council members who voted in the majority can sponsor an item for reconsideration, or in the negative on a tie vote, to move a reconsideration thereof at the same, or the next stated meeting."

They also cited a rule saying a petition cannot be reconsidered for at least a year.

Joel Messer, an outside attorney working for the town, said the rules on reconsideration govern only reconsideration at the same or next meeting, and that "petitions" are defined as requests that originate outside the council, not inside. And so, he argues, Sullivan was free to make his request.

Members of Citizens for a Green Scarborough said they're not finished. Some talked Wednesday about taking legal action, others threatened a referendum to bring back the organics-only policy.

"We are pursuing our options," said Peoples, who also runs an organic herb farm, MainelyHerbs, in Scarborough.

Much of the debate Wednesday centered around the peripheral issue of whether Sullivan – who runs a landscaping business, but has never been hired by the town – should have disclosed that his brother, Dan Sullivan, owns a landscaping business that does work for the town.

Rancourt accused Sullivan of violating a disclosure rule because his brother is paid \$40,000 by the Community Services Department for mowing and trimming. Town rules stipulate that councilors must file a disclosure statement if a member of his or her immediate family does more than \$1,000 of business with the town.

Sullivan said he has made no such disclosure, but that he doesn't believe he must because he has no reason to read contracts awarded by Community Services. When councilors vote on the budget, he said, they don't see every contract.

He said he barely talks to his brother, and that his brother doesn't even use pesticides.

"We don't have family functions, and we don't go on trips," he said. "I would never even know if my brother won or lost a contract."

Sullivan demanded that Rancourt retract her accusation. If she doesn't, he said, he will demand a council hearing.

Before leaving the meeting, D'Andrea also accused Town Manager Tom Hall of acting unethically for "not implementing the (organic) policy." Hall later said that no pesticide applications, organic or otherwise, have been made since September, with the exception of an emergency grub management application.

Even under the old policy, though, chemical pesticides may have been used in that case because of an emergency provision that allowed the town manager to opt out of organics.

After the meeting, one resident shouted at Councilor Jim Benedict, who voted for Sullivan's proposal. Others talked with Hall, who said he sought a legal opinion from the moment Sullivan asked about bringing the new policy forward.

Hall tried to assure residents that the council and his staff are still dedicated to using organic pest control techniques, and that the new policy allows them to do so.

"All is not lost, in fact a lot has been gained," he said.

But for some residents, it's not enough.

"You can't go half way on organics," Elisa Boxer-Cook said. "It's all or nothing."

Eddie Wooden, a local business owner and philanthropist who supports Citizens for a Green Scarborough, said the fight is not over.

"We're not going away," Wooden said. "We're going to be very aggressive about this."

Mario Moretto can be reached at 781-3661 ext. 106 or mmoretto@theforecaster.net. Follow him on Twitter: [@riocarmine](https://twitter.com/riocarmine).

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Scarborough councilors feud over pesticides, conflict-of-interest claims

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Posted: Wednesday, April 18, 2012 10:46 am | Updated: 3:10 pm, Wed Apr 18, 2012.

By Duke Harrington dharrington@keepmecurrent.com | 1 comment

SCARBOROUGH – Two Scarborough town councilors have accused a third of hiding a conflict of interest and are calling for his public censure, along with the removal of a pest control ordinance he authored from consideration at the council meeting set for Wednesday night, April 18.

However, Councilor Richard Sullivan is denying charges leveled by his peers, Carol Rancourt and Karen D'Andrea, that he'd benefit personally from rewriting a synthetic pesticide ban adopted by the council last fall. Instead, he's calling their accusations "dirty Washington politics come to small-town government," while returning that Rancourt has conflicts of her own to address.

In September, Sullivan was the only councilor to vote against a new pest control policy that banned the use of synthetic pesticides from use on town-owned property without permission of a newly created seven-person advisory committee. The policy, drafted by D'Andrea as an ordinance, was adopted 4-1 after more than a year of wrangling and downgrading at the committee level.

Sullivan's replacement version, based on "best management practices" adopted by the Maine Board of Pesticide Control in February, would encourage but no longer mandate the use of organic pesticides. Instead, it seeks to "minimize human exposure" through a system of postings and notifications.

Sullivan runs a landscaping company when not in uniform for the Portland Fire Department.

In letters addressed to Council Chairman Ron Ahlquist dated April 16 and 17, Rancourt and D'Andrea note that a company named "RJ Sullivan" appears on Scarborough's landscape maintenance vendors list, holding a \$40,000 contract to maintain grounds at the Scarborough Public Library and the town's three primary schools at Eight Corners, Pleasant Hill and Blue Point.

Although both women acknowledge in their letters that Sullivan's brother, Dan Sullivan, is principal of the company in question, both cast aspersions on Richard Sullivan's conduct. While D'Andrea claims Sullivan has pecuniary interests in his brother's contract with the town "based on information provided to me by a Scarborough citizen," Rancourt "respectfully demands" he be subject to a formal censure.

D'Andrea said in an email Tuesday afternoon that her "information" is held by Citizens for a Green Scarborough, an advocacy group that championed her version of the Organic Pesticide Management Policy now on the books. The Scarborough group could not be reached Tuesday.

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Taste of the Wild

Scarborough's Town Council Rules, Policies and Procedures Manual compels councilors to reveal conflicts of interests. It also requires that each councilor file with the town clerk in writing and under oath by April of each year the name of any person doing business with Scarborough in excess of \$1,000 from whom the councilor or an immediate family member receives "money or other thing of value" greater than \$1,000.

Assistant Town Clerk Carrie Noyes said she is not aware of any such disclosure ever made by Sullivan.

"That's because we're completely separate," said Sullivan. "I have never received any kind of gain from his business. We have nothing to do with each other."

Sullivan explained that his company, founded when he was 17, is RJ Sullivan Landscaping. His brother's company, inherited from their father, Richard J. Sullivan Sr., is RJ Sullivan Lawn Care. Not only are they two separate business, Sullivan said, there is no possibility of a financial link.

"My brother and I barely talk," he said Tuesday afternoon. "It's a sad thing, be we don't get along that good."

In an email Tuesday, D'Andrea repeated her claim that, "from evidence that was sent to me, it does appear that Richard does gain materially from the business."

"But even if he does not directly benefit from the business, he still must report the conflict of interest because of the relationship with his brother," said D'Andrea, via email. "The rules do not make an exception for people who barely speak to their immediate family member. The contract is for \$40,000 and is not a small amount of money no matter how you look at it.

"Councillor Sullivan also has a direct 'special interest' because of his business and that also must be disclosed," said D'Andrea. "There are no exceptions to this reporting requirement. There are very good reasons for requiring these disclosures and there is nothing unusual about these kinds of policies and rules. Bottom line is that he must report both the special and financial conflicts of interest which he did not report or disclose."

For his part, Sullivan said not only does he not benefit from his brother's business, his brother stands no chance of reaping rewards from his policy proposal, which would theoretically lower pest control costs, given the higher cost of organic treatments.

"He doesn't do anything like that," said Sullivan. "He just strictly mows lawns."

"Pest control services are not in the scope of Dan Sullivan's contract with the town," Town Manager Tom Hall confirmed Tuesday. Hall added that Sullivan would have no reason to recuse himself from any lawn care votes involving his brother, as Rancourt and D'Andrea claim, because that kind of contract bidding is handled entirely by community services. Councilors only see a bottom line dollar request from Community Services Director Bruce Gullifer, said Hall.

"My bother has had that mowing contract for 19 years – long before I became a councilor," said Sullivan, "but he could have lost it last year for all I'd know."

Ron Ahlquist said Tuesday he will be out of town, leaving control of the April 19 meeting to vice chairwoman Judy Roy. Roy could not be reached but Hall said that, as of 5 p.m., the pesticide policy proposal had not been stricken from the agenda.

"It better not be," said Sullivan. "I'll be ripped if we put that off another two weeks."

Sullivan's policy, set to supercede current rules, was originally on tap for the March 21 council meeting. However, it was pulled at the last minute by Ahlquist after Elizabeth Peoples, a Scarborough attorney and organic farmer working with Citizens for a Green Scarborough, questioned if council rules were followed in advancing the policy directly to the full council, with no initial stop in committee.

Sullivan said charges now leveled against him by Rancourt and D'Andrea are "just more delaying tactics to muddy the waters."

Still, he admits his policy rewrite does strike two positions on the pest management advisory committee, slated to go Wednesday to two people – Mark Follansbee and Marla Zando – whom he claims have signaled they "are absolutely against any kind of synthetic pesticides." The town needs the flexibility to use non-organic control measures, as necessary, he claims, to eradicate grubs on town athletic fields.

"They are really out of control," Sullivan said, "and this policy is a good common-sense compromise between organics people and synthetics people that's been endorsed by a bunch of state people."

But if push does come to shove Wednesday, Sullivan said he's ready to fight back. While he's never seen his brother's name connected to anything when casting votes, Sullivan said Rancourt does see the line-item for Scarborough's annual donation to her employer, the Maine Agency on

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Aging.

"Maybe I should make some ethics charges against her," said Sullivan.

"These are serious charges for councilors to level against one of their peers," said Hall, referring to the letters from Rancourt and D'Andrea. "But it would be unfortunate for things to devolve into that kind of tit-for-tat."

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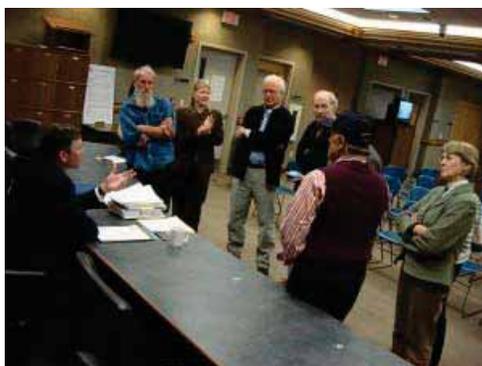
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Conflict charge on Scarborough pesticide flap heads to hearing

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Posted: Wednesday, April 25, 2012 12:56 pm

By Duke Harrington
dharrington@keepmecurrent.com | 0 comments

SCARBOROUGH – Within 24 hours late last week, Scarborough did two 180-degree turns on an updated pesticide-use policy, ending up exactly where it started – with the exception that one town councilor now faces a censure hearing regarding conflict-of-interest charges.

When the Town Council appeared to overturn its nascent pest control policy April 18 with one less restrictive of chemical use, Councilor Karen D'Andrea had her protests gavelled out of order. In response, she declared with a flourish of the arm, "You're all out of order." She then grabbed her jacket and left the meeting before it was over, telling a group of 37 residents gathered for the decision, "Sorry, folks. I tried my best."

But within 24 hours, Town Manager Tom Hall circulated an email advising that the decision had not carried after all, despite a 3-0 vote. Scarborough's Town Council Policy Manual requires four affirmative votes to pass any measure, he said. That was impossible thanks to the absence of Council Chairman Ron Ahlquist, the decision of Councilor Richard Sullivan to recuse himself because of conflict-of-interest charges, and abstentions by D'Andrea and Councilor Carol Rancourt.

Scarborough pesticide meeting

Scarborough Town Manager Tom Hall, seated, fields questions after the April 18 council meeting from residents unhappy with the adoption of a new policy governing use of pesticides on town property. Within 24 hours, the vote was ruled improper and the decision overturned. (Staff photos by Duke Harrington)

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Councilors James Benedict, Jessica Holbrook and Judith Roy all endorsed the update, which had been sponsored by Sullivan and is less demanding of organic pesticide use than the earlier version, written

by D'Andrea and adopted in September by a 4-1 vote, with only Sullivan objecting.

"I have to view the existing policy to be still in effect and will act accordingly unless directed otherwise," said Hall, on Friday.

That resets the status quo on the use of chemicals to fight grubs and other insect infestations in Scarborough – i.e., don't – but leaves Sullivan subject to censure proceedings for suggesting a different path.

In separate letters submitted by D'Andrea and Rancourt, Sullivan was accused of failing to disclose that his brother, Dan Sullivan, holds a \$40,000 contract with the town to mow lawns at Scarborough's public library and three elementary schools.

The council policy manual calls on each councilor to file with the town clerk by April 1 the name of any person holding a town contract worth more than \$1,000 from whom the councilor "or a member of his/her immediate family" received \$1,000 or more during the preceding year.

Sullivan volunteered to recuse himself from the vote, subsequently winning a 3-2 decision – with D'Andrea and Rancourt opposed – that allowed him to take part in debate. However, the fate of Sullivan's policy proposal has no bearing on disciplinary action leveled against him. Although not yet scheduled, a censure hearing will be held, said Hall.

"There's no pulling back," he said. "An ethics charge is a very serious thing. They made those allegations and it has been my advice to the council chair that they [the full council] should convene as a body, deliberate, and make a ruling."

Organic policy

The drive to ban synthetic pesticides in Scarborough began several years ago with local businessman and philanthropist Eddie Woodin. A lifelong birder, Woodin questioned if chemical pesticides could travel from weeds to worms to birds, much like the infamous, but once widely used insecticide, DDT. Woodin has more recently raised the specter of upwind turf chemicals being behind unexplained illnesses at Wentworth Intermediate School, rather than the aging building itself, as commonly supposed. In 2010, Marla Zando, then executive director of the Scarborough Land Trust, sounded a similar note of concern for children, citing reports that appear to link certain synthetic pesticides to an array of childhood cancers, as well as attention deficit hyperactivity disorder.

That prompted D'Andrea to draft an ordinance banning the use of synthetic pesticides, which, after a year of debate and the formation of Citizens for a Green Scarborough, eventually saw life in diluted form as a council policy, applicable only to town-owned lands.

Still, it specifically banned the use of synthetic pesticides without a waiver issued by a newly created pest control advisory committee or an "emergency" application approved by the town manager.

That was seen as a win by members of Citizens for a Green Scarborough like Mark Follansbee, who has a doctorate in pharmacology from Penn State. As a 15-year contracted toxicologist for the Environmental Protection Agency, Follansbee was concerned that, prior to policy adoption, no apparent preference was given to the use of organic pesticides in public parks and athletic fields.

"During one Ordinance Committee workshop, Community Services was kind enough to give us an accounting of products used in 2010," recalled Follansbee on Friday. "The information provided showed that only synthetic herbicides were being applied. No less-toxic approaches were apparently even attempted."

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Follansbee was first to sign up for the seven-member advisory group created in the new policy. However, in addition to relaxing restrictions on synthetic pesticides, Sullivan's policy also knocked the committee from seven to five members, specifically targeting posts for which Follansbee and Zando had been nominated.

"They are out of control – absolutely against any kind of synthetic pesticides," Sullivan explained in an interview before Wednesday's debate.

Integrated policy

A career landscaper when not fighting fires for the city of Portland, Sullivan first tried in March to present an "integrated policy" – one that encourages but does not absolutely require the use of organic pesticides.

Sullivan's first draft, based on "best management practices" adopted by the Maine Board of Pesticide Control in February, was pulled at the last minute from the March 21 agenda when questioned by Elizabeth Peoples, an attorney for the citizens group and a Scarborough resident.

Among other issues, Peoples argued that by voting against D'Andrea's organics-only policy in September, Sullivan was prevented from presenting any alternative for at least one year.

In an April 18 memo, Town Attorney Joel Messer of Bernstein Shur said rules on reconsideration apply only to the first meeting immediately following a decision. The one-year limit applies only to citizen petitions seeking to repeal an ordinance vote, he said.

Once the question of standing was sorted out, Sullivan tried again, albeit with a few changes. His initial proposal, for example, had completely exorcised the citizens advisory committee. Sullivan also took time to incorporate a matrix from the state plan that separates soils into four classes, advising the type of pesticide treatment best suited to each under certain circumstances.

Elisa Boxer-Cook, a member of the green group, chided the council by saying, "You can't go half way on organics – it's all or nothing." But Sullivan countered that it takes both time and money to prep soils for organic treatments.

That transition can be expensive, he said, and cost taxpayers more than an approach that tries to prevent infestations, rather than fighting them.

Violations of policy

Sullivan was not the only person accused of trying to overturn the organics-only policy. D'Andrea also suggested Hall may be culpable.

"It is perhaps another violation of our rules when the town manager is directed to implement a policy and does not do so," she said, referring to the seven-month lag between creation of the pesticide advisory committee and presentation Wednesday of a slate of appointees.

Hall said Friday there simply weren't any applicants until word circulated of Sullivan's initial plan to eliminate the committee, other than Follansbee and Zando, and they were invited to comment on bid specs for this year's turf work. However, Hall did admit the openings "were not well advertised," adding that "as early as March," when he learned of Sullivan's proposal, he slacked off on filling a committee that might cease to exist.

Still, Hall said, no pesticide of any kind has been applied to public property in Scarborough since September, other than one "emergency" grub treatment in the days after the new policy passed. Therefore, there was nothing for the advisory committee to advise on.

But Peoples said Citizens for a Green Scarborough members were being discouraged as early as January.

"We were told by [Community Services Director] Bruce Gulifer not to worry about it, that the policy probably would never be implemented," she said, following Wednesday's meeting.

Hall said that after Sullivan's first attempt to alter the pest policy was pulled in March, the citizens group was invited to a work session to craft a compromise policy.

"They're the ones who refused to work with us," agreed Sullivan.

But Peoples said there was a good reason for that.

"Tom Hall requested that it be a private meeting and we thought that was a violation," she explained, saying only councilors Ahlquist and Sullivan would have participated, to keep from triggering public meeting laws. "He wanted it kept quiet and we thought it should be a public process, just like the first policy went through for more than a year."

Despite clamoring about conflicts of interest, which Sullivan called "showboating" to derail his proposal, most of the dozen speakers at the meeting last Wednesday seemed most concerned

with an apparent rush to judgement. In fact, more than one person, including D'Andrea and Rancourt, said the two policies share more in common than not.

Even Follansbee, in comments Friday, acknowledged that the best management practices at the core of Sullivan's approach "are outstanding." The only concern, he said, is that the new policy would have "turned back the clock" to a time when Scarborough supported organic treatments in principle, but not practice.

"I am mostly disappointed that the opinion and will of one individual is being put ahead of the will of the citizens, disappointed that a quick and closed approach is being used to circumvent policy that was established after a year-long, inclusive process," he said.

"I am very well aware of the policies and procedures of the Town Council," agreed former two-term councilor Sue Foley-Ferguson. "They are put there so that there is a public process – not just for one individual who is concerned about a policy that they didn't win the vote on."

"We sat on this policy for six months and not one person had a complaint," said D'Andrea. "Then, all of a sudden, boom, one day there's a new policy. We get to vote on it once. There's no time for public process. There's no workshop. There's no explanation. All we hear is, 'It's a new common-sense approach.' That's baloney. It reeks of horse hockey."

"Well, that's quite enough of the superlatives at this time," said Roy, acting as chairwoman in Ahlquist's absence, as she called for the vote.

In his email about Wednesday's vote, Hall pointed out that abstentions by D'Andrea and Rancourt were improper under the policy manual. Outside of a "real or perceived conflict of interest," the rules say, "every member of the council present must vote" on every issue.

"Nobody can force me to vote on something that I consider [to be] an illegal action," said Rancourt in a telephone interview on Friday.

Still, neither woman faces disciplinary action at this time. Instead, it is Sullivan who is on the hot seat and predicting he will be "completely vindicated."

Sullivan said after the meeting last Wednesday that he would decide after consulting with his attorney if he wants his hearing held in public or in executive session.

Sullivan argued he has no conflict of interest because he makes no money from his brother's business, and because his brother only mows lawns. His proposals would not have impacted his brother's contract with the town at all, said Sullivan, adding that he had not registered the relationship in the clerk's office because he interpreted the rule to mean he had to benefit personally from the business to warrant giving notice. Also, he pointed out, Gulifer only presented a bottom-line dollar figure when bringing lawn care contracts to the council.

"We barely talk to each other," Sullivan said of his brother. "He could have lost that contract for all I'd have known."

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Selectmen Vote Not To Support Pesticide Article [POLL]

Town officials said Tuesday that Needham's Integrated Pest Management system was already aimed at the reduction of chemical applications and that a ban proposed by citizen's petition on the May 7 Annual Town Meeting warrant was unnecessary.

By [Becca Manning](#) | [Email the author](#) | April 26, 2012

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Needham selectmen are not supporting a change in the town's land management policy that would require departments with pests such as mosquitoes and invasive plants and insects.

The board voted 5-0 on Tuesday, April 24 against endorsing an article that appears on the May 7 Annual Town Meeting petition, the article seeks to require organic lawn and garden care on all town-owned properties in an effort to create a healthy environment.

On Tuesday, selectmen heard a presentation on the town's existing [Integrated Pest Management](#) program from representatives of the Public Works and Park and Recreation departments as well as Parks and Forestry Superintendent Ed Olsen and Director of Public Works Ed Olsen.

Olsen said the town already uses few pesticides, often turning to these chemicals only as a last resort to deal with serious departments reserved the right to use chemicals if needed, such as by the [Norfolk County Mosquito Control Project](#) or plant-damaging insects such as Japanese moths.

Board of Health member Stephen Epstein said there were actually risks in not using pesticides in some cases, pointing to Massachusetts from the mosquito-borne West Nile virus.

Later in the meeting, selectmen said they were satisfied that the town was already doing what it could to reduce the application of chemicals on town properties.

"I do believe the goal of trying to get to a point where we're not using pesticides at all is a good goal and an admirable goal," Selectman Jerry Wasserman said. "But I don't believe we can meet the requirement for this article right now and still maintain our fields."

Selectman Moe Handel said the town was already working toward a more organic approach to pest control.

"I think the article is unnecessary with respect to our current policy," he said.

Should Needham use only organic materials instead of chemical pesticides?

- Yes
- No
- Undecided

Total votes: **18**

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Class to teach principles of healthy homes

[Connections](#) | Wednesday, March 21, 2012

RUMFORD — The River Valley Healthy Communities Coalition and the Maine Health Access Foundation are sponsoring a Healthy Homes Training from 9 a.m. to 3:30 p.m. Thursday, March 29, at 49 Franklin St.

Many people live in homes and apartments that put them and their families at risk of sickness and injury.

Participants will learn about the seven core principles of a healthy home and about the Maine Healthy Homes Initiative.

Speakers will include Eric Frohberg, Maine Healthy Homes/Lead Prevention; Gary Fish, Board of Pesticides Control; Christine Crocker, Maine Indoor Air Quality Council; Katharyn Zwicker, Maine Injury and Violence Prevention Program; Jim Braddick, Maine Asthma Prevention and Control Program; and Tina Pettingill, Smoke Free Housing Coalition of Maine.

The registration fee is \$15. For more information, contact Patty Duguay at 364-7408 or email rvhcc@zwi.net.

April 15

Deirdre Fleming: Talks hope to calm Lyme fears

The news last week that the Maine Center for Disease Control predicts 2012 will be the worst year for Lyme disease in Maine did not shock the people of Long Island.

LYME DISEASE PANEL

WHAT: A panel of experts who will speak about the threat of Lyme disease and prevention

WHEN: 11 a.m., Saturday

WHERE: Long Island Learning Center

COST: Free, open to public

PANELISTS: Gary Fish, Maine Board of Pesticides; Chuck Lubelczyk, Maine Vector-borne Disease Laboratory; Joe Poisson, Atlantic Pest Solutions; Sherry Juris, Atlantic Pest Solutions biologist; Kate Colby, Maine Center for Disease Control; Sara Robinson, Maine CDC; Scott Lindsay, Maine Department of Inland Fisheries and Wildlife; Craig Holbrook, veterinarian

The **Maine CDC** is also holding free seminars at the following locations:

- **L.L. Bean**, Freeport, Thursday and Friday
- **Epic Sports Gear**, Bangor, May 4
- **Cabela's**, Scarborough, May 26

And the report released last week by the Maine Medical Center's Vector-Borne Disease Laboratory that showed the disease will spread across Maine by 2050 did not rattle this small island town in Casco Bay.

They confronted their Lyme disease nightmare two years ago when it became clear the number of confirmed cases on the island was mounting.

This is why Emily Jacobs, the town's health officer, put together an impressive panel of Lyme disease experts who will gather on the island Saturday.

"The safest thing to do is to personally protect yourself. But you can't tell people to dress up like it's January, all covered up when it's a hot summer day. I'm hoping a large audience shows up, and everyone hears the same thing," Jacobs said.

After seeing more and more cases of Lyme disease on the island, Jacobs decided to try to get a handle on how prevalent the disease was there. So two years ago, she sent out a questionnaire to as many island residents and summer visitors as she could.

The response showed there were at least 43 people who spent time on Long Island who had a confirmed case of Lyme disease. On an island that has 220 year-round residents and as many as 900 summer

visitors, Jacobs said the number was alarming.

Moreover, among the 220 year-round residents, there were 27 who reported to have a confirmed case of Lyme disease, more than 10 percent of the island's winter population.

"Once I put out the statistics, suddenly there was a feeling on the island that we have an epidemic," Jacobs said.

Concern has not died down. And two weeks ago, a 10-year-old boy became the most recent case of Lyme disease among the year-round residents.

So this spring, Jacobs decided to do the best thing she could for her island community. She asked as many experts on Lyme disease in Maine as she could find to come speak on the island.

She didn't just ask for a biologist from the Maine CDC, she asked for two, as well as a biologist from a private pesticide company, one from the Maine Board of Pesticides, a biologist from the Maine Vector-borne Disease Laboratory, and also one from the Maine Department of Inland Fisheries and Wildlife.

In all, eight experts will address the topic at 11 a.m. Saturday.

Vicky Delfino, president and founder of MaineLyme, a nonprofit that holds seminars to educate Lyme disease victims, said she's never seen a Lyme seminar in Maine that will speak so thoroughly to causes and prevention.

"I think Long Island has been concerned for a while. I did a presentation for them two years ago before MaineLyme was formed. I know they had a number of cases of Lyme then, a large number of cases," said Delfino, who has had Lyme disease for several years.

Jacobs said she felt that to quell the mania and help protect her neighbors and friends, they needed to be completely educated together.

"People are confused by it. I hope summer people come open up their cottages and come out, and everyone hears the same thing. That's my purpose, good or bad. So we can understand it together, and so the mania dies down," Jacobs said.

Staff Writer Deirdre Fleming can be contacted at 791-6452 or at: dfleming@pressherald.com

Twitter: [Flemingpph](#)

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Ticks a Growing Problem in Maine

04/09/2012 10:20 AM ET

A warmer than normal winter and an early start to spring have Maine health officials concerned about tick-borne diseases.

PORTLAND, Maine (AP) _ Susan Elias, a biologist at Maine Medical Center's Vector-borne Disease Laboratory, said after two days of unusually high temperatures in March reports of ticks started "rolling in."

The biggest problem in Maine is the deer tick, which spreads Lyme disease in humans and pets.

Adult ticks are active and looking to feed earlier, and a lack of snow has meant it's easier for them to find a host, animal or human.

Sheila Pinette, director of the Maine Center for Disease Control, tells The Portland Press Herald there were a record-high 981 reported cases of Lyme disease in the state last year, and about the same are expected this year.

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Fairfield Company Develops Natural Tick Pesticide

Tick-Ex, which is made from a strain of fungus, will be commercially available in 2014.

By [Chandra Johnson Greene](#) | [Email the author](#) | March 8, 2012

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Related Topics: [Connecticut](#), [Ticks](#), and [deer population](#)



A Fairfield-based company has developed a natural pesticide made from a fungus that could help control the tick population, [according to the Connecticut Post](#).

The product, which has been named Tick-Ex, is based on field trials performed by the Connecticut Agricultural Experiment Station and will be available to the public in 2014.

The strain of *Metarhizium anisopliae* fungus used in the product is deadly to the black-legged tick, but won't harm other insect like synthetic pesticides do. The fungus is found naturally in soil and after being tested on residential properties in northwestern Connecticut, 74 percent fewer ticks were found.

Researchers in Maine are encouraged by the news, particularly since it could be used as an alternative to pesticides, according to [a report on the Main Public Broadcasting Network](#).

The Connecticut Post article reported that Connecticut has the highest number of [Lyme disease](#) cases in the U.S. and has been rising steadily due to the high deer population. "Local Voices" blogger [Peter Wild](#), who is the executive director of Stamford-based Time for Lyme, warned readers last month that the [unusually warm winter season has allowed ticks to remain active](#).

For more information on Lyme disease:

- [Lyme Disease Education](#) Web page at the University of Connecticut website
- [U.S. Centers for Disease Control](#) Web page on Lyme Disease

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Tick-Attacking Fugus Shows Promise in Battle Against Lyme Disease

03/05/2012 Reported By: [Tom Porter](#)

Researchers in Maine are encouraged by the development of a new, natural pesticide for controlling the population of black-legged ticks that carry lyme disease. State scientists in Connecticut have been working with a European biotech company to devise a non-synthetic pesticide based on a strain of fungus which kills the ticks, but appears to cause little damage to the environment.



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The proper name for the latest weapon in the ongoing war against Lyme Disease is the "F52 strain of Metarhizium Anisopliae fungus." But for the sake of simplicity, let's use the product name, "Tick-Ex."

It could be commercially available in two years time, and Chuck Lubelczik (above) is excited by its potential. "I think it's actually a really good idea for product like this to come out now," he says.

Lubelczik is a field biologist with Maine Medical Center's Vector-Borne Disease Lab in South Portland, a non-profit research institute dedicated to studying and controlling lyme disease and other emerging tick-borne diseases. "We are going through a period, at least in Maine, where a lot of folks that are having problems with ticks are actually running into concerns

about using things like synthetic pesticides to control ticks," he says.

Tick-Ex uses no synthetic chemicals, relying instead on a strain of fungus that occurs naturally in soil, and that targets pretty much little else other than the black-legged tick. "This product, if it proves to be pretty effective, would be something that you could spray potentially near a wetland, close to the coast," Lubelczik says. "So you might not have impacts to things like shellfish or vernal pools, and you'd still be able to control your ticks in those areas."

Lubelczyk says the institute wants to start trying out Tick-Ex as soon as possible. "We're hoping to be one of the product-testers for this when it comes out in a trial phase this spring," he says. "We're going to be applying, along with several other people, for one of 27 sites on the eastern seaboard to be doing field tests.

Lyme disease is caused by the prolonged bite of an infected tick--often a deertick. Symptoms can include joint pain and fatigue, but if not treated it can damage the heart and nervous system. The number of reported cases began to soar in Maine a few years ago, and now stands at around 1,000 a year.

The infectious disease was first identified in the U.S. in the town of Lyme, Connecticut, in the mid-1970s, and the Constitution State is still very much ground zero in the struggle against the illness. It's appropriate, then, that researchers in Connecticut should identify the deer tick's possible "fungal nemesis."

"It's another valuable tool for the control of ticks--I mean lyme disease cases continue to increase," says Kirby Stafford, the chief entomologist for the state of Connecticut. Stafford helped conduct the field trials that have already taken place. While not as effective as synthetic treatments, initial trials still indicate a pretty high success rate for Tick-Ex.

"We got about up to 75 percent control, sometimes a little better with it," Stafford says, "as opposed to synthetic chemical insecticides, where you're looking at 85 to 100 percent."

And Stafford says these numbers were good enough for federal regulators. "As result of those studies, which are published in part, they were able to get U.S. EPA registration, as well as registration in all 50 states," he says.

Stafford says much of the the initial work identifying the fungus and studying its potential was done by a Fairfield-based research group called Earth Biosciences. But he says they didn't really have the financial clout to develop it.

Then in 2006, Earth Biosciences was acquired by a company from Denmark called Novozymes. Self-described as a "world leader in bio-innovation," Novozymes employs more than 5,000 people in 30 countries.

Francis Leier is the company's global business development manager. He hopes the product will hit the shelves by 2014, but because field trials are still ongoing, he can't indicate how much Tick-Ex will cost. He says that depends on finding other applications for the product.

"The more uses we have, the better the cost position will be on it," Leier says. Right now, he says, the fungus is being tested as an effective pest-control agent for a number of fruits and vegetables. "There's more uses outside and beyond the use of ticks."

The product's tick-killing potential, though, is the aspect that's attracted most attention in Maine. "Anything that can decrease the ticks decreases the exposures to humans and decreases the possibility of Lyme disease," says Maine State Epidemiologist Dr. Stephen Sears.

Sears says even if your backyard is fully sprayed with pesticide, you should still cover up when walking in the woods, and check yourself for ticks. While it's too early to say how many ticks will be around this summer, Dr. Sears says the mild winter and the comparative lack of snow means more people could be potentially exposed to deerticks.

Photos by Tom Porter.

 [Return](#)

BANGOR DAILY NEWS

Scientists find fungus that kills Lyme disease-carrying ticks

By Vinti Singh, Connecticut Post
Posted March 04, 2012, at 5:58 a.m.

BRIDGEPORT, Conn. — Local scientists have found a way to control the ticks responsible for passing Lyme disease on to humans. A new natural pesticide, derived from a strain of fungus that is deadly to the black-legged tick could help keep tick populations under control.

Unlike some synthetic pesticides that can be dangerous for more than just ticks, the fungus does not harm honeybees, earthworms or other beneficial insects.

The product was developed by a Fairfield-based company that was bought out by the Danish industrial biotechnology company [Novozymes](#).

The Connecticut Agricultural Experiment Station's field trials of the fungus helped obtain federal Environmental Protection Agency registration. Novozymes has built a plant in Canada to mass produce the product, Tick-Ex.

It will be commercially available in 2014, said Kirby Stafford, the station's vice director and chief entomologist.

"A lot of people do have their yards sprayed with pesticides, and they are quite effective, because synthetic materials will give you an 85 to 100 percent success rate," Stafford said. "But there are a special number of people who don't want to use them. The (organic product) may be slightly less effective, but it's giving people options. It certainly would fit in to organic land care."

The pesticide is made of the F52 strain of the *Metarhizium anisopliae* fungus, which occurs naturally in soil. The station tested it on residential properties in northwestern Connecticut and found up to 74 percent fewer ticks after treatment.

Although rates dipped slightly in 2010, the number of people in Connecticut with Lyme disease has been steadily rising, according to the federal Centers for Disease Control and Prevention. Connecticut has the nation's highest number of cases, relative to population. The first symptoms of the disease include headache, fever and rashes. But if left untreated, the disease can spread to the joints, heart and nervous system.

The overabundant deer population is one reason the disease is so widespread, according to the state Department of Public Health. Black-legged ticks feed on large mammal hosts, which in Connecticut are usually deer.

Many Lyme disease experts have said the solution is to cull the deer, but research shows that is only really effective when the deer are culled to very low numbers, said Louis Magnarelli, director of the Connecticut Agricultural Experiment Station.

The station has researched a number of methods to control Lyme disease.

It found nootkatone, a component of essential oil from Alaskan Yellow Cedar and grapefruit is toxic to ticks, and is highly effective.

As tests wind down, there is a small chance a company will pick it up because the cedar oil is only produced at a grade suitable for cosmetics and foods, making it expensive. Until production is scaled up for more commercial uses, it won't be used to eradicate ticks, Stafford said. The station has also tested a garlic spray product, which suppresses tick activity for around two weeks. Scientists in Maine discovered that a rosemary oil product, EcoEXEMPT, will eradicate ticks for at least two weeks.

The nationwide tick control research community is pretty small, Stafford said. Between 2001 and 2012, the state

Department of Health and the agricultural experiment station have received a little more than \$2 million for public outreach and tick control research from the CDC. The CDC was expected to hand out two tick control grants in 2011, but based on available funds ended up only distributing one, which went to a research laboratory in Rhode Island.

Studies have found the fungus strain is also effective in killing bed bugs, but it won't be marketed for that use just yet.

"I can't see spreading the spores of this fungus into a bedroom," Stafford said. "But it begs for a formulation of how you expose it to just the targets and not the rest of the environment."

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<http://bangordailynews.com/2012/03/04/health/scientists-find-fungus-that-kills-lyme-disease-carrying-ticks/> printed on May 3, 2012

BANGOR DAILY NEWS

Weather conditions could mean bee die-off in Maine

The Associated Press

Posted April 30, 2012, at 6:26 a.m.

AUGUSTA, Maine — A mild winter and unseasonably warm early spring have created conditions reminiscent of 2010, when an explosion in mite populations killed off many bee colonies in Maine.

Tony Jadcak, state apiarist and bee inspector, is warning beekeepers to monitor for the varroa mite, an external parasitic mite that attacks European honeybees.

“The bees are coming out, but so are the parasitic mites,” Jadcak [told the Kennebec Journal](#). “What I’ve seen in my inspections is elevated mite loads because of the good health of the honeybees. If it tracks like it did in 2010, we’ll have a huge die-off in the fall and winter.”

Maine beekeepers have suffered enormous losses since the parasite from the Asian honeybee was introduced into the United States in the mid-1980s.

And parasitic mites are not the only concern for beekeepers.

Beekeepers and some scientists say pesticides are killing bees and weakening their immune systems, making them more susceptible to pathogens. They say it could contribute to colony collapse disorder, in which all the adult honeybees in a colony suddenly disappear or die.

Bees are vital to U.S. agriculture because they pollinate many flowering crops, including blueberries.

Maine doesn’t have enough bees in the state to pollinate all the crops, so thousands of bee hives are brought in by commercial beekeepers every year.

<http://bangordailynews.com/2012/04/30/outdoors/expert-maine-conditions-could-mean-a-bee-die-off/> printed on May 4, 2012

Ash pest found closer to New England

By Associated Press | Wednesday, April 18, 2012 | <http://www.bostonherald.com> | [Local Coverage](#)

ALBANY, N.Y. — The invasive beetle that has destroyed tens of millions of ash trees over the past decade has been found east of the Hudson River for the first time, marking its closest known threat to New England, researchers in New York told The Associated Press Wednesday.

But the discovery of an emerald ash borer infestation in the Dutchess County village of Rhinecliff last month may signal a victory in the battle to stem the pest's spread: Foresters believe the colony was caught less than a year after it got established, a big step given that the beetle can go unnoticed for years.

The larval beetle tunnels under the bark, eventually destroying a tree without any sign until its foliage yellows and dies. The shiny green adults are only about half an inch long and tend to fly well above the ground, making them hard to spot.

"It's rare that infestations are found this early," said Nate Siegert, a U.S. Forest Service entomologist who has been working in Rhinecliff this month. He credited state Department of Environmental Conservation foresters for taking steps that led to the discovery.

Ash trees, prized as a commercial hardwood and a feature in urban plantings, have been ravaged through much of the Midwest and into the mid-Atlantic and Northeast since the Chinese beetle was first discovered near Detroit in 2002. Borer infestations were found in western New York in 2009, but experts say the Hudson Valley colony could have started years before that, possibly after catching a ride across the state in a load of wood.

The main population has been spreading gradually at a pace of about 2 to 3 miles a year, but "satellite" colonies leapfrog ahead, mostly by hitchhiking in loads of logs or firewood.

New York became a leading edge for research and control efforts after a major infestation was discovered on the west shore of the Hudson in 2010, about 150 miles east of colonies discovered elsewhere in New York since 2009.

Researchers set out purple traps and stripped bark from trees last year, eventually mapping finds of beetle larvae in a 225-square-mile area running north from just below Kingston, bounded on the east by the river and parts of the Catskills in the west.

Jeff Rider, a DEC supervising forester, said 28 "trap" trees on the east shore were also girdled — stripped of a band of bark — to attract any beetles that may have made it across.

Three of those trees just below the Kingston-Rhinecliff Bridge about 25 miles from the Connecticut and Massachusetts borders were found with small infestations in March, he said. That sent researchers ranging through a 3-mile radius around each, taking samples from 78 other ash trees. Rider said none of those trees was infested, but an additional 100 trees have now been girdled in the area.

He said plans are being made to quarantine moving ash material in Dutchess County, but he thinks that may be limited to particular towns, not entire counties like across the river. People can be fined for moving firewood 50 miles beyond its origin, a regulation meant to thwart ash borers and other invasive pests.

Rider thinks the latest infestation involved adults that crossed the river during last summer's flying season.

Forestry experts in New England have been watching for any sign of the ash borer, typically relying on the familiar purple traps.

"They're gearing up, knowing they're eventually going to have it," Rider said. "We're just trying to buy them some time."

"This is a battle worth fighting," said Chris Martin, the state forester in Connecticut. "The ash tree resources in New England are phenomenal."

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March 11

Maine Gardener: Rest for pest police? Not gonna happen

By TOM ATWELL

Pests from both the plant and animal kingdoms are continuing to invade our local gardens, and professionals in the industry have been getting reports at their winter meetings. All of the news is not bad, however. Some problems have eased over time, and some are not as bad as people initially thought.



Boxwood Blight has not been found in Maine yet, but it does exist in Massachusetts. Its potential for destruction has plant pros worried.

Courtesy UMass Extension

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One big problem is Boxwood Blight, which has not been found in Maine yet but has made it to Massachusetts and is likely to arrive in Maine soon.

Bruce Watt, a plant pathologist with the University of Maine Cooperative Extension in Orono, told professional landscapers meeting at O'Donal's Nursery in Gorham that the blight is a fungus that first shows up as brown cankers on boxwood leaves and defoliates the plant. The blight also infects the stems, causing dark brown or black lesions.

So far, the blight has been found to damage all varieties of boxwood, and there is no known control.

"You can't eradicate it," Watt said. "I would be hesitant to recommend that people plant boxwood in the future."

The blight was first discovered in the United Kingdom in the mid-1990s. It is not known how it came to the United States.

I wrote in January about the spotted wing drosophila, European crane fly and marmorated stink bug, and they continue to cause concern.

But some pests that were problems in the past are beginning to come under control, said Richard

Casagrande, a professor of entomology who specializes in biological controls.

Casagrande told a class last month at New England Grows in Boston that he has been having some success releasing wasps that kill the lily leaf beetle, and that two populations of them have established in Maine. The *Tetrastichus setifer* has colonized around Orono, and *Diaparsis jucunda* has been established in southern Maine.

But some gardeners might have to change their usual gardening practices. It still will take time for those colonies to expand their range to the rest of the state.

"Mulching the lilies is not good for the beneficial wasps," Casagrande said.

If you don't think biological controls work, think back to the 1970s and 1980s, when the gypsy moth caterpillar was decimating Maine's softwood forests.

"Then in 1989, a fungus showed up in the population which controlled the gypsy moth and spread over the entire range," Casagrande said. "When we have a drought, we will have localized outbreaks of gypsy moth" because the fungus does not do well in dry conditions.

Another pest that has made it to New Hampshire but not yet Maine is the mile-a-minute plant, *Persicaria perfoliata*, a barbed trailing vine. This vine grows in disturbed ground and in ditches next to roads, and will climb and smother all vegetation in its way.

"A weevil native to China has been quite effective where it was released in Delaware," Casagrande said.

He noted that biocontrols don't always work, but when they do, they are more cost-effective and less harmful to the environment than using chemical weed killers.

The biocontrols have to go through a long period of testing before they can be released. The scientists want to know that the predator insect or fungus will control the pest, and that it will damage only the pest. The worst thing that could happen would be for something to be released that would hurt native species.

Swallowwort -- an invasive vine that looks a bit like morning glories, is related to milkweed and develops pods that look like milkweed pods -- has been around for years, but has more problems than I thought. Yes, it will entwine among plants that you like, but it is also harmful to Monarch butterflies, which require milkweed in the reproduction cycle.

"Butterflies lay their eggs on swallowwort, but they don't survive," Casagrande said.

Lois Berg Stack, an ornamental horticulture specialist with the University of Maine Cooperative Extension in Orono, reported at New England Grows that Japanese Stilt Grass is another invasive that has reached southern New England. It looks like many native grasses, but is more aggressive, survives in full sun to deep shade, and grows in disturbed soil and along stream beds.

The best way to control it, she said, would be to find it wherever it first reaches the state, watching closely at areas where it is likely to grow, and remove it before it can spread.

"Eradication is less effective than prevention," she said. "We try to practice early detection and rapid response. We have to be diligent."

Berg Stack did outline ways to remove invasives when they arrive, including chemicals, weed wrenches and smothering with plastic, mulch and sheet vinyl. But it is easier to keep pests out in the first place.

Jeff O'Donal, speaking at the same meeting as Watt, said he is confident that the hemlock woolly adelgid

will not decimate Maine's hemlocks, although it has decimated the hemlock populations in Connecticut and other places farther south.

"If you looked at those trees even before the adelgid, they didn't look healthy," he said.

And while the adelgid has been found in southern Maine, most of the infected trees are healthy enough to fight it off.

Another bit of hope, O'Donal said, is that the viburnum leaf beetle that hit with a vengeance about 15 years ago is not doing as much damage as it has in the past, even on varieties of viburnum that seem to be susceptible to it. Either something has arrived to keep the beetle under control, or the beetle is just less prevalent.

Tom Atwell has been writing the Maine Gardener column since 2004. He is a freelance writer who gardens in Cape Elizabeth, and can be contacted at 767-2297 or at:

tomatwell@me.com

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Conn. company pitches Gouldsboro salmon farm

4/2/2012

A Connecticut-based company is applying to build a salmon aquaculture operation on former Navy property in Gouldsboro. Local officials have approved the preliminary proposal.

Palom Aquaculture LLC is seeking local, state and federal permits for the salmon farm in Gouldsboro's village of Corea at the Navy's former Schoodic Point, according to the **Bangor Daily News**. The company hopes to acquire two lots, where it will build a facility to house 20 salmon-raising tanks that will be grown without the use of pesticides, antibiotics or growth hormones. The company hopes to produce up to 2 million pounds of salmon a year by 2017 and employ seven to 10 people.

Local officials have given their preliminary approval but are still waiting to see blueprints of the proposed facility before issuing a permit, according to the paper. Palom is seeking approval from the Maine Department of Environmental Protection and the U.S. Army Corps of Engineers to extract water from and discharge it to Prospect Harbor. If it receives all necessary permits, the company hopes to begin construction this year and bring its first product to market in 2014.

Other parts of the former Navy site are also being eyed for aquaculture work. A facility for Maine Halibut Farms, which currently operates at the University of Maine Center for Cooperative Aquaculture Research in Franklin, is in development, and Eastern Maine Development Corp. wants to bring fisheries businesses to the 40 acres it owns.

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Baykeeper wins EPA lifetime achievement award

Written by David Carkhuff

Friends of Casco Bay's Joe Payne has won recognition for his lifetime advocacy for clean water. The U.S. Environmental Protection Agency yesterday presented its annual Environmental Merit Awards for 2011.

Payne was joined by Jeff Emery of the Maine Department of Environmental Protection in receipt of the merit awards, which recognize valuable contributions to environmental awareness and problem solving, the EPA's New England Regional Office reported.

Emery was noted as an environmental scientist "and as a leader in such collaborations as those with such national and regional organizations as EPA, Northeast States for Coordinated Air Use Management, the National Park Service as well as other jurisdictions that include Canadian Provinces, Maine Indian Tribes and other states."

Payne won an Environmental Merit Award for Lifetime Achievement at a ceremony in Boston. U.S. Rep. Chellie Pingree congratulated Payne.

"For 20 years, Joe Payne has worked tirelessly to protect Casco Bay from a number of environmental threats — storm-water runoff, industrial pollution and oil spills among them," Pingree said in a press release. "Everyone who values and makes their living from Casco Bay has benefited from this exceptional scientist and committed advocate. The health of the bay couldn't be in better hands than Joe Payne's."

Payne has been a baykeeper on Casco Bay for 20 years. In 1991, he was hired as the first employee of Friends of Casco Bay, a grassroots conservation organization in southern Maine, the EPA noted. Payne, a fisherman's grandson, has been a steward and voice for Casco Bay ever since.

Payne was saluted for his "science-based, collaborative approach to resolving threats to the bay's environmental health," the EPA press release noted.

"He has spearheaded numerous conservation campaigns that benefit the bay and the entire Maine coast," the EPA stated. "He created an award-winning volunteer water quality monitoring program and made

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Casco Bay one of the most thoroughly sampled water bodies in the country. The monitoring work allowed the organization to identify and eliminate sources of fecal coliform pollution and allowed hundreds of acres of clam flats to be re-opened to harvesters. His achievements also include launching a mobile pumpout service for recreational boats, which has kept over 125,000 gallons of raw sewage out of Casco Bay.”

The EPA added, “He sampled stormwater runoff for pesticides washing into the bay to support an education program to limit lawn chemicals, which is now a statewide effort, and initiated a lobster relocation project, rescuing 35,000 lobsters from the area to be dredged. He has also worked to raise awareness of the threat of coastal acidification from stormwater runoff and air deposits.”

Awarded by EPA since 1970, the merit awards honor individuals and groups who have shown particular ingenuity and commitment in their efforts to preserve the region’s environment. This year’s competition drew nearly 100 nominations from across New England.



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Schlein, Paul B

From: Fish, Gary
Sent: Thursday, May 03, 2012 12:34 PM
To: gary.fish@maine.gov
Subject: FW: Casco Baykeeper Accepts EPA's Lifetime Achievement Award

Huge congrats to Joe. One of the "Fathers" of BayScaping which morphed into YardScaping!

Gary Fish
Manager, Pesticide Programs
Maine Board of Pesticides Control
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207-287-7545
207-624-5020 fax
<http://www.ThinkFirstSprayLast.org>
<http://www.YardScaping.org>
<http://www.GotPests.org>

"Down the long lane of the history yet to be written America knows that this world of ours, ever growing smaller, must avoid becoming a community of dreadful fear and hate, and be instead, a proud confederation of mutual trust and respect." – Dwight D. Eisenhower

From: Cathy L. Ramsdell, CPA [mailto:jeff@cascobay.ccsend.com] On Behalf Of Cathy L. Ramsdell, CPA
Sent: Thursday, May 03, 2012 12:25 PM
To: Fish, Gary
Subject: Casco Baykeeper Accepts EPA's Lifetime Achievement Award

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Casco Baykeeper Accepts EPA's Lifetime Achievement Award



Mel Cote, Manager of the Ocean and Coastal Protection Unit, EPA Region I,
Casco Baykeeper Joe Payne, and
Curt Spaulding, Regional Administrator, EPA New England

On April 25th, the U.S. Environmental Protection Agency presented Casco Baykeeper Joe Payne with the 2012 Environmental Merit Award for a lifetime of advocacy for clean water. Friends of Casco Bay staff members, Joe's wife Kim (the Keeper of the Keeper), other New England waterkeepers, and EPA colleagues cheered Joe as he stepped up onto the stage at Boston's Faneuil Hall to receive his award for, as one EPA staffer put it, twenty years of "awesomeness." Afterwards, Joe said it was also pretty awesome to bask in the historic aura of the hall where George Washington and John Adams once orated.

Joe noted, "Because I am the face and voice of Friends of Casco Bay, the recognition I often receive should go to the entire staff and volunteers." It is because of the collaboration among our supporters - our members, donors, volunteers, and partners - that our work has received national recognition. As Joe points out, we are all working to make Casco Bay better.

The founders of Friends of Casco Bay said it best twenty years ago, shortly after hiring Joe: "The Casco Baykeeper has exceeded our dreams as steward and voice for Casco Bay. He is a listener, a fisherman's grandson who grew up working on the Bay, and a marine scientist whose actions are guided by both a passionate love for the Bay and an understanding of the physical and biological dynamics of the Bay."

Like Joe, we are all united in our "passionate love for the Bay." We all share in the Casco Baykeeper's success. Thank you!

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February 29

Organic farmers lament dismissal of Monsanto lawsuit

The group, led by a Maine farmer, had challenged Monsanto's patents on genetically modified seeds.

By [Avery Yale Kamilaakamila@mainetoday.com](mailto:Avery_Yale_Kamilaakamila@mainetoday.com)
Staff Writer

A national group of organic farmers headed by a potato grower from Maine was handed a legal setback Monday when a federal judge sided with agricultural and chemical giant Monsanto in a lawsuit challenging its patents of genetically modified seeds.



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Certified organic crops cannot contain genetically modified components. Such contamination could force farmers to lower prices for their crops or destroy them.

File Photo/Derek Davis/Staff Photographer

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- [Organic farmers appeal decision in Monsanto lawsuit](#)

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U.S. District Judge Naomi Buchwald of the Southern District of New York dismissed the lawsuit before it went to trial.

The lawsuit questioned the validity of Monsanto's patents on genetically modified seeds.

It also sought to give organic farmers blanket protection from lawsuits filed by the company claiming patent infringement should their crops be contaminated by Monsanto's genetically altered plants.

The lawsuit was filed in March 2011 by the Organic Seed Growers and Trade Association and more than 80 agricultural and consumer groups, with legal backing from the Public Patent Foundation, a nonprofit group that works to reduce abuses of the U.S. patent system.

By law, certified organic crops cannot contain genetically modified components. Such contamination could force farmers to lower prices for their crops or destroy them.

While acknowledging that some of the plaintiffs had stopped growing certain crops for fear of being sued, the judge ruled that the plaintiffs lacked standing to bring the lawsuit and called the farmers' claims that they could be subject to patent-infringement lawsuits "unsubstantiated" because "not one single plaintiff claims to have been so threatened."

Jim Gerritsen, who heads Wood Prairie Farm in Bridgewater and is the president of the Organic Seed Growers and Trade Association, said he was disappointed with the ruling.

"It was a flawed and erroneous ruling," Gerritsen said. "We have farmers who have already been impacted on their farms. We have farmers who've given up growing organic corn, organic soybeans and organic canola on their farms for fear of being sued. It's a very poor decision, full of error."

Monsanto praised the ruling saying in a statement it "makes it clear that there was neither a history of behavior nor a reasonable likelihood that Monsanto would pursue patent infringement matters against farmers who have no interest in using the company's patented seed products."

Monsanto has maintained throughout the case that it wouldn't sue farmers whose crops are inadvertently contaminated by its genetically engineered seeds.

Gerritsen dismissed those assurances.

"Monsanto's commitment is vague and not legally binding," Gerritsen said. "There is nothing that would prevent them from changing their mind tomorrow and pursuing us for patent infringement."

In her ruling, Buchwald cited a Monsanto blog as proof of the company's commitment not to sue organic farmers.

Buchwald acknowledges the threat of contamination is real, writing: "transgenic seeds may contaminate non-transgenic crops through a variety of means, including seed drift, or scatter, crosspollination, and commingling via tainted equipment."

The ruling also states that farmers shoulder the burden of maintaining transgenic-free crops by establishing buffer zones on their properties and paying to test their crops to make sure they're not contaminated.

According to the U.S. Department of Agriculture, genetically modified seeds accounted for more than 90 percent of soybeans and more than 70 percent of corn planted in 2011.

In the ruling, Buchwald stated that Monsanto brought 144 patent-infringement lawsuits against farmers from 1997 to 2010, or an average of 13 per year.

"This average of roughly thirteen lawsuits per year is hardly significant when compared to the number of farms in the United States, approximately two million," Buchwald wrote.

However, University of Maine School of Law professor Rita Heimes, who directs the Center for Law and Innovation, said Monsanto's patent-infringement lawsuits are unique in the intellectual property field.

"I think Monsanto's litigation strategy has been very aggressive compared to other patent holders," Heimes said. "Because Monsanto sues its customers, it does make all farmers nervous."

Most patent-infringement lawsuits involve one manufacturing company suing another for appropriating

patented technology, Heimes said.

"Had this judge ruled the other way this would have been a much bigger case in the field of intellectual property," Heimes said.

Daniel Ravicher, who heads the Public Patent Foundation and was the lead attorney for the farmers, said the plaintiffs are considering an appeal. They have until the end of March to decide.

"When (non-genetically modified organisms) farmers are under threat, that means all consumers are at risk of losing their access to good, clean food," Gerritsen said. "We have to wake people up to the injustice family farmers are up against. Our livelihoods are at stake."

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April 29

Mild winter could lead to huge honeybee die-off come fall

By [Mechele Coopermcooper@centralmaine.com](mailto:Mechele.Coopermcooper@centralmaine.com)
Staff Writer

Beekeepers need to be especially careful this year.



[click image to enlarge](#)

Bees climb over frames of an open box as Roy Cronkhite checked one of his hives in Livermore Falls to make sure the queen bee had plenty of empty cells left in the wooden frames of the hives to deposit eggs.

Staff photo by Joe Phelan



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Roy Cronkhite checked one of his hives in Livermore Falls to make sure the queen bee had plenty of empty cells left in the wooden frames of the hives to deposit eggs. He pulled out three frames until he found the queen. I see there's plenty of cells over here, so she's fine, he said. She has plenty of room.

A mild winter and unseasonably warm early spring have created conditions reminiscent of 2010, when beekeepers were caught off guard from an explosion of mite populations that killed off many honeybee colonies, according to a state expert.

“The bees are coming out, but so are the parasitic mites,” said Tony Jadczyk, state apiarist and bee inspector. “What I’ve seen in my inspections is elevated mite loads because of the good health of the honey bees. If it tracks like it did in 2010, we’ll have a huge die-off in the fall and winter.”

Varroa is one of the external parasitic mites that attacks European honeybees, along with nosema, an intestinal parasite, Jadczyk said.

He said most hives were strong in 2010 at the onset of the late spring and summer. Then, in mid- to late July, hive inspections indicated that many colonies were at or approaching levels at which they needed treatment for Varroa, Jadczyk said.

Those levels were reached at least a month earlier than normal.

Jadczyk said this year the bees are eager after the mild weather, the same as in 2010; so he’s advising Maine beekeepers to monitor their hives.

When the mite count exceeds recommended levels, it’s time to treat with soft chemicals, which are mainly organic acids from plant oils.

Jadczyk said bees should be managed according to weather conditions and plant phenology, not the calendar date. And monitoring varroa is crucial because mite populations can explode under certain circumstances, he said.

Jadczyk said Maine beekeepers suffered enormous losses since the parasite from the Asian honeybee was introduced into the United States in the mid-1980s. He said there’s also a viral complex associated with the exotic mite that honeybees in the U.S. have no defense against.

“Continue to monitor, but be ready to treat when the summer crop is done mid- to end of July, if we parallel 2010, which seems like what’s going on,” he said. “Weather is a big factor. Based on what I’m seeing, (bees are) running ahead of schedule.”

Honeybee decline

Parasitic mites are not the only concern for beekeepers.

The Maine Organic Farmers and Gardeners Association also is warning people about a class of pesticides that are increasingly linked to problems surrounding bee health, specifically a phenomenon called colony collapse disorder.

Russell Libby, executive director of MOFGA, said each year since 2006 U.S. beekeepers have lost on average a third of their hives.

At least one commercial beekeeper qualified for disaster relief from the U.S. Department of Agriculture because the loss of hives last year was so great.

Libby said Maine doesn’t have enough bees in the state to pollinate all the crops, so 70,000 bee hives are

brought in by commercial beekeepers every year. Libby is urging people to contact Maine's congressional delegation and ask that they pressure the U.S. Environmental Protection Agency to take swift action to protect the honeybee.

"The big issue really is to have EPA look more closely at these materials as they're approving pesticides for use," he said.

The Harvard School of Public Health released a study earlier this month that said the likely culprit in worldwide declines in honeybee colonies since 2006 is imidacloprid, one of the most widely used pesticides. Bees are exposed to the imidacloprid belonging to the group of pesticides called neonicotinoids when they feed on nectar and pollen. The pesticide interferes with the transmission of stimuli in the insect's nervous system and results in convulsions, paralysis and eventually death. The study is scheduled to appear in the June issue of the Bulletin of Insectology.

'There's no funding'

Roy Cronkhite, a beekeeper in Livermore Falls and president of the Kennebec Beekeepers Association, said beekeepers never had the luxury of federal or state funding like other agricultural entities until colony collapse disorder came to light.

"That really shook up a lot of people who said, 'Oh, my God,'" Cronkhite said. "If these large pollinators who go from state to state across the country are having this terrible problem, how will we get our crops pollinated? So they threw some money at it to do some research to find out the reason."

Cronkhite on Saturday checked his hives to make sure the queen bee had plenty of empty cells left in the wooden frames of the hives to deposit eggs. He pulled out three frames until he found the queen.

"I see there's plenty of cells over here, so she's fine," he said. "She has plenty of room." Cronkhite hasn't checked yet this year for mites, but he planned to do it later in the day with some friends. He planned to place mineral oil on the bottom board of the hive that the mites will stick to when they fall off the bees.

"You take the number of mites and divide it by the number of dead bees, and that comes up with a percentage," he said. "If it's greater than 10 percent, we have to treat."

He said regional bee clubs and the Maine State Beekeepers Association try to educate their members about colony collapse disorder. They rely on sources outside the state for information on the latest news regarding the problem, he said.

"We're hearing a lot of stuff about genetically engineered crops and wondering what the heck it is doing to the bees," Cronkhite said. "The young bees — the larva stage — they look like a little grub. They're fed flower nectar and pollen. The honeybee mixes that with their own enzymes, and that becomes food for the larva. If you have good food, you get the highest potential for growth and healthy bees. Anything less than that you're taking away from the bee, which can cause problems and weakness in the hives."

He said beekeepers continue to have the financial burden of replacing lost hives.

"There's no funding," he said. "It's just unfortunate."

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Controversial Pesticide Linked to Bee Collapse

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By Brandon Keim March 29, 2012 | 3:33 pm | Categories: [Animals](#), [Biology](#)

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A controversial type of pesticide linked to declining global bee populations appears to scramble bees' sense of direction, making it hard for them to find home. Starved of foragers and the pollen they carry, colonies produce fewer queens, and eventually collapse.

The phenomenon is described in two new studies published March 29 in *Science*. While they don't conclusively explain global bee declines, which almost certainly involve a combination of factors, they establish neonicotinoids as a prime suspect.

"It's pretty damning," said David Goulson, a bee biologist at Scotland's University of Stirling. "It's clear evidence that they're likely to be having an effect on both honeybees and bumblebees."

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Neonicotinoids emerged in the mid-1990s as a relatively less-toxic alternative to human-damaging pesticides. They soon became wildly popular, and were the fastest-growing class of pesticides in modern history. Their effects on non-pest insects, however, were unknown.

In the mid-2000s, beekeepers in the United States and elsewhere started to report sharp and inexplicable declines in honeybee populations. Researchers called the phenomenon colony collapse disorder. It was also [found in bumblebees](#), and in some regions now threatens to extirpate bees altogether.

Many possible causes were suggested, from viruses and mites to industrial beekeeping practices and climate change. Pesticides, in [particular neonicotinoids](#), also came under scrutiny.

Leaked internal reports by the Environmental Protection Agency showed that industry-run studies used to demonstrate some neonicotinoids' environmental safety [were shoddy and unreliable](#). Other researchers found signs that neonicotinoids, while they didn't kill bees outright, affected their ability to learn and navigate.

Those results came from laboratory situations, with no guarantee of real-world applicability, but they were troubling.

"Bees' ability to navigate is very important. When they leave their nest, they fly miles to gather food. Anything that makes them even a little bit worse at navigating or learning could be a disaster in those circumstances," said Goulson. "The research suggested effects on their learning ability, but it was all done in confined situations. What we and the French group did is something more natural."

'Anything that makes bees even a little bit worse at navigating or learning could be a disaster.'

In the first study, led by biologist Mickaël Henry of INRA, a French agricultural research institute, free-roaming honeybees were tagged with RFID chips that allowed researchers to track their movements. When dosed with a neonicotinoid, bees were more than twice as likely as non-dosed controls to die outside their hives. They seemed to get lost.

When the researchers added their results to computer simulations of honeybee dynamics, the model populations crashed.

Penn State entomologist James Frazier, who was not involved in the study, called it "the best study to date" on neonicotinoids' real-world effects on foraging.

The result dovetailed with the findings of Goulson's group, who exposed developing bumblebees to varying neonicotinoid levels and set them loose to forage in an enclosed field. Measured after six weeks of growth, pesticide-dosed colonies were stunted, weighing about 10 percent less and producing 85 percent fewer queens.

"Nests have annual cycles. They start with a single queen, and the nest grows through the season. If it doesn't get big enough, it doesn't have the resources to pour into rearing queens," Goulson said. "The French study shows that exposure to neonicotinoids make honeybees less likely to find their nest. That's likely the mechanism that led to our nests growing more slowly."

However, biologist Jerry Bromenshenk of the University of Montana was critical of the results. Goulson's results were interesting but the researchers weren't careful enough in verifying the doses given to their bees, and Henry's group administered an unrealistically high dose, said Bromenshenk.

The latter's dosing "is not what I would consider to be a field-relevant, low dose," wrote Bromenshenk in an email, citing [another recent study that used RFIDs to track bees](#) given what he considers a more realistic dose. "At truly field representative, sublethal doses — no effect," Bromenshenk wrote.

Both Goulson and Mace Vaughan, pollinator program director at the Xerces Society, an invertebrate conservation group, said

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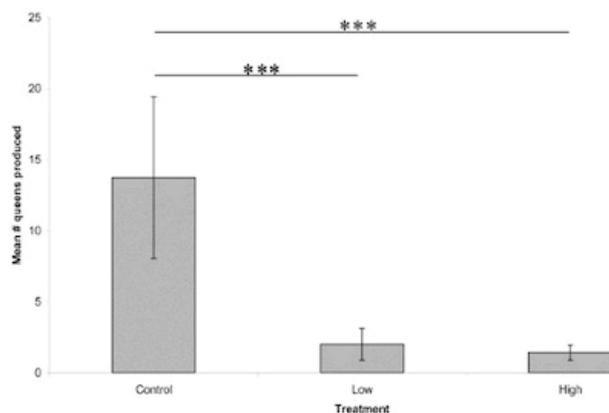
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neonicotinoids won't be the only cause of colony collapse disorder.

"If it was as simple as that, the answer would have been discovered a long time ago," said Goulson. "I'm sure it's a combination of things. I'm sure that disease is a part of it, and maybe the two interact." He noted a study in which honeybees exposed to neonicotinoids [were especially vulnerable to a common bee parasite](#). Another study found that neonicotinoids [dramatically increase the toxicity of fungicides](#).



A comparison of bee queen production in colonies treated (middle, right) and untreated (left) with a neonicotinoid pesticide. Image: Whitehorn et al./Science

Vaughan raised the issue of industrial-scale beekeeping practices, which have also been linked to bee declines. "We've potentially created a situation where behavioral impacts, compounded with a lack of genetic diversity and the food they eat, results in something like colony collapse disorder," he said.

"My only caution is that farmers use neonicotinoids for a reason," said Goulson. "If they were banned, farmers would have to use something else. The question is, what would that be? Would it be better? Would it also have harmful effects?"

While it's unlikely that neonicotinoids will be banned outright in the United States, where they're now used on more than 100 million crop acres and an unknown area of home gardens and urban vegetation, Vaughn said they could be used differently.

"I would call for a ban on their use without a demonstrated pest threat. If you have corn rootworm, and need to address that, then use neonicotinoid-coated seeds," he said. "But if it's a vague threat that you haven't identified, you shouldn't be using them. Maybe it makes you a few bucks, and certainly makes the seed companies a lot of money, but it's potentially killing bees across the country."

Heather Pilatic of the Pesticide Action Network recommended a return to pest management strategies used widely through the 1990s, when the rise of pesticide-treated seeds and genetically modified crops allowed farmers to change their growing strategies.

"When you plant the same crop, year after year, you're creating the conditions for a pest infestation," Pilatic said. "In the mid-1990s, we were doing a really good job of pest management with corn in particular. With the introduction of treated seeds, and in particular of genetically engineered corn, it all unraveled. But we know how to do it. We were doing it 20 years ago."

Penn State's Frazier said that the Environmental Protection Agency, which recently received a [1.25 million-signature-strong petition to ban neonicotinoids](#), is slowly becoming better at risk assessment, though the agency is still heavily influenced by chemical companies and opaque in its workings.

The fundamental problem isn't neonicotinoids, but our society's relationship to chemicals, said Frazier. "We're making ourselves the guinea pigs," he said. "I don't think that's what a rational society should be doing."

Image: [Jack Wolf/Flickr](#)

Citations: "Neonicotinoid Pesticide Reduces Bumble Bee Colony Growth and Queen Production." By Penelope R. Whitehorn, Stephanie O'Connor, Felix L. Wackers, Dave Goulson. *Science*, Vol. 335 No. 6076, March 30, 2012.

"A Common Pesticide Decreases Foraging Success and Survival in Honey Bees." By Mickaël Henry, Maxime Beguin, Fabrice Requier, Oriane Rollin, Jean François Odoux, Pierrick Aupinel, Jean Aptel, Sylvie Tchamitchian, Axel Decourtye. *Science*, Vol. 335 No. 6076, March 30, 2012.

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ARE NEONICOTINOIDS KILLING BEES?

A Review of Research into the Effects of Neonicotinoid Insecticides
on Bees, with Recommendations for Action

Jennifer Hopwood
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EXECUTIVE SUMMARY

Neonicotinoid pesticides were first registered for use in the mid 1990s. Since then, these chemicals have become widely adopted for use on farm crops, ornamental landscape plants, and trees. Of the six neonicotinoids commonly used on plants, the most widely used is imidacloprid. Neonicotinoids are systemic chemicals; they are absorbed by the plant and are transferred through the vascular system, making the plant itself toxic to insects.

The impact of this class of insecticides on pollinating insects such as honey bees and native bees is a cause for concern. Because they are absorbed into the plant, neonicotinoids can be present in pollen and nectar, making these floral resources toxic to pollinators that feed on them. The long-lasting presence of neonicotinoids in plants, although useful from a pest management stand-

point, makes it possible for these chemicals to harm pollinators even when the initial application is made outside of the bloom period. In addition, neonicotinoids persist in the soil and in plants for very long periods of time.

Across Europe and the United States, a possible link to honey bee die-offs has made neonicotinoids controversial. Several European countries have reexamined the use of neonicotinoids in crops such as corn, canola, and sunflower. In the United States and elsewhere, a number of opinion articles, documentary films, and campaigns have called for the ban of neonicotinoids.

This report reviews research on the impact of these pesticides on bees. We also identify knowledge gaps, highlight research needs, assess current regulations, and make recommendations for protecting bees.

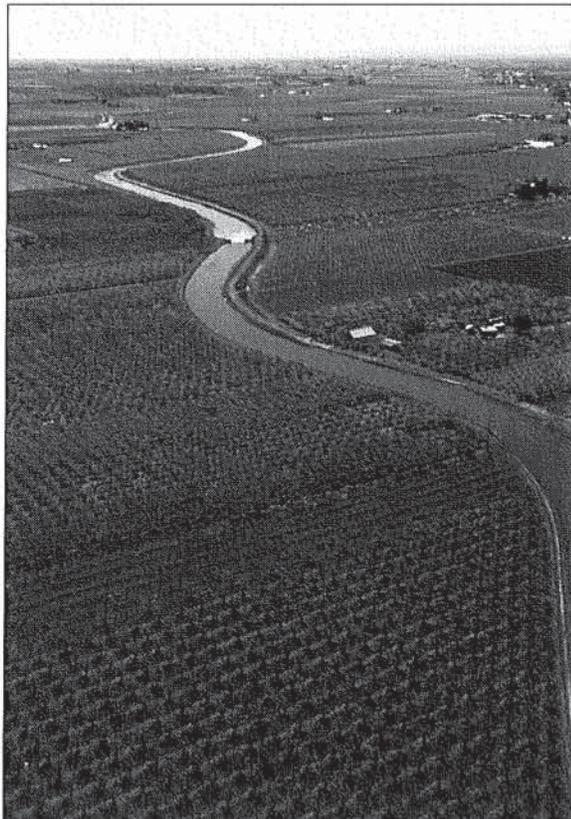
Findings

The following findings are divided into three sections. In the first section, we present clearly documented information about neonicotinoid impacts on bee, i.e., facts that are supported by an extensive body of research. The second section covers what can be inferred from the available research. This includes possible effects for which there is currently only limited research or the evidence is not conclusive. In the third section, we identify knowledge gaps in our understanding of pollinator and neonicotinoid interactions. Filling these gaps will allow better-informed decisions about the future use and regulation of these products.

Clearly Documented Facts

Exposure of bees to neonicotinoids

- ⇒ Neonicotinoid residues found in pollen and nectar are consumed by flower-visiting insects such as bees. Concentrations of residues can reach lethal levels in some situations.
- ⇒ Neonicotinoids can persist in soil for months or years after a single application. Measurable amounts of residues were found in woody plants up to six years after application.
- ⇒ Untreated plants may absorb chemical residues in the soil from the previous year.
- ⇒ Products approved for home and garden use may be applied to ornamental and landscape plants, as well as turf, at significantly higher rates (potentially 120



Neonicotinoid insecticides have been applied to hundreds of thousands of acres of farmland. Impacts on bees have been demonstrated, but there are still many things that are not known about the effects neonicotinoids have on these and other pollinators. (Photograph: USDA-ARS/Brian Prechtel.)

times higher) than those approved for agricultural crops.

- ⊕ Direct contact with foliar neonicotinoid sprays is hazardous to pollinators, and foliar residues on plant surfaces remain toxic to bees for several days.
- ⊕ Neonicotinoids applied to crops can contaminate adjacent weeds and wildflowers.

Effects on honey bees

- ⊕ Imidacloprid, clothianidin, dinotefuran, and thiamethoxam are highly toxic to honey bees.
- ⊕ Thiacloprid and acetamiprid are mildly toxic.
- ⊕ After plants absorb neonicotinoids, they slowly metabolize the compounds. Some of the resulting breakdown products are equally toxic or even more toxic to honey bees than the original compound.
- ⊕ Honey bees exposed to sublethal levels of neonicotinoids can experience problems with flying and navigation, reduced taste sensitivity, and slower learning of new tasks, which all impact foraging ability.

Effects on bumble bees

- ⊕ Laboratory studies demonstrate that imidacloprid and clothianidin are highly toxic to bumble bees.
- ⊕ Bumble bees exposed to sublethal amounts of neonicotinoids exhibit reduced food consumption, reproduction, worker survival rates, and foraging activity.

Bumble bees and solitary bees respond differently to neonicotinoids than do honey bees. Current regulatory testing doesn't address these differences. (Photograph: Mace Vaughan/The Xerces Society.)



Effects on solitary bees

- ⊕ Clothianidin or imidacloprid spray is toxic to blue orchard and alfalfa leafcutter bees.
- ⊕ Residue of imidacloprid on alfalfa foliage increases rates of mortality of alfalfa leafcutter and alkali bees.
- ⊕ Blue orchard bee larvae required more time to mature after consuming sublethal levels of imidacloprid in pollen.

Inferences from Research Results

Exposure of bees to neonicotinoids

- ⊕ Application methods other than seed coatings (foliar sprays, soil drenches, and trunk injections) apply a higher dosage per plant and may result in much higher—even toxic—levels of neonicotinoid residues in pollen and nectar.
- ⊕ Application of neonicotinoids shortly before and during bloom may lead to higher residue levels in pollen and nectar.
- ⊕ Application by soil drench or trunk injection may result in high residue levels in blossoms of woody ornamental species more than a year after treatment.

Effects on pollinators

- ⊕ There is no direct link demonstrated between neonicotinoids and the honey bee syndrome known as Colony Collapse Disorder (CCD). However, recent research suggests that neonicotinoids may make honey bees more susceptible to parasites and pathogens, including the intestinal parasite *Nosema*, which has been implicated as one causative factor in CCD.
- ⊕ Neonicotinoids may synergistically interact with demethylase inhibitor (DMI) fungicides. DMI fungicides significantly increased the toxicity of neonicotinoids to honey bees in laboratory tests, but the full effects of this interaction in field settings are unclear.
- ⊕ Bumble bees and solitary bees respond differently to neonicotinoids than do honey bees.
- ⊕ Pesticide residues from seed treatment have been found in hives. Neonicotinoid-treated corn seed is planted on millions of acres annually in the United States. Although we do not know the full scope of the impact of this exposure on bees, we do know that bees close to corn fields can come into contact with lethal levels of abraded seed coatings and dust, bees may collect contaminated pollen, and that plants (e.g., weeds) growing around seed-treated fields can become contaminated with systemic insecticides.

Knowledge Gaps

Exposure of bees to neonicotinoids

- ↻ How do residue levels in pollen and nectar increase in concentration over time with repeated application? Given that residues can persist for long periods, repeated applications to perennial plants may cause concentrations to accumulate to sublethal or lethal levels. These data are critical for managing impacts to pollinators.
- ↻ How do residues from repeated applications and/or repeated planting of seed treated annual crops accumulate in the soil over time, resulting in higher residue levels in the pollen and nectar of annual crops, as well as in crop weeds?
- ↻ What is the degree of risk posed by neonicotinoid contamination of non-target plants growing near treated plants?
- ↻ How soon after product application do neonicotinoid residues appear in pollen and nectar, and does its appearance vary with application method?
- ↻ Is the combined presence of neonicotinoids and their break-down products in pollen or nectar more toxic to bees than the individual chemicals? As a neonicotinoid breaks down inside a plant, bees may be exposed to residues of both the parent compound and its metabolites.
- ↻ Does the movement of neonicotinoids vary with the type of plant (e.g., herbaceous vs. woody), by functional group (e.g., forbs vs. legumes vs. grasses), or by the size of plant?
- ↻ How do residue levels vary in plants grown under differing field conditions (e.g., drought), soil types (e.g., sandy vs. loam), or under variable nutrient levels?

Effects on pollinators

- ↻ Do honey bees experience delayed effects of neonicotinoids during adverse weather conditions (e.g., winter or drought) when stored foods are consumed? Because honey bees store food for times of dearth, chemical exposure is likely delayed beyond field study timelines.
- ↻ What are the acute and chronic toxicities of neonicotinoids to bees other than honey bees? Given the contribution of bumble bees and solitary bees to agricultural and native plant pollination in temperate landscapes, it is vital that we better understand the effects of these chemicals on all bees.
- ↻ What is the full extent of the sublethal effects of neonicotinoids on foraging, reproduction, and other behaviors of adult bees?
- ↻ What is the full extent of the sublethal effects of neonicotinoids on larval bees?
- ↻ What effects do soil residues have on ground-nesting bees—the majority of bee species—exposed to neonicotinoids through soil applications (drenches, chemigation, granules)?
- ↻ What are the effects of neonicotinoid residues on bees that construct nests from contaminated plant tissues? About 30% of bee species construct nests by using leaf pieces, plant resins, or holes in stems and tree trunks.
- ↻ How do neonicotinoids affect other pollinators such as butterflies, moths, beetles, flies, and wasps? Although these insects make minor contributions to crop pollination, they serve important roles within crop systems and other ecosystems.

Recommendations

Bees provide essential services in agriculture, in natural ecosystems, and in the support of overall biodiversity. A large—and growing—body of research demonstrates that neonicotinoid insecticides harm multiple bee species, yet substantial knowledge gaps remain. Based on the findings, the Xerces Society for Invertebrate Conservation makes six major recommendations:

1 The bee safety of currently approved uses of products containing neonicotinoid insecticides should be reassessed and all conditional registrations reexamined and/or suspended until we understand how to manage the risk to bees. The risk from exposure to neonicotinoid insecticides then needs to be evaluated

against the risk posed to bees by alternative control measures

2 Before registration for a specific crop or ornamental plant species, research facilities should investigate the influences of application rate, application method, target plant species, and environmental conditions on levels of neonicotinoid residues in pollen and nectar.

3 The US Environmental Protection Agency should adopt a more cautious approach to approving all new pesticides, using a comprehensive assessment process that adequately addresses the risks to honey bees, bumble bees, and solitary bees in all life stages.

- 4 All neonicotinoid products used by commercial and agricultural applicators should include a clearly stated and consistent (standardized) warning on the label about the hazard to bees and other pollinators, including the unique exposure issues posed by contaminated pollen and nectar. This is particularly important for products marketed for garden and ornamental use.
- 5 Products marketed to homeowners for use on garden, lawn, or ornamental plants should all have a

clear warning label that prominently states, "Use of this product may result in pollen and nectar that is toxic to pollinators."

- 6 Legislators, regulators, and municipal leaders across the country should consider banning the use of neonicotinoid insecticides for cosmetic purposes on ornamental and landscape plants (as the ban now in force in Ontario, Canada). Approved application rates for ornamental and landscape plants, as well as turf, are often much higher than for farm crops.

In addition, we urge that the following issues are addressed.

Pesticide Risk Assessment and Registration

- ⊕ Regulators should evaluate neonicotinoid use and toxicity in mixtures that include fungicides and/or surfactants, including rigorous statistical tests.
- ⊕ Risk managers need to know how systemic insecticides accumulate in pollen and nectar after repeated use over multiple growing seasons, as well as the sublethal and lethal impacts of these concentrations.
- ⊕ More data on the lethal and sublethal impacts of neonicotinoids on bees are needed, particularly on those products other than imidacloprid, which to date has been the subject of most studies.
- ⊕ Regulators, researchers, and pesticide manufacturers should develop more comprehensive laboratory tests that assess the effects of neonicotinoids on multiple life stages of honey bees, bumble bees, and solitary bees during the registration process.
- ⊕ Regulatory standards for neonicotinoid testing should be changed to require that tests have adequate replication and sample size.
- ⊕ Testing must be subjected to rigorous statistical analyses so that significant independent variables can be identified.
- ⊕ Field tests completed during registration should include treated areas of at least 5 acres (2 hectares) and use managed solitary bees such as alfalfa leucutter and blue orchard bees that have a shorter foraging range than honey bees.
- ⊕ Methods should be developed for ongoing, post-registration assessment of the effects of neonicotinoids on bees at the landscape scale, under real world pest management conditions (e.g., repeated applications on 10s or 100s of acres), over multiple years.
- ⊕ Regulators should require multi-year tests to examine potential accumulation of residues in soil from

repeated annual plantings (e.g., via treated seeds or soil applications) and the impacts of these on ground-nesting bees.

Risk Management

- ⊕ The academic research community should develop IPM protocols that result in recommendations for the lowest effective dose for both specific crops and specific pests, as well as methods for reducing risk to non-target beneficial insects such as pollinators.
- ⊕ Licensed crop advisors and pesticide applicators should be required to understand the unique risks posed by neonicotinoids to bees and other flower visitors.
- ⊕ For all foliar applications, every attempt should be made to minimize direct contact with bees and other non-target insects.

Restrictions on Use

- ⊕ Until we know it is safe for bees, the use of neonicotinoids on crops such as apples and blueberries that bloom for a specific period of time each year should not be allowed during or before bloom. Such applications likely increase residue levels in pollen and nectar and increases exposure and risk to bees.
- ⊕ For crops that bloom continuously or over a long period of time (e.g., squash or tomato), academic IPM professionals should develop clear methods for how neonicotinoids can be used, so that concentrations of these products in crop pollen and nectar stays below sublethal levels.
- ⊕ Until a ban on cosmetic use of insecticides goes into effect, all neonicotinoid products marketed for non-agricultural use (e.g., homeowner products) should have label restrictions that limit application times, and reduce application rates on plants visited by bees.

Neonicotinoids in bees: a review on concentrations, side-effects and risk assessment

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Abstract Neonicotinoid insecticides are successfully applied to control pests in a variety of agricultural crops; however, they may not only affect pest insects but also non-target organisms such as pollinators. This review summarizes, for the first time, 15 years of research on the hazards of neonicotinoids to bees including honey bees, bumble bees and solitary bees. The focus of the paper is on three different key aspects determining the risks of neonicotinoid field concentrations for bee populations: (1) the environmental neonicotinoid residue levels in plants, bees and bee products in relation to pesticide application, (2) the reported side-effects with special attention for sublethal effects, and (3) the usefulness for the evaluation of neonicotinoids of an already existing risk assessment scheme for systemic compounds. Although environmental residue levels of neonicotinoids were found to be lower than acute/chronic toxicity levels, there is still a lack of reliable data as most analyses were conducted near the detection limit and for only few crops. Many laboratory studies described lethal and sublethal effects of neonicotinoids on the foraging behavior, and learning and memory

abilities of bees, while no effects were observed in field studies at field-realistic dosages. The proposed risk assessment scheme for systemic compounds was shown to be applicable to assess the risk for side-effects of neonicotinoids as it considers the effect on different life stages and different levels of biological organization (organism versus colony). Future research studies should be conducted with field-realistic concentrations, relevant exposure and evaluation durations. Molecular markers may be used to improve risk assessment by a better understanding of the mode of action (interaction with receptors) of neonicotinoids in bees leading to the identification of environmentally safer compounds.

Keywords Honey bee · Bumble bee · Solitary bee · Lethal toxicity · Sublethal effects · Reproduction · Behavioral effect · Risk assessment · Neonicotinoids · Residues

Introduction

Bees, including honey bees, bumble bees and solitary bees, are the prominent and economically most important group of pollinators worldwide; 35% of the world food crop production depends on pollinators (Klein et al. 2007; Velthuis and van Doorn 2006), accounting for an annual value of 153 billion Euros (Gallai et al. 2009). In Europe, for instance, the production of 84% of crop species is to some extent depending on animal pollination (Williams 1994). Bees also provide important pollination services to wild plants, of which in Europe 80% need insects for pollination (Kwak et al. 1998), so confirming their ecological importance. The decline of pollinating species, which has grown over the last decades, may lead to a

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parallel decrease of plant species, or vice versa (Biesmeijer et al. 2006; National Research Council of the National Academies 2007; Goulson et al. 2008). More specifically, there is a great concern about the decline of the honey bee (*Apis mellifera*) in several parts of the world (Oldroyd 2007; Stokstad 2007; VanEngelsdorp and Meixner 2010). It is now accepted that the abundance of pollinators in the environment is influenced by multiple factors, including biotic ones like pathogens, parasites, availability of resources due to habitat fragmentation and loss; and abiotic ones like climate change and pollutants (Decourtye et al. 2010; Neumann and Carreck 2010; Kluser et al. 2011). Although the putative causes are still currently analyzed, the extensive use of chemical pesticides against pest insects for crop protection may have contributed to the loss of pollinators.

To feed the fast growing global population, chemical insecticides are important to crop productivity in intensive farming systems where they preserve about one-fifth of the crop yield (Oerke and Dehne 2004). Good examples are the major staple crops like cereals, soybeans, maize, and many fruit and vegetable crops. Within the different insecticide classes, the neonicotinoid insecticides, which include imidacloprid, acetamiprid, clothianidin, thiamethoxam, thiacloprid, dinotefuran and nitenpyram, are an important group of neurotoxins specifically acting as antagonists of the insect nicotinic acetylcholine receptors (nAChR) (Matsuda et al. 2001; Elbert et al. 2008). Since the introduction of imidacloprid in the early 1990s, the use of different neonicotinoid insecticides has grown considerably. They are used extensively for the control of important agricultural crop pests by spraying and also widely used in seed dressings and soil additions. In the latter two cases residues of these systemic insecticides can be present at 'trace' levels in the plant pollen and nectar. So potentially, bees could be exposed at a large scale to insecticide residues originating from crop seed dressings.

To date in the international scientific literature >100 papers appeared with the keywords "neonicotinoids/imidacloprid" and "bee", the first being published in 1992, and an impressive cumulative number of citations near to 1,500. In addition many reports have appeared in different types of the public media, highlighting the awareness by the different stakeholders in the field related to pesticides, bees, environment, toxicology, pollination and agriculture.

This review gives, for the first time, a summary of the data published over the last 15 years on concentrations of neonicotinoid insecticides recovered in plants and bees and their products. This analysis of the literature took into consideration the different crops, the methods of application and the importance of metabolism, and covered data from different countries and continents. Second, the publicly available data on side-effects of the different neonicotinoid insecticides towards honey bees, bumble bees and

other bee species are summarized, and critically analyzed with a special emphasis on sublethal effects on reproduction, foraging behavior, memory/learning abilities and overwintering success. A third part focuses on the potential applicability of the new stepwise risk assessment scheme as proposed for systemic pesticides (Alix et al. 2009; Thompson 2010), for more adequately assessing risks for side-effects by neonicotinoid insecticides. The latter assessment took into account the characteristics of doses of neonicotinoid insecticides in their field-realistic range and followed the classical tiered approach from the laboratory to field-related conditions and from exposure of individual bees to the colony level. The importance of the use of adults and larvae (brood) together with the scoring of lethal and sublethal biological endpoints is also discussed. Points of comparison and experimental advantages and difficulties between honey bees, bumble bees and other bees are discussed. Attention is paid to the use of mixtures containing neonicotinoid insecticides that can synergize their hazards for bees. Our paper concludes with some targets for research and recommendations for future risk assessment studies, specifically with the aim to assess the global bee colony health status.

Concentrations and metabolism of neonicotinoid insecticides in plants and bees in relation to pesticide application

Translocation of residues in plants, nectar and pollen

Several studies have examined the translocation of imidacloprid from seed treatment to different parts of sunflower (*Helianthus annuus*) plants. In a greenhouse experiment with sunflowers treated with 0.7 mg ^{14}C -imidacloprid per seed (Gaucho WS, 700 g kg^{-1}) average imidacloprid concentrations amounted $3.9 \pm 1.0 \mu\text{g kg}^{-1}$ in pollen and $1.9 \pm 1.0 \mu\text{g kg}^{-1}$ in nectar (Schmuck et al. 2001). Nectar contained only imidacloprid and in pollen 85% of the ^{14}C -residues were present as imidacloprid (no metabolites were detected). In a field study at the dosage of 1 mg per seed (i.e. 30% higher than the recommended dose) no imidacloprid or metabolites were found in nectar and pollen, while the leaves of the sunflowers contained imidacloprid at $7 \mu\text{g kg}^{-1}$ and the hydroxy-metabolite at $<5 \mu\text{g kg}^{-1}$ (Schmuck et al. 2001). Only 5% of the ^{14}C -imidacloprid dose (1 mg per seed) was taken up from the seed after 4 weeks of sunflower growth in a climate-controlled cabinet. At flowering 90% of the dose was estimated to be still present in the soil. In the plant leaves mainly imidacloprid (approximately 50% of total ^{14}C) was found together with three metabolites (30–50% of ^{14}C). Imidacloprid concentrations decreased from the first leaves to the top leaves;

levels in sunflower pollen were $<0.5\text{--}36 \mu\text{g kg}^{-1}$ (Laurent and Rathahao 2003). Sunflower plants showed decreasing imidacloprid levels with time till the moment of capitule (flower head of Asteraceae) formation, but thereafter concentrations increased again. Imidacloprid concentrations in plants differed between sunflower varieties with average concentrations in the flowers between 5 and $10 \mu\text{g kg}^{-1}$ (Bonmatin et al. 2003). The latter study also determined imidacloprid residues in pollen samples of maize and sunflower that received a seed treatment. In 58% of the pollen samples imidacloprid was found with an average concentration of $3 \mu\text{g kg}^{-1}$ (range $1\text{--}11 \mu\text{g kg}^{-1}$) for sunflower. In 80% of the maize pollen samples imidacloprid was found at an average concentration of $2 \mu\text{g kg}^{-1}$ (5 samples only; range $1\text{--}3 \mu\text{g kg}^{-1}$) (Bonmatin et al. 2003), while a follow-up of this study reported an average concentration of $3.0 \mu\text{g kg}^{-1}$ (Charvet et al. 2004).

When sunflower and maize (without seed treatment) were planted on soils still containing imidacloprid at $2\text{--}18 \mu\text{g kg}^{-1}$ from earlier treatments, no imidacloprid was detected in pollen and nectar (Schmuck et al. 2001; Charvet et al. 2004).

Girolami et al. (2009) found that part of the imidacloprid taken up by maize seedlings can be eliminated through the guttation fluid, i.e. the droplets on the leaf tip. Excretion of guttation fluid seems limited to the first 3 weeks after germination (Girolami et al. 2009; Thompson 2010) and is affected by humidity, temperature, growth stage, water stress, root depth and soil water potential (Tapparo et al. 2011). During the first 3 weeks after emergence, imidacloprid concentrations can be very high. From a seed treatment of 0.5 mg per seed (Gaucho 350 FS), the imidacloprid concentrations in the guttation fluid of plants grown in the laboratory ranged between 47 ± 9.9 and $83.8 \pm 14.1 \text{ mg l}^{-1}$ (Girolami et al. 2009). Similarly, residues of clothianidin ($23.3 \pm 4.2 \text{ mg l}^{-1}$ from plants treated with 1.25 mg per seed as Poncho) and thiamethoxam ($11.9 \pm 3.32 \text{ mg l}^{-1}$; 1 mg per seed as Cruiser 350 FS) were found in the guttation fluid (Girolami et al. 2009). Tapparo et al. (2011) reported a decline of imidacloprid concentrations in the guttation fluid of maize plants that were dosed at 0.5 mg per seed (Gaucho) and grown in the greenhouse, from 80.1 mg l^{-1} after 1 day to 17.3 mg l^{-1} after 8–10 days, but the concentrations increased again to 60.1 mg l^{-1} during the next 10 days. At a dose of 1.25 mg per seed, imidacloprid concentrations in guttation drops that were collected during the first 6 days after emergence at the top of the leaves, ranged between 103 and 346 mg l^{-1} , while at the crown they amounted $8.2\text{--}120 \text{ mg l}^{-1}$. In the guttation fluid collected from plants grown in the field during the first day after emergence, imidacloprid concentrations ranged between 77 and 222 mg l^{-1} (Tapparo et al. 2011). Similar patterns were also seen for clothianidin ($7.3\text{--}102 \text{ mg l}^{-1}$)

and thiamethoxam ($2.9\text{--}40.8 \text{ mg l}^{-1}$) (Tapparo et al. 2011). Thiamethoxam concentrations in guttation fluid increased with decreasing soil moisture content, from 14 to 155 mg l^{-1} for plants grown under wet conditions to $34\text{--}1,154 \text{ mg l}^{-1}$ under dry conditions (Tapparo et al. 2011). The guttation fluid from plants growing on a field next to a plot planted with clothianidin-treated maize seeds (1.25 mg per seed; Poncho) always contained $<30 \mu\text{g l}^{-1}$ clothianidin (Marzaro et al. 2011).

Residues in bee-collected pollen, bees, honey and wax

Neonicotinoid residues in plants and plant parts only become of importance for bees once they are exposed. The most relevant measures of exposure are the concentrations in bee-collected plant materials, such as pollen, bee products like bee bread, honey and beeswax, and in the bees themselves. Table 1 summarizes reports on neonicotinoid insecticide concentrations in bee-related products as published in the literature.

Several studies were performed across Europe as well as North America (one study). Some studies involved a large scale analysis of samples collected over an extended area and in different years (Genersch et al. 2010; Chauzat et al. 2011), while others did a more or less nation-wide survey in one or two sampling years (Pirard et al. 2007; Nguyen et al. 2009; Bernal et al. 2010; Garcia-Chao et al. 2010; Mullin et al. 2010). A few studies focused on a limited number of samples (Bacandritsos et al. 2010) or did not mention the number of samples analyzed (Cutler and Scott-Dupree 2007). In some studies, a wide range of pesticides was measured in different bee-related products (Bernal et al. 2010; Chauzat et al. 2009; Mullin et al. 2010; Genersch et al. 2010), while others solely focused on neonicotinoid pesticides. Only few studies did include the analysis of metabolites.

An extensive inventory of imidacloprid in bee-collected pollen, honey and bees was performed by Chauzat et al. (2006, 2009, 2011), involving five sites across France with sampling of bee hives of five beekeepers in each area for 3 years and with four sampling events per year. Imidacloprid was found in 40.5 and 21.8% of the pollen and honey samples, respectively. The metabolite 6-chloronicotinic acid was present in 33.0 and 17.6% of the respective samples. The sampling took place in four agricultural areas and one natural area. Using a χ^2 test, frequency of imidacloprid + metabolite detection in pollen was shown to be significantly higher in 2003 compared to 2005; there was no difference for honey samples (Chauzat et al. 2011). No significance difference was found in the frequency of pesticide residue detection in pollen and honey between the different sampling areas (Chauzat et al. 2006, 2009). It is not known at what scale imidacloprid was applied in the

Table 1 Overview of literature data on neonicotinoid residues in bee-collected pollen, honey and bees

Substrate	Country/area/land use/ecosystem	Dominant crop/plant species	Experimental design and statistical analysis	Chemical	No. of samples	% Samples positive	Concentration ($\mu\text{g kg}^{-1}$ fresh weight)		Notes	References
							Mean ^a	Range in positive samples		
Pollen	France; 4 agricultural + 1 natural region	No data	24 Sites, 120 colonies; 4 × per year; Oct 2002–Sept 2005; χ^2 analysis to compare frequencies of pesticide occurrence between years and areas	imi	185	40.5	0.9	>0.2–5.7	No significant differences between areas; frequency in 2003 sign. higher than in 2005; many other pesticides found	Chauzat et al. (2006, 2009, 2011)
	Spain, 11 different regions	Sunflower dominant in 11.5%, wild vegetation in 78.7% and mixture in 9.8% of samples	Pollen from comb cells; χ^2 analysis to compare pesticide occurrence in unhealthy (depopulated) and asymptomatic colonies	imi	61	0	<0.4		No significant difference in pesticide residue levels between unhealthy and healthy populations; nine other pesticides detected	Higes et al. (2010)
	Spain, 17 regions	Wild vegetation in 47.8%, crops in 38.3% of samples (10.4% sunflower, 7.8% <i>Medicago</i> , 9.5% cereals)	Stored pollen from 448 + 92 spring and 397 + 84 autumn samples in 2006 + 2007, respectively; statistical analysis not applicable to imidacloprid	imi	1,021	0	<0.4		Many pesticides detected in 42% and 31% of spring and autumn samples, respectively	Bernal et al. (2010)
	USA (Florida, California, Pennsylvania or 13 states) + samples from outside USA and Canada	No data	2007/2008 Survey incl. 13 apiaries in Florida + California, 47 colonies in Pennsylvania orchards and from 'other' samples; no further data analysis	imi thm ace thc	350	2.9 0.29 ^b 3.1 5.4	3.1 53.3 1.9 1.3	<2.0–9.12 <5.0–124 <1.0–115	Many pesticides detected	Mullin et al. (2010)
	Germany, throughout the country	Main nectar flow plants: oil seed rape (<i>Brassica napus</i> ; 11–37%), sunflower (<i>H. annuus</i> ; 3–4%) and corn (<i>Zea mays</i> ; 16–22%)	Nation-wide survey: many beekeepers; 2005 + 2006 (50 apiaries; $n = 105$) and 2007 ($n = 110$ apiaries); sampling after rapeseed flowering; no further data analysis	imi thc clo ace	215	0.47 ^b 33 0 0.93	3 – <0.1 –	>1.0–199 >1.0	Many pesticides detected	Genersch et al. (2010)
	Guelph, Canada	<i>Brassica napus</i>	1 ha fields; treated 400 g AI kg^{-1} seed (= 32 g ha^{-1}) + control; 4 bee colonies per field for 21 days; sampling at 14 day intervals; samples pooled per field and sampling day; no further data analysis	clo	No data	Few	–	<0.5–2.59 (treated); <0.5 (control)		Cutler and Scott-Dupree (2007)

Table 1 continued

Substrate	Country/area/land use/ecosystem	Dominant crop/plant species	Experimental design and statistical analysis	Chemical	No. of samples	% Samples positive	Concentration ($\mu\text{g kg}^{-1}$ fresh weight)		Notes	References	
							Mean ^a	Range in positive samples			
Honey	France; 4 agricultural + 1 natural region	No data	24 Sites, 120 colonies; 4 × per year; Oct 2002–Sept 2005; χ^2 analysis to compare frequencies of pesticide occurrence between years and areas	imi	239	21.8	0.7	>0.3–1.8	No differences between areas or years	Chauzat et al. (2006, 2009, 2011)	
		Belgium, different areas	No data	Monitoring; no further details	imi	109	4.6	<0.084			Pirard et al. (2007)
	Belgium, different areas	Maize (0.05–2.48% of crop treated)	August–October 2004 (after flowering of <i>Zea mays</i>); no further data analysis	imi	48	8.4	0.275	>0.05 <0.5		Nguyen et al. (2009)	
	North-West Spain	No data	73 Apiaries; no further data analysis	imi	91	0	<2.33			Garcia-Chao et al. (2010)	
Honey bees	Guelph, Canada	<i>Brassica napus</i>	1 ha fields; treated 400 g AI kg ⁻¹ seed (= 32 g ha ⁻¹) + control; 4 bee colonies per field for 21-d; sampling at 14-d intervals; no further data analysis	thm	No data	Few	–	<0.5–0.93 (treated); <0.5 (control)		Cutler and Scott-Dupree (2007)	
				clo							
	France; 4 agricultural + 1 natural region	No data	24 Sites, 120 colonies; 4 × per year; Oct 2002–Sept 2005; χ^2 analysis to compare frequencies of pesticide occurrence between years and areas	imi	187	11.2	1.2	>0.3–11.1	No differences between areas or years	Chauzat et al. (2006, 2009, 2011)	
	Belgium, different areas	No data	Monitoring; no further details	imi	99	0	<0.1			Pirard et al. (2007)	
USA (Florida, California, Pennsylvania or 13 states) + outside U.S. and Canada	Belgium, different areas	Maize (0.05–2.48% of crop treated)	August–October 2004 (after flowering of <i>Zea mays</i>); no further data analysis	imi	48	0	<0.05			Nguyen et al. (2009)	
				imi							
	Greece, Peloponnesus region	No data	No data	Apiaries with bees exhibiting atypical behavior; no further data analysis	imi	5	60	27	14–39	Detection limits not given	Bacandritsos et al. (2010)
					clo			<LOD			
USA (Florida, California, Pennsylvania or 13 states) + outside U.S. and Canada	No data	No data	2007/2008 Survey of 13 apiaries in Florida + California, from 47 colonies in Pennsylvania orchard and from 'other' samples; no further data analysis	imi	140	0	<2.0		Many pesticides detected	Mullin et al. (2010)	
				thm			<1.0				
Greece, Peloponnesus region	No data	No data	Apiaries with bees exhibiting atypical behavior; no further data analysis	ace		0	<1.0				
				ace			<5.0				

Table 1 continued

Substrate	Country/area/land use/ecosystem	Dominant crop/plant species	Experimental design and statistical analysis	Chemical	No. of samples	% Samples positive	Concentration ($\mu\text{g kg}^{-1}$ fresh weight)		Notes	References
							Mean ^a	Range in positive samples		
Bees wax	Belgium, different areas	No data	Monitoring; no further details	imi	98	0	<0.1			Pirard et al. (2007)
	Belgium, different areas	Maize (0.05–2.48% of crop treated)	August–October 2004 (after flowering of <i>Zea mays</i>); no further data analysis	imi	48	0	<0.05			Nguyen et al. (2009)
	USA (Florida, California, Pennsylvania or 13 states) + outside USA and Canada	No data	2007/2008 Survey of 13 apiaries in Florida + California; from 47 colonies in Pennsylvania orchard; from ‘other’ samples; no further data analysis	imi thc thm ace	208	0.96 1.9 0 0	– 0.1 <1.0 <5.0	2.4–13.6 <1.0–8.0	Many pesticides detected	Mullin et al. (2010)
	Guelph, Canada	<i>Brassica napus</i>	1 ha fields; treated 400 g AI kg^{-1} seed (= 32 g ha^{-1}) + control; 4 bee colonies per field for 21-d; sampling at 14-d intervals; no further data analysis	clo	No data	Few	–	<0.5 (treated and control)		Cutler and Scott-Dupree (2007)

ace acetamiprid, *clo* clothianidin, *imi* imidacloprid, *thc* thiacloprid, *thm* thiametoxam

^a When no residues are detected, the limit of detection (LOD) is given

^b Only one sample was positive

agricultural areas where sampling took place. Neither is known what were the main plant species represented by the pollen samples collected.

As presented in Table 1, the average imidacloprid residue levels in positive pollen samples ranged between 0.9 and 3.1 $\mu\text{g kg}^{-1}$, while levels in honey and beeswax were generally lower. Concentrations of 6-chloronicotinic acid were only exceeding the limit of detection in the studies of Chauzat et al. (2006, 2009, 2011), with average concentrations of 1.2 (>0.3 – 9.3) $\mu\text{g kg}^{-1}$ and 1.2 (>0.3 – 10.2) $\mu\text{g kg}^{-1}$ in pollen and honey, respectively. Other studies reported in general lower frequencies of imidacloprid presence in pollen, honey and beeswax samples. Nguyen et al. (2009), who sampled in an area with 13.2% of the maize crop receiving seed dressing, detected imidacloprid in 8.4% of the honey samples, but levels were always below the limit of quantification (0.5 $\mu\text{g kg}^{-1}$). In a study in northern America, thiacloprid and acetamiprid were present in 5.4% of the pollen samples, while thiacloprid was also measured in 1.9% of the beeswax samples (Mullin et al. 2010). Also in Germany, thiacloprid was the most abundant neonicotinoid as it was detected in 33% of the pollen samples at concentration levels up to 199 $\mu\text{g kg}^{-1}$ (Genersch et al. 2010) (Table 1). In pollen collected at 1 and 6 days after spraying of apple trees in Slovenia with Calypso 480 SC at a dose of 0.2 kg ha^{-1} (approximately 0.1 kg AI ha^{-1}), respective thiacloprid levels of 60 and 30 $\mu\text{g kg}^{-1}$ were recorded. In bee bread, no thiacloprid was detected (detection limit 10 $\mu\text{g kg}^{-1}$) (Smodis Skerl et al. 2009).

The best measure of exposure and bioavailability are concentrations in honey bees. The study of Chauzat et al. (2011) found imidacloprid in 11.2% of the honey bee samples, while the main metabolite 6-chloronicotinic acid was detected in 18.7% of the samples. Average concentrations were 1.2 (>0.3 – 11.1) and 1.0 (>0.3 – 1.7) $\mu\text{g kg}^{-1}$, respectively. Also for honey bees, there were no significant seasonal and geographic differences in the frequencies of imidacloprid or 6-chloronicotinic acid residue detection (Chauzat et al. 2011). For honey bees, other studies did not detect imidacloprid in the bees. Only in the study of Bacandritsos et al. (2010) higher imidacloprid concentrations were measured in honey bees. This study however, concerned only five samples. As shown in Table 1, no other neonicotinoid insecticides were detected in honey bees in the other inventories performed across Europe and North America.

The low residue levels in honey bees probably are best explained from the fast imidacloprid metabolism by the honey bee *A. mellifera*. After exposure to sugar water dosed at 20, 50 or 100 $\mu\text{g }^{14}\text{C-imidacloprid kg}^{-1}$ honey bee, half-lives were 4–5 h (Suchail et al. 2004a, b). The major metabolites are 4- and 5-hydroxy-imidacloprid and olefin. Olefin peaked after about 4 h, while the hydroxy metabolite(s) appeared either immediately after termination of

exposure and then decreased in concentration (Suchail et al. 2004b) or showed a peak after about 4 h (Suchail et al. 2004a). The total amount of imidacloprid and metabolites in honey bees decreased with a half-life of 25 h (Suchail et al. 2004a). Imidacloprid was the main compound in the abdomen (38% of accumulated ^{14}C) directly after treatment. In the head, four metabolites were detected with imidacloprid levels always being $\leq 5\%$ of the ingested dose, and olefin and 4- and 5-hydroxy-imidacloprid being the main metabolites after 24 and 30 h, respectively. Imidacloprid and its metabolites were also detected in other body parts of the honey bee (hemolymph, midgut, rectum) with highest amounts in the thorax (Suchail et al. 2004a). It should be noted that dosages applied in these metabolism studies are much higher than the levels found in the field and might even be in the toxic range. The relevance of these data for the metabolism at field-realistic concentrations therefore remains uncertain.

Acetamiprid was also rapidly metabolized in bees, with a half-life of 25 min after oral administration with sugar water (100 $\mu\text{g kg}^{-1}$) and producing four metabolites. The major metabolite had a peak corresponding to approximately 48% of the dose after 8 h, and the other three metabolites reached maximum levels of 22–25%. After 72 h, the bees contained only metabolites. The metabolism of ^{14}C -acetamiprid seems to be tissue specific and showed a similar distribution pattern in the honey bee as imidacloprid (Brunet et al. 2005).

Side-effects of neonicotinoid insecticides in bees

Acute lethal toxicity

To date the evaluation of potential risks of insecticides is directed by guidelines like the Directive 91/414 in Europe and the Federal Insecticide, Fungicide and Rodenticide Act in the USA. Measurements of lethal toxicity are conducted by scoring the numbers of dead bees after 24–48 h and then the corresponding median lethal dose/concentration (LD_{50} and/or LC_{50}) is calculated. Tables 2 and 3 give an overview of the reported acute LD_{50} and LC_{50} values for neonicotinoid insecticides at the individual (organism) level. Based on this it is clear that several factors play a role:

Toxicity is dependent on the route of exposure with contact being less toxic than oral. The oral LD_{50} s, however, showed large variability over the different studies with neonicotinoids (Decourtye and Devillers 2010; Laurino et al. 2011). The process of trophallaxis may have contributed to differences in the uptake and accumulation of insecticide among the worker bees, and high imidacloprid doses may cause a reduction of sugar water consumption (Nauen et al. 2001).

Table 2 Overview of the lethal and sublethal side-effects by imidacloprid to individual (organism level) honey bees (*A. mellifera*), bumble bees (*B. impatiens*) and solitary bees as determined in different studies by oral/contact exposure under laboratory and (semi-)field conditions

Species	Exposure	Side-effects	References
<i>A. mellifera</i>	Lab + contact: acute (no information on concentration range), individual bees	LD ₅₀ -24 h: 18 ng bee ⁻¹	Iwasa et al. (2004)
<i>A. mellifera</i>	Lab + oral: acute exposure to 0.12 and 12 ng bee ⁻¹	Reduction of associative learning at 12 ng bee ⁻¹	Decourtye et al. (2004a, b)
<i>A. mellifera</i>	Lab + semi-field: oral exposure: 24 µg kg ⁻¹ in syrup	Decreased foraging activity on the food source and on the hive entrance; effect on the learning performance	Decourtye et al. (2004a, b)
<i>A. mellifera</i>	Lab + oral: acute exposure to 0.2–3.2 mg l ⁻¹	LD ₅₀ -48 h: 30 ng bee ⁻¹	Decourtye et al. (2003)
<i>A. mellifera</i>	Lab + oral: chronic exposure (no information on concentration)	LOEC on survival of winter bees: 24 µg kg ⁻¹	Decourtye et al. (2003)
<i>A. mellifera</i>	Lab + contact: acute exposure to 1.25–20 ng bee ⁻¹	LOEC on associative learning via PER assay on winter bees (12 µg kg ⁻¹) and summer bees (12 µg kg ⁻¹) LOEC for PER habituation: 1.25 ng bee ⁻¹	Lambin et al. (2001)
<i>A. mellifera</i>	Lab + oral: acute exposure to 0.1 and 81 ng bee ⁻¹	LOEC for mobility: 1.25 ng bee ⁻¹ ; mobility reduced at 2.5–20 ng bee ⁻¹ LD ₅₀ -48 h: between 41 and >81 ng bee ⁻¹ ; NOED: ≤1.25 ng bee ⁻¹ ; reduced sucrose uptake by 33% at 81 ng bee ⁻¹	Nauen et al. (2001)
<i>A. mellifera</i>	Lab + contact: acute exposure to 40–154 ng bee ⁻¹	LD ₅₀ -48 h: between 49 and 104 ng bee ⁻¹	Nauen et al. (2001)
<i>A. mellifera</i>	Lab + oral: acute exposure to 0.7 mg seed ⁻¹	LD ₅₀ -48 h: 4–41 ng bee ⁻¹	Schmuck et al. (2001)
<i>A. mellifera</i>	Lab + oral: chronic exposure (39 days) to sunflower nectar contaminated with 0.002–0.02 µg kg ⁻¹	NOEC for mortality, feeding activity, wax comb production, breeding performance and colony vitality: 0.02 µg kg ⁻¹	Schmuck et al. (2001)
<i>A. mellifera</i>	Summary of data of more than 30 lab and (semi-)field tests	Repellent antifeedant effect at 500–1,000 µg l ⁻¹ No adverse effects expected at residue levels of <20 µg l ⁻¹	Maus et al. (2003)
<i>A. mellifera</i>	Field + oral: chronic exposure	NOEC for the intraspecific communication: 10 µg l ⁻¹	Kirchner (1999)
<i>A. mellifera</i>	Lab + oral: acute exposure to 10–10,000 µg l ⁻¹	NOEC for survival, foraging activity, colony development, brood status and changes in pollen/nectar stores: 20 µg l ⁻¹	Suchail et al. (2001)
<i>A. mellifera</i>	Lab + oral: young bees chronically (10 days) exposed to 0.1, 1 and 10 µg l ⁻¹	LD ₅₀ -48 h: 60 ng bee ⁻¹ 50% Mortality	Suchail et al. (2001)
<i>B. impatiens</i>	Lab + contact: acute exposure via a Potter spray tower (no information on concentration range)	LC ₅₀ -48 h: 322 mg l ⁻¹	Scott-Dupree et al. (2009)
<i>O. lignaria</i>		LC ₅₀ -48 h: 7 mg l ⁻¹	
<i>M. rotundata</i>		LC ₅₀ -48 h: 17 mg l ⁻¹	

NOEC no-observed effect concentration, NOED no-observed effect dose, LOEC lowest observed effect concentration, PER proboscis extension reflex

Upon topical treatment, nitro-containing neonicotinoids (imidacloprid, clothianidin, thiamethoxam, nitenpyram and dinotefuran) were more toxic than the cyano-group containing ones (acetamiprid and thiacloprid) (Iwasa et al. 2004; Laurino et al. 2011). A similar high toxicity of imidacloprid and thiamethoxam was also found for the bumble bee *Bombus terrestris* (Mommaerts et al. 2010). The lower toxicity of the cyano-group neonicotinoids can be attributed to their fast biotransformation (Suchail et al. 2004a, b; Brunet et al. 2005) and the existence of different nAChR subtypes (Jones et al. 2006). For contact exposure Iwasa et al. (2004) ranked the neonicotinoid insecticides based on their 24-h LD₅₀ as follows: for the nitro-group: imidacloprid (18 ng bee⁻¹) > clothianidin (22 ng bee⁻¹) > thiamethoxam (30 ng bee⁻¹) > dinotefuran (75 ng bee⁻¹) > nitenpyram (138 ng bee⁻¹); and for the cyano-group: acetamiprid (7 µg bee⁻¹) > thiacloprid (15 µg bee⁻¹).

Metabolites of neonicotinoids were shown to contribute to the toxicity (Table 3) (Nauen et al. 2001, 2003; Suchail et al. 2001; Decourtye et al. 2003) except for acetamiprid with none of the metabolites being toxic (Iwasa et al. 2004). So far, most studies were conducted on metabolites of imidacloprid: those with a nitroguanidine-group (olefin-, hydroxy-, and dihydroxy-imidacloprid) were more toxic (oral LD₅₀) compared to the urea-metabolite and 6-chloronicotinic acid (Nauen et al. 2001). The metabolite of thiamethoxam, clothianidin was highly toxic for bees (Nauen et al. 2003).

For imidacloprid the toxicity varied upon insect-related factors such as the age of the bee, the colony, the subspecies used (Suchail et al. 2000, 2001; Nauen et al. 2001; Guez et al. 2003) and the health of the bees with sub-optimal protein feeding (Wehling et al. 2009) or *Nosema ceranae* infestation (Alaux et al. 2010; Vidau et al. 2011) making the bees more sensitive. Stark et al. (1995) found no effect of bee genera as the 24-h-contact LD₅₀s for imidacloprid were similar in both social bees (*A. mellifera*) and solitary bees (*Megachile rotundata* and *Nomia melanderi*) (Table 2). Similar conclusions were also drawn for thiamethoxam with an LD₅₀ of 30 ng bee⁻¹ for *A. mellifera* and 33 ng bee⁻¹ for *B. terrestris* (Iwasa et al. 2004; Mommaerts et al. 2010). Scott-Dupree et al. (2009), however, found that bumble bees (*Bombus impatiens*) were more tolerant to clothianidin and imidacloprid than *Osmia lignaria* and *M. rotundata*.

Chronic lethal toxicity

Chronic oral/contact exposure during 10–11 days to 1 µg bee⁻¹ acetamiprid and 1 ng bee⁻¹ thiamethoxam caused no significant worker mortality (Aliouane et al. 2009). For imidacloprid, laboratory tests showed high worker loss when honey bees consumed contaminated pollen

(40 µg kg⁻¹) (Decourtye et al. 2001, 2003) and sugar water (0.1, 1.0 and 10 µg l⁻¹) (Suchail et al. 2001). These results were in disagreement with field studies. Schmuck et al. (2001) reported no increased worker mortality when honey bee hives were exposed during 39 days to sunflower nectar contaminated with imidacloprid in a range of 2.0–20 µg kg⁻¹. Also Faucon et al. (2005) and Cresswell (2011) concluded that oral exposure to food contaminated with imidacloprid at realistic field concentrations did not result in worker mortality. A possible explanation for this discrepancy between laboratory and field studies may be differences in experimental methodology. Indeed the toxic effect on an individual may depend on its initial physiological state and on the longevity of nest mates (Decourtye and Devillers 2010). In addition, the social interaction should be taken into consideration with exposure of honey bees over a longer period. For bumble bees the chronic toxicity of compounds (exposure time up to 11 weeks) can be determined using micro-colonies (Mommaerts and Smaghe 2011).

Sublethal effects on reproduction

Reproduction is an important process to assure the further existence of the colony. Indeed, a loss of reproduction (brood) might be more detrimental for the colony than the loss of older bees (foragers) (Decourtye and Devillers 2010). This is further supported by studies on the division of tasks in bee colonies. For example in bumble bees (*B. impatiens*) task division is a dynamic process (weak task specialization) and so workers perform multiple tasks during their lifespan (Jandt and Dornhaus 2009). Therefore it is not unlikely that foragers are replaced by other bees when enough nurses are present in the hive. A few studies have demonstrated the adverse effects on larval development following exposure to imidacloprid (Tasei et al. 2000, 2001; Decourtye et al. 2005; Abbott et al. 2008; Gregorc and Ellis 2011). Decourtye et al. (2005) reported a delay in the time needed for honey bee larvae to hatch or develop as an adult when fed with food contaminated with imidacloprid at 5 µg kg⁻¹. Similar observations were also made by Abbott et al. (2008) for *O. lignaria* when imidacloprid was dosed at 30–300 µg kg⁻¹ food. Also for bumble bees (*B. terrestris*) a reduction of the brood (larvae) was seen in micro-colonies orally exposed to contaminated sugar water (10 µg kg⁻¹ imidacloprid) + pollen (6 µg kg⁻¹ imidacloprid) (Tasei et al. 2000) (Table 4).

Sublethal effects on behavior

Sublethal effects which interfere with the process of food collection and subsequent social colony life and pollination

Table 3 Lethal effect concentrations of neonicotinoids for workers of the honey bee (*A. mellifera*) by oral and contact exposure as determined in different laboratory studies

Neonicotinoid	Exposure	LD ₅₀ ($\mu\text{g bee}^{-1}$)	References
Parent compound			
Acetamiprid	Contact: individual bee (acute; no info on concentration range)	24 h: 7.07	Iwasa et al. (2004)
Acetamiprid	Contact + oral: individual bee (acute; no information on concentration range)	48 h: 14.5 (oral) + 8.09 (contact)	Decourtye and Devillers (2010)
Acetamiprid	Contact to dry residue + oral: 100 mg l ⁻¹ (acute; 2 days exposure for contact and 3 days for oral exposure)	Harmless	Laurino et al. (2011)
Clothianidin	Contact: individual bee (acute; no information on concentration range)	24 h: 0.022	Iwasa et al. (2004)
Clothianidin	Contact + oral: individual bee (acute; no information on concentration range)	48 h: 0.044 (contact) + 0.003 (oral)	Decourtye and Devillers (2010)
	Contact to dry residue: 75–1.5 mg l ⁻¹ (2 days exposure)	No LD ₅₀ determined but 100% loss at 15 mg l ⁻¹ after 48 h	Laurino et al. (2011)
	Oral: 75 mg l ⁻¹ to 7.5 $\mu\text{g l}^{-1}$ (acute; 3 days)	48 h: 0.003 $\mu\text{g l}^{-1}$	
Dinotefuran	Contact: individual bee (acute; no information on concentration range)	24 h: 0.075	Iwasa et al. (2004)
Dinotefuran	Oral: individual bee (acute; no information on concentration range)	48 h: 0.023	Decourtye and Devillers (2010)
Nitenpyram	Contact: individual bees (acute; no information on concentration range)	24 h: 0.138	Iwasa et al. (2004)
Thiacloprid	Contact: individual bees (acute; no information on concentration range)	24 h: 14.6	Iwasa et al. (2004)
Thiacloprid	Contact: individual bees (acute; no information on concentration range)	24 h: 24.2	Elbert et al. (2000)
Thiacloprid	Contact + oral: individual bees (acute; no information on concentration range)	48 h: 38.8 (contact) + 17.3 (oral)	Decourtye and Devillers (2010)
Thiacloprid	Contact dry residue + oral: 144 mg l ⁻¹ (acute; 2 days exposure for contact)	Harmless	Laurino et al. (2011)
Thiamethoxam	Contact: individual bees (acute; no information on concentration range)	24 h: 0.03	Iwasa et al. (2004)
Thiamethoxam	Contact + oral: individual bees (acute; no information on concentration range)	48 h: 0.024 (contact) + 0.005 (oral)	Decourtye and Devillers (2010)
Thiamethoxam	Contact dry residue: 100–1 mg l ⁻¹ (2 days exposure)	No LD ₅₀ determined but 100% loss at 100 mg l ⁻¹ after 6 h	Laurino et al. (2011)
Thiamethoxam	Oral: 100 mg l ⁻¹ to 10 $\mu\text{g l}^{-1}$ (3 days exposure)	48 h: 0.004 $\mu\text{g l}^{-1}$	Laurino et al. (2011)
Metabolite (parent compound)			
N-demethyl acetamiprid (acetamiprid)	Contact: individual bees (acute; no information on concentration range)	24 h: >50	Iwasa et al. (2004)
6-Chloro-pyridinylmethyl alcohol (acetamiprid)	Contact: individual bees (acute; no information on concentration range)	24 h: >50	Iwasa et al. (2004)

Table 3 continued

Neonicotinoid	Exposure	LD ₅₀ ($\mu\text{g bee}^{-1}$)	References
6-Chloro-nicotinic acid (acetamiprid)	Contact: individual bees (acute; no information on concentration range)	24 h: >50	Iwasa et al. (2004)
Oleofin (imidacloprid)	Oral: acute (no information on concentration range)	48 h: >0.036	Nauen et al. (2001)
Oleofin (imidacloprid)	Oral: 10–10,000 $\mu\text{g kg}^{-1}$ (acute)	48 h: 0.028 (acute)	Suchail et al. (2001)
	Oral: 0.1–10 $\mu\text{g l}^{-1}$ (chronic: 10 days)	no LD ₅₀ determined (chronic) but 30% mortality with 1 $\mu\text{g l}^{-1}$ after 125 h	
5-OH-imidacloprid (imidacloprid)	Oral: acute (no information on concentration range)	48 h: 0.159	Nauen et al. (2001)
5-OH-imidacloprid (imidacloprid)	Oral: 1.25–20 mg l^{-1} (acute)	48 h: 0.153	Decourtye et al. (2003)
5-OH-imidacloprid (imidacloprid)	Oral: 10–10,000 $\mu\text{g kg}^{-1}$ (acute)	48 h: 0.258 (acute)	Suchail et al. (2001)
	Oral: 0.1–10 $\mu\text{g l}^{-1}$ (chronic: 10 days)	no LD ₅₀ determined (chronic) but 40% mortality with 1 $\mu\text{g l}^{-1}$ after 125 h	
Di-OH-imidacloprid (imidacloprid)	Oral: acute (no information on concentration range)	48 h: >0.049	Nauen et al. (2001)
Urea-metabolite (imidacloprid)	Oral: acute (no information on concentration range)	48 h: >100	Nauen et al. (2001)
6-Chloronicotinic acid (imidacloprid)	Oral: acute (no information on concentration range)	48 h: >122	Nauen et al. (2001)

need to be considered (Thompson and Maus 2007; Desneux et al. 2007; Mommaerts and Smagge 2011). Over the past years several laboratory and (semi-) field tests have been developed to investigate the effect of neonicotinoid insecticides on motor and sensory functions linked to the foraging capacity of bees.

Neonicotinoid insecticides act as neurotoxic agents and affect the mobility of bees by inducing symptoms such as knockdown, trembling, uncoordinated movements, hyperactivity and tremors (Lambin et al. 2001; Nauen et al. 2001; Suchail et al. 2001; Medrzycki et al. 2003; Colin et al. 2004). These symptoms are easy to observe at high exposure levels, while the effect of a lower dose might be more difficult to see. El Hassani et al. (2005) therefore developed a new laboratory test consisting of a plastic box with a transparent plate that was illuminated, enabling to record the vertical displacement of the bees. Contact exposure to imidacloprid at 1.25 ng bee^{-1} and to acetamiprid at $\leq 0.5 \mu\text{g bee}^{-1}$ increased locomotor activity, whereas imidacloprid at 2.5 ng bee^{-1} significantly decreased bee mobility (Lambin et al. 2001). No negative effects on the locomotor activity were found after acute and chronic (11 days) exposure (oral) to acetamiprid at 0.1 $\mu\text{g bee}^{-1}$ and after acute exposure (contact and oral) to thiamethoxam at 1 ng bee^{-1} (El Hassani et al. 2008; Aliouane et al. 2009).

Another sublethal endpoint affected by neonicotinoids (acetamiprid and thiamethoxam) is the proboscis extension reflex (PER) following perception of sucrose and water (El Hassani et al. 2008; Aliouane et al. 2009). The effect was demonstrated to be dependent on the route, duration and dose of exposure (El Hassani et al. 2008; Aliouane et al. 2009). In addition, by conditioning of the PER using an odor, various studies demonstrated changes in the olfaction learning of bees upon exposure to neonicotinoids. Learning was reduced after chronic (up to 11 days) exposure to imidacloprid (winter bees: 48 $\mu\text{g kg}^{-1}$; oral), the metabolite 5-hydroxy-imidacloprid (winter bees: 120 $\mu\text{g kg}^{-1}$; oral) and thiamethoxam (0.1 ng bee^{-1} ; contact) (Decourtye et al. 2003; El Hassani et al. 2008; Aliouane et al. 2009). By expanding the PER test also more information was gained on how neonicotinoids interfere with the memory process. Oral uptake of 0.1 $\mu\text{g bee}^{-1}$ acetamiprid induced long-term memory impairments, whereas chronic contact to 1 ng bee^{-1} thiamethoxam (corresponding with 1/5 of the LD₅₀) did not cause long-term effects as recovery of memory was seen after 48 h (El Hassani et al. 2008; Aliouane et al. 2009). For imidacloprid, different authors reported on medium-term memory effects (Table 2) (Decourtye et al. 2001, 2003, 2004a; Lambin et al. 2001). Decourtye et al. (2004b) documented that such effects may result from an increase of the cytochrome oxidase activity, related with aberrations of the mushroom bodies in the brain. The effects of imidacloprid

Table 4 Overview of the concentrations of imidacloprid causing lethal and sublethal effects on (micro-)colony level in honey bees (*A. mellifera*) and bumble bees (*B. terrestris*, *B. impatiens*) as determined in different studies by oral/contact exposure under laboratory and (semi-)field conditions

Species	Exposure	Toxicity	References
<i>A. mellifera</i>	Field + oral: 0.5–5 $\mu\text{g l}^{-1}$ in syrup (chronic)	NOEC for survival: 5 $\mu\text{g l}^{-1}$	Faucon et al. (2005)
<i>A. mellifera</i>	Lab + oral: 100–1,000 $\mu\text{g l}^{-1}$ (acute)	NOEC for brood, adult foraging activity, adult bee population level, number of frames with brood after overwintering and general colony vitality: 5 $\mu\text{g l}^{-1}$ 500–1,000 $\mu\text{g l}^{-1}$; bees disappeared at the hive/feeding site up to 24 h	Bortolotti et al. (2003)
<i>A. mellifera</i>	Lab + oral: 0.12 and 12 ng bee^{-1} in syrup (acute)	100 $\mu\text{g l}^{-1}$; no effect on homing rate	Decourtye et al. (2004a, b)
	Lab + oral: 24 $\mu\text{g kg}^{-1}$ in syrup (24 h)	Increase of the cytochrome oxidase labeling, negative effect on the PER assay with 12 ng bee^{-1} but not with 0.12 ng bee^{-1}	
	Semi-field + oral: 24 $\mu\text{g kg}^{-1}$ in syrup (24 h)	Negative effect on the PER assay	
<i>A. mellifera</i>	Field + oral: foraging on maize fields treated with imidacloprid (chronic; no information about the dose)	Decreased foraging activity, hive entrance activity, sucrose consumption and brood size	Nguyen et al. (2009)
<i>A. mellifera</i>	Semi-field + oral: 40–6,000 $\mu\text{g l}^{-1}$ in syrup (acute)	No relation between imidacloprid treated maize fields and bee mortality in apiaries	Yang et al. (2008)
	Lab + oral: >100 $\mu\text{g kg}^{-1}$ (chronic)	LOEC for foraging behavior: 50 $\mu\text{g l}^{-1}$ >1,200 $\mu\text{g l}^{-1}$ abnormalities in revisiting the feeding site	
<i>A. mellifera</i>	Lab + oral: 10–25 $\mu\text{g kg}^{-1}$ in syrup + 6–16 $\mu\text{g kg}^{-1}$ in pollen (chronic exposure: 85 days)	NOEC survival: 2–20 $\mu\text{g kg}^{-1}$ in sunflower nectar 20 $\mu\text{g l}^{-1}$; decrease in foraging activity; >100 $\mu\text{g l}^{-1}$; reduce in foraging behavior for 30–60 min >50 $\mu\text{g l}^{-1}$; increase in interval between successive visits at a feeder	Schmuck (1999); Schmuck et al. (2001)
<i>A. mellifera</i>	Semi-field + oral: 48 $\mu\text{g kg}^{-1}$ in syrup (chronic)	Affected syrup consumption, foraging activity and brood size	Ramirez-Romero et al. (2005)
<i>B. terrestris</i>	Lab + oral: 10–25 $\mu\text{g kg}^{-1}$ in syrup + 6–16 $\mu\text{g kg}^{-1}$ in pollen (chronic exposure: 85 days)	Increased worker mortality after 30 days	Tasei et al. (2000)
<i>B. terrestris</i>	Field + oral: foraging on plants grown from imidacloprid-treated sunflower seeds treated with 0.7 mg seed^{-1} (chronic exposure: 9 days)	No effect on food consumption and male emergence; but 10 $\mu\text{g kg}^{-1}$ in syrup + 6 $\mu\text{g kg}^{-1}$ in pollen: reduction in brood size	Tasei et al. (2001)
	Lab + oral: 200 mg l^{-1} to 10 $\mu\text{g l}^{-1}$ in sugar water (chronic 11 weeks: test without foraging; test with foraging)	No effect on foraging and colony vitality	
<i>B. terrestris</i>	Semi-field + oral: 20–2 $\mu\text{g l}^{-1}$ in sugar water (chronic 2 weeks)	Test without foraging: LC ₅₀ : 59 $\mu\text{g l}^{-1}$; EC ₅₀ : 37 $\mu\text{g l}^{-1}$; NOEC for reproduction: 20 $\mu\text{g l}^{-1}$	Mommaerts et al. (2010); Mommaerts and Smaghe (2011)
<i>B. terrestris</i>	Lab + oral: 7–30 ng g^{-1} pollen (chronic: twice weekly)	Test with foraging: LC ₅₀ : 20 $\mu\text{g l}^{-1}$; EC ₅₀ : 3.7 $\mu\text{g l}^{-1}$; NOEC for reproduction: <2.5 $\mu\text{g l}^{-1}$	Mommaerts et al. (2010)
<i>Bombus occidentalis</i> , <i>B. impatiens</i>		NOEC for brood, colony growth and foraging activity: 2 $\mu\text{g l}^{-1}$ NOEC for brood, numbers of males, queens and workers, worker weight, pollen consumption, forage ability on complex artificial flowers: 7 ng g^{-1}	Morandin and Winston (2003)

Table 4 continued

Species	Exposure	Toxicity	References
<i>B. impatiens</i>	Field + dry residue (acute exposure) Granules + spray: 0.45 kg ha ⁻¹ Spray + irrigation: 0.34 kg ha ⁻¹ Spray + non-irrigated: 0.34 kg ha ⁻¹	No effect on colony vitality and worker behaviour No effect on colony vitality, worker defensive response and on foraging preference Reduction in numbers of brood chambers, honey pots, workers, colony weight and on foraging preference	Gels et al. (2002)

NOEC no-observed effect concentration, LOEC lowest observed effect concentration, PER proboscis extension reflex

on habituation of PER depended on the age of the bees tested and thus on their task within the colony (Guez et al. 2001, 2003). Although it is obvious that neonicotinoids can interfere with the olfactory learning process in different ways, extrapolation of these laboratory effects to a real exposure situation in the field therefore is complex and difficult.

Neurotoxic compounds such as neonicotinoids were also reported to interfere with the orientation process of honey bees. Associative learning between a visual mark and a reward (sugar solution) in a complex maze showed that only 38% of the bees found the food source after oral ingestion of thiamethoxam at 3 ng bee⁻¹ compared to 61% in the control group (Decourtye and Devillers 2010). In another study using marked foragers that were first trained to forage on artificial feeders, Bortolotti et al. (2003) noticed that a 500 m distance between the hive and the feeding area resulted in no foragers at the hive/feeding area up to 24 h after treatment when foragers were fed with imidacloprid at 500 and 1,000 µg l⁻¹ (Table 4). The latter authors also found that a lower concentration (100 µg l⁻¹ imidacloprid) caused a delay in the returning time (to hive or feeding area) of the foragers. This was confirmed by Ramirez-Romero et al. (2005) and Yang et al. (2008). Based on these results it is obvious that neonicotinoids interfere with the foraging capacity of bees. However, the different (semi-)field studies provide a mixed pallet of results. For instance, Cutler and Scott-Dupree (2007) reported no side-effects on honey bees foraging when hives were exposed to flowering canola grown from clothianidin-treated seeds. The same conclusion was drawn for imidacloprid (Schmuck et al. 2001; Faucon et al. 2005; Nguyen et al. 2009), but for thiacloprid foraging was only reduced up to 48 h after treatment (Schmuck et al. 2003). Similarly, there was no negative effect on *B. terrestris* foraging on imidacloprid- and thiamethoxam-treated plants (Colombo and Buonocore 1997; Tasei et al. 2001; Alarcón et al. 2005), and also no side-effects on *B. impatiens* exposed to weedy turf treated with imidacloprid by irrigation, to field residue levels of imidacloprid and to the highest residue level of clothianidin recovered in pollen (6 µg kg⁻¹) (Gels et al. 2002; Morandin and Winston 2003; Franklin et al. 2004). It needs to be remarked that the *B. impatiens* colonies, foraging on non-irrigated imidacloprid-treated weed, showed a significant reduction in nest development (brood chambers, honey pots and worker biomass) and foraging activity (Gels et al. 2002). From these observations it is clear that there exists a discrepancy between field and laboratory tests for sublethal effects. Decourtye and Devillers (2010) documented that this was due to the ability of bees to change their behavior in response to pesticide perception. Indeed, honey bees responded by rejection when they perceived a sucrose solution contaminated with 20 µg l⁻¹ imidacloprid, which resulted in a significant reduction of the foraging activity (Mayer and Lunden 1997;

Kirchner 1999; Schmuck 1999; Maus et al. 2003). This protective avoidance behavior of bees towards contaminated food might reduce risk of pesticide exposure and effects. Such behavior on the other hand contributed to a decrease in general fitness of the bees with 6–20%, as deduced from statistically fitted performance data (Cresswell 2011).

It has recently been shown that bees became exposed to neonicotinoids in seed-coated fragments also via guttation fluid. After feeding on dew no honey bee mortality was observed, but feeding guttation fluid from directly treated plants did result in high mortality (Girolami et al. 2009). Also direct exposure to dust from the planting machine resulted in high bee mortality (Marzaro et al. 2011). In the latter experiments, clothianidin residues in dead bees averaged 279 ± 142 ng bee⁻¹ at high humidity and 514 ± 174 ng bee⁻¹ at low humidity, which by far exceed the LD₅₀ of 21.8 ng bee⁻¹. Similar findings were also reported by Girolami et al. (2011), exposing honey bees to dust from clothianidin and imidacloprid-treated seeds. Their study showed that mortality of exposed honey bees only occurred at high air humidity.

Effects on overwintering of bees

During the last years a loss of overwintering bee colonies was noticed. Although identification of the causes of this disappearance is difficult, it was argued that reduced bee health might be initially caused by the chronic exposure to pesticides. So far only two studies have been conducted in this context for neonicotinoids. Using 8 honeybee colonies, Faucon et al. (2005) demonstrated that chronic exposure during the summer season (33 days) to 0.5 and 5.0 µg l⁻¹ imidacloprid in saccharose syrup did not affect the overwintering abilities of honey bees. Similarly, spring assessment of colony development (brood, worker biomass and colony health) was not affected in overwintered colonies that had foraged on flowering canola grown from seed treated with clothianidin at 0.4 mg kg⁻¹, representing the highest recommended rate (Cutler and Scott-Dupree 2007). In conclusion, these studies demonstrated no long-term effects on honeybee colonies of environmentally relevant concentrations.

Mixture toxicity

This section will focus on cases in which synergistic effects were found when exposing organisms to mixtures containing neonicotinoids insecticides.

Only one study is available on the toxicity of neonicotinoids in mixtures to pollinators. Iwasa et al. (2004)

found that addition of piperonyl butoxide and the fungicides triflumizole and propiconazole increased the acute toxicity (24-h LD₅₀, topical application) of acetamiprid and thiacloprid to honey bees (*A. mellifera*) by factors of 6.0, 244 and 105, and 154, 1141 and 559, respectively, but had little effect on the toxicity of imidacloprid (1.5–1.9 times more toxic). The toxicity of acetamiprid was 6.3–84 times increased by the fungicides triadimefon, epoxiconazole and uniconazole-P. All synergists were topically applied at a dose of 10 µg bee⁻¹ and 1 h before dosing the insecticides (Iwasa et al. 2004).

In grass shrimp larvae (*Palaemonetes pugio*) slightly synergistic effects were found when imidacloprid was applied together with atrazine (Key et al. 2007) with 96-h LC₅₀ values ranging between 0.83 and 0.93 toxic units.

The toxicity of mixtures of imidacloprid and thiacloprid for earthworms (*Eisenia fetida*) was sometimes higher than expected from the toxicities of the individual chemicals. This was especially the case for earthworm weight change in a clay loam soil, where a dose-ratio dependent deviation was seen suggesting a shift from antagonism to synergism when thiacloprid accounted for more than 88% of the toxicity of the mixture (Gomez-Eyles et al. 2009). For effects on the reproduction of both nematodes (*Caenorhabditis elegans*) and daphnids (*Daphnia magna*), the mixture of imidacloprid and thiacloprid showed a dose-level dependent deviation from additivity, with synergism at low and antagonism at high exposure levels. For nematodes, the switch occurred at approximately 95% of the EC₅₀ (Gomez-Eyles et al. 2009), while for daphnids this was the case at 1.5 times the EC₅₀ (Pavlaki et al. 2011). Gene response profiles (transcriptomics, proteomics) in marine mollusks (*Mytilus galloprovincialis*) showed different patterns for the mixture compared to the single compounds, suggesting that the mode of action at the molecular level may be quite distinct (Dondero et al. 2010).

Synergism for effects on the population growth rate of *Ceriodaphnia dubia* was found by Chen et al. (2010) when determining the toxicity of a mixture of the nonylphenol polyethoxylate R11 and imidacloprid. Results of this study are, however, hard to interpret as only one concentration was tested. A mixture of imidacloprid with nickel showed synergistic effects on body length development of *D. magna* (Pavlaki et al. 2011).

It remains unclear how these data can be extrapolated to bee-relevant exposure situations, although it may be noted that studies of Mullin et al. (2010), Genersch et al. (2010) and Bernal et al. (2010) showed the presence of large numbers of different pesticides in bee-collected products like pollen, honey and beeswax. The data do, however, not allow for a quantitative risk analysis of possible mixture exposure.

Risk assessment scheme for hazards by neonicotinoids in bees

A risk assessment for systemic compounds starts by identification of the exposure risk (Alix et al. 2009; Thompson 2010; Fischer and Moriarty 2011). In case exposure is likely to occur because bees are attracted to the crop and the compound can be translocated to the nectar and pollen further assessment is crucial. As given above, neonicotinoids show good systemic properties and are recovered in nectar and pollen, therefore suggesting this scheme for risk assessment can be applied for neonicotinoids.

At present Tier-1 recommends acute toxicity testing on adults and brood. However, to estimate the impact of neonicotinoids in the field a first screening should include environmental relevant doses. For neonicotinoids, contaminated food was already demonstrated to be transported to the hive where it can either be stored or used as food for larvae and adults or where it can enter the wax of the combs. In this context, Wu et al. (2001) found no larval mortality but demonstrated delayed worker development when brood was reared in highly contaminated (including low residue concentrations of several neonicotinoids) brood combs. Consequently, side-effects on brood by neonicotinoids must be assessed and no-observable effect levels (NOEL) need to be determined. When working with honey bees, care is needed as one bee gathers food and transmits it to nest mates by trophallaxis. A first study did not notice a difference between honey bees fed with imidacloprid individually or in a group as the 48-h LD_{50} of 25 ng bee^{-1} was equal for both (Decourtye and Devillers 2010). Nonetheless, future studies should give more attention to this as dilution of the product is likely to occur when food is transmitted between nest mates.

In Tier-2 the NOEL as determined under Tier-1 is used to determine the chronic oral toxicity for individual adult bees. Acute toxicity gives a first indication of the real risk but it is still an incomplete measurement. Therefore potential side-effects after long-term exposure (contact and acute) to neonicotinoids need to be evaluated. Honey bees have been exposed for a maximum of 10–11 days and 39 days in the different respective laboratory and field tests reported so far. Indeed the need for a more standardized approach on bee age, colony size and appropriate exposure was also confirmed by the Cox proportional hazard model of Dechaume-Moncharmont et al. (2003) during a 60-day dietary exposure with imidacloprid at 4 and $8 \mu\text{g l}^{-1}$. Tier-2 testing requires to consider both adult and larval stages because residues are recovered in their food, which includes pollen and nectar. Adult bees consume more nectar than pollen, while larval stages consume more pollen than nectar (Rortais et al. 2005). For the adults, a good knowledge on their foraging behavior on the crop is

crucial: for instance, is the bee attracted to nectar or pollen or to both? As documented above, neonicotinoids may be translocated to both compartments of nectar and pollen, however, residue analyses so far have mainly focused on pollen. As a consequence, more data on nectar contamination need be collected since it is difficult to extrapolate toxicity data obtained with pollen to nectar. Halm et al. (2006) also confirmed the need for a better standardization of the bee categories in risk assessment as the calculated exposure to imidacloprid was higher for the group of winter bees, nectar foragers and nurses than for the group of workers and drone larvae, wax-producing bees and pollen foragers. The latter authors propose to use the predicted environmental concentration/predicted no effect concentration (PEC/PNEC) ratio approach to determine the risk instead of using LD_{50} or LC_{50} values.

Higher tier risk assessments are conducted on the colony level to include the effect of social interaction. This phase of the assessment is needed to enable drawing firm conclusions on the compatibility of the compound under field conditions. The results obtained so far for neonicotinoids (mainly for imidacloprid) under laboratory conditions do not give a good estimation of the real effect on honey bees under field conditions. Indeed honey bees only needed to use a limited number of cues in a complex maze in laboratory studies, whereas visual learning in the field is more complex. Yang et al. (2008) reported on the use of foraging bees that have been trained prior to the risk assessment test, however, the marking is very labor intensive. Alternatively, Decourtye et al. (2011) connected a microchip to the honey bee body to assess sublethal effects on the number of foraging trips by low concentrations of fipronil. For bumble bees specifically, Mommaerts et al. (2010) developed a “foraging behavior” bioassay that allows to assess in the laboratory the sublethal effects on foraging by imidacloprid, as observed in free-flying bumble bee workers in the greenhouse.

As already mentioned at Tier-1, further improvement of the reliability will be obtained when tests can be performed with environmentally relevant concentrations. The field risk assessment studies should cover all potential routes of exposure. Exposure to neonicotinoids in dust from the planting machine has been reported to result in high bee mortality, especially at high air humidities (Girolami et al. 2011; Marzaro et al. 2011). Further, exposure might also occur via the ingestion of contaminated guttation fluid. Although this route of exposure has been considered important, the data so far are not clear. As Thompson (2010) reported, the liquid is mainly present early in the morning and it remains unclear whether that corresponds to the time when bees or other pollinators are active and to what extent they ingest this fluid. In addition, it is not clear whether residues after drying of the liquid on the leaves

remain a source of exposure (Thompson 2010). Tapparo et al. (2011) also reported that imidacloprid concentrations in guttation fluid did show a clear correlation with the dose applied to the seeds. Therefore, as long as no firm conclusion can be drawn, it is advisable to include this route of exposure into a risk assessment scheme for neonicotinoids.

In conclusion, assessment of risks for side-effects by use of field trials remains the final step as the field is a complex environment in which different factors may influence neonicotinoid toxicity. Concerning the effect of social interaction it needs to be remarked that for other non-*Apis* genera such as bumble bees potential side-effects on colony level can be evaluated earlier in the risk assessment, namely under Tier-2. Indeed, a standardized test with micro-colonies allows evaluating lethal and sublethal effects of neonicotinoids on bee reproduction and behavior. Micro-colonies are nests made of 3–5 new-born workers (the same age). Then, after 1 week one worker becomes dominant, like a queen in greenhouse colonies, and starts laying unfertilized eggs that develop into males while the other workers take care of the brood and forage for food. The dominant worker functions as a pseudo queen and the others as nurses and foragers. Food consists of commercial sugar water and pollen. Subsequently, the impact of neonicotinoids can be tested via different routes of exposure, namely contact exposure and orally via the drinking of treated sugar water and by eating treated pollen for 7 weeks. Other advantages of this method are the low cost, the ease of use, the possibility to work with standardized protocols and with multiple replicates resulting in sufficient statistical power to obtain reproducible data. The experimental set-up also allows social interaction to take place. Lethal effects are evaluated by scoring the number of dead workers per nest while evaluation of sublethal effects occurs by scoring the presence of honey pots, the number of dead larvae and the number of males produced per nest (Mommaerts et al. 2006a, b; Besard et al. 2011). Based on the latter endpoints, Mommaerts et al. (2010) could determine that the NOEC values for imidacloprid using such micro-colonies were equal to those obtained when using queenright colonies in the greenhouse test.

Conclusions and targets for research and recommendations

Neonicotinoids are an important group of insecticides effective in the control of economically important pests such as aphids, leafhoppers and whiteflies. The wide application of these insecticides with a worldwide annual market of \$1 billion is attributed to their selective mode of action at low doses (Aliouane et al. 2009). Neonicotinoids act as neurotoxins on the insect nervous system by

interaction with the insect nAChR. In order to identify potential hazards of neonicotinoids to bees this study summarized all available data.

Via the plant sap transport neonicotinoids are translocated to different plant parts. In general, the few reported residue levels of neonicotinoids in nectar (average of $2 \mu\text{g kg}^{-1}$) and pollen (average of $3 \mu\text{g kg}^{-1}$) were below the acute and chronic toxicity levels; however, there is a lack of reliable data as analyses are performed near the detection limit. Similarly, also the levels in bee-collected pollen, in bees and bee products were low. But before drawing a conclusion, it is strongly encouraged to conduct more studies as so far only a few large studies have been undertaken in apiaries in France, Germany and North America. Moreover, the wide and increasing application of neonicotinoids in pest control will likely cause an accumulation of neonicotinoids in the environment in the future.

Many lethal and sublethal effects of neonicotinoid insecticides on bees have been described in laboratory studies, however, no effects were observed in field studies with field-realistic dosages.

The risk assessment scheme for soil-applied systemic pesticides proposed by Alix et al. (2009) and Thompson (2010) seems adequate for assessing the risks of side-effects by neonicotinoids as it takes into account the effect on different stages (adult versus larvae) and on different levels of biological organization (organism versus colony). Nevertheless, there is still a need for testing field-realistic concentrations at relevant exposure and durations and, especially for honey bees, to continue side-effect evaluation over winter and the next year in spring. The scoring of sublethal effects related to foraging behavior and learning/memory abilities, however, is very difficult. As the genomes of honey bees (*A. mellifera*) and bumble bees (*B. terrestris*, *B. impatiens*) are available, these may help to better understand the complex (network) mechanisms under natural conditions in bees. Then, treatment with pesticides like neonicotinoids will indicate which effects and responses take place at the molecular level and can be related to the exposure. A good example is the availability of a microarray of the brain of honeybees (Alaux et al. 2009). After validation, such gene/transcriptome responses can be employed as molecular ecotoxicological markers, which in turn can improve risk assessment. These molecular markers can be complementary to the robust classical endpoints of mortality and reproduction, which are assessed using individual insects and (micro-)colonies in accordance with the tier-level. These new molecular insights can also contribute to better understanding the mechanisms of action of neonicotinoids like their interaction with different nAChR in bees, also in relation to their pharmacokinetics and metabolism. The newer and safer

neonicotinoids, e.g. using the cyano-group instead of the nitro-group, are good examples for further development of environmentally safer compounds employing the existence of different nAChRs in the insect nervous system. The toxicity of neonicotinoids may, however, increase by synergistic effects with other compounds as was demonstrated by Iwasa et al. (2004) for mixtures containing a cyano-group neonicotinoid. Therefore, screening for safer compounds should also include gathering more information on potential synergistic effects of mixtures containing neonicotinoids as this is currently lacking.

Finally, during the preparation of this review it was observed that results/data on concentrations, side-effects and risk assessment studies are available, but that many data are scattered and/or not publicly available. A better communication between industry, academia and government may help for a “better” risk assessment. The latter can also help to provide answers to the questions/concerns as present in the public media/society.

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From: Pat Kwiatkowski [mailto:pat.kwiatkowski@bayer.com]
Sent: Wednesday, April 18, 2012 2:49 PM
To: Jennings, Henry
Subject: FW: Re: Recent Public Activity Surrounding Neonicotinoids

Dear Mr. Jennings,

Neonicotinoids have recently attracted considerable media attention due to a few highly publicized studies reported in the literature on alleged neonicotinoid impact on bees and a recent petition to EPA requesting a stop sale order be issued for Clothianidin based primarily on the alleged role of neonicotinoids in the decline of bee health. As Bayer CropScience is a major registrant of neonicotinoid products registered in your State, we would like to provide you with more information on the events leading up to the Clothianidin petition, an update on federal regulatory activities covering the neonicotinoid class of chemistry and the current scientific consensus on bee health.

As background to the current Clothianidin petition, in December 2010 EPA Administrator Jackson received a letter signed by several environmental organizations and the major beekeeper and honey producer associations requesting that EPA stop sale of Clothianidin due to its adverse impact on bee health. February 8, 2011, EPA responded that they were not aware of any data that reasonably demonstrated that bee colonies are subject to elevated losses due to chronic exposure to Clothianidin, and that they did not intend to initiate suspension or cancellation. At the same time, however, EPA committed to accelerating a comprehensive review of the neonicotinoid class of insecticides, indicating they would open the docket for Registration Review of Clothianidin before the end of 2011. The docket for public comment on Clothianidin opened on December 21, 2011 and closed on February 21, 2012. During this 90 day period, many comments were posted supporting the important role that Clothianidin plays in agriculture. There were also a significant number of comments in the docket against the continued use of Clothianidin products, but Bayer CropScience review found no new bee related data previously unknown to EPA.

Despite EPA's transparent and participative approach to the issue, a petition was filed with the Environmental Protection Agency (EPA) on March 20, 2012 signed by four environmental and consumer organizations (Beyond Pesticides, Center for Food Safety, International Center for Technology Assessment, Pesticide Action Network North America [PANNA]) requesting that EPA suspends the registration of Clothianidin and stop sales. In contrast to the 2010 petition, only a few individual beekeepers were supporters; the major beekeeper and honey producer associations did not sign this petition.

The petition has coincided with a well-coordinated PR campaign that has been used to completely overstate the importance of a few studies published in late March claiming that scientists have *at last* determined that neonicotinoid insecticides are the cause of honey bee declines around the world. This culminated in a press release accompanying a study to be published in the *Bulletin of Insectology*, which claims imidacloprid is the "likely culprit" behind the worldwide decline in honey bee populations.

Expert opinion is that the study is heavily biased, poorly designed, factually inaccurate and seriously flawed, both in its methodology and conclusions. Although the study claims to have established a link between imidacloprid and bee colony collapse, the symptoms observed in the study bees are not consistent with, or even remotely similar to, those of Colony Collapse Disorder (CCD). The work has been heavily criticized by academia, beekeepers and the food industry alike, in particular the hypothesis that bees are exposed via residues present in corn syrup based on the use of these chemicals as corn seed treatments.

Most experts consider that the decline in honey bee populations is due to combination of factors, particularly parasitic mites and associated pathogens. Poor bee health correlates extremely well with the presence of Varroa mites and diseases, but does not correlate at all with pesticides. In 2012 alone there have been several extensive reviews published confirming this, including the update from the University of Georgia as leader of the USDA Managed Pollinator CAP program, a 17-member consortium of university and federal bee labs "dedicated to the reversal of honey bee decline."

Bayer CropScience is committed to ensuring robust bee health as a fundamental component of sustainable agriculture (we have recently announced the establishing of Bayer Bee Care Centers in Europe and North America). We are committed to effective stewardship to help ensure all products, including neonicotinoids, are used according to label and in such a manner as to minimize exposure. We are also committed to on-going and targeted research on bee health issues. Bayer CropScience believes that EPA is currently following an appropriate and well defined process to ensure that the neonicotinoids are regulated under a robust science-based risk assessment process that takes account of all appropriate data, and we continue to work with them to ensure appropriate research and studies are conducted to address scientifically valid hypotheses put forth by credible experts.

Scientific, risk based decisions are made on a product's complete body of safety data. As a responsible regulatory authority, I am sure you agree that as for any pesticide, neonicotinoids should not be regulated based on media attention around a published research study of limited scope and claims professing impact that clearly extends far beyond the study's range. However, Bayer CropScience understands the public pressure that may ensue due to media activities, and I would like to assure you that we are available to answer any questions you may have pertaining to this issue. Please contact me if you have any questions on the current situation or the robustness of the data supporting the registration of neonicotinoids in your state. I can be reached by telephone at (919) 549-2480 or by e-mail at pat.kwiatkowski@bayer.com. I will also be attending the upcoming May SFIREG POM and June Full SFIREG meetings if you also happen to be attending and would like to discuss any aspect in person.

Sincerely,

Pat Kwiatkowski
Director State Regulatory Affairs and Documentation Services
Bayer CropScience LP
Telephone (919) 549 2480

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Relyea, Rick A. 2012. New effects of Roundup on amphibians: Predators reduce herbicide mortality; herbicides induce antipredator morphology. *Ecological Applications* 22:634–647. <http://dx.doi.org/10.1890/11-0189.1>

Articles

New effects of Roundup on amphibians: Predators reduce herbicide mortality; herbicides induce antipredator morphology

Rick A. Relyea¹

Department of Biological Sciences, University of Pittsburgh, Pittsburgh, Pennsylvania 15260 USA

The use of pesticides is important for growing crops and protecting human health by reducing the prevalence of targeted pest species. However, less attention is given to the potential unintended effects on nontarget species, including taxonomic groups that are of current conservation concern. One issue raised in recent years is the potential for pesticides to become more lethal in the presence of predatory cues, a phenomenon observed thus far only in the laboratory. A second issue is whether pesticides can induce unintended trait changes in nontarget species, particularly trait changes that might mimic adaptive responses to natural environmental stressors. Using outdoor mesocosms, I created simple wetland communities containing leaf litter, algae, zooplankton, and three species of tadpoles (wood frogs [*Rana sylvatica* or *Lithobates sylvaticus*], leopard frogs [*R. pipiens* or *L. pipiens*], and American toads [*Bufo americanus* or *Anaxyrus americanus*]). I exposed the communities to a factorial combination of environmentally relevant herbicide concentrations (0, 1, 2, or 3 mg acid equivalents [a.e.]/L of Roundup Original MAX) crossed with three predator-cue treatments (no predators, adult newts [*Notophthalmus viridescens*], or larval dragonflies [*Anax junius*]). Without predator cues, mortality rates from Roundup were consistent with past studies. Combined with cues from the most risky predator (i.e., dragonflies), Roundup became less lethal (in direct contrast to past laboratory studies). This reduction in mortality was likely caused by the herbicide stratifying in the water column and predator cues scaring the tadpoles down to the benthos where herbicide concentrations were lower. Even more striking was the discovery that Roundup induced morphological changes in the tadpoles. In wood frog and leopard frog tadpoles, Roundup induced relatively deeper tails in the same direction and of the same magnitude as the adaptive changes induced by dragonfly cues. To my knowledge, this is the first study to show that a pesticide can induce morphological changes in a vertebrate. Moreover, the data suggest that the herbicide might be activating the tadpoles' developmental pathways used for antipredator responses. Collectively, these discoveries suggest that the world's most widely applied herbicide may have much further-reaching effects on nontarget species than previous considered.

Key words: [American toads \(*Bufo americanus* or *Anaxyrus americanus*\)](#), [amphibian decline](#), [dragonflies \(*Anax junius*\)](#), [glyphosphate](#), [inducible defense](#), [leopard frogs \(*Rana pipiens* or *Lithobates pipiens*\)](#), [newts \(*Notophthalmus viridescens*\)](#), [phenotypic plasticity](#), [synergy](#), [wood frogs \(*Rana sylvatica* or *Lithobates sylvaticus*\)](#)

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[Joanna Hopkins, President, Blue River Watershed Group Board](#)

[6:45 p.m. How Low Will It Go: 2012's Historically Low Snowpack](#)

[7:00 p.m. Dillon Reservoir Operations](#)

[7:20 p.m. Green Mountain Operations](#)

[7:40 p.m. Update: The Colorado River Cooperative Agreement](#)

[8:00 p.m. Water Wranglers:](#)

Popular weedkiller causes deformities in amphibians

Posted on April 3, 2012 by Bob Berwyn

Biologist 'shocked' to see morphological changes in vertebrates

By Summit Voice

SUMMIT COUNTY — Exposure to sub-lethal doses of a widely used weed killer caused tadpoles to grow abnormally large tails, according to University of Pittsburgh biologist [Rick Relyea](#), who has been studying ecotoxicology and ecology for two decades.



Tadpoles exposed to Roundup® grow abnormally large tails. PHOTO COURTESY FRIEDRICH BOEHRINGER VIA THE CREATIVE COMMONS.

Relyea has conducted extensive research on the toxicity of Roundup® to amphibians. Monsanto has challenged some of the studies and Relyea has responded to the criticism on [this website](#).

In his latest study, Relyea set up large outdoor water tanks that contained many of the components of natural wetlands. Some tanks contained caged predators, which emit chemicals that naturally induce changes in tadpole morphology (such as larger tails to better escape predators). After adding tadpoles to each tank, he exposed them to a range of Roundup® concentrations. After 3 weeks, the tadpoles were removed from the tanks.

"It was not surprising to see that the smell of predators in the water induced larger tadpole tails," Relyea said. "That is a normal, adaptive response. What shocked us was that the Roundup® induced the same changes. Moreover, the combination of predators and Roundup® caused the tail changes to be twice as large."

Because tadpoles alter their body shape to match their environment, having a body shape that does not fit the environment can put the animals at a distinct disadvantage.

According to Relyea, this is the first study to show that a pesticide can induce morphological changes in a vertebrate animal.

Predators cause tadpoles to change shape by altering the stress hormones of tadpoles, says Relyea. The similar shape changes when exposed to Roundup® suggest that Roundup® may interfere with the hormones of tadpoles and potentially many other animals.

"This discovery highlights the fact that pesticides, which are important for crop

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production and human health, can have unintended consequences for species that are not the pesticide's target," Relyea said. "

Herbicides are not designed to affect animals, but we are learning that they can have a wide range of surprising effects by altering how hormones work in the bodies of animals. This is important because amphibians not only serve as a barometer of the ecosystem's health, but also as an indicator of potential dangers to other species in the food chain, including humans."

The research was published today in [Ecological Applications](#).

Relyea is a University of Pittsburgh professor of biological sciences in the Kenneth P. Dietrich School of Arts and Sciences and director of Pitt's [Pymatuning Laboratory of Ecology](#).

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NEW STUDY: Genetically Modified Corn Toxic To Humans

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MAINE—(ENEWSPF)—February 20, 2012. A study published last week in the peer-reviewed **Journal of Applied Toxicology** revealed that genetically modified corn containing the genes for Bt (*Bacillus thuringiensis*) is toxic to humans. The study further revealed that the herbicide known as Roundup is toxic to humans, even at small exposures. The vast majority of the corn grown in the U.S. has been engineered to contain Bt and is sprayed with Roundup during the growing process.

PRESS RELEASE 2/20/2012

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Genetically engineered maize: New indication of health risks

Insecticidal Bt toxins such as those produced in genetically engineered plants can be detrimental to human cells. This is a result of recent research led by researchers at the University of Caen (France). Their experiments showed that toxins produced in, for example, the genetically engineered maize MON810, can significantly impact the viability of human cells.

The effects were observed with relatively high concentrations of the toxins, nevertheless there is cause for concern. According to companies like Monsanto, which produces genetically engineered maize with these toxins, the toxins are supposed to be active only against particular insects and should have no effect on mammals and humans at all.

For the first time, experiments have now shown that they can have an effect on human cells. These kinds of investigations are not a requirement for risk assessment in Europe or in any other region.

Another finding of the researchers concerns a herbicide formulation sold under the brand name Roundup. Massive amounts of this herbicide are sprayed on genetically engineered soybean crops and its residues can be found in food and feed. According to the new publication, even extremely low dosages of Roundup (glyphosate formulations) can damage human cells. These findings are in accordance with several other investigations highlighting unexpected health risks associated with glyphosate preparations.

“We were very much surprised by our findings. Until now, it has been thought almost impossible for Bt proteins to be toxic to human cells. Now further investigations have to be conducted to find out how these toxins impact the cells and if combinatorial effects with other compounds in the food and feed chain have to be taken into account,” says Gilles-Eric Séralini from the University of Caen, who supervised the experiments. “In conclusion, these experiments show that the risks of Bt toxins and of Roundup have been

underestimated.”

Bt toxins and tolerance to herbicides are broadly used in genetically engineered plants. Bt proteins only naturally occur in soil bacteria. By introducing the modified toxin gene into the plants, the structure of the toxins is modified and may

thereby cause selectivity to be changed. The content of the proteins within the plants is highly variable. Many genetically engineered plants contain several Bt toxins at the same time. For example, SmartStax produces six different Bt toxins and therefore has a higher overall content of the proteins. In addition, it was made tolerant to herbicides. So far, there has been no investigation of the combinatorial effects of these toxins and residues from spraying, or their potential risks for human health, which was considered unlikely. The researchers have now shown that interactivity does occur. Under the specific conditions of their experiment, the modified Bt toxin lowered the toxicity of Roundup. Further investigations are necessary to examine other potential combinatorial effects under varying conditions.

“These results are pretty worrying. Risk assessment requirements for genetically engineered plants and pesticides need to be rigidly enforced. In the light of these findings, we think that the commercialization of these plants is not in accordance with EU regulations”, says Christoph Then at Testbiotech. Testbiotech is closely following risk assessment at the European Food Safety Authority EFSA and has repeatedly brought attention to gaps in risk assessment.

The research was supported by GEKKO foundation (Germany). CRIIGEN Association (France) and Testbiotech (Germany) were involved in planning the experiments and the discussion of results. Findings were published after peer review process.

Professor Gilles-Eric Séralini, France: criigen@unicaen.fr, www.criigen.org

Christoph Then, Testbiotech, Germany: info@testbiotech.org, www.testbiotech.org

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Short Communication

Cytotoxicity on human cells of Cry1Ab and Cry1Ac Bt insecticidal toxins alone or with a glyphosate-based herbicide

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ABSTRACT

The study of combined effects of pesticides represents a challenge for toxicology. In the case of the new growing generation of genetically modified (GM) plants with stacked traits, glyphosate-based herbicides (like Roundup) residues are present in the Roundup-tolerant edible plants (especially corns) and mixed with modified *Bt* insecticidal toxins that are produced by the GM plants themselves. The potential side effects of these combined pesticides on human cells are investigated in this work. Here we have tested for the very first time Cry1Ab and Cry1Ac *Bt* toxins (10 ppb to 100 ppm) on the human embryonic kidney cell line 293, as well as their combined actions with Roundup, within 24 h, on three biomarkers of cell death: measurements of mitochondrial succinate dehydrogenase, adenylate kinase release by membrane alterations and caspase 3/7 inductions. Cry1Ab caused cell death from 100 ppm. For Cry1Ac, under such conditions, no effects were detected. The Roundup tested alone from 1 to 20 000 ppm is necrotic and apoptotic from 50 ppm, far below agricultural dilutions (50% lethal

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concentration 57.5 ppm). The only measured significant combined effect was that Cry1Ab and Cry1Ac reduced caspases 3/7 activations induced by Roundup; this could delay the activation of apoptosis. There was the same tendency for the other markers. In these results, we argue that modified *Bt* toxins are not inert on nontarget human cells, and that they can present combined side-effects with other residues of pesticides specific to GM plants. Copyright © 2012 John Wiley & Sons, Ltd.

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ASPCRO Position on Legislation Requiring Schools Pest Management

In the past 20 years since the U.S. Environmental Protection Agency has officially encouraged schools to adopt integrated pest management (IPM), implementation of this practice has been slow to be adopted. Adoption of IPM can be enhanced in states where some form of mandatory school IPM legislation has been passed and those laws are enforced. As an association of structural pest control regulators responsible for enforcing laws that cover pest management, ASPCRO also understands pest management in this nation's schools is primarily the responsibility of school systems across the U.S., and therefore, recognizes the importance and value of partnering with other state agencies and licensed pest control companies responsible for pest control and children's health and safety. For this reason ASPCRO supports states' efforts in developing enforceable mandatory school IPM standards for the performance of interior and exterior pest management.

ASPCRO also recognizes the value, and sometimes necessity, of legislation that holds schools to a higher standard in their pest management activities. Such legislation should recognize the importance of science based pest management practices that manage risks of both pests and the pesticides used to control them. Legislation introduced in the past, especially on the federal level, has not always balanced these two concerns well.

Unfortunately, proposed federal legislation to date has tended to focus on restricting how, when and what pesticides can be used in and around schools. Rather than encouraging schools to embrace and adopt the IPM concept, which includes emphasis on a variety of control tactics that are compatible with human and environmental health, we believe this legislation would result in greater resistance to IPM and restrictions on schools' abilities to manage pests effectively. For this reason, we offer the following recommendations for federal and state legislators considering introduction of mandatory school IPM laws.

- Require IPM for all pest control activities. Schools should include in their official district or administrative policy a requirement that contractors and staff will implement integrated pest management (IPM) practices for all pest control activities. Part of this policy will state the district's commitment to use monitoring and surveillance to demonstrate pest presence prior to pesticide use, establishment of action thresholds for key pests, use of multiple control tactics for control of pests, and the education and involvement of school personnel in the school IPM program.
- Training and certification requirements for anyone applying pesticides. To ensure the use of pesticides according to EPA labels, legislation should require that only state certified personnel be allowed to handle and apply pesticides to school property.
- Use efficacious "least risk" options first, when pesticides are required. Effective IPM relies on a variety of pest control tactics, including pesticides. Good school

IPM legislation should not prohibit the use of registered pesticides in schools. We support the product registration system used by the U.S. Environmental Protection Agency (EPA) to ensure that pesticides, when used according to label directions, pose no unreasonable adverse effects to people or the environment, including schools.

We acknowledge, however, that some pesticides pose inherently lower risks for human exposure and have lower toxicity ratings. Schools should be encouraged to use such lower exposure, lower risk products that have proven efficacy.

- Funding. Any federal or state legislation should include funding for education and compliance assistance. When IPM programs fail, it is due to lack of education and training, lack of follow up, and lack of follow through. In addition, legislation should include funding for improvements to school facilities, the building envelope. Many of the nations aging school buildings require improvements to exclude access to pests. Rapid reductions in pest problems can be realized by simply improving failing structural elements in school facilities.
- Require schools to appoint an IPM coordinator. To ensure responsibility and better understanding of pest control decisions, schools should be required to appoint at least one IPM Coordinator per district, whose job it is to ensure that the school follows its IPM policy and procedures. The IPM Coordinator should have a basic understanding of IPM and the legal requirements for pest control in schools in the state. States should be required to set training and competency standards for IPM Coordinators.
- Posting and notification requirements. Notification of parents, guardians and staff of pesticide applications is an addition to a responsible part of any school IPM program. States are encouraged to develop protocols for the notification of pesticide applications of routine, emergency and broadcast pesticide applications when school is in session. Exemptions of notification should be considered for baits and the use of other non synthetic or exempted pesticides
- Re-entry intervals and treating when children are present. Common sense suggests that pesticide applications should not generally be made when students are present nearby. States should be responsible for developing rules relating to distances between children and pesticide applications, and reasonable waiting periods between pesticide applications and when students may re-enter treated areas.

Acute Pesticide Poisoning Among Agricultural Workers in the United States, 1998–2005

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Background *Approximately 75% of pesticide usage in the United States occurs in agriculture. As such, agricultural workers are at greater risk of pesticide exposure than non-agricultural workers. However, the magnitude, characteristics and trend of acute pesticide poisoning among agricultural workers are unknown.*

Methods *We identified acute pesticide poisoning cases in agricultural workers between the ages of 15 and 64 years that occurred from 1998 to 2005. The California Department of Pesticide Regulation and the SENSOR-Pesticides program provided the cases. Acute occupational pesticide poisoning incidence rates (IR) for those employed in agriculture were calculated, as were incidence rate ratios (IRR) among agricultural workers relative to non-agricultural workers.*

Results *Of the 3,271 cases included in the analysis, 2,334 (71%) were employed as farmworkers. The remaining cases were employed as processing/packing plant workers (12%), farmers (3%), and other miscellaneous agricultural workers (19%). The majority of cases had low severity illness (N = 2,848, 87%), while 402 (12%) were of medium severity and 20 (0.6%) were of high severity. One case was fatal. Rates of illness among various agricultural worker categories were highly variable but all, except farmers, showed risk for agricultural workers greater than risk for non-agricultural workers by an order of magnitude or more. Also, the rate among female agricultural workers was almost twofold higher compared to males.*

Conclusion *The findings from this study suggest that acute pesticide poisoning in the agricultural industry continues to be an important problem. These findings reinforce the need for heightened efforts to better protect farmworkers from pesticide exposure.* Am. J. Ind. Med. 51:883–898, 2008. Published 2008 Wiley-Liss, Inc.†

KEY WORDS: pesticides; surveillance; poisoning; agriculture; farmworkers

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The findings and conclusions in this report are those of the authors and do not necessarily represent the views of the National Institute for Occupational Safety and Health or each author's state agency.

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INTRODUCTION

Pesticides are widely used in agriculture to control insects, microorganisms, fungi, weeds, and other pests. The control of these pests serves to increase crop yield and decrease manual labor [Litchfield, 2005]. In 2000 and 2001, over 5 billion pounds of pesticides were used annually throughout the world. The United States was responsible for 24% of this total usage [Kiely et al., 2004]. Within the US, the agricultural industry accounts for approximately 75% of the annual poundage used.

Farming is an essential component of our economy, but agricultural workers suffer elevated rates of injuries, hearing loss, and respiratory disease [Rust, 1990; Linaker and Smedley, 2002; Tak and Calvert, 2008]. Pesticides are also an important source of injury and illness among farmers and farm workers [Calvert et al., 2004]. Previous work has suggested that the agricultural industry's disproportionately high pesticide use puts farmers and farm workers at greater risk of pesticide exposure than others [Reeves and Schafer, 2003; Calvert et al., 2004]. Farmers and farmworkers may be exposed by mixing, loading and applying pesticides, or while performing duties not involved with pesticide application (e.g., weeding, harvesting, thinning, irrigating, or planting).

Recognizing the need for increased worker protections from pesticide exposures, the U.S. Environmental Protection Agency (EPA) promulgated rules in 1974 known as the Worker Protection Standard for Agricultural Pesticides (WPS; 40 CFR 170) and aimed at reducing pesticide exposures among agricultural workers. However, by 1992, EPA estimated that hired farmworkers alone experienced up to 10,000–20,000 illnesses and injuries from pesticide exposures each year [US EPA, 1992] and concluded that the WPS was inadequate in its requirements and scope of coverage. That year, EPA revised and expanded the WPS rules to include changes in labeling, coverage of more workers and agricultural operations, prohibition of employer retaliation against workers attempting to comply with the standard, and the following requirements: notification of workers about pesticide applications; restriction of re-entry into pesticide-treated areas; and, provision of personal protective equipment (PPE), decontamination supplies, emergency assistance, and pesticide safety training. Detailed information on the magnitude, characteristics and trend of acute pesticide poisoning since the revised WPS went into effect in 1995 are unavailable.

The National Institute for Occupational Safety and Health (NIOSH) developed the Sentinel Event Notification System for Occupational Risks-Pesticides (SENSOR-Pesticides) program [Calvert et al., 2001] to monitor risks from pesticide exposure. Data from this program are available beginning in 1998, when standardized definitions for cases and data elements were adopted [Calvert et al., 2001]. The California Department of Pesticide Regulation (CDPR) has a similar surveillance program that has been tracking pesticide-related

illnesses for more than 30 years [Calvert et al., 2001]. To assess the magnitude, characteristics and trend of acute pesticide poisoning among agricultural workers in the United States since the revised WPS went into effect in 1995, an analysis of data obtained from these surveillance systems was performed.

MATERIALS AND METHODS

Data were obtained on individuals age 15 through 64 who developed an acute pesticide-related illness or injury and who were employed in the agricultural industry when the occupational pesticide exposure occurred. Census 1990 industry codes (1990 CIC) and Census 2002 industry codes (2002 CIC) were used to identify cases employed in agriculture [US Bureau of the Census, 1992; US Census Bureau, 2005]. The agricultural industry was defined as: agricultural production, excluding livestock (1990 CIC = 010; 2002 CIC = 0170); agricultural production, including livestock (1990 CIC = 011; 2002 CIC = 0180); and agricultural services (1990 CIC = 030; 2002 CIC = 0290). All agricultural industry cases also had their occupation coded using Census 1990 occupation codes (1990 COC) and Census 2002 occupation codes (2002 COC) [US Bureau of the Census, 1992; US Census Bureau, 2005]. Agricultural occupations included: farmworkers (1990 COC = 477, 479, 484; 2002 COC = 6050, 6120, 8710, 8960); farmers (1990 COC = 473–476; 2002 COC = 0200, 0210); processing/packing plant workers (1990 COC = 488, 699; 2002 COC = 6040, 7830, 7850, 8640, 8720, 8800, 8860, 9640); and, other miscellaneous agricultural workers (workers employed in agriculture but whose 1990 COC and 2002 COC did not match any of those specified for the other three agricultural occupations). A pesticide handler was defined as an individual who mixed, loaded, transported and/or applied pesticides, or an individual who repaired or maintained pesticide application equipment at the time of pesticide exposure (insufficient information was available to determine pesticide handler status for 68 individuals). This analysis excluded illnesses associated with non-occupational exposures and illnesses associated with intentional (e.g., suicidal, malicious intent) exposures.

Cases under 15 years of age and those 65 years and older were omitted from analysis. The age range was chosen a priori, and is considered to include the vast bulk of workers who are gainfully employed. A total of 66 cases age 65 and older were identified but not included in this analysis (this represents a rate of 13/100,000 agricultural workers age 65 and older). Furthermore, Current Population Survey (CPS) data, the source of our denominator data, are unavailable on workers less than 15 years of age.

Data Sources

Data for this analysis were obtained from CDPR and the SENSOR-Pesticides program. State health departments in

ten states participated in the SENSOR-Pesticides program and contributed data. These ten state health departments were the: Arizona Department of Health, California Department of Public Health (CDPH), Florida Department of Health, Louisiana Department of Health and Hospitals, Michigan Department of Community Health, New Mexico Department of Health (through an agreement with the University of New Mexico), New York State Department of Health, Oregon Department of Human Services, Texas Department of State Health Services, and the Washington State Department of Health. The time frame for data availability varied according to state agency. The years of data availability are provided in Table I. Each of these agencies maintains its own passive population-based surveillance system for acute pesticide-related illness or injury with occasional outreach to potential reporters to stimulate reporting (e.g., contacting poison control centers to encourage them to report or reviewing physician reports submitted to workers' compensation insurance carriers to identify eligible cases) [Calvert et al., 2001, 2004]. Each agency obtains case reports from many different sources. All require physician reporting of pesticide-related illness cases. Other sources of case reports vary by state and include poison control centers, state agencies with jurisdiction over pesticide use (e.g., departments of agriculture), and workers' compensation claims. Because each state removes any personal identifiers from the data prior to submission to the Centers for Disease Control and Prevention this study was exempt from consideration by the federal Human Subjects Review Board.

Once a case report is received, the state agency determines whether the subject was symptomatic and whether the involved chemical is a pesticide. If so, attempts are made to interview the poisoned subject or their proxy to obtain details on the poisoning event, and any medical records are requested. Besides identifying, classifying, and tabulating pesticide poisoning cases, the states periodically perform in-depth investigations of pesticide-related events, and develop interventions aimed at particular industries or pesticide hazards.

Cases obtained from CDPR were cross-referenced with cases from the CDPH based on age, gender, date of exposure, and pesticide name. Matching cases were assumed to be the same individual and were counted only once.

Information Available on Each Case

Data collected for each case by the SENSOR-Pesticides and CDPR surveillance systems include case demographics, signs and symptoms of illness or injury, illness severity, EPA toxicity category, identity of implicated pesticides and the target (e.g., crop) of their application (if any), information on factors that may have contributed to the pesticide exposure that precipitated illness, and the source of the case report.

EPA evaluates the toxicity of and assigns a toxicity category to each pesticide product. The categories range

from I to IV, with I representing the most toxic and IV the least toxic substances [US EPA, 1975]. The toxicity category for each case was obtained by the relevant state agency conducting pesticide poisoning surveillance. When toxicity category data was not given, the category was determined by NIOSH based on standardized criteria from a dataset provided by EPA. Cases exposed to more than one pesticide product were assigned the toxicity category representing the pesticide product with the greatest toxicity.

Case Definition

A standardized case definition is used by all participating SENSOR-pesticides states. Cases of acute pesticide poisoning are included in the analyses if they were classified as definite, probable, possible or suspicious. A classification category is assigned to a case based on three factors: (1) the strength of evidence that a pesticide exposure occurred; (2) whether adverse health effects were observed by a healthcare professional versus being self-reported; and (3) the presence of sufficient evidence that the known toxicology of the agent was consistent with the observed health effects. Cases exposed to pesticides for which there is limited toxicological data were classified as suspicious [CDC, 2001a]. CDPR uses a comparable case definition [CDPR, 2006]. In this article, acute pesticide poisoning and acute pesticide-related illness and injury are used interchangeably.

Illness severity was assigned to all cases using standardized criteria which were based on signs and symptoms, medical care received, and lost time from work [CDC, 2001b]. *Low severity illness/injury* consist of illnesses and injuries that generally resolve without treatment and where minimal time (<3 days) is lost from work. Such cases typically manifest as eye, skin and/or upper respiratory irritation. *Moderate severity illness/injury* consists of non-life-threatening health effects that are generally systemic and require medical treatment. No residual disability is detected, and time lost from work is less than 6 days. *High severity illness/injury* consists of life threatening health effects that usually require hospitalization, involve substantial time lost from work (>5 days), and may result in permanent impairment or disability. *Death* pertains to fatalities resulting from exposure to one or more pesticides.

Data Analysis

SAS v. 9.1 was used for data management and analysis [SAS Institute Inc, 2003]. Chi square statistical analyses were performed on categorical data. Incidence rates (IR) for acute occupational pesticide poisoning were calculated for those employed in agriculture. Rates were calculated for occupational categories within agriculture, for each year studied, by age group, and for three geographic regions in the US. The numerator represents the number of relevant cases

TABLE I. Data on Demographics, Pesticide Toxicity, Pesticide Handler, Pesticide Functional Class, and Application Target for 3,271 Acute Pesticide Poisoning Cases in the Agricultural Industry by Severity Category, 1998–2005

	Fatal, N	High Severity, N	Medium Severity, N	Low Severity, N	Total, N (%) ^a
Total	1	20	402	2,848	3,271
Age					
15–17	0	0	5	19	24 (1)
18–24	0	1	82	521	604 (18)
25–34	0	2	109	786	897 (27)
35–44	0	7	103	630	740 (23)
45–54	0	3	59	358	420 (13)
55–64	1	5	28	138	172 (5)
Unknown	0	2	16	396	414 (13)
State where illness identified (years of data availability)					
Arizona (1998–1999)	0	0	4	17	21 (1)
California (1998–2005)	1	10	274	2,235	2,520 (77)
Florida (1998–2005)	0	0	23	109	132 (4)
Louisiana (2001–2005)	0	4	14	27	45 (1)
Michigan (2001–2005)	0	1	7	14	22 (1)
New Mexico (2005 only)	0	0	2	10	12 (1)
New York (1998–2005)	0	0	6	7	13 (1)
Oregon (1998–2005)	0	0	6	37	43 (1)
Texas (1998–2005)	0	3	40	146	189 (6)
Washington (2001–2005)	0	2	26	246	274 (8)
Gender					
Female	0	6	114	934	1,054 (32)
Male	1	14	288	1,886	2,189 (67)
Unknown	0	0	0	28	28 (1)
Year exposed					
1998	0	2	64	358	424 (13)
1999	1	1	85	337	424 (13)
2000	0	4	64	315	383 (12)
2001	0	0	30	236	266 (8)
2002	0	0	26	576	602 (18)
2003	0	0	43	279	322 (10)
2004	0	11	35	396	442 (14)
2005	0	2	55	351	408 (12)
Toxicity category ^b					
I	1	11	232	1,418	1,662 (51)
II	0	1	68	599	668 (20)
III and IV	0	6	82	792	880 (27)
Unknown	0	2	20	39	61 (2)
Pesticide handler					
Yes ^c	1	10	190	867	1,068 (33)
No	0	10	200	1,925	2,135 (65)
Unknown	0	0	12	56	68 (2)
Pesticide functional class ^d					
Insecticides—all	1	10	210	1,540	1,761 (54)
Insecticides only	0	7	115	747	869 (27)
Insecticides combined	1	3	95	793	892 (27)
Fungicides—all	1	4	90	734	829 (25)
Fungicides only	0	2	28	147	177 (5)
Fungicides combined	1	2	62	587	652 (20)

TABLE I. (Continued)

	Fatal, N	High Severity, N	Medium Severity, N	Low Severity, N	Total, N (%) ^a
Disinfectants—all	0	2	56	389	447 (14)
Disinfectants only	0	2	48	238	288 (9)
Disinfectants combined	0	0	8	151	159 (5)
Herbicides—all	0	1	56	400	457 (14)
Herbicides only	0	1	42	318	361 (11)
Herbicides combined	0	0	14	82	96 (3)
Fumigants—all	0	4	44	416	464 (14)
Fumigants only	0	4	44	416	464 (14)
Fumigants combined	0	0	0	0	0 (0)
Other	0	1	18	130	149 (5)
Application target					
Fruit crops	0	3	112	1,047	1,162 (36)
Vegetable crops	0	2	45	411	458 (14)
Soil	0	2	20	316	338 (10)
Grains, grasses and fiber crops	0	1	58	201	260 (8)
Landscape/ornamental	0	1	18	159	178 (5)
Undesired plant	0	0	6	74	80 (2)
Beverage crops	0	0	7	35	42 (1)
Crops that cross categories	0	0	6	32	38 (1)
Building structure/surface/space	0	1	8	35	44 (1)
Oil crops	0	0	5	15	20 (1)
Miscellaneous field crops	0	0	8	11	19 (1)
Veterinary (livestock or domestic)	0	0	4	13	17 (1)
Other	1	2	18	182	203 (6)
Not applicable	0	4	55	161	220 (7)
Unknown	0	4	32	156	192 (6)

^aPercentages may not sum to 100 due to rounding.

^bAcute pesticide toxicity category as defined by the U.S. EPA.

^cA pesticide handler was defined as an individual who mixed, loaded, transported and/or applied pesticides, or an individual who repaired or maintained pesticide application equipment at the time of pesticide exposure.

^dCases may be exposed to more than one functional class. The rows labeled with "combined" pertain to cases exposed to more than one pesticide active ingredient, some of which belong to the pesticide functional class specified in the row label and others belonging to other pesticide functional classes.

captured by CDPR and SENSOR-Pesticides from 1998 to 2005. Denominator data, including employment counts and the hours worked estimate, were obtained from the CPS [BLS, 2007]. The hours worked data were used to derive full-time equivalent (FTEs) estimates, with one FTE equivalent to 2,000 hr worked. Denominator data correspond to the states and time periods of data availability (Table I). Although rates were calculated with the two denominator estimates (employment counts and FTE estimates), the rates calculated with FTEs as the denominator are given prominence as they have previously been shown to be conceptually preferable over the use of raw employment counts [Ruser, 1998]. The comparison group consisted of all workers not employed in agriculture. IR for workers employed in non-agricultural industries were similarly calculated, with the numerator and denominator data obtained from the same agencies (SENSOR/CDPR and CPS, respectively) that provided the data on agricultural workers. Finally, incidence rate ratios (IRR)

were calculated to determine the risk of acute pesticide poisoning while working in agriculture. This ratio was calculated by dividing the IR among agricultural workers with that among non-agricultural workers. A ratio greater than one suggests an increased risk in farmers or farmworkers, while a ratio less than one suggests a decreased risk. Confidence intervals (95% CIs) were calculated for each rate ratio as described by Rothman [1986].

RESULTS

From 1998 to 2005, 3,271 case reports met inclusion criteria (Table I). Of these, 1,078 (33%) were identified by the SENSOR-Pesticides program and 2,193 (67%) originated from CDPR (527 cases were identified by both SENSOR and CDPR and were included in the CDPR total only). There were 1,942 separate pesticide exposure events, 1,762 of which (91%) involved only one ill agricultural worker. Of the

180 (9%) multi-victim events, the median number of ill agricultural workers was 3 (range 2–123). The number of pesticide exposure events decreased over the time period studied but the average number of cases per event increased (in 1998 there were 308 events with an average of 1.4 cases per event whereas in 2005 there were 209 events with an average of 2.0 cases per event).

Description of the Three Largest Events

More than three quarters of the cases ($N = 2,520$, 77%) occurred in California. Among these cases, we found a small number of events that exposed large numbers of agricultural workers. In two separate 2002 incidents, irritant vapors drifted from soil treatments with metam-sodium and caused low severity illness in 123 vineyard workers and in 72 workers at a carrot processing facility, respectively [see

O'Malley et al., 2005 for detailed information on the event involving 72 workers]. The second largest incident occurred in 2004, when 121 peach harvesters became ill after exposure to drift from an application of methamidophos and mancozeb to a nearby potato field. Most of these workers experienced low severity illness ($N = 111$, 92%), and the other 10 workers (8%) experienced moderate severity illness.

Incidence Rates

Tables II and III and Figure 1 summarize IRs for agricultural workers and non-agricultural workers from 1998 to 2005. Overall, the average annual IR among agricultural workers was 53.6/100,000 FTEs and 1.38/100,000 FTEs among all non-agricultural workers combined (IRR = 38.9 95% CI 37.2, 40.6). Agricultural workers' annual rates fluctuated between 33.8/100,000 FTEs (2001) and 79.9/

TABLE II. Incidence Rates by Industry, Year of Exposure, Age Group and US Region for 3,271 Acute Pesticide Poisoning Cases, 1998–2005

	Agricultural workers			Non-agricultural workers			
	Count	FTE estimate ^a	Incidence rate ^b	Count	FTE estimate ^a	Incidence rate ^c	Incidence rate ratio ^d
Year of exposure							
1998	424	790,837	53.6	762	40,792,468	1.9	28.7
1999	424	781,985	54.2	656	42,040,152	1.6	34.7
2000	383	781,654	49.0	577	41,041,774	1.4	34.9
2001	266	787,481	33.8	552	49,456,474	1.1	30.3
2002	602	753,595	79.9	598	49,110,280	1.2	65.6
2003	322	756,610	42.6	694	50,151,930	1.4	30.8
2004	442	735,270	60.1	716	50,989,934	1.4	42.8
2005	408	710,851	57.4	638	53,000,554	1.2	47.7
Age group (years)							
15–17	24	99,364	24.2	69	3,140,858	2.2	11.0
18–24	604	792,852	76.2	851	43,536,103	2.0	39.0
25–34	897	1,392,263	64.4	1,246	92,090,687	1.4	47.6
35–44	740	1,601,894	46.2	1,303	106,763,138	1.2	37.9
45–54	420	1,276,042	32.9	968	89,437,936	1.1	30.4
55–64	172	935,868	18.4	356	41,614,845	0.9	21.5
Unknown	414	—	—	400	—	—	—
US region							
West ^e	2,858	3,168,485	90.2	3,556	149,655,538	2.4	38.0
South ^f	366	2,258,774	16.2	1,225	141,962,800	0.9	18.8
East ^g	45	671,024	7.0	412	84,965,229	0.5	13.8
Total	3,271	6,098,283	53.6	5,193	376,583,567	1.4	38.9

^aFTE, full-time equivalent.

^bIncidence rate per 100,000 FTEs. Includes agricultural workers in Arizona, California, Florida, Louisiana, Michigan, New Mexico, New York, Oregon, Texas, and Washington.

^cIncidence rate per 100,000 FTEs. Includes non-agricultural workers in Arizona, California, Florida, Louisiana, Michigan, New Mexico, New York, Oregon, Texas, and Washington.

^dCompares the rate of acute pesticide poisoning among agricultural workers for a given year with non-agricultural workers. Cases are identified by participating SENSOR-Pesticides states and CDPR. All IRRs were significantly elevated ($P < 0.0001$).

^eArizona, California, New Mexico, Oregon, and Washington.

^fFlorida, Louisiana, and Texas.

^gMichigan, New York.

TABLE III. Incidence Rates by Occupation for 3,271 Acute Pesticide Poisoning Cases in the Agricultural Industry, 1998–2005

Occupation	Number	Percent	FTE estimate ^a	Incidence rate ^b
Farmworker—all	2,334	71	3,119,402	74.8
Farmworker—male	1,620	69	2,625,146	61.7
Farmworker—female	701	30	494,256	141.8
Farmer	89	3	1,852,030	4.8
Farmer—male	80	90	1,510,632	5.3
Farmer—female	9	10	341,398	2.6
Processing/packing plant worker	394	12	108,646	362.6
Processing/packing plant worker—male	108	27	21,094	512.0
Processing/packing plant worker—female	279	71	87,552	318.7
All other agricultural occupations	454	14	1,018,205	44.6
All other agricultural occupations—male	381	84	674,521	56.5
All other agricultural occupations—female	65	14	343,684	18.9
Total ^c	3,271	100	6,098,283	53.6
Total—male	2,189	67	4,831,393	45.3
Total—female	1,054	32	1,266,890	83.2

^aFTE, full-time equivalent.

^bIncidence rates per 100,000 FTEs. Includes agricultural workers in Arizona, California, Florida, Louisiana, Michigan, New Mexico, New York, Oregon, Texas, and Washington.

^cSex was unknown for 28 cases (farmworkers = 13, processing/packing plant worker = 7, all other agricultural occupations = 8).

100,000 FTEs (2002), driven primarily by the occurrence of large California events (Fig. 1). Limiting the analysis to the five states (California, Florida, New York, Oregon, Texas) that provided data for all 8 years had little effect on the plot in the Figure 1. By US geographic region, the IR for agricultural

workers was highest in the West. The rates in the West were largely driven by California and Washington State, where the rates were 100.8 and 113.0/100,000 FTEs, respectively.

Information on age was available for 87% of the cases (N = 2,857; Table I). The median age was 33 years (range

**FIGURE 1.** Incidence rates for acute pesticide poisoning cases among agricultural and non-agricultural workers by year, age 15–64 years, 1998–2005.

15–64), and over half of the cases were between the ages of 25 and 44 years ($N = 1,637$, 57%). The IR was highest among agricultural workers age 18–24 years (76.2/100,000 FTEs; Table II).

Because agricultural workers employed in the states and time periods covered in this study worked 2,173 hr per year on average, using FTEs in the denominator produced rates that were approximately 7% lower compared to when employment counts were used in the denominator. In contrast, because non-agricultural workers worked 1,935 hr per year on average, using FTEs in the denominator produced rates that were approximately 4% higher compared to when employment counts were used in the denominator. The overall average annual IR among agricultural workers using employment counts in the denominator was 57.6/100,000 workers, and was 1.33/100,000 FTEs among all non-agricultural workers combined (IRR = 43.2, 95% CI 41.4, 45.1).

Occupations of the Affected Agricultural Workers

Most of the 3,271 affected agricultural workers were employed as farmworkers ($N = 2,334$, 71%; Table III). The 394 affected processing/packing plant workers (12%) represented a disproportionately large share of people so employed, while farmers ($N = 89$, 3%) seemed less at risk than hired agricultural workers. Most of the “other miscellaneous agricultural workers” were employed as pest control operators ($N = 255$, 56%). Because CPS data for pest control operators were too sparse (e.g., in 1999 and 2001 no CPS data were available for this occupation), IRs were not calculated for this occupation.

Gender was reported for all but 28 (1%) of the cases. Males predominated in each occupational category except processing/packing plant workers. Paradoxically, IRs were higher among female than male farmers and farmworkers, but higher among male than female processing/packing plant workers. Females were less likely than males to be pesticide handlers (females = 8%, males = 45%, $P < 0.001$). Information on race and ethnicity was available for 727 cases (22%). A total of 502 (69%) were Hispanic, 187 (26%) were non-Hispanic white, 12 (2%) were black, and the remaining 26 (4%) recorded various other races.

Severity and Description of Fatal Case

A vast majority of the illnesses were of low severity (2,848 cases, 87%), while 402 (12%) were of medium severity and 20 (0.6%) were high severity (Table I). One case was fatal. The fatal case occurred in 1999 and involved a 59-year-old Hispanic male who was employed as an irrigator and farmworker supervisor. He was found dead in an orange grove in California, with packages of hotdogs and packets of

methomyl near his body. This led investigators to suspect he had violated regulations by opening water-soluble methomyl packets and using the potent carbamate insecticide to contaminate hot dogs for use as bait to kill coyotes. His autopsy found a small amount of methomyl in his gastric contents but none in his blood. His blood and bile also contained a relatively large concentration of benzothiazole, an industrial chemical and a metabolite of cyprodinil (a toxicity category III fungicide). The large concentration of benzothiazole was suggestive of chronic exposure, or heavy acute exposure at least 24 hr earlier. The medical examiner concluded that the cause of death was likely due to an interaction between the methomyl and cyprodinil. The source of the exposure to benzothiazole or cyprodinil was not known. Whether the exposures were accidental or intentional (i.e., suicidal) could not be distinguished.

Signs and Symptoms

Table IV lists the signs and symptoms most often reported in this cohort. It also provides information on the health effects among those exposed to the four pesticide chemical classes most commonly involved in illness.

Pesticides Responsible for Illness

Information on the pesticides responsible for illness is provided in Tables I, IV and V. Insecticides (alone or in combination with other pesticides) were implicated in more than half of the illnesses ($N = 1,761$, 54%). Cholinesterase inhibitors (organophosphates and *N*-methyl carbamates) were prominent among the insecticides ($N = 892$, 51%), particularly chlorpyrifos ($N = 190$), methamidophos ($N = 130$), dimethoate ($N = 84$), malathion ($N = 78$), and diazinon ($N = 70$). Over half of the cases ($N = 1,662$, 51%) were exposed to toxicity category I pesticides, the most toxic category as defined by EPA (Table I). We found little variation in illness severity by pesticide category.

Activity at Time of Exposure

Information on activity at time of pesticide exposure was available for 3,203 (98%) of the affected workers. Of these, 33% ($N = 1,068$) were pesticide handlers and 67% ($N = 2,135$) were doing routine work not involved with a pesticide application. Most of the handlers (71%) were exposed while making applications. Among the 2,135 doing routine work, half were exposed to off-target drift of pesticide from a nearby application ($N = 1,068$), and 35% ($N = 744$) had contact with pesticide residues present on a treated surface (e.g., plant material or treated animal).

Table I lists the targets to which pesticides were applied in incidents that resulted in human illness. Among the

TABLE IV. Illness Characteristics by Pesticide Chemical Class for 3,271 Acute Pesticide Poisoning Cases in the Agricultural Industry, 1998–2005

Signs and symptoms	Pesticide chemical class ^{a,b}				
	All ^a , N = 3,271 (%)	Cholinesterase inhibitors, N = 892 (%)	Pyrethroids, N = 182 (%)	Inorganics, N = 567 (%)	Dithiocarbamates, N = 512 (%)
Nervous/sensory	1,743 (53)	672 (75)	120 (66)	241 (43)	237 (46)
Headache	1,268 (39)	499 (56)	94 (52)	164 (29)	185 (36)
Dizziness	708 (22)	297 (33)	39 (21)	88 (16)	85 (17)
Muscle weakness	243 (7)	126 (14)	10 (5)	29 (5)	23 (4)
Blurred vision	204 (6)	86 (10)	8 (4)	34 (6)	29 (6)
Paresthesias	198 (6)	76 (9)	15 (8)	31 (5)	25 (5)
Muscle pain	98 (3)	44 (5)	7 (4)	7 (1)	15 (3)
Diaphoresis	94 (3)	59 (7)	6 (3)	8 (1)	7 (1)
Salivation	63 (2)	48 (5)	3 (2)	2 (<1)	19 (4)
Fasciculation	47 (1)	32 (4)	3 (2)	3 (1)	3 (1)
Confusion	46 (1)	19 (2)	1 (1)	3 (1)	2 (<1)
Gastrointestinal	1,300 (40)	510 (57)	91 (50)	174 (31)	188 (37)
Nausea	1,063 (33)	438 (49)	74 (41)	131 (23)	152 (30)
Vomiting	582 (18)	261 (29)	39 (21)	73 (13)	91 (18)
Abdominal pain/cramping	371 (11)	161 (18)	15 (8)	34 (6)	75 (15)
Diarrhea	148 (5)	80 (9)	8 (4)	8 (1)	17 (3)
Ocular	1,300 (40)	272 (30)	54 (30)	243 (43)	297 (58)
Irritation/pain/inflammation	1,112 (34)	208 (23)	48 (26)	222 (39)	262 (51)
Lacrimation	443 (14)	92 (10)	14 (8)	51 (9)	166 (32)
Conjunctivitis	80 (2)	8 (1)	5 (3)	23 (4)	1 (<1)
Dermatologic	1,077 (33)	235 (26)	57 (31)	191 (34)	96 (19)
Pruritis	580 (18)	106 (12)	26 (14)	125 (22)	50 (10)
Rash	571 (17)	98 (11)	17 (9)	126 (22)	64 (13)
Erythema	349 (11)	52 (6)	14 (8)	76 (13)	27 (5)
Irritation/pain	321 (10)	81 (9)	34 (19)	48 (8)	23 (4)
Respiratory	1,074 (33)	329 (37)	56 (31)	232 (41)	152 (30)
Upper respiratory pain/irritation	645 (20)	183 (21)	35 (19)	142 (25)	103 (20)
Dyspnea	408 (12)	115 (13)	19 (10)	91 (16)	60 (12)
Cough	278 (9)	67 (8)	5 (3)	75 (13)	32 (6)
Cardiovascular	211 (6)	77 (9)	7 (4)	43 (8)	31 (6)
Chest pain	131 (4)	45 (5)	4 (2)	32 (6)	18 (4)
Tachycardia	33 (1)	17 (2)	1 (1)	2 (<1)	3 (1)

^aMore than one sign/symptom may be reported by a case, and therefore the sum of the specific clinical effects may not equal the total number of system effects.

^bCases may be exposed to more than one chemical class. Columns include cases exposed to the labeled chemical class only as well as those exposed to mixtures containing that and other chemical classes.

fruit crops, the most common application targets were small fruits (e.g., grapes; N = 529, 46%), tree nuts (N = 181, 16%), citrus fruits (N = 175, 15%), and pome fruits (e.g., apples; N = 151, 13%). Among the most common vegetable crop targets were root and tuber vegetables (e.g., onions and potatoes; N = 185, 40%), leafy vegetables (N = 180, 39%) and fruiting vegetables (e.g., eggplant, tomatoes, and peppers; N = 48, 10%). Among grain, grass and fiber crops, the most common pesticide application targets were cotton (N = 140, 54%), and cereal grains (N = 61, 23%).

Factors That Contributed to Pesticide Exposure

We identified factors that contributed to pesticide exposure in 1,926 (59%) of the cases (Table VI). The most common factors identified were off-target drift (N = 1,216, 63%), early reentry into a recently treated area (N = 336, 17%), and use in conflict with the label (N = 319, 17%). In 992 (30%) cases, no obvious contributory factors could be identified (e.g., restricted entry interval was observed but worker still became ill; wore all required PPE but still

TABLE V. Fifteen Most Common Active Ingredients for 3,271 Acute Pesticide Poisoning Cases in the Agricultural Industry by Severity Category, 1998–2005

Active ingredient	Functional class (chemical class)	High severity/			N (%) ^a
		fatal	Moderate severity	Low severity	
Sulfur	Insecticide and fungicide (inorganic)	2	45	421	468 (14)
Metam-sodium	Fumigant (dithiocarbamate)	1	5	279	285 (9)
Glyphosate	Herbicide (phosphonate)	3	25	223	251 (8)
Mancozeb	Fungicide (dithiocarbamate)	1	17	184	202 (6)
Chlorpyrifos	Insecticide (organophosphate/cholinesterase inhibitor)	0	33	157	190 (6)
Sodium hypochlorite	Disinfectant (halogen)	2	35	149	186 (6)
Methamidophos	Insecticide (organophosphate/cholinesterase inhibitor)	1	10	119	130 (4)
Abamectin	Insecticide (microbial)	0	18	108	126 (4)
Imidacloprid	Insecticide (neonicotinoid)	4	5	104	113 (3)
Methomyl	Insecticide (<i>N</i> -methyl carbamate/cholinesterase inhibitor)	1	7	101	109 (3)
Myclobutanil	Fungicide (triazole)	1	11	97	109 (3)
Propargite	Insecticide (sulfite ester, inhibits oxidative phosphorylation)	0	21	77	98 (3)
Spinosad	Insecticide (derived from <i>Saccharopolyspora spinosa</i>)	1	10	85	96 (3)
Methyl bromide	Fumigant (halocarbon)	2	29	60	91 (3)
Dimethoate	Insecticide (organophosphate/cholinesterase inhibitor)	4	6	74	84 (3)

^aPercentages do not sum to 100, as not all cases are included in this table.

TABLE VI. Factors That Contributed to Pesticide Exposure and/or Illness for 3,271 Acute Pesticide Poisoning Cases in the Agricultural Industry by Severity Category, 1998–2005

Exposure/illness factor ^a	Medium or higher severity, N (%)	Low severity, N	Total, N (%)
All factors combined	219 (11)	1,707	1,926 (59)
Drift	118 (10)	1,098	1,216 (37)
Early reentry	41 (12)	295	336 (10)
Use in conflict with label	40 (13)	279	319 (10)
Failure to use required equipment	19 (12)	139	158 (5)
Oral notification of pesticide application not provided	9 (6)	143	152 (5)
PPE not worn	19 (16)	98	117 (4)
Training not provided or inadequate (excludes applicators)	30 (29) ^c	75	105 (3)
Hazard communication or other OSHA violation	9 (9)	86	95 (3)
Transport for care not provided	5 (6)	84	89 (3)
Application site not posted/notification posters incorrect	3(4)	81	84 (3)
Decontamination facilities inadequate	16 (26) ^c	46	62 (2)
Unsafe equipment/failure	8 (14)	49	57 (2)
Inadequate record keeping	7 (20)	28	35 (1)
Worker not told of health effects caused by pesticides	0 (0)	32	32 (1)
Person in treated area during application	2 (10)	19	21 (1)
Unspecified worker protection standard violation	3 (15)	17	20 (1)
PPE in poor repair	3 (23)	10	13 (<1)
FIFRA-other and unspecified ^b	6 (27) ^c	16	22 (1)
None identified	126 (13)	866	992 (30)
Unknown	78 (22)	275	353 (11)

^aOne factor was identified for 1,279 cases. Two or more factors were identified for 647 cases.

^bIncludes situations when a licensed applicator was not on site (N = 1) and when an applicator was not properly trained or supervised (N = 9).

^cThe proportion with medium or higher severity among cases with the factor of interest was significantly greater than the proportion with medium or higher severity in all other cases ($P < 0.05$). Those with insufficient information to identify factors (i.e., unknown category) were excluded from this analysis.

became ill; all pesticide label requirements appeared to have been followed). Compared to cases where no obvious contributory factors could be identified, identification of a contributory factor was not found to be significantly associated with severity of illness ($P = 0.33$). For 353 (11%) cases, insufficient information was available to identify factors that may have contributed to the pesticide exposure.

Among the 2,367 cases with personal protection equipment (PPE) usage information, 1,157 (49%) wore PPE (Table VII). Women were far less likely to wear PPE (females = 27%, males = 40%, $P < 0.01$). Pesticide handlers were more likely to use PPE (65% overall, 66% among men and 51% among women) compared to non-handlers (21% overall, 18% among men and 26% among women). Those exposed to toxicity category II pesticides were more likely to wear PPE (61%) than those exposed to toxicity category I or III/IV pesticides (53% and 54%, respectively). Table VII also provides information on the health effects experienced by those who used each type of protective equipment. Compared to those who used no protective equipment or had unknown information on its use, those who used protective equipment were less likely to have health effects involving the nervous, gastrointestinal and respiratory systems, but were more likely to have ocular and dermatologic health effects.

Report Source

Case reports were received from many different sources. The three leading sources of case reports were workers' compensation ($N = 1,109$, 34%), other government agencies (e.g., county health departments and the state department of agriculture; $N = 901$, 28%), and poison control centers ($N = 407$, 12%). A variety of sources accounted for the

remaining cases including health care professionals, employers, worker representatives (union, legal services), and self-report. The specific number of cases reported by these other sources is unavailable. Females were more likely to have been reported by one of these other sources (females = 41%, males = 23%, $P < 0.01$), and less likely to be identified by workers' compensation (females = 25%, males = 39%, $P < 0.01$) or poison control centers (females = 8%, males = 15%, $P < 0.01$). Females and males were equally likely to have been reported by other government agencies (28%).

DISCUSSION

It is important to conduct surveillance of acute occupational pesticide poisoning to determine whether policies, practices and regulations are effective in preventing hazardous pesticide exposures. National estimates of hospitalized pesticide poisonings in the 1970s and early 1980s, including agricultural workers, are available [Keefe et al., 1985, 1990]; but to the best of our knowledge this is the first detailed multi-state assessment of both hospitalized and non-hospitalized acute pesticide poisoning among agricultural workers. Our findings indicate that despite strengthening of the WPS in 1995, agricultural workers continue to have an elevated risk for acute pesticide poisoning. The pesticide poisoning incidence among US agricultural workers was found to be 39 times higher than the IR found in all other industries combined.

Improvement Compared to the 1980s

Although there was not a clear trend in the rates of poisoning during the time period that we studied, there is evidence to suggest that the counts of pesticide poisoning

TABLE VII. Illness Characteristics by Type of Protective Equipment Used 1998–2005

Type of protective equipment	Number that used protective equipment	Of workers who used protective equipment, the number (%) who had signs/symptoms involving these organs/systems				
		Nervous/sensory	Gastro-intestinal	Ocular	Respiratory	Dermatologic
Any PPE	1,157	557 (48)	404 (35)	472 (41)	301 (28)	437 (38)
Air-purifying respirator	261	121 (46)	91 (35)	92 (35)	49 (19)	94 (36)
Dust mask	40	16 (40)	12 (30)	19 (48)	18 (45)	15 (38)
Chemical resistant gloves	700	306 (44)	208 (30)	293 (42)	150 (21)	254 (36)
Chemical resistant clothing	542	230 (42)	177 (33)	214 (39)	105 (19)	213 (39)
Chemical resistant boots	367	170 (46)	110 (30)	152 (41)	63 (17)	127 (35)
Cloth/leather gloves	298	192 (64)	138 (46)	92 (31)	114 (38)	140 (47)
Goggles/eye protection	488	193 (40)	126 (26)	198 (41)	80 (16)	196 (40)
Engineering controls ^a	100	48 (48)	34 (34)	23 (23)	20 (20)	41 (41)
No or unknown PPE use ^b	0	1,186 (56)	896 (42)	828 (39)	773 (37)	640 (30)

^aEngineering controls included such things as enclosed tractor cabs or closed mixing/loading systems.

^bA total of 2,114 individuals used no protective equipment or had unknown information on use of protective equipment.

cases among agricultural workers have decreased since the 1980s. Mehler et al. [1992] reported an annual average of 723 cases of pesticide illness or injury in California arising from agricultural establishments from 1982 to 1990. In contrast, the California surveillance programs reported an average of 315 cases per year from 1998 to 2005. The numbers are not entirely comparable. Mehler characterized poisoning cases as being agricultural if the poisoned subject was a worker and the exposure arose from an agricultural establishment. Her definition included non-agricultural workers (e.g., truck drivers, construction workers, school employees), while we included only workers employed in agriculture.

True Incidence Remains Uncertain: Comparison With Data From the Bureau of Labor Statistics, the National Agricultural Workers Survey, and the Agricultural Health Study

In order to put our findings in perspective, acute pesticide poisoning annually accounts for a small percentage of the total occupational illnesses experienced by agricultural workers. The 2005 Bureau of Labor Statistics (BLS) survey of illnesses and injuries (SOII), which excludes agricultural production establishments with 10 employees or fewer, reports an annual injury rate of 5.7% and an annual illness rate of 0.4% (3% of which involve poisonings) among workers in farming, forestry and fishing [Myers, 2007]. Most of these illnesses consisted of dermatitis, respiratory conditions, and other conditions not specified (e.g., musculoskeletal conditions arising from cumulative trauma). We report a rate of pesticide poisoning five times higher than the SOII rate for all poisonings. This may indicate that pesticide poisonings are concentrated among the small establishments excluded from SOII, that under-reporting to SOII is more extreme than SENSOR-pesticide under-ascertainment, or that SENSOR-pesticide classification standards accept a large number of cases that BLS does not count.

Data from the National Agricultural Workers Survey (NAWS), by contrast, suggest an incidence of acute pesticide poisoning among agricultural workers [US Department of Labor, 2004] an order of magnitude greater than that found in this study. NAWS is a nationally representative annual survey of US crop workers conducted by the US Department of Labor. In 1999, NAWS included questions to determine if crop workers were poisoned by pesticides. This information was collected in two parts. First NAWS asked the crop worker if they were exposed to pesticides by “having them sprayed or blown on you,” “spilled on you,” or “when cleaning or repairing containers or equipment used for applying or storing pesticides.” NAWS then asked if the crop worker became “sick or [had] any reaction because of this incident.” Our analysis of these data found that 3.2% of crop workers acknowledged exposure during the previous 12 months, of

whom 43.4% reported getting sick or having a reaction. That is, 1.4% of US crop workers attributed health effects such as skin problems (59%), eye problems (24%), nausea/vomiting (30%), headache (26%), and numbness/tingling (12%) to pesticide exposure during the preceding 12 months. In a separate NAWS question, 0.6% of all US crop workers reported that in the last 12 months they had “received medical attention by a doctor or nurse due to pesticide exposure.” To our knowledge, neither these nor similar questions to assess the incidence of pesticide poisoning were included in NAWS surveys before or after 1999. In comparison, we found an average annual acute occupational pesticide poisoning IR of 0.05% among all agricultural workers, and 0.07% among farmworkers.

Acute pesticide poisoning IR were also assessed among farmers participating in the Agricultural Health Study (AHS), a prospective cohort study sponsored by the National Institutes of Health (i.e., the National Cancer Institute and the National Institute of Environmental Health Sciences) and EPA [AHS, 2007]. The AHS cohort consists of 52,395 farmers, 32,347 spouses of these farmers, and 4,916 commercial pesticide applicators residing in Iowa or North Carolina. In a nested case-control analysis involving 16,416 farmers/pesticide applicators who were interviewed by telephone in 1999–2000, 54 (0.33%) reported “an incident with fertilizers, weed killers, or other pesticides that caused an unusually high personal exposure” in the previous 12 months that resulted in physical symptoms [Bell et al., 2006]. Among these 54 individuals, only 7 (13%, or .04% of the entire subcohort) sought medical care. In contrast, we found an average annual acute occupational pesticide poisoning IR of 0.005% among farmers. However, as was observed in our study, the findings from AHS and NAWS suggest that the risk of pesticide poisoning is lower among farmers compared to farmworkers.

The true incidence of pesticide poisoning among agricultural workers remains uncertain. Our findings (51/100,000) fall between the low SOII estimate (less than 10/100,000) and the high rates elicited by NAWS interviews (1,400/100,000 symptomatic, 600/100,000 sought medical care).

Limitations

One of this study’s major limitations is that under-reporting compromises, to varying degrees, all the surveillance systems that provided the data for this analysis. Factors that contribute to under-reporting include: affected people not seeking care, or consulting care providers outside the jurisdiction of surveillance programs; misdiagnosis of this uncommon condition; and health care provider neglect of legal requirements to report. The rates provided should be considered low estimates of the magnitude of acute pesticide poisoning among agricultural workers.

Disproportionate numbers of agricultural workers probably are deterred from seeking health care by lack of health insurance [US Department of Labor, 2005], unfamiliarity with workers' compensation benefits or inability to qualify for them, and fear of job loss if they miss time from work to seek health care [Das et al., 2001; Arcury and Quandt, 2007], as well as concerns related to immigration status. Similarly, a variety of interrelated problems may lead health care professionals to misdiagnose acute pesticide poisoning. Health professionals rarely receive much training in environmental toxicology generally or on pesticide poisoning specifically [Schenk et al., 1996]. Consequently, they may not collect a pesticide exposure history, which is necessary to make a diagnosis of acute pesticide poisoning [Balbus et al., 2006]. Pesticide poisoning is relatively rare in developed countries, and its signs and symptoms often resemble those of more common conditions, which may be diagnosed preferentially. The difficulty and delays of receiving reimbursement through workers' compensation may also bias health care providers against diagnosing and reporting pesticide poisoning. Even among those cases correctly diagnosed, some cases may escape report to public health authorities through ignorance of the requirement (despite the fact that 30 states have a mandatory reporting system of occupationally related pesticide poisoning [Calvert et al., 2001]) or because the health care professionals fear that their patients may be subject to retaliation. Other cases may go unreported because many farmworkers immigrate from Mexico [US Department of Labor, 2005], and some poisoned Mexican farmworkers may prefer to visit physicians in Mexico where cultural and linguistic barriers are removed and fees are lower [US EPA, 1992; Arcury and Quandt, 2007]. Our state-based surveillance partners received only nine reports of pesticide poisoning cases managed outside the US.

Another limitation in our study was that information was incomplete for some reported cases. Most cases lacked information on race and ethnicity. Missing information could lead to misclassification of severity, if not all signs and symptoms were reported, or to inappropriate exclusion of the case. More detailed information on the affected worker's activities, pesticide exposures and health effects might have increased our case totals. Some cases in this report may be false-positives, with compatible symptoms that are coincidental with but not caused by pesticide exposure. Finally, information on factors that contributed to illness was identified in only 1,926 (59%) cases. In many cases a timely and definitive investigation into the factors responsible for exposure and illness was not possible due to insufficient investigatory resources and/or because of tardy notification of the exposure to state authorities.

Rates of pesticide poisoning may also be distorted by inaccuracy in estimates of population at risk. The size of the agricultural worker population, including farmworkers and processing/packing plant workers, is difficult to estimate for

several reasons, including the transient employment of many seasonal and migrant farmworkers, migration into and out of the United States in a manner that is not entirely predictable, and the tendency of many farmworkers to avoid government contact [Rust, 1990]. Our agricultural worker population estimates were derived from the CPS, which is conducted by the BLS and the United States Census Bureau. The CPS goes to great lengths to capture reliable workforce data [Bureau of Labor Statistics, 2002]. Nevertheless, a population seeking to escape detection could well be under-counted, leading to inflated apparent rates of illness/injury. Finally, illness rates for those known to have occupational pesticide exposure are not available because the numbers of workers exposed to pesticides are unknown.

Reasons for Higher Poisoning Rates in Western States

Rates of both agricultural and non-agricultural acute pesticide poisoning are higher in the western states as compared to the south and eastern regions of the United States. It is credible that labor-intensive Western agriculture may impose excess risk for acute pesticide poisoning illness, but it is also important to note that California (especially the CDPR program) and Washington have well established, longstanding and experienced state-based surveillance programs with higher staffing levels compared to other states participating in the SENSOR-Pesticides program [Calvert et al., 2004]. In addition, these states were much more likely to be notified about cases through the state workers' compensation system (in Washington State 76% of the cases were so identified; in California 34% of reports were provided by physicians to a workers' compensation insurance carrier). In contrast, only two other states identified cases through their state workers' compensation system: Oregon and Texas (7% and 4%, respectively, of cases in these states were so identified).

Higher Poisoning Rates Among Female Agricultural Workers

Female agricultural workers experienced nearly twice the risk of pesticide poisoning of male agricultural workers (Table III), a finding that was quite unexpected. Before indulging in speculation about possible differences in susceptibility, risk of exposure, or rate of ascertainment, we plan to perform more detailed analyses by geographic region, activity at time of exposure, pesticide, protective equipment, and severity.

Higher Poisoning Rates Among Processing/Packing Plant Workers

Processing/packing plant workers were found to have the highest acute pesticide poisoning IR compared to all other

agricultural occupations. Many farms are increasing the amount of food processing that is performed on site [National Research Council and Institute of Medicine, 2008]. This is due to a variety of factors, including the advent of new technology, quest for improved quality control and freshness, and a desire to increase profit. The types of food processing activities performed on farms include cleaning, sorting, packing, and cooling/freezing. Among the 394 poisoned processing/packing plant workers, the pesticides most commonly responsible for illness were fumigants (N = 151, 38%), disinfectants (N = 151, 38%), and insecticides (N = 73, 19%). The fumigant exposures were commonly related to drift from a nearby field (N = 111), the disinfectant exposures were commonly related to malfunction of disinfecting equipment (N = 74) and cleaning produce in disinfectant solutions (N = 31) and the insecticide exposures were commonly related to pesticide residue present on the produce (N = 37). Because the WPS only covers workers involved in the production of agricultural plants, processing/packing plant workers may not be covered by WPS.

Chronic Health Effects Associated With Acute Pesticide Poisoning

In addition to acute morbidity with its attendant costs in health care resources, and time lost from work and normal daily activities, acute pesticide poisoning is also associated with chronic adverse health sequelae. For example organophosphate poisoning has been found to be associated with deficits in neurobehavioral and neurosensory function [Steenland et al., 1994]. In addition, the Agricultural Health Study found that those who sought medical care for pesticide poisoning or who experienced an incident involving “unusually high” pesticide exposure had an increased risk for chronic neurologic symptoms (Kamel et al. 2005). These “unusually high” pesticide exposures, which are labeled by the authors as high pesticide exposure events (HPEE), result in acute symptomatic illness about 50% of the time [Bell et al., 2006]. Those who ever experienced an HPEE also had an increased risk for farmers lung (Hoppin et al. 2006). A non-significant elevated risk for prostate cancer was observed among those who had ever experienced an HPEE (odds ratio = 1.11, 95% CI = 0.8, 1.6) [Alavanja et al., 2003]. To our knowledge, the AHS has not published findings on any other associations between HPEE and cancer.

Recommendations

The most common factors that contributed to pesticide exposure included off-target drift, early reentry into a treated area, and use in conflict with the pesticide label. These findings and the observations of other investigators [Arcury et al., 2001] suggest that improved compliance with and enforcement of existing pesticide regulations could achieve

important improvements in safety. Measures to minimize drift (including equipment specifications, establishment of buffer zones, and limitations on maximum wind speed conditions during an application) seem likely to provide the greatest benefit. Our finding that 992 (30%) cases had no obvious factors contributing to exposure suggests that pesticide regulations and label requirements may also need to be enhanced. Additionally, reduced-risk pest control measures such as integrated pest management should be adopted, which can achieve reductions in pesticide exposure and misuse [National Research Council, 2000]. The high poisoning rates observed among processing/packing plant workers and the increased amount of food processing performed on farms suggests that processing/packing plant workers should be covered under the WPS.

Given the limitations in this analysis, improved state-based surveillance programs for pesticide-related illness are also needed. A comprehensive system needs to address the limitations described above including: agricultural workers and health care providers need to recognize the pesticide-relatedness of the illness; disincentives to receiving health care, including lack of health insurance, must be overcome; the costs of evaluation and treatment of acute occupational pesticide poisoning should be paid for by workers' compensation; health care providers need to make timely reports to pesticide poisoning surveillance systems; and, surveillance systems need to optimize access to and use of workers' compensation data, poison control center data and data from other state agencies with jurisdiction over pesticides.

CONCLUSION

Agricultural workers are at increased risk of acute pesticide poisoning in comparison to non-agricultural workers, particularly through drift, early reentry into a treated area, and use in conflict with the label. The IR was almost twofold higher in female agricultural workers compared to males. In addition to acute intoxication, pesticide poisoning may also lead to chronic adverse health sequelae. Improved compliance with and stringent enforcement of laws and regulations regarding pesticide applications are needed. Alternative pest control measures such as integrated pest management reduce the use of pesticides and therefore the potential for adverse health effects.

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Acute Pesticide Illnesses Associated with Off-Target Pesticide Drift from Agricultural Applications: 11 States, 1998–2006

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BACKGROUND: Pesticides are widely used in agriculture, and off-target pesticide drift exposes workers and the public to harmful chemicals.

OBJECTIVE: We estimated the incidence of acute illnesses from pesticide drift from outdoor agricultural applications and characterized drift exposure and illnesses.

METHODS: Data were obtained from the National Institute for Occupational Safety and Health's Sentinel Event Notification System for Occupational Risks–Pesticides program and the California Department of Pesticide Regulation. Drift included off-target movement of pesticide spray, volatiles, and contaminated dust. Acute illness cases were characterized by demographics, pesticide and application variables, health effects, and contributing factors.

RESULTS: From 1998 through 2006, we identified 2,945 cases associated with agricultural pesticide drift from 11 states. Our findings indicate that 47% were exposed at work, 92% experienced low-severity illness, and 14% were children (< 15 years). The annual incidence ranged from 1.39 to 5.32 per million persons over the 9-year period. The overall incidence (in million person-years) was 114.3 for agricultural workers, 0.79 for other workers, 1.56 for nonoccupational cases, and 42.2 for residents in five agriculture-intensive counties in California. Soil applications with fumigants were responsible for the largest percentage (45%) of cases. Aerial applications accounted for 24% of cases. Common factors contributing to drift cases included weather conditions, improper seal of the fumigation site, and applicator carelessness near nontarget areas.

CONCLUSIONS: Agricultural workers and residents in agricultural regions had the highest rate of pesticide poisoning from drift exposure, and soil fumigations were a major hazard, causing large drift incidents. Our findings highlight areas where interventions to reduce off-target drift could be focused.

KEY WORDS: agriculture, drift, pesticides, poisoning, surveillance. *Environ Health Perspect* 119:1162–1169 (2011). doi:10.1289/ehp.1002843 [Online 6 June 2011]

Pesticide drift, which is the off-target movement of pesticides, is recognized as a major cause of pesticide exposure affecting people as well as wildlife and the environment. In the United States in 2004, > 1,700 investigations were conducted in 40 states because of drift complaints, and 71% of the incident investigations confirmed that drift arose from pesticide applications to agricultural crops (Association of American Pesticide Control Officials 2005). Pesticide drift has been reported to account for 37–68% of pesticide illnesses among U.S. agricultural workers [California Department of Pesticide Regulation (CDPR) 2008; Calvert et al. 2008]. Community residents, particularly in agricultural areas, are also at risk of exposure to pesticide drift from nearby fields. Agricultural pesticides are often detected in rural homes (Harnly et al. 2009; Quandt et al. 2004). Alarcon et al. (2005) reported that 31% of acute pesticide illnesses that occurred at U.S. schools were attributed to drift exposure.

The occurrence and extent of pesticide drift are affected by many factors, such as the nature of the pesticide (e.g., fumigants are highly volatile, which increases their propensity for off-site movement [U.S. Environmental Protection Agency (U.S. EPA) 2010], equipment and application techniques (e.g., size and height of the spray nozzles), the amount of pesticides applied, weather (e.g., wind speed, temperature inversion), and operator care (Hofman and Solseng 2001). Pesticide applicators are required to use necessary preventive measures and to comply with label requirements to minimize pesticide drift. Pesticide regulations such as the Federal Insecticide, Fungicide, and Rodenticide Act (FIFRA) and EPA's Worker Protection Standard require safety measures for minimizing the risk of pesticide exposure (U.S. EPA 2008, 2009), and many states have additional regulations for drift mitigation (Feitshans 1999).

Better understanding about the magnitude, trend, and characteristics of pesticide

poisoning from drift exposure of agricultural pesticides would assist regulatory authorities with regulatory, enforcement, and education efforts. The purpose of this study was to estimate the magnitude and incidence of acute pesticide poisoning associated with pesticide drift from outdoor agricultural applications in the United States during 1998–2006 and to describe the exposure and illness characteristics of pesticide poisoning cases arising from off-target drift. We also examined factors associated with illness severity and large events that involved five or more cases.

Materials and Methods

Data on acute pesticide poisoning cases were obtained from the National Institute for Occupational Safety and Health (NIOSH)'s Sentinel Event Notification System for Occupational Risks (SENSOR)-Pesticides program and CDPR's Pesticide Illness Surveillance Program (PISP). The SENSOR-Pesticides program has collected pesticide poisoning surveillance data from 12 states using standardized definitions and variables available since 1998 (Calvert et al. 2010). This study included data from 11 states for the following years: Arizona, 1998–2000; California, 1998–2006; Florida, 1998–2006; Iowa, 2006; Louisiana,

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The findings and conclusions in this report are those of the authors and do not necessarily represent the views of NIOSH or each author's state agency.

J.B. is assigned to the California Department of Public Health by his employer [Public Health Institute (PHI)] and has never been involved in any advocacy activities of PHI. The authors declare they have no actual or potential competing financial interests.

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2000–2006; Michigan, 2000–2006; New Mexico, 2005–2006; New York, 1998–2006; Oregon, 1998–2006; Texas, 1998–2006; and Washington, 2001–2006. North Carolina, which joined SENSOR-Pesticides in 2007, was not included. Because each state removes personal identifiers from the data before submission to the Centers for Disease Control and Prevention (CDC), this study was exempt from consideration by the federal Human Subjects Review Board.

Participating surveillance programs identify cases from multiple sources, including health care providers, poison control centers, workers' compensation claims, and state or local government agencies. They collect information on the pesticide exposure incident through investigation, interview, and medical record review. In California, on some occasions, such as large drift events, active surveillance is undertaken for further case finding by interviewing individuals living or working within the vicinity affected by the off-target drift (Barry et al. 2010). Although the SENSOR-Pesticides program focuses primarily on occupational pesticide poisoning surveillance, all of the SENSOR-Pesticides state programs except California collect data on both occupational and nonoccupational cases. In California, PISP captures both occupational and nonoccupational cases. SENSOR-Pesticides and PISP classify cases based on the strength of evidence for pesticide exposure, health effects, and the known toxicology of the pesticide and use slightly different criteria for case classification categories (Calvert et al. 2010). This study restricted the analyses to cases classified as definite, probable, possible, or suspicious by SENSOR-Pesticides and definite, probable, or possible by PISP. We also performed analyses restricted to definite and probable cases only. Because the findings from these restricted analyses were similar to those that included all four classification categories (i.e., definite, probable, possible, or suspicious), only the findings that used the four classification categories are reported here.

In this study, a drift case was defined as acute health effects in a person exposed to pesticide drift from an outdoor agricultural application. Drift exposure included any of the following pesticide exposures outside their intended area of application: *a*) spray, mist, fumes, or odor during application; *b*) volatilization, odor from a previously treated field, or migration of contaminated dust; and *c*) residue left by offsite movement. Our drift definition is broader than U.S. EPA's "spray or dust drift" definition, which excludes post-application drift caused by erosion, migration, volatility, or windblown soil particles (U.S. EPA 2001). A drift event was defined as an incident where one or more drift cases experienced drift exposure from a particular source.

Both occupational and nonoccupational cases were included. An occupational case was defined as an individual exposed while at work. Among occupational cases, agricultural workers were identified using 1990 and 2002 Census Industry Codes (CICs): 1990 CICs, 010, 011, 030; 2002 CICs, 0170, 0180, 0290 (U.S. Census Bureau 1992, 2005).

Figure 1 presents the process of case selection. We selected cases if exposed to pesticides applied for agricultural use including farm, nursery, or animal production, and excluded cases exposed by ingestion, direct spray, spill, or other direct exposure. We then manually reviewed all case reports and excluded persons exposed to pesticides used for indoor applications (e.g., greenhouses, produce packing facilities), persons exposed within a treated area (e.g., pesticide applicators exposed by pesticides blown back by wind, workers working within or passing through the field being treated), and persons exposed to pesticides being mixed, loaded, or transported. Drift cases therefore represented the remaining 9% and 27% of all pesticide illness cases identified by the SENSOR-Pesticides and PISP, respectively. We also searched for duplicates from the two programs identifying California cases. Because personal identifiers were unavailable, date of exposure, age, sex, active ingredients, and county were used for comparison. A total of 60 events and 171 cases were identified by both California programs. These were counted only once and were included only in the PISP total.

Drift events and cases were analyzed by the following variables: state, year, and month

of exposure, age, sex, location of exposure, health effects, illness severity, pesticide functional and chemical class, active ingredient, target of application, application equipment, detection of violations, and factors contributing to the drift incident. U.S. EPA toxicity categories ranging from toxicity I (the most toxic) to IV (the least toxic) were assigned to each product (U.S. EPA 2007). Cases exposed to multiple products were assigned to the toxicity category of the most toxic pesticide they were exposed to. Illness severity was categorized into low, moderate, and high using criteria developed by the SENSOR-Pesticides program (Calvert et al. 2010). Low severity refers to mild illnesses that generally resolve without treatment. Moderate severity refers to illnesses that are usually systemic and require medical treatment. High severity refers to life-threatening or serious health effects that may result in permanent impairment or disability. Contributing factors were retrospectively coded with available narrative descriptions. One NIOSH researcher (S.J.L.) initially coded contributing factors for all cases. Next, for SENSOR-Pesticides cases, state health department staff reviewed the codes and edited them as necessary. Any discrepancies were resolved by a second NIOSH researcher (G.M.C.). For PISP cases, relatively detailed narrative descriptions were available for all incidents. These narratives summarize investigation reports provided by county agriculture commissioners, who investigate all suspected pesticide poisoning cases reported in their county. After initial coding, the two

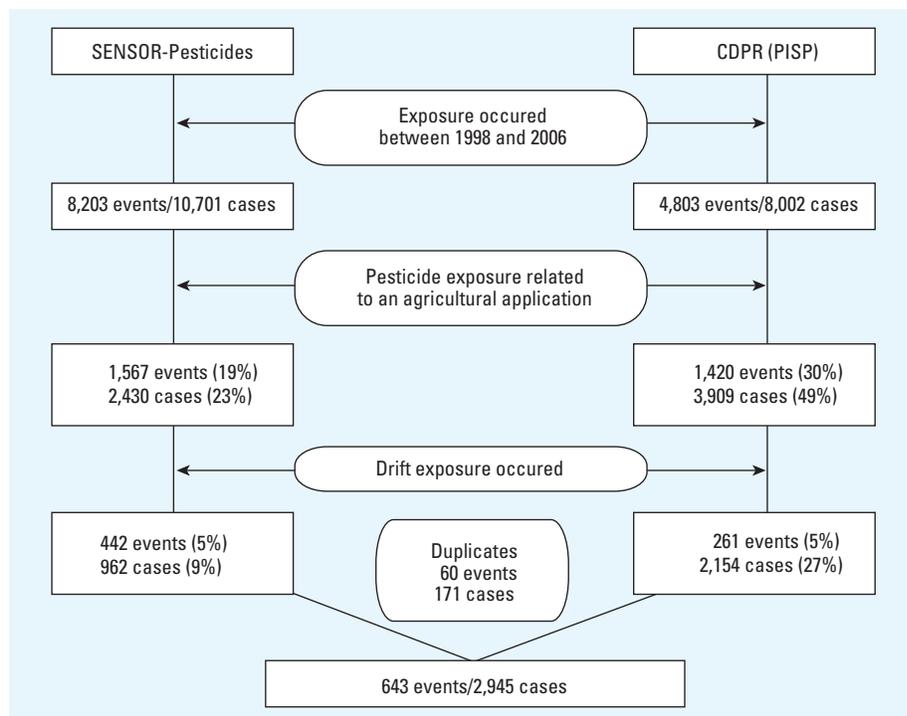


Figure 1. Eligible pesticide drift events and cases, 11 states, 1998–2006.

NIOSH researchers discussed those narratives that lacked clarity to reach consensus.

Data analysis. Data analysis was performed with SAS software (version 9.1; SAS Institute Inc., Cary, NC). Descriptive statistics were used to characterize drift events and cases. Incidence rates were calculated by geographic region, year, sex, and age group. The numerator represented the total number of respective cases in 1998–2006. Denominators were generated using the Current Population Survey microdata files for the relevant years (U.S. Census Bureau 2009). For total and nonoccupational rates, the denominators were

calculated by summing the annual average population estimates. A nonoccupational rate for agriculture-intensive areas was calculated by selecting the five counties in California where the largest amounts of pesticides were applied in 2008 (Fresno, Kern, Madera, Monterey, and Tulare) (CDPR 2010). For occupational rates, the denominators were calculated by summing the annual employment estimates including both “employed at work” and “employed but absent.” The denominator for agricultural workers was obtained using the same 1990 and 2002 CICs used to define agricultural worker cases (U.S. Census Bureau

1992, 2005). Moreover, in California, where data on pesticide use are available, incidence was calculated per number of agricultural applications and amount of pesticide active ingredient applied (CDPR 2009). Incidence trend over time was examined by fitting a Poisson regression model of rate on year and deriving the regression coefficient and its 95% confidence interval (CI).

Drift events were dichotomized by the size of events into small events involving < 5 cases and large events involving ≥ 5 cases. This cut-point was based on one of the criteria used by the CDPR to prioritize event investigations

Table 1. Number and incidence rate^a of off-target drift events and pesticide poisoning cases by year, region, sex, and age, 11 states, 1998–2006.

Variable	Drift events Count (%)	Drift cases											Total rate
		All cases			Nonoccupational cases		Occupational cases						
		Count	Population estimate ^b	Rate	Count	Rate ^c	Agricultural worker cases		Other worker cases				
						Count	Employment estimate ^{b,d}	Rate	Count	Employment estimate ^b	Rate		
Total	643 (100)	2,945	1,004.1	2.93	1,565	1.56	1,010	8.83	114.33	370	468.0	0.79	2.89
Year of exposure (no. states included)													
1998 (6)	60 (9.3)	130	93.6	1.39	46	0.49	45	1.11	40.46	39	43.2	0.90	1.90
1999 (6)	82 (12.8)	407	95.0	4.28	273	2.87	72	1.12	64.22	62	44.1	1.41	2.97
2000 (8)	64 (10.0)	193	110.3	1.75	76	0.69	93	1.24	74.94	24	51.8	0.46	2.21
2001 (8)	88 (13.7)	177	112.6	1.57	98	0.87	43	1.12	38.47	36	52.5	0.69	1.47
2002 (8)	81 (12.6)	580	113.7	5.10	271	2.38	281	1.11	252.33	28	52.2	0.54	5.80
2003 (8)	75 (11.7)	348	116.4	2.99	265	2.28	43	0.79	54.64	40	53.7	0.74	1.52
2004 (8)	47 (7.3)	232	117.4	1.98	43	0.37	177	0.75	235.33	12	54.7	0.22	3.41
2005 (9)	70 (10.9)	642	120.6	5.32	409	3.39	168	0.75	224.77	65	56.8	1.14	4.05
2006 (10)	76 (11.8)	236	124.5	1.90	84	0.67	88	0.84	104.53	64	59.1	1.08	2.54
Region													
West ^e	433 (67.3)	2,484	397.9	6.24	1,240	3.12	933	4.44	210.20	311	184.9	1.68	6.57
South ^f	193 (30.0)	426	365.6	1.17	311	0.85	59	3.25	18.17	56	170.7	0.33	0.66
East/central ^g	17 (2.6)	35	240.6	0.15	14	0.06	18	1.15	15.68	3	112.5	0.03	0.18
Sex													
Male	NA	1,560	491.6	3.17	742	1.51	554	6.90	80.27	264	251.6	1.05	3.16
Female	NA	1,360	512.5	2.65	807	1.57	448	1.93	231.90	105	216.5	0.49	2.53
Unknown	NA	25	—	—	16	—	8	—	—	1	—	—	—
Age (years)													
< 15	NA	418	221.2	1.89	415	1.88	3	—	—	0	—	—	—
15–24	NA	398	142.0	2.80	182	1.28	182	1.44	126.39	34	67.8	0.50	3.12
25–34	NA	453	140.0	3.24	140	1.00	240	1.81	132.53	73	106.8	0.68	2.88
35–44	NA	458	156.7	2.92	181	1.16	187	2.08	89.89	90	122.3	0.74	2.23
45–54	NA	306	136.1	2.25	172	1.26	78	1.59	49.00	56	104.6	0.54	1.26
55–64	NA	164	90.9	1.80	103	1.13	37	1.10	33.61	24	52.0	0.46	1.15
≥ 65	NA	92	117.2	0.78	80	0.68	9	0.81	11.11	3	14.6	0.21	0.78
Unknown	NA	656	—	—	292	—	274	—	—	90	—	—	—

Abbreviations: —, the denominator was not available and thus a rate was not calculated, NA, for sex and age, counting the number of events was not applicable. ^aPer 1,000,000 persons. ^bCases and employment estimates of agricultural workers were defined with 1990 and 2002 CICs (010, 011, 030 and 0170, 0180, 0290, respectively). ^cNumbers (in millions) were estimated using the Current Population Survey data (U.S. Census Bureau 2009). Participating years vary by state; only years of participation were included. ^dDenominators were population estimates. ^eArizona, California, New Mexico, Oregon, Washington. ^fFlorida, Louisiana, Texas. ^gIowa, Michigan, New York.

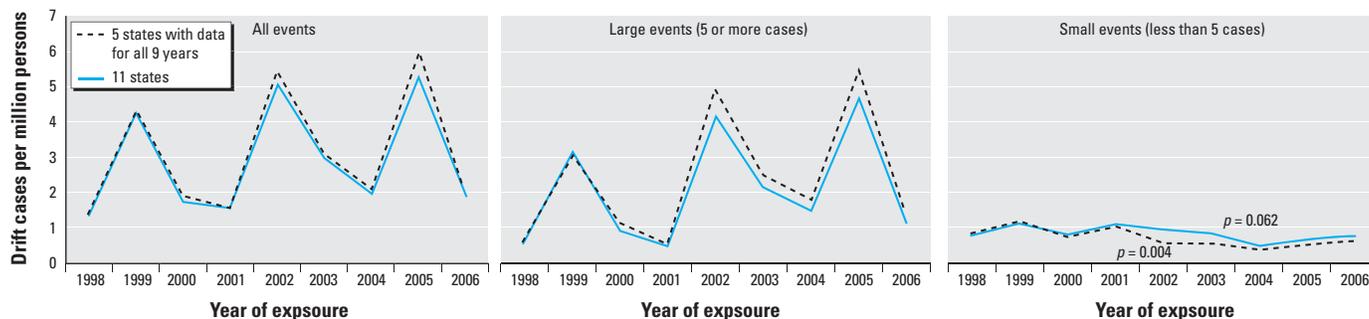


Figure 2. Incidence rate of pesticide poisoning associated with off-target drift exposure over time, 11 states, 1998–2006.

(CDPR 2001). Illness severity was dichotomized as low and moderate/high. Simple and multivariable logistic regressions were performed. Odds ratios (ORs) and 95% CIs were calculated.

Results

Number and incidence of drift events and cases. From 1998 through 2006, we identified 643 events and 2,945 illness cases associated with pesticide drift from agricultural applications (Figure 1). Of these, 382 events (59%) and 791 cases (27%) were identified by SENSOR-Pesticides (excluding 60 events and 171 cases also identified by PISP), and 261 events (41%) and 2,154 cases (73%) were identified by PISP. Drift cases consisted of 53 definite (1.8%), 2,019 probable (68.6%), 823 possible (27.9%), and 50 suspicious (1.7%) cases. Among drift cases, 1,565 (53%) were nonoccupational and 1,380 (47%) were occupational. Agricultural workers accounted for 73% (*n* = 1,010) of the occupational cases. A total of 340 events (53%) occurred between May and August, and these involved 1,407 cases (48%).

The overall incidence rate of drift-related pesticide poisoning was 2.93 per million person-years (Table 1). The rates of nonoccupational and occupational drift-related pesticide poisoning were 1.56 and 2.89 per million persons-years, respectively. Among occupational cases, the rate was 114.3 for agricultural workers and 0.79 for all other workers. Among nonoccupational cases identified in California, the rate was 42.2 for residents in the five agriculture-intensive counties and 0.61 for residents of all other California counties (data not shown). The rate was highest in the western states for both nonoccupational and occupational cases (Table 1). In California, per 100,000 agricultural applications, 1.6 drift events and 11.8 cases were identified; per 10 million pounds applied, 1.9 events and 14.4 cases were identified (data not shown).

The total annual incidence rate ranged from 1.39 to 5.32 per million persons over the 9-year time period (Table 1). Over time, the rate of drift cases involved in large events showed the same pattern as the rate of all drift cases, showing a spike every 3 years (Figure 2). The rate of drift cases involved in small events varied within a narrow range from 0.49 to 1.11, and we found no significant rate change over this time period; however, for the five states that provided data for all 9 years, we found a significant decrease in the rate (i.e., an estimated 9% decrease per year; 95% CI, 3–15%; *p* = 0.004).

Men comprised 53% of all cases (Table 1). The rate by sex was similar among non-occupational cases. For occupational cases, the rate was 1.25 times higher in male workers than in female workers but 2.89 times higher in female agricultural workers than in male

agricultural workers. Among nonoccupational cases, children < 15 years of age accounted for 33% of cases with known age and showed the highest rate (1.88/million person-years; Table 1).

Responsible pesticides, application targets, and application equipment. In 430 (67%) of 643 drift events, exposure was to pesticides from a single functional class (Table 2). Insecticides were the most commonly identified (31% of events), accounting for 23% (*n* = 678) of all cases. Fumigants were involved in only 8% of drift events but accounted for 45% (*n* = 1,330) of all cases. Organophosphorus compounds were the most common pesticide chemical class involved in drift events (28%). Most cases (66%) were exposed to toxicity I (high toxicity) pesticides.

For the intended application targets, 71% of events involved applications to fruit, grain/fiber/grass, or vegetable crops (Table 2). Soil

applications accounted for 9% of drift events and 45% of all cases. For application equipment, aerial applications (e.g., by airplane) were responsible for 39% of drift events, accounting for 24% of all cases. Chemigation (i.e., application via an irrigation system) or soil injectors were used in 7% of drift events and accounted for 44% of cases. All soil injector events and 95% of chemigation events involved the use of fumigants applied to soil (data not shown).

Location of exposure and health effects. Common exposure locations were private residences (44%) and farms/nurseries (37%; Table 3). More than half of cases experienced ocular (58%) or neurological (53%) symptoms or signs, and illness severity was low for most cases (92%; Table 3). Moderate/high severity illness was significantly associated with females, older age groups, and exposure to multiple active ingredients, before and after

Table 2. Off-target drift events and pesticide poisoning cases by pesticide and application characteristics, 11 states, 1998–2006.

Variable	Drift events (<i>n</i> = 643) <i>n</i> (%)	Drift cases		
		Total (<i>n</i> = 2,945) <i>n</i> (%)	Occupational <i>n</i> = 1,380 (%)	Nonoccupational <i>n</i> = 1,565 (%)
Pesticide functional class				
Insecticide only	198 (30.8)	678 (23.0)	32.9	14.3
Herbicide only	108 (16.8)	195 (6.6)	4.0	8.9
Fungicide only	29 (4.5)	64 (2.2)	3.7	0.8
Fumigant only	52 (8.1)	1,330 (45.2)	27.0	61.2
Other, single	43 (6.7)	87 (3.0)	2.8	3.1
Multiple	207 (32.2)	585 (19.9)	29.4	11.4
Unknown	6 (0.9)	6 (0.2)	0.2	0.2
Common pesticide chemical class^a				
Organophosphorus compound	181 (28.1)	660 (22.4)	36.7	9.8
Inorganic compound	87 (13.5)	231 (7.8)	11.1	5.0
Pyrethroid	52 (8.1)	207 (7.0)	9.6	4.7
Dithiocarbamates ^b	47 (7.3)	726 (24.7)	22.5	26.5
<i>N</i> -Methyl carbamates	33 (5.1)	71 (2.4)	4.1	1.0
Chlorophenoxy compound	26 (4.0)	47 (1.6)	0.9	2.2
Triazines	11 (1.7)	34 (1.2)	1.1	1.2
Maximum toxicity category				
I	203 (31.6)	1,944 (66.0)	59.9	71.4
II	167 (26.0)	468 (15.9)	21.2	11.2
III	154 (24.0)	327 (11.1)	13.6	8.9
Unknown	119 (18.5)	206 (7.0)	5.2	8.6
Application target				
Fruit crops	189 (29.4)	588 (20.0)	27.6	13.2
Grain/fiber/grass crops	185 (28.8)	411 (14.0)	12.8	15.0
Vegetable crops	85 (13.2)	374 (12.7)	22.9	3.7
Soil	55 (8.6)	1,337 (45.4)	27.5	61.2
Landscape/forest	32 (5.0)	64 (2.2)	2.8	1.7
Undesired plants	29 (4.5)	44 (1.5)	0.9	2.0
Other (e.g., miscellaneous crops, seed, livestock farm)	27 (4.2)	66 (2.2)	2.0	2.5
Unknown	41 (6.4)	61 (2.1)	3.6	0.8
Application equipment				
Aerial applicator	249 (38.7)	695 (23.6)	32.0	16.2
Handheld or backpack sprayer	24 (3.7)	63 (2.1)	3.8	0.6
Chemigation	22 (3.4)	752 (25.5)	16.4	33.5
Soil injector	20 (3.1)	558 (18.9)	10.0	26.8
Other ground applicator	254 (39.5)	747 (25.4)	32.6	19.0
Multiple	8 (1.2)	41 (1.4)	0.2	2.4
Unknown	66 (10.3)	89 (3.0)	4.9	1.4

^aCategories with the largest numbers of cases. Events and cases can be exposed to multiple categories. ^bMostly from single products.

controlling for other case and pesticide characteristics ($p < 0.05$; Table 4). Compared with fumigants, exposures to herbicides, insecticides, or multiple classes were significantly associated with moderate/high illness. Table 5 lists 15 active ingredients most commonly found among drift cases and their distribution according to illness severity.

Size of drift events. Most drift events involved a single case ($n = 387$, 60%). For multiperson events, 168 events (26% of the total) involved 2–4 cases, 78 events (12%) involved 5–29 cases, and 10 events (1.5%) involved ≥ 30 cases. Table 6 provides details

on the 10 largest events. Detailed investigation reports of some of these events are available elsewhere (Barry et al. 2010; CDC 2004; O'Malley et al. 2005). The occurrence of large versus small events (events with ≥ 5 vs. < 5 cases) was significantly associated with the use of fumigants (compared with insecticides) and applications to soil, small fruit crops, or leafy vegetable crops (compared with other targets; $p < 0.05$; Table 7).

Contributing factors to drift incidents. Of 299 drift events with information on violations of pesticide regulations, 220 (74%) had one or more violations and accounted

for 2,093 cases (89% of cases with violation information; Table 8). However, not all of the observed violations may have directly contributed to the drift exposure. Factors contributing to the drift exposure were identified in 164 events, accounting for 1,544 (52%) cases. Common contributing factors identified for drift events included applicators' carelessness near or over nontarget sites (e.g., flew over a house, did not turn off a nozzle at the end of the row), unfavorable weather conditions (e.g., high wind speed, temperature inversion), and poor communication between applicators or growers and others. Improper seal of the fumigation site (e.g., tarp tear, early removal of seal), which were identified in nine events, accounted for the largest proportion (60%) of cases with contributing factors identified.

The distance between the application and exposure site was identified in 1,428 (48%) cases (Table 8). Occupational cases accounted for 68% of cases exposed within 0.25 miles of the application site, and nonoccupational cases accounted for 73% of cases exposed > 0.25 miles away.

Discussion

To our knowledge, this is the first comprehensive report of drift-related pesticide poisoning in the United States. We identified 643 events involving 2,945 illness cases associated with pesticide drift from outdoor agricultural applications during 1998–2006. Pesticide drift included pesticide spray, mist, fume, contaminated dust, volatiles, and odor that moved away from the application site during or after the application. Although the incidence for cases involved in small drift events (< 5 cases) tended to decrease over time, the overall incidence maintained a consistent pattern chiefly driven by large drift events. Large drift events were commonly associated with soil fumigations.

Occupational exposure. Occupational pesticide poisoning is estimated at 12–21 per million U.S. workers per year (Calvert et al. 2004; Council of State and Territorial Epidemiologists 2010). Compared with those estimates, our estimated incidence of 2.89 per million worker-years suggests that 14–24% of occupational pesticide poisoning may be attributed to off-target drift from agricultural applications. Our study included pesticide drift from outdoor applications only and excluded workers exposed within the application area. Our findings show that the risk of illness resulting from drift exposure is largely borne by agricultural workers, and the incidence (114.3/million worker-years) was 145 times greater than that for all other workers. Current regulations require agricultural employers to protect workers from exposure to agricultural pesticides, and pesticide

Table 3. Location of exposure, health effects, and illness severity of drift cases ($n = 2,945$).

Variable	Percent
Location of exposure	
Private residence	44.5
Farm/nursery	36.7
Road/right-of-way	5.6
School	3.6
Agricultural processing facility	2.4
Other/unknown	7.2
Health effect^a	
Eye (e.g., pain/irritation/inflammation, lacrimation)	58.2
Neurological (e.g., headache, paresthesia, dizziness)	52.8
Respiratory (e.g., dyspnea, respiratory tract pain/irritation, cough)	47.8
Gastrointestinal (e.g., vomiting, nausea, diarrhea, abdominal pain)	41.5
Skin (e.g., pruritus, pain/irritation, rash)	14.7
Cardiovascular (e.g., chest pain)	5.1
Other (e.g., fatigue, fever)	11.4
Illness severity	
Low	92.2
Moderate	7.3
High	0.5

^aCases may have been included in multiple categories.

Table 4. Illness severity by case and pesticide characteristics.

Variable	Moderate/high severity ($n = 230$)	Low severity ($n = 2,715$)	Moderate/high severity (vs. low)	
	n (%)	n (%)	OR (95% CI)	Adjusted OR ^a (95% CI)
Sex^b				
Female	126 (54.8)	1,234 (45.5)	1.43 (1.09–1.87)	1.53 (1.15–2.04)
Male	104 (45.2)	1,456 (53.6)	Reference	Reference
Age (years)				
< 15	16 (7.0)	402 (14.8)	Reference	Reference
15–24	28 (12.2)	370 (13.6)	1.90 (1.01–3.57)	1.34 (0.68–2.62)
25–34	48 (20.9)	405 (14.9)	2.98 (1.66–5.33)	1.95 (1.02–3.71)
35–44	48 (20.9)	410 (15.1)	2.94 (1.64–5.27)	1.91 (1.02–3.58)
45–54	38 (16.5)	268 (9.9)	3.56 (1.95–6.52)	2.34 (1.24–4.41)
55–64	21 (9.1)	143 (5.3)	3.69 (1.87–7.27)	2.42 (1.20–4.91)
≥ 65	16 (7.0)	76 (2.8)	5.29 (2.54–11.03)	3.67 (1.72–7.86)
Unknown	15 (6.5)	641 (23.6)	0.59 (0.29–1.20)	0.63 (0.30–1.33)
Work related				
Yes	126 (54.8)	1,254 (46.2)	1.41 (1.08–1.85)	0.99 (0.70–1.40)
No/unknown	104 (45.2)	1,461 (53.8)	Reference	Reference
No. active ingredients				
1	90 (39.1)	1,719 (63.3)	Reference	Reference
> 1	140 (60.9)	996 (36.7)	2.72 (2.07–3.58)	1.42 (1.02–1.99)
Pesticide functional class				
Fumigant	35 (15.2)	1,295 (47.7)	Reference	Reference
Herbicides	33 (14.3)	162 (6.0)	7.54 (4.56–12.46)	4.10 (2.34–7.19)
Insecticide	79 (34.3)	599 (22.1)	4.88 (3.24–7.35)	3.34 (2.10–5.32)
Fungicides	2 (0.9)	62 (2.3)	1.19 (0.28–5.08)	0.77 (0.18–3.37)
Multiple	71 (30.9)	514 (18.9)	5.11 (3.37–7.76)	3.09 (1.85–5.16)
Other/unknown	10 (4.3)	83 (3.1)	4.46 (2.13–9.32)	2.82 (1.29–6.15)

^aAdjusted for all other variables. ^bExcluded unknown cases.

product labels instruct applicators to avoid allowing contact with humans directly or through drift (U.S. EPA 2009).

Our study found that the incidence of drift-related pesticide poisoning was higher among female and younger agricultural workers and in western states. These groups were previously found to have a higher incidence of pesticide poisoning (Calvert et al. 2008). It is not known why the incidence is higher among female and younger agricultural workers, but hypotheses include that these groups are at greater risk of exposure, that they are more susceptible to pesticide toxicity, or that they are more likely to report exposure and illness or seek medical attention. However, we did not observe consistent patterns among workers in other occupations. This finding requires further research to identify the explanation. The higher incidence in the western states may suggest that workers in this region are at higher risk of drift exposure; however, it may also have resulted from better case identification in California and Washington states through their higher-staffed surveillance programs, extensive use of workers' compensation reports in these states, and use of active surveillance for some large drift events in California.

Nonoccupational exposure. This study found that more than half of drift-related pesticide poisoning cases resulted from nonoccupational exposures and that 61% of these nonoccupational cases were exposed to fumigants. California data suggest that residents in agriculture-intensive regions have a 69 times higher risk of pesticide poisoning from drift exposure compared with other regions. This may reflect California's use of active surveillance for some large drift events. Children had the greatest risk among nonoccupational cases. The reasons for this are not known but may be because children have higher pesticide exposures, greater susceptibility to pesticide toxicity, or because concerned parents are more likely to seek medical attention. Recently several organizations submitted a petition to the U.S. EPA asking the agency to evaluate children's exposure to pesticide drift and adopt interim prohibitions on the use of drift-prone pesticides near homes, schools, and parks (Goldman et al. 2009).

Contributing factors. Soil fumigation was a major cause of large drift events, accounting for the largest proportion of cases. Because of the high volatility of fumigants, specific measures are required to prevent emissions after completion of the application. Given the unique drift risks posed by fumigants, U.S. EPA regulates the drift of fumigants separately from nonfumigant pesticides. The U.S. EPA recently adopted new safety requirements for soil fumigants, which took effect in early 2011 and include comprehensive measures designed to reduce the potential for direct

fumigant exposures; reduce fumigant emissions; improve planning, training, and communications; and promote early detection and appropriate responses to possible future incidents (U.S. EPA 2010). Requirements for buffer zones are also strengthened. For

example, fumigants that generally require a > 300 foot buffer zone are prohibited within 0.25 miles (1,320 feet) of "difficult-to-evacuate" sites (e.g., schools, daycare centers, hospitals). We found that, of the 738 fumigant-related cases with information on

Table 5. Fifteen most common active ingredients for drift cases and percentage of moderate/high severity.

Active ingredient	Functional class	Chemical class	Cases ^a (n = 2,945)	Cases exposed to single active ingredient	
				Total (n = 1,809)	Percent moderate/high severity (n = 90) ^b
Metam-sodium	Fumigant	Dithiocarbamate	664	664	3
Chloropicrin	Fumigant	Trichloronitromethane	637	532	1
Chlorpyrifos	Insecticide	Organophosphate	240	49	10
Sulfur	Insecticide/fungicide	Inorganic compound	147	32	25
Mancozeb	Fungicide	Dithiocarbamate	144	4	0
Methamidophos	Insecticide	Organophosphate	133	0	0
Malathion	Insecticide	Organophosphate	122	96	11
Spinosad	Insecticide	Spinosyn	107	1	0
Methyl bromide	Fumigant	Alkyl bromide	84	11	27
Dimethoate	Insecticide	Organophosphate	68	10	20
Cyfluthrin	Insecticide	Pyrethroid	59	2	0
Methomyl	Insecticide	N-Methyl carbamate	56	13	15
Atrazine	Herbicide	Triazine	54	8	0
λ-Cyhalothrin	Insecticide	Pyrethroid	52	39	3
Propargite	Acaricide/miticide	Sulfite ester	52	10	30

^aCan be exposed to other active ingredients also. ^bHigh, n = 7; moderate, n = 83.

Table 6. Ten largest drift events, 1998–2006.

State	Year	Cases			Pesticide application		
		Total (n = 1,293)	Occupational (n = 452)	Nonoccupational (n = 841)	Target	Equipment	Active ingredient
California	1999	170	6	164	Soil	Chemigation	Metam-sodium
California	2000	33	33	0	Almonds	Aerial application	Chlorpyrifos, propargite
California	2002	250	72	178	Soil	Soil injector	Metam-sodium
California	2002	123	123	0	Soil	Chemigation	Metam-sodium
California	2003	161	10	151	Soil	Soil injector	Chloropicrin
California	2004	122	122	0	Potatoes	Aerial application	Methamidophos
California	2005	324	1	323	Soil	Chemigation	Chloropicrin
California	2005	42	42	0	Soil	Chemigation	Metam-sodium
California	2005	34	34	0	Oranges	Ground sprayer	Cyfluthrin, spinosad
Texas	2005	34	9	25	Cotton	Ground sprayer	λ-Cyhalothrin

Table 7. Factors associated with large drift events (≥ 5 cases).

Factor	Small event (n = 555)	Large event (n = 88)	Large event (vs. small), OR (95% CI)
	n (%)	n (%)	
Pesticide functional class			
Insecticide	172 (31.0)	26 (29.5)	Reference
Fumigant	29 (5.2)	23 (26.1)	5.25 (2.64–10.41)
Multiple combination	178 (32.1)	29 (33.0)	1.08 (0.61–1.91)
Other single pesticide class or unknown	176 (31.7)	10 (11.4)	0.38 (0.18–0.80)
Application target			
Soil	31 (5.6)	24 (27.3)	8.50 (4.57–15.79)
Small fruit crops ^a	38 (6.8)	14 (15.9)	4.04 (2.03–8.06)
Leafy vegetable crops ^b	25 (4.5)	8 (9.1)	3.51 (1.49–8.27)
Other ^c	461 (83.1)	42 (47.7)	Reference
Application method			
Aerial application	223 (40.2)	26 (29.5)	0.91 (0.54–1.53)
Chemigation	20 (3.6)	22 (25.0)	8.58 (4.31–17.09)
Other ^d	312 (56.2)	40 (45.5)	Reference

^aFor example, berries, grapes, currants. ^bFor example, beets, celery, broccoli, lettuce, spinach. ^cIncludes tree fruit or other vegetable crops, other crop categories, landscape and forest, undesired plants, livestock farms, unknown. ^dIncludes other ground application equipment, multiple, and unknown.

distance, 606 (82%) occurred > 0.25 miles from the application site, which suggests that the new buffer zone requirements, independent of other measures to increase safety, may not be sufficient to prevent drift exposure.

This study also shows the need to reinforce compliance with weather-related requirements and drift monitoring activities. Moreover, applicators should be alert and careful, especially when close to nontarget areas such as adjacent fields, houses, and roads. Applicator carelessness contributed to 79 events (48% of 164 events where contributing factors were identified), of which 56 events involved aerial applicators. Aerial application was the most frequent application method found in drift events, accounting for 249 events (39%). Drift hazards from aerial applications have been well documented (CDC 2008; Weppner et al. 2006). Applicators should use all available drift management measures and equipment to reduce drift exposure, including new validated drift reduction technologies as they become available.

Limitations. This study requires cautious interpretation especially for variables with missing data on many cases (e.g., age, violation, contributing factors, distance). This study also has several limitations. First, our findings likely underestimate the actual

magnitude of drift events and cases because case identification principally relies on passive surveillance systems. Such underreporting might have allowed the totals to be appreciably influenced by a handful of California episodes in which active case finding located relatively large numbers of affected people. Pesticide-related illnesses are underreported because of individuals not seeking medical attention (because of limited access to health care or mild illness), misdiagnosis, and health care provider failure to report cases to public health authorities (Calvert et al. 2008). Data from the National Agricultural Workers Survey suggests that the pesticide poisoning rates for agricultural workers may be an order of magnitude higher than those identified by the SENSOR-Pesticides and PISP programs (Calvert et al. 2008). Second, the incidence of drift cases from agricultural applications may have been underestimated by using crude denominators of total population and employment estimates, which may also include those who are not at risk. On the other hand, the incidence for agricultural workers may have been overestimated if the denominator data undercounted undocumented workers. Third, the data may include false-positive cases because clinical findings of pesticide poisoning are nonspecific and diagnostic tests are not

available or rarely performed. Fourth, when we combined data from SENSOR-Pesticides and PISP, some duplication of cases and misclassification of variables may have occurred, although we took steps to identify and resolve discrepancies. Also, SENSOR-Pesticides and PISP may differ in case detection sensitivity because the two programs use slightly different case definitions. Lastly, contributing factor information was not available for 48% of cases, either because an in-depth investigation did not occur or insufficient details were entered into the database. We often based the retrospective coding of contributing factors on limited data, which may have produced some misclassification.

Conclusion

These study findings suggest that the incidence of acute illness from off-target pesticide drift exposure was relatively low during 1998–2006 and that most cases presented with low-severity illness. However, the rate of poisoning from pesticide drift was 69 times higher for residents in five agriculture-intensive California counties compared with other counties, and the rate of occupationally exposed cases was 145 times greater in agricultural workers than in nonagricultural workers. These poisonings may largely be preventable through proper prevention measures and compliance with pesticide regulations. Aerial applications were the most frequent method associated with drift events, and soil fumigations were a major cause of large drift events. These findings highlight areas where interventions to reduce pesticide drift could be focused.

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Table 8. Violation in and contributing factors to occurrence of drift incidents/exposures.

Variable	Drift events (n = 643) n (%)	Drift cases	
		Occupational (n = 1,380) n (%)	Nonoccupational (n = 1,565) n (%)
Violation of federal/state pesticide regulation			
Yes	220 (73.6) ^a	971 (85.6)	1,122 (93.2)
No	79 (26.4)	164 (14.4)	82 (6.8)
Unknown/pending	344	245	361
At least one contributing factor identified ^b	164 (100)	486 (100)	1,058 (100)
Applicator carelessness near nontarget sites ^c	79 (48.2)	49 (10.1)	98 (9.3)
By aerial applicator	56 (34.1)	21 (4.3)	66 (6.2)
Weather (wind, temperature inversion)	75 (45.7)	309 (63.6)	593 (56.0)
Poor/ineffective communication	19 (11.6)	102 (21.0)	11 (1.0)
Improper seal of fumigation site ^d	9 (5.5)	94 (19.3)	837 (79.1)
Inappropriate monitoring ^e	7 (4.3)	118 (24.3)	199 (18.8)
Applicator not properly trained or supervised	5 (3.0)	45 (9.3)	0 (0.0)
Excessive application	4 (2.4)	20 (4.1)	6 (0.6)
Use of inadequate equipment ^f	2 (1.2)	125 (25.7)	2 (0.2)
Other ^g	8 (4.9)	28 (5.8)	206 (19.5)
Distance from application site	NA	700 (100)	728 (100)
≤ 50 feet		66 (9.4)	54 (7.4)
> 50–100 feet		77 (11.0)	29 (4.0)
> 100–300 feet		113 (16.1)	69 (9.5)
> 300 feet–0.25 mile		267 (38.1)	93 (12.8)
> 0.25–0.5 mile		175 (25.0)	256 (35.2)
> 0.5–1 mile ^h		0 (0.0)	116 (15.9)
> 1 mile ⁱ		2 (0.3)	111 (15.2)

NA, for distance from application site, drift events were not applicable. All percentages for "At least one contributing factor identified" and "Distance from application site" were calculated only for cases with available data.

^aThe CDPR identified 159 (72%). ^bCases may have been included in multiple categories. ^cFor example, the applicator did not turn off a nozzle at the end of the row, or the crop duster flew overhead. ^dFor example, leakage from torn tarp, early removal of seal, or use of contaminated water. ^eFor example, did not measure wind speed or did not monitor drift from the application site. ^fFor example, used longer spray boom than specified on the label or used sprinklers without required calibration device. ^gFor example, treated additional rows without permission, permeable soil type, aerial application with very low height, or building/vehicle ventilator system sucking outside air in. ^hCases are from three events in California, Louisiana, and Washington. ⁱCases are from two events in California.

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From: Richard E. Stevenson, Jr. [mailto:richstevenson@Modernpest.com]
Sent: Sunday, February 26, 2012 9:43 PM
To: Jennings, Henry
Subject: Fwd: From February 25 Southern Ohio Fox Affiliate-Family Displaced After Bed Bug Control Efforts Go Awry

For distribution...

Begin forwarded message:

From: Bob Rosenberg <rosenberg@pestworld.org>
Date: February 26, 2012 5:55:27 PM EST
To: Bob Rosenberg <rosenberg@pestworld.org>
Subject: From February 25 Southern Ohio Fox Affiliate-Family Displaced After Bed Bug Control Efforts Go Awry

to: NPMA Bed Bug Division, et. al.

Family of 6 displaced after bedbug killing chemicals and smoking start fire



Four children and two adults are without a home after improper use of chemicals to kill bedbugs ignited after someone was smoking in the room.

Colerain Firefighters responded to a fire in a second floor apartment in the 3500 block of West Galbraith Road around 9:30 Saturday night.

The fire started when someone was smoking in a room which had been sprayed with Isopropyl Alcohol to exterminate bedbugs.

Isopropyl Alcohol is an effective home-remedy to kill bedbugs, but due to its volatile nature is extremely flammable, and even more flammable when using a spray bottle. Smoking in the area is extremely hazardous in this situation.

No firefighters or residents were injured during the fire. A damage estimate has not been determined; however the apartment and structure suffered moderate damage but will be able to be repaired. The family is receiving assistance from the Red Cross.

Springfield Township and Green Township Fire Departments also helped putting out the fire.

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News — April 24, 2012 6:04 am

Bug sensor earns UCR professor \$300,000 prize

Written by [Vy Nguyen](#)

Courtesy of UCR Today

Computer Science and Engineering Professor Eamonn Keogh has been awarded \$300,000 to help support his creation of a wireless bug sensor that can help protect food crops from natural pests and insects. Keogh won the first place prize in a competition hosted by the Vodafone Americas Foundation Wireless Innovation Project. The sensor is able to detect and classify any insect that flies through an opening and will then transmit data to farmers who can determine where to concentrate their pesticide treatments.

The sensors operate by detecting the speed and wing beats of flying insects, which in turn reveal the species and gender of the insect. "This method allows farmers a more targeted approach than mass intervention, reducing costs for labor and pesticides," stated the Vodafone Americas Foundation's website.

Keogh has noted that wide-scale implementation of the sensors can keep track of massive amounts of data and reveal trends in the movement patterns of insects. "Given the importance of insects in human affairs, it is somewhat surprising that computer science has not had a larger impact in entomology.

About five years ago, I decided that someone in computer science needed to lead the charge, and to take the power of computer science to entomology," stated Keogh in an interview with the Highlander.

Keogh hopes that his team's research will help shine light on the issue of insect invasion on healthy food crops—a matter that many farmers around the world must deal with every day. Prosperous places like the United States have been depending on the use of pesticides to keep the pest population under control; however, many developing countries cannot afford to use pesticides on their crops. As such, the cost-saving potential of the sensors could help promote more successful farming in developing countries where farmers would benefit the most from the technology.

Daniel Liao, a UCR alumnus in mechanical engineering, expressed his intrigue with the invention but identified an area of concern: the small triangular opening in which insects must pass through in order to be detected. "In order for the device to detect the species of the insects, the insects must go through the opening. The downside to that is the price in order to produce a large enough device to cover the many acres of crops," stated Liao.

Keogh admits that perfecting the wireless bug sensor will not be an easy task. "We are building simple low cost sensors [so] we can get accurate counts of flying insects in real-time. This information can be used by health care workers to plan interventions to kill mosquitoes (for malaria), or by farmers to control crop pests (for agriculture)," noted Keogh. "This is a very hard problem; there are 3,528 different species of mosquitoes alone. Only some moths cause problems for agriculture, but there are 150-250,000 species of moths."

The weeks leading up to Vodafone's phone call were a difficult and exciting time for Keogh, who stated that he would often look at his office phone in anxiety. "I spend the full day in my office trying to work, but really looking at the phone out of the corner of my eye. At about 4:00 p.m. the phone rang, and they told me I won. I tried to play it cool on the phone, but as soon as I hung up, I shouted 'yes!' so loud [that] people in adjacent offices came running to see what was happening," said Keogh.

Keogh and his fellow award winners convened at Washington D.C. last week to receive their awards at the Global Philanthropy Forum.

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Clues to Species Decline Buried in Pile of Bird Excrement

by Helen Fields on 17 April 2012, 7:01 PM | [2 Comments](#)

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In 2009, while searching for ways to help endangered birds, research technician Chris Grooms heard that a chimney on his university campus used to host a migratory species known as the chimney swift. When he investigated, he found a pile of bird excrement 2 meters deep. The poop lay at the bottom of a five-story-high chimney and had been deposited over 48 years by the birds, which had roosted there until the top was capped in 1992. Now, Grooms and his colleagues have dug into that pile of guano, revealing new clues about why the chimney swift and other species like it have begun to disappear.

Grooms volunteers for an environmental group in Ontario, Canada, that's trying to conserve local wildlife. He also works in a lab at Queen's University in Kingston that studies sediments in lakes. As dirt and dead things sink to the bottom of these bodies of water, they preserve a record of environmental conditions. Grooms wondered if the same thing had happened with his pile of bird poop. He brought the idea to the head of the lab, ecologist John Smol. Smol was intrigued: "It could be 2 meters of bird poop, or it could be a pretty important story."

The researchers entered the chimney through a little door near the bottom that was only big enough to crawl through. Behind the door was the wall of poop. It took 2 days to dig out enough of the crumbly, dark-gray, dry excrement so that the researchers could stand up. After 20 years, the poop had lost its smell, but the researchers wore respirators just in case some pathogen was hanging around.

With the help of [radioisotopes produced by nuclear bomb tests](#), which linger in sediments and can be used for dating, the researchers worked out that the deposit built up between 1944 and 1992. A team at the University of Ottawa measured levels of DDE, a chemical that comes from the pesticide DDT, to see if DDT affected what insects the birds were eating. Another set of samples went off to Joseph Nocera, a conservation biologist at the Ontario Ministry of Natural Resources in Peterborough, who sorted out insect remains. Most were beetles; the next most common remains were from the Hemiptera, an order known as "true bugs" that includes stink bugs and cicadas.

As DDE increased through the lower layers of the deposit, [beetles showed up less often in the birds' diets and true bugs became more common](#), the researchers report online today in the *Proceedings of the Royal Society B*. This result agrees with other reports that DDT is hard on beetles, while true bugs can evolve resistance quickly. The change in diet may also help explain why chimney swifts have declined so precipitously, Nocera says.

Canadian surveys have found that the number of chimney swifts dropped 95% between 1968 and 2005. Some researchers have suggested that part of the reason is that chimneys like this one, swifts' preferred habitat, have been capped or redesigned, making it harder for birds to get in. But the new work suggests that the decline may be diet related. Beetles generally contain more calories than do true bugs. Swifts need a ton of energy—they spend a lot of time on the wing, looking for food. A change in their diet, like substituting less-nutritious true bugs, could have

[ENLARGE IMAGE](#)



Hidden treasure. Chimney swifts (bottom left) roosted in this chimney until it was capped in 1992; researchers dug out their poop and studied the hard remains of insects they'd eaten (top right).

Credit: Chris Grooms/Queen's University; (bird, left inset) Bruce Di Labio

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a big impact. DDT was banned in the 1970s, but the beetles never seem to have gotten back their original place in the food web, Nocera says.

Nocera thinks DDT and other pesticides may have effects far beyond their well-known impacts on the eggshells of large birds, such as taking away the foods that chimney swifts, barn swallows, flycatchers, and other insect-eating birds relied on. He says he doesn't know of any other studies that have looked at a pile of bird poop on the scale of decades—other studies have looked at older guano. There are probably many more archives like this in the chimney swift's range, he says, and this study shows that it's possible to get useful information out of them.

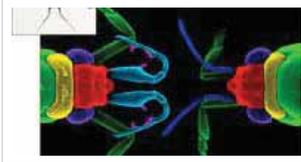
Çağan Şekercioğlu, a conservation ecologist at the University of Utah in Salt Lake City, agrees with the team's conclusions. Pesticides get more concentrated as they move up the food chain, which means they can be worse for insect-eating birds than for birds that eat fruit or nectar, he says. Still, Şekercioğlu says he would have liked to see more discussion of how the loss of nesting and roosting sites—like the chimney in this study—affected chimney swift populations. But "it's a very good historical data set," he says. "We don't have that opportunity for almost any other bird species. It's a brilliant idea and very well thought out, and the fact that they found this potential link to DDT is fascinating."

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Not Exactly Rocket Science

Scientists crawl into tower of poo to understand reasons for swift decline



For some scientists, an academic career can feel like crawling into a tower of crap. For other scientists, an academic career actually *involves* crawling into a tower of crap.

Since 1928, thousands of chimney swifts have roosted at Fleming Hall, a university building in Kingston, Ontario. For decades, they fed on local insects, and excreted the remains down one of the building's chimneys. Around 2 centimetres of droppings, or 'guano', built up every year until the chimney was finally capped in 1992. To this date, Fleming Hall contains a hardened guano tower, two metres tall and 64 years in the making, which preserves a layered record of the swifts' meals.

Now, a team of scientists, led by Joseph Nocera, have used this archive of historical poo to explain why the swift populations have fallen by 90 per cent since their heyday.

The guano tower was discovered by Chris Grooms from the Kingston Field Naturalists, who brought it to the team's attention. They reached it via a 2-foot-wide square door at the bottom of the chimney, and found a two-metre-tall column. "One has to be somewhat of a contortionist to get in," says Nocera. "The guano is compacted and very dry, like a popcorn-cake. It has a slightly musty smell, and the area is very dusty. Overall, it's not a terribly comfortable place to work!"

The team cut slices down the entire length of the column. They studied the insect remains within it, the levels of different chemical elements, and the amounts of pesticides such as DDT.

The insect shells revealed that, during the 1940s, the swifts were mostly eating beetles. As the 50s came around, they shifted towards 'true bugs' (a term referring to a specific group of insects, rather than 'bugs' in general). This coincides with the introduction of DDT, which hit beetles more than many other insect

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Ed Yong is an **award-winning** British science writer. His work has appeared in New Scientist, the Times, WIRED, the Guardian, Nature and more. Not Exactly Rocket Science is his attempt to talk about the awe-inspiring, beautiful and quirky world of science to as many people as possible.

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groups. Starved of their main prey, the swifts turned to bugs, which are more resilient to DDT sprays and quicker to evolve resistance.

Once the use of DDT started to decline, the beetles rebounded and regained their prime position on the swifts' menu. The use of the pesticide reached its nadir in the 1970s, when it was banned from agricultural use under the Stockholm Convention. However, DDT is still used in countries that didn't sign up to the convention, and more broadly to control malarial insects.

In fact, Nocera's study shows that DDT levels have risen slightly since the 1970s, possibly because of this background use and the pesticide's infamous ability to persist in the environment. And, at the same time, the swifts made yet dietary shift from beetles to bugs.

Nocera thinks that these changing diets were important for the swifts. Bugs make for harder meals because they have a greater range of chemical defences, and they provide fewer calories than beetles. "It could take a lot of small bugs to equal the content of catching one large beetle," he says. "Chimney swifts spend most of the day in flight and are on tight energy budgets. Any disruption to that would result in negative consequences, such as fewer resources to successfully rear chicks. The dietary change we observed was likely a trigger of swift population declines."

That might explain why the swifts started to disappear, but Nocera thinks that other factors helped to perpetuate the decline, including changing climate. It could also be that the communities of insects that feed the swifts have permanently changed as a result of the early DDT wave.

To test that, Nocera's team is planning to analyse the DNA of the guano tower's insect remains, to identify the species that the swifts were eating. "We want to test whether the most common prey items in recent years are the same as the most common prey items in previous years," he says.

They are also going to study guano columns from other chimneys around North America, to see if the fate of the Queen's University swifts represents continent-wide on changes. They already have samples from places in Quebec, Manitoba and Connecticut.

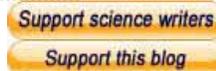
Reference: Nocera, Blais, Beresford, Finity, Grooms, Kimpe, Kyser, Michelutti, Reudink & Smol. 2012. Historical pesticide applications coincided with an altered diet of aerially foraging insectivorous chimney swifts. Proc Roy Soc B. <http://dx.doi.org/10.1098/rspb.2012.0445>



April 17th, 2012 by Ed Yong in [Animal behaviour](#), [Animals](#), [Birds](#), [Conservation](#), [Environment](#), [Select](#) | [5 comments](#) | [RSS feed](#) | [Trackback >](#)



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March 5, 2012

To: Dr. Steven Bradbury
U.S. Environmental Protection Agency
Office of Pesticide Programs
Mail Code 7501P
Ariel Rios Building
1200 Pennsylvania Avenue NW
Washington, D.C. 20460-0001

From: 22 Members and Participants of North Central Coordinating Committee
NCCC46 and Other Corn Entomologists:
Patrick Porter, Past Chair, Texas AgriLife Extension, Texas A&M University
Eileen Cullen, Chair, University of Wisconsin
Thomas Sappington, Chair-elect, USDA-Agricultural Research Service
Arthur Schaafsma, Secretary-Treasurer, University of Guelph
Steve Pueppke, Administrative Advisor to NC-205 and NCCC46, Michigan State
University
David Andow, University of Minnesota
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Larry Buschman, Kansas State University
Yasmin J. Cardoza, North Carolina State University
Christina DiFonzo, Michigan State University
B. Wade French, USDA-Agricultural Research Service
Aaron Gassmann,, Iowa State University
Michael E. Gray, University of Illinois
Ronald B. Hammond, The Ohio State University
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Jonathan G. Lundgren, USDA-Agricultural Research Service
Kenneth R. Ostlie, University of Minnesota
Elson Shields, Cornell University
Joseph L. Spencer, University of Illinois
John F. Tooker, Pennsylvania State University
Roger R. Youngman, Virginia Tech

We write as a group of public-sector corn entomologists to provide commentary about western corn rootworm (*Diabrotica virgifera virgifera*) resistance to Cry3Bb1, specifically the repeated reports of “greater than expected damage” to Cry3Bb1 rootworm-protected transgenic corn. We are troubled about the immediate implications of these observations for the durability of pyramid toxin rootworm-protected corn, as well as their potential long-term impact on corn production. This letter articulates our concerns about Cry3Bb1 resistance and then assesses the issue of resistance to Bt in the context of integrated pest management (IPM) of corn rootworm.

Executive Summary

Bt technology has been valuable in terms of reducing insecticide use and increasing farm income. The first documented case of field-evolved resistance to a Bt transgenic hybrid in the continental U.S. provides an opportunity to assess and respond to the current situation, one that should be acted upon carefully, but with a sense of some urgency. On-farm planting and other rootworm management decisions will alter the future course of resistance evolution, and we believe it is critical for industry, regulatory agencies and university and government scientists to work together to provide science-based, practical information to corn growers, consultants and the agricultural industry.

Likely contributing factors to the problem include: the widespread use of Bt corn hybrids (or Bt corn + insecticide) where it is not economically justified, the repeated deployment of hybrids expressing the same toxin in the same fields year after year, violation of stewardship requirements for refuges, and decreased options to employ alternative forms of pest management. Effective long-term corn rootworm management and sustainable use of Bt hybrid technology require an integrated approach that is not overly reliant on any single tactic.

Experience with commercialized rootworm-protected transgenic corn

Rootworm-protected transgenic corn hybrids were initially marketed to prevent economic yield loss while simultaneously reducing the use of broad-spectrum soil insecticides. They have proven to be an effective and environmentally responsible means of controlling corn rootworm.

Hybrids expressing the first rootworm Bt, Cry3Bb1 (trade named YieldGard® RW), were commercially planted in 2003, followed by those expressing Cry34/35Ab1 (trade named Herculex® RW) in 2005 and mCry3A (trade named Agrisure® RW) in 2006. All of these hybrids had a required 20% non-Bt refuge. More recently, hybrids with pyramids of rootworm traits Cry3Bb1 + Cry34/35Ab1 (trade named SmartStax®) and mCry3A + Cry34/35Ab1 (trade named Agrisure 3122™ Refuge Renew) were approved with a reduced refuge size. Delivery of the refuge “in the bag” had previously been approved for Cry34/35Ab1 (trade named Optimum AcreMax™ RW) at 10% refuge and is now approved for Cry3Bb1 + Cry34/35Ab1 at 5% refuge.

Convergence of evidence on field-evolved resistance

Greater than expected damage to Bt corn hybrids expressing the Cry3Bb1 protein was first observed across a wide geographic area during the 2009 growing season. By 2011, problem areas had been reported in northwestern and north-central Illinois, northeastern Iowa, southern Minnesota, northeastern Nebraska, and eastern South Dakota. Common features of affected fields in these areas included a history of continuous planting to corn and the use of Cry3Bb1-expressing hybrids for multiple years.

The first published report of field-evolved resistance by western corn rootworm to a Bt toxin, Cry3Bb1, also appeared in print in 2011. In this peer-reviewed paper, Gassmann et al. (2011) confirmed rootworm resistance to Cry3Bb1 corn and demonstrated that this was not accompanied by an increase in tolerance to Cry34/35Ab1 corn.

The circumstances surrounding the appearance of field-evolved resistance and its documentation by Gassmann et al. (2011) are consistent with laboratory selection studies, which revealed rapid evolution of resistance to Cry3Bb1 in nine of nine experiments (Meihls et al. 2008, Meihls 2010, Oswald et al. 2011). All available evidence thus converges in implicating field-evolved resistance to Cry3Bb1 as the most likely cause of “greater than expected damage” in rootworm problem fields.

Resistance to Cry3Bb1 threatens hybrids carrying two toxins

Confirmation of Cry3Bb1 resistance in field populations of western corn rootworm raises deep concerns about the durability of the Cry3Bb1 + Cry34/35Ab1 toxin pyramid in SmartStax hybrids. The appearance of Cry3Bb1 resistance is particularly troubling given the decreased non-Bt refuge requirements (from 20% to 5%) for these hybrids.

Reduction in refuge size was, of course, predicated on the effectiveness of both toxins against corn rootworm. Under these conditions, evolution of resistance to either single toxin would be slowed in fields having doubly susceptible insects. But this assumption is no longer valid in problem areas such as those described above, which are characterized by reduced efficacy of Cry3Bb1. Here Cry34/35Ab1 receives only partial protection from Cry3Bb1 and is vulnerable to insects quickly evolving resistance, especially with only a 5% refuge.

Continued reliance on smaller refuges in conjunction with pyramids planted in problem areas may slow the spread of Cry3Bb1 resistance into susceptible areas. This would occur because smaller refuge size reduces the total number of rootworms carrying resistance alleles. Thus, the Cry34/35Ab1 toxin serves to decrease the size of the local population where Cry3Bb1 resistance is building, in the process limiting the total number of Cry3Bb1 resistance alleles that can be spread by emigrating beetles.

However, use of the smaller refuge size in problem areas to slow resistance evolution to Cry3Bb1 would likely have the opposite effect of hastening evolution of

resistance to Cry34/35Ab1. This is highly undesirable, because it would compromise the durability of the Cry34/35Ab1 proteins in both current and future non-pyramided and pyramided Bt corn hybrids. We strongly recommend that this possibility be taken into account in determination of the appropriate refuge size for SmartStax corn in problem areas of resistance to Cry3Bb1. It is crucial that susceptibility to Cry34/35Ab1 be preserved, in part because it has now been approved in pyramid with mCry3A and is the common toxin in two different pyramids from two registrants. A third registrant is also seeking to register mCry3A+Cry34/35Ab1.

The ultimate impact of increasing the SmartStax refuge requirement in problem areas hinges on allele frequency and fitness costs, and more research is required before we can assess the impact. If the Cry3Bb1 resistance allele frequency is low and fitness costs are high, then planting a larger refuge would likely manage both Cry3Bb1 resistance as well as delay Cry34/35Ab1 resistance evolution. However, if the frequency of Cry3Bb1 resistance alleles is high and fitness costs are low, then planting a larger refuge in problem areas could lead to a population density increase of Cry3Bb1 resistant insects over time, because resistant insects are not exposed to Cry34/35Ab1 on non-Bt plants. This in turn would accelerate spread of local Cry3Bb1 resistance. Additionally, in this scenario if there is cross resistance to Cry3Bb1 and mCry3A then resistance to mCry3A could be accelerated.

What have we learned and what can be done to protect future transgenics

The appearance of resistance to Cry3Bb1 corn has reinforced some of our underlying concerns about the role of Bt hybrids in IPM of corn rootworm. Rootworm-protected transgenic corn was introduced into an existing IPM-based system that promoted multiple practices to control the insect. These include crop rotation, scouting, and application of insecticides when and where necessary.

Although many factors come into play, the always “on” nature of transgenic toxins means they cannot be deployed or withdrawn in response to changing pest densities. Selection for resistance thus occurs wherever Bt corn is grown and susceptible insects are present. The response to this selection depends on many factors including resistance allele frequency, fitness costs, toxin dose etc. Avoidance of undue resistance risk forms the rationale for Insect Resistance Management (IRM) plans that are required for Bt crops. The current toxins deployed for control of corn rootworm are considered low to moderate dose, and it is more difficult to prevent resistance to low to moderate dose toxins than it is to the high dose Bt toxins deployed against Lepidoptera. For some Lepidoptera, especially the stalk borers European corn borer and southwestern corn borer, the toxins in Bt corn are truly high dose. However, they are less than high dose for other key Lepidoptera species like fall armyworm, corn earworm and western bean cutworm.

- *Rotate Bt corn hybrids to expose rootworms to different Bt toxins*

An essential component of IPM as practiced with conventional insecticides is the alternation of modes of action to avoid repeated selection, and a fundamental

principle of resistance management is to cease use of an insecticide if resistance is developing. Rotation of toxins in hybrids as a strategy has been neglected as transgenic corn acreage has increased across the Corn Belt. In areas with significant corn rootworm pressure, hybrids expressing the same toxin(s) are often planted in the same field year after year. This practice is not a sound component of effective IPM.

- *Plant non-Bt corn and avoid prophylactic planting of Bt corn*

As described above, substitution of Cry34/35Ab1 or Cry3Bb1 + Cry34/35Ab1 hybrids for Cry3Bb1 hybrids may slow evolution of resistance to Cry3Bb1. Unfortunately, this could actually accelerate resistance development to Cry34/35Ab1, especially with reduced refuge. In short, planting more of a failing toxin and/or more of an effective toxin over a larger area poses significant risk.

Rootworm-protected Bt corn is being used prophylactically in areas with little or no need for it. This unwarranted use occurs in part because genes to produce rootworm Bt toxins (and toxins active against Lepidoptera) are incorporated into elite germplasm with the highest yield potential. Thus growers often have few options other than to plant stacks and pyramids if they wish to use the hybrids with best yield potential. When growers do not want to use Bt corn, many report increasing difficulty in obtaining non-transgenic seed. Scarcity of non-Bt seed may become more acute as the seed industry transitions to a refuge “in the bag” approach for resistance management.

Planting non-Bt corn can be profitable and should be one of the IPM tools to maintain susceptibility to rootworm-protected transgenic corn. After all, whether used in conjunction with soil-applied insecticides or not, conventional hybrids cause no selection for resistance to any Bt toxin. It is ironic that the decreasing availability of non-Bt hybrids erodes the ability of producers to move to a more integrated system of corn rootworm management, one that protects the value of Bt hybrids. As a component of effective IPM for corn rootworms, attention should be given to increasing the supply of elite hybrids that do not contain Bt.

- *Bt Resistance has real economic and environmental costs*

IPM emphasizes a minimal environmental footprint that is consistent with grower profitability, and it is crucial to note that resistance to Cry3Bb1 corn threatens both. Reduction in the use of broad-spectrum insecticides has been asserted to be one benefit of the registration of Bt rootworm corn. Yet in problem areas where Cry3Bb1 resistance is present, the management plan proposed by the registrant recommends the use of soil insecticides against larvae and/or foliar insecticide sprays against adult beetles in conjunction with Bt corn hybrids.

We can envision that multiple approaches might be necessary under special circumstances if, for example, growers in problem areas have purchased Cry3Bb1 seed for the coming season. But in general, treatment of rootworm protected transgenic corn with insecticides is not a recommended control strategy. It elevates

production costs, reduces profits, selects for resistance to the insecticides, and masks the geographic extent and in-field severity of Cry3Bb1 resistance.

Conclusions

The widespread recommendations to apply insecticides to protect transgenic Bt corn rootworm corn strikes us as a clear admission that the Cry3Bb1 toxin is no longer providing control adequate to protect yield, and that economic value derived from the toxin is declining. Pyramided Cry3Bb1 + Cry 34/35Ab1 corn should not need insecticidal protection to protect yield given that the Cry34/35Ab1 toxin is still effective. Any insecticide use would therefore be for preservation of rootworm susceptibility to the toxins. It is unfortunate that the widespread adoption of transgenic technology has now left many growers without the equipment necessary to apply soil insecticides if needed. Similarly, Cry34/35Ab1 corn should not need insecticide other than as protection for the 20 percent refuge in areas with extreme rootworm populations.

Finally, we note that there is an escalating use of insecticides directed at western and northern corn rootworm in areas of the Corn Belt where rootworm densities are low and the likelihood of economic injury is minimal. When insecticides overlay transgenic technology, the economic and environmental advantages of rootworm-protected corn quickly disappear. We are concerned that high commodity prices and other factors may have fueled an insurance-based approach to corn rootworm management, one that violates many tenets of IPM and that will only increase insect resistance development in the long term.

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Signatures follow on the next page.

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EPA Denies Petition on 2,4-D Pesticide

For Release: April 9, 2012

New! [FR Notice Published](#) - April 18, 2012

In a petition filed on November 6, 2008, the Natural Resources Defense Council (NRDC) requested that EPA cancel all product registrations and revoke all tolerances (legal residue limits in food) for the pesticide 2,4-dichlorophenoxyacetic acid, or 2,4-D. After considering public comment received on the petition and all the available studies, EPA is denying the request to revoke all tolerances and the request to cancel all registrations.

By way of background, in 2005, as part of the regulatory process to ensure pesticides meet current regulatory standards, EPA completed a review on the registration and on the safety of the tolerances for 2,4-D. EPA determined that all products containing 2,4-D are eligible for reregistration, provided certain changes were incorporated into the labels and additional data were generated and submitted to the EPA for review.

During the recent review of the petition from NRDC to revoke the tolerances, EPA evaluated all the data cited by NRDC and new studies submitted to EPA in response to the reregistration decision. Included in the new studies is a state-of-the-science extended one-generation reproduction study. That study provides an in-depth examination of 2,4-D's potential for endocrine disruptor, neurotoxic, and immunotoxic effects. This study and EPA's comprehensive review confirmed EPA's previous finding that the 2,4-D tolerances are safe.

EPA also carefully reviewed NRDC's request that the Agency cancel all 2,4-D product registrations. Based on studies addressing endocrine effects on wildlife species and the adequacy of personal protective equipment for workers, the Agency concluded that the science behind our current ecological and worker risk assessments for 2,4-D is sound and there is no basis to change the registrations.

2,4-D is a phenoxy herbicide and plant growth regulator that has been used in the U.S. since the 1940s. It is currently found in approximately 600 products registered for agricultural, residential, industrial, and aquatic uses. There are 85 tolerances for 2,4-D. EPA published the NRDC petition for public comment on December 24, 2008.

Below are EPA documents responding to NRDC's petition on 2,4-D including a pre-publication copy of the agency's Federal Register Order. These documents are also available on EPA's website at www.epa.gov/pesticides. A 60-day period for filing objections and requests for a hearing on the Order runs from the date of publication in the Federal Register Notice [EPA-HQ-OPP-2008-0877](#).

Related Documents:

- [April 18, 2012, FR Notice: 2,4-D; Order Denying NRDC's Petition to Revoke Tolerances](#)
- [April 7, 2012, Letter to NRDC: EPA Denial of November 6, 2008 NRDC Petition to Cancel All 2,4-D Registrations](#)
- [April 7, 2012, Memorandum to Public Docket: EPA Response to Issues Raised in Public Comments, but Unrelated to Issues in NRDC 2,4-D Petition](#)

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http://www.epa.gov/oppfead1/cb/csb_page/updates/2012/2-4d-petition.html

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Wind tunnel drawing a lot of attention

By **HEATHER JOHNSON**

hjohnson@nptelegraph.com | Posted: Friday, April 27, 2012 12:00 am

A new tool is causing quite the stir at the West Central Research and Extension Center in North Platte. The first of two wind tunnels is up and running, and it appears that everyone wants to see it.

"We've had several hundred people go through here since we started using it in February," said Greg Kruger, WCREC cropping systems specialist. "We've had over 100 people in the last week."

Located in an old hog barn, the tunnel is used to study how wind affects chemical spray applications in farmers' fields. The goal is to try to reduce pesticide drift, and thereby reduce human health risks, lawsuits, waste and contamination of adjacent crops among other concerns.

The influence that different pressures, nozzles and solutions have on droplet size are all taken into consideration during the testing. According to Kruger, the smaller the droplet, the greater the drifting potential. The more pressure that's applied, the smaller the droplet will be.

During the tests, a fan at one end of the 48-foot long Plexiglass tunnel forces wind through a "honeycomb," which makes the air travel in a straight line. At the opposite end of the tunnel is a single-nozzle sprayer that emits the various chemicals.

The droplets are blown in front of a laser, which automatically records their sizes and charts them on a line graph in a nearby computer. A scrubber system pulls the particles and air out of the tunnel after each test, so subsequent ones aren't affected.

The tunnel is designed to mimic ground applications and is capable of producing winds up to 30 miles per hour. Another shorter tunnel is expected to be ready in a couple of weeks that will be able to create winds over 200 miles per hour. Much of the equipment is interchangeable for both tunnels.

"We saved a lot of money that way," said Kruger. "We've got almost \$1 million in this project as it is." According to him, funding came in several forms, including a loan, grants and donations from private companies.

"We do grant research, but we also do a lot of contract work for pesticide companies and nozzle manufacturers," he said. Tests will be conducted year round.

The goal is to take the information gathered from the studies and make it accessible to farmers instantaneously via an iPhone application. That capability is still a few months out.

The tunnels are the second of their kind in the U.S. and only the fourth of their kind in the world.



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Tics and Toxins: Pesticide Ban, Rainfall Could Point to Poisonous Fungus as Factor in Student Outbreaks



By Dan Olmsted and Mark Blaxill

LEROY, N.Y., February 6 -- Last year, during the wettest spring ever recorded across large swaths of New York state, a little-noticed law took effect: As of May 18, pesticide use was banned from the grounds of every school in the state. That same month, a girl at the junior/senior high school here, and another at a high school near Albany, developed a mysterious tic disorder. The total number of cases in LeRoy has now risen to 15.

This convergence adds a new possibility to the list of suspects already being scrutinized in this picturesque Western New York village of 4,400, suspects that range from a 1970 train derailment that spewed toxic chemicals, to an autoimmune disorder called PANDAS, to leaks from gas wells on school grounds that may or may not have employed "fracking." The new possibility: Poisoning from a fungus that grows on a grass commonly planted on school grounds.

The fungus is called ergot, and it can grow when ryegrass – used on most athletic playing fields – sprouts a floweret that gets infected. That most often happens during wet spring months and on low-lying or marshy areas. (This photo was taken on school grounds last week.)

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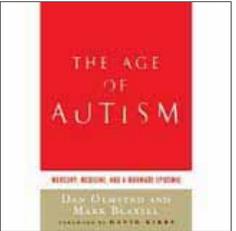
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Two other tic cases have been reported in girls who attend Corinth High School, north of Albany. Both are members of the school softball team; the first girl collapsed unconscious in May during the first inning of a softball game and began twitching and convulsing, according to the Albany Times-Union; in LeRoy, at least 6 of the first 12 cases were among athletically active girls – four cheerleaders and two members of the soccer team.

And as we have reported, a 35-year-old man in the village of Bath, about 70 miles from LeRoy, was stricken with the same symptoms in September. He lives close to a field that was planted last spring in rye and not harvested; there is a swamp and a levee nearby; and his water comes from a well in his yard. (This is a photo of the swamp.)



In Corinth, the first girl was affected in May; according to a report from the New York State Department of Health released Friday, the first case in LeRoy was also in May, followed by three more cases in weeks that began in September, two cases in October, one case in November, and one case in December. The state report dismissed environmental or infectious factors and embraced the official diagnosis of "conversion disorder," in which stress or trauma are subconsciously converted into physical symptoms (several cases at once is called "a mass psychogenic event").

The report was released a day ahead of a community meeting Saturday at the high school, but did little to assuage community concerns.

At the meeting, Superintendent Kim Cox attempted to reassure parents that the school is safe, even as she said more environmental testing would be done on air quality. Some residents, including parents of stricken girls, said the school had not done enough to rule out environmental hazards on the school grounds.

In response to a question about an orange colored substance that has oozed out of the ground and gotten on some students' shoes and clothing, officials described it as a "harmless" and "nontoxic" rust fungus that grows on grasses.

Our attention to the possibility of ergot poisoning evolved from a discussion with Bryan Tremblay, the man in Bath, about 70 miles away from LeRoy, who was struck in September with a similar affliction. The farm field behind his house, normally planted with corn, was planted in rye last spring, left to lie fallow, and not harvested this fall. His water came from a well in his backyard.

As we walked across the field, Tremblay, a history buff, remarked that some historians believe the women accused in the Salem witch trials may actually have been victims of ergot

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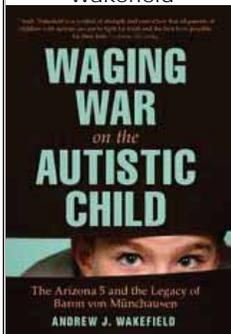
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poisoning. That made us curious and we decided to look deeper. We learned:

- Ergot poisoning, or ergotism, is caused by toxic excretions from a common fungus
- The so-called ergot fungi (any species from the genus *claviceps*) that cause ergotism grow most commonly on rye and ryegrasses
- Ryegrass is widely used on school athletic fields
- When ergot fungi infect a plant, they produce a growth called a sclerotium that contains spores as well as toxic alkaloid compounds
- When eaten, these toxic alkaloids are known to produce severe neurological symptoms, including twitches, seizures, headaches and trouble walking
- The onset of spring and rainy weather causes the sclerotium to germinate, release its spores and spread the ergot infection to other grasses
- In especially rainy conditions, these sclerotia can be infected by another type of fungus called rust
- One common type of rust known to infect ergot fungi, *fusarium*, can take on an orange color
- The sclerotium of an ergot fungus, although typically not orange, can appear in many colors as well

So when school officials dismiss the orange substance on the school grounds as a "a form of nontoxic rust fungi," they may be overlooking an important clue to a potentially toxic exposure. Alternatively, infected ryegrass could be located elsewhere at the school, including the marshy areas we've described, or rye could be grown on nearby farm fields.

The law that banned all pesticide use at schools was passed in 2010. It took effect that year for daycare sites, and in May 2011 for schools statewide. As summarized in a Cornell University publication: "Pesticides are substances intended to prevent, destroy, repel or mitigate pests and any substance or mixture of substances intended as a plant growth regulator, defoliant or desiccant. They include insecticides, fungicides, herbicides and plant growth regulators. All are banned by this law for use on grounds at schools ..."

Pesticide logs we obtained from the LeRoy Central School District under a Freedom of Information request show the only pesticides applied at the school last year were ant and wasp sprays from a can, allowed under the new law.

Because school officials have declined to talk to us, permitted no independent testing, and released no results of any tests outside the school building, there is much we don't know. We don't know if ryegrass is actually used at the school (it would be unusual if it were not). We don't know if a harmful fungus actually developed in the grass. We don't know how students might have been exposed or why only girls appear to be affected. We haven't found any reports in the medical literature of ergot poisoning from contact with ryegrass at a school.

We also don't know if there is evidence for the school's assurance that the orange ooze is "harmless." The matter is not mentioned in the state report Friday, though it did describe tests of water inside the building and at the junction connecting it to the Monroe County water supply that serves the village (the water is safe, according to the tests). In fact, there is still no evidence that officials have tested anything at all on the school grounds.

At the Saturday meeting, residents pressed for soil testing, but officials said they first wanted to retest air. It could take three weeks to do that and receive results; residents wanted to know why soil testing couldn't begin now.

As we've reported, epic rains occurred in New York state last year, including the wettest spring on record in Buffalo and the second-wettest in Rochester (LeRoy is located between the two). Albany, which is near Corinth, and Binghamton, near Bath, also had massive downpours in 2011 including rainfall from Hurricanes Irene and Lee.

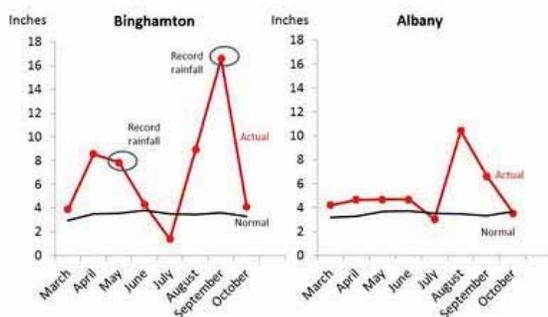
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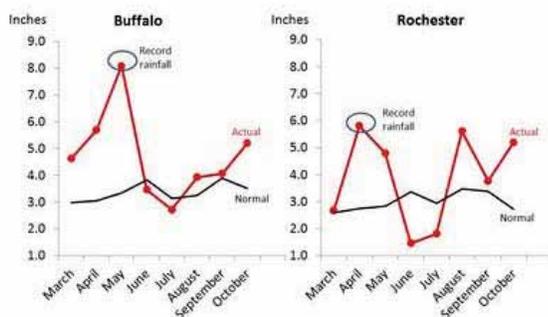
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Heavy rains in Upstate NY from March to October 2011



Heavy rains in Upstate NY from March to October 2011



Another LeRoy resident has come forward to say she also developed severe tics in October that were also diagnosed as conversion disorder. The woman, Marge Fitzsimmons, 36, has no connection to the school. Her address puts her home next to a farm field in the Town of LeRoy just outside the village, with a small creek apparently on the other side of the property. The LeRoy school is also just outside the village boundary.

Famed environmental activist Erin Brockovich stirred controversy a week ago when she sent a representative, accompanied by media, to the school to attempt to take soil samples from playing fields. School officials called the event a publicity stunt and "criminal," had him escorted off school grounds, and then padlocked gates to the playing fields. If you were at the bar last week at Larry's Steak House in nearby Batavia, the Genesee County seat, you would have heard complaints about Brokovich but also suspicions about why the school district wouldn't welcome outside help – often from the same person.

Brockovich and others have pointed to a train derailment a few miles from the school in 1970 as a likely cause of the outbreak. The derailment spewed cyanide and a toxic manufacturing chemical called TCE into the ground. Officials acknowledge that gravel from a quarry near the derailment was used as fill at the school but say it is not toxic.

Recent attention has focused on gas wells on the school grounds, several of which have leaked and spread liquid nearby. Other theories include a possible autoimmune reaction to infection, called PANDAS, which can have neurological consequences including tics. (The National Institutes of Health has offered to examine the girls for this, as well as evaluate them for an ongoing study on conversion disorder.) Vaccine concerns have been raised. The state report on Friday said not all the girls had the Gardasil shot to prevent HPV infection. They did not address the issue of flu shots, most of which contain mercury, an established cause of tics.

If a toxin generated from schoolyard grass were the cause, ironically, it would appear to absolve LeRoy as some sort of toxic wastebasket, although the question of why the school was sited where it is might become more pertinent. We learned that in 2000, the district ignored an offer of free land within the village of LeRoy and instead bought land for the new school from the brother and mother of the school board president.

As we reported here, several of the fields sit atop a federally designated FEMA flood hazard area.



Former students and townspeople have told us that flooding and settling problems have plagued the school since it opened in 2003, and that ball fields and a soccer field had to be dug up and rebuilt in the past year or so because of water woes.

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March 13, 2012

[CropLife America \(CLA\)](#) president and CEO Jay Vroom spoke last Friday about the history of advancements in the [crop protection industry](#) with attendees of the University of Utah's Wallace Stegner Center's 17th Annual Symposium, "Silent Spring at 50: The Legacy of Rachel Carson," in Salt Lake City, Utah. Vroom presented to an audience of approximately 150 law students, faculty, alumni, and interested community representatives about the influence of Rachel Carson's seminal book, and its impact on the regulatory framework, environmental awareness, and development of crop protection products.

Other conference speakers represented the fields of academia, public health and conservation, including: Dr. Philip Landrigan of Mt. Sinai School of Medicine; Mark Lytle of Bard College; Paul Holthus of the World Ocean Council; and Terry Collins of Carnegie Mellon University. In his presentation, Vroom emphasized that whether it's through improved chemical formulas, more precise applications or integrated pest management programs, the crop protection industry continues to advance and help growers safely and responsibly produce food, fiber and renewable fuel.

"Silent Spring launched the modern environmental movement, and it is important to reflect on the impacts made on U.S. agricultural policy and the regulation of crop protection products," said Vroom. "By looking back at the societal changes ignited by Ms. Carson's writing, we can also look forward to future research and development opportunities and the creation of better modern agricultural tools. CropLife America is excited to engage in an open dialogue about this important book, discuss the improvements the crop protection industry has made in its wake, the advancements still happening every day, and how modern agriculture better interfaces with today's environmental concerns."

In his presentation, Vroom highlighted specific changes spurred by the 1962 publication of Silent Spring:

- **The creation of the U.S. Environmental Protection Agency (EPA):** Amid growing consumer concerns about environmental protection, President Nixon created the U.S. EPA in 1970 to protect human health and the environment. The creation of EPA marked a transition to a more rigorous crop protection registration and regulatory program. It also created a collaborative atmosphere between industry and the Agency.
- **A revised Federal Fungicide, Insecticide and Rodenticide Act (FIFRA):** With the creation of EPA, FIFRA was revised to provide new safety measures. Three separate amendments from 1972 through 1992 significantly updated the original 1947 law, and established additional rigorous standards for crop protection including: transferring pesticide regulation from the U.S. Department of Agriculture (USDA) to EPA; re-registering older pesticides to ensure compliance with new standards; and new worker protection measures. In addition, the 1996 Food Quality Protection Act adds special safety margins for infants and children, and the Pesticide Registration Improvement Act (PRIA), passed first in 2002, increased industry fees to enable EPA to expand scientific evaluation capacity and enhance timely decision-making.
- **A dedication to research & development:** Research and development (R&D) is a core pillar of the crop protection industry. Information from the USDA Economic Research Service shows that private investment in R&D for crop protection products has grown significantly, from \$42 million nominal in 1962, to \$793 million in 2010.

"The crop protection industry is committed to hearing and responding to consumer questions and concerns about U.S. agriculture, and to better communicating our investment and dedication to protecting human health and the environment," continued Vroom. "Speaking at this conference with the University Of Utah College Of Law is a unique opportunity to join in this dialogue surrounding Silent Spring and take this conversation one step further."

Thorough testing, science-based regulation, and continued investment in modern agricultural tools and techniques all contribute to the success of U.S. farming. With the collaboration of scientists, industry and regulatory agencies, agriculture looks vastly different than what was portrayed by Silent Spring 50 years ago.

To view a special interview that Vroom recently held with Ken Cook, president of the Environmental Working Group, on these issues and more, visit www.croplifeamerica.org/news/multimedia-resources/Jay-Vroom-and-Ken-Cook-Discuss-Silent-Spring. For additional information on the regulation and safety of crop protection products, visit www.croplifeamerica.org.

Dr. Seuss, Pesticide Shill?

Before he was Dr. Seuss, the beloved children's author Ted Geisel inked comics for a pesticide giant.

By [Kate Sheppard](#) | Mon Apr. 9, 2012 3:00 AM PDT

Dr. Seuss is best known for his allegorical children's books on themes like protecting the environment, shunning materialism, and embracing multiculturalism. But many people don't realize that before writing those children's books, Seuss also worked on commercial art for a pesticide company.

As farmer and author Will Allen noted in his 2007 book [The War on Bugs \[1\]](#), Seuss also created illustrations for pesticides in the late 1920s. The book's publisher, Chelsea Green, has [made the full chapter \[2\]](#) of the book available online for a limited time.

Back when Theodor Seuss Geisel was a young cartoonist, Standard Oil—a major player in the petroleum industry that had branched out into making bug sprays—noticed that he'd used their Flit spray guns in several illustrations. Standard decided to hire Seuss to make funny cartoon advertisements, which appeared in national magazines and newspapers. He did work for the company between 1928 and 1943, and "is generally acknowledged to be responsible for greatly popularizing the use of household poisons," writes Allen.

Certainly no fan of chemicals, he continues:

Seuss helped America become friendly with poisons; we could laugh at ourselves while we went about poisoning things. In the process, the public grew comfortable with the myth that pesticides were absolutely necessary.

That work also helped Seuss, who was then working for a national humor magazine, pay the bills and work on the beloved books he would later become famous for writing. But anyone who's seen Seuss' books warning about the dangers of industrialism might wonder what the heck happened. Allen offers a possible explanation:

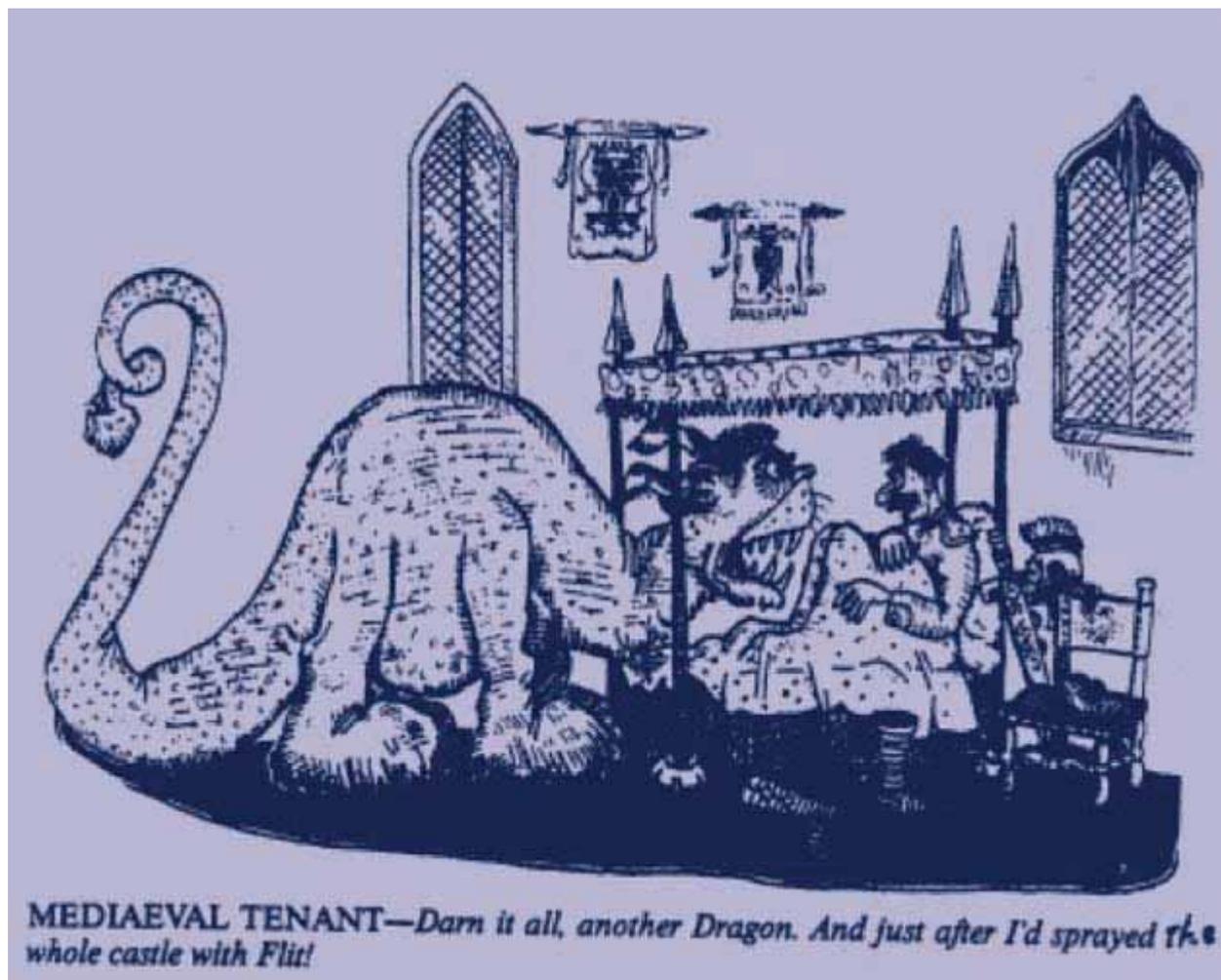
Perhaps Dr. Seuss realized his earlier mistakes and indiscretions with Standard Oil's Flit and tried to make amends with *The Lorax*. Geisel must



Seuss created this

have known that Flit's cartoons and his World War II cartoons for DDT had an enormous impact on the public's use of pesticides and acceptance of DDT.

cartoon for Flit bug spray.



After Seuss used Flit as a prop in this cartoon, Standard offered him a job.



Another cartoon Seuss drew for Standard's Flit ad campaign.

Source URL: <http://www.motherjones.com/blue-marble/2012/04/dr-seuss-pesticide-shill-flit-bug-spray>

Links:

[1] http://www.chelseagreen.com/bookstore/item/the_war_on_bugs:paperback

[2] <http://www.chelseagreen.com/content/dr-suess-petrochemicals-and-the-war-on-bugs/>