Maine Board of Pesticides Control

Miscellaneous Pesticides Articles October–November 2012

(***identified by Google Alerts or submitted by individuals***)

Maine Government News

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Maine Identifies First Case of West Nile Virus In Human

October 31, 2012
Human Services

AUGUSTA – The Maine Center for Disease Control and Prevention announced today that it has identified the state's first-ever case of West Nile virus (WNV) in a Maine resident from Cumberland County. The patient, who has now recovered, presented with symptoms including fever, encephalitis, meningitis, weakness and double vision. The symptoms began on October 1. Since the patient had not traveled during the exposure period, this is considered a locally acquired case. WNV is a virus that is transmitted through the bite of an infected mosquito. It can cause serious illness in humans, large animals and some species of birds. Nationally and regionally, 2012 has been an extremely active year for WNV. Prior to this human case, seven mosquito pools in Maine tested positive for WNV in August and September. Five of the positive mosquito pools came from Cumberland County and the other two were in York County. A Pennsylvania resident vacationing in Maine in August also tested positive for WNV earlier this year; however the infection was likely contracted out of state. While winter is approaching in Maine, the temperatures are still high enough to support mosquito activity. "As long as the temperatures remain above freezing there is potential for West Nile virus transmission," said Dr. Stephen Sears, State Epidemiologist. Mosquitoes will remain active until the first hard frost of the season. While some parts of Maine have already experienced their first hard frost, others have yet to reach freezing temperatures, making it important to remember to protect against mosquito exposures.

Maine CDC recommends the following preventative measures to protect against WNV and other mosquito-borne illnesses:

- Use an EPA approved repellent when outdoors, especially around dawn and dusk – always follow the instructions on the product's label
- Wear protective clothing when outdoors, including long-sleeved shirts, pants, and socks
- Use screens on windows and doors to keep mosquitoes out of the home
- Avoid or limit time outdoors at dawn and dusk when many species of mosquitoes are most active
- Practice household mosquito-source reduction: standing water should be removed from artificial water-holding containers in and around the house

The MeCDC will continue to monitor mosquitoes and expects to see the virus again in the spring when it will be brought by migrating birds. The Department of Agriculture, Conservation and Forestry notes that both WNV and Eastern Equine Encephalitis are reportable diseases in animals as well. Dr. Beth McEvoy, Acting State Veterinarian, reminds horse owners that both viral diseases are preventable with vaccines.

Portland Press Herald

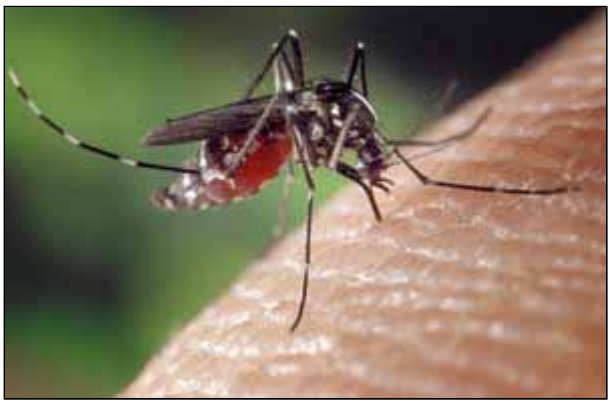
October 31

Maine confirms first-ever case of West Nile Virus

The Cumberland County man is recovering at home after being hospitalized for a disease now in 48 states.

By Leslie Bridgerslbridgers@pressherald.com
Staff Writer

A 34-year-old man from Cumberland County who was hospitalized in October has been confirmed as the first person known to contract the West Nile virus in Maine, state health officials said Wednesday.



The man, who suffered from encephalitis and meningitis as a result of the virus, has been released from the hospital and is recovering well, said officials, who would not disclose the man's name or hometown. They did not know how long he had been in the hospital.

Alaska and Hawaii are now the only states that haven't reported a case of West Nile virus. Each of the other 48 states reported at least one case this year, according to the U.S. Centers for Disease Control and Prevention.

The 4,891 cases so far in 2012, which included 223 deaths through the end of October, are the most reported since 2003, according to the federal CDC.

More than a quarter of the cases this year have been in Texas, where 1,665 people have been diagnosed with West Nile virus and 75 have died.

State epidemiologist Dr. Stephen Sears said Wednesday that federal health officials had confirmed within the past day that blood and cerebral spinal fluid samples taken from the Cumberland County man tested positive for West Nile.

He said no other people in Maine are suspected to have the disease.

A vacationer from Pennsylvania was diagnosed with the West Nile disease in Maine about two months ago, but that person felt sick before arriving in the state and likely contracted the virus in Pennsylvania,

officials said.

The Cumberland County man had not recently left the state, said Sears.

Seven mosquito pools in the state -- five in Cumberland County and two in York County -- contained mosquito larvae that tested positive for West Nile virus in August and September, the Maine Department of Health and Human Services said in a press release Wednesday.

The pools were located in Biddeford, Scarborough, Gorham, Lebanon and Standish, according to the department.

Dr. Sheila Pinette, director of the Maine Center for Disease Control & Prevention, said state health officials were "kind of expecting" there would be a human case of West Nile this year, given the prevalence of infected mosquitoes in Maine.

Still, she said, "It kind of disappoints us -- not what we wanted to hear."

Because there has not been a hard frost in southern Maine this fall, mosquitoes are still around and "there's still a risk," said Sears.

Pinette advised people to empty standing water from pots or tire swings and to fix screens on doors and windows.

"Just be aware that it's out there," she said.

Staff Writer Leslie Bridgers can be contacted at: 791-6364 or at

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11/1/2012 11:46:00 AM

Maine Identifies First Case of West Nile Virus in a Human

Mosquito carriers will be active until first hard frost -

The Maine Center for Disease Control and Prevention (CDC) announced on Wednesday, October 31, that it had identified the state's first-ever case of West Nile virus (WNV) in a Maine resident. The WNV virus, transmitted through the bite of an infected mosquito, can cause serious illness in humans, large animals and some species of birds. The patient from Cumberland County, who has now recovered, presented with symptoms including fever, encephalitis, meningitis, weakness and double vision. The symptoms began on October 1; since the patient had not traveled during the exposure period, this is considered a locally acquired case.

A Pennsylvania resident vacationing in Maine tested positive for WNV earlier this year; however, the infection was likely contracted out of state. In August and September, seven mosquito pools in Maine tested positive for WNV, five in Cumberland County and the other two in York County.

Mosquitoes will remain active until the first hard frost of the season. "As long as the temperatures remain above freezing there is potential for West Nile virus transmission," says Dr. Stephen Sears, state epidemiologist. Parts of Maine have not yet experienced their first hard frost; Maine CDC recommends the following:

• Use an EPA-approved repellent when outdoors, especially around dawn and dusk; always follow the instructions on the product's label

• Wear protective clothing when outdoors, including long-sleeved shirts, pants, and socks

• Avoid or limit time outdoors at dawn and dusk, when many species of mosquitoes are most active

• Use screens on windows and doors to keep mosquitoes out of the home

• Minimize mosquito breeding sites: inside and around the house, standing water should be removed from containers.

Maine CDC will continue to monitor mosquitoes and expects to see the virus again in the spring, when it will be brought by migrating birds. The Department of Agriculture, Conservation and Forestry notes that both WNV and Eastern equine encephalitis are reportable diseases in animals as well. Dr. Beth McEvoy, acting state veterinarian, reminds horse owners that both viral diseases are preventable with vaccines.

Maine records 1st West Nile virus case

AUGUSTA, Maine (AP) — Maine health officials say they've identified the state's first case of West Nile virus.

The patient, who was not identified by the state, is a resident of Cumberland County and has recovered.

The Maine Center for Disease Control and Prevention says the patient had symptoms including fever, encephalitis, meningitis, weakness and double vision. The symptoms began on Oct. 1.

West Nile is transmitted through the bite of an infected mosquito. It can cause serious illness in humans, large animals and some species of birds. Nationally and regionally, 2012 has been an extremely active year for the disease. Seven mosquito pools in Maine tested positive for West Nile in August and September.

BANGOR DAILY NEWS

Cumberland County man Maine's first case of West Nile in a human

By Jackie Farwell, BDN Staff
Posted Oct. 31, 2012, at 3:17 p.m.

A Cumberland County man has been confirmed as Maine's first case of West Nile virus in a human, the Maine Center for Disease Control and Prevention announced Wednesday.

The 34-year-old man who contracted the potentially deadly disease, which is carried by mosquitoes, has recovered. His symptoms began on Oct. 1 and included fever, weakness and double vision, as well as swelling of the brain and meningitis.

He was hospitalized and has been released, according to Dr. Sheila Pinette, director of the Maine CDC. Tests last week confirmed the disease, she said.

In August, a Pennsylvania resident vacationing in Maine tested positive for West Nile but health officials said the infection was likely contracted out of state. The Cumberland County patient had not recently traveled and is believed to be the first in Maine's history to acquire the disease in state.

West Nile reappeared in Maine this year in August in mosquitoes in York County. It subsequently showed up in one other mosquito trap in York County and five traps in Cumberland County.

With Wednesday's case, Maine became the last of the lower 48 states this year to report West Nile infection among residents, according to U.S. CDC data. The virus made a comeback in 2012, and has infected nearly 5,000 people, the most reported to the CDC through the last week of October since 2003.

More than 230 people have died.

Texas accounts for about a third of this year's cases.

"We were one of the few states that had not had it yet, so it was sort of expected, although disappointing," Pinette said.

Only Hawaii and Alaska have been spared from West Nile.

The virus first was detected in the United States in 1999. Health officials aren't sure what's causing the rise in West Nile this year, but suspect the weather may be playing a role.

Temperatures are still warm enough for mosquitoes to survive and spread the disease, according to state epidemiologist Dr. Stephen Sears. Mosquitoes pick up the virus from biting infected birds and then transmit it to people.

"As long as the temperatures remain above freezing there is potential for West Nile virus transmission," Sears said in a news release.

Mosquitoes remain active until the first hard frost of the season. Some parts of Maine have seen a hard frost, but other regions have yet to reach freezing temperatures.

Most people infected with West Nile don't show any symptoms. About one in five gets sick with a fever, body aches, vomiting and joint pain that can last from a few days to several weeks. Symptoms typically appear between three and 14 days after a bite from an infected mosquito.

In the rare severe cases, which are more common in people 50 and older, the virus causes neurological problems such as brain swelling that can lead to confusion, coma, seizures and permanent damage.

To survive the serious neurological complications, patients typically must have a healthy immune system, Pinette said.

There is no human vaccine or cure for West Nile. Patients with the virus can be treated for their symptoms, by administering oxygen and intravenous nutrition and fluids.

Horses can be vaccinated against both West Nile and Eastern equine encephalitis, another mosquito-borne illness.

West Nile is not spread by close contact with someone infected with the virus.

Maine CDC plans to continue monitoring mosquitoes for the virus and expects West Nile to crop up again in the spring as it is carried by migrating birds.

To lower your risk of getting infected with West Nile and other diseases carried by mosquitoes, Maine CDC recommends:

• Using insect repellent containing an EPA-registered active ingredient when outdoors.

• Trying to stay indoors at dusk and dawn, when mosquitoes are most active.

• Wearing long sleeves and pants if you plan to be outside.

• Installing door and window screens and repairing any holes to keep mosquitoes out.

• Emptying standing water where mosquitoes can breed, such as from flower pots, buckets and barrels. Change the water in pet dishes and replace the water in bird baths every week. Drill holes in tire swings to allow water to drain. Empty kiddie pools when they are not in use and store them on their side.

• Don't handle a dead bird with your bare hands. Contact your local health department for instructions on reporting and disposing of it.

EPI Information
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- A - Z Index of Epidemiology Diseases
- Disease Reporting
- Publications
- Request for Data
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Information for
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211 Get Connected. Get Answers.
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Arboviral Surveillance

Eastern Equine Encephalitis (EEE) and West Nile Virus (WNV) are arboviral illnesses spread by the bite of an infected mosquito. Mosquito, animal, and human specimens are tested for the presence of EEE and WNV at Maine's Health and Environmental Testing Laboratory (HETL). Animal specimens may also be tested through the National Veterinary Services Laboratory (NVSL). The state of Maine organizes routine mosquito trapping at selected sites, and routinely tests those mosquitoes for disease. The mosquito trapping season runs from July to September. Maine no longer routinely tests birds.

2012 Maine Arboviral Positive Results

Surveillance	Species	Collection Date	Town	County	Agent
Human				Cumberland	WNV
Mosquito	Culiseta melanura	08/01/2012	Lebanon	York	WNV
Mosquito	Culex pipiens/restuans	08/01/2012	Gorham	Cumberland	WNV
Mosquito	Culiseta melanura	08/23/2012	Gorham	Cumberland	WNV
Mosquito	Culiseta melanura	08/23/2012	Standish	Cumberland	WNV
Mosquito	Culiseta melanura	08/30/2012	Scarborough	Cumberland	WNV
Mosquito	Culiseta melanura	08/31/2012	Scarborough	Cumberland	WNV
Animal	Pheasant	09/06/2012	Lebanon	York	EEE
Mosquito	Culex pipiens/restuans	09/06/2012	Biddeford	York	WNV

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- **2011**

Resources

- Massachusetts Arbovirus Information
- New Hampshire Arboviral Test Results
- Vermont Arboviral Surveillance
- National Arbonet Maps (provided by USGS)
- Federal CDC Site

Pesticide Risks....topic of discussion on the UMFK campus

October 26, 2012
NR12108

The University of Maine at Fort Kent's Environmental Studies Speaker Series will welcome Dr. Lebelle Hicks, from the Maine Board of Pesticides Control, who will deliver a presentation on “Pesticide Risks in 2012,” on Friday, November 9 at 11 a.m. in Nadeau Hall teleconference room. The presentation is free and open to the public.

Dr. Hicks will speak on chemicals, exposure, and toxicity. How can you use pesticides and not poison everything?

Dr. Hicks received a Ph.D. in Food and Nutrition Sciences from the University of Maine in 1999, and a Master of Science in Biology and Toxicology from Northeastern University in 1983.

She has been certified by the American Board of Toxicology in general toxicology since 1991. Dr. Hicks has worked as a pesticide toxicologist for the last 27 years, first in Massachusetts and currently in Maine. Her duties include addressing pesticide concerns from the general public, other state agencies, and grower groups.

She also provides written and oral presentations of hazard reviews and risk assessments relating to human health or environmental exposure. Dr. Hicks' duties with the Maine Department of Agriculture cover a wide range of pesticide and non-pesticide food safety issues.

In 2012, Dr. Hicks was appointed as adjunct faculty at the University of Maine and will be teaching a course in Risk Assessment, Risk Management and Risk Communication on the Orono campus through the Food Science and Human Nutrition Program.

The Environmental Studies Speaker Series at UMFK regularly brings speakers to campus. These presentations cover such topics as water quality, waste management, wind power, endangered species, forestry and marine science. The program is offered in collaboration with the UMFK Environmental Studies degree program. Speakers only are scheduled during the academic year.

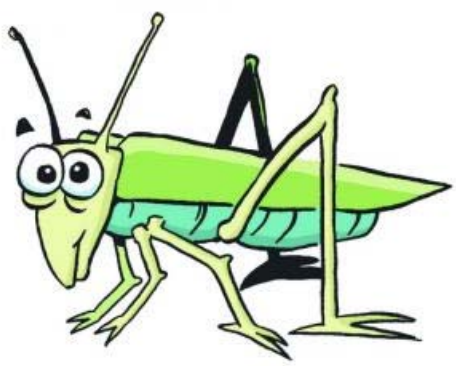
For more information on the presentation or the Environmental Studies Speaker Series, contact Dr. Steven Selva at (207) 834-7617 or by e-mail at: sselva@maine.edu

Maine Board of Pesticide Control speaker presents "Pesticide Risks in 2012"

News Local News
Send by email

CONTRIBUTED
8 November 2012

FORT KENT - UMFK's Environmental Studies Speaker Series welcomes Dr. Lebelle Hicks, from the Maine Board of Pesticides Control, who will deliver a presentation on "Pesticide Risks in 2012," on Friday, November 9 at 11 a.m. in the Nadeau Hall teleconference room. The presentation is free and open to the public.



Dr. Hicks will speak on chemicals, exposure, and toxicity, and will address the question, "How can you use pesticides and not poison everything?"

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Chris Prior Passes Maine Pesticide Exam

Chris Prior Passes Maine Pesticide Exam

Posted on October 23, 2012 by alicia

EnviroVantage is proud to say that their Director of Residential Sales, Chris Prior, has passed the Maine Pesticide 7C3 Master certification exam, specifically for projects dealing with mold remediation and water damage restoration. He is now one of only 14 people that possess this specific license.

On top of it being the law in Maine to possess this certification when working in situations involving pesticides, it also ensures responsibility and shows a commitment to safety on the part of the company. EnviroVantage knows how important it is to keep all employees up-to-date in training as well as to obtain new certifications as they plan to grow throughout New England.

Way to go, Chris!

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Pesticide Exam

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Popular Post

Morning Sentinel

November 10

Monmouth experimental farm leads fight against new fruit fly threat

BY JOHN HALE, Correspondent

MONMOUTH -- Situated atop Norris Hill on U.S. Route 202 in the western part of town, Highmoor Farm has been a center for research into apple growing, other fruit and vegetables, and crop pests for more than a century.

click image to enlarge

The Highmoor Farm in Monmouth includes a large farmhouse, two large barns, two laboratories, a shop, 10 cold storage lockers, two hoop houses and a greenhouse on 278 acres with 17 acres of apple orchards.

Contributed photo

Now the University of Maine Agricultural Experiment Station is leading the fight against a newly discovered strain of fruit fly that poses a serious threat Maine's berry crops.

David Handley, an extension agent who has worked at Highmoor since 1983, is behind the efforts to stop spotted wing drosophila, the breed of fruit fly that originated in Asia and then migrated to the American South. It first turned up in the Northeast in the summer of 2011.

Drosophila can lay eggs on strawberries and raspberries before they are ripe. The larvae then hatch after the berries are ripe and begin eating the fruit, making the berries soft and mushy, Handley said.

"It's fairly easy to kill, if you have the right chemicals," he said. "A problem is you might want to use a pesticide at the same time that you want to harvest the fruit."

Highmoor Farm is one of five farms run by the University of Maine College of Natural Sciences, Forestry and Agriculture that aim to help Maine farmers find crops with the best yields and quality.

The University of Maine Cooperative Extension Service also has a presence at Highmoor Farm, doing

outreach and education with about 200 commercial growers in Maine. That research now is centered on the newly discovered strain of fruit fly.

Greg Koller, superintendent at Highmoor Farm since 2004, lives year-around at Highmoor Farm with his wife, Sheri, and their two high school-age sons. Koller said scab tolerance in cucumbers was an important vegetable feature that was developed at Highmoor Farm. Brock apples, a sweet and juicy variety that ripens in early October, also were developed at Highmoor.

"We research integrated pest management," Koller said. "It used to be that people sprayed on a schedule, no matter what. Now we try to spread it out and only spray when we need to. With this new fruit fly, we hope to use other natural enemies of the fly."

During the summer, Handley uses four University of Maine students who go out on the road to berry farms to check on traps that are set for drosophila flies and other pests.

Many pests can't survive winter in Maine, Handley said, but drosophila might be able to so.

Handley said the pesticide Spinosad, derived from a fungus, so far has proved most effective against the flies.

"We can control it, but it's a lot of effort and a lot of cost," Handley said. "It's a new pest, so we haven't developed alternatives for this. I don't think there's an easy answer for this."

MORE ABOUT HIGHMOOR

According to an article by David C. Smith, "A History of the Maine Agricultural Experiment Station, 1885-1978," the farm buildings at Highmoor were built by James Roscoe Day in 1880 as a summer home. Day also planted some 5,000 apple trees.

By 1907, the farm was owned by F.H. Mundy. When the state of Maine bought the farm and turned it over to the University of Maine in 1909, the apple orchards had deteriorated and there were only 3,100 apple trees left. Most of them were in poor condition.

By 1910, Highmoor's orchard had been culled to a total of 2,300 trees. Highmoor produced only 200 barrels of apples in 1909, but by 1912, its production had risen to 3,200 barrels.

Highmoor Farm today includes a large farmhouse, two large barns, two laboratories, a shop, 10 cold-storage lockers, two hoop houses and a greenhouse. The farm encompasses 278 acres, with 17 acres of apple orchards and 5 acres of tilled fields for vegetable and small fruit research.

Besides Greg Koller and David Handley, staff members at Highmoor Farm include apple specialist Renae Moran, researcher and Extension agent Mark Hutton and Mark Hutchinson, who runs the composting education program.

Koller said Highmoor hosts the Maine Compost School twice a year, for one week each session. He said it attracts students from around the world. Highmoor also cooperates with the town of Monmouth on composting.

Portland Press Herald

October 21

Maine Gardener: Extension unleashes Green Bug on pesky pests

By TOM ATWELL

For the past couple of weeks, I have been writing about pests, and there do seem to be a lot more of them to write about. This week, the topic is a new program to help people deal with those pests.

The University of Maine Cooperative Extension, in cooperation with a number of garden centers and other pesticide retailers, has recently launched the Green Bug Education Program designed to help people deal with pests without using unnecessary pesticides.

"It's an amazing thing," said James Dill, a pest management specialist with the extension, "but from 1995 to 2007, the use of lawn pesticides in Maine has gone from 800,000 pounds a year to 6.2 million pounds. There is really no need for that kind of increase."

Under the Green Bug program, seven retailers so far have agreed to charge an extra 20 cents for every package of lawn or garden pesticides sold to help pay for the extension's Home and Garden Integrated Pest Management Program.

Integrated Pest Management is an approach that uses a variety of methods, including changing physical structures of growing areas, biological controls, accurate identification of the specific pest causing the problem and more to make sure pests are controlled in the most environmentally sound way possible.

The extra 20 cents per package will be a tax-deductible donation for the retailers. "People paying $19 on a package of weed and feed aren't going to mind paying $19.20," Dill said.

The retailers will put up a Green Bug logo near their supplies of pesticides, and also may carry a rack of cards describing a number of the pests.

Dill said his office gets more than 3,000 calls and specimens in the mail each year from people wanting to know what is damaging their plants.

"We don't mind that," he said. "It is what we are here to do."

But the Home and Garden IPM website at umaine.edu/home-and-garden-ipm has a variety of ways that people can identify pests on their own, including a photo gallery of pests and other insects divided into whether they affect fruits, vegetables, ornamental gardens, households or other areas.

It also shows beneficials, and has a section called "Curiosities" about some of the unusual things you might find.

If you think you know what you have, an easier way to check it might be the alphabetical list.

For more information about the Green Bug program, go to the Home and Garden IPM website and click on the green bug at the left of the page.

GETTING AWAY from pests, the Maine Landscape and Nursery Association earlier this month landscaped a group home in South Portland for people with traumatic brain injuries as its annual volunteer

BANGOR DAILY NEWS

Maine tick myths, revealed

Victoria Arocho | AP

A female deer tick is shown under a microscope.

By Aislinn Sarnacki, BDN Staff
Posted Nov. 07, 2012, at 1:38 p.m.

Hunters, beware. You aren't the only ones in the woods searching for a set of antlers.

Winter ticks have just hatched, and they're looking for warm hosts, preferably deer or moose. But because the two of you are looking for the same thing, your odds of running into each other are pretty high, and ticks aren't known to be picky.

You might just stumble through a nest and look down to see 50 newly-hatched ticks crawling up your camouflage. They'll latch onto your skin and suck your blood, which is thoroughly disturbing, but no research shows them transmitting any diseases.

Unfortunately, deer ticks are also out and about, and one in four carries Lyme disease, according to Jim Dill, pest management specialist with the University of Maine Cooperative Extension.

"This was a pretty high deer tick year in comparison to other years," Dill said. "The dog tick population this year was fairly high, too, especially south of Bangor."

And when it comes to ticks, what you don't know can indeed hurt you, so let's lay to rest a few common misconceptions about these tiny pests.

Myth: Ticks die in the winter.

Deer ticks live for an average of two years, and they certainly don't migrate. Therefore, it's safe to assume they survive the winter. When the weather is freezing, they simply become inactive and often burrow into leaf litter.

"What we're finding about this time of year is that the deer tick is active as long as it's 38 degrees and above," said Dill. "Because of that, you can find deer ticks out any time of year. You can find them out in the middle of winter."

"Last winter was so mild that children were playing outside without the snow and coming in with ticks," said Bob Maurais, co-owner of Mainely Ticks, a tick management business based in southern Maine. "It's not a function of season, it's a function of the temperature and ground condition."

Myth: There are two types of ticks in Maine, dog ticks and deer ticks.

Winter ticks are actually a different species than dog or deer ticks. Also called the "moose tick," the winter tick is usually found on moose and deer, and occasionally on horses, cows, dogs and humans, mainly in central and northern Maine.

"We are still finding deer ticks, as we have all along, but we're having a lot of hunters call us with winter ticks," Dill said. "They cause a lot of problems with moose in the winter."

Hundreds of winter ticks can attach to a moose when it walks through their nest, which is often located in underbrush.

"What happens then is [the ticks] weaken the animal because they're drawing so much blood out of the animal," Dill said. "The moose is more susceptible to disease and predation, even cold temperatures."

Maine is also home to tick species such as the woodchuck tick, squirrel tick, rabbit tick, brown dog tick and Lone Star tick, according to the Maine Medical Center Research Institute.

Myth: If you find a tick latched onto you, you should set a hot match against its back end.

When a person finds a tick on them, the natural reaction is to get it off right away.

Not so fast.

"If you want to remove it, you have to be careful," Dill said. "Don't follow the old wives' tale and set a hot match on its butt or try to get it off with nail polish. If you think about it, if you do that, what [the tick] is probably going to do is regurgitate more stuff into the wound."

And you certainly don't want a tick to be vomiting anything into your bloodstream.

If it's a deer tick, the contents could include a corkscrew-shaped bacteria that causes Lyme disease, an illness that frequently starts with a rash and flu-like symptoms, and if untreated, may progress to cause arthritis and neurological problems.

To remove a tick, use fine-pointed tweezers or a tick removal device and grasp the tick as close to your skin as possible. Then apply slow, steady pressure, pulling outward to remove it, Dill said.

Usually, it takes a deer tick a minimum of 24 hours to transmit Lyme disease, but even if you remove the tick before it's engorged, it's always a good idea contact a doctor to talk about whether you should take antibiotics.

Myth: If you don't have a rash, you don't have Lyme disease.

In humans, Lyme disease typically causes a red rash to expand from the site of the tick bite. But this rash occurs in only 70-80 percent of Lyme disease patients, according to the Maine Center for Disease Control and Prevention.

Myth: Deer ticks and Lyme disease are only a problem in Southern Maine.

The first documented case of Maine-acquired Lyme disease was diagnosed in 1986. Since 2003, when 175 cases were confirmed, the numbers of reported cases have increased each year through 2011 (1,002 cases), with the exception of 2010, according to a 2012 report by the Maine Center for Disease Control and Prevention.

While the vast majority of Lyme disease cases reported in the 1990s occurred among residents of south coastal Maine, in recent years, the disease incidence has increased steadily in the northern parts of the state. Since 2008, no Maine county

has escaped at least one new case of Lyme disease each year.

Myth: Only people can get Lyme disease.

Lyme disease affects many animals, including horses, cats and especially dogs.

“The state no longer requires that small animal veterinary clinics report cases of the disease in dogs because we were getting so flooded with reports,” said Elizabeth McEvoy, acting state veterinarian.

“We’re seeing more and more Lyme disease cases every day now,” said Jessica Jones, a veterinarian at Brewer Veterinary Clinic. “We’re actually diagnosing it every day in dogs.”

At the Brewer clinic, the test for Lyme disease in dogs takes about eight minutes and three drops of blood, said Jones. And if the dog tests positive for the disease, they put the animal on a 30-day regimen of antibiotics.

“A lot of the dogs that we diagnose with Lyme disease, we wouldn’t even know they have it,” said Jones. “And the dogs that show symptoms will often have swollen joints, lameness or lethargy. In a small percentage of dogs, it causes fatal kidney failure.”

Maine veterinary clinics offer a Lyme disease vaccination for dogs that has become increasingly popular, taking care of the problem before it starts.

Myth: The only disease ticks transmit is Lyme disease.

Two other tick-transmitted diseases, human granulocytic ehrlichiosis (HGE) and babesiosis, have been found in Maine during the last few years, according to the Maine Medical Center Research Institute. For more information, visit mmcri.org.

Myth: You can check your body for ticks with your eyes.

Two summers ago, a woman from southern Maine called Maurais at Mainely Ticks after finding a nymph (young) tick in the belly button of her 10-month-old son. At first, she thought it was dirt, but when she tried to move it, it wouldn’t come out. With a magnifying glass, she looked again and saw that the “dirt” had legs.

“Had she not checked and had her son been infected, who knows what problems that youngster would have,” Maurais said.

The tick has three life stages: larva, nymph, and adult. Because the nymphs are about the size of a poppy seed, they often go unnoticed until fully engorged, and are therefore responsible for nearly all of human Lyme disease cases, according to the American Lyme Disease Foundation.

“It surprises people when we talk about the nymph tick, which is the size of the head of a pin,” Maurais said. “It could be nestled behind your ear and you’d never know it. When you ask someone to do a tick check, they’re subconsciously looking for a big tick.”

Maurais suggests that people who spend time outdoors — for example, hunters — should conduct a daily tick check with their fingertips, feeling for small bumps along their skin. A thorough check should take about 30-45 seconds, he said.

And you don’t need to stumble through a winter tick nest while moose hunting to be a likely host. If your outdoor cat sleeps on your pillow at night or you’ve been raking the leaves off your lawn, you might want to go ahead and check.

For a fact sheet on ticks by pest management specailist Jim Dill and insect diagnostician Clay Kirby, visit umaine.edu/ipm/ipddl/publications/5047e/.

KENNEBEC JOURNAL

PRINT THIS

October 21

Nonprofit taking over LakeSmart program

By Matt Hongoltz-Hetlingmhhetling@mainetoday.com
MaineToday Media

A program that rewards lakefront property owners for good environmental stewardship will soon be run by a private nonprofit lakes association instead of the state.

click image to enlarge

PROTECTORS: Phyllis and Lynn Matson stand beside a wooded buffer zone between their home and Long Pond in Rome. The buffer helps restrict erosion that contains phosphorus and other nutrients that can adversely affect the water. The Matsons received a LakeSmart award for their efforts.

Staff photo by David Leaming

An official with the **Department of Environmental Protection**, which created the LakeSmart program in 2004, said the decision to transfer it was made because a local nonprofit is more likely to get widespread participation than the state.

The Maine Congress of Lakes Associations, which is poised to fully take over the program on Nov. 1, is seeking funding to help keep the program going.

Samantha DePoy-Warren, department spokeswoman, said no new lakes have joined LakeSmart over the last year or two; while 33 lakes have participated over the course of the program's history, only about 10 lakes had active participation this year.

Property owners, especially those who would benefit from the exposure to the program's educational message, are wary of inviting state inspectors onto their land, knowing that those same inspectors have the power to fine them for environmental violations.

"We feel that more lakefront property owners, including those that need the extra education this program provides, would feel more comfortable inviting LakeSmart onto their property," DePoy-Warren said.

Maggie Shannon, president of the congress, agreed.

"It's hard for a regulatory agency that goes out with a stick to dangle a carrot and have people respond,"

she said. "This is the ultimate carrot."

LakeSmart is meant to encourage responsible lakefront stewardship by awarding highly visible lawn signs to those who meet certain criteria during a visit from certified inspectors.

For example, an applicant might receive points for planting vegetative buffers that slow pollutant-bearing runoff, but lose points for owning a dog that leaves waste by the water's edge.

Phyllis Matson and her husband, Lynn Matson, have participated in the program for the last three and a half years, both as property owners and as volunteer inspectors in a pilot LakeSmart training program run by the department and the Belgrade Lakes Association.

Over that time, she estimates that they've screened about 45 properties on Long Pond and Great Pond, which are part of the Belgrade Lakes system.

She said property owners understand the difference between her and a state regulator.

"We make clear to them that we're not the lake police," she said.

For Matson, she said that she will continue to participate in the program as long as it continues to protect the lakes.

"As long as standards are kept the same," she said, "I don't care who runs it."

LakeSmart was begun on the idea that, after a certain number of signs are awarded on a given lake, a tipping point would be achieved that would put social pressure on other property owners to have their land evaluated and certified as well.

That tipping point comes around the 15 percent mark, said Shannon.

"This is based on a whole body of knowledge that studies human behavior. We don't do what we're told to do. We do what our neighbors do," Shannon said.

The problem, said DePoy-Warren, is that the program hasn't been very popular under the state's supervision, so the theoretical tipping point is not being achieved.

She estimates that, after spending an estimated $1 million on LakeSmart over the years, mostly in staff time, the program has reached fewer than 500 of the tens of thousands of lakefront property owners in the state. Of Maine's 2,300 lakes, only 33 have had even a single participating landowner.

"If you break out the numbers, it's been about $2,000 per property," she said. "I need a program that, for that amount of money, is more far-reaching. I need to think about educating more owners than 500 homes over 10 years. "

Shannon said that, with an army of volunteers from 120 member lake associations, her group can also bring down the costs of the program while increasing participation.

While the department has estimated the program's costs to be about $100,000 per year, Shannon said the congress can run it in 2013 for between $65,000 and $70,000.

The money, however, is not yet in place.

DePoy-Warren said that the department, which will continue to offer technical support for LakeSmart, helped the congress to apply for some grants.

"They're in a much better place to solicit private and nonprofit and public sector funding in a way that the department is just not able to pursue," she said.

Shannon said that a fundraising campaign will begin in earnest in November.

"We think a lot of individuals and local businesses would be interested ins supporting this and being recognized," she said. "Many towns depend on these lakes for essential services. In my town, Rome, 60 percent of the economic activity comes from lakefront properties. They're life-giving."

The goal is to get 60 lakes involved in the program by 2018, an increase of about 10 lakes per year, according to a transition summary from the congress.

For 2013, the plan is to maintain LakeSmart's presence in lakes that currently participate. At the same time, Shannon said that there will be "an energetic and systematic review" of the program to figure out how to improve it. The established criteria to qualify for a LakeSmart award will not be changed, she said.

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Winter Moth Spread Prompts Community Action

jeanne posted on November 01, 2012 15:47

By Douglas Rooks, Editor

The defoliating insect known as winter moth has been identified in two parts of the Maine coast, but the affected communities are making determined efforts to control its spread. Charlene Donahue, forest entomologist at the Maine Forest Service, said that an infestation identified last year in Harpswell has now been matched by a new, relatively small one, on Vinalhaven.

And Donahue has now been able to positively identify the cause of the outbreak – contaminated nursery stock from sections of eastern Massachusetts that have long hosted populations of winter moth. “What seems to be happening is that stock transported over the winter is thawing in the spring and producing the larva,” she said. The caterpillar stage of the insect (*see photo*) is hardly distinguishable from other common species. But when the moth emerges late in the season – usually in November, hence the name “winter moth” – it quickly begins feeding on a variety of trees, including oak, maple, birch, cherry and important shrubs such as blueberry.

Once the moth becomes established, it can start defoliating whole trees, and mortality becomes a significant issue. The Harpswell outbreak, near the village center, is now fairly extensive and covers about 400 acres, Donahue said. And that’s prompted community action, after a series of meetings in which Donahue presented options for quarantine and control.

“They really stepped up to the plate,” she said of townspeople. “They were even willing to cancel a plant sale that had been one of the big community fundraisers,” adding that there’s no way to tell just what plants have been infested before the caterpillars emerge. Statewide, Donahue hopes that homeowners will get the word that they shouldn’t import any nursery stock from the infested areas of Massachusetts – one of several states south of Maine where winter moth is found.

One of the reasons Massachusetts didn’t respond to the original outbreak, Donahue said, is that the moth was misidentified. Its real identity didn’t become apparent until trees started to be denuded on a large scale, by which time control is less effective.

There is some good news in the picture, however. Control measures – through a parasitic fly (*Cyzenis albicans*) that’s just now being deployed in Massachusetts – can be effective. In fact, after the moth was accidentally imported from Europe, the first major outbreak was found in Nova Scotia in the 1930s, where it did significant damage. But the flies have been so effective in Canada that the moth has been reduced to minimal levels, suggesting that Maine could also have effective control of the pest. “It takes 10 years or longer, but the parasite does become well established,” she said.

But scientists aren’t sure they’ve completely figured out the winter moth lifecycle. Studies of other outbreaks suggest that the moth may interbreed with other species, which could produce either less damaging moths, or ones resistant to the parasitic fly.

Donahue managed to obtain a few of the parasitic flies on a trial basis this year, but hopes to have a lot more by next April, when they could be released in Harpswell. The flies lay eggs on leaves, which are then ingested by the winter moth caterpillars. The eggs then hatch inside the caterpillars, preventing them from spinning cocoons and ultimately killing them. Now that winter moth is here, it’s unlikely to be eradicated, she said, though through control efforts its effects can be minimized – much like other, better-known pests like gypsy moth and browntail moth, whose populations wax and wane, often from reasons obscure even to entomologists.

MFS is receiving some financial assistance for contending with winter moth. Donahue has been busy writing grant proposals, and the state should be eligible for control funds through the U.S. Forest Service. “Fortunately, it’s one of the things they will fund,” she said, despite overall reduced levels of federal assistance.

Vigilance is another important source of protection, Donahue said. “We found out about the Harpswell outbreak when a landowner called us and said, ‘What are these moths flying around here in November?’ ” Because moths are so unusual at that time of year, they stand out. “Look out your window,” Donahue advises. “If you see something unusual, call us.”

(*The Maine Forest Service welcomes reports of unusual insect activity. Call 287-2431.*)

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WCSH6.com Portland, ME

Emerald Ash Borer recently detected in Massachusetts

4:44 PM, Sep 14, 2012 | Written by Tim Goff

Related Links
- Maine Forest Service Emerald Ash Borer Information Website

AUGUSTA, Maine (NEWS CENTER) -- The emerald ash borer (EAB), an invasive insect first detected in the Midwest a decade ago, continues to quickly spread throughout the US and Canada. Last week, US forestry officials confirmed the tiny, metallic green beetles had been found in the western Massachusetts town of Dalton.

In Maine, entomologists with the Maine Forest Service are wrapping up a massive surveillance effort, inspecting nearly a thousand purple insect traps they placed strategically throughout the state to attract and capture the insect to see if it is already in the area.

"The purple color is slightly attractive to EAB, and then we have a little lure inside of it that has the scent of a stressed tree," explained Maine Forest Service entomologist, Colleen Teerling. "The idea is that if there is an insect around, they will be attracted by the color purple and by the scent of the lures, fly in and be stuck to the glue on the outside of the trap."

So far, none of the traps have captured an emerald ash borer, but news that they have spread into Massachusetts and Connecticut means that it may only be a matter of time.

"It is probably inevitable that at some point in time it will come to Maine, because it is firmly established in the US and it is spreading. It is just a matter of when. Is it going to be next year, or is it going to be 5 years or 10 years down the road?"

"The longer we can delay it coming to Maine, the more management tools we are going to have for options," stated Teerling.

"It is not an automatic, immediate death sentence for every ash tree in the state if we find it. So we do have hope to slow it down, to keep some ash trees alive longer, to contain the spread or at least manage the spreads so that it spreads very slowly as opposed to spreading like wildfire, which it has in the past."

NEWS CENTER

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Deer Hunt Set for Maine Island

11/19/2012 10:11 AM ET

The Department of Inland Fisheries and Wildlife has signed off on a controlled deer on Islesboro hunt to run from Dec. 10 through the end of the year.

ISLESBORO, Maine (AP) _ Islesboro residents are having a special deer hunt on their island to reduce the number of Lyme disease cases and protect the island's woodlands.

The Department of Inland Fisheries and Wildlife has signed off on a controlled deer hunt to run from Dec. 10 through the end of the year.

The island allows deer hunting from September into early December, but only with bows and arrows.

The special season allows shotguns, the first that's ever happened on the island. It's open only to residents and family members.

Voters last year approved a plan to cull the deer herd from about 50 deer per square mile to about 10 per square mile.

Of the island's 600 year-round residents, dozens have contracted the tick-borne Lyme disease over the years.

Return!

Sable Oaks Golf Course gains Audubon certification

By Duke Harrington dharrington@keepmecurrent.com | Posted: Wednesday, October 17, 2012 11:02 am

SOUTH PORTLAND – The 13th fairway at the Sable Oaks Golf Course in South Portland is about to get a little more difficult.

“This already is one of the most challenging greens in the state. It’s an extremely hard hole,” course superintendent Matt TenEyck said on Monday. “What we’re doing now with this is, we are actually changing the way this hole is played, making it potentially even harder.”

But those changes are not being made merely to frustrate area divot divas. The plan is to restore that natural conditions of Long Creek, which flows through the middle of the course, adding a 50-foot buffer zone to an area of the fairway that, not so long ago, was tended right up to the edge of the stream. It is part of an effort to restore habitat for bugs in Long Creek, to bring back trout and meet certain water-quality standards by 2020, the ultimate goal of the Long Creek Watershed Plan, created in July 2009.

“I think it takes a true stormwater geek to get really excited about the bugs, but generally, when a golf course is more environmentally friendly, it’s just a nice place – it’s a more beautiful golf course,” said Tamara Lee Pinard, executive director of the Long Creek Management District, as well as stormwater program manager at the Cumberland County Soil and Water Conservation District.

“I play golf all over New England and Sable Oaks has some of the best greens and fairways,” said Bell, whose firm is helping Sable Oaks obtain the Audubon certification. “It proves that ‘environmentally friendly’ can translate easily into ‘great conditions.’”

“Reclaiming this much, this is a lot,” said Forrest Bell, senior scientists for Portland-based FB Environmental Associates. “To leave a buffer of this width is a really big deal.”

On Tuesday, officials from city, county and state government were on hand to help plant red osier dogwoods, choke berry bushes and other plants along the stream that separates tee from cup on the Par 3 green. That digging was done in part to celebrate Sable Oaks’ recent certification in environmental planning from the Audubon Cooperative Sanctuary Program for Golf Courses, the first of six steps leading to recognition as a Certified Audubon Cooperative Sanctuary.

“We welcome Sable Oaks Golf Club’s commitment to the environment and management of the golf course with wildlife in mind,” said Jim Sluiter, staff ecologist for Audubon International, in a release announcing Sable Oaks’ effort to stand alongside the Portland Country Club, the only golf course in Maine with full sanctuary certification.

According to Pinard, Sable Oaks, at 154 acres, is the largest single lot of 127 along the 10 miles of Long Creek and its associated tributaries. Impervious surfaces – roofs, parking lots and other hard areas that speed stormwater runoff into the stream – cover between 11 and 67 percent of each of those lots. Eight percent is enough to impact water quality, says Pinard. Because of that heavy development, Long Creek does not meet minimum state water-quality standards.

That realization led to the creation of the Long Creek Management District, encompassing 2,240-acres and four-

municipalities, from Blanchette Brook tributary at the Colonel Westbrook Industrial Park, past the Maine Mall to Clark's Pond and out to the Fore River.

When the management district was formed two years ago, landowners of an acre or more of impervious surface were given a choice for how to meet Maine Department of Environmental Protection standards created to restore the stream to former health. They could obtain individual permitting or join the district under a general permit, at a cost of $3,000 per acre, for 10 years.

"We operate on a cash basis because it was really important to the landowner that we not be taking out loans and that sort o stuff," said Pinard, pointing to work on settlement basis at Texas Instruments slated for next year, as well as a 2014 mitigation project around the Mall.

"This is a real public-private partnership," said Pinard. "We want to do what's best for the stream but we also want to do what's best for the property owners in the district."

According to Pinard, only one property owner, who controls four of the 127 district parcels, is not in compliance. Of the rest, one, the MTA Administrative Building, already has met state standards, while three – UPS, the Wyndham Hotel and Capital Automotive – have pressed on with individual permits in progress when the Long Creek Management District was launched. The remaining 119 parcels fall under district auspices, said Pinard.

But given the amount of development surrounding Long Creek, Sable Oaks presents some of the best available wildlife habitat in the watershed.

"Preserving that habitat and improving the water quality in Long Creek, made joining Audubon's Cooperative Sanctuary Program a win-win for both the watershed and the golf course," said TenEyck.

That's true in more ways than one. Because it is creating the buffer zone on its own dime, the golf course will get a discount on its watershed dues.

"Instead of Long Creek having to come in and do these plantings, they're doing it, so they get that in-kind value," said Pinard.

However, Sable Oaks also got to make a trade with the Department of Environmental Protection, which allowed TenEyck to cut back some areas along Long Creek had become overgrown with "larger woody materials that were obstructing the playability of the golf course," particularly at the course's signature 14th hole.

"The Long Creek Advisory Board approved the design plan for new plantings on Hole 13, and DEP allowed me to do what I needed to, to make the restoration efforts easier," said TenEyck.

At one time, said TenEyck, the greens on Hole 13 were tended right up to the edge of Long Creek. That made life easy for golfers, who could easily recover the results from an errant swing. Now, those dimpled balls, which can run between $3 and $5 each, are lost forever when they fall into a newly created "enhanced riparian zone" that extends 25 feet from the stream. That and another 25-foot-long "no-mow zone" are designed to slow runoff into the creek, absorb chloride and other toxins and shade the stream bed, to both lower water temperature and increase dissolved oxygen.

"It all helps the stream act more like a stream," said Pinard. "The warmer the water is, the less oxygen it can hold."

TenEyck credits Sable Oaks owner Ocean Properties with getting on board with the mitigation project, along with part-time landscaper Al Hardy, the course's "No. 1 ecologist."

"The long-term goal for Sable Oaks is to have the water cleaner when it exits the golf course that it was when it came in," he said.

But then there's the matter of how the change will affect players. Sure, says Bell, the 13th hole with a wild swath through the middle of it is nicer to look at, but as TenEyck notes, it may take an extra stroke to make it over the stream, from a newly created "drop zone" for balls lost in the off-limits rough.

"I don't know how much some will like that," said TenEyck, "but I haven't been giving out my cell number."

County farm fills East Coast's broccoli niche

BY **JAMES MCCARTHY**

11/12/2012

PHOTO/JAMES MCCARTHY

Lance Smith oversees a conveyor space where broccoli bunches are wrapped.

If you go into a supermarket anywhere east of the Mississippi River, odds are high the broccoli displayed in the fresh produce section came from Smith's Farm's fields in Aroostook County … or its fields in northeast Florida.

Of the three retail broccoli growers in Maine selling beyond local markets, two are located in Aroostook County. And of those two, Smith's Farm, by far, is the largest, with a distribution range extending to the Mississippi River. But you won't see the Smith name on that broccoli bunch, only a stag on the rubber band holding the stalks together.

Make no mistake, though, this sixth-generation farming family based in Presque Isle is proud of its broccoli, potatoes and grains. The Stag brand has been the farm's premium label for more than 50 years — initially for potatoes but increasingly over the last 25 years for broccoli as well. What it brands, according to the company's website, is quality, freshness and the integrity of the Smith family's farming practices.

How then does the Stag brand help Smith Farms, if consumers have no way of connecting the label to the family name?

“That's a good question,” replies Tara Smith Vighetti, a sixth-generation family member who serves as Smith's Farm's director of marketing. “What we've found based on research is that our 'customer' isn't necessarily the buyer at the grocery store: It's the people who make the buying decisions at the retail level. Our retail buyers along the East Coast know our story, our legacy, our name and brand. We do a lot to let them know who we are.”

Smith's Farm Inc./H. Smith Packing Corp.

Address: 99 Fort Road, Presque Isle
Founded: 1859
Co-owners: Lance Smith, Greg Smith, Emily Smith, Zachary Smith, Tara Smith Vighetti
Employees: 25 year round; 250 seasonal workers
Acres in production: 4,000 broccoli; 3,000 in potatoes, grains
Annual revenue: $25 million gross

A September visit to the Smith's Farm's processing and distribution center in Westfield — operated by the family's sister company H. Smith Packing Corp. — drives home the point that potatoes aren't the only crop grown in Aroostook County, although 90% more acreage is devoted to potatoes there than broccoli, according to the Maine Department of Agriculture, Conservation and Forestry.

“We're lucky we found a niche for a product that people wanted,” says Lance Smith, who with his cousin, Greg, represents the fifth-generation of Smiths farming in The County. “People can have good confidence of our broccoli's freshness and quality. The air is clean, the water is clean and it's harvested so close to the markets it's in the grocery store only a day or two after it's harvested. We've spoiled the buyers in New

Contact: 800-393-9898
www.smithsfarm.com

England. The broccoli order that came in at 11 a.m. was harvested this morning. We'll ship it out by truck at 4 p.m. It will be in Boston at 3 a.m. and in the store that very day."

To which Greg Smith adds: "The 'impossible' is done immediately. Miracles take a little longer."

At the Westfield distribution center, trucks line up at mid-morning waiting to be loaded with iced boxes of broccoli cut that morning. Inside the facility, a front loader deposits ice into a pulverizer, which transforms it into a watery slush that's funneled into a box of broccoli waiting below. The slush pours in for a few seconds, then the box moves mechanically onto a conveyor, replaced by a new box that will get the same treatment. As water drains from the doused box, the remaining slush forms an icy armor around the broccoli.

It's fast, efficient and locks in the fresh flavor that consumers along the East Coast take for granted when they select a bunch of broccoli from the produce bin at their local supermarket. Other truckloads will ship broccoli that's shrink-wrapped and iceless, the preference of buyers in Southern supermarkets. Broccoli will ship out in 14- and 18-count bunches, as crowns, Asian crowns and florets — the various cuts catering to consumer preferences.

Lance Smith points to a stack of boxes to be loaded into the trailer of a waiting truck. Inside are broccoli crowns, the preferred cut for Smith Farm's growing Asian market in the Chinese communities of Boston, New York City and Philadelphia. The label is written in Chinese. He acknowledges the company hired a translator for that specialty job.

"There's a huge market for broccoli crowns," Smith says. "We do a lot of business in New York City. We've got two or three tractor trailers delivering broccoli into [Chinatown] each day."

Each carton heading out of the H. Smith Packing Corp.'s distribution center has a bar code conveying specific information about the broccoli packed inside: the field where it came from, crew that did the harvesting, date and time. That way, if there's ever a problem, it can be pinpointed back to the particular shipment and source quickly. It facilitates the quality-control review that will follow. Smith's Farm is all about freshness and consistency, says Lance, and holding everyone accountable for quality — from the field workers to packers to the truckers delivering the produce to the market — is how the Stag brand keeps its standing in the market.

He says Smith's Farm's proximity to the large metropolitan markets of the East Coast gives it an edge over its California competitors whose produce might take four or five days to arrive. But that competitive edge is never taken for granted.

"When we say 'It ships today,' we have to know we can fulfill that promise," Lance says, noting that his daughter Emily is the one who coordinates that aspect of the family business. "When we say 'yes' we mean 'yes.'"

East Coast markets within overnight delivery range extends to mid-state New York and eastern Pennsylvania and south to coastal Delaware, Maryland and Virginia. Second-day delivery extends through western Michigan and Ohio down through Kentucky, Tennessee and South Carolina. By day three, Smith's Farm broccoli has been delivered all the way to the Mississippi River.

Walter Whitcomb, commissioner of the Maine Department of Agriculture, Conservation and Forestry, says there's no question Smith Farms enhances Maine's reputation as a "place to grow a good product."

"They're a significant broccoli grower east of the Mississippi — if not the most significant," he says. "They understand marketing. They've worked hard on finding varieties that suit Maine's climate and soils and meet the market's needs. They think 'big' and have made the investments they needed to make. They certainly are among the best. They take a lot of pride in what they do."

Greg Smith says his family originally got into the broccoli business in the 1980s because it's a good rotation crop with potatoes. When it became obvious that broccoli was valuable in its own right as an agricultural crop, the company expanded the acreage devoted to it. There are now roughly 4,000 acres devoted to broccoli — out of 7,000 farm acres — with a crop rotation cycle ranging from three to seven years that alternates broccoli, potatoes, barley, wheat and soybeans.

Additional acreage is leased from neighboring potato farms (which, in turn, lease land from Smith's Farm for their crop rotation needs), rounding out the 16,000 acres Smith Farms requires to maintain a 4,000-acre broccoli rotation.

Each rotational crop adds nutrients to the soil needed by the next crop in the cycle — thereby reducing fertilizer costs. It also helps to control erosion by providing ground cover when broccoli or potatoes aren't planted.

Barley also happens to be a good cash crop for Smith's Farm. Much of it is sold to the Canada Malting Co., which uses it to produces malt for the brewing, distilling and food markets. Grain not used by the brewing market can be sold to Pineland Farms Natural Meats and Aroostook cattle farmers as feed.

The farm uses a mix of old and new techniques. Global-positioning systems guide tractors when plowing and planting broccoli, ensuring that the rows are perfectly straight for optimum crop maintenance and harvesting. The resulting efficiency lowers overall costs and improves erosion control in the acreage devoted to broccoli each season.

Harvesting is another story: There's no technology invented yet that can do better than an experienced harvester with a razor-sharp knife cutting the broccoli one stalk at a time.

“You still need eyes to tell if the broccoli is ready,” says Lance.

For that work, Smith's Farm flies in between 150 to 250 migrant workers from California to do its broccoli harvesting in The County. The migrant workers, some of whom have been harvesting broccoli for Smith's Farm for 20 years, are highly skilled specialists who work six days a week, making $10 to $12 an hour, with housing and transportation provided. Cutters cut, packers pack, boxers box: That's what the workers prefer, Lance Smith says, expressing surprise that no one switches jobs to break the monotony.

“They like working in Maine, they can make more money here,” he says, noting that in California the workers have to pay their own transportation and housing costs.

The Smith family isn't hung up on formal job titles. Lance and his daughters Emily and Tara, his cousin Greg and Greg's son Zachary are identified simply as the “farm team.”

Even so, Lance acknowledges each family member brings particular strengths to the team and they've naturally gravitated to the assignments best suited to them. He credits the up-and-coming generation with helping Smith's Farm keep pace with the complex challenges of following sustainable farming practices while also keeping a close eye on costs, quality control and marketing.

“They are a lot better versed in the science of agriculture than we are,” he says. “You've got to be able to look at the numbers, look at the cash flow. There's a huge investment out there.”

The weather, not surprisingly, is a critical variable that can suddenly put the brakes on a season that had been going well, or turn those blue-green broccoli fields into a bumper crop. And Smith's Farm is additionally challenged in sustaining operations in Maine — where the harvest typically runs from July through early November — and its farm in Florida, where broccoli is grown December through April.

In 2001 Smith's Farm started growing broccoli in northeastern Florida to close what had been a seventh-month opening for its California competitors to sell broccoli in Eastern grocery stories. Now Smith's Farm broccoli is out of the market only during April, May and June. Lance Smith says the Florida operation carries its own set of weather-related challenges.

“It can be 80 degrees today, 25 degrees tomorrow and two days later back up to 85 degrees,” he says.

On Oct. 29, as the remnants of Hurricane Sandy bore down on Maine, Smith's Farm completed its broccoli harvest in Aroostook County. The final accounting hasn't been completed, but Tara Smith Vighetti says it looks like it was an “average year” for broccoli.

Standing at the edge of a 140-acre field of broccoli being harvested in early September, her father gestures across the field and notes his father's potato farm was about that size. His gaze takes in that blue-green field of broccoli and The County's wide-open vistas and gently rolling hills that surround it.

“My father would be just flabbergasted to see the acreage we have now,” he says.

October 27. 2012 3:00PM

Print Page

New Web feature helps protect pesticide-sensitive areas

- The Colorado Department of Agriculture is proud to announce the implementation of Driftwatch, a web-based program designed to help protect pesticide-sensitive crops and habitats in Colorado.

LAKEWOOD, Colo. – The Colorado Department of Agriculture is proud to announce the implementation of Driftwatch, a web-based program designed to help protect pesticide-sensitive crops and habitats in Colorado.

"Colorado is the eighth state to integrate the Driftwatch program; it was developed to help pesticide applicators, specialty crop growers and stewards of at-risk habitats to communicate more effectively to protect pesticide-sensitive areas," said CDA's Pesticides Program Manager, John Scott. "The Colorado Department of Agriculture regulates pesticide distribution and use in the state and Driftwatch will be a powerful tool to help protect the environment."

Managers of ecologically sensitive areas and owners of commercial fields and apiaries may register the areas on Driftwatch. Pesticide applicators can then log in to see the location of those sites. Driftwatch is not intended to be a registry for homeowners or sites less than half an acre of agriculture production.

The site features an easy-to-use Google Map interface that clearly shows applicators the locations of registered areas so they can take the appropriate precautions and, if need be, communicate with a Driftwatch participant before they spray.

Some of the sensitive crop areas, or data layers, that CDA intends to register in Colorado include beehives, certified organic crops, fruits, grapes, nursery crops, pumpkins, melons, tomatoes and vegetables. The Department will review and consider adding additional sensitive sites upon request by growers or applicators.

Registration is voluntary and simple. For agricultural producers, it involves going to the Colorado Driftwatch site, which will be accessible through www.Driftwatch.org or through CDA's website at
http://www.colorado.gov/cs/Satellite/ag_Plants/CBON/1251631971141
where they will be directed how to register their field sites, commodity and contact information. When commercial applicators register, they will designate the area within the state they perform work, which will allow them to see Driftwatch participants located within that area. Applicators can also receive an email notifying them of newly registered growers in their designated area.

Driftwatch was designed in 2008 by staff from the Purdue University Agricultural and Biological Engineering and Agricultural Communications departments with input and support from Purdue University Cooperative Extension Specialists.

Portland Press Herald

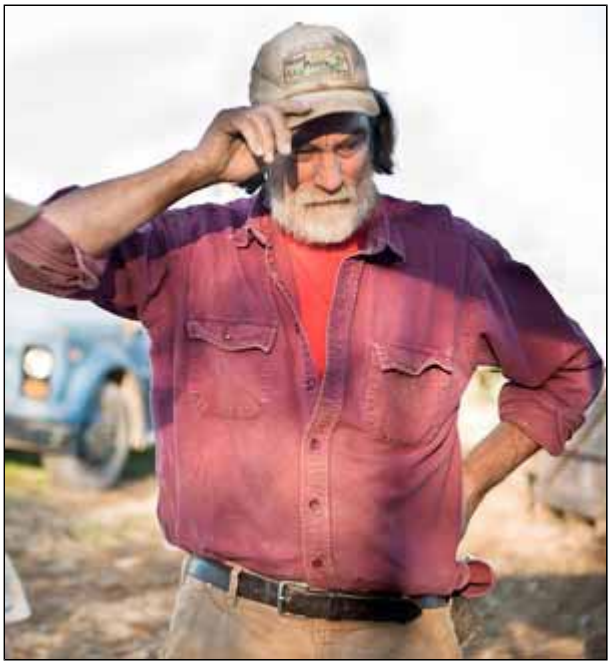
November 23

Maine farmers, others have court date to refute Monsanto ruling

At issue in the appeal is Monsanto's right to hold seed patents and farmers' need to be exempt from lawsuits.

By Avery Yale Kamilaakamila@mainetoday.com
Staff Writer

A lawsuit filed by a nationwide consortium of farmers against the chemical giant Monsanto concerning genetically modified seeds is headed for court again.



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Jim Gerritsen, Maine potato-seed farmer and president of the national Organic Seed Growers and Trade Association.

Contributed photo

The U.S. Court of Appeals for the Federal Circuit in Washington, D.C., will hear oral arguments in the case on Jan. 10 and is expected to rule within three months of the hearing.

The case questions Monsanto's legal basis for genetically modified seed patents, and seeks blanket protection from patent-infringement lawsuits for farmers, should their crops be contaminated through unwanted pollination by Monsanto's genetically altered plants. The plaintiffs include Maine farmers.

By law, certified organic crops cannot contain genetically modified material.

While most of the plaintiffs are organic farmers, some are conventional farmers who farm with seed that hasn't been genetically modified and face the same risks of contamination.

Genetically modified seeds are protected by patents. Farmers who grow genetically modified crops must buy new seeds each year, and cannot use traditional seed-saving practices.

In February, U.S. District Judge Naomi Buchwald of the Southern District of New York dismissed the case brought by the national, nonprofit Organic Seed Growers and Trade Association, which is based in Washington, Maine, and whose board president is a Maine potato-seed farmer, Jim Gerritsen of Wood Prairie Farm in Bridgewater.

The trade association seeks to have the judgment reversed and the case sent back to federal district court. Monsanto will argue that Buchwald's decision should stand.

The lawsuit was filed in March 2011 by the trade association and more than 70 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.

In dismissing the case, Buchwald acknowledged that some of the plaintiffs had stopped growing certain crops for fear of being sued, but ruled that the plaintiffs lacked standing to bring the lawsuit.

The judge also 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."

The plaintiffs claim that Buchwald ignored Supreme Court precedent relating to intellectual property law and patent infringement litigation.

Calling the case one of basic property rights, Gerritsen said, "what our briefs show is that (Buchwald) committed certain legal and factual errors that led her to the wrong conclusion and led her to dismiss the case."

The trade association's brief names specific farmers who have stopped growing certain crops for fear of contamination and subsequent lawsuits by Monsanto. The brief also names plaintiffs, including Maine-based Fedco Seeds, that have discovered unwanted genetic contamination when they have sent their seed out for third-party testing.

St. Louis-based Monsanto has maintained throughout the case that it doesn't sue farmers whose crops are inadvertently contaminated by its genetically modified seeds. Representatives of Monsanto did not return calls seeking comment on Friday.

In a statement issued after Buchwald dismissed the lawsuit, Monsanto said the judge's ruling "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."

According to a report from the Center for Food Safety, a nonprofit environmental advocacy group based in Washington, D.C., Monsanto annually investigates about 500 farmers for possible patent infringement. The same report says Monsanto sued 144 farmers from 1997 to 2010, and settled 700 cases out of court in that period.

The trade association is raising money to enable its member farmers to go to Washington, D.C., to hear the oral arguments in January. During oral arguments this year in New York, 60 farmers from more than 20 states and Canadian provinces filled the courtroom.

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BANGOR DAILY NEWS

Maine farmers get second chance in court against giant Monsanto

By Whit Richardson, BDN Staff
Posted Nov. 23, 2012, at 6:06 p.m.

WASHINGTON, Maine — A lawsuit brought by a Maine-based agriculture group against global giant Monsanto will receive its day in a federal appeals court after being dismissed by another judge earlier this year.

Oral arguments in the case brought by the Organic Seed Growers and Trade Association, which is based in Washington, Maine, against St. Louis-based Monsanto will be heard Jan. 10, 2013, at the Court of Appeals for the Federal Circuit in Washington, D.C., according to court documents.

The organic seed growers association, along with 82 plaintiffs, in March 2011 sued Monsanto in federal district court in New York. The suit challenges the validity of several patents the company holds for genetically modified crops.

The farmers are also seeking protection from lawsuits in case Monsanto's genetically modified seed inadvertently contaminates their crops through natural causes such as seed drift and cross pollination.

However, federal judge Naomi Buchwald in February dismissed the suit before it went to trial, saying the plaintiffs' claims of being in fear of patent infringement lawsuits from Monsanto were unsubstantiated and that "these circumstances do not amount to a substantial controversy and that there has been no injury traceable to defendants."

Jim Gerritsen, a seed potato farmer in Bridgewater and president of the organic seed growers association, told the Bangor Daily News the judge made "numerous legal and factual errors" that led to her decision to dismiss the case.

The newly scheduled oral arguments will give the plaintiffs, who are represented pro bono by the nonprofit Public Patent Foundation, an opportunity to explain to three appellate court judges how "reversible errors were committed" and why the case should be allowed to continue, Gerritsen said.

The appellate judges also will consider two amicus briefs — one by 11 law professors and the other by 14 nonprofit consumer and food safety nonprofits — that were filed in support of the farmers' position.

The plaintiffs need two of the three judges to vote in favor of sending the case back to district court, Gerritsen said. "We hope we are given a fair hearing by honorable judges that will take [Judge Buchwald's] ruling, critique it and put it on a level field," he said.

Gerritsen said the case is extremely important because of the implications it could have on a farmer's ability to farm how they please without fear of being targeted by a company such as Monsanto, which Gerritsen points out has a 75-person in-house legal team that independent farmers can't compete against. Gerritsen said Monsanto has sued farmers 144 times, "and in each and every one they make the farmer out to be the villain."

"The fact is we are all at jeopardy, our livelihoods are at stake," Gerritsen said, adding that Monsanto has a record of intimidating and suing farms where their genetically modified crops have shown up. "If Monsanto can gain ownership of our crops when they contaminate them, how can we possibly continue farming?"

For its part, Monsanto claims it is not its policy "to exercise [their] patent rights where trace amounts of [their] seed or traits are present in [a] farmer's fields as a result of inadvertent means," according to court documents.

The 83 plaintiffs are made up of independent farms, seed companies and agricultural associations around the country. Plaintiffs from Maine include the Maine Organic Farmers and Gardeners Association in Unity and Fedco Seeds in Waterville. Gerritsen said the plaintiffs collectively represent approximately 300,000 people and probably 25 percent of all certified organic farms in the United States and Canada.

Now that the appeals court date has been set, Gerritsen is busy reestablishing the Farmer Travel Fund, which will help fund farmers' ability to take time away from their farms to attend the oral arguments in Washington, D.C.

"Farmers in general don't get away from their farms, so we will need all this time to just prepare a plan so the farmers can get away and travel to Washington, D.C.," Gerritsen said.

The farmers will attend the event "to observe this oral argument and bear witness to the functioning of the judicial process, and also show by their presence how important this is to them and their livelihood and their communities, and that it's not just some academic exercise in refining patent law."

http://bangordailynews.com/2012/11/23/business/maine-farmers-get-second-chance-in-court-against-giant-monsanto/ printed on November 26, 2012

Seeds of Discontent

Newsletter Weight:
5

By Virginia M. Wright

For nearly three decades Jim Gerritsen quietly farmed organic seed potatoes in Aroostook County. These days he is in the international spotlight as the spokesman for the lead plaintiff in a lawsuit against Monsanto Corporation, the world's largest producer of genetically modified seeds.

Gerritsen is president of the Organic Seed Growers and Trade Association (OSGATA), whose lawsuit asks to have Monsanto's patents for genetically altered seeds invalidated.

OSGATA, which has been joined in lawsuit by eighty-two seed businesses, family farmers, and trade organizations, including the Maine Organic Farmers and Gardeners Association, wants court protection for farmers against possible lawsuits for patent infringement should Monsanto's transgenic crops be found in their harvest as a result of accidental cross pollination.

Monsanto makes its transgenic seeds by transferring the genes of one species into the DNA of another. Among its products is Roundup Ready transgenic seed, which grows crops that tolerate the company's weed killer, Roundup.

The agriculture giant's reputation for suing or threatening to sue farmers for patent infringement has been widely reported; nevertheless, U.S. District Judge Naomi Reice Buchwald dismissed OSGATA's lawsuit in February, saying the plaintiffs' allegations were unsubstantiated. The case is now before the United States Court of Appeals for the Federal Circuit in Washington, D.C. Gerritsen expects oral arguments will likely be heard this winter.

In the meantime, Gerritsen, who was named an Utne Reader visionary in 2011 for his leadership on the OSGATA case, has been traveling around the country, speaking about the lawsuit and its relationship to other concerns about genetically modified foods, including safety, consumer choice, and farm independence.

Tell us a little about your farm, Wood Prairie Farm.

We've been farming organically in the town of Bridgewater for thirty-six years. We've got a small farm — as a potato farm in Aroostook County, we're not even on the radar screen. We're surrounded by woods. Route 11, twenty-five miles west of here, interrupts the north Maine woods, but then they go on all the way to Quebec City. That kind of isolation is ideal for growing anything, but it's particularly good for growing organic seed, so that's what we've focused on for the last twenty-five years — primarily seed potatoes, but we also grow other seed crops like corn, peppers, tomatoes, squash. We're blessed to be in Aroostook County. It is one of the world-class areas for raising potatoes. You can't find anyplace better.

Why is isolation a good thing?

Two things come to mind immediately. Isolation is important for genetic purity of seed growing. You need geographical isolation to prevent cross-pollination of crops like corn or squash, whose pollen will travel great distances.

The other aspect is that a farmer's troubles seem to come from his neighbor. It's great for, say, a potato farmer to have a buffer between him and another farmer's potatoes because that's where a pest like the Colorado potato beetle is going to come from. The more isolated you are, the more protected you are.

That ties into the topic of OSGATA's lawsuit against Monsanto, which stems from farmers' concerns that transgenic seeds might contaminate their crops. How are organic crops threatened by genetically engineered (GE) crops?

It all comes down to the lessons that we learn back in high school biology class: Each farm crop has its own biological qualities that are at stake when it comes to reproduction. Pollen can be dispersed great distances by wind and insects, and if pollen goes off one farm to another, that can cause trouble.

I'll give you an example: One of the crops we grow is organic sweet corn seed. Genetic purity is necessary for good quality seed. If a neighbor were to grow a type of corn that a cow would eat either for silage or dry corn, the pollen from that corn not only could make our sweet corn genetically impure, but it also would lower its quality because it would be less sweet and kind of starchy.

When you get into genetically engineered crops, you're facing a whole different level of concern. Many countries, as well as the organic and non-GMO [genetically modified organism] conventional markets in this country, will not accept crops that have GE content. A number of the farmers who are plaintiffs in our lawsuit are conventional farmers who do not want to grow GMO crops, and in many cases they have developed a nice market niche for a crop like corn that is free of transgenic content.

Genetic engineering is a process that would never, ever be found in nature. For example, researchers once spliced a gene from a flounder fish into a strawberry in order to try to give the strawberry increased frost resistance. Well, obviously if you set a strawberry and a flounder in a room for a million years you would never get any crossing going on. This concerns us because the biotech companies say the kind of genetic manipulation they're doing is what farmers have been doing for thousands of years, and that is absolutely false. These are humanly engineered in the laboratory.

I for one have no confidence in the studies that the Food and Drug Administration (FDA) relies upon in their regulatory decision-making process. Many of the federal regulators come from the biotech industry — they spend a few years supposedly regulating the crop, then they go back to the biotech industry. The other factor is, the federal government has no research function of its own. They simply do a document review of what the biotech companies provide to them.

What kinds of crops are most vulnerable to accidental pollination by genetically modified plants?

Canola is wicked for reestablishing itself. They've now found that GMO canola is cross-breeding with wild species of canola. It is getting way out of hand in North Dakota, Manitoba, Saskatchewan. These are weeds that grow in the shoulders of the road and at the end of culverts. They're spreading like mad. It's because the pollen of canola is extremely mobile, as it is for corn, and it can spread quite a distance.

There are some crops not yet approved by the federal government whose potential for spread is mindboggling. In Oregon, GMO bentgrass was found growing fifteen miles from the research plot where it is being developed. Imagine if you were a seed farmer in Oregon raising bentgrass seed and you wanted nothing to do with GMOs. You'd have to be assured that there wasn't GMO bentgrass being grown within a radius of fifteen or twenty miles of your farm or else you've got a big problem, especially if you are selling your bentgrass seed to Europe or Asia, where they do not allow it. You'd have no market; the value of that crop would be evaporated.

[The bentgrass to which Gerritsen refers was discovered in eastern Oregon's Malheur County in 2010, according to the Capital Press, an agriculture newspaper. It tested positive for a transgenic gene that makes it resistant to Roundup, Monsanto's weed killer. Oregon State University weed scientist Carol Mallory-Smith speculated that the plants originated from seed that had spread from a field near Parma, Idaho, where Roundup Ready bentgrass is being developed for golf courses by Scotts Co. and Monsanto.]

In dismissing OSGATA's lawsuit, Judge Buchwald said the farmers were creating a controversy where none exists.

We feel her ruling is filled with factual and legal errors that led her to dismiss our case, and that's the basis on which we are filing our appeal. She had done inadequate research on the entire reason Congress passed the Declaratory Judgment Act. We are a classic case. She said we haven't suffered any economic harm, and in our briefs we expressed to her, yes, we have. Farmers within our plaintiff group have suspended growing crops like organic corn, organic canola, and organic soybeans because of this certainty that they will be contaminated by Monsanto seed being grown by their neighbors.

The judge actually acknowledged that this contamination is going to occur, but then for whatever reason she did not make the connection that possession is the problem. Here's how patent law works: Once a corporation is given a patent on something — in this case, genetically engineered crops — they have total ownership of that material. The only way someone can be in possession of that is if they have signed a licensing agreement and paid royalty on that licensed

right to use. It doesn't matter whether you intend to possess. It doesn't even matter whether you're knowledgeable of possession. If you are in possession without a licensing agreement, you are in violation of a patent right and you are subject to patent infringement violation. That's why we needed to go court. We have no protection.

The lawsuit seeks to invalidate Monsanto's patents on genetically altered seeds. That would protect farmers from patent infringement lawsuits, but how does it protect your crops from being contaminated by transgenic seeds?

Here's the situation right now: If our corn crop should become contaminated by Monsanto's corn, there is zero value in the organic market place for it. We might as well throw it away. So that's one immediate impact.

But if I should say, "Jeepers, this is threatening the survival of our farm. This corn seed crop is so important to us, and it wasn't right for them to trespass onto our farm. I'm going to get a lawyer and I'm going to file a lawsuit to try to recover damages." We are concerned that Monsanto will countersue us and say, "You've got our technology. You don't have permission to have our technology, and we're suing you for patent infringement." Then we go bankrupt trying to defend ourselves in this secondary lawsuit.

We believe that the contamination is occurring, and farmers are scared to death to seek compensation for fear they'll be countersued. Once we gain court protection under the Declaratory Judgment Act, we will have the legal tools that we need to properly get compensation if Monsanto hurts us.

Why should consumers be concerned about this?

The demand for organic food is steadily increasing. People are putting two and two together, and they're seeing that the food that they purchase is an important part of their family's well-being and health. So from the consumer's standpoint, this protection of farmers is essential to protecting their right of access to something besides genetically engineered foods.

There are campaigns around the country, including Maine, to require labeling of genetically engineered food. Why do you think mandatory labels are necessary? What don't consumers know about the food they are buying?

They don't know if it's been genetically engineered or not. National polling in the United States has consistently indicated that 90 percent of the American public favors mandatory labeling of GE crops — 90 percent! It goes across all party lines — Republicans, Democrats, independents are in favor of this. And why? It's because America is a democracy. Democracy is based on the idea of informed citizenry making the best decisions. To deny that kind of information to Americans is un-American and it's outrageous. Over fifty countries, including China, have mandatory GMO labeling, but in America, which prides itself on being the world's leading democracy, we don't. What's going on?

We have caloric intake on the labels. We have "be careful peanuts may have been used in this" labels. Many other things are on the labels. Many of us have concerns about genetically

engineered foods. In September, scientists at CRIIGEN (Committee for Research and Independent Information on Genetic Engineering) published the results of a two-year study, which found that rats fed a diet of Roundup-resistant corn or water containing Roundup [at levels permitted in drinking water and GM crops in the United States] developed increased amounts of cancerous tumors and had shortened lifespans. Here is the real interesting thing to me: The rats started to express these problems four months into the study. The studies that biotech submits to the FDA are based on ninety-day tests.

Here's another important thing to remember: When the biotech industry goes to the FDA, they say their crops are substantially equivalent to traditional crops and for that reason we shouldn't label them. Then the industry goes to the U.S. Patent Office and says, you know, this invention we have here is new and wonderful, it's different, and it deserves a patent. Which is it? Either it's new and different and deserving of a patent, in which case it must be labeled, or it's substantially equivalent and in that way not deserving of a patent.

How prevalent are genetically engineered crops in Maine?

There are about 25,000 acres of genetically engineered corn grown primarily in the dairy belt in central Maine. There are GMO soybeans, and there is some GMO canola being grown up here in Aroostook County. Sugar beets are another crop that has been made resistant to Roundup, and I would imagine they're making their way into Maine, but I have not heard any numbers. That's one of the problems: There is no requirement for farmers growing GMO crops to let their neighbors know or to file a report to the state. Since the crops look the same, you don't know if one is GMO or not. That doesn't seem fair. It seems reasonable that they should have to register so a neighbor could find out what's going on in their vicinity.

Currently there aren't any genetically engineered varieties of potatoes, which are one of Maine's biggest crops. Why is that?

Interestingly, genetically engineered potatoes were one of the first crops that were introduced in the mid-1990s by Monsanto. They called the product New Leaf. They had gene-sliced a bacterial toxin into a potato plant. The target insect was the Colorado potato beetle, a serious insect that plagues potatoes in every temperate potato-producing region in the world. The idea was that the bug would eat it and it would die. One of the two New Leaf seed potato propagation facilities was here in Maine, in Island Falls, so this was a big area for producing that early generation stock, and the crops were planted here.

After five or six years, New Leaf potatoes were withdrawn from the market. The problem is, when you gene-splice, it's not like the bacterial toxin stops at the leaf and doesn't go through the stem and tuber. Every single cell, including those in the tuber, had that bacterial toxin. As you might imagine, there was concern among the public — do we really want to be eating this? In the early 2000s McDonald's announced it would no longer be buying French fries made with New Leaf potatoes. Then McCain, the local French fry processor, stopped using GMO potatoes, and Monsanto voluntarily removed them from the market.

Here's something illuminating: Under Maine law, Monsanto was required to register those potatoes as a pesticide with the Maine Board of Pesticides Control. It met the legal definition of a pesticide, even though it was a potato that people were supposed to have confidence eating.

Do you find that Mainers are generally well informed about food quality and choices?
More so than other parts of the country, partly because we have a rural heritage and many people still have gardens. But there also has been a growing interest in food across the country in the last ten to twenty years. In the fifties and sixties, people got into the convenience of having the food prepared in the factory. Now the pendulum is swinging. People want to know more about their food. They've made the connection between their family's health and the food they consume.

Small family farms are on the rise in Maine. Do they stand to benefit if the labeling of transgenic foods become law?
Oh, absolutely, absolutely. You know, I don't want to feed GMOs to my family. The corporations like them because they control the seed supply and they gain royalty payments every time a farmer uses their seed. But from the standpoint of an organic farmer or a consumer who wants access to good food, GMO foods have not been proven to be safe.

As for the growth of small farms and farmers' markets, it's all tied together with the increased interest in food and eating healthy. We're a seed company, and the last five years have been the best years for the seed industry going back to World War II. More people are planting gardens, and people who already had gardens are doubling their size. These are good traditional Maine and New England values, the idea of self-reliance, cutting your own firewood, growing your own garden, making do for yourself.

For decades the numbers of farms in every state was going down, down, down. We have turned that around, and Maine is toward the top in terms of the number of new farms. These new farmers are the people you see at the farmers' markets in Portland, Yarmouth, and other places. They're highly educated, and they've decided they want a life with meaning. My wife and I came to that conclusion thirty-five years ago: This is a great place and a great way to raise our kids. We're in wonderful rural communities with wonderful hardworking neighbors. There's so much going for it that I think these young people are making the choice to farm in Maine because it's a great life.

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PLOS ONE

Increasing Cropping System Diversity Balances Productivity, Profitability and Environmental Health

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Abstract

Balancing productivity, profitability, and environmental health is a key challenge for agricultural sustainability. Most crop production systems in the United States are characterized by low species and management diversity, high use of fossil energy and agrichemicals, and large negative impacts on the environment. We hypothesized that cropping system diversification would promote ecosystem services that would supplement, and eventually displace, synthetic external inputs used to maintain crop productivity. To test this, we conducted a field study from 2003–2011 in Iowa that included three contrasting systems varying in length of crop sequence and inputs. We compared a conventionally managed 2-yr rotation (maize-soybean) that received fertilizers and herbicides at rates comparable to those used on nearby farms with two more diverse cropping systems: a 3-yr rotation (maize-soybean-small grain + red clover) and a 4-yr rotation (maize-soybean-small grain + alfalfa-alfalfa) managed with lower synthetic N fertilizer and herbicide inputs and periodic applications of cattle manure. Grain yields, mass of harvested products, and profit in the more diverse systems were similar to, or greater than, those in the conventional system, despite reductions of agrichemical inputs. Weeds were suppressed effectively in all systems, but freshwater toxicity of the more diverse systems was two orders of magnitude lower than in the conventional system. Results of our study indicate that more diverse cropping systems can use small amounts of synthetic agrichemical inputs as powerful tools with which to tune, rather than drive, agroecosystem performance, while meeting or exceeding the performance of less diverse systems.

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Introduction

One of the key challenges of the 21st century is developing ways of producing sufficient amounts of food while protecting both environmental quality and the economic well-being of rural communities [1,2]. Over the last half century, conventional approaches to crop production have relied heavily on manufactured fertilizers and pesticides to increase yields, but they have also degraded water quality and posed threats to human health and wildlife [3–6]. Consequently, attention is now being directed toward the development of crop production systems with improved resource use efficiencies and more benign effects on the environment [1,7]. Less attention has been paid to developing better methods of pest management, especially for weeds. Here we explore the potential benefits of diversifying cropping systems as a means of controlling weed population dynamics while simultaneously enhancing other desirable agroecosystem processes [8]. We focus on crop rotation, an approach to cropping system diversification whereby different species are placed in the same field at different times.

Rotation systems have been used for millennia to maintain soil fertility and productivity and to suppress pests, and can increase yields even in situations where substantial amounts of fertilizers and pesticides are applied [9,10]. Rotation systems also foster spatial diversity, since different crops within the rotation sequence are typically grown in different fields on a farm in the same year. Diversification through crop rotation can be an especially useful strategy in farming systems that integrate crop and livestock production. The addition of forage crops, including turnips and clovers, to cereal-based systems in northwestern Europe and England in the 1600s and 1700s enhanced nitrogen supply through fixation by legumes, and increased nutrient cycling due to greater livestock density and manure production. These changes allowed the intensification of both crop and livestock production and increased yields substantially [11,12]. Integrated crop–livestock systems remained widespread in northern Europe, England, and much of the humid, temperate regions of North America until the 1950s and 1960s, when increased availability of relatively low-cost synthetic fertilizers made mixed farming and nutrient recycling biologically unnecessary and specialized crop and livestock production more economically attractive. In recent years, there has been interest in reintegrating crop and livestock systems as a strategy for reducing reliance on fossil fuels, minimizing the use of increasingly expensive fertilizers, and

limiting water pollution by nutrients, pathogens, and antibiotics [13,14].

Weeds are a ubiquitous and recurrent problem in essentially all crop production systems, and chemicals applied for weed control dominate the world market for pesticides [15]. With the introduction of crop genotypes engineered to tolerate herbicides, especially glyphosate, and with the continuing availability of older, relatively inexpensive herbicides, such as atrazine, successful weed management in conventional crop production systems has been largely taken for granted since the mid-1990s. Now, however, with expanded recognition of herbicides as environmental contaminants [4] and the increasing prevalence of herbicide resistant weeds [16], there is an important need to develop weed management strategies that are less reliant on herbicides and that subject weeds to a wide range of stress and mortality factors [17]. We believe that cropping system diversification may play an important role in the development of such strategies.

Here, we report the results of a large-scale, long-term experiment examining the consequences of cropping system diversification on agronomic, economic, and environmental measures of system performance. The experiment was conducted during 2003–2011 in Boone County, Iowa, within the central U.S. maize production region, and comprised three contrasting cropping systems varying in length of crop sequence, levels of chemical inputs, and use of manure. We compared a conventionally managed 2-yr rotation (maize-soybean) that received fertilizers and herbicides at rates comparable to those used on surrounding commercial farms with two more diverse cropping systems: a 3-yr rotation (maize-soybean-small grain + red clover) and a 4-yr rotation (maize-soybean-small grain + alfalfa-alfalfa) managed with reduced N fertilizer and herbicide inputs and periodic applications of composted cattle manure. Triticale was used as the small grain crop in 2003–2005; oat was used in 2006–2011. The 2-yr rotation is typical of cash grain farming systems in the region, whereas the 3-yr and 4-yr rotations are representative of farming systems in the region that include livestock. Details of the experimental site, management practices, sampling procedures, and data analyses are provided in the online SI section (Text S1, Figure S1, Tables S1-S4).

A central hypothesis framing our study was that cropping system diversification would result in the development of ecosystem services over time that would supplement, or eventually displace, the role of synthetic external inputs in maintaining crop productivity and profitability. Based on this hypothesis, we predicted that input requirements of the more diverse systems would initially be similar to that of the less diverse system, but would increasingly diverge from the less diverse system over time as the systems matured. We also predicted that crop yields, weed suppression, and economic performance of the three systems would be similar throughout the study. Finally, we predicted that reduced requirements for external synthetic inputs for pest management would result in a lower toxicological profile of the more diverse systems compared to the less diverse system.

Results

Crop Yields and Net Profitability

Cropping system diversification enhanced yields of maize and soybean grain and system-level harvested crop mass (grain, straw, and hay) while maintaining economic returns. The most parsimonious linear statistical models for each of these measures of system performance contained terms for main effects of *year* and *system*, but no interaction term ($AIC_{with\ interaction} = 319$; $AIC_{no\ interaction} = 315$). Over the 2003 to 2011 period, maize grain yield was on average 4% greater in the 3-yr and 4-yr rotations than in the 2-yr rotation (means for the 2-yr, 3-yr and 4-yr rotations are hereafter referred to as μ_2, μ_3 and μ_4, respectively; $\mu_2 = 12.3 \pm 0.1$ Mg ha^{-1}; $\mu_3 = 12.7 \pm 0.2$ Mg ha^{-1}; $\mu_4 = 12.9 \pm 0.2$ Mg ha^{-1}; pre-planned 1 d.f. contrast of *system*: $F_{1,7} = 8$, $P = 0.03$), and similar in the 3-yr and 4-yr rotations (Fig. 1a). Soybean grain yield during the same period was on average 9% greater in the 3-yr and 4-yr rotations than in the 2-yr rotation ($\mu_2 = 3.4 \pm 0.07$ Mg ha^{-1}; $\mu_3 = 3.8 \pm 0.08$ Mg ha^{-1}; $\mu_4 = 3.8 \pm 0.08$ Mg ha^{-1}; $F_{1,7} = 11.3$, $P = 0.01$) and similar in the 3-yr and 4-yr rotations (Fig. 1b). Harvested crop mass, averaged over the various crop phases comprising each cropping system, followed a similar pattern to maize and soybean grain yields. Mean crop biomass for 2003 to 2011 was 8% greater in the 3-yr and 4-yr rotations than in the 2-yr rotation ($\mu_2 = 7.9 \pm 0.08$ Mg ha^{-1}; $\mu_3 = 8.5 \pm 0.1$ Mg ha^{-1}; $\mu_4 = 8.6 \pm 0.2$ Mg ha^{-1}; *system*: $t_6 = 5.1$, $P = 0.002$), and similar in the 3-yr and 4-yr rotations (Fig. 1c).

We examined system profitability by calculating net returns to land and management, which represent profits to a farm operation without accounting for costs of land (e.g., rent or mortgage payments), management time (e.g., marketing), and federal subsidies. Profitability was analyzed for two temporal periods. From 2003 to 2005, considered the "startup" phase for the study, there were no differences among cropping systems in net profit, either through an analysis of main effects of *system* ($\mu_2 = \$448 \pm 17$ ha^{-1}; $\mu_3 = \$402 \pm 17$ ha^{-1}; $\mu_4 = \$457 \pm 15$ ha^{-1}; $F_{2,6} = 0.12$, $P = 0.89$) or by pre-planned 1-d.f. contrasts (2-yr vs. 3-yr and 4-yr rotations: $F_{1,7} = 0.10$, $P = 0.77$) (Fig. 1d). From 2006 to 2011, the "established" phase of the study, there were again no differences among systems, either through main effects of *system* ($\mu_2 = \$953 \pm 36$ ha^{-1}; $\mu_3 = \$965 \pm 34$ ha^{-1}; $\mu_4 = \$913 \pm 26$ ha^{-1}; $F_{2,6} = 0.62$, $P = 0.57$) or by pre-planned 1-d.f. contrasts (2-yr vs. 3-yr and 4-yr rotations: $F_{1,7} = 0.03$, $P = 0.86$).

Stability of system performance over time, as measured through a comparison of variances for the various products of the system, was similar for maize grain yield ($F_{2,6} = 2.4$, $P = 0.17$), soybean grain yield ($F_{2,6} = 0.95$, $P = 0.44$) and net returns to land and management during the startup phase of the study, 2003 to 2005 ($F_{2,6} = 0.05$, $P = 0.95$). Two system products, harvested crop mass from 2003 to 2011 and profit during the established phase of the study, 2006 to 2011, showed considerable differences in system stability over time, but in contrasting ways. Variance in mean harvested crop mass was greater in the 3-yr and 4-yr rotations than in the 2-yr rotation ($\sigma_2{}^2 = 0.27$; $\sigma_3{}^2 = 0.60$; $\sigma_4{}^2 = 0.95$; $F_{1,7} = 16$, $P = 0.005$). Conversely, cropping system diversification was associated with lower variance in profit during the established phase of the study. Variance in profit from 2006 to 2011 was lower in the 3-yr and 4-yr rotations than in the 2-yr rotation ($\sigma_2{}^2 = 1.5 \times 10^5$; $\sigma_3{}^2 = 8.1 \times 10^3$; $\sigma_4{}^2 = 6.3 \times 10^3$; $F_{1,7} = 16$, $P = 0.005$).

Agrichemical, Labor and Energy Inputs

Application rates of the primary agrichemicals used in this study, manufactured N fertilizer ($F_{2,14} = 117$, $P < 0.0001$) and herbicides ($F_{2,14} = 287$, $P < 0.0001$), both showed strong effects of cropping system. Manufactured N fertilizer applications were higher in the 2-yr rotation than in the 3-yr and 4-yr rotations ($\mu_2 = 80 \pm 3$ kg N ha^{-1}; $\mu_3 = 16 \pm 3$ kg N ha^{-1}; $\mu_4 = 11 \pm 2$ kg N ha^{-1}; $F_{1,17} = 16$, $P = 0.005$), with the difference between systems increasing over the course of the study ($F_{2,14} = 11.6$, $P = 0.001$) (Fig. 1e). Herbicide application rates followed a similar pattern, with greater amounts of herbicide applied in the 2-yr rotation than in the 3-yr and 4-yr rotations ($\mu_2 = 1.9 \pm 0.06$ kg a.i. ha^{-1};

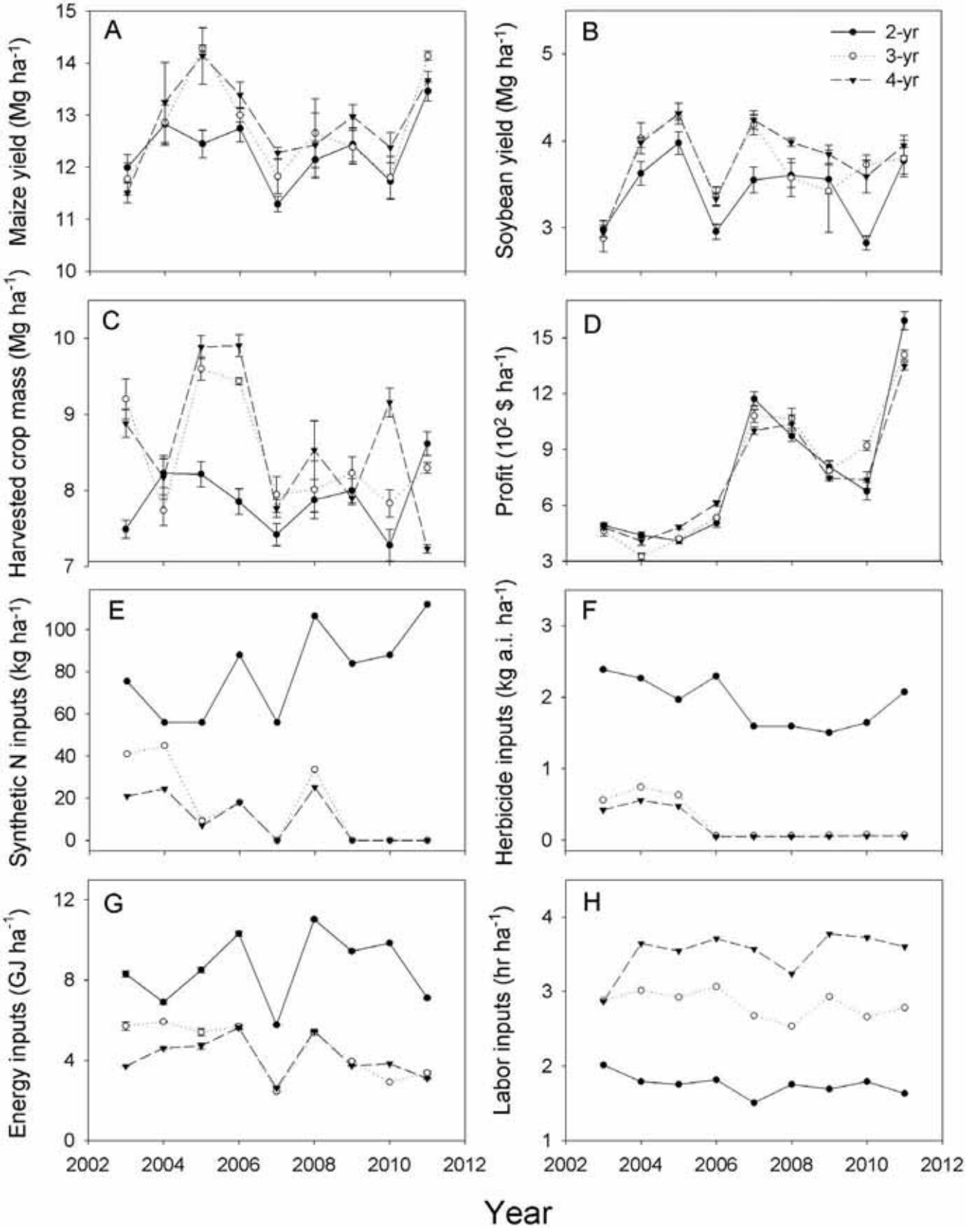

Figure 1. Cropping system performance over time. Annual performance of maize-soybean (2-yr), maize-soybean-small grain/red clover (3-yr), and maize-soybean-small grain/alfalfa-alfalfa (4-yr) cropping systems in Boone, IA, from 2003 to 2011. Performance metrics included: a) maize yield, b) soybean yield, c) rotation-level harvested crop mass, d) net returns to land and management, e) manufactured N fertilizer application rate, f) herbicide

application rate, g) fossil energy use, and h) labor requirements. Symbols represent the mean ± SEM of four replicate experimental blocks (N = 36 per cropping system).
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$\mu_3 = 0.26 \pm 0.05$ kg a.i. ha^{-1}; $\mu_4 = 0.20 \pm 0.03$ kg a.i. ha^{-1}; $F_{1,17} = 610$, $P < 0.0001$); differences among systems, however, did not increase over time (Fig. 1f).

Fossil energy use was strongly influenced by cropping system in both the startup ($F_{2,6} = 94$, $P < 0.0001$) and established ($F_{2,6} = 116$, $P < 0.0001$) phases of the study, with no difference in energy use between experimental phases ($F_{1,92} = 0.39$, $P = 0.53$) (Fig. 1g). From 2003 to 2011, inputs of energy were greater in the 2-yr rotation than in the 3-yr and 4-yr rotations ($\mu_2 = 8.6 \pm 0.1$ GJ ha^{-1}; $\mu_3 = 4.5 \pm 0.1$ GJ ha^{-1}; $\mu_4 = 4.2 \pm 0.04$ GJ ha^{-1}; $F_{1,7} = 55$, $P = 0.0001$). The partial correlations between energy use in a given cropping system and energy use in the maize phase of that rotation, taking into account the amount of N fertilizer applied to maize, were 0.94, 0.81 and 0.70 in the 2-yr, 3-yr and 4-yr systems, respectively (SI, Table S5). This indicated that synthetic N fertilizer use in the maize phase of the various cropping systems drove energy use within the maize phase, which in turn drove energy use by a given cropping system.

Demand for labor differed among the three cropping systems in both the startup ($F_{2,4} = 26$, $P = 0.005$) and established ($F_{2,10} = 299$, $P < 0.0001$) study phases, but followed a contrasting pattern to energy requirements (Fig. 1h). Labor inputs were more than 33% lower in the 2-yr rotation than in the 3-yr and 4-yr rotations from 2003 to 2005 ($F_{1,5} = 35$, $P = 0.002$) and from 2006 to 2011 ($F_{1,11} = 59$, $P < 0.0001$). Overall, there was a strong negative correlation ($r = -0.79$, $P < 0.0001$) between fossil energy and labor inputs over time in the three cropping systems.

Divergent Weed Management Systems

Two lines of evidence indicate that weeds were managed effectively in all three cropping systems in both the 'startup' and 'established' phases, in spite of reducing herbicide use by 88% in the 3-yr and 4-yr rotations compared to the 2-yr rotation. First, weed seedbanks declined at an equal rate in all study systems (Fig. 2a). Selection among linear mixed effects regression models incorporating temporal autocorrelation among seedbank measurements over time supported different intercepts (*system*: $F_{2,6} = 16.8$, $P = 0.0035$) but did not support inclusion of a *year* by *system* interaction term ($AIC_s = 182$; $AIC_{s*y} = 185$), thus indicating a common slope ($b_1 = -0.18$). For all three systems, the time to decline to 95% of the weed seedbank levels in 2003 was 16.6 years. Declines in weed seedbanks reflected a focus of management attention on the timing of weed management activities and herbicide choices in all three systems, as well as the increased number and diversity of stress and mortality factors present in the 3-yr and 4-yr rotations [8,21]. Higher densities of weed seeds in the 3-yr and 4-yr rotations, as indicated by their greater intercept values than for the 2-yr rotation (Fig. 2a.), were the result of poorer weed control in the 3-yr and 4-yr rotations during the set-up of the experiment plots in 2002.

The second line of evidence concerns weed biomass, which was very low in all three cropping systems for the duration of the study (Fig. 2b), never exceeding 0.3% of harvested crop mass. Weed biomass was the same within a given crop phase, regardless of the cropping system in which it occurred (main effect of *system*: maize, $F_{2,6} = 1.47$; $P = 0.30$; soybean, $F_{2,6} = 0.88$; $P = 0.46$; small grain, $F_{1,3} = 1.24$; $P = 0.31$). There were differences in mean weed biomass among cropping systems ($\mu_2 = 0.0003 \pm 0.00007$ Mg ha^{-1}; $\mu_3 = 0.0076 \pm 0.0012$ Mg ha^{-1}; $\mu_4 = 0.009 \pm 0.001$ Mg ha^{-1}; $F_{2,6} = 12.7$; $P < 0.007$). These differences arose mainly due

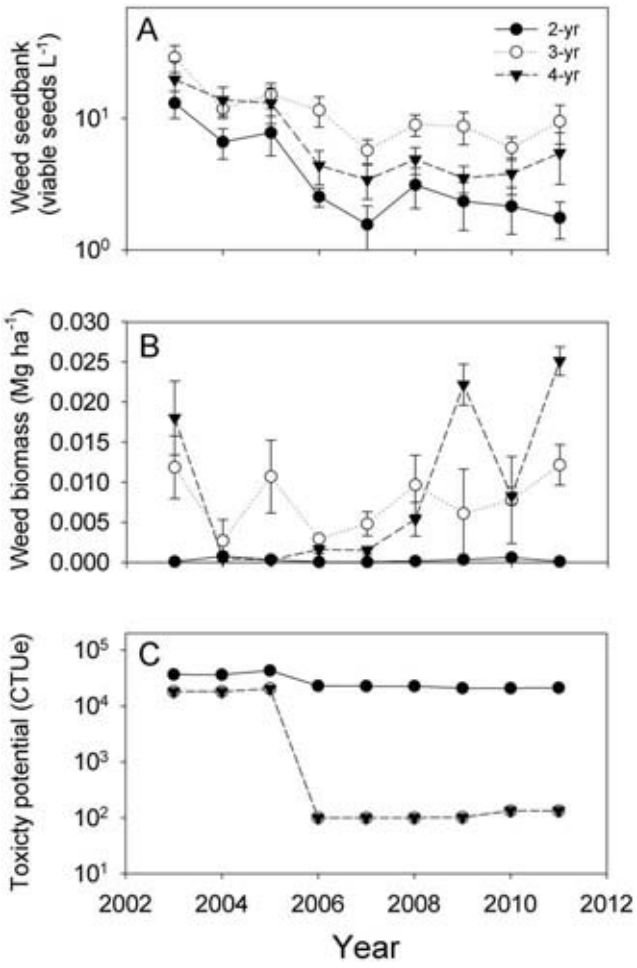

Figure 2. Divergent weed management systems. Weed management characteristics in maize-soybean (2-yr), maize-soybean-small grain/red clover (3-yr), and maize-soybean-small grain/alfalfa-alfalfa (4-yr) cropping systems in Boone, IA, from 2003 to 2011. Performance metrics included a) weed seed density in soil, b) weed aboveground biomass, and c) freshwater toxicity potential expressed in comparative toxic units (CTU_e). Symbols represent the mean ± SEM of four replicate experimental blocks (N = 36 per cropping system).
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to the presence of a small grain phase in the 3-yr and 4-yr rotation crop sequences. Weed biomass did not differ between maize and soybean in any of the cropping systems ($F_{1,202} = 2.1$; $P = 0.15$), however weed biomass in the small grain phase of the 3-yr and 4-yr rotations was greater than weed biomass in the maize and soybean phases ($F_{1,206} = 174$; $P < 0.0001$). In the 4-year system, weed biomass in alfalfa was intermediate between weed biomass levels in the maize/soybean and small grain phases.

Environmental toxicity, in relation to ecotoxicological profiles for herbicides used in this study (Fig. 2c), showed a strong effect of *system* ($F_{2,14} = 1673$, $P < 0.0001$), with lower toxicity potential in the 3-yr and 4-yr rotations compared to the 2-yr rotation (*type*: $F_{1,17} = 2691$, $P < 0.0001$). Ecotoxicity in the diversified and conventional systems diverged as the systems matured over time [*type* x *phase*: $F_{1,16} = 7.4$, $P = 0.015$], transitioning from a two-fold

difference during 2003 to 2005 to a two hundred-fold difference in toxicity from 2006 to 2011 (Fig. 2c).

Discussion

Our results support the hypothesis that the development of ecosystem services over time in more diverse cropping rotations increasingly displaces the need for external synthetic inputs to maintain crop productivity. From 2003 to 2011, as predicted, the desired products (crop yield, weed suppression, and economic performance) of the more diverse and less diverse cropping rotations were similar, whereas external inputs and environmental impacts differed greatly among the systems (Fig. 3). Comparing these metrics of system performance by experimental phase (initial three years of system establishment versus the following six years) confirmed our prediction that system inputs and environmental impacts would diverge over time, whereas yield and profit would remain similar among more diverse and less diverse rotations. In the more diverse rotations, small amounts of synthetic agrichemical inputs thus served as powerful tools with which to tune, rather than drive, agroecosystem performance.

Grain production in the U.S. is dominated by short rotation systems designed to maximize grain yield and profit. These are important goals but represent only a portion of the many ecosystem services that managed lands may provide [18] and that should be considered when evaluating alternative production systems [1,19]. We believe that these functions are complementary, rather than competing, considerations for agroecosystem design. The results of this study demonstrate that more rotationally diverse cropping systems may be optimized in multiple dimensions, leveraging small agrichemical inputs with biological synergies arising from enhanced diversity of crop species and management tactics.

An example of the synergizing effects of cropping system diversification can be found in weed management in the 3-yr and 4-yr rotations. Weeds were suppressed as effectively in these systems as in the 2-yr rotation, with declining soil seedbanks and negligible weed biomass, yet herbicide inputs in the 3-yr and 4-yr rotation plots were 6 to 10 times lower, and freshwater toxicity 200 times lower, than in the 2-yr rotation. Improved efficiency and environmental sustainability of weed management in the 3-yr and 4-yr rotations resulted from integrating multiple, complementary tactics in an ecological weed management framework [8,20]. Mounting evidence for unintended effects resulting from heavy reliance on herbicides highlights the need to re-think the role of herbicides in weed management. Non-target impacts of herbicides include reproductive abnormalities and mortality in vertebrates [5,21–23] and potential for diminished non-crop nectar resources for key pollinator species [17,24,25]. Herbicide overuse has also resulted in widespread, accelerating evolution of weed genotypes resistant to one or more modes of herbicide action [26,27]. Our

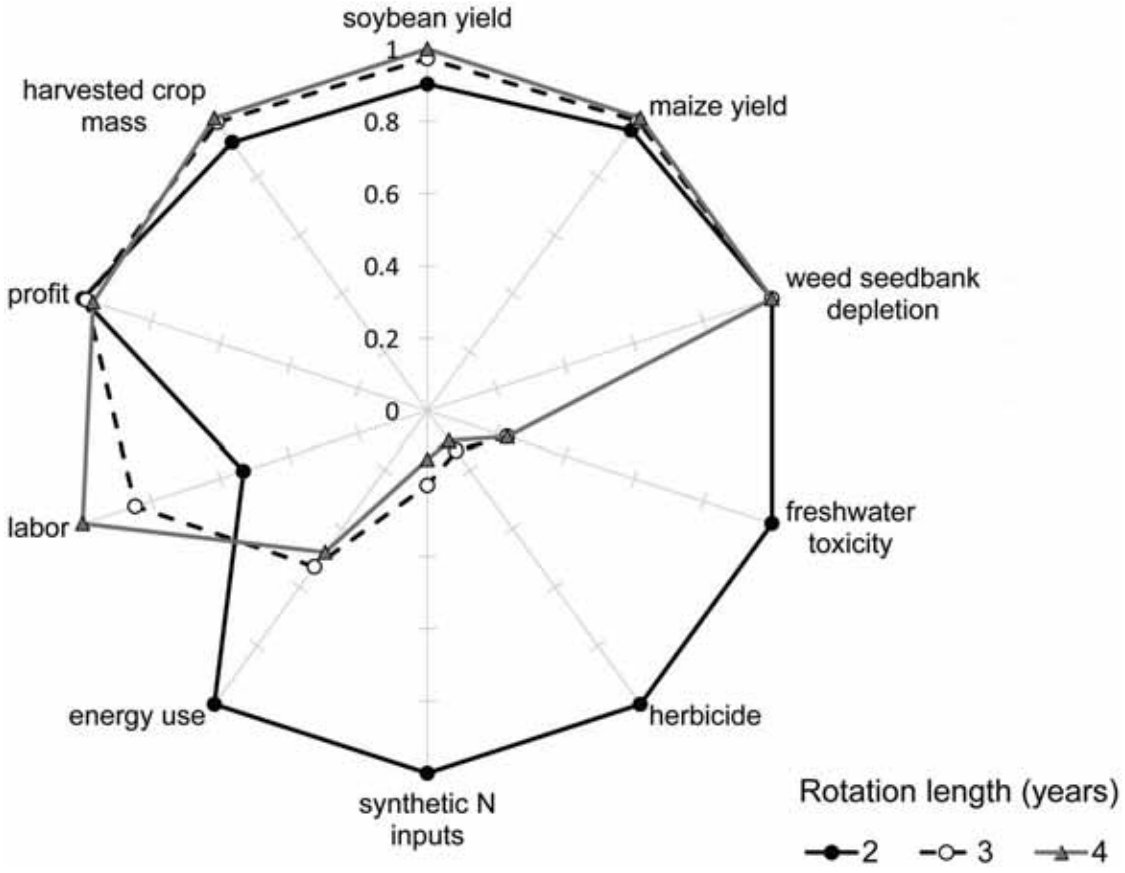

Figure 3. Multiple indicators of cropping system performance. Comparative long-term performance of maize-soybean (2-yr), maize-soybean-small grain/red clover (3-yr), and maize-soybean-small grain/alfalfa-alfalfa (4-yr) cropping systems in Boone, IA, averaged over the 2003–2011 study period. Variable means are normalized on a 0 to 1 scale, with 1 representing the cropping system with the largest absolute value for that variable (N = 36 per cropping system). Performance metrics included: maize and soybean yield, rotation-level harvested crop mass, net returns to land and management, manufactured N fertilizer and herbicide application rate, fossil energy use, labor requirements, freshwater toxicity potential and weed seedbank decline (measured as exponential decay constant).
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data indicate that, in the context of a cropping system with weed suppressive characteristics, small herbicide inputs may contribute to a diverse suite of tactics that cumulatively provide effective, reliable, and more durable weed management.

The diversity-productivity-stability relationship has long been a key theme in ecology [28,29]. Recently, it has been applied in the context of bioenergy crop production to describe increases in biomass and ecosystem services, such as C sequestration, associated with increasing species diversity in polycultures of bioenergy feedstock crop species [30]. Our work supports the application of this concept to cropping systems more broadly. Future gains in more diverse systems may depend upon the application of ecological principles surrounding this relationship to cropping system design [31,32]. Cropping system diversification in this study included both crop species and management practices. In contrast to the 2-yr rotation, with two species, both of the 3-yr and 4-yr rotations included four crop species. In the 4-yr rotation, further temporal diversification was achieved by including a perennial-only crop phase (alfalfa hay) for one quarter of the rotation sequence. Our results showed productivity gains associated with greater diversity in system-level harvested crop mass and maize and soybean seed yields. We also observed increased stability of profit, with similar long-term means, in the 3-yr and 4-yr rotations compared to the 2-yr rotation.

Similar profits were attained through different pathways in the 3-yr and 4-yr rotations and the 2-yr rotation (Fig. 3). Increased labor, information intensive management and ecosystem services arising from increased biological N fixation (via the clover and alfalfa crops) and contrasting crop phenologies and competitive abilities were substituted in 3-yr and 4-yr rotations for the higher inputs of manufactured N, herbicides and energy from fossil fuels driving the 2-yr rotation. Energy use in maize drove differences among the cropping systems, and manufactured N inputs to maize contributed most strongly to energy balances for this crop. The high sensitivity of agricultural energy use to N fertilizer inputs provides a high-priority target for the redesign of cropping systems for increased sustainability.

Reintegration of crop and livestock production, as represented by the forage legumes and manure applications present in the more diverse systems, is not simply another aspect of cropping system diversification. Instead, it embodies an important principle in sustainable agriculture: system boundaries should be drawn to minimize externalities. Animal manure is produced regardless of whether feed grains are shipped to centralized concentrated animal feeding operations, or produced within integrated crop-livestock farming operations. In the former case, the manure may become a waste product and water pollutant if quantities exceed available land area for field application [33], whereas in the latter case, it contributes directly to crop nutrient requirements, improves soil quality, and reduces fossil fuel subsidies associated with grain transport and external N fertilizer inputs [14].

Substantial improvements in the environmental sustainability of agriculture are achievable now, without sacrificing food production or farmer livelihoods. When agrichemical inputs are completely eliminated, yield gaps may exist between conventional and alternative systems [19]. However, such yield gaps may be overcome through the strategic application of very low inputs of agrichemicals in the context of more diverse cropping systems. Although maize is grown less frequently in the 3-yr and 4-yr rotations than in the 2-yr rotation, this will not compromise the ability of such systems to contribute to the global food supply, given the relatively low contribution of maize and soybean production to direct human consumption and the ability of livestock to consume small grains and forages [34]. Through a balanced portfolio approach to agricultural sustainability, cropping system performance can be optimized in multiple dimensions, including food and biomass production, profit, energy use, pest management, and environmental impacts.

Materials and Methods

Site Details and Agronomic Management

To investigate the relative performance of conventional and more diverse cropping systems, we conducted a 9-hectare experiment at the Iowa State University Marsden Farm (Figure S1), in Boone County, IA (42°01′ N; 93°47′ W; 333 m above sea level). The experiment site lies within a region of intensive rain-fed maize and soybean production and is surrounded by farms with high levels of productivity. Soils at the site are deep, fertile Mollisols. The experimental cropping system treatments included a conventionally managed 2-yr rotation (maize/soybean) that received agrichemicals at rates comparable to those used on commercial farms in the region, and more diverse cropping systems – a 3-yr rotation (maize/soybean/small grain + red clover green manure) and a 4-yr rotation (maize/soybean/small grain + alfalfa/alfalfa hay) – managed with reduced N fertilizer and herbicide inputs.

The entire site was planted with oat in 2001 and the cropping systems experiment was established in 2002 using a randomized complete block design with each crop phase of each rotation system present every year in four replicate blocks. Plots were 18 m x 85 m and managed with conventional farm machinery. Spring triticale was used as the small grain in 2003–2005, whereas oat was used in 2006–2010. Synthetic fertilizers were applied in the 2-yr rotation at conventional rates based on soil tests. In the 3-yr and 4-yr rotations, composted cattle manure was applied before maize production at a mean dry matter rate of 8.3 Mg ha^{-1} and substantial amounts of N were added through fixation by red clover and alfalfa [35,36,37]. Manure and legume N-fixation in the 3-yr and 4-yr rotations were supplemented with synthetic fertilizers based on soil tests, including the late-spring soil nitrate test for maize production [38]. Weed management in the 2-yr rotation was based largely on herbicides applied at conventional rates. In the 3-yr and 4-yr rotations, herbicides were applied in 38-cm-wide bands in maize and soybean and inter-row zones were cultivated; no herbicides were applied in small grain and forage legume crops. Choices of post-emergence herbicides used in each of the systems were made based on the identities, densities, and sizes of weed species observed in the plots. Other details of the farming practices used in the different cropping systems are described in Liebman et al. [39] and in the online SI materials (Text S1). Sampling procedures for determining crop yields, weed biomass and weed seed densities in soil are also described in the online SI materials (Text S1).

Energy and Economic Analyses

Energy inputs were divided into five categories: seed, fertilizer, pesticides, fuel for field operations, and propane and electricity used for drying maize grain after harvest. Data were obtained from logs describing all field operations, material inputs, and crop moisture characteristics for the experimental plots during the study period. Economic analyses measured performance characteristics of whole rotation systems under contrasting management strategies. We evaluated net returns to land and management on a unit land area basis, with land units divided in two equal portions for maize and soybean in the 2-yr rotation; three equal portions for maize, soybean, and small grains with red clover in the 3-yr rotation; and four equal portions for maize, soybean, small grains with alfalfa, and alfalfa in the 4-yr rotation. Net returns to land and management represented returns to a farm operation calculated

without accounting for costs of land (e.g., rent or mortgage payments), management time (e.g., marketing), or possible federal subsidies. Data sources and assumptions for the energy and economic analyses are shown in the online SI materials.

Ecotoxicological Calculations

Freshwater ecotoxicity of pesticide use was estimated using the USEtox model [40–42]. Characterization factors (CFs) of ecotoxicity potential for active ingredients included transport to freshwater via surface water, soil, and air. CFs were available for eight of ten active ingredients applied in the three rotations. The two active ingredients for which CFs were unavailable are not of particular concern for freshwater ecotoxicity due either to their low toxicity (mesotrione) or low infiltration and persistence in freshwaters (lactofen) [43].

Statistical Analyses

The experiment was arranged in a randomized complete block design, with all entry points of the three crop rotations (i.e. all crops within each of the rotations) represented in four replicate blocks in each year of the study, for a total of 36 plots. Cropping system effects in time series data were analyzed using hierarchical linear mixed effects repeated measures models, modeling temporally correlated errors with an ARMA (auto-regressive moving average) correlation structure in the *nlme* package of R v.2.14.1 [44,45]. Fixed effects included *cropping system* and *experimental phase* (startup = 2003 to 2005; established = 2006 to 2011), and random effects included *replicate block* nested within *cropping system* and *year*. Partial correlations were estimated using the *corpcor* package in R v.2.14.1. In contrast to data for quantitative observations (e.g. crop yield or weed biomass) that varied by replicate block and year, data for input variables, such as synthetic fertilizer or herbicides and associated environmental toxicity metrics, did not vary among blocks for a particular rotation entry point in a given year, but did vary among years. Therefore, site-year was treated as the source of experimental replication for these latter variables in our statistical tests for effects of *cropping system* and *experimental phase*. This led to contrasting degrees of freedom in reported F-tests for these two data types. Finally, for variables with non-constant variance among cropping systems over time (crop biomass and profit), we used the 'varIdent' variance function within the *nlme* package to explicitly model differences in variances among cropping systems for these variables within our mixed effects models.

Supporting Information

Figure S1 Aerial view of Marsden Farm study, Boone IA. Crop abbreviations: m = maize, sb = soybean, g = small grain, a = alfalfa.
(TIF)

Table S1 Mean monthly air temperature and total monthly precipitation during the 2003–2011 growing seasons, and long-term temperature and precipitation averages. Data were collected about 1 km from the experimental site in Boone Co., IA.
(DOCX)

Table S2 Crop identities and seeding rates in 2003–2011.
(DOCX)

Table S3 Macronutrients applied in manufactured fertilizers, herbicide adjuvants, and manure in 2003–2011. Manufactured N, P, and K fertilizers were applied at rates that varied among years and rotations in response to soil test results. Manure was applied at a rate of 15.7 Mg ha^{-1} in maize phases of the 3-year and 4-year rotation systems, but moisture and nutrient concentrations varied among years, resulting in variable rates of macronutrient additions.
(DOCX)

Table S4 Herbicide applications in 2003–2011 to maize and soybean in the three rotation systems. No herbicides were used for triticale, oat, red clover, and alfalfa grown within the 3-yr and 4-yr systems. Reported application rates reflect the effect of banding of herbicides over crop rows in the 3-yr and 4-yr systems.
(DOCX)

Table S5 Simple and partial correlations between energy use within a given crop phase and mean rotation energy use and between energy use within a given crop phase and N fertilizer application rates.
(DOCX)

Text S1 Detailed description of experimental site, management practices, scientific methods and statistical approach.
(DOCX)

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Author Contributions

Conceived and designed the experiments: ASD ML. Performed the experiments: CAC AMJ ML. Analyzed the data: ASD JDH CAC AMJ ML. Contributed reagents/materials/analysis tools: ML. Wrote the paper: ASD JDH ML.

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The New York Times
Opinionator

OCTOBER 19, 2012, 1:05 PM

A Simple Fix for Farming

By MARK BITTMAN

IT'S becoming clear that we can grow all the food we need, and profitably, with far fewer chemicals. And I'm not talking about imposing some utopian vision of small organic farms on the world. Conventional agriculture can shed much of its chemical use - if it wants to.

This was hammered home once again in what may be the most important agricultural study this year, although it has been largely ignored by the media, two of the leading science journals and even one of the study's sponsors, the often hapless Department of Agriculture.

The study was done on land owned by Iowa State University called the Marsden Farm. On 22 acres of it, beginning in 2003, researchers set up three plots: one replicated the typical Midwestern cycle of planting corn one year and then soybeans the next, along with its routine mix of chemicals. On another, they planted a three-year cycle that included oats; the third plot added a four-year cycle and alfalfa. The longer rotations also integrated the raising of livestock, whose manure was used as fertilizer.

The results were stunning: The longer rotations produced better yields of both corn and soy, reduced the need for nitrogen fertilizer and herbicides by up to 88 percent, reduced the amounts of toxins in groundwater 200-fold and didn't reduce profits by a single cent.

In short, there was only upside - and no downside at all - associated with the longer rotations. There was an increase in labor costs, but remember that profits were stable. So this is a matter of paying people for their knowledge and smart work instead of paying chemical companies for poisons. And it's a high-stakes game; according to the Environmental Protection Agency, about five billion pounds of pesticidesare used each year in the United States.

No one expects Iowacorn and soybean farmers to turn this thing around tomorrow, but one might at least hope that the U.S.D.A.would trumpet the outcome. The agency declined to comment when I asked about it. One can guess that perhaps no one at the higher levels even knows about it, or that they're afraid to tell Monsantoabout agency-supported research that demonstrates a decreased need for chemicals. (A conspiracy theorist might note that the journals Science and Proceedings of the National Academy of Sciences both turned down the study. It was finally published in PLOS One; I first read about it on the Union of Concerned Scientists Web site.)

Debates about how we grow food are usually presented in a simplistic, black-and-white way, conventional versus organic. (The spectrum that includes conventional on one end and organic on the other is not unlike the one that opposes the standard American diet with veganism.) In farming, you have loads of chemicals and disastrous environmental impact against an orthodox, even dogmatic method that is difficult to carry out on a large scale.

But seeing organic as the only alternative to industrial agriculture, or veganism as the only alternative to supersize me, is a bit like saying that the only alternative to the ravages of capitalism is Stalinism; there are other ways. And positioning organic as the only alternative allows its opponents to point to its flaws and say, "See? We have to remain with conventional."

The Marsden Farm study points to a third path. And though critics of this path can be predictably counted on to say it's moving backward, the increased yields, markedly decreased input of chemicals, reduced energy costs and stable profits tell another story, one of serious progress.

Nor was this a rinky-dink study: the background and scientific rigor of the authors - who represent the U.S.D.A.'s Agricultural Research Service as well as two of the country's leading agricultural universities - are unimpeachable. When I asked Adam Davis, an author of the study who works for the U.S.D.A., to summarize the findings, he said, "These were simple changes patterned after those used by North American farmers for generations. What we found was that if you don't hold the natural forces back they are going to work for you."

THIS means that not only is weed suppression a direct result of systematic and increased crop rotation along with mulching, cultivation and other nonchemical techniques, but that by not poisoning the fields, we make it possible for insects, rodents and other critters to do their part and eat weeds and their seeds. In addition, by growing forage crops for cattle or other ruminants you can raise healthy animals that not only contribute to the health of the fields but provide fertilizer. (The same manure that's a benefit in a system like this is a pollutant in large-scale, confined animal-rearing operations, where thousands of animals make manure disposal an extreme challenge.)

Perhaps most difficult to quantify is that this kind of farming - more thoughtful and less reflexive - requires more walking of the fields, more observations, more applications of fertilizer and chemicals if, when and where they're needed, rather than on an all-inclusive schedule. "You substitute producer knowledge for blindly using inputs," Davis says.

So: combine crop rotation, the re-integration of animals into crop production and intelligent farming, and you can use chemicals (to paraphrase the report's abstract) to fine-tune rather than drive the system, with no loss in performance and in fact the gain of animal products.

Why wouldn't a farmer go this route? One answer is that first he or she has to hear about it. Another, says Matt Liebman, one of the authors of the study and an agronomy professor at Iowa State, is that, "There's no cost assigned to environmental externalities" - the

environmental damage done by industrial farming, analogous to the health damage done by the "cheap" standard American diet - "and the profitability of doing things with lots of chemical input isn't questioned."

This study not only questions those assumptions, it demonstrates that the chemicals contributing to "environmental externalities" can be drastically reduced at no sacrifice, except to that of the bottom line of chemical companies. That direction is in the interest of most of us - or at least those whose well-being doesn't rely on that bottom line.

Sadly, it seems there isn't a government agency up to the task of encouraging things to move that way, even in the face of convincing evidence.

This post has been revised to reflect the following correction:

Correction: October 19, 2012

An earlier version of this column incorrectly listed Nature as one of several scientific journals that had turned down the Marsden Farm study. While Science and Proceedings of the National Academy of Sciences did not accept it, the study was not submitted to Nature.

The New York Times

November 3, 2012

Did Farmers of the Past Know More Than We Do?

By VERLYN KLINKENBORG

A couple years ago, I saw a small field of oats growing in northwest Iowa — a 40-acre patch in a sea of genetically modified corn and soybeans. It was an unusual sight. I asked my cousins, who still farm what my dad always called the "home place," whether someone had added oats to the rotation of crops being planted. The answer was no.

The purpose of that patch of oats was manure mitigation. The waste that had been sprayed on that field came from a hog confinement operation, and oats were the only crop that would put such concentrated, nearly toxic manure to nutritional use and do it quickly.

Oats used to be a common sight all over the Midwest. They were often sown with alfalfa as a "nurse crop" to provide some cover for alfalfa seedlings back when alfalfa was also a common sight. Until about 30 years ago, you could find all sorts of crops growing on Iowa farms, and livestock. Since then two things have happened. All the animals have moved indoors, into crowded confinement operations. And the number of crops has dwindled to exactly two: corn and soybeans.

My uncle Everon, who died last summer, farmed the home place when I was growing up. He would have been surprised to learn that he was following the principles of an early 18th-century agricultural experimenter named Charles Townshend, who, apart from his fascination with turnips, was every inch a viscount. Townshend's discovery — borrowed from Dutch and Flemish farmers — was that crops grow better, with fewer weeds and pest problems, if they are rotated in a careful sequence.

Townshend's rotation — like the ones George Washington and Thomas Jefferson used — included clover, wheat, other small grains and turnips, which made good winter food for sheep and cattle. My uncle grew no turnips, but he, like all his neighbors, was using his own version of the four-crop system, at the heart of which was alfalfa.

Getting to the four-crop rotation wasn't easy, historically speaking. The Romans knew about crop rotation, but by the Middle Ages, farming was based on the practice of letting the land lie fallow, unplanted — resting it, in other words. The purpose of that practice, like crop rotation itself, is to prevent the soil from becoming exhausted when the same crop is sown over and over again. In early American agriculture, only sophisticated farmers like Washington and Jefferson were using crop rotations in their fields. There was simply too much good land available. It was too easy to farm a piece and then move on when the soil was depleted.

In one sense, that is still how modern agriculture works. You look to the future and discard the past. A

modern rotation includes only corn, soybeans, fertilizer and pesticides. Whatever you may think about genetically modified crops, the switch to those varieties has driven the rush to the two-crop system. Those crops are designed to tolerate the presence of herbicides. The result is that farmland has been inundated with glyphosate, the herbicide genetically modified crops are engineered for.

The very structure of the agricultural system, as it stands now, is designed to return the greatest profit possible, not to the farmers but to the producers of the chemicals they use and the seeds they plant. And because those chemicals depend on fossil energy, the entire system is inherently unsustainable. What farmers used to return to the soil in the form of labor and animal manure — not the toxic kind you find in livestock confinement systems — they now must purchase, just the way they buy diesel for their tractors.

In fact, as a recent study by agronomists from the Department of Agriculture, Iowa State University and the University of Minnesota shows, there's nothing obsolete about four-crop rotation. It produces the same yields, it sharply reduces the toxicity of freshwater runoff, and it eliminates many of the problems associated with genetically modified crops, including the emergence of glyphosate-resistant weeds. It's also simply better for the soil. A four-crop rotation using conventional crop varieties, along with much lower applications of fertilizer and herbicides and some animal manure, works every bit as well as the prevailing monotony of corn and soybeans.

This study is a reminder of something essential. Modern agriculture is driven by diminishing biological diversity and relentless consolidation, from the farms themselves to the processors and the distributors of the crops and livestock. But you cannot consolidate the soil. It is a complex organism, and it always responds productively to diversity. The way we farm now undervalues and undermines good soil. Our idea of agricultural productivity and efficiency must include the ecological benefits of healthy soil. The surest way to improve the soil is to remember what industrial agriculture has chosen to forget.

DROVERS America's Beef Business Source CattleNetwork

Three or four year crop rotation vs corn/soybean rotation

University of Tennessee | Updated: November 6, 2012

In many circles, it is taken as a matter of fact that to be able to feed an additional 2 billion people by 2050, farmers everywhere are going to have to adopt the intensive agricultural practices that have been perfected in the US heartland where massive amounts of corn and soybeans are harvested almost every year. For the most part, this production system also separates crop agriculture from livestock agriculture composed of large chicken complexes, huge hog production facilities, and massive feedlots.

The implication of all of this is that only with this type of system will agriculture be able to meet the food demands of a rapidly growing population. Sustaining this model of agricultural production involves the heavy use of herbicides, insecticides, and synthetic fertilizers, all of which have significant environmental impacts. The system is also heavily dependent upon the use of fossil-based energy to produce the synthetic fertilizers that are crucial to the system and the fuel that is needed to cultivate fields, plant the crops, harvest them, and transport the corn and beans to feed mills that prepare the rations used in the various meat production systems.

Iowa State University with funding from the United States Department of Agriculture (USDA), the Leopold Center for Sustainable Agriculture, the Iowa Soybean Association, and the Organic Center conducted a field study from 2003-2011 that compared the typical 2-year corn/soybean rotation, with 3-year and 4-year rotations that included both crops and livestock.

The results of their study was published in a PLOS-One paper titled, "Increasing cropping system diversity balances productivity, profitability, and environmental health" . The researchers "hypothesized that cropping system diversification would promote ecosystem services that would supplement, and eventually displace synthetic external inputs used to maintain crop productivity."

The authors write "One of the key challenges of the 21st century is developing ways of producing sufficient amounts of food while protecting both environmental quality and the economic well-being of rural communities. Over the last half century, conventional approaches to crop production have relied heavily on manufactured fertilizers and pesticides to increase yields, but they have also degraded water quality and posed threats to human health and wildlife. Consequently, attention is now being directed toward the development of crop production systems with improved resource use efficiencies and more benign effects on the environment."

They conducted their study at the Marsden Farm and used a randomized block design to be able to compare the results of the three cropping systems. The 3-year and 4-year systems added the production of a small grain (triticale and oat) along with the use of a legume and composted animal manure. Small amounts of synthetic fertilizer and herbicides were used in the 3-year and 4-year rotations while the 2-year rotation used conventional amounts of these products.

The first finding that they discussed in their report was that the diversified rotations were just as profitable as the standard corn/soybean rotation that is being used by most farmers in Iowa and throughout the US Midwest. The profitability was measured without accounting for costs of land, management time, and federal subsidies.

The researchers found that "weeds were managed effectively in all three cropping systems in both the 'startup' and 'established' phases, in spite of reducing herbicide use by 88 percent in the 3-year and 4-year

rotations compared to the 2-year rotation.”

The use of labor in the 3-year and 4-year rotations was greater than in the 2-year rotation, but the cost of this was compensated by lower use of synthetic nitrogen inputs and reduced herbicide inputs.

“Weeds were suppressed as effectively in [the 3-year and 4-year] systems as in the 2-year rotation, with declining soil seedbanks and negligible weed biomass, yet herbicide inputs in the 3-year and 4-year rotation plots were 6 to 10 times lower, and freshwater toxicity 200 times lower, than in the 2-year rotation.”

The authors discussed their findings writing, “Reintegration of crop and livestock production, as represented by the forage legumes and manure applications present in the more diverse systems, is not simply another aspect of cropping system diversification. Instead, it embodies an important principle in sustainable agriculture: system boundaries should be drawn to minimize externalities. Animal manure is produced regardless of whether feed grains are shipped to centralized concentrated animal feeding operations, or produced within integrated crop-livestock farming operations. In the former case, the manure may become a waste product and water pollutant if quantities exceed available land area for field application, whereas in the latter case, it contributes directly to crop nutrient requirements, improves soil quality, and reduces fossil fuel subsidies associated with grain transport and external N fertilizer inputs.

“Substantial improvements in the environmental sustainability of agriculture are achievable now, without sacrificing food production or farmer livelihoods. When agrichemical inputs are completely eliminated, yield gaps may exist between conventional and alternative systems. However, such yield gaps may be overcome through the strategic application of very low inputs of agrichemicals in the context of more diverse cropping systems. Although maize is grown less frequently in the 3-year and 4-year rotations than in the 2-year rotation, this will not compromise the ability of such systems to contribute to the global food supply, given the relatively low contribution of maize and soybean production to direct human consumption and the ability of livestock to consume small grains and forages.”

Moving to a rotation system such as used in this study would require a willingness and managerial ability by the farm operator to reintegrate small-scale livestock production into a moderate-sized cropping system—something that was common place 5 decades ago but rare today.

The environmental advantages are compelling. The economics appear encouraging. The true test will be when the system moves from university plots to full-time farms, including those that are no longer “livestock-ready” for example. What size of livestock and crop operation would be needed to provide an adequate level of family income for a full-time farm operation? Or would a rotation system of livestock and crop farming, such as the one in this study, be one of several specialty farming segments that are currently thriving but in limited numbers?

Source: Daryll E. Ray and Harwood D. Schaffer, Agricultural Policy Analysis Center, University of Tennessee

Medical Daily

Scientists Perform 'Germ Warfare' on Bedbugs With Fungus More Effective Than Pesticides

Researchers found that we may be able to rid of the insects for good - and without the use of pesticides.

BY MAKINI BRICE | NOV 23, 2012 10:47 AM EST

Bed bugs have become an exploding problem in recent years. While the tiny insects do not carry any disease, their blood-sucking bites cause distressing itches and the pests are close to impossible to be rid of because they hide in spaces difficult to reach.

Enlarge

(Photo : South Carolina Department of Health and Environmental Control) When researchers monitored the critters, they found that 100 percent of the bugs who had been exposed to the fungus spores died within five days, a success rate that not even chemical pesticides can boast.

But a new study, published in the Journal of Invertebrate Pathology by researchers from Penn State University, has found that we may be able to rid of the insects for good - and without the use of pesticides. The researchers conducted a study using paper and cotton jersey, commonly used in linens. They treated one sheet with oil. The other, they treated with fungus spores from the *Beauveria bassiana*.

The surfaces dried overnight. Then researchers put 10 bed bugs at a time on the sheet material in a Petri dish.

When researchers monitored the critters, they found that 100 percent of the bugs who had been exposed to the fungus spores died within five days, a success rate that not even chemical pesticides can boast.

What's more, the insects do not even need to be directly exposed to the biopesticide. When one bed bug is exposed to the fungus, he returns to the group's hideaway and contaminates nearly all of the other insects.

That ability is incredibly important, because the bugs generally live in hideaways behind light switches, under loose wallpaper, behind baseboards, and other hard-to-reach spots.

"They don't even need to be directly exposed, and that's something chemicals cannot do," researcher Nina Jenkins said in a press release. "If you have bedbugs in your house ... what you really want to know is if they've all gone at the end of the treatment, and I think that's something that this technology could offer."

Researchers need to test exposure times and conduct more research before the product is ready for the market, but the timing is ripe. Bed bug infestation has been on the rise over the years; some reports say that infestation levels are the worst that they have been since the 1940s.

Read more at http://www.medicaldaily.com/articles/13239/20121123/scientists-perform-germ-warfare-bedbugs-fungus-more.htm#esxkBZrJ4ubpE7ch.99

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Short Communication

A preliminary evaluation of the potential of *Beauveria bassiana* for bed bug control

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ABSTRACT

Residual biopesticide treatments of *Beauveria bassiana* were tested against the bed bug *Cimex lectularius*. An oil formulation of conidia was applied to different substrates. Bed bugs were exposed for 1 h, transferred to an unsprayed environment and monitored for mortality. Separate bioassays evaluated the effect of bed bug strain, sex, life stage, and exposure substrate on mortality. Rapid mortality was observed in all bioassays, with bed bugs exposed to treated jersey knit cotton dying most rapidly. A further assay demonstrated efficient autodissemination of conidia from exposed bed bugs to unexposed bed bugs within artificial harborages.

1. Introduction

The human bed bug *Cimex lectularius* is a hematophagous insect that requires blood meals for growth and development throughout its life cycle. Over the past decade, bed bug infestations have grown virtually exponentially in both North America and Europe (Hwang et al., 2005). This resurgence in bed bug infestations has been linked to increased international travel, changes in pest management practices (including increased regulatory constraints removing certain chemical insecticides from operational use (Boase, 2007)) and the wide scale spread of insecticide resistance (Moore and Miller, 2006; Romero et al., 2007; Seong et al., 2010). Insecticide resistance, together with concerns over extensive use of chemicals in the domestic environment (Sanborn et al., 2002), create a need for safe alternative methods of bed bug control. One candidate approach is the formulation of fungal entomopathogens as novel biopesticides.

Entomopathogenic fungi lend themselves to development as biopesticides because, like many conventional chemical insecticide active ingredients, they act through contact. Fungal species such as *Beauveria bassiana* and *Metarhizium anisopliae* are capable of infecting a broad range of insect hosts and several biopesticide products have been developed for use in horticulture and agriculture (Lacey et al., 2008). Recently, research has extended to blood feeding insects and disease vectors including mosquitoes (Scholte et al., 2005; Blanford et al., 2005, 2011; Darbro et al., 2011), ticks (Fernandes et al., 2011), tsetse flies (Maniania and Odulaja, 1998) and triatomid bugs (Pedrini et al., 2009).

To date, there are no published studies on the efficacy of entomopathogenic fungi against bed bugs. In this study, we evaluated the efficacy of one candidate isolate of *B. bassiana* as a residual biopesticide against the common bed bug in laboratory conditions, considering effects of feeding status, sex, bed bug strain, life history stage, and exposure substrate. Additionally, we evaluated autodissemination of conidia as a means to spread infection among bed bug populations in untreated, inaccessible areas.

2. Materials and methods

2.1. Bed bugs

A pyrethroid-susceptible laboratory strain (Harlan; cultured without introductions nor pesticide exposure since 1973) of bed bugs (designated HS) was obtained from Virginia Polytechnic Institute and State University. A second 'field' strain (an amalgam of several populations collected from cities across the US in 2005; designated FS) was obtained from University of Minnesota. Both strains were reared in our lab under standard conditions of 27 °C, 50% relative humidity (RH), and 14:10 (L:D) in glass rearing jars containing folded filter paper (Whatman No. 1, 90 mm) for a harborage and offered a blood meal weekly via an artificial feeding system (Montes et al., 2002).

2.2. Fungal isolate

B. bassiana I93-825 was maintained in long-term storage at −80 °C on microporous beads (Pro-Lab Diagnostics, Austin, TX, USA). Conidia were mass-produced using our standard 2-stage production system on barley flake (Jenkins et al., 1998; Anderson et al.,

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2011). Conidia were harvested using a Mycoharvester (Acis Manufacturing, Devon, UK), dried to 5% moisture content over silica gel, sealed in foil laminated sachets and stored at 5 °C until use.

Conidia were formulated in oil containing 80% Isopar M (Exxon Mobil) and 20% Ondina 22 (Shell) and adjusted to a concentration of 1.6×10^9 conidia/mL (viability 94–98%).

2.3. Application of conidia to exposure substrate

Conidial formulations were applied to substrates (paper or cotton jersey) at a rate of 3×10^6 conidia/cm^2 using an airbrush sprayer (Anderson et al., 2011), to give an equivalent volume application rate of 20 mL/m^2. Spore formulations were applied to HP™ Color-Laser Paper or jersey knit cotton, which were then cut into 9 cm circles. Control substrates were sprayed with blank oil formulation only. After spraying, substrates were allowed to dry at room temperature overnight. Coverage of conidia was verified by extracting the conidia from three replicate, 2 cm^2 swatches in Isopar M and counting the resulting conidial suspension using an improved Neubauer hemocytometer.

2.4. Exposing bed bugs to fungal spores

FS bed bugs were used in all experiments except that which compared the susceptibility of the two strains. Most experiments used adult bed bugs of mixed sex, which were removed from the colony one day prior to exposure and fed 12 h prior to experimentation. Variations to this procedure are described in the specific methods for each study.

In all studies, three replicates of 10 bed bugs were exposed to each treatment by placing them on pre-sprayed, dry substrate in a Petri dish for 1 h. After exposure, bed bugs were placed on clean filter paper in a Petri dish. All bed bugs were fed on day 7, 14, and 21 following exposure and mortality recorded daily for 21 d. Cadavers were incubated under high humidity to confirm mycosis. Survival data were analyzed using Kaplan–Meier survival analysis (SPSS, software version 18). Differences in median survival time between treatments were compared using the log-rank test.

2.5. Impact of feeding status, sex, and strain

All bioassays to evaluate the effect of feeding status, sex and strain of bed bugs were conducted on HP™ Color-Laser Paper as the test substrate. To evaluate the effect of feeding status, 60 adult bed bugs of mixed sex were randomly selected from the FS colony prior to feeding. Thirty bed bugs were left unfed (no blood meal for 14 d), while the remaining 30 were blood fed 12 h prior to exposure and the bioassay conducted on three replicates of 10 bed bugs as per the standard bioassay procedure.

To evaluate sex and strain differences, males versus females or mixed sex populations from the HS and FS colonies were used, respectively.

2.6. Impact of sprayed substrate on conidial transfer

Adults of mixed sex from the FS colony were fed 12 h prior to exposure and placed on either sprayed HP™ Color-Laser Paper or jersey knit cotton for 1 h.

2.7. Impact of life history stage

FS bed bugs were grouped according to instar. Adult, first and fifth instar bed bugs were selected as these life stages were most easily distinguishable. All bed bugs were fed 12 h prior to exposure. Three replicate groups of 10 bed bugs from each instar were placed on treated jersey knit cotton for 1 h.

2.8. Autodissemination of conidia

Bed bugs were removed from the FS colony, fed, and placed into 30 mL diet cups in six groups of 20 and left overnight. The following day, 10 bed bugs were removed at random from each group and exposed to either treated or unsprayed jersey cotton (three replicates) and allowed to remain in contact with the substrate for 1 h. After exposure, bed bugs were returned to their respective diet cups to comingle with the 10 unexposed bed bugs. A sterile filter paper harborage was provided. Mortality was assessed daily as above.

3. Results

There was no difference in mean survival times (MST) of treated bed bugs regardless of feeding status (MST fed 4.30 ± 0.160 days, unfed 4.17 ± 0.230 days, chi-square = 0.714, d.f. = 1, $p = 0.398$), sex (MST males 4.60 ± 0.214 days, females 5.60 ± 1.070 days, chi-square = 0.328, d.f. = 1, $p = 0.567$), or strain (MST HS 5.03 ± 0.559 days, FS 5.10 ± 0.552 days, chi-square = 0.259, d.f. = 1, $p = 0.611$) (Fig. 1A–C). Mycosis was confirmed in 100% of cadavers.

Mean survival times of bed bugs exposed to sprayed jersey knit cotton were significantly shorter than those exposed to sprayed paper (MST jersey 3.03 ± 0.580 days, paper 4.30 ± 0.160 days chi-square = 43.382, d.f. = 1, $p \leqslant 0.001$) (Fig. 1D).

All bed bug instars tested were susceptible to infection following exposure to sprayed jersey material (MST 1st instar 3.00 ± 0.00 days, 5th instar 4.00 ± 0.048 days, adults 3.03 ± 0.033 days) (Fig. 1E).

Bed bugs sharing harborages with conidia-exposed individuals experienced significantly more mortality than in the control harborages (chi-square = 124.04, d.f. = 1, $p \leqslant 0.000$). Mean survival times for adult bed bugs in the exposed treatment was 5.42 ± 0.532 days. There was no control mortality in this experiment. Overall mortality in the treated group was 95%, demonstrating that practically all of the unexposed beg bugs became infected when sharing the harborage with recently exposed individuals (Fig. 1F).

4. Discussion

B. bassiana (I93-825) was highly virulent to bed bugs, causing rapid mortality (3–5 days) following short-term exposure to spray residues. Infection levels were generally 100% indicating complete susceptibility to fungal infection under these exposure conditions. In a couple of assays 5–8% of individuals did not die, but re-exposure of these few survivors resulted in infection and mortality (results not shown), suggesting sub-optimal pick up of spores (especially from the paper substrate) rather than any physiological resistance. Results were robust across six separate assays.

There were no striking differences in susceptibility due to bed bug feeding status, sex, strain, or life stage. With respect to test substrates, jersey knit cotton was a better substrate for conidial transfer than paper, probably due to the relatively contoured surface resulting in more conidia coming into contact with the insect cuticle. These results demonstrate that choice of substrate is important in both bioassay design and end product development. Studies exploring transfer of conidia to mosquitoes following short-term residual exposure also show substrate type to effect infection levels and spore persistence (Farenhorst et al., 2011).

The current study focused on the lethal effects of infection, not least because our bioassay system resulted in such rapid and extensive mortality. In 'field' settings (i.e. in domestic environments where the fungus would be deployed) it is possible that bed bugs might experience lower doses via transient exposures, or when fungal spray residues begin to decay, resulting in slower

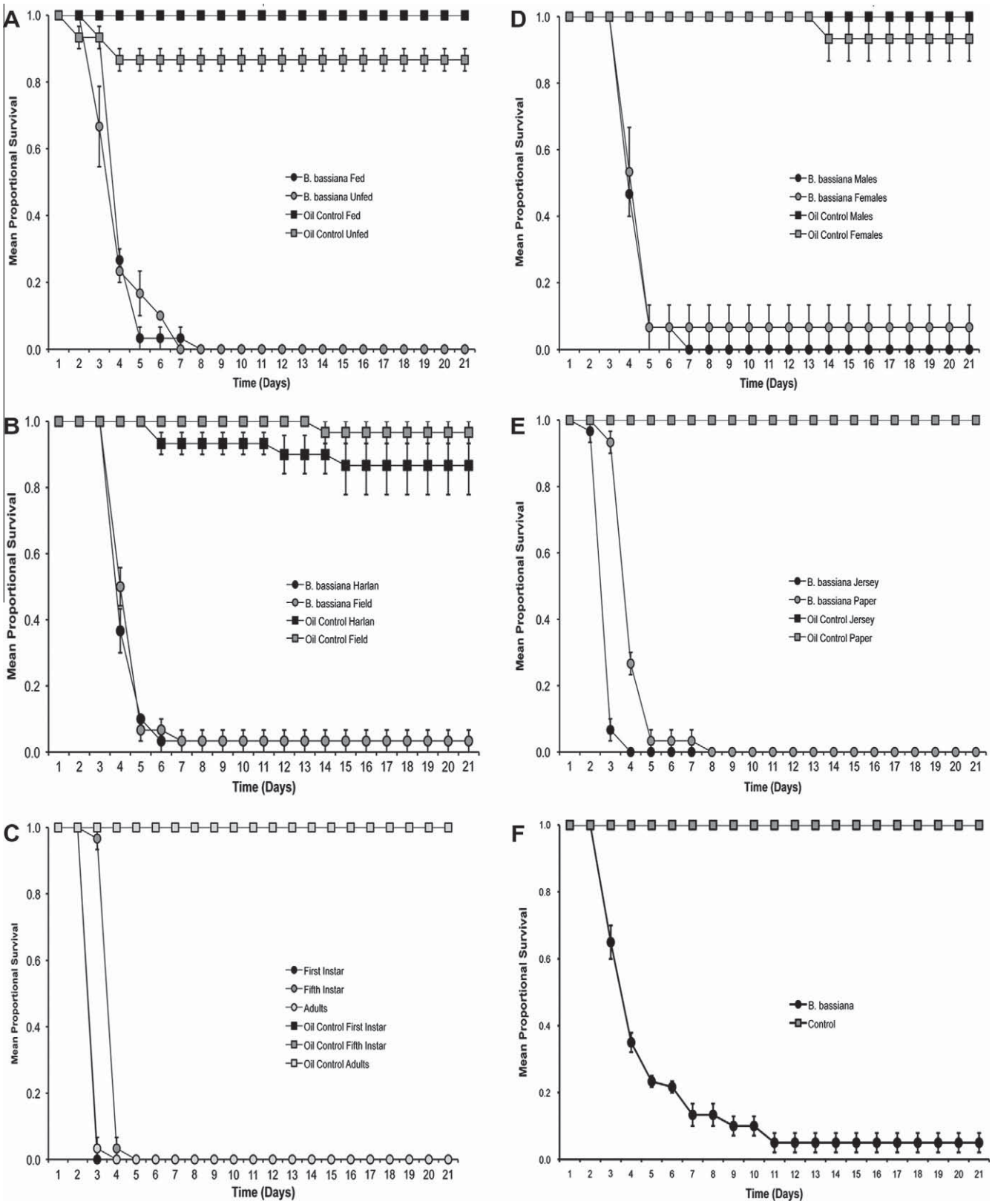

Fig. 1. (A–C) Mean proportional survival of bed bugs exposed to paper sprayed with oil formulation of *B. bassiana* conidia at 3×10^6 conidia/cm^2 (circles), or blank oil formulation (squares) for 1 h. (A) Bed bugs blood-fed 12 h prior to exposure (black circles), or left unfed prior to exposure (grey circles). (B) Male (black circles) and female (grey circles) bed bugs. (C) Harlan strain (black circles) and field strain (grey circles). (D) Mean proportional survival of adult field strain bed bugs exposed to sprayed paper (grey circles) or cotton jersey (black circles) for 1 h. (E) Mean proportional survival of first instar (black circles), fifth instar (grey circles) and adult (open circles) bed bugs, exposed to treated and untreated cotton jersey. (F) Mean proportional survival of adult bed bugs where only 50% of the population was exposed to fungus-treated cotton jersey (black circles) or blank formulated control (squares), and the remaining bed bugs sharing the harborages were unexposed. All data points represent the mean (± SE) of three replicates of 10 bed bugs except (F) where three replicates of twenty bed bugs were used.

mortality. However, slower speed of kill might be of relatively little consequence with respect to population suppression and ultimate elimination from a residence. Bed bug nymphs typically take 4–5 weeks to complete development and reach sexual maturity (Omori, 1941). This relatively slow development provides many days for a fungus to act while still preventing reproduction. Furthermore, sub- or pre-lethal effects of fungal infection, which include reduced feeding, mobility, and fecundity, are well documented in other systems (Blanford and Thomas, 2001; George et al., 2011; Howard et al., 2010) and have the potential to supplement lethal effects substantially.

Elimination of established bed bug infestations is challenging because it is difficult to identify and target all concealed harborages. However, bed bugs make nightly excursions in search of a blood meal (Mellanby, 1939; Usinger, 1966). Therefore, development of delivery systems based on barrier treatments, such as a 'bed skirt', positioned between the harborages and the human host show potential for effective control. In addition, our results suggest the potential for efficient autodissemination of conidia via contact with contaminated individuals. Our assay demonstrated that when only 50% of a bed bug population was directly exposed to fungus, total mortality exceeded 95%. Other studies have demonstrated autodissemination of conidia (Scholte et al., 2004) and potential for disease cycling following biopesticide spray applications (Arthurs and Thomas, 1999; Thomas et al., 1995). Since bed bugs are highly gregarious with all life stages aggregating in confined harborages with humid microclimates (Usinger, 1966), horizontal transmission could greatly increase the impact of fungal treatments relative to conventional chemicals.

Overall, this study represents an important first step in developing *B. bassiana* as a biopesticide for use against bed bugs within novel strategies of integrated pest management. Further research is now required to develop appropriate formulations and delivery systems to investigate population level impact under more realistic 'semi-field' and 'field' settings.

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Antibiotic resistance killing off bees

Photo by Michael McHugh.

BY EMMA GOLDBERG
CONTRIBUTING REPORTER
Tuesday, November 6, 2012

Recommended

- Blumenthal starts antibiotics arms race
- MCDONALD: Trying to GAIN an edge on 'superbugs'
- Antibiotics linked with childhood asthma
- Suspected TB case surfaces
- Undergraduate in isolation with assumed tuberculosis

Yale researchers have identified a potential culprit in a nationwide increase of honeybee deaths — resistance to an important antibiotic.

In the Oct. 30 issue of the mBio journal, Yale professor of ecology and evolutionary biology Nancy Moran published a study showing that beneficial bacteria found in the guts of honeybees have acquired genes that make bees resistant to tetracycline, an antibiotic used to prevent colony-destroying infections and other bacterial diseases. Moran's research identified eight tetracycline-resistant genes in American honeybees that were absent in honeybee populations where such antibiotic treatment is banned, suggesting that use of tetracycline has genetically altered beneficial bacteria and made colonies more prone to infection.

"We noticed that the [first] colony collapses happened in 2006, which is when the antibiotic was first introduced," Moran told the News. "But it's really a very speculative concept right now."

Researchers in Moran's lab are looking to confirm the link they have established between antibiotics and colony collapses so as to impact policy-making in the beekeeping community.

"The beekeepers are interested in healthy bees. If we can show that antibiotics affect honey production or colony survival, we can draw their attention," Moran said.

To test bee genes for resistance to antibiotics, researchers in Moran's lab isolated all of the bacterial DNA in bee guts and transferred them into independent DNA molecules called plasmids. These plasmids were then put into E. coli and sequenced so that the tetracycline-resistant genes could be identified. To determine how different bee populations interact with antibiotics, researchers used a technique called polymerase chain reaction to amplify bee DNA samples collected from various locations in the United States, New Zealand, the Czech Republic and Switzerland.

"We found that bees from the USA, which had a long treatment history [with tetracycline], carried the most resistant genes," said Waldan Kwong GRD '16, one of the authors of the paper and a researcher in Moran's lab.

If confirmed, Moran's research could have wide-reaching impact on American crop growth and production. Honeybee pollination plays a critical role in the $15 billion U.S. agriculture industry. The industry has been plagued by recent bee colony collapses, due primarily to bees' contraction of the bacterial disease foul brood.

University of California, Davis apiculture professor Norman Gary stressed the magnitude of the colony collapse disorder.

"Only recently has the true value of honeybees been appreciated by people in this country," Gary told the News. "The colony collapse disorder is a complex issue and many scientists are advancing theories to explain its cause."

Kwong said he hopes the research conducted in Moran's lab will encourage beekeepers to exercise caution when introducing new antibiotics into bee colonies.

"Beekeepers and the general public should be aware that application of antibiotics not only affects pathogens but also the normal healthy microbes that coexist with the host," he said. He added, however, that further research and consultation with the beekeeping community are needed before any new policies can be introduced and implemented.

Gary said bee die-offs are likely the result of multiple factors rather than a single central cause. "In the scientific community we're hoping that honeybees will develop a resistance to the cause of the colony collapses," he said.

Moving forward, researchers in Moran's lab are studying the health benefits and hazards posed by gut bacteria in bees. They are studying the microbes that have become resistant to tetracycline to understand the beneficial functions they perform, such as pathogen defense, as well as the negative impact they can have on bees' immune systems.

"We want to understand how bacteria function in bees," Moran said. Her lab is currently working on an experiment that exposes bees to antibiotics and analyzes their long-term effects. Though she said the team has evidence that bacteria can help bees digest food, Moran said they hope to discover other health benefits bacteria provide bees.

"It's basic work, but nothing like it has ever been done before," she said.

The lead author of the paper was former Yale postdoctoral researcher Baoyu Tian, and other authors included Yale researchers Nibal H. Fadhil and J. Elijah Powell. The research was funded through a grant from the National Science Foundation.

American Academy of Pediatrics

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AAP Makes Recommendations to Reduce Children's Exposure to Pesticides

11/26/2012

For Release: November 26, 2012

Children encounter pesticides every day and are uniquely vulnerable to their toxicity. A new policy statement from the American Academy of Pediatrics (AAP) outlines the harmful effects of pesticides on children and makes recommendations on how to reduce exposure. The policy statement, "Pesticide Exposure in Children," and an accompanying technical report are published in the December 2012 issue of Pediatrics (released online Nov. 26). Prenatal and early childhood exposure to pesticides is associated with pediatric cancers, decreased cognitive function and behavioral problems. According to the AAP, recognizing and reducing children's exposure to pesticides will require improved medical training, public health tracking, and regulatory approaches. The AAP recommends pediatricians become familiar with the effects of acute and chronic exposures to pesticides; learn what resources are available for both treatment of acute poisoning and addressing lower dose chronic exposures in children; and understand pesticide labeling. Pediatricians should ask parents about pesticide use around the home and yard, offer guidance about safe storage, and recommend parents choose lowest-harm approaches when considering pest control. Pediatricians should also work with schools and government agencies to advocate for the least toxic methods of pest control, and to inform communities when pesticides are being used in the area. The policy statement also makes a number of recommendations for government, including specific recommendations related to marketing, labeling, use and safety of pesticides to minimize children's exposure.

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The American Academy of Pediatrics is an organization of 60,000 primary care pediatricians, pediatric medical subspecialists and pediatric surgical specialists dedicated to the health, safety and well-being of infants, children, adolescents and young adults. For more information, visit www.aap.org.

PEDIATRICS
OFFICIAL JOURNAL OF THE AMERICAN ACADEMY OF PEDIATRICS

Pesticide Exposure in Children

COUNCIL ON ENVIRONMENTAL HEALTH

Pediatrics; originally published online November 26, 2012;
DOI: 10.1542/peds.2012-2757

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FROM THE AMERICAN ACADEMY OF PEDIATRICS

Organizational Principles to Guide and Define the Child Health Care System and/or Improve the Health of all Children

POLICY STATEMENT

Pesticide Exposure in Children

COUNCIL ON ENVIRONMENTAL HEALTH

KEY WORDS
pesticides, toxicity, children, pest control, integrated pest management

ABBREVIATIONS
EPA—Environmental Protection Agency
IPM—integrated pest management

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PEDIATRICS (ISSN Numbers: Print, 0031-4005; Online, 1098-4275).

abstract

FREE

This statement presents the position of the American Academy of Pediatrics on pesticides. Pesticides are a collective term for chemicals intended to kill unwanted insects, plants, molds, and rodents. Children encounter pesticides daily and have unique susceptibilities to their potential toxicity. Acute poisoning risks are clear, and understanding of chronic health implications from both acute and chronic exposure are emerging. Epidemiologic evidence demonstrates associations between early life exposure to pesticides and pediatric cancers, decreased cognitive function, and behavioral problems. Related animal toxicology studies provide supportive biological plausibility for these findings. Recognizing and reducing problematic exposures will require attention to current inadequacies in medical training, public health tracking, and regulatory action on pesticides. Ongoing research describing toxicologic vulnerabilities and exposure factors across the life span are needed to inform regulatory needs and appropriate interventions. Policies that promote integrated pest management, comprehensive pesticide labeling, and marketing practices that incorporate child health considerations will enhance safe use. *Pediatrics* 2012;130:e1757–e1763

INTRODUCTION

Pesticides represent a large group of products designed to kill or harm living organisms from insects to rodents to unwanted plants or animals (eg, rodents), making them inherently toxic (Table 1). Beyond acute poisoning, the influences of low-level exposures on child health are of increasing concern. This policy statement presents the position of the American Academy of Pediatrics on exposure to these products. It was developed in conjunction with a technical report that provides a thorough review of topics presented here: steps that pediatricians should take to identify pesticide poisoning, evaluate patients for pesticide-related illness, provide appropriate treatment, and prevent unnecessary exposure and poisoning.[1] Recommendations for a regulatory agenda are provided as well, recognizing the role of federal agencies in ensuring the safety of children while balancing the positive attributes of pesticides. Repellents reviewed previously (eg, N,N-diethyl-meta-toluamide, commonly known as DEET; picaridin) are not discussed.[2]

SOURCES AND MECHANISMS OF EXPOSURE

Children encounter pesticides daily in air, food, dust, and soil and on surfaces through home and public lawn or garden application, household insecticide use, application to pets, and agricultural product

TABLE 1 Categories of Pesticides and Major Classes

Pesticide category	Major Classes	Examples
Insecticides	Organophosphates	Malathion, methyl parathion, acephate
	Carbamates	Aldicarb, carbaryl, methomyl, propoxur
	Pyrethroids/pyrethrins	Cypermethrin, fenvalerate, permethrin
	Organochlorines	Lindane
	Neonicotinoids	Imidacloprid
	N-phenylpyrazoles	Fipronil
Herbicides	Phosphonates	Glyphosate
	Chlorophenoxy herbicides	2,4-D, mecoprop
	Dipyridyl herbicides	Diquat, paraquat
	Nonselective	Sodium chlorate
Rodenticides	Anticoagulants	Warfarin, brodifacoum
	Convulsants	Strychnine
	Metabolic poison	Sodium fluoroacetate
	Inorganic compounds	Aluminum phosphide
Fungicides	Thiocarbamates	Metam-sodium
	Triazoles	Fluconazole, myclobutanil, triadimefon
	Strobilurins	Pyraclostrobin, picoxystrobin
Fumigants	Halogenated organic	Methyl bromide, Chloropicrin
	Organic	Carbon disulfide, Hydrogen cyanide, Naphthalene
	Inorganic	Phosphine
Miscellaneous	Arsenicals	Lead arsenate, chromated copper arsenate, arsenic trioxide
	Pyridine	4-aminopyridine

residues.[3–9] For many children, diet may be the most influential source, as illustrated by an intervention study that placed children on an organic diet (produced without pesticide) and observed drastic and immediate decrease in urinary excretion of pesticide metabolites.[10] In agricultural settings, pesticide spray drift is important for residences near treated crops or by take-home exposure on clothing and footwear of agricultural workers.[9,11,12] Teen workers may have occupational exposures on the farm or in lawn care.[13–15] Heavy use of pesticides may also occur in urban pest control.[16]

Most serious acute poisoning occurs after unintentional ingestion, although poisoning may also follow inhalational exposure (particularly from fumigants) or significant dermal exposure.[17]

ACUTE PESTICIDE TOXICITY

Clinical Signs and Symptoms

High-dose pesticide exposure may result in immediate, devastating, even lethal consequences. Table 2 summarizes features of clinical toxicity for the major pesticides classes. It highlights the similarities of common classes of pesticides (eg, organophosphates, carbamates, and pyrethroids) and underscores the importance of discriminating among them because treatment modalities differ. Having an index of suspicion based on familiarity with toxic mechanisms and taking an environmental history provides the opportunity for discerning a pesticide's role in clinical decision-making.[18] Pediatric care providers have a poor track record for recognition of acute pesticide poisoning.[19–21] This reflects their self-reported lack of medical education and self-efficacy on the topic.[22–26] More in-depth review of acute toxicity and management can be found in the accompanying technical report or recommended resources in Table 3.

The local or regional poison control center plays an important role as a resource for any suspected pesticide poisoning.

There is no current reliable way to determine the incidence of pesticide exposure and illness in US children. Existing data systems, such as the American Association of Poison Control Centers' National Poison Data System or the National Institute for Occupational Safety and Health's Sentinel Event Notification System for Occupational Risks,[27,28] capture limited information about acute poisoning and trends over time.

There is also no national systematic reporting on the use of pesticides by consumers or licensed professionals. The last national survey of consumer pesticide use in homes and gardens was in 1993 (Research Triangle Institute study).[29]

Improved physician education, accessible and reliable biomarkers, and better diagnostic testing methods to readily identify suspected pesticide illness would significantly improve reporting and surveillance. Such tools would be equally important in improving clinical decision-making and reassuring families if pesticides can be eliminated from the differential diagnosis.

The Pesticide Label

The pesticide label contains information for understanding and preventing acute health consequences: the active ingredient; signal words identifying acute toxicity potential; US Environmental Protection Agency (EPA) registration number; directions for use, including protective equipment recommendations, storage, and disposal; and manufacturer's contact information.[30] Basic first aid advice is provided, and some labels contain a "note for physicians" with specific relevant medical information. The label does not specify the pesticide class or "other"/"inert" ingredients that may have significant toxicity and can account for up to 99% of the product.

Chronic toxicity information is not included, and labels are predominantly available in English. There is significant use of illegal pesticides (especially in immigrant communities), off-label use, and overuse, underscoring the importance of education, monitoring, and enforcement.[31]

TABLE 2 Common Pesticides: Signs, Symptoms, and Management Considerations[a]

Class	Acute Signs and Symptoms	Clinical Considerations
Organophosphate and N-methyl carbamate insecticides	• Headache, nausea, vomiting, abdominal pain, and dizziness • Hypersecretion: sweating, salivation, lacrimation, rhinorrhea, diarrhea, and bronchorrhea • Muscle fasciculation and weakness, and respiratory symptoms (bronchospasm, cough, wheezing, and respiratory depression) • Bradycardia, although early on, tachycardia may be present • Miosis • Central nervous system: respiratory depression, lethargy, coma, and seizures	• Obtain red blood cell and plasma cholinesterase levels • Atropine is primary antidote • Pralidoxime is also an antidote for organophosphate and acts as a cholinesterase reactivator • Because carbamates generally produce a reversible cholinesterase inhibition, pralidoxime is not indicated in these poisonings
Pyrethroid insecticides	• Similar findings found in organophosphates including the hypersecretion, muscle fasciculation, respiratory symptoms, and seizures • Headache, fatigue, vomiting, diarrhea, and irritability • Dermal: skin irritation and paresthesia	• At times have been mistaken for acute organophosphate or carbamate poisoning • Symptomatic treatment • Treatment with high doses of atropine may yield significant adverse results • Vitamin E oil for dermal symptoms
Neonicotinoid insecticides	• Disorientation, severe agitation, drowsiness, dizziness, weakness, and in some situations, loss of consciousness • Vomiting, sore throat, abdominal pain • Ulcerations in upper gastrointestinal tract	• Supportive care • Consider sedation for severe agitation • No available antidote • No available diagnostic test
Fipronil (N-phenylpyrazole insecticides)	• Nausea and vomiting • Aphthous ulcers • Altered mental status and coma • Seizures	• Supportive care • No available antidote • No available diagnostic test
Lindane (organochlorine insecticide)	• Central nervous system: mental status changes and seizures • Paresthesia, tremor, ataxia and hyperreflexia	• Control acute seizures with lorazepam • Lindane blood level available as send out
Glyphosate (phosphonate herbicides)	• Nausea and vomiting • Aspiration pneumonia type syndrome • Hypotension, altered mental status, and oliguria in severe cases • Pulmonary effects may in fact be secondary to organic solvent	• Supportive care • Pulmonary effects may be secondary to organic solvent
Chlorophenoxy herbicides	• Skin and mucous membrane irritation • Vomiting, diarrhea, headache, confusion • Metabolic acidosis is the hallmark • Renal failure, hyperkalemia, and hypocalcemia • Probable carcinogen	• Consider urine alkalinization with sodium bicarbonate in IV fluids
Rodenticides (long-acting anticoagulants)	• Bleeding: gums, nose, and other mucous membrane sites • Bruising	• Consider PT (international normalized ratio) • Observation may be appropriate for some clinical scenarios in which it is not clear a child even ingested the agent • Vitamin K indicated for active bleeding (IV vitamin K) or for elevated PT (oral vitamin K)

IV, intravenous; PT, prothrombin time.

[a] Expanded version of this table is available in the accompanying technical report.[1]

CHRONIC EFFECTS

Dosing experiments in animals clearly demonstrate the acute and chronic toxicity potential of multiple pesticides. Many pesticide chemicals are classified by the US EPA as carcinogens. The past decade has seen an expansion of the epidemiologic evidence base supporting adverse effects after acute and chronic pesticide exposure in children. This includes increasingly sophisticated studies addressing combined exposures and genetic susceptibility.[1]

Chronic toxicity end points identified in epidemiologic studies include adverse birth outcomes including preterm birth, low birth weight, and congenital

TABLE 3 Pesticide and Child Health Resources for the Pediatrician

Topic/Resource	Additional Information	Contact Information
Management of acute pesticide poisoning		
Recognition and Management of Pesticide Poisonings	Print: fifth (1999) is available in Spanish, English; 6th edition available 2013	http://www.epa.gov/pesticides/safety/healthcare/handbook/handbook.htm
Regional Poison Control Centers		1 (800) 222-1222
Chronic exposure information and specialty consultation		
The National Pesticide Medical Monitoring Program (NPMMP)	Cooperative agreement between Oregon State University and the US EPA. NPMMP provides informational assistance by E-mail in the assessment of human exposure to pesticides	npmmp@oregonstate.edu or by fax at (541) 737-9047
Pediatric Environmental Health Specialty Units (PEHSUs)	Coordinated by the Association of Occupational and Environmental Clinics to provide regional academically based free consultation for health care providers	www.aoec.org/PEHSU.htm; toll-free telephone number (888) 347-AOEC (extension 2632)
Resources for safer approaches to pest control		
US EPA	Consumer information documents • Household pest control • Alternatives to chemical pesticides • How to choose pesticides • How to use, store, and dispose of them safely • How to prevent pesticide poisoning • How to choose a pest-control company	www.epa.gov/oppfead1/Publications/Cit_Guide/citguide.pdf
Citizens Guide to Pest Control and Pesticide Safety		
Controlling pests	Recommended safest approaches and examples of programs	www.epa.gov/pesticides/controlling/index.htm
The University of California Integrative Pest Management Program	Information on IPM approaches for common home and garden pests	www.ipm.ucdavis.edu
Other resources		
National research programs addressing children's health and pesticides	• NIEHS/EPA Centers for Children's Environmental Health & Disease Prevention Research • The National Children's Study	www.niehs.nih.gov/research/supported/centers/prevention www.nationalchildrensstudy.gov/Pages/default.aspx
US EPA	Pesticide product labels	www.epa.gov/pesticides/regulating/labels/product-labels.htm#projects
The National Library of Medicine "Tox Town"	Section on pesticides that includes a comprehensive and well-organized list of web link resources on pesticides	http://toxtown.nlm.nih.gov/text_version/chemicals.php?id=23

anomalies, pediatric cancers, neurobehavioral and cognitive deficits, and asthma. These are reviewed in the accompanying technical report. The evidence base is most robust for associations to pediatric cancer and adverse neurodevelopment. Multiple case-control studies and evidence reviews support a role for insecticides in risk of brain tumors and acute lymphocytic leukemia. Prospective contemporary birth cohort studies in the United States link early-life exposure to organophosphate insecticides with reductions in IQ and abnormal behaviors associated with attention-deficit/hyperactivity disorder and autism. The need to better understand the health implications of ongoing pesticide use practices on child health has benefited from these observational epidemiologic data.[32]

EXPOSURE PREVENTION APPROACHES

The concerning and expanding evidence base of chronic health consequences of pesticide exposure underscores the importance of efforts aimed at decreasing exposure.

Integrated pest management (IPM) is an established but undersupported approach to pest control designed to minimize and, in some cases, replace the use of pesticide chemicals while achieving acceptable control of pest populations.[33] IPM programs and knowledge have been implemented in agriculture and to address weeds and pest control in residential settings and schools, commercial structures, lawn and turf, and community gardens. Reliable resources are available from the US EPA and University of California—Davis (Table 3). Other local policy approaches in use are posting warning signs of pesticide use, restricting spray zone buffers at schools, or restricting specific types of pesticide products in schools. Pediatricians can

play a role in promotion of development of model programs and practices in the communities and schools of their patients.

RECOMMENDATIONS

Three overarching principles can be identified: (1) pesticide exposures are common and cause both acute and chronic effects; (2) pediatricians need to be knowledgeable in pesticide identification, counseling, and management; and (3) governmental actions to improve pesticide safety are needed. Whenever new public policy is developed or existing policy is revised, the wide range of consequences of pesticide use on children and their families should be considered. The American Academy of Pediatrics, through its chapters, committees, councils, sections, and staff, can provide information and support for public policy advocacy efforts. See http://www.aap.org/advocacy.html for additional information or contact chapter leadership.

Recommendations to Pediatricians

1. Acute exposures: become familiar with the clinical signs and symptoms of acute intoxication from the major types of pesticides. Be able to translate clinical knowledge about pesticide hazards into an appropriate exposure history for pesticide poisoning.
2. Chronic exposures: become familiar with the subclinical effects of chronic exposures and routes of exposures from the major types of pesticides.
3. Resource identification: know locally available resources for acute toxicity management and chronic low-dose exposure (see Table 3).
4. Pesticide labeling knowledge: Understand the usefulness and limitations of pesticide chemical information on pesticide product labels.
5. Counseling: Ask parents about pesticide use in or around the home to help determine the need for providing targeted anticipatory guidance. Recommend use of minimal-risk products, safe storage practices, and application of IPM (least toxic methods), whenever possible.
6. Advocacy: work with schools and governmental agencies to advocate for application of least toxic pesticides by using IPM principles. Promote community right-to-know procedures when pesticide spraying occurs in public areas.

Recommendations to Government

1. Marketing: ensure that pesticide products as marketed are not attractive to children.
2. Labeling: include chemical ingredient identity on the label and/or the manufacturer's Web site for all product constituents, including inert ingredients, carriers, and solvents. Include a label section specific to "Risks to children," which informs users whether there is evidence that the active or inert ingredients have any known chronic or developmental health concerns for children. Enforce labeling practices that ensure users have adequate information on product contents, acute and chronic toxicity potential, and emergency information. Consider printing or making available labels in Spanish in addition to English.
3. Exposure reduction: set goal to reduce exposure overall. Promote application methods and practices that minimize children's exposure, such as using bait stations and gels, advising against overuse of pediculicides. Promote education regarding proper storage of product.
4. Reporting: make pesticide-related suspected poisoning universally reportable and support a systematic central repository of such incidents to optimize national surveillance.
5. Exportation: aid in identification of least toxic alternatives to pesticide use internationally, and unless safer alternatives are not available or are impossible to implement, ban export of products that are banned or restricted for toxicity concerns in the United States.
6. Safety: continue to evaluate pesticide safety. Enforce community right-to-know procedures when pesticide spraying occurs in public areas. Develop, strengthen, and enforce standards of removal of concerning products for home or child product use. Require development of a human biomarker, such as a urinary or blood measure, that can be used to identify exposure and/or early health implications with new pesticide chemical registration or reregistration of existing products. Developmental toxicity, including endocrine disruption, should be a priority when evaluating new chemicals for licensing or reregistration of existing products.
7. Advance less toxic pesticide alternatives: increase economic incentives for growers who adopt IPM, including less toxic pesticides. Support research to expand and improve IPM in agriculture and nonagricultural pest control.
8. Research: support toxicologic and epidemiologic research to better identify and understand health risks associated with children's exposure to pesticides. Consider supporting another national study of pesticide use in the home and garden setting of US households as a targeted initiative or through cooperation with existing research opportunities (eg, National Children's Study, NHANES).
9. Health provider education and support: support educational efforts to increase the capacity of pediatric health care providers to diagnose and manage acute pesticide

poisoning and reduce pesticide exposure and potential chronic pesticide effects in children. Provide support to systems such as Poison Control Centers to provide timely, expert advice on exposures. Require the development of diagnostic tests to assist providers with diagnosing (and ruling out) pesticide poisoning.

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Pesticide Exposure in Children

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TECHNICAL REPORT

Pesticide Exposure in Children

James R. Roberts, MD, MPH, Catherine J. Karr, MD, PhD, and COUNCIL ON ENVIRONMENTAL HEALTH

KEY WORDS
pesticides, toxicity, children, pest control, integrated pest management

ABBREVIATIONS
CDC—Centers for Disease Control and Prevention
CI—confidence interval
2,4-D—2,4-dichlorophenoxyacetic acid
DDE—*p,p'*-dichlorodiphenyldichloroethylene
EPA—Environmental Protection Agency
ES—Ewing sarcoma
GI—gastrointestinal
INR—international normalized ratio
IPM—integrated pest management
NPDS—National Poison Data System
OP—organophosphate
OR—odds ratio
PT—prothrombin time
RR—relative risk
SGA—small for gestational age
Th2—T helper 2

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abstract

FREE

Pesticides are a collective term for a wide array of chemicals intended to kill unwanted insects, plants, molds, and rodents. Food, water, and treatment in the home, yard, and school are all potential sources of children's exposure. Exposures to pesticides may be overt or subacute, and effects range from acute to chronic toxicity. In 2008, pesticides were the ninth most common substance reported to poison control centers, and approximately 45% of all reports of pesticide poisoning were for children. Organophosphate and carbamate poisoning are perhaps the most widely known acute poisoning syndromes, can be diagnosed by depressed red blood cell cholinesterase levels, and have available antidotal therapy. However, numerous other pesticides that may cause acute toxicity, such as pyrethroid and neonicotinoid insecticides, herbicides, fungicides, and rodenticides, also have specific toxic effects; recognition of these effects may help identify acute exposures. Evidence is increasingly emerging about chronic health implications from both acute and chronic exposure. A growing body of epidemiological evidence demonstrates associations between parental use of pesticides, particularly insecticides, with acute lymphocytic leukemia and brain tumors. Prenatal, household, and occupational exposures (maternal and paternal) appear to be the largest risks. Prospective cohort studies link early-life exposure to organophosphates and organochlorine pesticides (primarily DDT) with adverse effects on neurodevelopment and behavior. Among the findings associated with increased pesticide levels are poorer mental development by using the Bayley index and increased scores on measures assessing pervasive developmental disorder, inattention, and attention-deficit/hyperactivity disorder. Related animal toxicology studies provide supportive biological plausibility for these findings. Additional data suggest that there may also be an association between parental pesticide use and adverse birth outcomes including physical birth defects, low birth weight, and fetal death, although the data are less robust than for cancer and neurodevelopmental effects. Children's exposures to pesticides should be limited as much as possible. *Pediatrics* 2012;130:e1765–e1788

INTRODUCTION

Pesticides represent a broad classification of chemicals that are applied to kill or control insects, unwanted plants, molds, or unwanted animals (eg, rodents). "Pesticide" is a collective term for a wide array of products but is often inappropriately used in reference to only insecticides. The universe of pesticide types and products is broad, and

a comprehensive review of all active ingredients is beyond the scope of this report. This review focuses on select insecticides, herbicides, and rodenticides and specific chemical classes within these groups that have the greatest acute and chronic toxicity for children on the basis of historical experience and/or emerging evidence (Table 1).

Several types of pesticides are not discussed in this report. Fumigants and fungicides, although potentially toxic, are less commonly involved in acute childhood exposure and poisoning, in general, so these are not included. Wood preservatives containing arsenic are also not included in this report. The specific compound containing arsenic, copper chromium arsenate, has been removed from the market since January 2004. Older wood structures treated with copper chromium arsenate may still be found in homes, on playgrounds, and in yards and should be treated yearly with a waterproof sealant.[1] Insect repellents, including *N*, *N*-diethyl-meta-toluamide and picaridin, are different from most pesticides in that they are a product purposefully applied to human skin to prevent insect bites and are, in fact, not insecticides. These compounds are unique and have been reviewed recently.[2]

Although the severity of pesticide exposures and toxicity may be greater in developing countries where regulatory oversight and information is limited, the content of this technical report is oriented toward exposures most relevant to children residing in the United States. Commonly used insecticides, including the organophosphates (OPs), carbamate, and pyrethroid classes, are discussed, as are the relatively new neonicotinoids. Other pesticides that will be discussed in some detail include the phosphonate herbicides (eg, glyphosate), chlorophenoxy herbicides, and long-acting anticoagulants (rodenticides). For a more comprehensive survey of the acute toxicity from the spectrum of pesticide active ingredients and products, see other sources.[1,3]

CHILDREN'S EXPOSURE: VULNERABILITY, MECHANISMS, AND SOURCES OF EXPOSURE

Children's Unique Vulnerabilities

Children are uniquely vulnerable to uptake and adverse effects of pesticides because of developmental, dietary, and physiologic factors. Exposure occurs through ingestion, inhalation, or dermal contact. Unintentional ingestion by children may be at a considerably higher dose than an adult because of the greater intake of food or fluids per pound of body weight. Children exhibit frequent hand-to-mouth activity, and this is an important source of increased exposure in comparison with adults.[4,5]

Residential Factors

Fortunately, acute toxicity attributable to pesticide poisoning is relatively uncommon in US children, and a pediatrician in general practice may not encounter such an event. However, subacute and chronic low-level exposure is common. Residential factors that influence chronic exposure include the use of insecticides and rodenticides in the home, and herbicide and fungicide use on lawns, as well. Indoors, broadcast applications including sprays, "flea bombs," and foggers can leave lingering residues in the air, carpet, toys, and house dust.[6–9] Typical exploratory behavior, including playing on and crawling across the floor, increases the risk of dermal, inhalation, and oral exposure to residues on surfaces or the air as it settles.[10] Repeated and cumulative incidental exposure can also occur. Pesticides can be measured in indoor air samples and persist in dust vacuumed from carpeted areas, upholstered objects, and children's toys, such as stuffed animals, and can also be brought home from the workplace.[11–14] Herbicides applied on the lawn or garden can be tracked into the home, with residues building up over time.[15] Applications of diazinon to lawns have been demonstrated to be carried indoors via the paws of pet dogs.[16] Residential pesticide residue levels also vary geographically according to the specific pesticide needs in the area. In Los Angeles, high levels of chlorpyrifos and other insecticides were found because of the large numbers of crawling insects, fleas, and termites. Conversely, in Iowa, there were high levels of the herbicides 2,4-dichlorophenoxyacetic acid (2,4-D) and dicamba because of weed-control applications.[17]

Residentially related sources may be relevant in other settings where children spend time, including school, child care, a relative's home, etc, depending on indoor and outdoor pesticide use patterns and proximity to pesticide use. In a North Carolina study of 142 urban homes and preschools, chlorpyrifos was detected in all indoor air and dust samples.[18]

Biomonitoring Data for Exposure Assessment

The Centers for Disease Control and Prevention (CDC) conducts a population-based biomonitoring program associated with the NHANES.[19] The most recent report includes biomarker data for many organochlorine, OP, and carbamate insecticides; herbicides; pyrethroid insecticides; and some other pesticides. Testing of 44 pesticide metabolites revealed that 29 were detectable in most people from whom samples were analyzed (ages 6–59 years), with OP and organochlorine insecticides reported to be most prevalent in the US population.[19] Although the health implications of these "snapshot" sampling data are largely unknown, they do

TABLE 1 Major Pesticide Classes and Selected Examples

Pesticide Class	Examples	Toxicity	Comment, Uses
Organochlorines	DDT, endrin, aldrin, chlordane, lindane	• High toxicity	• Many organochlorines now banned in the United States • Lindane has been banned in California, elsewhere used for control of lice and scabies • DDT and other organochlorines have long metabolic disposition and are stored in fatty tissues and can persist in the environment
Organophosphates	Parathion, chlorpyrifos, dichlorvos, acephate, methyl-parathion, malathion, phorate	• Most OPs are highly toxic • Malathion is considered relatively less toxic than other OPs	• Parathion is banned for use in the United States • Chlorpyrifos is no longer approved for residential use • Most others are used for insect control in both agricultural and home settings • Malathion is an approved treatment of head lice
N-Methyl carbamates	Aldicarb, carbaryl, carbofuran, pirimicarb, propoxur	• Aldicarb and carbaryl are both highly toxic • Other carbamates have a relatively moderate toxicity	• Insect control in agricultural and home settings
Pyrethrins and pyrethroids	Permethrin, cyano-pyrethroids: deltamethrin, cypermethrin, fenvalerate	• Permethrin has relatively low toxicity • Other pyrethroids have moderate toxicity	• Permethrin is a common pediculicide • Most other pyrethroids are commonly used to control insects, often used in home and garden
Neonicotinoids	Imidacloprid	• Relatively newer class of insecticides	• Selective affinity toward insect nicotinic acetylcholine receptors compared with mammalian nicotinic acetylcholine receptors
		• Have relatively lower toxicity than OPs and carbamates	• Often used as spot-on flea control for domestic animals
N-Phenylpyrazole insecticides	Fipronil	• Relatively newer class of insecticides	• Often used as spot-on flea control for domestic animals • Yard treatments for insect control
Phosphonate herbicides	Glyphosate	• Because of primary mechanism of action, has relatively low toxicity from active ingredient. • Toxicity often due to the accompanying organic solvent	• Acts on plant cell wall • Commercially available in many products
Chlorophenoxy herbicides	2,4-D, 2,4,5-T	• Moderate toxicity	• Weed control
Dipyridyl herbicides	Paraquat, diquat	• Highly toxic	• Infrequently used • Paraquat toxicity often requires lung transplant
Long-acting anticoagulants	Brodifacoum (superwarfarins)		• Rodenticides • Longer-acting than warfarin • Recently eliminated packaging as loose pellets

2,4,5-T, 2,4,5-trichlorophenoxy acetic acid.

provide a reference point on pesticide metabolite distributions. Periodic reassessment also allows for evaluations of population-level exposure trends.

As noted previously, children's unique behaviors and metabolic rate often place them at risk for absorption of higher doses from contaminated environments in comparison with adults. One example evident from the biomonitoring data is chlorpyrifos, a nonpersistent OP insecticide. Although banned in 2000 for use inside the home, it continues to be used in agriculture, including orchard fruits, such as apples and pears, and other dietary staples of children. In the CDC biomonitoring data, chlorpyrifos-specific urinary metabolites were highest for the youngest age group assessed (6–11 years) compared with older children and adults.[19] In contrast, biomonitoring of serum markers of organochlorine insecticides and their metabolites, such as DDT, dieldrin, and chlordane, many of which were banned from use in the United States in the 1970s and 1980s, revealed lower concentrations in the youngest age group monitored (12–19 years). Despite relatively lower concentrations, the ongoing detection and the higher levels with increasing age likely reflect the influence of the accumulation of these fat-soluble, persistent compounds over a lifetime.

Exposures From the Food Supply

In the general population, the food supply represents the most important source of exposure for organochlorines and OPs. For pyrethroids, both food residues and household pest control products are important sources.[20] The US Environmental Protection Agency (EPA) regulates exposure to pesticides in food by setting "tolerances," which are the maximum amount of pesticides that may legally remain in or on food

and animal feed. The US Food and Drug Administration is responsible for enforcement of these tolerances, which includes a modest monitoring program, which analyzed 7234 total samples in 2003. Among the domestically produced samples, 49% of fruit, 29% of vegetables, 26% of grain products, 24% of fish/shellfish, and 0% of milk/dairy tested had detectable but legally allowable pesticide residues. Only fruit and vegetables had residues above the legal tolerance (approximately 2% each). Overall, the detection of residues in the samples from imported fruits and vegetables tested were less, but the exceedances of legal tolerances were greater (5%–7% of imported fruits/vegetables sampled).[21] Consumption of organic food may lower pesticide exposure, as demonstrated by a study in which children were placed on an organic diet for a period of 5 consecutive days. A rapid and dramatic drop in their urinary excretion of metabolites of malathion and chlorpyrifos OP insecticides during the organic diet phase was observed.[22]

Agriculturally Related Exposures

Proximity to pesticide-treated agricultural areas or household members that work with pesticides presents another opportunity for contamination of the residential environment for some children. In a Washington State study of children of agricultural workers and nonagricultural workers in an agricultural setting, pesticide levels in carpet dust and pesticide metabolites in urine of residents increased with self-reported proximity of homes to orchard fields and during the pesticide application season.[9,23] Similarly, in an agriculture center in California, pesticide residues of 3 chemicals used recently on crops were significantly correlated with house dust samples in nearby homes and urine samples among their inhabitants. The findings were noted in both farmworkers and nonfarmworkers.[24] The presence of an agricultural worker in the home also increases pesticide levels through "take-home" exposures.[23] Children living on a farm had higher urinary pesticide metabolite levels than children not living on a farm.[25] Children themselves may participate in agricultural work that involves the use of pesticides or contact with pesticide-treated foliage.[26–28]

Exposures From Drinking Water

Contamination of drinking water presents another potential source of exposure, particularly for herbicides. A 10-year study (1992–2001) by the US Geological Survey's National Water-Quality Assessment program provided a national-scale view of pesticide occurrence in streams and groundwater. Overall, pesticides were detected in more than 50% of sampled wells from shallow groundwater tapped beneath agricultural and urban areas as well as in 33% of the deeper wells that tap major aquifers used for water supply. The concentrations associated with these detections rarely exceeded water quality health reference levels (approximately 1% of the 2356 domestic and 364 public-supply wells that were sampled). Herbicides, particularly the triazine class, were the most frequently detected pesticide group in agricultural areas. (It should be noted that atrazine and other triazine herbicides were monitored from surface water.) In urban areas, both herbicides and insecticides (particularly diazinon and carbaryl) were frequently detected. The greatest proportion of wells exceeding a health reference level was for those tapping shallow groundwater beneath urban areas. It is noteworthy that the detection of pesticides usually occurred as mixtures, and health reference levels reflected exposure to a single agent.[29]

NATIONAL DATA ON ACUTE EXPOSURE, MORBIDITY, AND MORTALITY

Although some states (eg, California and Washington) mandate the reporting of pesticide-related illness, there is no national surveillance system for pesticide exposure and poisoning. The American Association of Poison Control Centers' National Poison Data System (NPDS [formerly known as the Toxic Exposure Surveillance System]) compiles annual data on pesticide exposures. Incidents reported by the NPDS are categorized by age (<6 years, 6–19 years, and >19 years), reason (unintentional, intentional, other, adverse reaction), and outcome (none [no morbidity], minor, moderate, major, or death). However, these data represent self-reports from patients and/or family members and calls from medical treatment facilities. Although they are useful to describe trends, they do not indicate true prevalence or incidence. Data are reported annually and, since 2005, have been published in *Clinical Toxicology*.[30]

In 2009, pesticides were the tenth most frequently involved substance in human exposure (3.9% of all NPDS reports) and the ninth most common substance encountered in children (3.3% of pediatric NPDS reports). Nearly 55.8% of all single-substance pesticide exposures involved children ≤19 years of age, and 94% of all pesticide ingestions were unintentional. Twenty-one of the reports from pesticide exposure resulted in death; however, these were not categorized by age.[30] Rates (calculated by using US census data for the catchment area served by the poison control center as the denominator) of reported pesticide poisonings described as moderate, major, and fatal declined from 1995 to 2004 by approximately 42%. The sharpest declines in poisonings were from OP and carbamate insecticides,

likely reflecting EPA regulatory action to discontinue residential use of several previously widely available OP and carbamate insecticides on the basis of child health concerns.[31]

ACUTE TOXICITY MECHANISMS AND CLINICAL MANIFESTATIONS

OP and Carbamate Insecticides

OP and carbamates insecticides have been widely used for insect control in the home and in agriculture since the 1960s. During this period, OP and carbamates usage largely replaced the use of organochlorines because of environmental and human health concerns of the latter class. In the past 10 years, chemical products in the OP and carbamate group have come under scrutiny, with subsequent regulatory action based on human health concerns. Examples include 2 commonly used OPs with high acute toxicity: parathion (banned) and chlorpyrifos (no longer allowed for residential use). Other OPs that remain widely used include dichlorvos, acephate, methyl-parathion, and malathion. Malathion has relatively lower acute toxicity among the OPs and is registered for the treatment of head lice (Ovide). A well-known example of a carbamate is aldicarb, although use has largely been curtailed by regulatory action because of its high toxicity. Commonly used carbamates include carbaryl and pirimicarb.[1,3]

Toxicity, Clinical Signs, and Symptoms

OPs and carbamates exert a common mechanism of action by inhibiting the acetylcholinesterase enzyme, thereby producing accumulation of acetylcholine at the synapses, neuromuscular junction, and end organs, which results in excessive stimulation at those sites. The reaction is generally an irreversible binding by OPs and a reversible binding by carbamates, and it influences treatment approaches for each class of insecticides. Consequently, acute poisoning by OPs tends to be more severe and refractory than that of carbamates; however, variations are observed in each class. There are some notable carbamates (such as aldicarb) that have equal if not greater toxicity than some OPs.[1,3]

Acute clinical manifestations reflect the development of cholinergic crisis and can arise from stimulation of muscarinic, nicotinic, and/or central nervous system receptors (Table 2). Early findings can often mimic a flu-like illness and include hypersecretion. Miosis is a helpful diagnostic sign. The classic cardiovascular sign is bradycardia, although early on, tachycardia may be present initially because of nicotinic stimulation. Progressive symptoms lead to muscle and respiratory problems. The central nervous system may also be affected, signifying severe poisoning, particularly in children.[1,3,32–34] Reviews of case series indicate that between 20% and 30% will have seizures, and between 50% and 100% of children will have lethargy, stupor, or coma.[32–34] A high clinical suspicion plus directed and persistent environmental history taking to identify potential exposures are necessary to identify these poisonings. Reviews of pediatric poisonings note that, historically, most children were transferred to a referral center with the wrong preliminary diagnosis and parents initially denied any exposure history.[33,34]

Laboratory Evaluation and Treatment

Poisoning with OPs and carbamates can be detected on the basis of clinical findings and history of exposure. Laboratory confirmation can assist in the diagnosis by using red blood cell and plasma cholinesterase levels; both are typically depressed with acute poisoning, although there is some variation among active ingredients as well as variation in levels by severity of poisoning.[35] Measurement techniques and resultant levels vary among laboratories; therefore, clinicians will need to check with their own laboratory for reference values. Red blood cell cholinesterase levels typically are more specific for acute poisoning and will be depressed longer than plasma cholinesterase levels (often 1–3 months) until enzyme is replaced.[3] Interpretation of results can be discussed with a pediatric environmental health specialist or clinical toxicologist.

The parent active ingredient cannot typically be measured in biological specimens. These compounds undergo metabolic transformation in the liver and are excreted in the urine mostly in their metabolized form, most of which are nonspecific metabolites for all OPs.[19] Exceptions include parathion, methyl-parathion, and chlorpyrifos, all of which have their own specific metabolite in addition to the nonspecific metabolites. Urinary metabolites can be measured, and human data are available from the CDC on a nationally representative sample.[19] However, an evidence base to support clinical interpretation of urinary concentrations is lacking.

Treatment of OP poisoning (and this applies to the acute treatment of any other pesticide as well) begins with the basics of advanced life support, with any necessary airway or breathing support as needed. Gastrointestinal (GI) decontamination is controversial. The American Academy of Clinical Toxicology and the European Association of Poisons Centres and Clinical Toxicologists issued a joint statement on the use of single-dose charcoal for poisoned patients (inclusive of all types of poisonings). They stated that activated charcoal is most effective when given within 1 hour after the ingestion of a poison, but routine administration in all poisonings is not recommended.

TABLE 2 Clinical Signs and Symptoms

Class of Compounds	Signs and Symptoms	Special Notes, Laboratory Evaluations, Specific Treatments, or Antidote
Organophosphate and carbamate insecticides	• Nonspecific early symptoms: headache, nausea, vomiting, abdominal pain, and dizziness	• Red blood cell and plasma cholinesterase levels
	• Sometimes hypersecretion: sweating, salivation, lacrimation, rhinorrhea, diarrhea, and bronchorrhea	• Measure nonspecific metabolites for most OPs
	• Progressive symptoms: muscle fasciculation, muscle weakness, and respiratory symptoms (bronchospasm, cough, wheezing, and respiratory depression)	• Specific metabolites can be measured for chlorpyrifos and parathion
	• Bradycardia is typical, although early in acute poisoning, tachycardia may be present	• Atropine is primary antidote
	• Miosis	• Pralidoxime is also an antidote for OP and acts as a cholinesterase reactivator
	• Central nervous system: respiratory depression, lethargy, coma, and seizures	• Because carbamates generally produce a reversible cholinesterase inhibition, pralidoxime is not indicated in these poisonings
Pyrethroids	• Dermal: skin irritation and paresthesia	• At times have been mistaken for acute OP or carbamate poisoning and treated with atropine with potentially adverse or disastrous results
	• Nonspecific symptoms including headache, fatigue, vomiting, diarrhea, and irritability	• Symptomatic treatment
	• Similar findings found in OPs, including hypersecretion, muscle fasciculation, pulmonary symptoms and seizures	• Vitamin E oil for dermal symptoms
Neonicotinoids	• Disorientation, agitation—severe enough to require sedation, drowsiness, dizziness, weakness, and, in some situations, loss of consciousness	• Supportive care
	• Vomiting, sore throat, abdominal pain	• No available antidote
	• Ulcerations in upper GI tract	• No available diagnostic test
Fipronil (*N*-phenylpyrazole insecticides)	• Nausea and vomiting	• Supportive care
	• Aphthous ulcers	• No available antidote
	• Altered mental status and coma	• No available diagnostic test
	• Seizures	
Organochlorines	• Central nervous system: mental status changes and seizures	• Control acute seizures with lorazepam
	• Paresthesia, tremor, ataxia, and hyperreflexia	
Glyphosate (phosphonate herbicides)	• Nausea and vomiting	• Supportive care
	• Aspiration pneumonia type syndrome	
	• Hypotension, altered mental status, and oliguria in severe cases	
	• Aspiration pneumonia type syndrome	
	• Pulmonary effects may in fact be secondary to organic solvent	
Chlorophenoxy herbicides	• Skin and mucous membrane irritation	• Consider forced alkaline diuresis with sodium bicarbonate in IV fluids
	• Vomiting, diarrhea, headache, confusion	
	• Metabolic acidosis is the hallmark	
	• Renal failure, hyperkalemia, and hypocalcemia	
Long-acting anticoagulants (rodenticides)	• Bleeding: gums, nose, and other mucous membrane sites	• Consider PT (INR) or observation
	• Bruising	• Vitamin K indicated for bleeding (IV vitamin K) or for elevated PT (INR) (oral vitamin K)

IV, intravenous.

Activated charcoal is contraindicated if the patient does not have a protected or intact airway.[36] A randomized controlled trial evaluating the effect of multiple-dose charcoal for pesticide-poisoned patients in Asia found no benefit, as measured by a reduction in mortality.[37] Skin decontamination also is critically important, and clothing should be removed. Medical personnel should take measures to protect themselves from contaminated skin and clothing, because numerous cases of hospital-acquired OP poisoning have been documented.[38] Parents or other family caregivers may also be at risk for skin contamination. Seizures should be controlled with intravenous lorazepam.[3]

Atropine can be given as a nonspecific antidote in both OP and carbamate poisoning. It will reverse the muscarinic effects of the poisoning; however, it is less effective on central nervous system effects. It is given as a dose of

0.05 to 0.1 mg/kg per dose and may be given as often as every 15 minutes until respiratory secretions are controlled.[3] Notably, this dose is 10 times the usual dose given during a resuscitation situation, because the purpose is to overcome complete blockade of the muscarinic channel. Pralidoxime is also given as a specific antidote to reverse the acetylcholinesterase inhibitor complex. The use of pralidoxime continues to be of interest, particularly in developing countries, although most studies have been performed with adult patients.[39,40] The World Health Organization recommends its use for all patients who require atropine.[41] Its use is indicated for OP poisoning, because cholinesterase inhibition usually is permanent in OP poisoning. Use of pralidoxime usually is not necessary or recommended for carbamate poisoning, because this inhibition is reversible.[3]

Pyrethrins and Pyrethroid Insecticides

Pyrethrins and pyrethroids are a relatively more recent class of insecticides that have been largely replacing the use of cholinesterase-inhibiting insecticides, especially in the consumer market. These insecticides are used for structural pest control in urban areas, in gardening or agriculture for row crops and orchards, and in the home for pet sprays and shampoo.

The pyrethrins are botanically derived from pyrethrum, an extract of the chrysanthemum plant. For these consumer products, pyrethrins are usually combined with another active ingredient: either a longer-acting synthetically derived pyrethroid or one of the cholinesterase inhibitors. Pyrethrins are not stable in heat or sunlight and, therefore, are usually used more for indoor application. Permethrin is the most widely known example of a pyrethrin and is one of the few products licensed for use to apply to human skin, because it is commonly used as a pediculicide.[3,42,43]

Pyrethroids are synthetically derived compounds that have been modified to be more stable in sunlight and heat and are, therefore, used more widely for insect control, especially outdoors. Toxicity varies widely among pyrethrins and pyrethroids, and, although they are less acutely toxic as a class than the cholinesterase insecticides, there is a subgroup of these compounds that has been modified with a cyano side chain. This modification creates a compound that is significantly more resistant to degradation and potentially more acutely toxic than other pyrethroids. Commonly used chemicals in this subgroup include deltamethrin, cypermethrin, and fenvalerate—these are the insecticides to which the majority of toxic signs and symptoms in the next section apply.[43]

Toxicology, Clinical Signs, and Symptoms

Pyrethroids exert their toxic effect by blocking the sodium channel at the level of the cell membrane. Most clinical reports of poisoning occur either through excessive skin contact or through ingestion or inhalation. The result is continued hyperpolarization, effectively inhibiting cell function. Some types of pyrethroids also work at other sites, including voltage-dependent chloride channels and γ-aminobutyric acid–gated chloride channels. This appears to be one of the reasons for a variety of toxicity found among pyrethroid insecticides.[42,43] Pyrethroids with a cyano group, also known as type II pyrethroids, constitute most cases of human poisoning.[42,43] Pyrethroids are well absorbed across the GI tract, but limited penetration occurs across the skin barrier, which can limit acute toxicity.[42,44] Some pyrethroids have a high acute toxicity, usually after ingestion.[42,45] Pyrethroids are metabolized by the liver and excreted in their metabolic forms.

Pyrethroids have adverse effects on the nervous system, GI tract, and skin. Specific signs and symptoms are found in Table 2. Similar to OPs, muscle fasciculation, weakness, an altered level of consciousness, and seizures can develop after exposures to some pyrethroids.[42–45] Of note, paresthesias, including burning, tingling, stinging, and eventually numbness, are characteristic of pyrethroid exposure.[46,47] The paresthesias appear to be dose-dependent and occur at pyrethroid dosages lower than what would cause systemic toxicity, thereby acting as a warning of exposure. The paresthesias are self-limiting once exposure is eliminated.[48]

Laboratory Evaluation and Treatment

Pyrethroid toxicity is identified through clinical history and knowledge of exposure to the agent. There are no rapidly available diagnostic laboratory tests. Most pyrethroids are metabolized to 3-phenoxybenzoic acid, which can be recovered in the urine. CDC national surveys provide biomonitoring information on pyrethroid urinary metabolites and can act as comparison for background measures of exposure in the general population. However, in the clinical setting, results of metabolite levels are usually obtained from specialty laboratories and are not immediately available; therefore, these results not useful in acute clinical management.

Paresthesias are generally self-limiting and resolve within 24 hours.[46,48] If exposure is interrupted after the onset of paresthesias and other dermal findings, no additional treatment is necessary. Vitamin E oil or cream has been shown to improve the

symptoms associated with the paresthesias.[47] The mechanism is not completely clear; however, in experimental studies, vitamin E (α-tocopherol) blocked tetramethrin-modified sodium channels.[49]

Treatment of systemic pyrethroid poisoning is supportive, in general, and there are no specific antidotes. Because of the similar features of cholinesterase inhibitor poisoning, some patients have been treated erroneously with high atropine, sometimes with disastrous results.[45] Efforts have been aimed at antagonizing the sodium current resulting from the pyrethroid blockade. Several medications have been tested in the animal model, but, to date, none have been considered effective antidotes for systemic pyrethroid poisoning in humans. For significant neurologic effects, patients should have standard decontamination, including GI tract decontamination, supportive respiratory care, seizure control with diazepam or lorazepam, and careful dosing of atropine for excessive salivation.[42] Proper identification of the offending agent is imperative to distinguish these poisonings from OPs and often requires a high index of suspicion and a thorough exposure history.

Organochlorine Insecticide (Lindane)

The discussion of acute toxicity for organochlorines is focused on lindane, because most other organochlorine compounds have been banned for use in the United States. Other organochlorines, including DDT and some of the cyclodienes, including chlordane and dieldrin, are important compounds, because they can still persist in human and environmental samples. These chronic exposures are of continuing concern for developmental health effects, including immunotoxicity, endocrine disruption, and neurodevelopmental insults (see Chronic Health Effects of Pesticide Exposure).

Lindane, also known technically as the γ-isomer of hexachlorocyclohexane, is still approved in some states for control of lice and scabies. However, in a comparison of in vitro activity against lice with other pediculicides, it was the least effective.[50] It is efficiently absorbed across the skin (approximately 9%) and even more so across abraded skin, such as with severe excoriations from scabies.[51,52] Signs and symptoms are noted in Table 2. Treatment is supportive and includes decontamination and the control of seizures with lorazepam. There is no specific antidote. Lindane has been banned in California because of high levels found in the water supply.[53]

Neonicotinoids

Neonicotinoids are a new class of insecticides based on metabolic alterations of nicotine. They are used primarily in agriculture and are gaining widespread use for flea control on domestic animals. They act on the nicotinic *N*-acetylcholine receptors and selectively displace acetylcholine. They do have a relatively selective affinity for insects as opposed to mammals, although there have been a few reports of human poisoning.[54–56] The most commonly used neonicotinoid in the United States is imidacloprid. Information about toxicity and signs and symptoms can be found in Tables 1 and 2.

N-Phenylpyrazoles

Fipronil is the primary representative of this class and was developed in the mid-1990s. It is widely used in flea control on domestic pets. It is also used in ant and roach bait stations, agriculture crops, and lawn treatments. It acts by inhibiting γ-aminobutyric acid–gated chlorine channels. The inhibition will block chloride passage and result in hyperexcitability of the cell.[57–59] Signs and symptoms are reported in Table 2.

HERBICIDES

Chlorophenoxy Herbicides

Chlorophenoxy herbicide compounds are often mixed with fertilizers and are used both in agriculture and on residential lawns. These compounds are well absorbed from the GI tract but are not well absorbed after inhalational or dermal exposure.[60] Examples of commonly used chlorophenoxy herbicides are 2,4-D and 2,4,5-trichlorophenoxy acetic acid. The half-lives of these compounds range between 13 and 39 hours. They are mostly excreted unchanged in the urine; excretion can be greatly enhanced in an alkaline environment.[3,61,62] More toxic substances that can be produced during the manufacture of these herbicides include dioxins, which were contaminants of the herbicide Agent Orange and were found in the Love Canal chemical dump site.[63]

Primary initial effects are on the skin and mucous membranes. Severe poisoning will result in metabolic acidosis and possibly renal failure.[3,61,64] Specific symptoms are discussed in Tables 1 and 2. The compounds can be measured in the urine, although similar to pyrethroid insecticides, analyses are generally performed at specialty laboratories, so results are usually not immediately available to clinicians. Treatment is primarily supportive and may also include forced alkaline diuresis by adding sodium bicarbonate to the fluids and establishing a high urine pH and high urine flow.[3,61,65]

Phosphonate Herbicides (Glyphosate)

Glyphosate is a commonly used herbicide and is commercially available in

many products. Glyphosate acts on the cell wall of plants, so, theoretically, it should have no effect on human cells, at least by way of its primary mechanism of action. Despite this, there are numerous reports in the medical literature of adverse events after human exposure, particularly unintentional ingestions. Patients have presented with signs and symptoms consistent with an aspiration pneumonia–like syndrome, and the offending agent may be the hydrocarbon solvent with which the glyphosate is mixed. Treatment is primarily supportive, and providers should be vigilant for aspiration pneumonia.

RODENTICIDES (LONG-ACTING ANTICOAGULANTS)

Most currently used rodenticides belong to the class of warfarin-type anticoagulants. Unlike warfarin, the superwarfarin agents, such as brodifacoum, have a much longer half-life. Although they have traditionally been available as pellets that can be spread around or in a box that the rat can consume, the EPA has recently changed the type of products that are available to consumers. Since 2008, superwarfarins can only be sold as a child-resistant bait station instead of loose pellets.[66]

The mechanism of action is inhibition of the synthesis of vitamin K–dependent clotting factors. As such, the primary manifestations of toxicity are bleeding and easy bruisability. In severe cases, bleeding may be life-threatening. Clinicians who suspect that their patients may have ingested a superwarfarin should consider obtaining a prothrombin time (PT; also known as the international normalized ratio [INR]).[3] However, several studies that have analyzed cohorts of exposed children have found very few subjects with an elevated PT (INR) or active bleeding. Therefore, in situations in which it is unclear whether a child ingested more than a few pellets, it is reasonable to simply observe the child.[67–70] Most patients can be managed in the outpatient setting as long as the ingestion has been recognized early.[71]

Treatment is vitamin K and should be reserved for patients with elevated PT (INR) levels or active bleeding. With severe bleeding or shock, a transfusion of blood or plasma is indicated as well.[3]

CHRONIC HEALTH EFFECTS OF PESTICIDE EXPOSURE

The health implications of the nonacute, relatively low, but often repetitive and combined exposures encountered routinely by children are an ongoing focus of concern and inquiry for scientists, regulators, and parents.[72,73] Pediatricians are well placed to provide guidance to parents about potential long-term or subtle health effects from pesticide residues on food, in water, or used in homes or schools and on exposure-reduction strategies. However, surveys suggest pediatricians often feel ill-prepared with training in this topic, underscoring the importance of improving educational opportunities for clinical providers.[74–76]

The associated health effects of chronic pesticide exposure in children vary, reflecting the diversity of toxicological properties of this broad group of differing chemicals. Some of the important end points of concern include an increased risk of cancer, abnormal neurodevelopment, asthma, perturbation of gestational growth, and endocrine-mimicking effects. Health effects of pesticides and the current relative strength of the evidence base are reviewed in subsequent sections for each of these health outcomes.

Childhood Cancer

All pesticides undergo in vitro and animal testing to determine their likelihood of causing cancer. The EPA maintains a list and classification of all active ingredients in pesticides and their potential for carcinogenicity. The method of identifying potential carcinogenicity has changed. Before 1996, pesticides were assigned a letter classification (eg, pesticides with the "C" classification were considered "possibly carcinogenic"). Subsequently, pesticides have been assigned a category such as "likely to be carcinogenic to humans," "suggestive evidence of carcinogenic potential," "inadequate evidence," and "not likely." These categories are not directly comparable, so both classifications (before 1996) and categories (after 1996) continue to exist.

The pesticides that are categorized as "possibly carcinogenic" or "likely to be carcinogenic to humans" are available from the EPA via an e-mailed report.[77] Included in this report are some well-known and widely used OPs, carbamates, pyrethroids, and fungicides. Within classes of pesticides, variation in carcinogenicity potential exists. Note that a pesticide, such as cypermethrin, that has "replaced" use of cancer-causing OPs has cancer-causing potential.

A substantial amount of observational epidemiological data demonstrate a link between pesticide exposure and childhood cancers.[78–87] However, the evidence base includes studies that found no association between childhood cancers and pesticides or few associations that cannot be ruled out as a chance finding.[88,89] Overall, the most comprehensive reviews of the existing literature implicate an association of pesticides with leukemia and brain tumors.[78,79]

Leukemia

In 1998, Zahm and Ward[79] reviewed 18 studies assessing the relationship between pesticide exposure and leukemia; 13 studies found an elevated risk, and,

for 6 of those studies, the association was statistically significant. The most frequently occurring associations among the studies were between pesticide exposure and acute lymphocytic leukemia.

A 2007 review by Infante-Rivard and Weichenthal[78] summarized the 1998 review of Zahm and Ward and updated findings from recent studies. Although it was previously postulated that childhood exposure to agricultural products or proximity to an agricultural setting would present the highest risks, the most commonly associated pesticide exposure in childhood acute lymphocytic leukemia studies was household insecticide use. Cases were more likely to have had preconception exposure and/or exposures in utero in most studies. The main limitations with the studies in the 1998 review included crude exposure assessment, concern for recall bias, small numbers of exposed cases, and mixing of different leukemia types.[78]

In the updated review, 5 of 6 recent case-control studies found a statistically significant relationship between pesticide exposure and leukemia.[84,85,90–92] In particular, 2 studies included the most detailed exposure assessment to date and reported findings related to a dose/exposure–response gradient.[84,85] The primary risk factors were maternal exposure to pesticide between the periods of preconception through pregnancy. The largest of the 2 studies had 491 cases and an equal number of controls, focused only on acute lymphocytic leukemia, included a measure of frequency of use, and considered genetic susceptibility. For maternal use of herbicides, plant insecticides, and pesticides for trees during pregnancy, the odds ratio (OR) was 1.84 (95% confidence interval [CI], 1.32–2.57), 1.97 (95% CI, 1.32–2.94), and 1.70 (95% CI, 1.12–3.59), respectively. For parental use during the child's postnatal life, OR was 1.41 (95% CI, 1.06–1.86), 1.82 (95% CI, 1.31–2.52), and 1.41 (95% CI, 1.01–1.97) after exposure to herbicides, plant insecticides, and pesticides for trees, respectively.[84]

To further explore associations between pesticides and leukemia, a group of authors conducted 2 meta-analyses. They provided similar and additional support to the associations described previously. One examined studies that included parental occupational exposure (prenatally and in early childhood) and leukemia in their offspring. Maternal occupational exposure, but not paternal occupational exposure, was found to be associated with leukemia. The reported OR was 2.09 (95% CI, 1.51–2.88) for overall pesticide exposure, 2.38 (95% CI, 1.56–3.62) for insecticide exposure, and 3.62 (95% CI, 1.28–10.3) for herbicide exposure.[93] The second meta-analysis assessed pesticide exposure in the home and garden setting. In this meta-analysis, 15 studies were included, and exposures during pregnancy to unspecified pesticides, insecticides, and herbicides were all associated with leukemia (OR, 1.54 [95% CI, 1.13–2.11], 2.05 [95% CI, 1.80–2.32], and 1.61 [95% CI, 1.2–2.16], respectively).[94]

Brain Tumors

Zahm and Ward's 1998 review included 16 case-control studies examining associations between brain tumors and pesticide exposures. Of these, 12 found an increased risk estimate of brain tumors after pesticide exposure; 7 of these findings reached statistical significance. Associated exposures were most often from parental use of pesticides in the home, in the garden, and on pets. Interpretation of these studies is difficult given the inadequate exposure assessments, small numbers because of a relatively rare childhood outcome, and a mixture of brain tumor types among cases.[79]

Since 1998, 10 additional studies have been published, all but one of which demonstrated an increased risk estimate of cancer with maternal and/or paternal exposure, although not all studies demonstrated statistical significance. Some of the more robust findings come from a case-control study with 321 cases of astrocytomas. The risk estimate from maternal occupational exposure to insecticides before or during pregnancy was 1.9 (95% CI, 1.1–3.3). The risk estimates for paternal exposure for insecticides, herbicides, and fungicides were 1.5, 1.6, and 1.6, respectively. These risk estimates were just short of reaching statistical significance.[87] In a cohort study of more than 200 000 patients, paternal exposure in any occupation and in agricultural/forestry preceding conception was associated with an increased risk of central nervous system tumors (relative risk [RR], 2.36 [95% CI, 1.27–4.39] and RR, 2.12 [95% CI, 1.08–4.39], respectively).[83] For all studies, it appears that prenatal exposure to insecticides, particularly in the household, as well as both maternal and paternal occupational exposure before conception through birth represent the most consistent risk factors.[83,86,87,95–100]

Ewing Sarcoma

Two case-control studies were performed to evaluate potential parental occupational exposures and the development of Ewing sarcoma (ES). One study of 196 cases and matched controls found an association between ES in boys age 15 years or younger and household pesticide extermination (OR, 3.0; 95% CI, 1.1–9.2). There was no association between parental occupational exposure to pesticides and ES.[101] A study in Australia compared 106 cases of either ES or peripheral primitive neuroectodermal tumor with 344 population-based controls. Exposures

included prenatal exposure from conception through pregnancy and also included parental exposures through the time of the child's diagnosis. Notable elevated risks were observed for mothers who worked on farms (OR, 2.3; 95% CI, 0.5–12.0), mothers who handled pesticides (OR, 2.3; 95% CI, 0.6–8.5), patients who ever lived on a farm (OR, 2.0; 95% CI, 1.0–3.9), and farming fathers at the time of conception and/or pregnancy (OR, 3.5; 95% CI, 1.0–11.9).[102] Of note in this study, all 95% CIs include 1.0, so they did not reach statistical significance, although some ORs approached it.

In summary, there is some evidence of increased risk of developing several childhood cancers after preconception and/or prenatal exposure to pesticides. The strongest evidence appears to be for leukemia, which is a relatively more common type of childhood cancer than brain tumors. Maternal exposure to insecticides and paternal occupational exposure appear to carry the greatest risk.

Neurodevelopment/ Neurobehavioral Effects

Many pesticides have well-described acute neurotoxicant properties that have been described previously in this report in relation to human poisoning episodes and acute toxic mechanisms. However, information on the potential neurodevelopmental toxicity arising from chronic, low-level exposure in gestational or postnatal life is inadequate or lacking for most pesticides in use. There is a growing available evidence base supporting an adverse effect on neurodevelopment from 2 classes of insecticides, the organochlorines (specifically DDT and its metabolite *p,p*′-dichlorodiphenyldichloroethylene [DDE]) and, most recently, OPs. Several recent reviews of the evidence base are now available.[103–105]

Although chronic neurologic sequelae after acute OP poisoning have been observed in multiple adult studies, the epidemiological data on children are limited.[106,107] A recent neuropsychological evaluation of healthy school-aged children who had experienced hospitalization for acute OP poisoning before the age of 3 years found subtle but significant deficits in their ability to restrain and control their motor behaviors compared with both children who had no history of poisoning and children who had a history of early life poisoning with kerosene.[108]

Of greater public health concern is the potential neurotoxicity from routinely encountered chronic exposures. This is the subject of study in ongoing, large National Institutes of Health/EPA-sponsored prospective birth cohorts. Studies in 2 urban settings and a rural farmworker community have enrolled women during pregnancy with an objective assessment of exposure by the use of environmental measurements and biological monitoring.[104,109,110] Follow-up assessment of neurodevelopment and neurobehavior in their children with the use of validated tools such as the Brazelton Neonatal Assessment Scales, the Bayley Scales of Infant Development, the Child Behavior Checklist, and IQ testing at comparable intervals is being conducted. To date, remarkably similar findings relating adverse neurodevelopmental and neurobehavioral outcomes associated with prenatal OP exposure have been made in these distinct cohort studies. For example, in 2 cohorts, the Brazelton Neonatal Behavioral Assessment Scale was administered in the first weeks of life. In both, deficits in the primitive reflex domain were noted with the other 6 of 7 Brazelton Neonatal Behavioral Assessment Scale domains not associated with prenatal OP exposure.[111,112] Two of the cohorts have published their Bayley Mental and Psychomotor Developmental Index results conducted during the toddler years (ages 2–3).[113,114] Significantly poorer mental development was associated with higher OP exposure in both, whereas one of the cohorts also observed OP-associated deficits in the motor scale at 3 years of age. Results of Child Behavior Checklist assessments are also available for 2 cohorts, conducted at 2 years of age in one and 3 to 4 years of age in the other. Significantly increased scores representative of pervasive developmental disorder were associated with higher OP exposure in both.[113,114] One cohort also had increased scores for inattention and attention-deficit/hyperactivity disorder subscales.[114] All 3 cohorts have found decrements in IQ testing associated with higher prenatal exposures at the time of follow-up at 7 years of age.[115–117]

In one of the cohorts, postnatal exposure effects in the child have been investigated and reported. Interestingly, improved mental development based on Bayley's Index at 12 and 24 months of age is associated with higher contemporary child excretion of OP urinary metabolites. Explanations for this are debated but include theories that children with higher cognitive abilities may explore their environments more thoroughly and, as such, experience higher exposure.

Recently, a US-based cross-sectional analysis demonstrated that children with high urinary concentrations of OP metabolites were more likely to have a diagnosis of attention-deficit/hyperactivity disorder. This study used data from a representative sample of 8- to 15-year-old children collected as part of the NHANES conducted by the CDC.[118]

One study based in Ecuador has examined the relationship of OP exposure on neurodevelopment in school-aged children.[119] Prenatal exposure (based

on mother occupational history questionnaire) was associated with a decrease on the Stanford-Binet copying test among the study subjects at 7 years of age. Their concurrent exposure (on the basis of OP urinary metabolites) was associated with an increase in simple reaction time.

The toxicological mechanisms that underlie the adverse neurodevelopmental observations are also under investigation. Interestingly, noncholinergic mechanisms are being deciphered in animal models and in vitro studies, distinct from the well-described mechanism of acute OP toxicity (cholinesterase inhibition) and occurring at doses much lower than required to inhibit cholinesterase.[120]

Well-designed recent cohort studies and previous work including animal models suggest that OP exposures that are being experienced by US children may have adverse neurodevelopmental consequences. The plasticity of these effects and clinical implications are as yet unclear, although continued assessments as these cohorts age and enter school age are planned and may add clarity. The potential modification of these effects on the basis of genetic factors, specifically metabolic enzymes involved in pesticide detoxification pathways, are also being explored in these cohorts. For example, preliminary analyses indicate that children with a particular variant of the paraoxonase I gene, which is associated with lower levels of this OP-metabolizing enzyme, may be at higher risk of health consequences from OP exposure.[121,122]

Although DDT has not been used since the early 1970s, its persistence in the environment and fat solubility results in ongoing detection of the parent compound and breakdown product (DDE) in contemporary US populations.[19] The potential adverse neurodevelopmental consequences of prenatal DDT (2 studies) and DDE (several studies) was studied in one of the recent cohorts described previously in this report, which was a predominately Mexican American farmworker population. In this cohort, maternal serum DDT levels were negatively associated with mental development and psychomotor development at 12 and 24 months.[123] Maternal serum DDE was associated with reduced psychomotor development at 6 months and mental development at 24 months. A review of the overall evidence base reveals that studies of in utero DDE exposure and neurodevelopment are mixed, with at least 2 studies showing decrements in psychomotor function. Both of the 2 studies that have evaluated effects of DDT exposure observed cognitive deficits.[103]

In summary, the existing and recently emerging evidence base suggests that organochlorine and OP exposure in early life, particularly prenatally, may have adverse consequences on child neurodevelopment.

Physical Developmental Effects

In addition to neurodevelopmental toxicity, there is also considerable concern of physical developmental toxicity to the embryo and fetus from pesticide exposure. These concerns arise from multiple epidemiological studies that have investigated their relationship to adverse pregnancy outcomes including intrauterine growth retardation, preterm birth, fetal death, and congenital anomalies. The available studies are heterogeneous in design, are conflicting in results, and often have an insufficient exposure assessment. Nonetheless, pesticides remain one of the most common environmental exposures of concern cited in relation to adverse pregnancy outcomes and have been the focus of recent reviews on the topic, which include weight of the evidence evaluations.[124–126]

Among studies that are able to address specific types of pesticide exposures, there are more data focused on the organochlorine and OP insecticides or phenoxy or triazine herbicides. These represent the currently or historically (eg, organochlorine) most heavily used pesticides. This review summarizes the highlights of the existing evidence base with a focus on studies that incorporate direct measures of exposure for individual study subjects.

Fetal Death and Birth Defects

A California-based case-control study found an increased risk of fetal death attributable to congenital anomalies when OP application occurred in the residential area of the mother during weeks 3 through 8 of pregnancy—consistent with organogenesis.[127] One other study found an elevated risk of spontaneous abortion associated with chlorophenoxy herbicides. However, as with some studies of birth defects discussed previously, this study also relied on self-report and less reliable means of exposure assessment.[128] Results are not consistent, because other studies have not found association of parental exposure to OPs with spontaneous abortion or stillbirth.[129–131]

Birth defects will be discussed first, followed by other adverse birth outcomes. The more common birth defects include orofacial clefts, limb defects, and neural tube defects, which are generally the defects studied in relationship to pesticide exposures. Although several studies have found associations of maternal or paternal exposures with a wide variety of birth defect categories, all of the studies used indirect measures of exposure and most were ecological study designs, making interpretation of the adverse birth outcome evidence base inadequate and unreliable.[125]

A 1995 review article discussed the available evidence for associations between birth defects and potential

pesticide exposure.[132] Five studies were included that assessed various birth defects (central nervous system, oral cleft, limb defects) compared with maternal agricultural occupation. Four of those 5 reported an elevated RR or an OR ranging from 1.6 to 5.0; however, only 2 were statistically significant.[133–137] Of note, in these studies, there was not an assessment to any single pesticide; rather, the "exposure" was maternal occupation.

Six additional studies from this period evaluated maternal pesticide exposure at work and the development of birth defects. Of the 5 studies with an elevated OR or RR, ranging from 1.3 to 7.5,[138–142] 3 were statistically significant. Unfortunately, some of these studies included small numbers of cases, and others were likely to have significant exposure misclassification. The conclusion of this review was that there are some indications of elevated risk but no clearly convincing evidence.[143]

Two studies from Minnesota have reported a relationship between physical defects in children and paternal occupation of pesticide applicator. The first study compared data from a birth registry between 1989 and 1992. A geographic section of Minnesota that had the highest agriculture activity and highest frequency of use of chlorophenoxy herbicides and fungicides was also found to have the highest rate of birth defects (30.0/1000). By comparison, the general population in this same region had a birth defect rate of 26.9/1000. Interestingly, there was a seasonal effect, with the highest frequency occurring in infants who were conceived in the spring, the same time as most herbicide and some fungicide application (OR, 1.36; CI, 1.10–1.69).[144] The second study is a cross-sectional study that used a survey of licensed applicators and subsequently more in-depth interviews of either/both the applicator and female partners of licensed applicators when possible. The study eventually included live births fathered by 536 applicators. The birth defect rate in this study was 31.3/1000, which is statistically significantly higher than what the previous study found for the general population. Again, there was a significant difference in season of conception (7.6% in spring versus 3.7% in other seasons).[145]

Studies of birth defects often include all types within the analysis because of insufficient numbers of individual defects to allow adequate power of statistical analyses. A meta-analysis used 19 studies that had sufficient data to be included to estimate the effects of pesticides on orofacial clefting. Maternal occupational exposure to pesticides was associated with orofacial clefts (OR, 1.37; 95% CI, 1.04–1.81). There was a weaker association for paternal occupation (OR, 1.16; 95% CI, 0.94–1.44).[146] Studies on 3 other birth defects—cryptorchidism, hypospadias, and polythelia—will be discussed in the section on endocrine effects.

In summary, a small risk elevation is noted for birth defects and pesticide exposure, but the findings are not robust, and the data specific to pesticide subtypes are not adequate.

Adverse Birth Outcomes (Low Birth Weight, Decreased Gestational Age)

DDT (and its major metabolite DDE) is the organochlorine that has been most extensively examined in relation to birth defects, fetal death, and fetal growth, with mixed findings. Fetal exposures, as determined by maternal serum or umbilical cord blood levels, have been associated with preterm birth, decreased birth weight, and intrauterine growth retardation.[147–151] However, not all studies reported significant associations between exposure with infant birth weight or preterm birth, including a relatively recent study of Mexican American farmworking women in the United States with higher exposures in comparison with a similar group of a national sample of nonfarmworking Mexican American women.[142,152] In the largest cohort study to date (a US cohort of births between 1959 and 1966), DDE concentrations in maternal serum during pregnancy demonstrated a dose–response relationship to risk of preterm delivery and delivering small for gestational age (SGA) infants.[147]

Exposure to pesticides is associated with risk of decreased birth weight. In a study conducted before recent regulatory actions that reduced their residential use, exposure to the OPs chlorpyrifos and diazinon were associated with decreased birth weight in a New York City cohort.[110] In another New York City cohort, birth weight was reduced among mothers with higher OP exposure levels in pregnancy, but only among those with a genetic polymorphism of an OP detoxification enzyme (paraoxonase 1 or PON1).[150] In a similar longitudinal pregnancy cohort conducted among Latina farmworkers in agricultural California, no association of maternal pregnancy exposure to OPs and birth weight was determined, but a reduction in gestational age was associated.[153]

An ecological study determined that women in a rural region of Iowa with increased levels of triazine, metolachlor, and cyanazine herbicides in the drinking water had an elevated risk of delivering an infant with intrauterine growth retardation compared with women in other parts of the state.[154] A study based in France reported that atrazine levels in municipal drinking water throughout pregnancy were not associated with increased risk of delivering an SGA infant but that the

risk of delivering an SGA infant increased when the third trimester occurred in whole or in part during the period of May through September, when atrazine levels typically peak.[155]

Summary: Physical Developmental Defects

In summary, the true extent and nature of pesticide exposure on adverse fetal growth and birth outcomes is unknown despite suggestive epidemiological studies that link some of the most widely used pesticides to reduced intrauterine growth, fetal death, preterm birth, and congenital anomalies. Very little is known about many pesticide types in current use, including synthetic pyrethroids and carbamate insecticides, rodenticides, and fungicides. Studies that examine the timing and extent of exposure to pesticides and exposure to pesticide mixtures with validated exposure assessment techniques including biological markers are needed. The potential for differential vulnerabilities because of genetic polymorphisms that influence the toxicological properties of these exposures must also be explored.

ENDOCRINE EFFECTS

An emerging concern, although less well studied in humans, is the potential effects that some chemicals including pesticides may have on the endocrine system. Some of the most notable pesticides thought to have such effects are the organochlorine pesticides, such as DDT, endosulfan, methoxychlor, chlordecone, chlordane, and dieldrin. Other herbicides (atrazine, 2,4-D, and glyphosate) and fungicides (vinclozolin) also have some endocrine activity.[156–159] The associations are very complex and are primarily based on in vitro and animal studies. Estrogen-mimicking properties tend to be the most commonly reported, although effects on androgen and thyroid hormones, among others, are also reported. Feminization has been noted in alligators found in lakes highly contaminated by organochlorine pesticides.[160] Hayes et al[161] have studied the effects of atrazine on amphibians and have noted a 10-fold decrease in testosterone from exposure to 25 ppb of atrazine in mature male frogs. The mechanism of the latter appears to be activation of the enzyme aromatase, which promotes conversion of testosterone to estrogen.[162]

The human epidemiology literature is limited on endocrine effects from pesticides. One report from Macedonia noted some degree of early pubertal findings, primarily premature thelarche, which was hypothesized to be related to organochlorine pesticide exposure.[163] A study in 2000 with 48 patients, 18 of which had cryptorchidism, first raised the hypothesis about an association with organochlorine pesticides. An association between cryptorchidism and organochlorine pesticide levels has been hypothesized.[164] Since then, additional case-control studies have been conducted to examine the effects of organochlorines on endocrine-related birth outcomes, cryptorchidism, hypospadias, and/or polythelia. Two focused on fetal exposures from maternal levels of DDE alone and development of cryptorchidism and hypospadias.[165,166] Bhatia et al[165] calculated an OR of 1.34 (95% CI, 0.51–3.48) for the association of cryptorchidism and DDE and 1.18 (95% CI, 0.46–3.02) for the association of hypospadias and DDE. Longnecker et al[166] estimated an OR of 1.3 (95% CI, 0.6–2.4) for the association between DDE and cryptorchidism and an OR of 1.2 (95% CI, 0.6–2.4) the association between DDE and hypospadias. The modest association is felt to be inconclusive with the imprecision in risk estimates and suggests that a larger sample size may be needed. A third case-control study found inconclusive results on the effect of heptachlor and β-hexachlorocyclohexane levels in pregnant women on cryptorchidism. For heptachlor, the OR was 1.2 (95% CI, 0.6–2.6), and for β-hexachlorocyclohexane, the OR was 1.6 (95% CI, 0.7–3.6). The sample size in this study was 219 cases, compared with 564 controls.[167]

Two nested case-control studies have examined the possibility that multiple organochlorine compounds will have a cumulative effect on the development of urogenital abnormalities in boys.[168,169] Fernandez et al[168] reported that total xenoestrogens as well as detectable pesticide levels were associated with cryptorchidism and/or hypospadias. They found elevated ORs in the range of 2.19 for endosulfan to 3.38 for lindane. All 95% CIs were noted to be statistically significant. The study in Finland and Denmark reported a significant relationship between chlordane and cryptorchidism but no other relationships between 7 other individual organochlorines. However, combined analysis of the 8 persistent pesticides did demonstrate a statistically significant increase in cryptorchidism in exposed boys.[169]

Testing chemicals is an important and necessary step for the EPA to determine potential long-term risks from pesticide during the registration or re-registration process. There has been progress in the development of appropriate biomarkers to evaluate chemicals for the presence of endocrine-disruption qualities. The ability to measure DDE and dioxins from human milk has been developed.[170] More recently, a biomarker for xenoestrogen mixtures was developed in Spain.[171]

In summary, there is compelling basic science evidence for endocrine-mimicking effects of several pesticide chemicals that is sound and scientifically plausible. Human data

are slowly emerging but not yet conclusive.[172]

Asthma

Given the widespread use of pesticides and the high morbidity of asthma in children, questions have been raised regarding pesticides as triggers as well as risk factors for incident disease. Concern is raised by a mounting adult occupational literature associating pesticides with asthma or other measures of respiratory health. In addition, preliminary toxicological data provide mechanisms that link pesticides and asthma. An important limitation of most epidemiological studies to date is the lack of exposure specificity regarding pesticide chemicals or chemical classes. In addition, studies regarding children are few.

There is indirect evidence that pesticides skew the immune response toward the T helper 2 (Th2) phenotype associated with atopic disease. The National Institutes of Health/EPA-sponsored rural birth cohort described above regarding evaluation of neurodevelopmental effects has also observed that maternal agricultural work was associated with a 26% increase in proportion of Th2 cells in their 24-month-old infants' blood samples.[173] The percentage of Th2 cells was associated with both physician-diagnosed asthma and maternal report of wheeze in these infants. This population of largely Mexican American farmworkers was selected for study on the basis of the relatively high use of OP pesticides in this agricultural area.

Animal-based toxicological mechanistic models include OP-induced airway hyperreactivity via alteration in muscarinic receptor function in airway smooth muscle and oxidative stress induced by OP-related lipid peroxidation.[174–177]

The few epidemiological data on pesticides and respiratory health in children have mixed results. In a cohort of rural Iowan children, any pesticide use indoors or any outdoor use in the previous year was not significantly associated with asthma symptoms and prevalence.[178] Contrarily, a cross-sectional analysis of Lebanese children identified increased risk of chronic respiratory symptoms, including wheeze, among those with any pesticide exposure in the home, exposure related to parent's occupation, and use outside the home. The highest risk was observed for children whose parents had occupational exposure to pesticides (OR, 4.61; 95% CI, 2.06–10.29).[179] However, given this study's cross-sectional design, it is not possible to discern whether the pesticide exposure preceded the diagnosis of asthma.

Among exposures in the first year of life explored in a nested case-control study of the Southern California Children's Health Study, both herbicides and pesticides/insecticides had a strong association with asthma diagnosis before 5 years of age (OR, 4.58 [95% CI, 1.36–15.43] and OR, 2.39 [95% CI, 1.17–4.89], respectively).[180]

More published data are available regarding adult farmers and adult rural residents. These studies more consistently support a link between pesticides and respiratory symptoms or chronic respiratory disease, such as asthma.[181,182] For example, use of multiple individual pesticides was evaluated in relation to self-reported episodes of wheeze in the previous year in a large cohort of commercial pesticide applicators (adults) and farmers enrolled in the Agricultural Health Study.[182] Among the pesticides classes, several OPs showed associations with wheeze, including several that demonstrated a dose–response trend. Chlorpyrifos, malathion, and parathion were positively associated with wheeze among the farmers; for the commercial applicators, the OPs chlorpyrifos, dichlorvos, and phorate were positively associated with wheeze. Among commercial applicators, the strongest OR was for applying chlorpyrifos on more than 40 days per year (OR, 2.40; 95% CI, 1.24–4.65). Elevated risk for wheeze related to herbicide use was almost exclusively associated with chlorimuron-ethyl (urea-derivative class).

Similar studies addressing the respiratory health implications for children for specific pesticide chemical types or groups are rare. However, for DDT, there is some emerging evidence for a link between metabolites of DDT and asthma risk.[183,184] In a prospective cohort study of children in Spain, wheezing at 4 years of age increased with increasing levels of DDE at birth. The adjusted RR for the children with exposure in the highest quartile was 2.63 (95% CI, 1.19–4.69). The use of physician-diagnosed asthma (occurring in 1.9% of children) instead of wheezing as the outcome variable also resulted in a positive association, although it was not statistically significant.[184]

In summary, the available data regarding chronic exposure to pesticides and children's respiratory health remain limited. Studies that incorporate pesticide-specific exposure assessment and markers of biological mechanisms and consider the influence of timing of exposure across the life span are needed.

THE PESTICIDE LABEL

Pesticides for sale or use in the United States must be registered with the EPA, and this includes approval of the product label, which contains the EPA registration number. The pesticide label contains several types of information that may be important in understanding and preventing acute health consequences associated with their use.[185]

The product label identifies the active ingredient and provides the manufacturer's

contact information. The label does not specify the particular class of pesticide for the active ingredient, which may make it difficult for a physician to identify potential toxic effects. Information about "other" or "inert" ingredients, which may account for up to 99% of the product, is not required to be disclosed on the label. These constituents include chemicals with known toxicity. The physician treating a patient may request this from the manufacturer; however, delay in information may compromise optimal clinical care. The local or regional poison control center plays an important role as a resource for any suspected pesticide poisoning. The EPA is currently considering rulemaking changes that would expand the disclosure of information on inert ingredients. One of the options under consideration includes labeling 100% of the ingredients.[186]

The "directions for use" section on the label explains when, how, and where the pesticide may be applied. The label is considered the law; therefore, any use of the product in a manner inconsistent with the label is a violation of the Federal Insecticide, Fungicide, and Rodenticide Act (Pub L No. 80-104).[187] Information on recommended storage of the product and disposal of the container is also printed on the label.

The label will contain a signal word and symbol to identify acute toxicity potential: "danger" along with the word poison and the skull and crossbones symbol signifies high acute toxicity; "warning" signifies moderate acute toxicity; and "caution" represents slight acute toxicity. There is a section for precautionary statements regarding the potential hazards to people or pets and the actions that can be taken to reduce these hazards, such as wearing gloves or other protective equipment. Basic first aid advice for responding to dermal, inhalational, and/or oral exposure is provided. Some labels contain a "note for physicians" that includes specific medical information. The label does not provide any information or warnings about the potential for chronic toxicity arising from normal use or misuse of the pesticide. An example of an interactive pesticide label can be found at the EPA Web site.[188] It includes "pop-up" features that define each of the components on the pesticide label.

STATE OF PESTICIDE KNOWLEDGE AMONG PEDIATRICIANS

Self-reported medical education and self-efficacy suggests pediatricians are not well prepared to identify pesticide exposure and illness, including taking a relevant environmental history or discussing pesticide risks with their patients.[189–191] Even in agricultural areas of the Pacific Northwest, where pesticide use is heavy, a survey of health care providers who serve high volumes of agricultural farmworkers and their families found that 61% did not feel comfortable responding to patient/client questions regarding pesticides on the basis of their training, background, and experience.[75] Among academic pediatricians with an interest in pediatric environmental health, pesticides were among the topics they felt least prepared to teach to their trainees.[192] Given the widespread use of pesticides and concerns for child health, opportunities to increase pesticide competency in pediatric medical education are likely to prevent missed diagnoses and reduce exposure because of improved anticipatory guidance.

Clinicians must have a high index of suspicion to identify pesticide poisoning. Identification and treatment of acute pesticide poisoning requires familiarity with the toxic mechanisms and related signs and symptoms of the pesticide classes. For example, when evaluating a patient with status epilepticus or mental status changes, certain insecticides belong in the differential among the numerous and more common etiologies. Eliciting an environmental history will help decipher the relative importance of pesticides in further clinical decision-making. The environmental history is a general tool for addressing potentially hazardous environmental exposures and is discussed in detail in the Pediatric Environmental Health manual from the AAP.[193]

EFFORTS TO REDUCE PESTICIDE EXPOSURE

Dietary Considerations

Dietary modifications can help reduce pesticide exposure. As mentioned previously, consuming organic produce has shown a reduced amount of urinary pesticide levels in comparison with a conventional diet.[22] Because many food-based pesticide residues occur on the surface of food crops, other practical approaches may be used to reduce exposures by washing produce, peeling off outer layers of leafy vegetables, and removing peels from fruits and vegetables. Trimming fat from meat and fat and skin from poultry and fish may reduce residues of persistent pesticides, such as the organochlorines, that concentrate in animal fat.

Efforts to address and reduce chronic pesticide exposure via the food supply in children have included regulatory approaches that consider the unique vulnerability of the developing child in policy decision-making. For example, the 1996 Food Quality Protection Act (Pub L No. 104-170, Section 405) required that the EPA use an additional 10-fold margin of safety regarding limits of pesticide residues on food (unless there are data that show a less stringent residue level is safe for

prenatal and postnatal development; for description, see http://www.epa.gov/opp00001/factsheets/riskassess.htm).

Integrated Pest Management

In addition to food residues, use of pesticides in and around the home and other settings where children spend time (child care, school, and playgrounds and sports fields) is an important influence on the chronic and cumulative exposure to pesticides among US children. Most of the pest problems that occur indoors as well as control of lawn and garden pests can be addressed with least toxic approaches, including integrated pest management (IPM) techniques. IPM focuses on nontoxic and least toxic control methods to address pest problems have been promoted and adopted for residential, school, and agricultural settings (fact sheets available at http://www.epa.gov/opp00001/factsheets/ipm.htm).

"Integrated" refers to employment of complementary strategies of pest control, which may include mechanical devices; physical devices; genetic, biological, and cultural management; and chemical management. For example, to control cockroaches, a family could be counseled to keep garbage and trash in containers with well-fitted lids, eliminate plumbing leaks or other sources of moisture, store food in insect-proof containers, vacuum cracks and crevices, clean up spills immediately, and use the least-toxic insecticides, such as boric acid, in cracks and crevices or bait stations. The goal is to target the pest and limit the effect on other organisms and the environment. Although developed with a focus on agricultural insect pests, IPM programs and knowledge have extended to address weeds and pest control in residential settings and schools, commercial structures, lawn and turf, and community gardens.

Within agriculture, IPM has been recognized and promoted for decades; however, inadequate leadership, coordination, and management of US Department of Agriculture IPM programs were identified as impediments to adequate progress in a 2001 report.[194] The report provided the basis for an ongoing national roadmap effort to improve ongoing development of increased IPM in agriculture.

To protect children, IPM in schools has been recommended by the US Department of Agriculture, EPA, American Public Health Association, and National Parent Teacher Association. Many states and local municipalities have adopted programs and resources to encourage IPM in public places, in addition to homes and schools (see Table 3). IPM strategies seek to minimize insecticide use by applying strategies such as cleaning up food and water, sealing cracks and crevices, and using pesticides that are contained in baits or traps, which are far less likely to pose a health concern compared with any type of broadcast spray application. Avoiding combination products with pesticides and fertilizers (ie, "weed and feed" preparations) is advised for lawn maintenance, because these tend to result in overapplication of pesticides. Hand weeding is always a reasonable alternative to herbicides. However, if an herbicide is to be used, some (such as glyphosate) have better acute human toxicity profiles than others (such as 2,4-D). Even so, glyphosate is not without its risks. Most cases of moderate to severe toxicity have occurred after intentional (suicidal) ingestion.[195] Using safe storage practices (in a locked cabinet or building) and not reusing pesticide containers are important components toward the prevention of acute poisonings after unintentional ingestion by small children. Reliable resources for useful information on pest-control alternatives and safe use of pesticides are available from the EPA and University of California-Davis (Table 3).

Spraying in the Community: Right to Know

Although there is no federal mandate for notification of pesticide use in communities, many states, locales, or schools have implemented requirements for posting warning signs or developing registries to alert individuals of planned pesticide application (see Table 3). These are designed to allow the public to make decisions to avoid exposures during application or soon after from residues. Other local policies that have been developed include restricting spray zones that create buffers from schools or other areas or restrict specific types of pesticide products in schools. Pediatricians can play a role in the promotion of development of model programs and practices in the communities and schools of their patients. For example, in some communities, pediatricians have participated in local organizations that have successfully advocated for no pesticide application in schools.

SUMMARY

Pesticides are a complex group of chemicals with a wide range of acute and chronic toxicity. Poison control centers report lower rates of more severe poisonings but continue to report similar total numbers of acute exposures among children. There is a growing body of literature that suggests that pesticides may induce chronic health complications in children, including neurodevelopmental or behavioral problems, birth defects, asthma, and cancer. Pediatricians are a trusted source of information for families and communities, although current training focused on pesticide toxicity and environmental health, in

TABLE 3 Pesticide and Child Health Resources for the Pediatrician

Management of Acute Pesticide Poisoning		
Recognition and Management of Pesticide Poisonings		Print: fifth (1999) is available in Spanish, English (6th edition available 2013) http://www.epa.gov/pesticides/safety/healthcare/handbook/handbook.htm
Regional Poison Control Centers		1-800-222-1222
Chronic Exposure Information/Specialty Consultation		
The National Pesticide Medical Monitoring Program (NPMMP)	Cooperative agreement between Oregon State University and the EPA NPMMP provides informational assistance by e-mail in the assessment of human exposure to pesticides	npmmp@oregonstate.edu or by fax at 541-737-9047
Pediatric Environmental Health Specialty Units (PEHSUs)	Coordinated by the Association of Occupational and Environmental Clinics to provide regional academically based free consultation for health care providers	http://www.aoec.org/PEHSU.htm Toll-free telephone number 888-347-AOEC (2632)
Resources for Safer Approaches to Pest Control		
EPA	Consumer information documents	http://www.epa.gov/oppfead1/Publications/Cit_Guide/citguide.pdf
Citizens Guide to Pest Control and Pesticide Safety	• Household pest control • Alternatives to chemical pesticides • How to choose pesticides • How to use, store, and dispose of them safely • How to prevent pesticide poisoning • How to choose a pest-control company	
Controlling pests	Recommended safest approaches and examples of programs	http://www.epa.gov/pesticides/controlling/index.htm
The University of California Integrative Pest Management Program	Information on IPM approaches for common home and garden pests	http://www.ipm.ucdavis.edu
Other Resources		
National research programs addressing children's health and pesticides	NIEHS/EPA Centers for Children's Environmental Health & Disease Prevention Research The National Children's Study	www.niehs.nih.gov/research/supported/centers/prevention www.nationalchildrensstudy.gov/Pages/default.aspx
EPA	Pesticide product labels	www.epa.gov/pesticides/regulating/labels/product-labels.htm#projects
The National Library of Medicine "Tox Town"	Section on pesticides that includes a comprehensive and well-organized list of Web link resources on pesticides	http://toxtown.nlm.nih.gov/text_version/chemicals.php?id=23

NIEHS, National Institute of Environmental Health Sciences.

general, is limited. Pediatricians should be familiar with the common pesticide types, signs and symptoms of acute toxicity, and chronic health implications. Efforts should be made to limit children's exposure as much as possible and to ensure that products released to the marketplace have been appropriately tested for safety to protect fetuses, infants, and children from adverse effects.

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Pesticide Exposure in Children

James R. Roberts, Catherine J. Karr and COUNCIL ON ENVIRONMENTAL HEALTH

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Safety & Prevention

Pesticides, Herbicides and Children

Pesticides and herbicides are used in a variety of settings, including homes, schools, parks, lawns, gardens, and farms. While they may kill insects, rodents, and weeds, some are toxic to people when consumed in food and water.

More research is needed to determine the short- and long-term effects of pesticides and herbicides on humans. Although some studies have found connections between some childhood cancers and an exposure to pesticides, other studies have not reached the same conclusions. Many pesticides disrupt the nervous system of insects, and research has shown that they have the potential to damage the neurological system of children.

Prevention

Try to limit your child's unnecessary exposure to pesticides or herbicides. To reduce such exposure:

- Minimize using foods in which chemical pesticides or herbicides were used by farmers.
- Wash all fruits and vegetables with water before your child consumes them.
- For your own lawn and garden, use nonchemical pest control methods whenever possible. If you keep bottles of pesticides in your home or garage, make sure they're out of the reach of children to avoid any accidental poisoning.
- Avoid routinely spraying homes or schools to prevent insect infestations.

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topic landing page

Leading pediatricians' group recommends parents reduce pesticides at home

By

Published November 26, 2012 | MyHealthNewsDaily

Children are particularly vulnerable to the potentially toxic effects of pesticides, and steps should be taken to limit kids' exposure to these chemicals as much as possible, pediatricians say.

Kids are exposed to pesticides everyday through the air, dust and soil, and their food, according to the American Academy of Pediatrics, which published a report today (Nov. 26) about the topic.

Exposure to high doses of pesticides, which can occur if a child unintentionally ingests a product such as weed or insect killer, is known to cause serious problems, including vomiting, breathing problems, seizures and even death. [See Is Pesticide Spraying Safe?]

But there's also emerging evidence that long-term exposure to lower doses of pesticides can harm children, the AAP says. Studies have linked exposure to pesticides while in the womb with an increased risk of cancers in children, including brain cancer and leukemia. Parents in these studies, were exposed to pesticides either at their work, or while using pesticides around the house.

Other studies have linked prenatal exposure chemicals called organophosphates with lower IQ scores in children.

Parents can reduce pesticide exposure by aiming to control pests in homes and gardens in the least toxic ways, the AAP says. For instance, to control cockroaches, families can keep garbage in containers with lids, eliminate plumbing leaks and use the least toxic insecticides, such as boric acid, in cracks and crevices, the report says.

Families should avoid using lawn products that combine pesticides and fertilizers because use of these products tends to result in over-application of pesticides, the AAP said.

Eating organic foods may also help lower pesticide exposure. In one small study, children had lower levels of pesticides in their urine when they switched from a conventional diet to one with mostly organic foods. However, in a report published last month, the AAP noted it's not clear whether the lower levels of pesticides found in organic foods would make a difference in terms of health over a lifetime.

Washing produce and removing peels may also reduce pesticide exposure, the AAP said.

Pediatricians should ask parents about pesticide use in the home, and recommend minimal-risk products and safe storage practices (such as storing pesticides in a locked cabinet or building), the AAP said.

Government regulators should take steps to increase economic incentives for farmers who use less toxic pesticides, and support research to better understand the health risks of pesticides, the AAP said.

Top 5 Ways to Reduce Toxins in Homes
5 Experts Answer: What Are the Most Dangerous Items in a Home?

Schlein, Paul B

From: National Pest Management Association <npma@pestworld.org>
Sent: Thursday, November 29, 2012 4:17 PM
To: AF-Pesticides Internet
Subject: NPMA Response to AAP's "Pesticide Exposure in Children"

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Response to "Pesticide Exposure in Children"
Published by the American Academy of Pediatrics
PEDIATRICS Volume 130, Number 6, December 2012
Overview and Speaking Points
Prepared for the National Pest Management Association
By the Professional Pest Management Alliance

November 28, 2012

Background

On November 26, 2012, the American Academy of Pediatrics (AAP) posted a policy statement, **Pesticide Exposure in Children**, asserting that pesticides have acute and chronic health affects on children. The online article, will also run in the December 2012 print issue of its PEDIATRICS® journal.

The AAP states that children are exposed to pesticides (a collective term for chemicals intended to kill unwanted insects, plants, molds and rodents) every day and have unique susceptibilities to their potential toxicity. According to the AAP, this exposure can have both immediate and long-term health implications as evidenced by "associations between early life exposure and pediatric cancers, decreased cognitive function and behavior problems." The group also claims that these epidemiological findings are supported by related animal toxicology studies, which provide supportive biological

plausibility.

Highlights:

- The statement touches on diet, agricultural settings (spray drift, teen agricultural workers), unintentional ingestions and inhalational exposure.
- It references that "there is no current reliable way to determine the incidence of pesticide exposure and illness in US children," and calls for improved physician education, biomarkers and diagnostic testing methods to better track pesticide exposure and acute illnesses.
- Concedes that EPA labels contain information for understanding and preventing acute health consequences, but does not address chronic toxicity and labels are only in English. Labels also don't specify the pesticide class or list the "other"/"inert" ingredients that may have toxicity and can account for up to 99% of the product.
- Mentions significant use of illegal pesticides and off label use, underscoring the importance of education, monitoring and enforcement.
- States that IPM is "an established but undersupported approach to pest control."
- Urges pediatricians to become more knowledgeable in pesticide identification, counseling and management; Governmental actions needed to improve pesticide safety.

The policy statement has been summarized online with articles on FOXNews.com Yahoo! News, MyHealthNewsDaily.com and several more. We are closely monitoring this story to see whether it receives further attention by national and other local media outlets and we recognize it may generate inquiries from customers and/or employees.

NPMA Statement

"The professional pest management industry supports and has long advocated for Integrated Pest Management (IPM) approaches to control household pests. IPM is more difficult and requires a thorough knowledge of pest biology and behavior. Treatment options in IPM can vary from proactive measures like sealing cracks and removing food and water sources to reactive measures, such as utilizing pest control products, when necessary. It is important homeowners turn to pest professionals to eliminate a pest problem, because if pesticide treatment is necessary, professionals will ensure products are applied correctly, with minimal exposure, and in

strict adherence to product labels.

Products used by the industry are registered by the EPA after it has reviewed extensive heath and safety data and made a determination that the product meets or exceeds the federal government's rigorous health standards. Consumers should feel confident that the application of these products is safe to both family and home when applied correctly by qualified and licensed pest professionals as directed on the product label.

The professional pest management industry's primary concern is for the health, safety and protection of its customers, the American public and especially our children. Common household pests pose significant health risks including the transmission of bacteria and disease, and can exacerbate respiratory issues such as allergies and asthma, particularly in small children. As such, pest control should not be taken lightly, but rather it should be handled in partnership with a licensed pest professional to properly identify, assess and treat the infestation. Consumers should discuss Integrated Pest Management solutions with their pest professional."

Speaking Points

- IPM is a process involving common sense and sound solutions for controlling pests by helping to eliminate sources of food, water and shelter. Pest professionals work with customers every day to find the best strategy for dealing with pest problems - which often times isn't the simplest solution.
    - Trained and certified pest professionals never employ a "one-size-fits-all" method when faced with an infestation, but rather utilize a three-part practice: inspection, identification and treatment, and always in partnership with the customer.
- The professional pest control industry and the products used by pest professionals are highly regulated by the Environmental Protection Agency (EPA) as well as state agencies. The EPA regulates pesticides under the Federal Insecticide, Fungicide, and Rodenticide Act (FIFRA), the Federal Food, Drug, and Cosmetic Act (FFDCA) and the 1996 Food Quality Protection Act (FQPA).
    - Each of these acts has put in place requirements and protections to ensure there are no

unreasonable adverse effects on human health or environment as well as providing special protections for infants and children as they are recognized as a sensitive group.

- The professional pest management industry can confidently say that professional products used in the treatment of residential pest infestations are rigorously reviewed and registered by the EPA to be used by certified applicators for pest management. The National Pest Management Association works closely with the EPA to ensure that all products used in pest management are rigorously reviewed, re-registered and provided with accurate and comprehensive labeling for use.

- Additionally, licensed and trained pest professionals must keep abreast of any regulatory changes related to pesticides and their use in residential and commercial environments and must stop use of any products that are banned by the EPA.

- Professional pest management plays a vital role in protecting public health and property. Household pests pose serious risks to our health by spreading bacteria and disease, contaminating food and causing respiratory problems such as asthma. Some household pests can also severely damage property. Wood destroying insects eat away at a home's structural stability and rodents gnaw away at drywall and electrical wires, posing a serious risk for fires.

Additional background regarding FIFRA, FFDCA and FQPA

- Under the Federal Insecticide, Fungicide, and Rodenticide Act (FIFRA), EPA registers pesticides for use in the United States and prescribes labeling and other regulatory requirements to prevent unreasonable adverse effects on human health or the environment.

- Under the Federal Food, Drug, and Cosmetic Act (FFDCA), EPA establishes tolerances (maximum legally permissible levels) for pesticide residues in food.

- The 1996 Food Quality Protection Act (FQPA) established a more consistent, protective regulatory scheme, grounded in sound science by:

- mandating a single, health-based standard for all pesticides in all foods;
- providing special protections for infants and children;
- expediting approval of safer pesticides;
- creating incentives for the development and maintenance of effective products; and
- requiring periodic re-evaluation of pesticide registrations and tolerances to ensure that the scientific data supporting pesticide registrations will remain up to date in the future.

- Additionally, FQPA dramatically changed the safety standards EPA uses in evaluating potential pesticide risks, especially to infants and children. Since FQPA was enacted, effective protection of children, already a priority, received additional emphasis through the addition of an **extra tenfold Children's Safety Factor**. This additional factor is now standard in dietary risk assessments, unless reliable data support a different factor.

  - Other protective measures require EPA to assess the aggregate impact of exposure to pesticides in the food we eat and water we drink, along with exposures resulting from residential pesticide uses and other non-occupational sources of exposure. Finally, FQPA mandated that EPA's safety assessments consider the cumulative effects on health from exposures to multiple different pesticides that cause the same biological effects in humans.

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Organic Foods: Health and Environmental Advantages and Disadvantages

Joel Forman, Janet Silverstein, COMMITTEE ON NUTRITION and COUNCIL ON ENVIRONMENTAL HEALTH

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American Academy of Pediatrics
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CLINICAL REPORT

Organic Foods: Health and Environmental Advantages and Disadvantages

abstract

FREE

The US market for organic foods has grown from $3.5 billion in 1996 to $28.6 billion in 2010, according to the Organic Trade Association. Organic products are now sold in specialty stores and conventional supermarkets. Organic products contain numerous marketing claims and terms, only some of which are standardized and regulated.

In terms of health advantages, organic diets have been convincingly demonstrated to expose consumers to fewer pesticides associated with human disease. Organic farming has been demonstrated to have less environmental impact than conventional approaches. However, current evidence does not support any meaningful nutritional benefits or deficits from eating organic compared with conventionally grown foods, and there are no well-powered human studies that directly demonstrate health benefits or disease protection as a result of consuming an organic diet. Studies also have not demonstrated any detrimental or disease-promoting effects from an organic diet. Although organic foods regularly command a significant price premium, well-designed farming studies demonstrate that costs can be competitive and yields comparable to those of conventional farming techniques. Pediatricians should incorporate this evidence when discussing the health and environmental impact of organic foods and organic farming while continuing to encourage all patients and their families to attain optimal nutrition and dietary variety consistent with the US Department of Agriculture's MyPlate recommendations.

This clinical report reviews the health and environmental issues related to organic food production and consumption. It defines the term "organic," reviews organic food-labeling standards, describes organic and conventional farming practices, and explores the cost and environmental implications of organic production techniques. It examines the evidence available on nutritional quality and production contaminants in conventionally produced and organic foods. Finally, this report provides guidance for pediatricians to assist them in advising their patients regarding organic and conventionally produced food choices. *Pediatrics* 2012;130:e1406–e1415

Joel Forman, MD, Janet Silverstein, MD, COMMITTEE ON NUTRITION, and COUNCIL ON ENVIRONMENTAL HEALTH

KEY WORDS

organic food, produce, meat, dairy, growth hormone, antibiotic, farming, diet

ABBREVIATIONS

GH—growth hormone
NOP—National Organic Program
USDA—US Department of Agriculture

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DEFINITION AND REGULATION OF ORGANIC FOODS

Definition

Organic farming uses an approach to growing crops and raising livestock that avoids synthetic chemicals, hormones, antibiotic agents, genetic engineering, and irradiation. In the United States, the US Department of Agriculture (USDA) has implemented the National Organic Program (NOP)[1] in response to the Organic Foods Production Act of 1990.[2] The NOP set labeling standards that have been in effect since October 2002. NOP standards for organic food production include many specific requirements for both crops and livestock. To qualify as organic, crops must be produced on farms that have not used most synthetic pesticides, herbicides, and fertilizer for 3 years before harvest and have a sufficient buffer zone to decrease contamination from adjacent lands. Genetic engineering, ionizing radiation, and sewage sludge is prohibited. Soil fertility and nutrient content is managed primarily with cultivation practices, crop rotations, and cover crops supplemented with animal and crop waste fertilizers. Pests, weeds, and diseases are managed primarily by physical, mechanical, and biological controls instead of with synthetic pesticides and herbicides. Exceptions are allowed if substances are on a national approved list. Organic livestock must be reared without the routine use of antibiotic agents or growth hormones (GHs) and must be provided with access to the outdoors. If an animal is treated for disease with antibiotic agents, it cannot be sold as organic. Preventive health practices include vaccination and vitamin and mineral supplementation. The USDA certifies organic products according to these guidelines. Organic farmers must apply for certification, pass a test, and pay a fee. The NOP requires annual inspections to ensure ongoing compliance with these standards.

Labeling

Consumers are confronted with a wide range of food product marketing terms, some regulated and some not (Table 1). The labeling requirements of the NOP apply to raw, fresh products and processed products that contain organic agricultural ingredients. These labeling requirements are based on the percentage of organic ingredients in a product.[3] Products labeled "100% organic" must contain only organically produced ingredients and processing aids (excluding water and salt). Products labeled "organic" must consist of at least 95% organically processed ingredients (excluding water and salt); the remaining 5% of ingredients may be conventional or synthetic but must be on the USDA's approved list. Processed products that contain at least 70% organic ingredients can use the phrase "made with organic ingredients" and list up to 3 of the organic ingredients or food groups on the principal display panel. For example, soup made with at least 70% organic ingredients and only organic vegetables may be labeled either "soup made with organic peas, potatoes, and carrots" or "soup made with organic vegetables."

Related Terms

The NOP places no restrictions on the use of truthful labeling claims, such as "no drugs or growth hormones used," "free range," or "sustainably harvested."[3] The USDA regulates the term "free range" for poultry products; to use this term, producers must demonstrate that the poultry has been allowed "access to the outside."[4] According to Consumers Union's evaluation, this means that a poultry product comes from a bird that had at least 5 minutes of access to the outdoors each day.[4,5] No standard definition exists for all other products

TABLE 1 Commonly Used Food Product Marketing Terms

Term	Definition
100% organic	Must contain only organically produced ingredients and processing aids (excluding water and salt).
Organic	Must consist of at least 95% organically produced ingredients (excluding water and salt). Any remaining product ingredients must consist of nonagricultural substances approved on the National List.
Made with organic ingredients	Must contain at least 70% organic ingredients.
Natural	A product containing no artificial ingredient or added color and that is only minimally processed (a process that does not fundamentally alter the raw product). The label must explain the use of the term.
Free range	Producers must demonstrate to the USDA that the poultry has been allowed access to the outside.
No hormones (pork or poultry)	Hormones are not allowed in raising hogs or poultry. Therefore, the claim "no hormones added" cannot be used on the labels of pork or poultry unless it is followed by a statement that says "Federal regulations prohibit the use of hormones."
No hormones (beef)	The term "no hormones administered" may be approved for use on the label of beef products if sufficient documentation is provided to the USDA by the producer showing no hormones have been used in raising the animals.
No antibiotics (red meat and poultry)	The terms "no antibiotics added" may be used on labels for meat or poultry products if sufficient documentation is provided by the producer to the USDA demonstrating that the animals were raised without antibiotics.
Certified	"Certified" implies that the USDA's Food Safety and Inspection Service and the Agriculture Marketing Service have officially evaluated a meat product.
Chemical free	This term is not allowed to be used on a label.

There are no restrictions on use of other truthful labeling claims, such as "no drugs or growth hormones used," or "sustainably harvested."

carrying the "free range" label, such as beef, pork, or eggs; the use of the term, however, is allowed.

The term "natural" or "all natural" is defined by the USDA for meat and poultry and means that the products contain no artificial flavoring, color ingredients, chemical preservatives, or artificial or synthetic ingredients and are "minimally processed." Minimally processed means that the raw product was not fundamentally altered. Additional USDA definitions of other labeling terms can be found in publicly available USDA fact sheets.[4]

The term "raw" milk refers to unpasteurized milk. All milk certified as organic by the USDA is pasteurized. Raw milk can contain harmful bacteria, such as *Salmonella* species, *Escherichia coli* O157:H7, *Listeria* species, *Campylobacter* species, and *Brucella* species, and has been repeatedly associated with outbreaks of disease caused by these pathogens. The American Academy of Pediatrics, US Food and Drug Administration, and Centers for Disease Control and Prevention advise consumers not to consume raw milk.[6–8]

SCOPE OF CONSUMER USE, PRICES, AND TRENDS IN ORGANIC FOOD

In 2008, more than two-thirds of US consumers bought some organic products, and more than one-quarter bought organic at least weekly. The amount of US acreage dedicated to organic crops has doubled since 1997.[9] Consumers choose organic food in the belief that organic foods are more nutritious, have fewer additives and contaminants, and are grown more sustainably.[10] Some studies[11,12] suggest that families with children and adolescents or younger consumers in general are more likely to buy organic fruits and vegetables than are other consumers.[13] The factor most consistently associated with the increased propensity to purchase organic food is the level of consumer education.[14–21] Organic products, however, cost up to 40% more.

NUTRITIONAL QUALITY OF ORGANIC VERSUS CONVENTIONAL FOOD

Produce

Consumers believe that organic produce is more nutritious than conventionally grown produce, but the research to support that belief is not definitive. Many studies have demonstrated no important differences in carbohydrate or vitamin and mineral content.[22] Some studies have found lower nitrate content in organic foods versus conventionally grown foods, which is potentially desirable because of the association of nitrates with increased risk of gastrointestinal cancer and, in infants, methemoglobinemia. Higher vitamin C concentrations were found in organic leafy vegetables, such as spinach, lettuce, and chard versus the same conventionally produced vegetables in 21 of 36 (58%) studies.[22] Other studies have found higher total phenols in organic produce versus conventionally grown produce and have postulated health benefits from antioxidant effects.[23]

Several attempts have been made to review the relevant literature and draw conclusions on organic versus conventional foods, but the results are conflicting.[24–28] A large systematic review published in 2009 found that fewer than 20% of 292 articles with potentially relevant titles met criteria for quality, leaving only 55 studies to assess. The authors highlighted the fact that the nutrient content of produce is affected by numerous factors, including the geographic location of the farm, local soil characteristics, climactic conditions that can vary by season, maturity at time of harvest, and storage and time to testing after harvest. Because of the large number of nutrients reported in various articles, the authors grouped the nutrients into large categories. They found no significant differences in most nutrients, with the exception of higher nitrogen content in conventional produce and higher titratable acidity and phosphorus in organic produce.[29] Better-quality research that accounts for the many confounding variables is needed to elucidate potential differences in nutrients and the clinical importance of nutrients that may be different. At this time, however, there does not appear to be convincing evidence of a substantial difference in nutritional quality of organic versus conventional produce.

Milk

The composition of dairy products, including milk, is affected by many factors, including differences caused by genetic variability and cattle breed; thus, the results of studies assessing milk composition must be interpreted with caution. In general, milk has the same protein, vitamin, trace mineral content, and lipids from both organically and conventionally reared cows. Fat-soluble antioxidants and vitamins present in milk come primarily from the natural components of the diet or from the synthetic compounds used to supplement the feed ingested by lactating cows.[30]

One recent study examined antibiotic and microorganism content, hormone concentrations, and nutritional values of milk in 334 samples from 48 states labeled as organic, not treated with bovine GH (referred to as "GH-free"), or conventional. This study found that milk labeled "conventional" had lower bacterial counts than milk that was organic or GH-free, although this was not clinically significant. Estradiol and progesterone concentrations were lower in conventional milk than in organic milk, but GH-free milk had progesterone concentrations similar to conventional

milk and estradiol concentrations similar to organic milk. Macronutrient composition was similar, although organic milk had 0.1% more protein than the other 2 milk types.[31]

Several studies have demonstrated that organic milk has higher concentrations of antioxidants and polyunsaturated fatty acids. However, it is important to recognize that the composition of milk is strongly related to what the cows eat. This differs by time of year (outdoors in the summer, indoor forage in the winter) and whether the farms are high or low input. High-input farms supplement the diets of cattle with proprietary minerals and vitamins. Low-input farms use methods similar to those used in organic farming but do not follow all the restrictions prescribed by organic farming standards; they use mineral fertilizers but at lower levels than used by conventional high-input systems. One study comparing milk from all 3 production systems found milk from both the low-input organic and low-input nonorganic systems generally had significantly higher concentrations of nutritionally desirable unsaturated fatty acids (conjugated linoleic acid and omega-3 fatty acids) and fat-soluble antioxidants compared with milk from the high-input systems; milk derived from cows in both organic certified and nonorganic low-input systems was significantly higher in conjugated linoleic acid content than was milk from conventional high-input systems.[32]

HORMONES

GH

Hormone supplementation of farm animals, especially with GH, is one of the major reasons consumers state they prefer to buy organic foods. Bovine GH (ie, recombinant bovine somatotropin) increases milk yield by 10% to 15% and is lipotropic in cows. Because GH is degraded in the acidic stomach environment, it must be given by injection. GH is species-specific, and bovine GH is biologically inactive in humans. Because of this, any bovine GH in food products has no physiologic effect on humans, even if it were absorbed intact from the gastrointestinal tract. In addition, 90% of bovine GH in milk is destroyed during the pasteurization process. There is no evidence that the gross composition of milk (fat, protein, and lactose) is altered by treatment with bovine GH, nor is there any evidence that the vitamin and mineral contents of milk are changed by GH treatment.[31]

GH treatment of cows may actually have environmental benefits. GH increases milk production per cow, which could theoretically decrease the number of cows needed to produce a given amount of milk, with resultant need for fewer cows and, thus, less cultivated land needed to feed the cows. In addition, fewer cows would result in the production of less manure with resultant reduced methane production and less carbon dioxide production, with a resultant salutary effect on global warming.[33]

Sex Steroids

Treatment of cattle with sex steroids increases lean muscle mass, accelerates the rate of growth, and is an efficient way to increase meat yield. Estrogens are usually given by implantation of estrogen pellets into the skin on the underside of the ear, and the ear is discarded during slaughter. Unlike GH, sex steroids are not species-specific and may be given orally without degradation in the stomach. In 1998, the Food and Agriculture Organization of the United Nations and World Health Organization jointly concluded that meat from estradiol-treated animals was safe on the basis of data obtained from residue levels in meat from studies performed in the 1970s and 1980s using radioimmunoassay methods. One study demonstrated concentrations of estrogens found in meat residues were low and overlapped with concentrations found in untreated cows.[34] Gas chromatography measurements of sex steroids progesterone, testosterone, 17β estradiol, and estrone and their metabolites in meat products, fish, poultry, milk, and eggs revealed insignificant amounts compared with daily production of these steroids in adults and children.[35] Furthermore, 98% to 99% of endogenous sex steroids are bound by sex-hormone-binding globulin, rendering them metabolically inactive as only the unbound (free) forms of sex steroids are metabolically active. Synthetic sex steroids (zeranol, melengestrol, and trenbolone) commonly used in animals have lower affinities to sex-hormone-binding globulin and, therefore, are potentially more metabolically active unbound sex steroids. These hormones do not occur naturally in humans, and although the concentrations of these hormones are low in cattle, the biological effects in humans, if any, are unknown.

Ingestion of milk from estrogen-treated cows appears to be safe for children. Estradiol and estrone concentrations in organic and conventional 1%, 2%, and whole milk were the same, although the concentrations of sex steroids were higher as the fat content of the milk increased and were lower than endogenous production rates in humans. Estradiol concentrations in milk ranged from 0.4 to 1.1 pg/mL, and estrone concentrations ranged from 2.9 to 7.9 pg/mL, with the lowest concentrations in skim milk and the highest in whole milk.[36]

Endogenous estradiol concentrations are as high as 80 pg/mL in 2- to 4-month-old female infants and 40 pg/mL in 2- to 4-month-old male infants. Human milk has estradiol concentrations

as high as 39 pg/mL and estrone (which has approximately half the potency of estradiol) concentrations as high as 1177 pg/mL. Human colostrum has even higher estrogen concentrations of 500 pg/mL and 4000 to 5000 pg/mL for estradiol and estrone, respectively. Cow milk, by comparison, has estradiol concentrations of 4 to 14 pg/mL and estrone concentrations of 34 to 55 pg/mL.[37,38]

It has been postulated that ingested estrogen in food derived from sex-hormone-treated animals may play a role in earlier development of puberty and increasing risk of breast cancer. However, no studies have supported this hypothesis in humans. Studies in animals demonstrating carcinogenic and teratogenic effects of estrogens used high doses of estradiol and cannot be extrapolated to the low doses of sex steroids found in the food supply. Estrogen concentrations in the myometrium, breast, and vagina of postmenopausal women, although still low, are higher than those found in serum, and additional studies are needed to determine the significance of these low concentrations of sex steroids in estrogen-sensitive tissues.[39]

An association has been found between red meat consumption in high school girls and the development of breast cancer later in life. A 7-year prospective longitudinal study of 39 268 premenopausal women 33 to 53 years of age who filled out a comprehensive diet history of foods eaten while in high school in the 1960s and 1970s revealed a linear association between each additional 100 g of red meat consumed in high school per day with the risk of developing hormone-receptor-positive premenopausal tumors (relative risk, 1.36; 95% confidence interval, 1.08–1.70; $P = .008$). Red meat ingestion did not increase the risk of hormone-receptor-negative tumors. Although this intriguing study, which suggested that higher red meat consumption in adolescence may increase breast cancer risk, tracked cases of cancer prospectively after the dietary history was obtained, it was limited by a number of factors, including the dependence on subjects' long-term memory of amount of food eaten decades previously, the likelihood that hormone concentrations in meat were higher in that period, and the lack of direct measurement of hormonal exposure.[40] Longitudinal prospective studies are needed to compare the risk of breast cancer in women who eat meat from hormone-treated animals with the risk in women who eat meat from untreated animals.

Endocrine disrupters, chemicals that interfere with hormone signaling systems, are pervasive in our environment. Among the most commonly found endocrine disrupters are bisphenol A, found in industrial chemicals and plastics; phthalates, found in personal care items such as cosmetics; and lavender and tea tree oil, found in many hair products, soaps, and lotions; all have estrogenic properties. Endocrine disrupters are postulated to be involved in the increased occurrence of genital abnormalities among newborn boys and precocious puberty in girls. Recent literature on sex steroid concentrations and their physiologic roles during childhood indicate that concentrations of estradiol in prepubertal children are lower than originally thought and that children are extremely sensitive to estradiol and may respond with increased growth and/or breast development even at serum concentrations below the current detection limits.[41] No threshold has been established below which there are no hormonal effects on exposed children. Furthermore, the daily endogenous production rates of sex steroids in children estimated by the Food and Drug Administration in 1999 and still used in risk assessments are highly overestimated and should be reevaluated by using current assays.[41] It is therefore important to determine the relative importance of hormone treatment of animals in the context of other environmental endocrine disrupters through long-term longitudinal studies in children.

NONTHERAPEUTIC USE OF ANTIBIOTIC AGENTS

Conventional animal husbandry frequently includes the administration of antibiotic agents in nontherapeutic doses to livestock to promote growth and increase yields. Between 40% and 80% of the antimicrobial agents used in the United States each year are used in food animals, three-quarters of which is nontherapeutic. Many of these agents are identical or similar to drugs used in humans.[42] Evidence is clear that such nontherapeutic use promotes the development of drug-resistant organisms in the animals and that these organisms then colonize the intestines of people living on farms where this practice occurs.[43] Evidence is also ample that human disease caused by antibiotic-resistant organisms spread through the food chain.[44] Because organic farming prohibits the nontherapeutic use of antibiotic agents, it could contribute to a reduction in the threat of human disease caused by drug-resistant organisms.

SYNTHETIC CHEMICAL EXPOSURE

Pesticides

Pesticides have a host of toxic effects that range from acute poisonings to subtle subclinical effects from long-term, low-dose exposure.[45] Organophosphate pesticides are commonly used in agriculture, and poisoning is a persistent problem in the agricultural setting. From 1998 to 2005, 3271 cases of agricultural occupational acute pesticide poisoning were

reported to the California Department of Pesticide Regulation and the National Institute of Occupational Health's SENSOR-Pesticides program. This constitutes a rate of 56 cases per 100 000 full-time equivalents, 38 times the rate observed in nonagricultural occupations.[46] Chronic exposure among farm workers has been associated with numerous adult health problems, including respiratory problems, memory disorders, dermatologic conditions, depression, neurologic deficits including Parkinson disease, miscarriages, birth defects, and cancer.[47–50] Prenatal organophosphate pesticide exposure has been associated with adverse birth outcomes, such as decreased birth weight and length[51] and smaller head circumference.[52] A large prospective birth cohort study that measured pesticide exposure in pregnant farm workers in California and followed their offspring found lower mental development index scores at 24 months of age[53] and attentional problems at 3.5 and 5 years of age.[54] An analysis of cross-sectional data from the NHANES has demonstrated that within the range of exposure in the general US population, the odds of attention-deficit/hyperactivity disorder for 8- to 15-year-old children were increased 55% with a 10-fold increase in urinary concentrations of the organophosphate metabolite dimethyl alkylphosphate.[55]

The National Research Council reported in 1993 that the primary form of exposure to pesticides in children is through dietary intake.[56] Organic produce consistently has lower levels of pesticide residues than does conventionally grown produce,[57] and a diet of organic produce reduces human exposure. Several studies have clearly demonstrated that an organic diet reduces children's exposure to pesticides commonly used in conventional agricultural production. A small longitudinal cohort of children who regularly consumed conventional produce demonstrated that urinary pesticide residues were reduced to almost nondetectable levels (below 0.3 μg/L for malathion dicarboxylic acid, for example) when they were changed to an organic produce diet for 5 days.[58] In addition, residues varied with seasonal intake of produce, suggesting that dietary intake of organophosphate pesticides represented the major source of exposure in these young children.[59]

Although a common practice, rinsing conventionally farmed produce reduces some but not all pesticide residues on produce to varying degrees but has not been proven to decrease human exposure.[60]

Pesticide metabolite concentrations observed in studies that examined exposure in farming communities as well as in residential settings were in the same range as those observed in subjects consuming conventional produce in studies of biological exposure measures for organic versus conventional produce diets. For instance, the median concentration observed for malathion urinary metabolites in female farm workers whose offspring had significantly lower mental development index scores at 24 months of age was 0.82 μg/L,[53] which is close to the median concentration found in children in the initial conventional diet phase of the organic diet study of 1.5 μg/L, discussed previously.[58] Ranges for other pesticide metabolites were similar.

Although chronic pesticide exposure and measurable pesticide metabolite concentrations seem undesirable and potentially unhealthy, no studies to date have experimentally examined the causal relationship between exposure to pesticides directly from conventionally grown foods and adverse neurodevelopmental health outcomes. Most of the research implicating pesticides in these adverse health outcomes is from case-control or cross-sectional studies. These studies are limited by a number of factors, including difficulties measuring past exposures and the lack of a positive temporal relationship between exposure and outcome. It is difficult to directly extrapolate from these studies and draw conclusions about potential toxicity at the levels of pesticide exposure documented from dietary intake of conventional produce. Data derived from large prospective cohort studies may address some of these shortcomings.

ENVIRONMENTAL IMPACT AND PRODUCTION EFFICIENCY OF ORGANIC VERSUS CONVENTIONAL FARMING METHODS

Environmental Impact

A major subject in the organic debate is whether organic farming methods have less impact on the environment, can be equally as productive, and can be no more expensive than conventional approaches. A variety of surveys and studies have attempted to compare these issues for organic and conventional farming methods. Many believe that organic farming is less damaging to the environment because organic farms do not use or release synthetic pesticides into the environment, some of which have the potential to harm soil, water, and local terrestrial and aquatic wildlife.[61] In addition, it is thought that organic farms are better than conventional farms at sustaining diverse ecosystems, including populations of plants, insects, and animals, because of practices such as crop rotation. When calculated either per unit area or per unit of yield, organic farms use less energy and produce less waste.[62,63] Organically managed soil has been demonstrated to be of higher quality and have higher water retention, which may increase yields for organic farms in drought years.[64]

Production Efficiency

Critics of organic farming methods believe that organic farms require more land to produce the same amount of food as conventional farms. One study found a 20% smaller yield from organic farms.[65] Another study from the Danish Environmental Protection Agency found that, area for area, organic farms of potatoes, sugar beets, and seed grass produce as little as half the output as their conventional farm counterparts.[66]

It remains controversial whether organic farming is able to provide adequate food supply to sustain the world population. Norman Borlaug, considered to be the father of the "green revolution" and winner of the Nobel Peace Prize, believes that organic farming alone is incapable of feeding the world population and needs to be used in conjunction with genetically modified food.[67] On the other hand, a meta-analysis of 292 studies designed to assess the efficiency of both organic and conventional farming concluded that organic methods could produce enough food on a global per-capita basis to sustain the current human population and potentially an even larger population without increasing the agricultural land base.[68]

The largest prospective farming study to date is a comparative trial of more than 20 years' duration conducted by researchers from Cornell University. This study, conducted in Pennsylvania, compared various conventional and organic farming approaches in a controlled prospective design in which confounding influences such as weather and moisture were similar in the different systems. Over 20 years of observation, the organic fields had productivity that was generally comparable to the conventional fields, while avoiding environmental pollution with herbicides and pesticides and reducing fossil fuel consumption by 30%. Although costs were higher primarily because of increased labor costs (15%), the return for the organic plots was higher because of the higher prices commanded at the marketplace.[64]

THE DIFFERENCE IN PRICE OF ORGANIC VERSUS CONVENTIONAL FOODS

One major concern with organic food is its higher price to consumers. Organic products typically cost 10% to 40% more than similar conventionally produced products.[69] A number of factors contribute to these higher costs, including higher-priced organic animal feed, lower productivity, and higher labor costs because of the increased reliance on hand weeding. Of potential concern is that the higher price of organically produced fruits and vegetables might lead consumers to eat less of these foods, despite the well-established literature documenting the health benefits of eating fruits and vegetables, including lower rates of obesity, cardiovascular disease, and certain types of cancer. Fifty-five percent of children born in the United States are eligible for food packages under the Special Supplemental Nutrition Program for Women, Infants, and Children, and these food packages are currently giving families approximately $10 a month to spend on fruits and vegetables, so the money must be used wisely to maximize spending capacity for healthy foods.

SUMMARY

To demonstrate superiority of 1 food production method over another, it is important to show an advantage in terms of improved individual health or an important societal advantage. Organic diets have been convincingly demonstrated to expose consumers to fewer pesticides associated with human disease. Nontherapeutic use of antibiotic agents in livestock contributes to the emergence of resistant bacteria; thus, organic animal husbandry may reduce the risk of human disease attributable to resistant organisms. There is sound evidence that organic foods contain more vitamin C (ascorbic acid) and phosphorus than do conventional foods, but there is no direct evidence that this provides meaningful nutritional benefits to children eating organic foods compared with those who eat conventionally grown food products. Well-designed farming studies demonstrate that comparable yields can be achieved with organic farming techniques and that organic farming has a lower environmental impact than do conventional approaches. However, no well-powered human studies have directly demonstrated health benefits or disease protection as a result of consuming an organic diet. Such studies would be difficult to perform and require large prospective cohort populations or, better, randomly assigning subjects to interventions that increase organic versus conventional food intakes. Additional data are needed to identify relationships between diet and pesticide exposure and individual health outcomes. Pediatricians should incorporate this evidence when discussing the health and environmental impact of organic foods and organic farming while continuing to encourage all patients and their families to attain optimal nutrition and dietary variety by choosing a diet high in fresh fruits and vegetables, consistent with the USDA's MyPlate recommendations.

Key Points

1. Nutritional differences between organic and conventional produce appear minimal, but studies examining this have been limited by inadequate controls for the many subtle potential confounders, such as moisture, maturity of the produce, and measurement techniques.

No direct evidence of a clinically relevant nutritional difference between organic and conventional produce exists.

2. Organic produce contains fewer pesticide residues than does conventional produce, and consuming a diet of organic produce reduces human exposure to pesticides. It remains unclear whether such a reduction in exposure is clinically relevant.
3. Organic animal husbandry that prohibits the nontherapeutic use of antibiotic agents has the potential to reduce human disease caused by drug-resistant organisms.
4. There is no evidence of clinically relevant differences in organic and conventional milk.
   a. There are few, if any, nutritional differences between organic and conventional milk. There is no evidence that any differences that may exist are clinically relevant.
   b. There is no evidence that organic milk has clinically significant higher bacterial contamination levels than does conventional milk.
   c. There is no evidence that conventional milk contains significantly increased amounts of bovine GH. Any bovine GH that might remain in conventional milk is not biologically active in humans because of structural differences and susceptibility to digestion in the stomach.
5. Organic farming approaches in practice are usually more expensive than conventional approaches, but in carefully designed experimental farms, the cost difference can be mitigated.
6. The price differential between organic and conventional food might be reduced or eliminated as organic farming techniques advance and as the prices of petroleum products, such as pesticides and herbicides, as well as the price of energy, increase.
7. Organic farming reduces fossil fuel consumption and reduces environmental contamination with pesticides and herbicides.
8. Large prospective cohort studies that record dietary intake accurately and measure environmental exposures directly will likely greatly enhance understanding of the relationship between pesticide exposure from conventional foods and human disease and between consumption of meat from hormone-treated animals and the risk of breast cancer in women.

Advice for Pediatricians

1. Encourage patients and their families to eat an optimally health-promoting diet rich in fruits, vegetables, whole grains, and low-fat or fat-free milk and dairy products.
2. When approached by families interested in consuming organic foods, review key facts presented in this report to address the full range of relevant nutrition, human health, environmental, and cost issues. Be explicit about areas in which scientific evidence is strong as well as those in which it is uncertain.
3. When advice is sought by families concerned with the potential health impact of pesticide residues in food, direct them toward reliable resources that provide information on the relative pesticide content of various fruits and vegetables. Two such examples include:
   a. *Consumer Reports* article (September 2008) "Fruits and Vegetables, When to Buy Organic" (http://www.consumerreports.org/health/healthy-living/diet-nutrition/healthy-foods/organic-foods/overview/when-to-buy-organic.htm) and
   b. Environmental Working Group's "Shopper's Guide to Pesticides" (http://www.foodnews.org).

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Organic Foods: Health and Environmental Advantages and Disadvantages

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Are Organic Foods Healthier for Kids?

Kid's Doctor

Posted on November 8, 2012 at 5:01 AM

There's a bit of a battle brewing among some scientific communities over whether organic vegetables & meats are healthier for kids (and adults) in the long run. The controversy revolves around whether the amount of synthetic pesticides used in conventional farming is unsafe for consumers, particularly children whose bodies are still developing.

For the first time the American Academy of Pediatricians (AAP) is weighing in on the subject. The AAP said in a recent report, that at least with some foods, buying organic is worth the effort to avoid pesticide residue. That position is contrary to a recent study, released by Stanford University, that suggested organic foods and meats offer no health advantages for consumers. The Stanford study did show that 38% of conventional produce tested contained pesticide residue compared with only 7% of organic produce. However, the study did not address whether government standards for safe amounts of pesticide residue were sufficient to avoid health problems.

The AAP is concerned because babies of female farm workers in California showed small but significant developmental and motor delays when their mothers were exposed to pesticides at levels similar to those deemed acceptable in conventionally grown produce while pregnant.

While no studies have been done to see if exposure to similar levels of pesticides from simply eating produce causes similar problems, early exposure to lead and other toxins – even at low levels- is known to be harmful to children. The AAP believes that caution is advisable when considering conventionally grown produce.

"Clearly if you eat organic produce, you have fewer pesticides in your body," Joel Forman, an associate professor of pediatrics at Mount Sinai School of Medicine in New York and a lead author of the new report, tells NPR's *The Salt*. That's particularly important for young children, he says, because they are especially vulnerable to chemical exposure while their brains are developing.

Pediatricians have long encouraged parents to make sure that their children are getting plenty of fresh fruits and vegetables in their diet. Organic foods are typically more expensive and while parents might like to buy organic, they simply cannot afford the extra cost.

"We don't want to be telling people to eat organic if in the end, they eat less healthy," Forman says. Instead, he says, parents should think about buying organic for fruits and vegetables that are more likely to contain more pesticide residue, like spinach and celery, and going conventional for veggies like cabbage and sweet potatoes, which tend to have less. The new report recommends using the Environmental Working Group's Shopper's Guide to Pesticides in Produce to help decide. "It's a good resource," Forman says. "Nobody disputes the quality of the data."

The AAP sided with the federal government and the National Dairy Foundation by stating that there are no individual health benefits from purchasing un-pasteurized organic milk. The group has previously issued concerns about children drinking raw milk stating the risk of serious infection from bacteria including Salmonella, E. coli, Listeria, Campylobacter and Brucella.

The AAP suggests that organically raised meat and poultry may be a healthier choice for families. Large-scale ranching and poultry farms often add hormones and antibiotics to their feed to stimulate animal growth and prevent bacterial growth. Some studies have suggested that these additives may be contributing to the rise in deadly antibiotic resistant strains of bacteria, and hormonal changes in children and adults. The group is calling for more studies on these environmental exposures.

Vegetables, fruits, proteins, grains and protein are important to a well-rounded diet. If you can't afford to buy only organic foods, and many people can't, one way to lessen pesticide residue is to wash your produce before eating. The U.S. Food and Drug administration recommends washing produce with large amounts of cold or warm tap water. Scrub with a brush when appropriate but do not use soap. Throw away the outer layer of leafy vegetables such as lettuce and cabbage. Wash fruits in the same manner and peel the outer covering if possible. For meats and poultry, trim the fat. Some residues concentrate in animal fat.

Vegetables and fruits that have the highest pesticide residue are listed below in descending order. The first listed is the highest, then the second and so on.

- Apples
- Celery
- Sweet bell peppers
- Peaches
- Strawberries
- Nectarines-imported
- Grapes
- Spinach
- Lettuce
- Cucumbers
- Blueberries-domestic
- Potatoes

The group of vegetables and fruits shown to have the least amount of pesticide residue are:

- Onion
- Sweet Corn
- Pineapple
- Avocado
- Cabbage
- Sweet Peas
- Asparagus
- Mangoes
- Eggplant
- Kiwifruit
- Cantaloupe-domestic
- Sweet potatoes
- Grapefruit
- Watermelon

- Mushrooms

Washing produce also helps to remove dirt and some “veggie-washes” have been shown to reduce exposure to salmonella and E-coli, but none have been shown to completely eliminate pesticide residue.

Sources: **http://www.npr.org/blogs/thesalt/2012/10/22/163407880/docs-say-choose-organic-food-to-reduce-kids-exposure-to-pesticides**

http://www.aap.org

http://www.ewg.org/foodnews/summary/

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Monday, October 22, 2012

Report Supports Organic Produce, but Not Milk

By ANDREA PETERSEN

When it comes to feeding your children organic food, pediatricians have new advice for parents. Organic produce and meat might be worth the hefty price tag, but you can probably skip the organic milk.

The advice is part of a new clinical report released by the American Academy of Pediatrics and published online in the journal Pediatrics on Monday. It is the first time the organization, which is holding its annual meeting in New Orleans this week, has weighed in on the sometimes-controversial topic of the relative healthfulness of organic food.

It comes on the heels of a much-discussed study published in the Annals of Internal Medicine in September that concluded organic food isn't significantly more nutritious than conventionally grown food.

The American Academy of Pediatrics finally weighs in on organic foods for babies. The group says feeding children organic fruits and vegetables cuts down on the amount of potentially harmful chemicals and also may help prevent immunity to antibiotics. Andrea Petersen discusses on Lunch Break. Photo: Getty Images.

The pediatricians, who analyzed existing scientific evidence, also said there doesn't seem to be much difference in the vitamin and mineral content between organic and conventional foods. (Though they say some organic produce does have more vitamin C and phosphorus.)

Still, children may benefit from organic produce because it isn't grown with synthetic pesticides. The pediatricians cited several studies linking pesticide exposure to, for example, memory problems and cancer in adult farm workers and an increased risk of attention deficit hyperactivity disorder in children.

They also noted one study that showed that switching to organic produce for just five days dramatically reduced the levels of pesticide residue in the urine of children who usually ate conventional produce.

"Kids' nervous systems are developing. Exposure to toxins can have different and much more profound effects on children," said Joel Forman, an associate professor at the Mount Sinai School of Medicine in New York and a co-author of the study.

The researchers said organic milk doesn't seem to have significant health benefits for children. Many parents buy organic milk because of concerns about growth hormone and estrogen often given to conventionally raised

cows. But the pediatricians said growth hormone given to cows doesn't affect humans. "Ingestion of milk from estrogen-treated cows appears to be safe for children," they said, adding that there doesn't seem to be much difference in the sex-hormone concentrations in organic and conventional milk. Instead, to reduce exposure, drink skim. The more fat content in milk, whether organic or not, the higher the concentration of estradiol, the report found.

Regarding meat, the pediatricians focused on the risk of exposure to antibiotics from eating conventionally raised animals. Eating organic meat, they said, could reduce the risk of disease related to antibiotic resistant bacteria. They noted one study that showed that teenage girls who eat more red meat from animals treated with hormones have a great risk of breast cancer later in life. The pediatricians called for further research into the potential health effects of low-level estrogen exposure from food.

The pediatricians emphasized that the best diet for children includes a lot of fruits and vegetables. They also said there is no direct evidence that an organic diet will prevent disease or convey health benefits.

And they don't want the new recommendations to make parents—perhaps faced with a $6 half-pint of organic blueberries—to cut back on buying produce. Frugal parents might want to choose organic versions of produce that is known to have a lot of pesticide residue when conventionally grown, such as apples and celery, and skip it for low-pesticide products like onions and pineapples.

Alanna Levine, a pediatrician in Tappan, N.Y., who wasn't involved in the study, says the new recommendations won't change what she tells parents. "It is up to you," whether to buy organic food or not, she says. "There may be some benefit. It is unclear."

Write to Andrea Petersen at andrea.petersen@wsj.com

A version of this article appeared October 23, 2012, on page D4 in the U.S. edition of The Wall Street Journal, with the headline: Report Supports Organic Produce, but Not Milk.

Forbes

Henry I. Miller, Contributor
I debunk the worst, most damaging, most hypocritical junk science.

OP/ED | 11/07/2012 @ 9:35AM | 6,013 views

Is Organic Agriculture "Affluent Narcissism?"

By Henry I. Miller and Richard Cornett

As can be seen from the popularity of rip-off artists like Whole Foods markets, organic foods are popular. The U.S. market for organic produce alone was $12.4 billion last year.

Some of the devotion from consumers attains almost cult-like status, which is why a recent article by Stanford University researchers that was dismissive of health or nutritional benefits of organic foods created such a furor.

The study, by researchers in the university's Center for Health Policy and published in the Annals of Internal Medicine, was a meta-analysis in which results from the scientific literature were combined but no new, original laboratory work was conducted. Data from 237 studies were aggregated and analyzed to determine whether organic foods are safer or healthier than non-organic foods. They concluded that fruits and vegetables that met the criteria for "organic" were on average no more nutritious than their far cheaper conventional counterparts, nor were those foods less likely to be contaminated by pathogenic bacteria like E. coli or Salmonella.

The investigators themselves were surprised by the result. "When we began this project, we thought that there would likely be some findings that would support the superiority of organics over conventional food," according to physician Dr. Dena Bravata.

Many devotees of organic foods purchase them in order to avoid exposure to harmful levels of pesticides. But that's a poor rationale: Non-organic fruits and vegetables had more pesticide residue, to be sure, but more than 99 percent of the time the levels were below the permissible, very conservative safety limits set by regulators – limits that are established by the Environmental Protection Agency and enforced by the Food and Drug Administration.

Ironically, the designation "organic" is itself a synthetic construct of

bureaucrats that makes little sense. It prohibits the use of synthetic chemical pesticides – although there is a lengthy list of exceptions listed in the Organic Foods Production Act – but permits most "natural" ones (and also allows the application of pathogen-laden animal excreta as fertilizer).

These permitted pesticides can be toxic. As evolutionary biologist Christie Wilcox explained in a September 2012 Scientific American article ("Are lower pesticide residues a good reason to buy organic? Probably not."): "Organic pesticides pose the same health risks as non-organic ones. No matter what anyone tells you, organic pesticides don't just disappear. Rotenone is notorious for its lack of degradation, and copper sticks around for a long, long time. Studies have shown that copper sulfate, pyrethrins, and rotenone all can be detected on plants after harvest—for copper sulfate and rotenone, those levels exceeded safe limits. One study found such significant rotenone residues in olives and olive oil to warrant 'serious doubts…about the safety and healthiness of oils extracted from [fruits] treated with rotenone.'" (There is a well-known association between rotenone exposure and Parkinson's Disease.)

There is another important but unobvious point about humans' ingestion of pesticides: The vast majority of pesticidal substances that we consume occur in our diets "naturally," and they are present in organic foods as well as conventional ones. In a landmark research article published in the Proceedings of the National Academy of Sciences, University of California, Berkeley, biochemist Bruce Ames and his colleagues found that "99.99 percent (by weight) of the pesticides in the American diet are chemicals that plants produce to defend themselves. Only 52 natural pesticides have been tested in high-dose animal cancer tests, and about half (27) are rodent carcinogens; these 27 are shown to be present in many common foods."

The bottom line of Ames' experiments: "Natural and synthetic chemicals are equally likely to be positive in animal cancer tests. We also conclude that at the low doses of most human exposures the comparative hazards of synthetic pesticide residues are insignificant."

In other words, consumers who buy overpriced organic foods in order to avoid pesticide exposure are focusing their attention on 0.01% of the pesticides they consume.

There seems to be confusion about these issues even at the American Association of Pediatrics (AAP), which in October released a report that appeared to endorse organic produce because of its lower levels of pesticide residues, while at the same time admitting, "in the long term, there is currently no direct evidence that consuming an organic diet leads to improved health or lower risk of disease."

Perhaps the most illogical tenet of organic farming is the exclusion of "genetically engineered" plants – but only if they were modified with the newest, best, most precise and predictable techniques. Except for wild berries and wild mushrooms, virtually all the fruits, vegetables and grains in our diet have been genetically improved by one technique or another – often as a result of seeds being irradiated or genes being moved from one species or genus to another in ways that do not occur in nature. But because genetic engineering is more precise and predictable, the technology is at least as safe as – and often safer than – the modification of food products in cruder, "conventional" ways that can qualify as organic.

There are examples of new varieties of plants, including two varieties each of potatoes and squash and one of celery, that have sickened or killed consumers, but all of these were the result of conventional genetic modification – which would qualify for organic farming.

The organic community remains unswayed by either biology or history, however, and modern genetic engineering remains prohibited from organic agriculture. This bias against genetic engineering in organic agriculture makes recommendations such as those of the American Association of Pediatrics especially dubious because as genetically engineered "biofortified" foods with enhanced levels of vitamins, antioxidants and so on appear, none of them will be available to organophiles.

Another rationale for buying organic is that it's supposedly better for the natural environment. But the low yields of organic agriculture – typically 20-50 percent lower than conventional agriculture – impose various stresses on farmland and especially on water consumption. A British meta-analysis published in September of this year in the Journal of Environmental Management identified some of the environmental stresses that were higher in organic, as opposed to conventional, agriculture: "ammonia emissions, nitrogen leaching and nitrous oxide emissions per product unit were higher from organic systems," as was "land use, eutrophication potential and acidification potential per product unit."

An anomaly of the way that "organic" is defined is that it is not focused on the composition, quality or safety of the actual food; it is essentially a set of acceptable practices and procedures that a farmer intends to use. So, for example, chemical pesticide or pollen from genetically engineered plants wafting onto an organic crop from an adjacent field does not cause the harvest to lose its organic status.

In an article entitled "The Organic Fable," New York Times columnist Roger Cohen had some pithy observations stimulated by the Stanford study. "Organic has long since become an ideology, the romantic back-to-nature obsession of an upper middle class able to afford it and oblivious, in their affluent narcissism, to the challenge of feeding a planet whose population will surge to 9 billion before the middle of the century and whose poor will get a lot more nutrients from the two regular carrots they can buy for the price of one organic carrot."

Finally, many who are seduced by the romance of organic farming ignore the human toll it exacts. Missouri farmer Blake Hurst offers this reminder: "In the many places around the world where organic farming is the norm, a large proportion of the population is involved in farming. Not because they choose to do so, but because they must. Weeds continue to grow, even in polycultures with holistic farming methods, and without pesticides, hand weeding is the only way to protect a crop." He might have added that in many places, the back-breaking drudgery of hand-weeding falls largely to women and children.

Save your money. It's more cost-effective, environmentally responsible and humane to buy conventional food than the high-priced organic stuff.

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Western Plant Health Association, a nonprofit agricultural trade group based in Sacramento, Calif.

Top 10 Food Trends For 2012

LETTER

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Combined pesticide exposure severely affects individual- and colony-level traits in bees

Richard J. Gill[1], Oscar Ramos-Rodriguez[1] & Nigel E. Raine[1]

Reported widespread declines of wild and managed insect pollinators have serious consequences for global ecosystem services and agricultural production[1–3]. Bees contribute approximately 80% of insect pollination, so it is important to understand and mitigate the causes of current declines in bee populations [4–6]. Recent studies have implicated the role of pesticides in these declines, as exposure to these chemicals has been associated with changes in bee behaviour[7–11] and reductions in colony queen production[12]. However, the key link between changes in individual behaviour and the consequent impact at the colony level has not been shown. Social bee colonies depend on the collective performance of many individual workers. Thus, although field-level pesticide concentrations can have subtle or sublethal effects at the individual level[8], it is not known whether bee societies can buffer such effects or whether it results in a severe cumulative effect at the colony level. Furthermore, widespread agricultural intensification means that bees are exposed to numerous pesticides when foraging[13–15], yet the possible combinatorial effects of pesticide exposure have rarely been investigated[16,17]. Here we show that chronic exposure of bumblebees to two pesticides (neonicotinoid and pyrethroid) at concentrations that could approximate field-level exposure impairs natural foraging behaviour and increases worker mortality leading to significant reductions in brood development and colony success. We found that worker foraging performance, particularly pollen collecting efficiency, was significantly reduced with observed knock-on effects for forager recruitment, worker losses and overall worker productivity. Moreover, we provide evidence that combinatorial exposure to pesticides increases the propensity of colonies to fail.

The majority of studies to date have focused on pesticide exposure in honeybees, but bumblebees are also crucial pollinators and have smaller colonies, making them ideally suited to investigate effects at both the individual (worker) and colony level. This study mimicked a realistic scenario in which 40 early-stage bumblebee (*Bombus terrestris*) colonies received long-term (4-week) exposure to two widely used pesticides frequently encountered when foraging on flowering crops, the neonicotinoid imidacloprid and the pyrethroid λ-cyhalothrin. Imidacloprid is a systemic pesticide found in all plant tissues, including the pollen and nectar consumed by bees (oral exposure[18–20]). λ-cyhalothrin is sprayed directly on to crops, including their flowers, to which bees will be topically exposed (details in Supplementary Information). Foraging bees are thus simultaneously exposed to both chemicals in the field, making them excellent candidates to investigate the potential for combinatorial effects of pesticide exposure. Using a split block design (see Methods), we monitored colonies exposed to each pesticide independently and in combination (ten control colonies, ten exposed to imidacloprid (I), ten exposed to λ-cyhalothrin (LC) and ten exposed to I and LC (mix = M)). Imidacloprid (dissolved in 40% sucrose solution) was provided at a concentration (10 p.p.b. (parts per billion (10^9)) within the range found in crop nectar and pollen in the field[9,21]. λ-cyhalothrin was administered following label guidance for field-spray application (see Supplementary Information). Bees were able to forage in the field, providing a realistic and demanding behavioural setting, and the foraging behaviour of individual workers was recorded using radio frequency identification (RFID) tagging technology[10,11,22] (Supplementary Figs 1 and 2). Colonies were motivated to forage because we provided them with no pollen and limited amounts of sucrose solution.

During colony development, the production of workers (and their survival) is vital to colony success because workers provide the labour (for example, brood care and foraging) for the colony. Total worker production at the end of the experiment was significantly lower in imidacloprid-treated colonies (reduced by 27% in I and 9% in M colonies) compared to control colonies (mean ($\pm$ s.e.m.) workers per colony, I = 19.7 $\pm$ 3.0, M = 24.4 $\pm$ 3.2 versus control = 27.0 $\pm$ 4.0; linear mixed effects model (LMER), I, $Z = -3.71$, $P < 0.001$; M, $Z = -2.62$, $P = 0.009$; Fig. 1a). Two of the forty colonies, both M colonies, did not survive the experiment (they 'failed' after 3 and 8 days; see Supplementary Information), a colony failure rate significantly higher than other treatments (Fisher's Exact test: mid-P correction = 0.029). These two colonies were excluded from statistical analyses to provide a conservative assessment of worker production in M colonies (when included in analysis = 20.0 $\pm$ 3.9 workers). During the experiment, 223 (21% of total) workers were found dead inside nest boxes. On average, 36 $\pm$ 7.3% and 39 $\pm$ 7.5% of workers from LC and M colonies, respectively, died in the nest box; a figure four times higher than control (9 $\pm$ 3.4%) colonies (LMER, LC, $t = 4.31$, $P < 0.001$; M, $t = 4.23$, $P < 0.001$; Fig. 1b). Moreover, 43% of the workers found dead in LC and M colonies lived fewer than 4 days after eclosion—an apparent waste of resources required for future colony growth given that such young members are unlikely to have contributed any work (for example, foraging) to offset the resources invested to produce them. Queen loss occurred in 14 colonies, although loss rate did not differ significantly among treatments (control = 4; I = 5; LC = 2; M = 3; Fisher's exact test: mid-P-correction = 0.40) and we accounted for queen loss in our analyses (see Supplementary Information).

Daily counts of newly eclosed bees showed that worker production in I colonies did not become significantly lower than control colonies until the end of week 2, and for M colonies until the end of week 4 (Fig. 1c; see Supplementary Information and Supplementary Table 1). Daily counts of dead bees also revealed that worker mortality in LC colonies did not become significantly higher than that in control colonies until the end of week 3, but worker mortality in M colonies became significantly higher than that in control colonies as early as the end of week 1. The delayed effect of imidacloprid exposure on worker productivity in I and M colonies coincides with the time taken by workers to develop from egg to adult (approximately 22 days), suggesting that the observed effect is a result of imidacloprid on brood development. Indeed, the total number of larvae and pupae combined that were found in colonies at the end of the experiment ('brood number') was significantly lower in I and M colonies compared to control colonies (LMER, I, $Z = -6.23$, $P < 0.001$; M, $Z = -5.60$, $P < 0.001$). Overall, this represented a 22% reduction in brood production in I colonies and a 7% reduction in M colonies (mean ($\pm$ s.e.m.) brood number, I = 36 $\pm$ 8.0, M = 43 $\pm$ 11.7 (including failed colonies:

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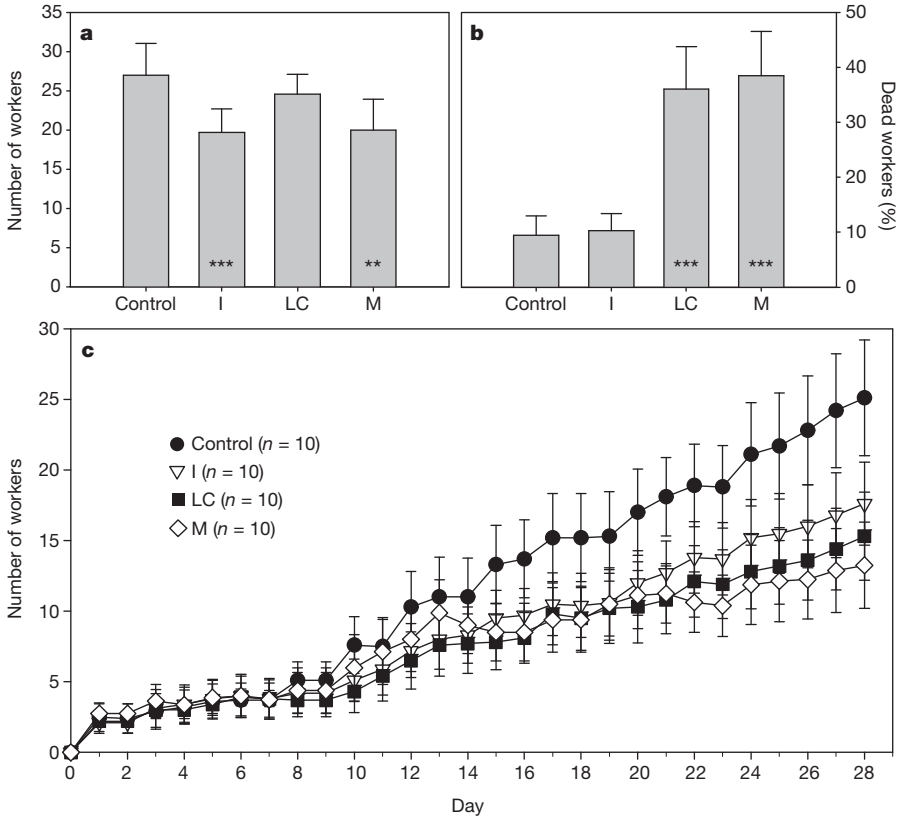

Figure 1 | Worker production and mortality. **a**, Mean (± s.e.m.) number of workers per colony that eclosed by the end of the experiment. **b**, Mean percentage of workers per colony found dead inside the nest box by the end of the experiment. **c**, Colony growth shown by daily counts of the cumulative number of workers eclosed minus the cumulative number of workers found dead (mean (± s.e.m.) per colony). Data shown on the *x* axis indicate the number of days since the start of the experiment (day 1 = 24 h after the start of experimentation). M treatment includes the two collapsed colonies. $*P \leq 0.05$, $**P \leq 0.01$, $***P \leq 0.001$ (comparison with control).

M = 39 ± 9.6) versus control = 46 ± 9.7). Despite this, there was no significant difference in the mass of the wax nest structure (see Supplementary Information for details) across treatments at the end of the experiment (LMER, I, $t = -1.12$, $P = 0.27$; M, $t = -1.22$, $P = 0.23$; Supplementary Fig. 3) indicating that I and M colonies attempted to raise similar brood numbers but that a lower proportion of larvae and pupae survived to eclosion.

Although imidacloprid could be directly affecting brood (physiological) development, it could also indirectly affect the brood by causing changes to colony behaviour and/or structure: for example, changes to foraging behaviour leading to food limitation[23,24]. We tested this hypothesis by studying worker foraging performance using RFID technology to automatically record the exact time workers left or entered each colony (Supplementary Figs 1 and 2). Overall, we collected data from 259 recognized foragers from 32 colonies (*n* colonies: control = 7; I = 10; LC = 8; M = 7) making 8,751 foraging bouts (median (interquartile range) per worker = 23 (10–44); for criteria used to classify foragers and foraging bouts see Methods). We examined whether pesticide treatment affected foraging activity and forager recruitment. We found that foragers from M colonies performed fewer foraging bouts compared to control colonies (LMER, $t = -2.55$, $P = 0.011$; Fig. 2a), and that there were significantly more foragers in both I and M colonies compared to control colonies over the 4 weeks (LMER, I, $Z = 4.20$, $P < 0.001$; M, $Z = 3.49$, $P < 0.001$; Fig. 2a). The higher number of foragers in I and M colonies (compared to control) is unlikely to be due to either pesticide causing a significant repellent or anti-feedant effect (this corroborates the lack of published evidence for pyrethroid repellency in bumblebees despite reports of pyrethroids being repellent to honeybees[25]). This is because workers did not have to visit the feeder, as they could forage for nectar outside, yet we found no difference among treatments in the amount of sucrose collected from feeders (LMER, $t \leq 1.63$, $P \geq 0.11$; Supplementary Fig. 6).

Given that I and M colonies recruited higher numbers of workers to forage compared to control colonies, we evaluated whether this was a response to reduced individual foraging efficiency by monitoring pollen foraging performance and observing the size of pollen loads (load size scored as: small = 1, medium = 2, large = 3; see Methods) brought back by foragers ($n = 20$ h of observation per colony). Crucially, imidacloprid-exposed foragers returned with significantly smaller pollen loads per foraging bout compared to control colonies (LMER, I, $t = -3.31$, $P = 0.0011$; M, $t = -3.38$, $P < 0.001$; Fig. 2b). Imidacloprid-exposed foragers collected pollen successfully in a significantly lower percentage of their foraging bouts (mean (± s.e.m.), I = 59 ± 7.3%, M = 55 ± 8.6% versus control = 82 ± 5.8%; LMER, I, $t = -3.16$, $P = 0.0018$; M, $t = -3.05$, $P = 0.0026$; Supplementary Fig. 4) and we also found that the average duration of successful foraging bouts (during which pollen was collected) was significantly longer for imidacloprid-exposed foragers than for control foragers (LMER, I, $t = 2.10$, $P = 0.037$; M, $t = 2.87$, $P = 0.005$; Fig. 2c). Together, these data show that imidacloprid-exposed workers were significantly less efficient at collecting pollen in the field.

A consequence of recruiting a greater number of workers to forage is that it increases the proportion of colony workforce going outside to undertake a potentially hazardous task[22]. Indeed, our RFID data show the number of foragers per colony was significantly correlated with the number of workers leaving the colony and getting 'lost' outside (that is, workers that did not return: Spearman's Rank, $\rho = 0.801$, $P < 0.001$; Supplementary Fig. 5). Consequently, we found that on average the percentage of workers getting lost in I and M colonies was 50% and 55% higher than control colonies (I = 30 ± 3.1%, M = 31 ± 5.3% versus control = 20 ± 2.9%; LMER, I, $t = 2.83$, $P = 0.008$; M, $t = 2.26$,

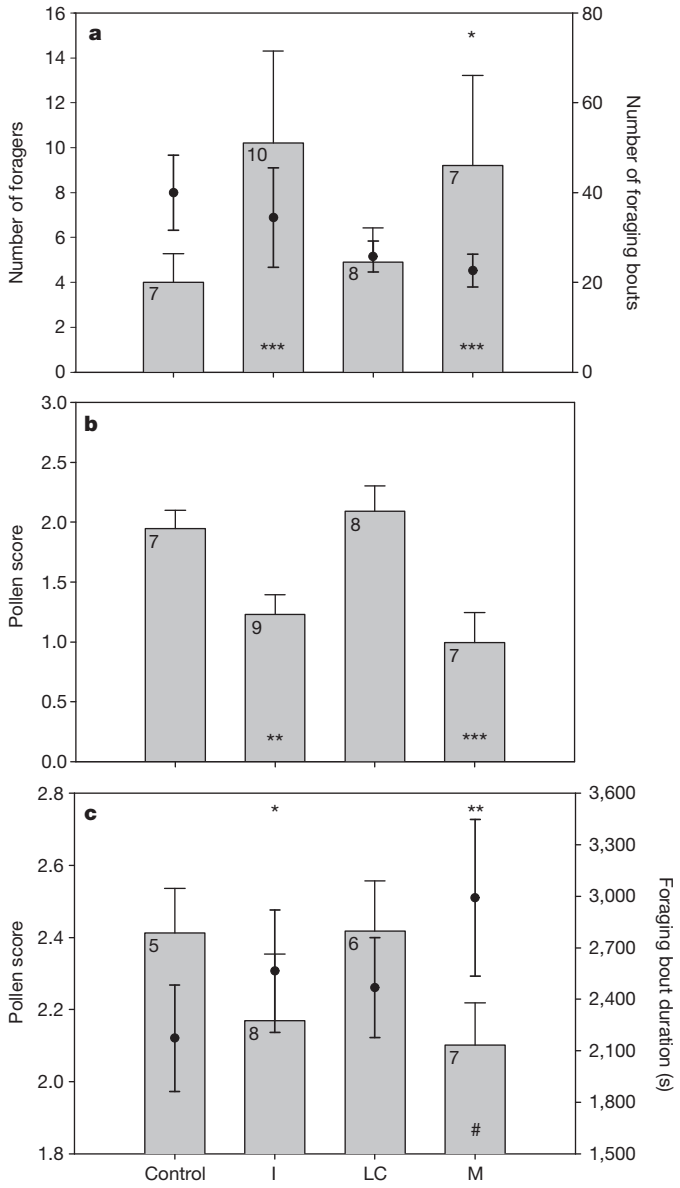

Figure 2 | Foraging performance. **a**, Mean (± s.e.m.) number of foragers per colony (column), and foraging bouts per worker per colony (filled circles: $n = 259$ foragers). **b**, Mean pollen score per worker per colony for all observed foraging bouts ($n = 228$ foragers). **c**, Mean pollen score per successful (pollen) foraging bout for each worker per colony (column), and mean duration of successful foraging bouts per worker per colony (filled-circles) ($n = 147$ foragers). *n* colonies shown in top left corner of columns. Significant differences from control treatment for column data are shown at the bases of columns, and for filled-circle data are shown above columns (**a** and **c**). $\#P \leq 0.1$, $*P \leq 0.05$, $**P \leq 0.01$, $***P \leq 0.001$ (comparison with control).

$P = 0.03$). Furthermore, when considering worker mortality and losses combined over the 4 weeks (mean (± s.e.m.): I = 41 ± 4.2%, LC = 51 ± 6.8%, M = 69 ± 7.1% versus control = 30 ± 5.0%, LMER, I, $t = 1.79$, $P = 0.08$; LC, $t = 3.25$, $P = 0.0026$; M, $t = 5.24$, $P < 0.001$; Table 1 and Fig. 3), we found that colonies treated with both pesticides (M) suffered most severely. Moreover, M colonies had significantly higher overall worker losses than either I colonies (LMER, $t = -3.69$, $P < 0.001$) or LC colonies (LMER, $t = -2.31$, $P = 0.027$).

We have shown that imidacloprid exposure at concentrations that can be found in the pollen and nectar of flowering crops causes impairment to pollen foraging efficiency, leading to increased colony demand for food as shown by increased worker recruitment to forage. However, imidacloprid-treated colonies (I and M) were still unable to collect as much pollen as control colonies. Such pollen constraints, coupled with a higher number of workers undertaking foraging rather than brood care, seemed to affect brood development, resulting in reduced worker production that can only exacerbate the problem of having an impaired colony workforce. These findings show a mechanistic explanation to link recently reported effects on individual worker behaviour[10,11,26–29] and colony queen production[12] as a result of neonicotinoid exposure. Moreover, exposure to a second pesticide λ-cyhalothrin (pyrethroid) applied at label-guideline concentration for crop use caused additional worker mortality in this study highlighting another potential risk. Bee colonies typically encounter several classes of pesticides when foraging in the field[13–15], potentially exposing them to a range of combinatorial effects. Indeed, M colonies in our study were consistently negatively affected in all our measures of worker behaviour, suffered the highest overall worker losses (worker mortality and forager losses), which were twice as great as for control colonies, and two colonies did in fact fail (Table 1).

Table 1 | Summary of observed pesticide effects for each treatment group (I, LC or M) in comparison to the control group

Effect level	Effect type	I	LC	M
Effects on individual behaviour	Number of foragers	+	ND	+
	Foraging bout frequency	ND	ND	−
	Amount of pollen collected	−	ND	−
	Duration of pollen foraging bouts	+	ND	+
Effects at colony level	Worker production	−	ND	−
	Brood number	−	ND	−
	Nest structure mass	ND	ND	ND
	Worker mortality	ND	+	+
	Worker loss	+	−	+
	Worker mortality & loss	ND	+	+
	Colony failure (*n* failed/*n* survived)	0/10	0/10	2/8

Significant decrease (−), significant increase (+) and no detected effect (ND) at the 5% significance level.

Pesticide-label-guidance concentrations and application rates are approved on the basis of ecotoxicological tests using single pesticides and set at a level for field use deemed 'sublethal' (below a dose lethal to 50% of animals tested (LD_{50})). However, the risk of exposure to multiple pesticides, or of the same pesticide being applied to different (adjacent) crops, is currently not considered when evaluating the safety of pesticides for bees. Given the serious impacts on M colonies it is concerning that pesticide products containing mixtures of neonicotinoids and pyrethroids are in current use[18]. At present there are also no guidelines for testing chronic or sublethal effects of pesticides on

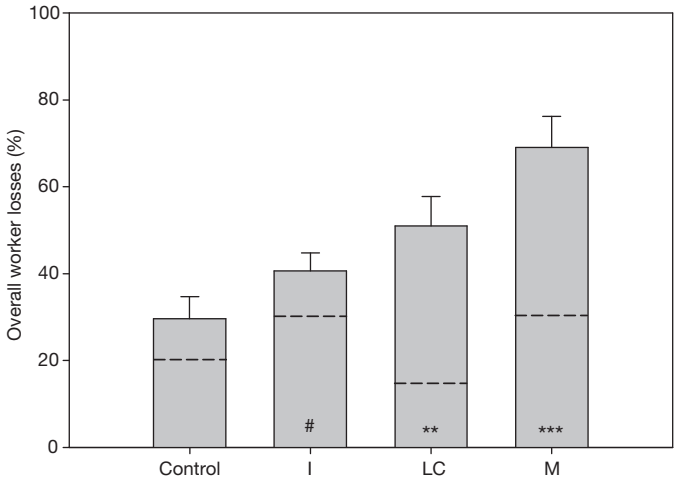

Figure 3 | Overall worker losses. Mean (± s.e.m.) overall percentage of workers lost per colony, including workers lost outside (below the dashed line) and worker mortality (dead workers found in nest box; above the dashed line), during the 4-week experiment. $n = 40$ colonies. $\#P \leq 0.1$, $**P \leq 0.01$, $***P \leq 0.001$ (comparison with control).

bees[30], and considering that we did not detect significant effects until 2 to 4 weeks into our study, the current European and Mediterranean Plant Protection Organisation (EPPO) and Organization for Economic Co-operation and Development (OECD) guideline of a maximum exposure of 96 h (for testing acute effects of pesticides on honeybees) appears to be insufficient. Our results emphasize the importance of recent recommendations by the European Food Safety Authority (EFSA) Panel on Plant Protection Products and their Residues (http://www.efsa.europa.eu/en/efsajournal/pub/2668.htm) proposing the need for longer term toxicity testing on both adult bees and larvae, new protocols to detect cumulative toxicity effects and separate risk assessment schemes for different bee species. Our findings have clear implications for the conservation of insect pollinators in areas of agricultural intensification, particularly social bees with their complex social organization and dependence on a critical threshold of workers performing efficiently to ensure colony success.

METHODS SUMMARY

Each colony contained a queen and ten or fewer workers at the start of the experiment, with no significant difference among treatments in worker number (Kruskal–Wallis: $H = 0.26$, $P = 0.97$). Colonies were housed in two-chambered nest boxes, with the rear chamber housing the nest and front chamber used for pesticide exposure (Supplementary Figs 1 and 6). Nest boxes were kept in the laboratory but connected via an outlet tube to the outside to allow natural foraging. Foraging activity of tagged workers was automatically recorded by RFID readers placed at the entrance to each nest box (Supplementary Fig. 2). The food chamber housed a feeder containing a specified volume (averaging 13ml) of control sucrose solution (control and LC) or 10 p.p.b. imidacloprid sucrose solution (I and M) provided every 2 to 3 days (Supplementary Table 2). The feeder was placed in a Petri dish lined with filter paper that was sprayed once at the start of each week with 0.69 ± 0.046 ml of control solution (control and I) or 37.5 p.p.m. (parts per million (10^6)) λ-cyhalothrin solution (LC and M). Workers walking across the filter paper to the feeder had contact exposure to λ-cyhalothrin (LC and M), and oral exposure to imidacloprid (I and M) when feeding. Colonies were not provided with pollen to motivate foraging behaviour. All workers were RFID tagged, with new workers tagged within 3 days of eclosion (Supplementary Fig. 2). We classified a foraging bout as a period of at least 5 minutes between a worker leaving and returning to a colony, and a forager as a worker that performed at least 4 foraging bouts. Pollen foraging was observed for 1 hour per colony per day (5 days per week) recording the presence and size of pollen loads collected (Supplementary Table 2). Colonies were frozen at the end of the experiment; the number of workers (and tag identifications) and brood was counted, and the mass of the nest structure was recorded.

Full Methods and any associated references are available in the online version of the paper.

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Supplementary Information is available in the online version of the paper.

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Author Contributions R.J.G., O.R.-R. and N.E.R. carried out the experiment; R.J.G. and N.E.R. designed the experiment and wrote the paper; N.E.R. conceived the project.

Author Information Reprints and permissions information is available at www.nature.com/reprints. The authors declare no competing financial interests. Readers are welcome to comment on the online version of the paper. Correspondence and requests for materials should be addressed to R.J.G. (richard.gill@rhul.ac.uk) or N.E.R. (nigel.raine@rhul.ac.uk).

METHODS

Experimental setup. Each colony contained a queen and an average of four workers (range = 0–10) at the start of the experiment, reflecting the development stage of natural colonies when crops tend to flower in Europe[31,32], and when most pesticide treatments are applied (March to June)[33,34]. We used a split block design to account for variation in colony size, developmental stage and potential seasonal variation between replicates (20 colonies in July, and 20 colonies in September: see Supplementary Information). For each replicate, colonies were ranked according to the number of workers and pupae, with the 4 highest-ranked (largest) colonies assigned to block 1, the next 4 highest ranked to block 2, and so on. Each replicate consisted of 5 blocks ($n = 20$ colonies). Within each block the 4 treatments (control, I, LC and M) were randomly assigned among the 4 colonies. There was no significant difference among treatments in either the number of workers or pupae present at the start of the experiment (Supplementary Information). Colonies were provided a two-chambered nest box; the rear chamber housing the nest ('brood chamber') and front chamber used for pesticide exposure ('food chamber'; Supplementary Figs 1 and 6). Nest boxes were kept in the laboratory but connected to the outside environment through an outlet tube leading to an exit hole in the laboratory window, allowing natural foraging (for details see Supplementary Information and Supplementary Fig. 1). Between the outlet tube and nest box were three sections of transparent tubing allowing observation of bees as they left or entered nest boxes (Supplementary Fig. 2). Two RFID readers (Maja IV reader modules with optimized antenna for mic3 transponders: Microsensys GmbH) at the nest entrance allowed automatic monitoring of all tagged workers as they entered and left the colony with minimal disturbance to natural foraging patterns[22].

Pesticide treatment. Bees were exposed to pesticide treatments in the food chamber using a gravity feeder placed on a Petri dish (90 mm diameter) lined with filter paper. The filter paper was sprayed with 0.69 ± 0.046 ml of either control solution (control and I) or 37.5 p.p.m. λ-cyhalothrin solution (LC and M); the maximum label-guidance concentration for spray application to oilseed rape in the United Kingdom. The gravity feeder contained either a control sucrose solution (control and LC) or 10 p.p.b. imidacloprid sucrose solution (I and M). This concentration falls within the range found in the pollen and nectar of flowering crops visited by bees[9,20,21,35–38] (for details on pesticide selection and application see Supplementary Information and Supplementary Box 1). During the experiment the sucrose treatment was applied every 2 days (3 days over weekends) between 13:00 and 14:00 (Supplementary Table 2). Before refilling feeders we measured the volume of any remaining solution to calculate what the bees had collected ($n = 12$ feeder replenishments per colony during the 28-day period). We provided 10 ml of sucrose treatment per application in week 1, with a 2-ml incremental increase in the volume of sucrose at the start of each subsequent week (week 2 = 12 ml, week 3 = 14 ml, week 4 = 16 ml) to reflect an increase in colony demand as they developed. The amount of sugar provided was less than each colony typically collects by nectar foraging[39], ensuring that workers were motivated to forage for nectar and pollen outside.

Spray treatments were applied once at the start of each experimental week (Supplementary Table 2) using a new piece of filter paper for each application. This follows label guidance for the maximum application of λ-cyhalothrin to crops that recommends at least 7 days between spraying events and a maximum of 4 applications within the flowering season.

Observations and measurements. To monitor colony condition and development, colonies were inspected every day to assess the number of newly eclosed (callow) workers, the number of dead workers (removed and frozen (–20 °C)), and queen condition. Three days before the start of the experiment faecal samples from each queen were checked for the presence of three parasites: the trypanosome *Crithidia bombi*, the microsporidian *Nosema bombi* and the neogregarine *Apicystis bombi*. This parasite assessment was repeated on the twenty-eighth experimental day using faecal samples from the queen (if present) and a subset of workers from each nest box (for details of parasite assessment see Supplementary Information).

To monitor foraging performance, all workers present at the start of the experiment (precise age unknown) were individually RFID tagged (for details see Supplementary Information), and during the experiment all newly produced workers were tagged within 3 days of eclosion (age known). Tagging stopped on the twenty-fourth day of the experiment because any workers emerging after this point were unlikely to become foragers[40]. In total, 854 workers were tagged, with each tag providing a unique (16-digit) code for unambiguous identification. We classified a foraging bout as a period of at least 5 minutes elapsing between a worker leaving and entering a colony. We also specified that workers must perform at least four foraging bouts to be considered a forager (for the rationale behind foraging rules see Supplementary Information).

Pollen foraging was observed in each colony for 1 hour per day (5 days a week) to record pollen foraging activity. Observation periods were always 2 h (at approximately 16:00) and 21 h (at approximately 10:00 the following day) after treatment application or renewal (Supplementary Table 2). We recorded the time that each tagged worker entered a colony (observing when it passed through the transparent tubes and under the RFID readers) using a stopwatch synchronised with the RFID (host) data logger. We scored the amount of pollen in the forager's corbiculae (pollen baskets) as small (score of 1), medium (score of 2) or large (score of 3) relative to the size of the worker.

Nest box entrances were closed after dark on the evening of the twenty-eighth experimental day. Each nest box, containing bees and brood, was placed in a freezer (–20 °C). Window exits remained open for 18 h with each outlet tube connected to an individual bottle trap to catch any returning foragers. All tagged workers were identified and recently eclosed (untagged) workers were assumed to have developed in the colony they were found in. Worker thorax width was measured using digital callipers. All pupae and larvae were dissected from each nest, counted and weighed to provide final measures of brood development, and the nest structure was also weighed.

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Cocktail of pesticides increases bee deaths, says study

Monday 22 October 2012 09:00

Chronic exposure to two commonly used pesticides in farming kills worker bees and damages their ability to forage for food, according to a new study.

Biologists at the University of London conducted field studies into bumblebees using neonicotinoids and pyrethroids at concentrations similar to field-level exposure.

The Gill/Raine study mimicked realistic scenarios in which crops were sprayed with different pesticides and dosages at different times.

Forty early stage bumblebee colonies received four-week exposure to two pesticides that are frequently encountered when foraging on flowering crops: the neonicotinoid Imidacloprid - an active ingredient of Bayer Crop Protection - and Lambda-Cyhalothrin, a generic active ingredient originally developed by Syngenta but now widely manufactured by many other producers.

Imidacloprid was provided in a sucrose solution at levels that could be found in nectar and cyhalothrin was administered following label guidance for field spray applications.

Bees were able to forage in the field providing a realistic behavioural setting, and the foraging behaviour of individual workers was recorded using radio frequency identification (RFID) tagging technology.

The researchers found that bees exposed to imidacloprid were less able to forage effectively, particularly when it came to collecting pollen.

This meant treated colonies had less food available to them, so could not raise as many new workers. On average, the percentage of workers leaving the colony and then getting lost was 55% higher in those receiving imidacloprid than those that were not exposed to pesticides.

"Currently pesticide usage is approved based on tests looking at single pesticides. However, our evidence shows that the risk of exposure to multiple pesticides needs to be considered, as this can seriously affect colony success."
Nigel Raine

"Chronic exposure... impairs natural foraging behavior and increases worker mortality, leading to significant reductions in brood development and colony success," said the researchers, in the report published in *Nature* journal.

Combined exposure to the two pesticides "increases the propensity of colonies to fail", added the researchers.

Ecologist and study co-author Nigel Raine said: "Currently pesticide usage is approved based on tests looking at single pesticides. However, our evidence shows that the risk of exposure to multiple pesticides needs to be considered, as this can seriously affect colony success."

Bee decline

Bees are vitally important to agriculture for pollinating our food crops and maintaining biodiversity in the rural environment.

A recent Friends of the Earth report estimated bees are worth £510m a year to the UK economy.

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However, bee numbers have been declining worldwide in recent years and conservationists claim that pesticides are a contributing factor, in particular neonicotinoids.

In June, the French government banned Cruiser OSR, a neonicotinoid pesticide made by Syngenta, citing scientific evidence from two French studies that linked neonicotinoids with bee colony disorder.

Last month, a **DEFRA report rejected the research** and said it would not change existing legislation.

Following the release of this latest report, a DEFRA spokesman said: "We are looking at the risks of pesticides to bees as we take any threat to them very seriously.

"However, until we have all the evidence back from our research we won't be putting in place any new restrictions. We will act if our new evidence shows the need."

Luke Gibbs, spokesman for Syngenta UK, branded the latest study "unrealistic" and said there was no evidence that pesticides damaged the health of bee populations.

"This study deliberately encouraged bumblebees to feed on a sucrose solution containing an insecticide for four weeks in the laboratory," said Mr Gibbs.

"To reach the solution they were forced into contact with a potentially lethal dose of another insecticide before leaving the lab to forage. The possibility of this scenario being repeated in the field is highly questionable."

More on this topic

Cruiser OSR study 'flawed', says British scientists

DEFRA launches consultation on farm pesticide use

Philip Case on G+

Cruiser OSR study 'flawed', say British scientists

Philip Case
Wednesday 26 September 2012 08:55

British scientists have branded a study that prompted a French ban on a valuable oilseed rape pesticide as "flawed".

France withdrew Syngenta's marketing licence for Cruiser OSR in June, amid claims in a study that neonicotinoids cause honeybee colony collapse.

But research from the Food and Environment Agency and the University of Exeter, published in the *Science* journal, has highlighted flaws with the original study.

The previous research, led by French scientist Mikaël Henry, showed that the death rate of bees increased when they drank nectar laced with a neonicotinoid pesticide, thiamethoxam, an active ingredient in Cruiser OSR, a pesticide produced by the Swiss company Syngenta.

It calculated that this would cause their colony to collapse.

However, new research by the British team explained that the calculation might have used an inappropriately low birth rate and underestimated the rate at which colonies could recover from the loss of bees.

Lead author James Cresswell, of Biosciences at the University of Exeter, said: "We know that neonicotinoids affect honeybees, but there is no evidence that they could cause colony collapse.

"When we repeated the previous calculation with a realistic birth rate, the risk of colony collapse under pesticide exposure disappeared."

Dr Cresswell could not, however, determine that pesticides were harmless to honeybees but he said the research showed that the effects of thiamethoxam were not as severe as first thought.

"We do not yet have definitive evidence of the impact of these insecticides on honeybees and we should not be making any decisions on changes to policy on their use," he added.

More research was needed to understand the real impact of neonicotinoids on honeybees, Dr Cresswell said.

Meanwhile, the Environmental Audit Committee has launched an inquiry looking at the effects of pesticide use in the UK on biodiversity with a specific focus on bees.

In a letter sent to The Guardian, Joan Walley, chair of the committee, said: "DEFRA ministers may want to start doing their homework on pesticide policy and biodiversity, because we will be calling them before parliament on these issues.

"In particular, we will be scrutinising the evidence behind the government's decision not to revise pesticide regulations or to follow other European countries in temporarily suspending the use of insecticides linked to bee decline."

Luke Gibbs, Syngenta UK spokesman, said he welcomed the inquiry and hoped it would focus on science and not the politics.

"It's an opportunity for the industry and the relevant government advisory agencies to outline the science that underpins neonicotinoid pesticides, to detail how these products protect crop yield and quality, and how in practice this advanced class of pesticide works to reduce adverse impacts on beneficial insects such as bees," he added.

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Neonicotinoids are among the most widely used agricultural insecticides and honeybees ingest residues of the pesticides as they gather nectar and pollen from treated plants.

The French ban does not affect British growers and Cruiser seed treatment is still free to use as normal in the current planting season.

More on this topic

DEFRA dismisses study linking bee deaths to pesticide use

France upholds ban on Syngenta's Cruiser OSR

Philip Case on G+

THE INDEPENDENT

Double dose of pesticide poses new danger for bumblebees

Government study suggests long-term exposure to chemicals can destroy colonies

Michael McCarthy

Monday, 22 October 2012

The combination of two pesticides commonly used on UK fields can have damaging effects on the behaviour of bumblebees and cause their colonies to collapse, new research by British scientists has found.

And long-term exposure to individual pesticides – for up to a month – is also likely to have damaging effects, the scientists say. They argue that current safety tests are insufficient, as guidelines only demand pesticides are tested on bees for four days.

The findings, which come from a Government-funded study, represent the fifth major piece of research to appear this year linking the worldwide and worrying declines of bees to pesticides, and in particular to the use of the relatively new nerve-agent pesticides, the neonicotinoids.

This new study is considered particularly important because bees forage widely so are likely to encounter more than one type of pesticide.

The research was carried out at Royal Holloway College, University of London, as part of the Insect Pollinators Initiative, a £10m British programme looking at threats to pollinating insects such as bees, butterflies, moths and hoverflies.

Published last night in Nature online, the study reports that exposure to two commonly used pesticides, one a neonicotinoid and the other from a different pesticide family, a pyrethroid, at concentrations approximating what might be found in the field, impaired the natural foraging behaviour of bumblebees. This led to increased numbers of deaths and in some cases the failure of colonies. The compounds involved were made by major agrochemical manufacturers: the most widely used neonicotinoid, imidacloprid, manufactured by Bayer and already implicated in problems with bees in earlier studies, was one, while the pyrethroid was lambda-cyhalothrin, originally developed by Syngenta.

The Royal Holloway researchers found that bees exposed over a month to imidacloprid were less able to collect pollen effectively, which meant that their colonies had less food available and so could not raise as many workers. On average, the percentage of workers leaving the colony and then getting lost was 55 per cent higher in those receiving imidacloprid than those that were not exposed to pesticides.

Two of the test colonies that were exposed to both pesticides together collapsed completely.

"The risk of exposure to multiple pesticides, or of the same pesticides being applied to different (adjacent) crops, is currently not considered when evaluating the safety of pesticides for bees," the researchers say.

On long-term effects, they say: "Considering that we did not detect significant effects until two to four weeks into our study, the current... guideline of a maximum exposure of 96 hours for testing acute effects of pesticides on honeybees appears to be insufficient."

A spokesman for Syngenta said yesterday: "There's no evidence that pesticides damage the health of bee populations but yet again we see unrealistic research being used to prove the opposite."

NATURE | **NEWS**

The buzz about pesticides

Common pesticides affect bumblebee foraging.

Charlotte Stoddart

21 October 2012

Bees, the most important pollinators of crops, are in trouble. All over the world, their populations are decreasing and scientists and farmers want to know why. In some cases, such as the widely reported colony collapses in North America in 2006, it is probably down to disease. But a blooming crop of research suggests that pesticides are also to blame[1–3].

Earlier this year, two studies published in *Science* showed that colonies are severely affected when bees are exposed to neonicotinoid pesticides of the kind commonly sprayed on crops. In one study[1], exposure led to a significant loss of queens in colonies of bumblebees (*Bombus terrestris*). In the other[2], on honeybees (*Apis mellifera*), the insecticide interfered with the foragers' ability to navigate back to the hive.

Now, in a study published in *Nature*[3], researchers at Royal Holloway, University of London, in Egham, UK, show that low-level exposure to a combination of two pesticides is more harmful to bumblebee colonies than either pesticide on its own. The results suggest that current methods for regulating pesticides are inadequate because they consider only lethal doses of single pesticides. As ecologist Nigel Raine explains in the video, low doses of pesticides have subtle effects on individual bees and can seriously harm colonies. He hopes that his work will feed into consultations on pesticide regulations that are happening now in Europe.

Nature doi:10.1038/nature.2012.11626

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Most produce sampled in California had no detectable pesticides

11/08/2012 4:35:00 AM
By Vicky Boyd


Vicky Boyd

The majority of fresh produce sold and sampled in California last year had no detectable pesticide residue, according to a report released by the California Department of Pesticide Regulation.

During 2011, the Sacramento-based department collected 2,707 samples of more than 160 diffrerent types of domestic and imported produce.

Of those, about 60 percent—or 1,647 samples—had no detectable pesticide residues.

Another 36 percent had residues within limits set by the U.S. Environmental Protection Agency.

In addition, 3.4 percent had illegal residues, although most were at such low levels they did not pose acute health risks, according to the report.

Most of those samples were imported from other countries.

Of the total samples, 988 were from California-grown produce, and 97.9 percent were in within legal limits.

Nearly one-quarter of the samples were analyzed with new technology that expanded the number of pesticides detected.

The new technology will be full implemented by 2014.

Rock Hill Herald Online

US Home & Garden Pesticides Market

Published: October 30, 2012

By Reportlinker

NEW YORK, Oct. 30, 2012 — /PRNewswire/ -- Reportlinker.com announces that a new market research report is available in its catalogue:

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US demand to rise 3.5% annually through 2016

US demand for home and garden pesticide products will increase 3.5 percent annually through 2016, driven by increased interest in home gardening activities and by continued concerns about pest-borne diseases. Insecticides are the largest product type by value, while herbicides are expected to show the strongest growth due to recovery in lawn and garden applications. Growth will also be promoted by the use of value-added, ready-to-apply products, and by sustained interest in less hazardous products like biopesticides. Greater public awareness of pest-borne diseases, such as hantavirus and West Nile Virus, will also contribute to demand for home pest control products and repellents.

Lawn and garden pesticides to outpace household types

Lawn and garden pesticide demand is expected to grow faster than demand for household pest control products, due primarily to a recovery in the housing market and improved economic prospects overall. Continuing the trends seen in the 2006-2011 period, which was characterized by "staycations" and do-it-yourself yard maintenance during the recession, demand for lawn and garden pest control products will be buoyed by interest in gardening (especially edible gardens) and other yard activities. Similar to household pest control, demand for lawn and garden pesticides will be strongly impacted by increased interest in less hazardous pesticides, such as natural pesticides and animal repellents. Some biopesticides, such as Bacillus thuringiensis, have already established a presence in the lawn and garden segment of the pesticide industry, but the natural and organic pesticide category as a whole remains less mature than conventional lawn and garden pesticides, offering plenty of room for expansion. In the larger household segment of the market, growth will be buoyed by concerns about pest-borne diseases, such as West Nile Virus, and by a greater level of interest in environmentally friendly pest control. The strongest gains are expected in products that can effectively protect people and the home from pest populations, while still remaining safe for people, pets, and the environment. For example, insect repellents are forecast to show faster growth than insect controls, and animal repellents are expected to overtake rodenticides by 2016. Although conventional insect repellents -- such as those containing DEET and picaridin -- will remain the most common types, biorepellents such as lemon eucalyptus oil are expected to show much faster growth as consumers become more interested in naturally derived alternatives.

Shift toward value-added products to boost prices

In addition to trends favoring safer pesticide products, the US home and garden pesticide industry is expected to continue to be affected by the shift toward value-added, ready-to-apply products available in a range of retail locations. Many of these products contain very little active ingredient, but can be sold at a premium price due to proprietary formulations that maximize a pesticide's efficacy in a particular climate or application. Additionally, many of these products are sold in special packaging, such as a specially designed spray bottle that enhances the ease of use and contributes to the value of the pesticide. The availability of pest control products in locations such as drug stores, garden supply centers, convenience stores, mass merchandisers, and home improvement stores contributes to industry growth as pesticides are readily available to consumers.

Study coverage

This upcoming industry study presents historical demand data (2001, 2006, 2011) and forecasts for 2016 and 2021 by product, application and raw material. The study also considers market environment factors, evaluates company market share and profiles industry players.

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Article published Nov 3, 2012

Deer to be tested for EEE today and Sunday

Volunteers with the Vermont Department of Health will be gathering blood samples from deer today and Sunday during youth deer hunting weekend to check for eastern equine encephalitis.

Samples will be taken from deer brought to check stations throughout the state for a project that has occurred over the last three years.

In the fall of 2010, the Health Department partnered with the Vermont Agency of Agriculture, Food and Markets and a researcher with the Centers for Disease Control and Prevention on the deer and moose survey to look for evidence of the EEE virus in Vermont.

This may be the last year of the deer survey, according to Erica Berl, a state infectious disease epidemiologist.

The project is not being modified or expanded because of two human deaths from EEE this year.

In 2010, deer and moose samples were collected from more than 500 animals during rifle season. Eleven percent of deer samples and 28 percent of moose samples tested positive.

Information on last year was not available.

2010 was the first time that evidence of EEE had been detected in Vermont.

CDC, state to conduct study in Vermont on eastern equine encephalitis

Posted By Andrew Stein On November 16, 2012 @ 8:21 pm In Recent | 1 Comment

Officials from the Vermont Department of Health and the U.S. Centers for Disease Control, or CDC, want to conduct a study in Vermont that hasn't been carried out in the U.S. for more than four decades.

On Town Meeting Day, they hope to test hundreds of Vermonters in three towns for the incidence rate of eastern equine encephalitis, or EEE.

Department Commissioner Harry Chen and four other state officials met with more than 50 concerned residents in Whiting's town hall on Thursday to talk about how the state and town might handle the incurable and potentially fatal mosquito-borne illness. Discussion wandered from the nature and history of the disease to the state's proposed research project to concerns about aerial spraying of insecticides and state practices.

Thursday's meeting follows the EEE-caused deaths of a Sudbury and a Brandon man this fall [1] and the aerial spraying of insecticides over large swaths of land in six Addison and Rutland county towns. [2]

In 2010, state officials discovered the virus for the first time in Vermont by sampling the blood of deer and moose brought in by hunters. The ruminants were carrying EEE antibodies but appeared to have successfully fought off the virus. Erica Berl, an infections disease epidemiologist for the Health Department, said mammals either fight off the virus or die.

EEE appeared again in September 2011, when the virus killed more than a dozen emus in Brandon. Then, this past summer, the state discovered EEE in humans and mosquitos for the first time.

Alan Graham of the Vermont Agency of Agriculture has sampled more than 1 million mosquitos over a 15-year period and did not come across the virus until 2012. He said that although he began using a more effective EEE test this year, the sheer volume of mosquitos found with EEE this summer and fall — the densest concentration of which was in Whiting — suggests to him that the disease spiked in Vermont this year, which mirrors a similar uptick in Massachusetts. The only species of mosquitos found to be carrying the disease in Vermont were Culista melanura.

The problem for officials looking to address the problem is that there is very little information and data on the virus and its effects on humans. It is not prominent in the winter, when mosquitos aren't active. But it will lay low for years, said Graham, and then, all of a sudden, it will explode.

"It just disappears," he said.

Chen told the room that very little research has been done on the virus.

"As much as everyone in this room wants certainty, there is none," said Chen.

A state-proposed study could bring some unknowns into focus — for both Vermont and the nation.

The study

Department of Health officials hypothesize that a number of Vermonters who live in the Addison and Rutland county areas with high EEE concentrations have contracted the disease without knowing it.

"What we don't know is how many people become infected that don't have severe symptoms or recover and don't have symptoms at all," said Chen.

To answer that question the department is proposing a human research project, where volunteers 12 and older from Brandon, Whiting and Sudbury would donate a blood sample on Town Meeting Day.

"It would be completely voluntary, but we'd love to get 100-200 individuals," said Patsy Kelso, a state epidemiologist who was present at the meeting. "We could really learn how many people have antibodies to EEE and were never infected, so that could really add a lot to our understanding of this and what the risk is of being infected and getting sick if you are infected."

The U.S. Centers for Disease Control (CDC) supports the proposal, and the federal body has agreed to carry out the testing for the state if it's approved.

Dr. John-Paul Mutebi, a CDC entymologist who specializes in vector-borne illnesses, said that the last time

such a study was conducted was in New Jersey in 1969.

“It would give us a pretty good idea of, one, the transmission potential of the virus, and, number two, how effective it really is,” he said. “This information would help us find a better way to protect people. Not only to use in public outreach, but also in planning control and prevention programs.”

Right now, a team of physicians from Fletcher Allen Health Care and the state Health Department are drawing up a study proposal for the Vermont Agency of Human Services’ Institutional Review Board, or IRB. The state IRB will evaluate the ethics and soundness of the proposed study. If the state IRB approves it, Kelso said the proposal would then go through an expedited IRB approval process via the Fletcher Allen and CDC boards.

The reason officials want to draw blood on Town Meeting Day is because a lot of people will be in one place at one time, and Kelso hopes 200 volunteers will participate in each town.

The tests won’t be conducted for diagnostic purposes, said Kelso, and no participants will find out the results.

“This is a research project,” said Kelso. “It’s a selfless act, giving blood and participating.”

Chen qualified that the aim of the study is purely for gathering information.

“There’s a heck of a lot we don’t know and one of the ways we can learn more is by doing studies,” said Chen.

Concerns

Those present at the meeting did not voice concern about the study, but many did take issue with other practices.

One concern that frequently came up was the state’s aerial application of insecticides in September, which was the first time the Agency of Agriculture targeted mosquitos from the sky.

One of the main reasons the state used this method, officials said, is because mosquito havens in remote Addison and Rutland county wetlands are not accessible from roads.

“We only sprayed because we saw repeated piles of mosquitos with EEE,” said Graham. “So the question is do we spray or not? Do we let you know there’s a high risk and not do anything? If we only spray part of it, we don’t really hit the problem.”

The state sprayed a synthetic insecticide called Anvil, which was used instead of its organic cousins in the pyrethrin pesticide family. The synthesized version was used to mitigate negative effects on bee populations. State officials said Anvil is designed to dissipate within hours, and field tests after the spraying showed bee populations weren’t affected, said Graham.

Anvil is classified as an “adulticide” and the state used it to target adult mosquitos. The reason the state didn’t go after the larvae of the Culista melanura, said Graham, is because they burrow deep into the ground where larvacides can’t penetrate.

Paul Quesnel, a local farmer, took issue with the USDA’s practice of buying up cropland and returning it to wetland. He said that land once used for crop cultivation has been turned into hotbeds for mosquito breeding.

“They’re using tax dollars to take this land out of production to create wetlands and now we’re being forced to pick up the price of spraying,” he said. “And who wants to spray insecticides widespread?”

Agency of Agriculture officials told him that the issue was beyond the scope of the meeting and that in some wetlands increased fish activity keeps mosquito populations down.

“We’re seeing fish in (Leicester) that we have not seen for 10 years,” said Graham. “There will not be mosquitos in areas that retain water like that with fish.”

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1 Comment (Open | Close)

1 Comment To "CDC, state to conduct study in Vermont on eastern equine encephalitis"

#1 Comment By Wendy Raven On November 17, 2012 @ 12:28 pm

I am wondering why the study is limited to EEE, when Lyme disease is actually a bigger ongoing threat…..I believe this should be expanded to include lyme disease.

Article printed from VTDigger: **http://vtdigger.org**

URL to article: **http://vtdigger.org/2012/11/16/cdc-to-conduct-study-in-vermont-on-eastern-equine-encephalitis/**

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[1] follows the EEE-caused deaths of a Sudbury and a Brandon man this fall: **http://vtdigger.orglink http://vtdigger.org/2012/09/18/second-vermonter-dies-from-eastern-equine-encephalitis/**
[2] large swaths of land in six Addison and Rutland county towns.: **http://vtdigger.org/2012/09/17/officials-say-mosquito-spraying-for-eastern-equine-encephalitis-at-least-60-percent-effective/**

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Article published Nov 9, 2012

Public EEE talks this month

By Cristina Kumka
STAFF WRITER

The state Health Department is getting a leg up on mosquito control for next year by holding three public meetings this month to inform residents and get more feedback from them.

Health Commissioner Dr. Harry Chen and experts from the department and the Agency of Agriculture, Food & Markets will discuss eastern equine encephalitis and mosquito control at three informational meetings. The first will be held from 6 to 8 p.m. Thursday, Nov. 15, at Whiting Town Hall, the second from 7 to 9 p.m. on Nov. 28 at Otter Valley Union High School in Brandon and the third from 7 to 9 p.m. on Nov. 29 at Sudbury Town Hall.

Two men — 86-year-old Richard Breen of Brandon and 49-year-old Scott Sgorbati of Sudbury — died from EEE in Vermont this year. Their deaths marked the first year the arbovirus was detected in the state and the first fatalities from EEE in Vermont.

The meetings are a proactive way for health and agriculture officials to address some lingering concerns of the public and inform them the state does indeed have a plan moving forward, according to Erica Berl, infectious disease epidemiologist with the Health Department.

According to the Health Department, at each meeting health and agriculture officials will review surveillance data, aerial spraying, other actions taken to control mosquitoes and plans in progress for tracking and control in 2013. The meetings will also bring to light what health officials know and don't know about the virus.

Berl said the public meetings are a few of many that will happen before next summer.

"While this is still sort of fresh in people's minds, we thought it was a good time to make ourselves available to answer questions people may have," Berl said. "People also need to know we are working on plans for next year."

Berl said that includes asking the state Legislature for more money for funding mosquito surveillance and testing in 2013, a sum that was cut by 75 percent over the last few years.

"We also want to hear what some other concerns are, so we can than think about them in the off-season when we aren't trying to react to it (EEE)," she said.

For more information on mosquito-borne illness visit Healthvermont.gov.

cristina.kumka@rutlandherald.com

West Nile Cases Still Climbing As Temperatures Drop

Helen Thompson November 7, 2012

Mosquitoes are sorted at the Dallas County mosquito lab in Dallas, Texas on Aug. 16, 2012. Dallas County has seen the highest number of cases of West Nile virus of any county in Texas: 379 this year, as of Oct. 25. (ASSOCIATED PRESS)

Summer may be long gone, but cases of West Nile virus are still popping up – making this year's outbreak the worst so far in the U.S. since 2003.

New tallies released today by the Centers for Disease Control and Prevention bring the total case count for the year to 5,054, and the death toll to 228 — more than the past four years combined.

The mosquito-borne virus has struck some states harder than others. Roughly 80 percent of cases occurred in the twelve states – Texas, California, Louisiana, Mississippi, Illinois, South Dakota, Michigan, Oklahoma, Nebraska, Colorado, Ohio, and Arizona. Texas has borne the brunt of the outbreak with 1,684 cases (about one third of the national total), and Dallas took its mosquito control measures at the end of the summer to the next level with city-wide spraying.

Epidemiologists believe West Nile first arrived in the U.S. 13 years ago as a strain that originated in Israel. Symptoms can be as mild as the flu, but severe forms can cause paralysis and inflammation in the spinal cord and brain. Such "neuroinvasive" infections account for 51 percent of all cases, according to the CDC.

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CDC officials said in September that they thought the worst was over. Now they're still asking: why so many cases this year? But at this point, it's too soon to pin it on any single factor.

One possible suspect is weather – a mild winter means more disease-carrying mosquitoes survive through the cold months.

On October 31, Maine reported its first confirmed human case of West Nile – ever. The virus had previously been detected in mosquitoes and birds. "As long as the temperatures remain above freezing there is potential for West Nile virus transmission," Stephen Sears, a state epidemiologist, said in a statement.

And some epidemiologists are wondering if this year's outbreak could be linked to larger patterns of climate change.

While the scientists parse through the data, your best bets to protect again infection are to use insect repellant, keep screen doors and windows well-maintained, reduce standing water in your backyard, and hope that this winter brings a good freeze.

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The Washington Post

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Hints of a more virulent, mutating West Nile virus emerge

By Brian Vastag, Published: November 8

The West Nile virus epidemic of 2012, the worst in a decade, may be notorious for yet another reason: The virus, in some cases, is attacking the brain more aggressively than in the past, raising the specter that it may have mutated into a nastier form, say two neurologists who have extensive experience dealing with the illness.

One doctor, Art Leis in Jackson, Miss., has seen the virus damaging the speech, language and thinking centers of the brain — something he has never observed before. The other, Elizabeth Angus in Detroit, has noticed brain damage in young, previously healthy patients, not just in older, sicker ones — another change from past years.

But a scientist for the Centers for Disease Control and Prevention said the federal agency has not seen any evidence that the virus is causing a different type of brain damage. He said doctors may be seeing more-serious cases this year because there are more cases overall. But he acknowledged that the CDC does not collect the granular data needed to quickly determine whether the virus is causing more-severe brain damage.

Still, Angus, who has treated West Nile patients for a decade, and Leis, who has more experience treating severe West Nile illness than perhaps any doctor in the country, both suspect the virus has changed — a view bolstered by a Texas virologist whose laboratory has found signs of genetic changes in virus collected from the Houston area.

“I’ve been struck this year that I’m seeing more patients where the brain dysfunction has been very much worse,” said Angus, of Detroit’s Henry Ford Hospital. “It makes you wonder if something’s different, if something’s changed.”

And while the virus in the past has typically invaded the brain and spinal cord only of people who have weakened immune systems, such as the elderly and transplant or cancer patients, Angus this summer treated a severely affected woman in her 20s and a man in his 40s.

Leis said he is seeing much more severe encephalitis — inflammation of the brain — than he has in the past. “It is clearly much more neuroinvasive, neurovirulent,” he said.

Four patients Leis treated this summer had lost their ability to talk or write. Another was paralyzed on one side, as often seen in strokes, not West Nile infections. Others experienced recurring seizures.

In all, 11 of the first 12 patients Leis saw this year at the Methodist Rehabilitation Center in Jackson had more severe brain damage than he had seen previously. The outlook for such patients varies, but most will

face years or a lifetime of disability.

“For the first time, we have radiographic evidence, clinical evidence of the virus attacking the higher cortical areas,” said Leis, who has published 15 scientific papers describing previous West Nile patients.

Marc Fischer, a CDC epidemiologist who tracks the West Nile virus, said the agency has not noticed the changes described by Leis and Angus. “There’s just a lot more cases this year than anybody has seen in at least 10 years,” he said. “You’re just going to see more severe cases and probably a broader variety of manifestations.”

Last month, Leis asked a Food and Drug Administration scientist who studies the genetics of the virus whether a new, more virulent strain was circulating.

“You are absolutely right . . . that new genetic variants of WNV might have appeared this year,” the scientist replied in an Oct. 23 e-mail obtained by The Washington Post. The scientist continued that “it is not easy to correlate” the new mutations with any specific type of brain damage.

Thirty minutes after Leis received the message, another e-mail from the same scientist arrived. It said the previous message had been “recalled.”

When contacted by phone, the FDA scientist, who works at the agency’s Center for Biologics Evaluation and Research, declined to discuss the messages, saying that his superiors had instructed him not to talk to reporters.

In an e-mail, FDA spokeswoman Heidi Rebello said that the agency is studying the genetics of West Nile viruses collected from 270 blood donors this year but that “it is premature for us to draw any conclusions about new genetic variants . . . or of any possible association of new genetic variants with increased virulence.”

West Nile virus, made of error-prone RNA instead of the hardier DNA found in human cells, can evolve rapidly. In 2002, a new strain appeared that quickly churns out copies of itself inside mosquitoes. This fast-replicating version swiftly replaced the earlier dominant variety.

In 2003, another genetic variant, now dubbed the Southwestern strain, appeared in New Mexico and Arizona.

The West Nile virus, first described in Uganda in 1937, arrived in New York City in 1999, killing eight in the city. Infected birds transmit the virus to mosquitoes, which then infect people, who cannot infect one another. By 2003, the virus had crossed the country.

So far this year, health authorities have reported more than 5,000 cases of West Nile illness and 228 deaths in 48 states, with Texas, California, Illinois and Michigan having the most cases. The CDC has classified about half of the illnesses as “neuroinvasive” — meaning the virus has gotten into the spinal cord or brain, causing encephalitis or other brain ailments. That’s the most dangerous type of illness caused by West Nile virus. In the other cases, patients come down with fevers or other flulike symptoms.

As of Nov. 6, there had been eight cases reported in the District this year, 45 in Maryland and 25 in Virginia, with new cases expected to plummet with the temperature.

In Texas, the state hit hardest by the epidemic, virologist Alan Barrett said samples of the virus taken from mosquitoes and birds in the Houston area show signs of genetic changes.

“This year’s virus looks more like the virus from 2002 and 2003” than the virus seen more recently, said Barrett, of the University of Texas Medical Branch in Galveston. Given that the Houston-Galveston area is a major flyway for birds, Barrett speculated that a different virus arrived in the area this year.

But it is too early to say whether this possible new strain is more virulent than those seen in years past, Barrett said. It will also take a while to study the genetics of viruses from other parts of the country. His

laboratory, one of the few studying West Nile genetics, is backlogged with samples. “We’re overwhelmed,” he said.

North Texas suffered the worst of this year’s epidemic, with 388 cases and 18 deaths in Dallas County alone. Authorities declared a public health emergency and sprayed insecticide from airplanes and helicopters for the first time since 1966.

The spraying worked, said Christopher Perkins, the county health department’s medical director. “We’re getting one or two new cases a week,” down from 20 to 30 in July and August, he said.

Neurologists in Dallas also witnessed devastating encephalitis this year, but in different areas of the brain than Leis described. Steven Vernino, a neurologist at the University of Texas Southwestern Medical Center in Dallas, said he saw damage to the lower brain stem in several patients but not to the higher language and thinking centers.

Barrett will look at the genetics of viruses from North Texas as soon he gets samples, which he expects any day. “Everybody wants to know what’s going on in Dallas,” he said.

Leis said it’s crucial to know whether the virus is mutating. “Otherwise,” he said, “we might be unprepared to deal with it in the future.”

Star Local News
community news & voices

Cases of West Nile down since spraying

Print Page

By Heather M. Goodwin, hgoodwin@starlocalnews.com

Published: Thursday, November 15, 2012 5:57 PM CST

Since opting in for the aerial spraying, less than 40 new cases of West Nile virus have been reported in Denton County - and none have been reported in Lewisville.

On Aug. 27, the Lewisville City Council members voted to opt in for the Denton County aerial spraying, which was completed Aug. 31-Sept. 2. During the Nov. 5 city council meeting, Bing Burton, director of the Denton County Health Department, presented the results of the spraying.

Councilman John Gorena, who requested the West Nile report, said there may have been other ways to combat the virus.

"I'm meaning telling our population to wear long clothes and spray ourselves," Gorena said. "I'm not saying spraying didn't do anything, I just think it was kind of a strong reaction."

Burton said in August, Denton County had the highest instance rate of West Nile in the state.

"Maybe we didn't get as many mosquitoes as we would like to have gotten, but I was delighted to see that 56 percent of mosquitoes in the spray area were killed," Burton said.

Burton also said that 60 percent of culex mosquitoes were killed. Culex mosquitoes serve as carriers of diseases, such as West Nile virus. He said West Nile cases "dropped dramatically after spraying."

"Aerial spraying was certainly a major factor, but there were other factors as well that led to the drop in September," Burton said. "There was a change in weather, change in temperature - there were a number of factors."

Burton said no other state had the numbers of West Nile cases like Texas. In Denton County there were 182 cases, Dallas County had 387 and Tarrant County had 272. Denton County sprayed 250,000 acres twice from Aug. 31-Sept. 2.

"The real heroes in this event were the doctors and hospitals who diagnosed early and kept the death rate low," Burton said.

The Lewisville City Council also tabled a variance request from Lewisville ISD. The district is asking to renew a variance granted in 2009 allowing an approximately 175 foot span of six-foot high chain link fence to remain in lieu of providing the required screening fence along the east property line of the site. The remainder of the screening fence complies with the ordinance.

The district constructed the Harmon 9th and 10th grade campus in 2010. Now, it is proposing to construct a 49,500 square foot natatorium located on the northern portion of the site, approximately 1,000 feet from the eastern property line that abuts multi-family zoning.

Claude King, city manager, said the city has sent emails and left messages with the district concerning the variance request. He said the city has been trying to reach district officials since January.

"Because LISD has chosen not to respond to our questions, we will table that item until we get some questions answered," said Mayor Dean Ueckert.

The item is set to be on Monday's city council agenda.

"We heard from district officials the very next morning after the meeting," said James Kunke, community relations and tourism director. "They've been positive, productive talks, but I'm not sure how that will affect the agenda item."

The city council will meet at 7 p.m. Monday in the council chambers, 151 W. Church St.

West Nile Outbreak Concerns Rise: Is Pesticide Spraying Safe?

Aug 17, 2012 | 2:09 PM ET | Rachael Rettner, MyHealthNewsDaily Staff Writer

Malaria is responsible for nearly 1 million deaths each year, mostly among children in sub-Saharan Africa. It is caused by a parasite and transmitted by a mosquito.

CREDIT: Dreamstime

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The use of pesticide spraying to combat mosquitoes and curb outbreaks of West Nile virus has sparked concern regarding the health effects of the chemicals used, but experts say, in these cases, the benefits of spraying far outweigh the risks.

Aerial pesticide spraying began on Thursday in Dallas, where an outbreak of West Nile virus has infected 200 people and killed 10. Pesticide spraying also recently began in parts of New York City.

There are several reasons why aerial pesticide spraying for West Nile is considered safe. For one, these sprayings use very small amounts of pesticides — much lower than the amounts used on agricultural crops, said Robert Peterson, a professor of entomology at Montana State University.

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Even if someone was outside during the spraying, "the amount of insecticides that they would be exposed to is below any amount known to cause any adverse effects," Peterson said.

Because the exposure to these pesticides is negligible, the risks to people's health are negligible, Peterson said.

During a spraying, a tiny cloud of aerosolized pesticide is released from a plane, Peterson said. The droplets are very small, and intended to fall on, and kill, mosquitoes. Even larger insects are typically not affected by the spraying, because the droplets bounce right off them, Peterson said.

In addition, the modern pesticides used in these sprayings have a very short life in the environment, and are degraded by sunlight into non-toxic chemicals, said David Savitz, an environmental epidemiologist at Brown University.

"It will kill the things you want it to, and disappear very quickly thereafter," Savitz said.

When public health is threatened, authorities must balance the risks of an action — in this case, exposure to pesticides — with the benefits — reducing the spread of West Nile, Savitz said.

In Dallas, where West Nile cases have reached a high level, authorities have made a sound judgment to use pesticide spraying, Savitz said.

To avoid direct exposure to pesticides, the New York City Department of Health recommends people stay indoors during the spraying when possible, and remove children's toys and belongings from outside.

While the chemicals used in these sprayings aren't known to be harmful to people "It's so easily avoided, it makes sense to try to avoid that possibility if you can," Savitz said.

Generally, there have been concerns over pesticide exposure for pregnant women, because it's known that the fetus is especially sensitive to environmental chemicals, Savitz said. However, there's no evidence that the chemicals used in pesticide sprayings for West Nile pose a risk to the fetus, Savitz said.

Exposure to a large amount of any substance, including pesticides, can be harmful. People exposed to large amounts of pesticides, can experience acute neurological problems, Savitz said.

Pass it on: The risks of aerial pesticides spraying on people are negligible.

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- 5 Things You Need to Know About West Nile Virus
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Information and Resources

Worst-Ever West Nile Epidemic: What Happened?

By Daniel J. DeNoon
WebMD Health News

Reviewed by Louise Chang, MD

Nov. 19, 2012 -- This week or next, 2012 officially will become America's worst year ever for death and brain damage from West Nile virus.

More than 2,600 Americans already suffer paralysis or other lingering neurological damage from the virus. At least 229 have died.

With reports still coming in and new cases continuing -- infected mosquitoes continue to bite in southern states -- the CDC expects the toll to pass 2002's record 284 deaths and 2,946 cases of neuro-invasive (brain/spinal cord) disease.

So how many people got a West Nile infection this year?

The best estimate comes from this year's 2,601-and-counting cases of neuro-invasive infection. This very serious illness puts virtually every patient in the hospital. For each such case, the CDC figures that 30 to 70 more people were infected. Some had severe fever lasting weeks; others with mild infections hardly felt ill at all.

"We estimate 78,000 to 182,000 cases so far this year," says Lyle Petersen, MD, MPH, CDC's director of vector-borne diseases. "Cases are still being reported. And states are still following up. So this year will be probably pretty close to or over the record."

As case counts rose every week this summer, they far surpassed counts for the same weeks of the large 2002 and 2003 epidemics.

Why Was 2012 a Record West Nile Season?

Birds carry West Nile virus. Mosquitoes bite the birds and then bite people. If there's enough virus in the mosquito -- that is, if conditions are right for the virus to multiply inside the insect -- people get infected.

Birds, bugs, and bites seem to happen every year. This year's big difference was the weather.

"The smoking gun is the abnormally warm spring and summer this year. In many parts of the country, it was the hottest year on record," Petersen says. "We know that when conditions are right, increasing temperatures promote virus growth in mosquitoes. This makes it easier for them to transmit the virus."

More West Nile Epidemics to Come

As temperatures drop and mosquitoes stop biting, the 2012 West Nile season finally is ending. Does this mean we've dodged the West Nile bullet?

Probably not. Some mosquito-borne diseases run in cycles in the U.S. West Nile virus arrived in New York only in 1999 and took until 2004 to spread from coast to coast -- so it's too soon to say whether natural cycles will appear.

One good thing about West Nile virus is that once you've had an infection, you're likely immune for life.

"We've never seen WNV twice in the same person," Petersen says.

It might seem that with so many infections this year, there wouldn't be many vulnerable people left to infect. But the CDC says many Americans remain vulnerable.

"Most people are still going to be susceptible to being infected in subsequent years," Petersen says. "Studies we have done in North Dakota -- the most heavily affected state since West Nile has been in the U.S. -- even there with very high incidence from year to year, only about 15% of the population has been infected. So in Texas [and other states hit hard this year], most people still are susceptible."

Stopping West Nile: Lessons Learned

There's no vaccine against West Nile virus. There's no treatment. The best advice for avoiding infection is CDC's "Fight the Bite" campaign. This means wearing protective clothing and insect repellent, avoiding the outdoors at dawn and dusk, and ridding your home of places mosquitoes breed.

One controversial measure has been the use of aerial spraying of insecticide to kill adult mosquitoes. While such spraying is considered safe, it's not totally without risk. And there have been questions about whether it works.

There's still no definitive answer to those questions. However, data strongly supporting aerial spraying comes from Texas, which saw some 30% of the nation's severe cases this year.

"In areas of Texas subjected to aerial spraying, the spray reduced the number of vector mosquitoes by more than 90%. Areas not sprayed had an increase in mosquitoes," Petersen says. "Once we analyze all the data, we expect that these control measures were effective -- probably highly effective -- in stopping the outbreak in those areas."

SOURCES:
CDC web site.
Lyle R. Petersen, MD, MPH, Director, Division of Vector-Borne Infectious Diseases, Centers for Disease Control and Prevention.
Texas Department of Health web site.

My Notes:

First Freeze Doesn't Chill West Nile Virus Worries

Experts warn that mosquitoes will be back out biting once warm temperatures return

By Kevin Cokely | Tuesday, Nov 13, 2012 | Updated 5:05 PM CST

Kevin Cokely, NBC 5 News

Experts say that even a freeze won't stop mosquitoes from biting back -- and spreading West Nile virus.

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The temperature may be dropping, but don't expect mosquitoes to drop like flies along with them.

Experts say that even biting cold won't stop mosquitoes from biting back -- and spreading West Nile virus.

"A freeze will slow them down, but it doesn't do away with them," said David Jefferson, manager of the Tarrant County health department's Environmental Health division.

Pete Esqueda of Arrow Exterminators in Addison agreed.

"If they can, they'll burrow themselves down into the ground," he said. "They'll sit there and just kind of slow their metabolism down [and] wait. Once it gets warm enough, they shoot off."

Many mosquitoes will die off in cold weather. But many others survive, mostly female mosquitoes, ready to get back to biting whenever temperatures rise.

"Just like us, once it gets nice weather, we all come rushing outside, go to the lake," Esqueda said. "These things just come out, and they look for the first thing they can find, which is a blood meal."

Spray trucks stopped running many weeks ago, but the need to protect yourself from West Nile virus still exists.

"If it's warm -- we have several warm days -- [it] wouldn't hurt to remember to use DEET as a repellent and still, always, always, all year round, get rid of the standing water," Jefferson said.

Mosquitoes that survive the cold are the ones smart enough to hang out near houses and other

buildings, where it's always a bit warmer and easier to find someone to bite.

West Nile Virus:
Click here for complete coverage of the outbreak of West Nile virus in North Texas. Find updated numbers of human cases, spraying schedules, and more FAQs about the disease.

Find this article at:
http://www.nbcdfw.com/news/health/First-Freeze-Doesnt-Chill-West-Nile-Virus-Worries-179044461.html

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Baiting Mosquitoes With Knowledge and Proven Insecticides

ScienceDaily (Nov. 13, 2012) — While one team of U.S. Department of Agriculture (USDA) scientists is testing the effectiveness of pesticides against mosquitoes, another group is learning how repellents work.

ARS entomologist Sandra Allan has found that five different classes of insecticides can be mixed with a sugar bait to attract and kill mosquitoes. (Credit: Stephen Ausmus)

At the Agricultural Research Service (ARS) Center for Medical, Agricultural and Veterinary Entomology (CMAVE) in Gainesville, Fla., entomologist Sandra Allan is using toxic sugar-based baits to lure and kill mosquitoes. Allan and her CMAVE cooperators are evaluating insecticides and designing innovative technology to fight biting insects and arthropods. ARS is USDA's principal intramural scientific research agency.

Allan studied 10 different commercial pesticides that contain additives that enable the pesticides to be dissolved in water and ingested by mosquitoes. Pesticides were combined with a sucrose solution and fed to females of three mosquito species that transmit pathogens such as West Nile virus and arboviruses. While only females feed on blood, all mosquitoes need to feed on sugar and will potentially be attracted to -- and ingest -- the toxic sugar bait.

Compounds from five different classes of insecticide-active ingredients -- pyrethroids, phenylpyroles, pyrroles, neonicotinoids and macrocyclic lactones -- were found to be toxic against all three mosquito species, *Culex quinquefasciatus*, *Anopheles quadrimaculatus* and *Aedes taeniorhynchus*.

Scientists at the ARS Henry A. Wallace Beltsville Agricultural Research Center in Beltsville, Md., are learning more about how mosquito repellents work. Entomologist Joseph Dickens and post-doctoral research associate Jonathan Bohbot found that several repellents -- DEET, 2-undecanone, IR3535 and picaridin -- affect specific odorant receptors in mosquitoes differently, thereby scrambling the insect's ability to detect chemical attractants.

In experiments, they injected frog eggs with odorant receptor genes. Molecular mechanisms within the eggs allowed these receptors to be reproduced in the outer cell membrane of the egg. Researchers then placed electrodes in the outer cell membrane and recorded electrical responses of the odorant receptors to chemical solutions.

Story Source:

The above story is reprinted from materials provided by **United States Department of Agriculture - Research, Education and Economics**. The original article was written by Sandra Avant.

Note: Materials may be edited for content and length. For further information, please contact the source cited above.

Everything Jersey

N.J. to release shrimp-like crustaceans to combat mosquito problem

Published: Thursday, June 09, 2011, 7:30 AM Updated: Thursday, June 09, 2011, 11:26 AM

By **Star-Ledger Staff**

Enlarge **Katie Colaneri/The Jersey Journal**

The battle to keep mosquitoes at bay gallery (11 photos)

State entomologists are breeding an army.

Though smaller than a thumbnail, these soldiers are the front line against this year's onslaught of mosquitoes, which is expected to be particularly bad because of the wet spring.

The troops are shrimp-like crustaceans — Macrocyclops albidus — and they have no weapons, save for their voracious appetites. Especially delicious to them are tasty tiny mosquito larvae.

Native to New Jersey, the shrimp feed on larvae in roadside ditches, small water pools, clogged downspouts and other, smaller wet areas where mosquitoes live.

They can devour up to 90 percent of larvae in a given area, said Jennifer DeSio, an entomologist who works at State Department of Agriculture's 21,000-square-foot laboratory where the crustaceans are raised.

After one of the soggiest springs in recent years, there will be plenty of bugs for them to eat, said Bob Kent, director of the state Department of Environmental Protection's Office of Mosquito Control Coordination.

"This is another environmentally friendly tool that can be used to battle mosquitoes, without having to resort to pesticides,'' Kent said.

Entomologists like DeSio will scour Mercer County's puddles and ponds looking for pregnant female crustaceans — identifiable by a little baby bump containing hundreds of eggs — which distend their bodies.

The females are taken back to the lab, where the eggs hatch. After a few generations, there are tens of thousands of soldiers ready for battle. The DEP and state Department of Agriculture have been exploring this mosquito-battling tool for several years, conducting field trials in Hunterdon, Morris, Monmouth, Ocean and Cumberland counties since 2006.

The hungry crustaceans are but one line of attack in New Jersey's skeeter war.

Mosquito-fighters will also use fish that can be deployed into bodies of water to eat the larval forms of the bugs, which are spawned in stagnant water. Using the mosquito-eating fish is the most efficient way to defeat the pests, Kent said, because it is environmentally friendly — and once a body of water is stocked, it does not have to be maintained.

The DEP will distribute between 100,000 and 300,000 fish this year, he said, depending on the severity of mosquito infestations this summer. There are several species that are used for different habitats, including Gambusia, fathead minnows, freshwater killifish and pumpkinseed sunfish. The DEP approves target spots before the fish are released.

Essex County recently stocked about 5,000 of the Gambusia species, said Eric Williges, county superintendent for mosquito control.

"We know it's been rough for residents so far, but we're working hard to get it under control," he said.

Those measures include spraying pesticides from helicopters to kill the adult mosquitoes, which fly, Williges said.

Morris County officials plan to order about 5,000 fish to stock self-contained flooded areas along the Passaic River, said Kris McMorland, the county's superintendent.

"Once you stock a location, you don't have to go back because the fish take care of the situation," McMorland said. "So that is helpful for us in terms of keeping the mosquitoes at a tolerable level."

Since March, the county has been treating the ground and spraying the air with larvacide, he said, but more rain means more waves of mosquitoes.

Spots in Morris Township and Parsippany have been approved by the DEP for the fish, he said, though authorities plan to carefully monitor additional sites in Lincoln Park and Montville.

There are several species of mosquito in New Jersey, and the type being targeted now is known as salt-marsh mosquitoes, said Deepak Matadha, an entomologist with the Middlesex County Mosquito Commission.

It takes about a week to 10 days for a mosquito to go from egg to adult, he said, so if local commissions don't take action, mosquito levels will rise each time a new batch grows, a week or so after water has accumulated from rain.

To avoid the pests, Matadha recommends using repellents with DEET and following application directions carefully; emptying all standing water from vases and birdbaths to eliminate potential breeding sites; and being careful about exposed skin during dusk and dawn, when mosquitoes are most active.

Still, the mosquito levels could improve by the end of the summer, Matadha said. If June and the beginning of July are on the dry side, the end of the summer would not be nearly as bad.

"It's difficult to project what will happen as the season progresses," he said. "Each time there is a new batch we have to go after them again, so we have to be vigilant the whole season."

By Aliza Appelbaum and Dan Goldberg / The Star-Ledger

Working out the bugs: State deploys crustaceans to control mosquito population

Published: Sunday, July 08, 2012, 7:45 AM

By **Eugene Paik/The Star-Ledger**

Jerry McCrea/The Star-Ledger

Ronnie Dumas, an inspector with the Morris County Mosquito Commission, uses a dipping stick with a cup on the end to gather water and look for mosquito larvae while out performing mosquito control in a wooded area in Morris Plains.

As summer deepens, the curse of mosquitos is once again afflicting the bodies and minds of New Jerseyans.

But once again, an army of tiny, shrimp-like creatures is being deployed to help control the perennial pests. And that army may have a wider reach than it did in 2011.

Fresh off a successful inaugural run last year, state officials have pushed for more counties to enlist the crustaceans, which have an appetite for mosquito larvae.

Morris, Bergen, Passaic and Cape May counties have received shipments of Macrocyclops albidus, the mosquito-hungry creatures also known as copepods, and a delivery to Ocean County will be made later this summer.

Four or five other counties, including Hunterdon County, may also join in, said Larry Hajna, spokesman for the state Department of Environmental Protection.

State officials have nurtured the program for about a decade, after they were inspired by a similar program in New Orleans.

Once it was learned the crustaceans could be found within New Jersey, they were harvested for tests in select counties.

The copepods are bred in a state laboratory in West Trenton, where they take six months to develop amid a mixture of distilled water, wheat seed and paramecium.

Much like the mosquito-eating fish used by the state, the copepods are used in pools of standing water that are either hard to reach or are in areas too sensitive for pesticides. They're more a preventive measure than an ultimate weapon, officials say, but they make a difference in narrowing the scale of the mosquito fight.

Never before had a state in the northeast U.S. used copepods for this purpose, said Robert Kent, administrator of the state Office of Mosquito Control. He believes New Jersey is the only state outside Louisiana to do so, though the practice is popular in countries such as Japan and Vietnam.

"Why nobody tried it before is a mystery to me," Kent said.

Jerry McCrea/The Star-Ledger

Mosquito larvae gathered in the field are reared through the larvae stage to adulthood at the offices of the Morris County Mosquito Commission to identify and catalog the more than 40 species that live in the county.

The state unrolled the program on a broad level for the first time last year, when Atlantic, Cumberland, Warren, Gloucester, Bergen, Monmouth, Cape May, Burlington, Hunterdon and Ocean counties took part.

This year, production of the copepods has been increased, already surpassing last year's total of 50,000. It's hoped to double that number in 2012, Kent said.

Morris County received 10,000 copepods this year, which were spread over two sites in Parsippany and one in Hanover, said Kristian McMorland, Morris County's superintendent of mosquito control.

Mosquitos haven't been too big a problem there this year, he said, but the copepods reduce the number of inspections county workers have to make.

The state has budgeted $35,000 to pay for the copepods, Kent said, but counties are responsible for maintaining and monitoring their sites.

That's a reason why counties such as Somerset and Middlesex counties have chosen to wait a little longer before participating.

"At this point, I don't have enough information," said Vincenzo Russo, who helps steer Somerset County's mosquito program. "We're going to wait until it's seasoned a little more."

But in the end, most of the counties could join in, like they eventually did with the state's mosquito-eating fish, Kent said.

"It's like any new program," Kent said. "You have to work out the bugs, no pun intended."

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Experimental Malaria Vaccine Disappoints, But Work Continues

Richard Knox November 9, 2012

LISTEN TO THIS STORY

A mother dresses her baby after doctors examined him during the malaria vaccine trial at the Walter Reed Project Research Center in Kombewa in Western Kenya in October 2009. (AP)

The public health world has waited for the results for more than a year. After a half-billion dollars in R&D, would the front-runner malaria vaccine protect the top-priority targets: young infants?

The results are disappointing. The vaccine — called RTS,S for its various molecular components — reduced infants' risk of malaria by about a third.

To be precise, malaria was 31 percent less frequent among 3,200 infants between 1 and 5 months old who got the vaccine compared to controls. The risk of severe malaria was 37 percent lower. The infants got three vaccine shots over three months.

The news was announced Friday at a meeting in South Africa and published online by the *New England Journal of Medicine*.

It was a letdown after the first results from the largest malaria vaccine trial — involving nearly 16,000 children in seven African countries. A year ago, researchers announced the

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vaccine lowered malaria incidence by 55 percent among children ages 5 to 17 months. The vaccine reduced their incidence of severe malaria by 47 percent.

'Disappointing'

Malaria experts hoped the efficacy would be as high in young infants. The World Health Organization's goal is a vaccine effective enough to be added to the Expanded Immunization Program for infants.

"The main issue from WHO was: Should we add this to the infant regimen?" Dr. Johanna Daily of Albert Einstein College of Medicine said in an interview with Shots. "And for this vaccine the answer is probably no.

"That's disappointing, but parasites are very good at avoiding our immune system," Daily says, "and [the malaria] parasite resides in our blood where all our immune cells and blood proteins are. So it has gotten particularly good at staying under the radar."

Moncef Slaoui, chairman of research and development for vaccine sponsor GlaxoSmithKline, expects the new results will be met with skepticism and disappointment.

"Everybody would have hoped for that number to be higher" than 31 percent, Slaoui told Shots.

But he still has a glass-half-full view of the results.

Impact

"This is the second demonstration of a significant effect of the vaccine," he says.

He points out that the vaccine reduced the incidence of malaria from 900 cases for every 1,000 infants (among those in a control group) to 640 cases.

GSK has invested $300 million in the RTS,S vaccine, and Laoui says the company expects to spend another $200 million to get the vaccine licensed. He says the company will file for licensure with the European Medicines Agency within the next few months — if it has the support of partners such as the Bill and Melinda Gates Foundation.

The main Gates-supported sponsor, the PATH Malaria Vaccine Initiative, also seems undeterred.

"Malaria is so prevalent in these African kids," Dr. David Kaslow of the MVI told Shots, "that even a modest protection translates into large public health impact, just given the sheer numbers. So [the vaccine] will reduce disease and it will save lives."

More than 200 million people get malaria each year, according to the WHO, and 655,000 people die from it. Most of the burden is in sub-Saharan Africa.

Still, Kaslow says he can't predict whether RTS,S will ever be widely deployed.

"Sitting with the data we have in hand, I can't answer that question," he says. "The honest answer is, we won't know until 2014."

Next Steps

Between now and then, RTS,S researchers will try to figure out why infants have a less robust immune response to the vaccine.

It could be one or more of several factors. Infants' immune systems are immature and less responsive to infections. Anti-malaria antibodies in their mothers' blood may interfere with infants' ability to make their own antibodies in response to the vaccine. Perhaps the administration of other infant vaccines interferes with their ability to make antibodies to malaria antigens in the vaccine.

Investigators don't know how long-lasting the vaccine's protection is, and how much difference a booster shot would make.

Beyond that, researchers want to investigate whether the vaccine's efficacy varies depending on the prevalence of malaria where they live.

Slaoui hypothesizes that the vaccine may be less effective among infants who live in areas where they are more likely to be bitten by malaria-infected mosquitoes.

"One would rationally expect when you're getting bitten three times a day by an infected mosquito, the chance that one of these would break through (the vaccine's protection) would be higher than if you have one bite per week or one per day," Slaoui says.

There was a wide range of malaria prevalence among the 11 African sites where the vaccine was tested. But so far the researchers haven't parsed vaccine efficacy by the different sites.

That could have important implications for decisions about how or whether to deploy the vaccine. An effective-enough vaccine might tip the balance in areas where malaria has been suppressed over the past 10 years by heavy investments in insecticide-treated bed nets, indoor spraying and use of effective anti-malaria drugs.

Expected Debate

Dr. John Lusingu helped test the vaccine in a district in Tanzania that used to be a high-prevalence area and now is low-to-moderate. He's disappointed the vaccine didn't lower malaria by at least half in infants. But he thinks it could still prevent malaria's high fevers, seizures, anemia and death in a lot of African children.

"Being a father from sub-Saharan Africa, I have witnessed my own children suffer from several episodes of malaria," Lusingu told Shots. "So I would highly encourage that this vaccine should be incorporated into other existing tools to control malaria."

That will be the subject of intense debate over the next few years, says Dr. Richard Feachem, director of the global health group at the University of California, San Francisco.

"Because the efficacy is disappointing and there are still some questions about the duration of its protection, those will be difficult discussions," Feachem told Shots.

The best childhood vaccines are 90 percent protective. But many argue that the perfect shouldn't be the enemy of the good-enough.

"A clear failure is a clear failure, and a 90 percent efficacy is, you know, a glass of champagne," Feachem says. "But this lies in the middle because there are so many uncertainties."

Whatever the ultimate fate of the RTS,S vaccine — or the 30 other malaria vaccines in the pipeline — Feachem says the results increase the urgency to develop new, better insecticides against malaria-infected mosquitoes, and new classes of drugs to cure those who get infected and circumvent the parasites' ability to evade the best current drugs.

More Photos

Transcript

STEVE INSKEEP, HOST:

Next, we have two reports on malaria. In the last decade, a lot of progress has been made to control that disease. But it still infects more than 200 million people per year, and kills

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around 650,000 - most of them, children. We'll look at why experts are worried about the emergence of a form of malaria in Southeast Asia, that's resistant to a commonly used drug. First, we have news about another way to control malaria - with a vaccine. NPR's Richard Knox has the latest on a study just out this morning, in the New England Journal of Medicine.

RICHARD KNOX, BYLINE: The public health world has been waiting for these results for over a year. The hope was the vaccine, tested in seven African countries, would be at least as effective in very young infants, as it proved to be in toddlers. But today's results are - well, disappointing. The vaccine reduced infants' risk of malaria by about a third. In contrast, the vaccine lowered the risk in toddlers by 50 percent, in a report released last October. But Dr. David Kaslow says a vaccine that protects only a third of infants, is enough to make a big difference.

DR. DAVID KASLOW: Malaria is so prevalent in these African kids. And so even a modest protection translates into large, public health impact. It will reduce disease, and it will save lives.

KNOX: Kaslow is director of a nonprofit called PATH Malaria Vaccine Initiative. It's funneled hundreds of millions of dollars into the vaccine study. Researcher John Lusingu helped test the vaccine in Tanzania.

JOHN LUSINGU: Being a father from sub-Saharan Africa, I have witnessed my own children suffering from several episodes of malaria.

KNOX: He's disappointed the vaccine didn't lower malaria by at least half, in infants. But he agrees with Kaslow that it could prevent high fevers, seizures, anemia and death, in a lot of African kids. Infants are considered the top priority group, partly because it was hoped malaria shots could be given to them along with other infant vaccines.

LUSINGU: So I would highly encourage that this vaccine should be incorporated into other, existing tools, to control malaria in sub-Saharan Africa.

KNOX: The question raised by the study is: How good does a malaria vaccine have to be, to justify its licensure in widespread use?

DR. MONCEF LAOUI: I think it is good enough.

KNOX: That's Dr. Moncef Laoui. He acknowledges he has a strong bias.

LAOUI: I have to tell you, I've been involved with the discovery of this vaccine from day one. I'm a co-inventor of this vaccine and therefore, I have a very strong passion, and commitment, to it.

KNOX: On top of that, Laoui is chief of research for the pharmaceutical giant GlaxoSmithKline, which has poured $300 million into developing and testing the vaccine. But he admits the new results are worse than expected.

LAOUI: I expect that the first reaction is one of a level of skepticism; a new level of disappointment with the number. I think part of that is, of course, real. Everybody would have hoped for that number to be higher.

KNOX: Still, it would be a mistake to write off the vaccine. Dr. Richard Feachem, of the University of California-San Francisco, says the best childhood vaccines are 90 percent protective; but the perfect shouldn't be the enemy of the good-enough.

DR. RICHARD FEACHEM: A clear failure is a clear failure, and a 90 percent efficacy is, you know, a glass of champagne. But this slides in the middle because there are so many uncertainties.

KNOX: Over the next couple of years, researchers will try to figure out why infants in this study didn't have a better immune response; and whether the vaccine performs better in places where malaria has been reduced by other means, such as insecticide-treated bed nets. Meanwhile, Feachem says the disappointing vaccine results increased the urgency to develop

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newer, better insecticides against malaria-infected mosquitoes; and new classes of drugs, to cure those who get infected.

Richard Knox, NPR News. Transcript provided by NPR, Copyright National Public Radio.

ORIGINAL ARTICLE

A Phase 3 Trial of RTS,S/AS01 Malaria Vaccine in African Infants

The RTS,S Clinical Trials Partnership

November 9, 2012 | DOI: 10.1056/NEJMoa1208394

Abstract | Article | References

BACKGROUND

The candidate malaria vaccine RTS,S/AS01 reduced episodes of both clinical and severe malaria in children 5 to 17 months of age by approximately 50% in an ongoing phase 3 trial. We studied infants 6 to 12 weeks of age recruited for the same trial.

Full Text of Background...

METHODS

We administered RTS,S/AS01 or a comparator vaccine to 6537 infants who were 6 to 12 weeks of age at the time of the first vaccination in conjunction with Expanded Program on Immunization (EPI) vaccines in a three-dose monthly schedule. Vaccine efficacy against the first or only episode of clinical malaria during the 12 months after vaccination, a coprimary end point, was analyzed with the use of Cox regression. Vaccine efficacy against all malaria episodes, vaccine efficacy against severe malaria, safety, and immunogenicity were also assessed.

Full Text of Methods...

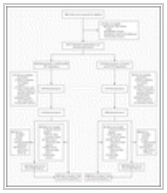
RESULTS

The incidence of the first or only episode of clinical malaria in the intention-to-treat population during the 14 months after the first dose of vaccine was 0.31 per person-year in the RTS,S/AS01 group and 0.40 per person-year in the control group, for a vaccine efficacy of 30.1% (95% confidence interval [CI], 23.6 to 36.1). Vaccine efficacy in the per-protocol population was 31.3% (97.5% CI, 23.6 to 38.3). Vaccine efficacy against severe malaria was 26.0% (95% CI, −7.4 to 48.6) in the intention-to-treat population and 36.6% (95% CI, 4.6 to 57.7) in the per-protocol population. Serious adverse events occurred with a similar frequency in the two study groups. One month after administration of the third dose of RTS,S/AS01, 99.7% of children were positive for anti-circumsporozoite antibodies, with a geometric mean titer of 209 EU per milliliter (95% CI, 197 to 222).

Full Text of Results...

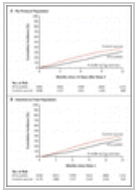
MEDIA IN THIS ARTICLE

FIGURE 1



Enrollment of Infants 6 to 12 Weeks of Age.

FIGURE 2



Cumulative Incidence of a First or Only Episode of Clinical Malaria (Primary Case Definition).

CONCLUSIONS

The RTS,S/AS01 vaccine coadministered with EPI vaccines provided modest protection against both clinical and severe malaria in young infants. (Funded by GlaxoSmithKline Biologicals and the PATH Malaria Vaccine Initiative; RTS,S ClinicalTrials.gov number, NCT00866619.)

Full Text of Discussion...

Read the Full Article...

Prop. 37: Genetic food labels loses

Stacy Finz

Updated 5:04 a.m., Wednesday, November 7, 2012

A measure that would require most foods made with genetically engineered ingredients to be labeled in California was significantly behind early Wednesday.

Supporters of Proposition 37 said consumers have a right to know whether food has been genetically altered, particularly when the long-term health impacts are unclear. Opponents argued that the labels would stigmatize foods that are scientifically proven to be safe.

With more than 94 percent the precincts reporting, voters rejected the proposed labeling law. California would have been the first state in the nation to pass such an initiative.

"We said from the beginning that the more voters learned about Prop. 37, the less they would like it," said Kathy Fairbanks, a spokeswoman for the opposition. "We didn't think they would like the lawsuits, more bureaucracy, higher costs, loopholes and exemptions. It looks like they don't."

The measure calls for genetically engineered foods to include labels on either the front or back of the product. Whole foods, such as sweet corn and salmon, would have a sign on the shelf. Products such as alcohol, beef, eggs and dairy are exempt.

"Whatever happens tonight, this is a win," said Grant Lundberg, CEO of Lundberg Family Farms, co-chair of Yes on 37. "Never before have millions of Californians come together to support giving consumers a choice about genetically engineered foods."

Opponents argued that the price of new California labels, or the cost manufacturers will incur by changing over to non-GMO ingredient, would be passed on to consumers. The No campaign calculated that households would pay as much as $400 more a year in grocery bills. But there is no independent study to show that.

Opponents, raising more than $45 million, had the backing of large agribusiness and chemical companies such as Monsanto and Dow, and food manufacturer giants, including PepsiCo. The Yes campaign raised about $6.7 million and was supported largely by the organic industry, consumer groups and alternative medicine organizations.

About 70 to 80 percent of processed foods sold in the United States are made with genetically engineered ingredients such as corn, soybeans, sugar beets and cottonseed oil. The seeds for these crops have been genetically altered in the lab to make them more resistant to pests and invasive weeds.

But proponents of Prop. 37 said research shows the risks of eating genetically engineered foods range from allergies to organ damage. They also contend that because weeds are rapidly becoming resistant to the genetic formula of these plants, more herbicides are being used.

Opponents argued that the fears are misguided. They say genetically modified crops are better suited to survive periods of bad weather and significantly increase per-acre yields, which means feeding more people for less money.

Stacy Finz is a San Francisco Chronicle staff writer. E-mail: sfinz@sfchronicle.com Twitter: @sfinz

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November 7, 2012
HUFF POST FOOD

GMOs in Food: Better Safe -- Or, at Least, Informed -- Than Sorry? (Or, Vote 'Yes' on California Proposition 37)

Posted: 11/06/2012 3:45 pm

Normally, I don't watch much television, but when the San Francisco Giants made the playoffs I made sure to catch every game. As an unfortunate but unavoidable side effect, I was exposed to many commercials and political messages. The ads -- which must have been extremely expensive ones -- were mostly insults to intelligence, but I was surprised and dismayed to see so many "No on Proposition 37" messages, some even featuring people I know and otherwise respect.

California's Proposition 37 would require labeling of some food items if they contain genetically-modified organisms (GMOs) -- making them genetically-engineered (GE) foods. Until recently it looked like a slam-dunk to pass, as it would seem difficult to argue against people having a right to know what's in what they eat -- a well-established right and practice, in fact, if you ever notice the ingredients lists on cans, bottles, and other packaging. But the barrage of opposing campaigning -- $36 million worth, by some counts -- seems to be working and support has dropped, despite very questionable and even fraudulent claims in some anti-37 material -- some which have even had to be withdrawn due to misrepresentation of facts and endorsements.

Here are a couple of things to consider (you can find much of this here as well, but this is the "yes on 37" site, so might be considered biased):

Cost: The anti-Proposition 37 campaign asserts that 37 will cost the average consumer something like $400 a year. This seemed a dubious assertion from the start, so I went looking for where it originates. The answer: Out of thin air, it seems, but pushed well-funded agribusiness interests opposed to 37. It's sad that media outlets such as newspapers have bought it, sadder still that some physicians and other presumably less-gullible professionals have as well.

For a reality check, here is a nonpartisan analysis by a professor at Emory University's School of Law. Its summary: "Consumers will likely see no increases in prices as a result of the relabeling required by the Right to Know Act."

Health: Quite a few outlandish claims have been made about GMOs, both pro and con. As is often the case, the truth is likely in the middle. GMOs will neither "save" nor destroy humanity. More hungry humans might get fed as a result of some GMOs, and be less vitamin-deficient, and that's a good thing in itself. On the other hand, there are some worrying studies, and a fair number of good, unbiased scientists who think GMOs may also turn out to increase health risks in humans in some ways. A recent study in rats had much media attention but was flawed in various ways and in any event, not enough to make any conclusive claims. But that's not enough to draw any conclusions on the broader issues. "More study is needed," as the researchers' mantra goes. Which is why even the American Medical Association has recently called for pre-market safety testing of GE foods: "Recognizing the public's interest in

the safety of bioengineered foods, the new policy also supports mandatory FDA pre-market systemic safety assessments of these foods as a preventive measure to ensure the health of the public," the AMA said in June. "We also urge the FDA to remain alert to new data on the health consequences of bioengineered foods."

After the American Association for the Advancement of Science issued a statement that labeling of GMOs is unwarranted, a group of 20 leading environmental scientists, physicians, and advocates (including this author) released a joint statement charging that the AAAS position "'ignores the broader life-cycle impacts' of genetically modified crops, in particular the safety of herbicides used to grow herbicide-resistant GM crops, and the potential spread of herbicide-resistance to other plants and weeds." A recent study about increased pesticide/herbicide use concludes, "The magnitude of increases in herbicide use on herbicide-resistant hectares has dwarfed the reduction in insecticide use on Bt crops over the past 16 years, and will continue to do so for the foreseeable future." This would be a sadly ironic effect, if true even in lesser effect, or even if the impact was neutral, as GMOs were developed at least in part to decrease such use. It is controversial, and reflects a "scientific deadlock" about the true impacts of GMOs.

However, this is no accident; as in other arenas, this "deadlock" is in fact the desired outcome of those who profit from the status quo. "Manufacturing uncertainty" is a time-tested tactic of, for example, the tobacco industry. But at this point, the fact is that we don't yet really know the broader, longer-term impacts of GMOs on human health and the environment, but there is cause for concern.

Back to Proposition 37: At this point, it's almost as much a philosophical decision as a scientific one. There are some important principles at play. One is the increasingly accepted "precautionary principle," which holds that in the face of scientific uncertainty about important issues, we err -- if indeed it is error -- on the side of safety, and wait for better scientific guidance. It's the "better safe than sorry" concept that guides our Food and Drug Administration regarding approval of medications, and what many parents have told children for centuries on a myriad of topics and risks. Another is that more modern "right to know" idea that is, again, well-established with respect to food ingredients and labeling in general, and with respect to GMOs in 67 other nations where some sort of labeling is required.

Along those lines, here's what a respected food geneticist says in supporting Proposition 37:

> "The question of whether to label genetically engineered (GE) foods, as Proposition 37 would require, is not about science. Prop 37 is about people having the right to know what's in their food and how it was produced. It's about making competition in a free market-the hallmark of capitalism-more transparent."

Or, if you like/trust famed food author Michael Pollan, who calls Proposition 37 a litmus test for democracy, here is what he says:

> "Americans have been eating genetically engineered food for 18 years, and as supporters of the technology are quick to point out, we don't seem to be dropping like flies. But they miss the point. The fight over labeling G.M. food is not foremost about food safety or environmental harm, legitimate though these questions are. The fight is about the power of Big Food. Monsanto has become the symbol of everything people dislike about industrial agriculture: corporate control of the regulatory process; lack of transparency (for consumers) and lack of choice (for farmers); an intensifying rain of pesticides on ever-expanding monocultures; and the monopolization of seeds, which is to say, of the genetic resources on which all of humanity depends."

So it is coming down to a question of who you trust more: "Big Food" and the people they convince -- or buy -- to spread their message in the interests of profit, or Consumer's Union, the American Public Health Association, California Nurses

Association, Breast Cancer Fund, Michael Pollan, and many, many others who want more truth in labeling, and consumer choice -- *your* choice.

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November 1, 2012
HUFFPOST HEALTHY LIVING

Can Genetically Engineered Foods Harm You?

Posted: 11/01/2012 8:49 am EDT

By Brooke Borel for YouBeauty.com

Genetically engineered (GE) foods are in the headlines again. Last month, a controversial French study claimed that a particular strain of GE corn causes cancer in lab rats. And in next week's election, Californians will vote on Proposition 37, which aims to require labeling for all genetically engineered foods.

So do GE foods truly pose a health threat to humans? Does mandatory GE labeling make sense? We take a look at what the science says.

The Lowdown On Genetically Engineered Foods

GE foods are often referred to as genetically modified organisms (GMOs), but that term is incorrect, explains Alison Van Eenennaam, Ph.D., a genomicist and biotechnologist at the University of California, Davis. All foods have been genetically modified in some way, whether through engineering in a lab or through traditional selective breeding in the field.

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New U.S.D.A Biobased Seal Encourages Healthier Products
Is It Organic? How to Read Labels
QUIZ: What's Your Eating Style?

GE foods have been genetically altered to give them a new, favorable characteristic, including pest or viral resistance or an improved nutrition profile. In some cases, scientists splice in DNA from another species that codes for a beneficial trait. In others, specific genes are essentially turned off so that they don't code for an unwanted trait.

For now, the only GE foods on the market are plants and microbes. Commercial GE plants include pest- or herbicide-resistant crops, such as corn and soy, and viral-resistant papaya and zucchini. GE microbes include an engineered curdling agent for vegetarian cheese to replace rennin, an enzyme that comes from the stomachs of cud-chewing animals used to make some cheeses, as well as engineered yeasts used to ferment some wines.

Today, the most widespread GE foods are pest- and herbicide-resistant crops, according to Van Eenennaam, which mainly come from multinational corporations including Monsanto, DuPont and Syngenta. And for now, these are the GE foods that have stirred up the biggest controversy.

The most common pest-resistant crops have been modified with bits of DNA from Bacillus thuringiensis (Bt), bacteria that naturally produce proteins that are fatal to a specific group of insects, including major crop pests, but are not toxic to mammals. Bt-based insecticides have been used in both conventional and organic farming since the 1950s in the U.S. and are still sprayed on crops today.

The most common herbicide-resistant plants also include major crops such as corn and soy. These have been genetically altered to withstand a specific type of herbicide (mostly Roundup Ready), which kills weeds while leaving the crop alive. Between 75 and 80 percent of processed foods in the U.S. include some sort of GE crop, according to the Grocery Manufacturers Association

What's The Worry?

When it comes to health and GE foods, most consumers are worried about the possibility of allergens or toxins. Many also have concerns over the crops' environmental impact or have a general distrust of the multinational corporations that control the crops (but those are topics for another time).

Allergic reactions and toxicity are also the main concerns both for the scientists who develop new GE foods and those who study food allergies

and toxins, explains Bruce Chassy, Ph.D., a food scientist and professor emeritus at the University of Illinois at Urbana-Champaign. All plant breeding, whether engineered or conventional, poses the risk of producing a plant that is allergenic, toxic or has other unwanted traits, according to a 2004 National Academy of Sciences (NAS) report on GE foods and human health, which ultimately determined that GE crops are safe.

But GE crops must jump through regulatory hoops that conventional crops do not, which means potentially dangerous traits are weeded out, making GE "the most researched and regulated food on the market," says Chassy.

Over the past two decades, adds Chassy, hundreds of studies have convinced major scientific societies including the NAS and the American Medical Association that GE crops "pose no new or different risks than any other crop, and there is no scientific reason to believe they would be any more risky."

Fear Of Allergies

Most consumer concerns regarding allergies center on Bt crops and whether eating proteins that the bacterial DNA produces will lead to new food allergies. It's not so far-fetched considering that most food allergies are an abnormal immune response to a protein, according to Stephen Taylor, Ph.D., a food toxicologist from the University of Nebraska-Lincoln. But as with all potential GE crops, the Bt protein was compared to an extensive database of all known allergen proteins before it was introduced to the marketplace and matched none.

Bt is also present in very small quantities in the edible parts of Bt crops and degrades after it is cooked, further decreasing the likelihood of an allergic reaction, adds Chassy. And there is no widespread evidence of Bt-related allergies over the 60 years that Bt insecticides have been used on conventional and organic crops.

The larger public health problem, according to both Chassy and Stevens, comes from a lack of public understanding of the eight major sources of food allergies, which include the most common foods in our diet: milk, egg, peanut, tree nut, fish, shellfish, wheat and soy.

Do GE Foods Cause Cancer?

In September, a new and widely-panned toxicity study claimed that Roundup Ready corn and herbicide caused tumors in rats. The paper was met with extensive criticism throughout the scientific community, including a negative review from the European Food Safety Authority. Major critiques noted that the study did not follow OECD guidelines for testing chemicals, the standard for toxicity tests; that the rats used in the test are known for developing spontaneous tumors, which means the tumors in the experiment may not have been due to the corn; and that the group failed to publish all of their data.

The paper's authors also tried to manipulate science journalists into one-sided coverage by asking them to sign confidentiality agreements in order to cover the work.

Not only was the study flawed, it was also not the first to look at the long-term effects of GE crops on animals. To date, at least 25 long-term, multigenerational animal studies have shown no evidence of GE crop toxicity, notes Marcel Kuntz, Ph.D., a biologist at the French National Center for Scientific Research, co-author of a recent literature review of the studies and author of a website that collects scientific studies on GE foods.

To Label Or Not To Label

Between the fears of allergies and toxicity, it's no surprise that some consumers want to know if the foods they're eating have been genetically modified. California's Prop 37 ballot initiative, if approved by voters, will require labels on products that have ingredients made through genetic engineering. It will also make it illegal to put labels on GE or processed foods indicating they are natural. Proponents argue it is simply a matter of the consumer's right to know what is in their food.

The Legislative Analyst's Office, a nonpartisan fiscal policy advisor to the California legislature, suggests the program's annual cost could run the state “from a few hundred thousand dollars to over $1 million” for regulation. Economists from the University of California, Davis suggest it will cost food processors an additional $1.2 billion annually (this research was, in part, funded by a group opposed to Prop 37). An industry report claims this will translate to an additional $350 to $400 in grocery bills per Californian family per year, although not everyone agrees.

Numbers and intentions aside, there is the issue of what information the labels would relay. Currently, mandatory labels provide information about the food's contents, including calories, and alerts to the presence of common food allergens. Despite Prop 37's intention to provide more information on food content to consumers, the proposed GE label provides no information about the contents of the food; rather, it focuses only on the process by which it was made.

"I don't like process-based labels," says Alan McHughen, a plant biotechnologist and geneticist from the University of California, Riverside, echoing the opinions of Van Eenennaam and Kuntz. "I fully support the current labeling regime that we have nationally where composition of the food is paramount." Even if allergens were a concern in GE foods, McHughen adds, the proposed labels wouldn't indicate which foods contain Bt crops and which contain other types of GE-made ingredients. Instead, they would simply indicate that genetically engineering was used at some point in making the product.

"Prop 37 is supposed to provide information to consumers so they can make an informed choice," says McHughen, referring to the fact that

other common food processes, including organic production, ionizing radiation or carcinogenic chemicals have no similar required labeling. "But if processed based labeling is such a good idea, why is it limited to genetically engineered food for one thing, and then only to a small set of those genetically engineered foods? If labeling for process is such a good idea, why is it so limited in scope?"

Regardless of whether genetically engineered foods will one day require labels, if you're concerned about GEs, go organic. A 2012 report from the Council on Science and Public Health, which notes no significant difference in the contents of GE versus traditional foods, points out that consumers who want to avoid GE foods already have that option by choosing foods labeled "USDA Organic."

For more on diet and nutrition, click here.

Research

Highly accessed Open Access

Impacts of genetically engineered crops on pesticide use in the U.S. -- the first sixteen years

Charles M Benbrook

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Environmental Sciences Europe 2012, **24**:24 doi:10.1186/2190-4715-24-24
Published: 28 September 2012

Abstract (provisional)

Background

Genetically engineered, herbicide-resistant and insect-resistant crops have been remarkable commercial successes in the United States. Few independent studies have calculated their impacts on pesticide use per hectare or overall pesticide use, or taken into account the impact of rapidly spreading glyphosate-resistant weeds. A model was developed to quantify by crop and year the impacts of six major transgenic pest-management traits on pesticide use in the U.S. over the 16-year period, 1996--2011: herbicide-resistant corn, soybeans, and cotton; Bacillus thuringiensis (Bt) corn targeting the European corn borer; Bt corn for corn rootworms; and Bt cotton for Lepidopteron insects.

Results

Herbicide-resistant crop technology has led to a 239 million kilogram (527 million pound) increase in herbicide use in the United States between 1996 and 2011, while Bt crops have reduced insecticide applications by 56 million kilograms (123 million pounds). Overall, pesticide use increased by an estimated 183 million kgs (404 million pounds), or about 7%.

Conclusions

Contrary to often-repeated claims that today's genetically-engineered crops have, and are reducing pesticide use, the spread of glyphosate-resistant weeds in herbicide-resistant weed management systems has brought about substantial increases in the number and volume of herbicides applied. If new genetically engineered forms of corn and soybeans tolerant of 2,4-D are approved, the volume of 2,4-D sprayed could drive herbicide usage upward by another approximate 50%. The magnitude of increases in herbicide use on herbicide-resistant hectares has dwarfed the reduction in insecticide use on Bt crops over the past 16 years, and will continue to do so for the foreseeable future.

The complete article is available as a provisional PDF. The fully formatted PDF and HTML versions are in production.

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To buy organic or not to buy organic: Both are choices that are well supported by science

Copper in Organic Foods?

- *Jack Dini* (Bio and Archives) Sunday, November 25, 2012
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Most people who buy organic produce wouldn't like the idea that they are getting quite a bit of copper (technically a heavy metal) as part of the bargain. Why copper? Because fungicides based on copper (copper hydroxide, copper sulfate pentahydrate…) are some of the limited options that an organic farmer has to control plant diseases caused by fungi and bacteria, reports Steve Savage. (1) The reason to bring this up is to point out that: 1- pesticides are used on organic foods, and 2- some of the pesticides that are used on organic are not necessarily safer than those used by 'conventional' growers.

Savage adds, "Many consumers believe that by buying organic they are eliminating exposure to pesticide residues. This erroneous concept is often encouraged by some of those who market organic products or those who advocate for organic. There is a long list (1700 products) of the materials allowed on organic published by OMRI (Organic Materials Review Institute). The pesticides on this list (including copper fungicides) are definitely real pesticides (they kill pests) and so they have to be registered for use by the EPA like any other pesticide. Copper fungicides are applied to crops frequently and at several pounds per acre each time. Many synthetic alternatives are applied at a few ounces per acre and less frequently. The best synthetic products not only generate far less hazard, their exposure is also far smaller, and they actually work better for the control of most diseases." (1)

However, don't be alarmed by mention of the word 'copper.' Copper is an essential element to all species and we would be sick without it. There are more than 10 copper-dependent enzymes which are required by all cells to produce energy. Also, there is little danger that our diet does not provide enough copper, because it is abundant in certain foods. Those with the most copper are sea-foods—oysters, crab and lobster; among meats it is land, duck, pork, and beef which have the most copper; the liver and kidneys of land and beef are especially rich in copper. The plant-derived foods with most copper are almonds, walnuts, Brazil nuts, sunflower seeds, mushrooms and bran. (2)

So, a miniscule amount of copper on organic produce is a non-issue. Even though the copper fungicides are toxic

to mammals at certain doses, the EPA risk analysis finds there is an acceptable margin between how much we can get from eating the produce and how much it would take to actually hurt us.

The EPA defines a range of toxicity categories from I to IV, with IV being the least toxic (essentially non-toxic to mammals, but their terminology is classic regulatory-cautious.) On a weight basis, the largest share of pesticides used in California in 2010 (62%) fall into the least toxic category. Since it's not easy for most people to relate to the EPA category descriptions, Steve Savage provides comparisons between pesticides and familiar chemicals in foods and pharmaceuticals: "Vitamin C is something which many people take in large, 250-1000 mg doses on a regular basis. Fifty-five percent (55%) of pesticides used in California in 2010 were less toxic than Vitamin C. Sixty-four (64%) were less toxic than Vitamin A. Seventy-one percent (71%) were less toxic than vanilla ice cream or lattes. Seventy-six percent (76%) of the pesticides were less toxic than ibuprofen in products like Advil. Ninety-seven (97%) of California pesticide use in 2010 was with products that are less toxic than the caffeine in our daily coffee, the aspirin many take regularly, or the capsaicin in hot sauces or curries. This is not the sort of image that most people visualize when they hear the word 'pesticide.'" (3)

One last item about pesticides is this: we get much more natural pesticides than synthetic pesticides in our diet and this has nothing to do with 'conventional' or 'organic' growers. Bruce Ames and his colleagues at the University of California, Berkeley, report that about 99.99 percent of all pesticides in the human diet are natural pesticides from plants. All plants produce toxins to protect themselves against fungi, insects and animal predators such as man. Tens of thousands of these natural pesticides have been discovered, and every species of plant contains its own set of different toxins.

When plants are stressed or damaged (such as during a pest attack), they increase their levels of natural pesticides manifold, occasionally to levels that are acutely toxic to humans. Ames estimates that Americans eat about 1,500 mg per person per day of natural pesticides and that a person annually ingests about 5,000 to 10,000 different natural pesticides and their breakdown products. (4)

The bottom line in the pesticide argument for buying organic is not compelling in a modern time-frame. If someone wants to spend the extra money for organic, that is their choice. Someone who does not want to buy organic should feel neither guilt nor fear about that decision. Both are choices that are well supported by science. (3)

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Jack Dini, Livermore, CA, writes a monthly column on science and environmental issues for Plating & Surface Finishing and also writes for other publications. He is the author of Challenging Environmental Mythology (2003). Jack can be reached at: jdini@comcast.net

U.S. NEWS | November 9, 2012, 9:13 p.m. ET

Organic Food To Be Tested Periodically

By BILL TOMSON

The U.S. Department of Agriculture said Friday it would require periodic tests of organic food starting next year to help ensure producers aren't using prohibited pesticides, genetically modified organisms or other nonorganic substances.

Under existing USDA regulations, organic-food producers must get an initial inspection before being certified to produce organic food. But the agency hasn't required they get continued periodic testing to ensure their products remain free of nonorganic material.

The USDA is mandating that agents test annually a minimum of 5% of the farms or production facilities they are contracted to monitor. That is enough testing, the USDA said, to discourage use of prohibited substances without raising costs to the organic industry. It will be up to the private, USDA-accredited agents as to which facilities to test.

The agency said the new testing requirements will protect the integrity of the industry by discouraging the mislabeling of organic food that consumers buy.

The 1990 law that ordered the creation of organic standards called for periodic testing of foods labeled organic, but regulators never put in place clear rules to do so, according to a 2010 inspector-general report. The USDA didn't put in place rules governing organic-food production until 2002, and only a small number of private agencies have been performing routine testing since then, mostly on a voluntary basis.

Organic standards in the U.S. are contained in a complex set of regulations that differ by product, but generally they ban many pesticides and other synthetic substances. Organic farmers aren't allowed to use the genetically modified organisms that are in most of the corn and soybeans grown in the U.S.

The Organic Trade Association, a group representing organic-food producers in

the U.S. and Canada, said the rules would help certifying agents identify and take enforcement action against farms and businesses intentionally using prohibited substances or methods.

Write to Bill Tomson at bill.tomson@dowjones.com

A version of this article appeared November 10, 2012, on page A4 in the U.S. edition of The Wall Street Journal, with the headline: Organic-Food Facilities To Face Periodic Testing.

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Bloomberg

Organic Holiday Fare to Face Pesticide Test Purists Call Flawed

By Andrew Zajac - Nov 21, 2012

A new rule designed to bolster the integrity of next year's organic holiday food by testing for pesticides and contaminants may be too weak to be effective.

Beginning in January, food inspectors in the U.S. will be required to test for at least one prohibited substance in one out of every 20 certified farms and processing facilities certified as organic by the U.S. Department of Agriculture.

Decisions about the products and facilities to be tested will be made by outside inspectors, who are paid by farmers and processors for organic certifications. Because the USDA doesn't plan to track the results, consumers -- who pay a premium for food assumed to be free of additives -- will be less able to make informed judgments about food purity, according to Charles Benbrook, member of a USDA advisory committee on biotechnology and agriculture.

"It's an opportunity missed to do something meaningful on the question of pesticides in organic food and food in general," said Benbrook, a research professor at Washington State University's Center for Sustaining Agriculture and Natural Resources. "It's the kind of behavior you might expect from an accountant who's trying to protect the client rather than solve a problem."

The flaws in the testing regime reveal the challenges faced by the USDA's National Organic Program, which serves as both a booster of organic food and a guarantor of standards in the sector. The program relies on third-party certifiers -- an amalgamation of private businesses, non-profits and local government agencies -- to make sure that farmers and food processors follow record-keeping and production requirements needed to meet organic standards.

USDA Report

Most certifiers currently don't survey farms for pesticides or other substances prohibited by the organic program, which include genetically modified foodstuffs, synthetic hormones, arsenic, herbicides and antibiotics, unless there are suspicions of non-compliance.

The rollout of the new testing mandate was triggered by a 2010 USDA report which found that none of four certifiers visited by agency investigators conducted periodic testing on the approximately 5,000 farms and processing facilities they inspected, even though such tests are required by a 1990 law governing the production of organic food.

“There was no assurance that certifying agents performed regular periodic testing at any of the approximately 28,000 certified organic operations worldwide,” the report found. “Without such testing, the potential exists that an operation’s products may contain substances that are prohibited for use in organic products.”

Worldwide Scope

The number of operations worldwide covered by the testing requirement has increased to more than 30,000 since the report was issued. About 60 percent of those facilities are in the U.S.

The new inspection rule was published “to further ensure organic integrity,” Soo Kim, a spokeswoman for USDA’s Agricultural Marketing Service, which includes the NOP, said in an e-mailed statement. “It wasn’t the intent to draw broad- based conclusions across specific commodities or category of products.”

The organic industry is among the fastest growing segments in agriculture. Sales in 2011 totaled $29.2 billion, compared with $6.1 billion in 2000, and now account for about 4.2 percent of all food sales, according to the Brattleboro, Vermont-based Organic Trade Association.

Purity Premium

Consumers typically pay 30 percent to 100 percent more for organic food products, according to David Sprinkle, publisher of Packaged Facts, a Rockville, Maryland-based market research service.

The USDA estimates that tests of soil, water, waste or food products will cost about $500 each, with a total annual expense to certifiers of $750,000.

The testing program invites conflict of interest because certifiers who have a financial relationship with producers pick which operations and products will be examined as well as which prohibited substances will be tested for, said Arthur Harvey, a blueberry farmer in Hartford, Maine. In 2005, Harvey successfully sued the USDA to restrict synthetic additives in organic food.

“Do you think they’re going to test the ones that are doubtful?” Harvey said. “The selection of which products to test should be done by an independent consumer group.”

There’s also a danger that certifiers will test for items they know aren’t present, he said.

“That’s such a tiny, tiny program,” Harvey said. “It’s as minimal as it’s possible to make it.”

Client Base

Patricia Kane, coordinator of the Port Crane, New York- based Accredited Certifiers Association Inc., a trade group for organic inspection companies, said the growth of the industry blunts concerns about certifiers being motivated to look the other way out of concerns they’ll lose business.

“There’s no shortage of clients,” said Kane, whose group represents 44 of 93 certifiers accredited by the USDA.

The rule requires inspectors to report pesticide residues or other contaminants that exceed limits set by the Food and Drug Administration and the Environmental Protection Agency to federal authorities.

Otherwise, inspectors are supposed to keep results on file, available to the public if requested.

By failing to collect the data, the agriculture department is falling short of its obligation to continuously improve organic standards, Benbrook said.

“It’s going to be more difficult to focus on the few residues that do pose risk,” Benbrook said. “Organic food is far safer from when it comes to pesticide levels, but USDA still owes it to the organic consumer to do everything it can to bring the risk level as close to zero as possible.”

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For Immediate Release

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Three Leading Scientific Societies Take an Objective Look at the Issues Associated with "Least Toxic Pesticides" Applied as a "Last Resort"

LAWRENCE, Kansas – November 12, 2012 – Recommendations and decisions to use "least toxic pesticides" and "pesticides as a last resort" have flourished in the last decade, but according to three scientific organizations – the Weed Science Society of America (WSSA), the American Phytopathological Society (APS) and the Plant-Insect Ecosystems Section of the Entomological Society of America (P-IE ESA) – these are not the correct approaches to the pesticide component of an Integrated Pest Management (IPM) program.

The three organizations have joined to take an objective look at the problems associated with "least toxic pesticides" applied as a "last resort" and today issued the following statement:

IPM is Fundamental Wherever Pests Must Be Controlled

It is essential to practice IPM, whether managing weeds, insect pests or plant diseases – on the farm, on commercial sites, on public lands, or in or around the home. Key components of IPM include making the habitat unfavorable for pests, excluding pests where feasible, using proper

sanitation practices, monitoring the infestation level, knowing the pest tolerance level for the specific situation and implementing the necessary management practices.

Judicious use of pesticides is a critical component of many IPM programs. Judicious (careful) use refers to various practices – following all label directions and making all appropriate stewardship decisions required in the particular situation. This includes applying a product registered for the target pest(s) *after* accurate pest identification, and consideration of the level of infestation and the potential for economic, health or other negative pest impacts. Careful use extends beyond pesticides to household chemicals, automobiles, medicines, alcoholic beverages, and countless other products that are part of our daily lives.

The Problem with Selecting Only "Least Toxic Pesticides"

- ✓ "Least toxic" implies there are pesticides available for every pest spectrum that are least toxic to everything else. This is not true. The toxicity of a pesticide depends on what is being evaluated – short-term or long-term toxicity – and who or what may be affected (e.g. applicators, farmworkers, livestock, wildlife, pets, birds, fish, beneficial insects, earthworms, sediment-dwelling organisms, crops). It is also important to remember that toxicity is not the same as risk, which is dependent on both toxicity and exposure.

- ✓ The risk associated with the use of pesticides and other chemicals is managed by establishing safe exposure levels based on the toxicity specific to each product. Assigning a "most" or "least" toxic rating does not equate to actual risk when the product is **properly** applied. For example, the label of a pesticide product that may cause skin irritation will also contain requirements for personal protective equipment that safeguards the skin, while a product that may affect fish will contain use directions, precautions and possibly even restrictions intended to protect fish. This is why the EPA-approved label instructions **must** be followed.

- ✓ All pesticides – including those referred to as "least toxic," "organic" and "natural" – are toxic to one or more pests and possibly humans and other organisms as well. Use of these terms can lead to false security regarding the need for careful handling of pesticides and proper environmental stewardship.

- ✓ Over-reliance on a "least toxic" pesticide can cause new problems. For example, glyphosate is considered a "least toxic" herbicide choice, but overreliance on it has led to significant weed resistance problems. Over-use or misuse of **any** pest management tactic can cause problems – for example, cultivation to control weeds on hilly land can

cause soil erosion, and excessive hand-hoeing can cause back injuries and increase the risk of skin cancer.

- ✓ Often, "least toxic" products do not work as well on the pest(s), leading to the need for re-treatment with another pesticide on larger and/or harder-to-control pest infestations. This can result in higher costs, reduced control and undesirable environmental effects attributable to the pest.

The Problem with Using Pesticides Only "As a Last Resort"

- ✓ "Last resort" implies that pesticides will work as well when every non-chemical control technique is attempted first. However, delaying application of a pesticide can cause buildup of the pest(s) in crops, gardens, buildings and other sites, with negative impacts on yield, quality and/or health. In fact, delaying treatment can significantly increase the ecological and economic damage to crop and non-crop areas.

- ✓ Using pesticides as the last line of defense can result in a more limited choice of pesticides, as well as reduced crop tolerance, the need for higher rates, and less effective control because of higher infestation levels and/or more tolerant pest stages. For example, seedling weeds and early-stage insect larvae and diseases are usually more easily controlled than later pest stages.

- ✓ Effective pesticide choices, when they are applied as a "last resort," means fewer options to rotate pesticides, which is a critical step in preventing a pest from becoming resistant to a pesticide. "Last resort" pesticide strategies may also increase the need for multiple products and higher application rates to control the pest effectively.

- ✓ "Last resort" suggests pesticides are always the worst choice, which is not true. First using non-chemical techniques that are ineffective or inefficient has the potential to add to the cost of pest management, intensify the pest problem or create new problems.

- ✓ Branding pesticides as the "last resort" choice certainly does not stimulate a strong public interest in funding education on their proper use. Pesticides are widely used, but discretionary federal funding of the U.S. Pesticide Safety Education Program has been eliminated in 2011 and 2012. This program is vital to educate pesticide users and dealers who must be certified to apply or sell pesticides, and to teach the public how to use pesticides safely.

There is no benefit or scientific basis to simplistic messages like “use least toxic pesticides as a last resort” for the large number of pesticide users who apply pesticides according to the label and practice good stewardship. Nor are these messages beneficial for those who neither seek training nor adequately read the label – believing instead that it is safe, practical, and effective to simply choose a product considered a “least toxic pesticide” and apply it only as a “last resort.” These messages hinder pesticide safety and stewardship education and practices that are in the best interest of the pesticide user, our food supply, public health and ecosystem preservation.

The WSSA, APS and P-IE ESA do not promote the use of pesticides above other pest management techniques. Pesticides should ONLY be used when needed, when risks to non-target organisms and habitats have been carefully considered, and when diligent attention will be given to following all label directions and other applicable laws. In addition, general and product-specific stewardship must always be practiced to prevent undesired effects under the particular application conditions.

Pesticides are an important component of many IPM programs for a variety of reasons. A fungicide, for example, may prevent disease, have curative effects, induce plant resistance to disease or promote plant health and yield. The most important message is to follow the label – the entire label, including all safety and other precautions – and practice good stewardship. Suggesting that only “least toxic pesticides” be used, as a “last resort,” ignores the extensive research, regulatory, educational and stewardship efforts that make important pesticide tools available and define their proper and safe use in Integrated Pest Management programs.

Societies Renew Their Endorsement of IPM Definition in USDA “National Road Map for Integrated Pest Management”

No pest management-related term has been defined in so many different ways as “Integrated Pest Management.” WSSA, APS and P-IE ESA strongly oppose a non-scientific approach to IPM and re-endorse the USDA National Road Map definition:

“Integrated Pest Management, or IPM, is a long-standing, science-based, decision-making process that identifies and reduces risks from pests and pest management related strategies. It coordinates the use of pest biology, environmental information and available technology to prevent unacceptable levels of pest damage by the most economical means, while posing the least possible risk to people, property, resources and the environment. IPM provides an effective strategy for managing pests in all arenas, from developed agricultural, residential, and public areas to wild lands. IPM serves as an umbrella to provide an effective, all encompassing, low-risk approach to protect resources and people from pests.” USDA National Road Map for Integrated Pest Management

Real examples of the risks when pesticides are used only as a "last resort" and the benefits of using appropriately timed pesticides as part of an integrated pest management program, as well as common questions and answers, are available online.

About the Weed Science Society of America
The Weed Science Society of America, a nonprofit scientific society, was founded in 1956 to encourage and promote the development of knowledge concerning weeds and their impact on the environment. The Weed Science Society of America promotes research, education and extension outreach activities related to weeds, provides science-based information to the public and policy makers, fosters awareness of weeds and their impact on managed and natural ecosystems, and promotes cooperation among weed science organizations across the nation and around the world. For more information, visit www.wssa.net.

About the American Phytopathological Society
The American Phytopathological Society (APS) is a nonprofit, professional scientific organization. The research of the organization's more than 5,000 worldwide members advances the understanding of the science of plant pathology and its application to plant health. For more information, visit www.apsnet.org.

About the Entomological Society of America
The Entomological Society of America (ESA) is the largest organization in the world serving the professional and scientific needs of entomologists and people in related disciplines. Founded in 1889, ESA today has more than 6,000 members affiliated with educational institutions, health agencies, private industry and government. Members are researchers, teachers, extension service personnel, administrators, marketing representatives, research technicians, consultants, students and hobbyists. For more information, visit www.entsoc.org.

KINGDOM

Ministry ban on toxic pesticides likely to boost organic farming

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Amal Al-Sibai
Saudi Gazette

JEDDAH — Fahd Balghunaim, Minister of Agriculture, has said the ministry recently banned 30 different agricultural pesticides after research has pointed to the dangers that these harmful chemicals pose to public health.

Despite intense objections and pressure from the manufacturers and importers of these toxic compounds, the ministry remained steadfast in its decision to ban their use as pesticides on crops intended for human consumption.

The Ministry of Agriculture has established a center with a total expenditure of SR70 million to promote organic agricultural methods throughout the Kingdom. Alternative organic farming methods will replace the conventional farming methods that rely heavily on chemical fertilizers and artificial pesticides, which have been linked to many types of cancers, neurological disorders, and hormone disruptions.

Funds will be directed toward breeding beneficial and farmer friendly insects and using them on crops to combat and kill pests that destroy crops, but in a natural, safe and non-toxic way. Natural methods of pest control are actually less expensive than buying potent pesticides, and it is safer for the crops, human health, wildlife and the environment.

Organic farming strictly limits the use of artificial fertilizers, pesticides, plant growth regulators such as hormones, antibiotics in livestock feed, food additives, and genetically modified organisms.

Balghunaim stressed that the Ministry of Agriculture is working diligently to stop the spraying of crops and fresh produce with toxic chemical pesticides. He said that each farmer must understand the grave responsibility of providing society with a safe source of nutrition and that the farmer will be held accountable by Allah if he applies dangerous chemicals on crops.

Organic farmers promote biodiversity by growing a variety of crops, rather than one single crop. Through several methods, organic farmers prevent soil erosion, improve soil fertility, conserve energy and help protect local wildlife and runoff water. Organic farming can greatly reduce the amount of greenhouse gases, such as carbon dioxide, that contribute to global warming.

Organic farming may be slower, harder, more complex and more labor-intensive but for the sake of the community's health and the global environment, it is worth the extra effort.

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