Maine Board of Pesticides Control

Miscellaneous Pesticides Articles
June 2014

(identified by Google alerts or submitted by individuals)

Pesticide illness cases show jump this spring in Washington

By Kate Prengaman of the Yakima Herald-Reupblic

- As of Tuesday, May 13, 2014

The state Department of Heath reports a spike in pesticide-related illnesses, with 60 people becoming ill so far this spring.

In total, 15 pesticide exposure events have been reported to the Health Department in the past two months, which is as many as the agency normally sees in a year, spokeswoman Kelly Stowe said.

All of the recent cases have occurred in Eastern Washington in counties with lots of orchards. Most of the exposures are believed to be the result of pesticide drift — when the chemical spray drifts away due to wind or improper application.

Stowe said the two cases reported in Yakima County both affected one person. In one case, an employee was exposed and had to be taken to the emergency room for treatment. In another, a bystander was exposed, but declined medical attention.

The state Department of Agriculture is investigating 13 drift complaints as of Monday, including nine that affected two or more people, said spokesman Hector Castro. The highest number of cases are in Grant and Chelan counties, he added.

“To see this many pesticide drift cases this early in the season is a concern,” Castro said.

The Agriculture Department is only investigating one case in Yakima, in which a fungicide drifted from an orchard onto a neighboring residence. Joel Kangiser, Pesticide Compliance Program manager, said it could be the same situation that the Health Department referred to as the bystander case, but he couldn’t be sure.

Incidents that involve workers exposed on the job are investigated by the Department of Labor and Industries, Kangiser added, which typically explains the difference in case numbers.

His office also issued a news release Monday reminding pesticide applicators to take steps to reduce drift and exposure.

Such steps include evaluating the winds, ensuring that all workers are wearing protective equipment and scouting adjacent areas to make sure no one is in the treatment zone.

Last year, the Agriculture Department pursued enforcement actions in 28 cases of pesticide drift.

Using beekeepers' real world experience to solve beekeepers' real world problems
Be Included, Be Involved, Bee Informed

Colony Loss 2013-2014

Posted on **May 15, 2014** by **The Bee Informed Team**

Preliminary Results: Honey Bee Colony Losses in the United States, 2013-2014

May 6, 2014

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Note: *This is a preliminary analysis. A more detailed final report is being prepared for publication at a later date.*

The Bee Informed Partnership (http://beeinformed.org), in collaboration with the Apiary Inspectors of America (AIA) and the United States Department of Agriculture (USDA), is releasing preliminary results for the eighth annual national survey of honey bee colony losses. For the 2013/2014 winter season, 7,183 beekeepers in the United States (U.S.) responded. Collectively, they managed 564,522 colonies in October 2013, 21.7% of the country's 2.6 million colonies.

For the winter of 2013/14, 23.2% of managed honey bee colonies in the U.S. died. Nearly two-thirds of the respondents (65.4%) experienced winter colony loss rates greater than the average self-reported acceptable winter mortality rate of 18.9%. The 2013/14 winter colony loss rate of 23.2% is 7.3 points (or 23.9%) lower than the previous years' (2012/13) estimate of 30.5% loss. (Figure 1) and is notably lower than the 8-year average total loss of 29.6% .

Preliminary results for the 2013/14 survey indicate that 20.0% of all colonies managed between April 1 2013 and Oct 1 2013 died. Responding beekeepers who managed bees over the entire April 2013 – April 2014 survey period reported losing 34.2% of the 670,568 colonies managed over this period. The annual loss differs from the sum of summer and winter losses reported above because the respondent pool differed as only respondents who reported for both the summer and winter period are included in the annual loss rate calculation.

The 2012/13 survey expanded beyond only winter mortality estimates to improve our understanding of colony losses by also reporting on summer and annual colony mortality rates. Results from the 2012/13 survey indicated that that summer colony losses (between April 1 2012 and Oct 1 2012) were 25.3%. Loss estimate for the 12-month period (between April 1, 2012 and March 30, 2013) was 45.2%.

This survey was conducted by the Bee Informed Partnership, which receives a majority of its funding from the National Institute of Food and Agriculture, USDA (award number: 2011-67007-20017).

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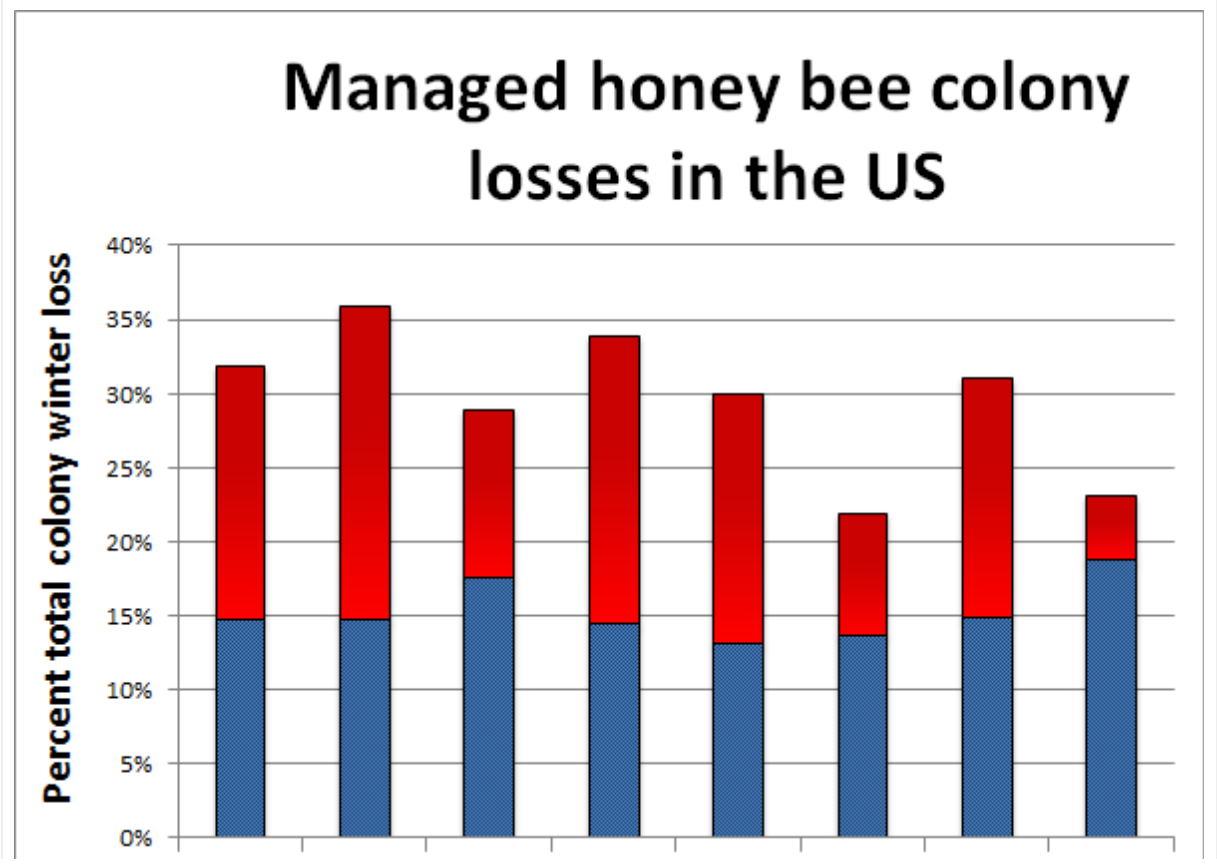
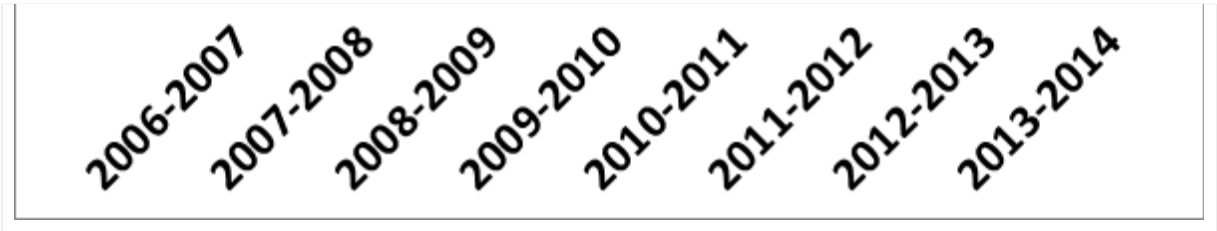
1. Based on NASS Honey report 2013 figures

2. Previous survey results found a total colony loss in the winters of 30.5% in the winter of 2012/2013, 21.9% in 2011/2012, 30% in 2010/2011, 34% in 2009/2010, 29% in 2008/2009, 36% in 2007/2008, and 32% in 2006/2007 (see figure attached)





– Figure 1: Summary of the total overwinter colony loss (October 1 – April 1) of managed honey bee colonies in the US across the 8 annual national surveys (red bars). The acceptable range (blue bars) is the average percentage of acceptable loss declared by the survey participants in each of the 8 years of the survey.

This entry was posted in **Results**, **Winter Loss Survey** by **The Bee Informed Team**. Bookmark the **permalink [http://beeinformed.org/2014/05/colony-loss-2013-2014/]** .

About The Bee Informed Team

The Bee Informed Team regularly announces the release of results at beeinformed.org. The Bee Informed Partnership is an extension project that endeavors to decrease the number of honey bee colonies that die over the winter. A listing of all project activity reports can be found at the **'Results'** tab above. Results published in scientific journals are included as links. Who's on the team? See the **Team Pages.**

View all posts by The Bee Informed Team →

7 THOUGHTS ON “COLONY LOSS 2013-2014”

Pingback: Updates: news releases and updates

Pingback: Bee losses highlight urgent need to restrict pesticides, shift to sustainable agriculture | Ecocide Alert

Pingback: Yearly Survey Shows Better Results for Pollinators, but Losses Remain Significant | AgNetWest

Pingback: Honeybee deaths went down last winter, survey finds | Science Recorder

Don Blume

on **May 15, 2014 at 5:40 pm** said:

It would be helpful to see a state-by-state or at least a regional breakdown of the data. Obviously, hives in the southwestern US this past winter were exposed to dramatically different conditions than here in Connecticut, where I am a suburban gardener.

I have yet to see a honeybee in my garden this year, and have been fortunate that queen bumblebees are present and have been busy as bees.

One more thought: what do we know about residential uses of neonicotinoids like Imidicloprid in lawn and garden products? Garden centers, Home Depots and Lowes in this area push these products hard.

D.B.

Pingback: US bee losses drop but not far enough : Nature News Blog

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Sub-lethal exposure to neonicotinoids impaired honey bees winterization before proceeding to colony collapse disorder

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Abstract

Honey bee (*Apis mellifera* L.) colony collapse disorder (CCD) that appeared in 2005/2006 still lingers in many parts of the world. Here we show that sub-lethal exposure of neonicotinoids, imidacloprid or clothianidin, affected the winterization of healthy colonies that subsequently leads to CCD. We found honey bees in both control and neonicotinoid-treated groups progressed almost identically through the summer and fall seasons and observed no acute morbidity or mortality in either group until the end of winter. Bees from six of the twelve neonicotinoid-treated colonies had abandoned their hives, and were eventually dead with symptoms resembling CCD. However, we observed a complete opposite phenomenon in the control colonies in which instead of abandonment, they were re-populated quickly with new emerging bees. Only one of the six control colonies was lost due to *Nosema*-like infection. The observations from this study may help to elucidate the mechanisms by which sub-lethal neonicotinoids exposure caused honey bees to vanish from their hives.

Key words: colony collapse disorder, CCD, honey bee, neonicotinoids, imidacloprid, clothianidin.

Introduction

Since its emergence in 2005/2006, the continuing significant losses of honey bees (*Apis mellifera* L.) colonies resulting from the symptomatic disease of colony collapse disorder (CCD) has demonstrated our inability to identify and eradicate the responsible cause(s) of CCD (BBC News, 2013; The New York Times, 2013; vanEngelsdorp *et al*., 2008). While the prevailing opinions suggest the linkage of CCD to multi-factorial causes including pathogen infestation, beekeeping practices (including malnutrition), and pesticide exposure in general (Cox-Foster *et al*., 2007; Blanchard *et al*., 2008; Higes *et al*., 2008; vanEngelsdorp *et al*., 2009; Alaux *et al*., 2010; de Miranda *et al*., 2010; Williams *et al*., 2010; Di Prisco *et al*., 2011; Vidau *et al*., 2011; USDA, 2013), this notion ignores the differential mortality symptoms; in particular hive abandonment in CCD *vs.* diseased colonies. However, recent scientific findings linking CCD with exposure to neonicotinoids, a group of systemic insecticides, appear to be gaining traction (Maini *et al*., 2010; Pareja *et al*., 2011; Lu *et al*., 2012; Farooqui, 2013; Matsumoto, 2013) and have led to new regulatory control (Erickson, 2012). In this study, we extend our previous study (Lu *et al*., 2012) showing that sub-lethal exposure of imidacloprid and clothianidin affected the winterization of healthy honey bee colonies that subsequently leads to CCD.

Materials and methods

In order to investigate the detrimental effects of sub-lethal neonicotinoid exposure in healthy honey bee colonies, we utilized the split-plot lifecycle study design in which honey bees are fed with pre-determined known amounts of neonicotinoids and allowed to freely forage in the environment. We then assessed their hive growth and strength, as well as their mortality and morbidity, throughout the lifecycle including multiple worker bee generations. The setup and management of eighteen study colonies (using 10-frame Langstroth pine hive) in three apiaries in central Massachusetts was identical to that previously described (Lu *et al*., 2012). At each apiary, we separated six colonies into two groups in which honey bees were fed with either sucrose water or high-fructose corn syrup (HFCS) over the study period. Each sugar group consisted of two neonicotinoid-treated and one control colonies replicated in each of the three apiaries. We purchased sucrose from a local food store and HFCS from a beverage company. Both sugar waters made of sucrose and HFCS were analyzed prior to be used in the experiment and found non-detectable residues of neonicotinoids using a published method (Chen *et al*., 2013). Starting from July 2nd 2012, we administered 258 µg of imidacloprid (1-(6-chloro-3-pyridinyl) methyl)-N-nitro-2-imidazolidinimine, CAS# 138261-41-3) or clothianidin (1-(2-chloro-1,3-thiazole-5-ylmethyl)-3-methyl-2-nitroguanidine, CAS# 210880-92-5) in 1.9 liter (0.5 gallon) of sucrose water and HFCS to the treated colonies each week, respectively, for thirteen consecutive weeks ending on September 17th 2012. Assuming each colony consisted of 50,000 bees at any given day in spring and summer, we administered 0.74 ng/bee/day of either imidacloprid or clothianidin to treated hives for 13 consecutive weeks. This dosage is far below the oral LD50 of 3.4 and 118.7 ng/bee for clothianidin and imidacloprid, respectively (Laurino *et al*., 2013). Control colonies were given neonicotinoid-free sucrose or HFCS throughout the experimental period. Sugar water (both types) was completely consumed by each colony at the end of each week during the 13-week neonicotinoids administration.

From June 29th to September 24th 2012, we assessed

the brood rearing production of all colonies on a bi-weekly basis using a modified brood assessment method as previously described (Lu *et al*., 2012). In brief, the 20-frames in each hive were scored cumulatively for the area covered by "sealed brood" which is the pupal stage of honey bee development. Brood was estimated by dividing the face of each side of frame into 32 squares (each square containing approximately 100 cells). All 20 frames in each hive were scored by visually estimating the number of squares of capped brood per frame face. All colonies were treated with Miteaway Quick strips for controlling *Varroa* mite on August 13th 2012, followed by Apistan strips from October 1st to November 15th 2012. The *Varroa* mite counts were assessed twice using the common alcohol wash method on August 13th (pre-Miteaway application) and August 22nd (post-Miteaway application). In addition, colonies were treated with Fumagillan-B [9.1 g dissolved in 7.6 liters (two gallons) of sucrose or HFCS] in early October 2012 to control *N. apis* and *N. ceranae*, two common intestinal parasites. Entrance reducers were installed before the hives were ready for winterization.

All colonies were monitored weekly beginning on late October 2012. Notes were taken on the size of the clusters observed by counting the numbers of frames containing honey bees from the top of the hive in which it generally took no more than 10 seconds. Starting from November 2012, hives were supplemented either with crystallized HFCS or with granular sucrose mixed into a thick water paste. The food was placed on waxed paper on top of the frames inside the inner covers. Data were analyzed using SPSS Statistics (version 20.0).

Results

We found honey bee colonies in both control and neonicotinoid-treated groups progressed almost identically, and observed no acute morbidity or mortality in either group until the arrival of winter. In addition, neither the locations where the hives were set up nor the type of sugar (high-fructose corn syrup vs. sucrose) fed to honey bees was associated with the brood rearing or the occurrence of CCD (one-way ANOVA). Therefore data from 3 apiary locations and two types of sugar were pooled in the data analysis. As temperatures began to decrease in late October 2012, we observed a steady decrease of bee cluster size in both control and neonicotinoid-treated colonies. While such decline was quickly reversed in the control colonies in January 2013, the neonicotinoid-treated hives continued to decline (figure 1). As shown in table 1, the numbers of frames containing bees were not significantly different among the treatments from 10/27/2012 to 12/29/2012 (one-way ANOVA), but became statistically significant different from 1/5/2013 to 4/4/2013 (one-way ANOVA, $p < 0.0001$). At the end of the experiment on 4/4/2013, there were 5.3, 2.0, and 2.9 frames of bees in the control, imidacloprid, and clothianidin-treated hives, respectively. The diminishing cluster size in the neonicotinoid-treated colonies led to the loss of six of the twelve (50%) with symptoms resembling CCD, whereas only 1 of the 6 control colonies was lost exhibiting *Nosema ceranae* like symptoms, although we did not perform any test to confirm *Nosema* infection in this control hive. No similar *Nosema*-like symptoms were

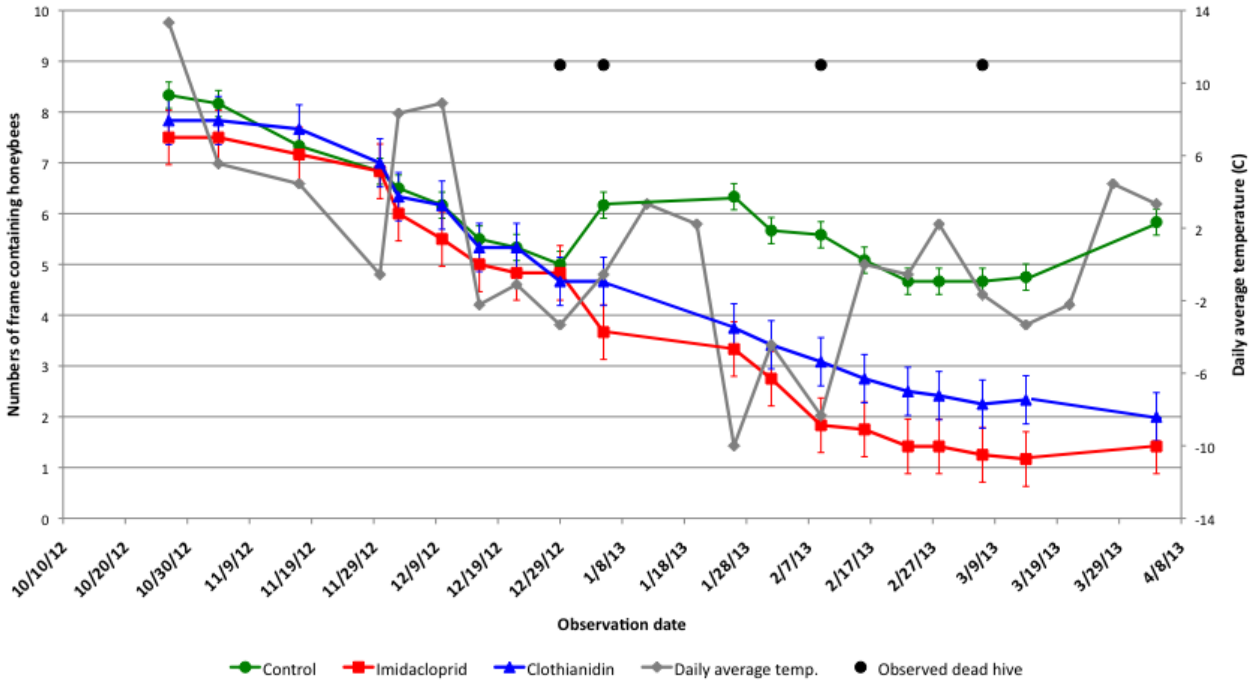

Figure 1. Average numbers of frame (standard deviations shown as error bars) containing honeybees for control-, imidacloprid-, and clothianidin-treated colonies and the corresponding daily average temperature at Worcester regional airport in Worcester MA recorded from October 2012 to April 2013. The daily average temperature readings were obtained from the NOAA website (http://cdo.ncdc.noaa.gov/qclcd/QCLCD).

Table 1. Field recording data from honey bee hives treated with control, imidacloprid, and clothianidin in sucrose water or high-fructose corn syrup (HFCS) from May 2012 to April 2013.

Treatment	Control		Imidacloprid		Clothianidin	
	Sucrose	HFCS	Sucrose	HFCS	Sucrose	HFCS
Honey bee hives	3	3	3	3	3	3
Average # of frame with bees (SD) Recorded from 10/27/2012 to 12/29/2012	6.3 (2)	6.8 (2)	6.0 (3)	6.3 (3)	6.6 (2)	6.3 (2)
Average # of frame with bees (SD) Recorded from 1/5/2013 to 4/4/2013	5.8 (1)	4.9 (3)	1.8 (2)	2.2 (2)	2.9 (2)	2.9 (2)
# of dead colony (%)	0 (0)	1 (33.3)	2 (66.7)	2 (66.7)	1 (33.3)	1 (33.3)
Date of dead colony observed		3/7/2013	1/5/2013 2/9/2013	1/5/2013 3/7/2013	1/5/2013	12/29/12
Average *Varroa* mite counts						
Before treatment (SD)	10 (6) [a]	11 (3) [a]	11 (2) [a]	10 (3) [a]	12 (2) [a]	9 (4) [a]
After treatment (SD)	2 (2) [b]	1 (1) [b]	1 (1) [b]	2 (1) [b]	1 (1) [b]	1 (1) [b]
Pooled Data [c]						
Honey bee hives	6		6		6	
Average # of frame with bees (SD) Recorded from 10/27/2012 to 12/29/2012	6.6 (2) [d]		6.1 (3) [d]		6.5 (2) [d]	
Average # of frame with bees (SD) Recorded from 1/5/2013 to 4/4/2013	5.3 (2) [e]		2.0 (2) [e]		2.9 (2) [e]	
# of dead colony (%)	1 (17)		4 (67)		2 (33)	
Average *Varroa* mite counts						
Before treatment (SD)	10 (4) [f]		12 (2) [f]		10 (3) [f]	
After treatment (SD)	2 (1) [f]		1 (1) [f]		1 (1) [f]	

[a] *Varroa* mite counts were not significantly different before Miteaway Quick strips treatment between sucrose and HFCS in control, imidacloprid, and neonicotinoid-treated hives (one-way ANOVA);
[b] *Varroa* mite counts were significantly different after Miteaway Quick strips treatment between sucrose and HFCS in control, imidacloprid, and neonicotinoid-treated hives (one-way ANOVA);
[c] Data from two sugar treatments were pooled for control, imidacloprid, and neonicotinoid-treated hives;
[d] Numbers of frame containing bees were not significantly different among control, imidacloprid, and neonicotinoid-treated hives during this period of time (one-way ANOVA);
[e] Numbers of frame containing bees were significantly different among control, imidacloprid, and neonicotinoid-treated hives during this period of time (one-way ANOVA, $p < 0.0001$);
[f] *Varroa* mite counts were significantly different before and after Miteaway Quick strips treatment in control, imidacloprid, and neonicotinoid-treated hives (paired t-test, $p < 0.0001$).

observed in the treated hives. Upon close examination of colonies in early April 2013, we found that the majority of bees in all neonicotinoid-treated colonies, regardless of whether they survived or not, had abandoned their hives during the course of winter. However, we observed a complete opposite phenomenon in the control colonies in which instead of abandonment, hives were re-populated quickly with new emerging bees. The honey bee clusters in the six surviving neonicotinoid-treated colonies were very small, and were either without queen bees, or had no brood.

We found no significant difference in the degree of *Varroa* mite infection between the control and neonicotinoid-treated colonies. The average mite counts were 10-12 per 150 bees in the control and neonicotinoid-treated colonies, respectively, as assessed in mid-August 2012 (table 1). We later reduced the mite counts in all colonies to 1-2 mites per 150 bees after the applications of Miteaway Quick strips, a commonly used medicinal treatments prior to the arrival of winter in which it significantly reduced mite counts from 10-12 to 1-2 mites per 150 bees, respectively, in control, imidacloprid, and neonicotinoid-treated hives (paired t-test, $p < 0.0001$). We also found that neonicotinoids do not appear to affect the quality of brood rearing during summer and fall (figure 2). The sealed brood counts for both control and neonicotinoid-treated colonies decreased significantly in parallel from July to September 2012 (Pearson, 2-tails, $p < 0.0001$). This decreasing (slope = −0.62) trend has been reported previously (Lu *et al*., 2012), and is consistent with a dirth of nectar that is common in the New England area during the summer, and is therefore independent of neonicotinoid exposure.

Discussion

The results from this study not only replicate findings from the previous study on imidacloprid and extend to clothianidin, but also reinforce the conclusion that sublethal exposure to neonicotinoids is likely the main culprit for the occurrence of CCD (Lu *et al*., 2012). The survival of 5 out of 6 control colonies in the same apiaries where the neonicotinoid-treated colonies were set up augment this conclusion. The observation of winter temperature modulating the severity of CCD associated

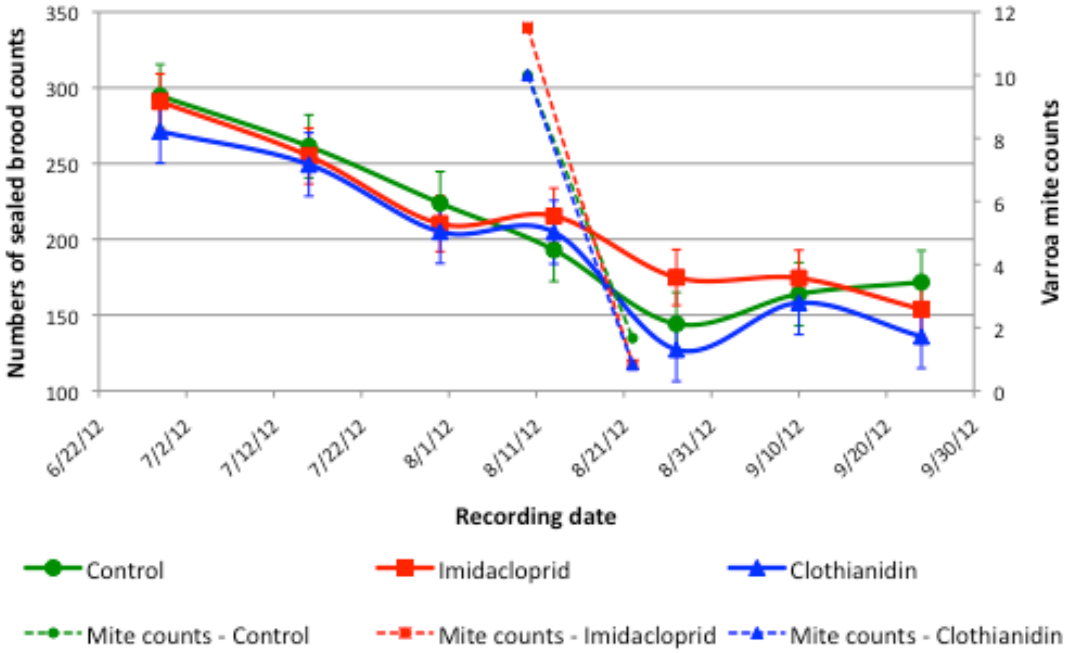

Figure 2. Average numbers of sealed brood count (standard deviations shown as error bars) for control-, imidacloprid-, and clothianidin-treated colonies during the dosing period (from 6/29/2012 to 9/24/2012), and the average numbers of *Varroa* mite counts recorded before and after Miteaway Quick strip treatment on 8/13/2012. Sealed brood counts were neither significantly different between sugars (one-way ANOVA) nor among treatments (one-way ANOVA). However, sealed brood counts were significantly decreased for all colonies from 6/29/2012 to 9/24/2012 (Pearson 2-tails, $p < 0.0001$).

with sub-lethal neonicotinoid exposure coincides with reports that CCD often occurs in the winter season. The modification of the sub-lethal effect of neonicotinoid by the severity of winter might be significant, and should not be overlooked in the evaluation of CCD epidemic. The previous study conducted during a colder winter reported 100% mortality of CCD in colonies treated with 0.1 ng/bee/day of imidacloprid (Lu *et al*., 2012), one-seventh of the dose used in the present study.

We found that chronic sub-lethal neonicotinoid exposures do not appear to compromise honey bees' immune resistance to pathogen infection in this study. This is in contrast to several earlier reports suggesting that the increased CCD mortality of honey bee colonies is due to reduced resistance toward common pathogens, such as increased susceptibility of *Nosema* infection, caused by neonicotinoid exposures (vanEngelsdorp *et al*., 2009; Alaux *et al*., 2010; Vidau *et al*., 2011; Pettis *et al*., 2012). The similar degree of *Varroa* mite infection in both control and neonicotinoid-treated colonies disagrees with the findings that CCD hives are often associated with significantly higher pathogen infestations than non-CCD hives exposures (vanEngelsdorp *et al*., 2009; Alaux *et al*., 2010; Vidau *et al*., 2011). In addition, a recent re-analysis of genomic data previously generated from RNA pools of CCD colonies has also excluded the association of pathogen infection and CCD (Tokarz *et al*., 2011). It is imperative to emphasize that while pathogen infections are common and serious diseases found in honey bees that often lead to colony death, the post-mortem examinations of the pathogen-caused dead colonies are vastly different to those suffered from CCD (Anderson and East, 2008; Lu *et al*., 2012). One of the defining symptomatic observations of CCD colonies is the emptiness of hives in which the amount of dead bees found inside the hives do not account for the total numbers of bees present prior to winter when they were alive (figure 3). On the contrary, when hives die in the winter due to pathogen infection, like the only control colony that died in the present study, tens of thousands of dead bees are typically found inside the hives (figure 4). The absence of dead bees in the neonicotinoid-treated colonies is remarkable and consistent with CCD symptoms.

Two critical questions remain to be answered in order to solve the CCD puzzle. First, why do neonicotinoid-treated colonies lose their ability to renew brood rearing toward the end of winter when temperatures began to rise? Considering that neonicotinoid-treated and control colonies had identical brood rearing performance prior to the arrival of winter (figure 1), the failure of neonicotinoid-treated colonies to resume brood rearing, in particular during the transition from winter to spring might be part of the interplay between sub-lethal neonicotinoid exposure and CCD. While it is true that the lack of brood rearing might simply be due to smaller surviving clusters during cold winter months, the surviving neonicotinoid-treated colonies never re-initiated the brood rearing into warm weather. We found that the severity of CCD caused by sub-lethal neonicotinoid exposures might be modulated by winter temperature. A colder and prolonged winter in 2010/2011 in central Massachusetts rendered a higher CCD mortality rate of 94% (Lu *et al*., 2012) than the current 50% in 2012/2013. Such disparity

Figure 3. Picture of the bottom board taken from one of the dead neonicotinoid-treated colonies on March 1st, 2013. The numbers of dead bees in the six dead CCD colonies ranged from 200-600 dead bees.

Figure 4. Picture of the bottom board taken from the only dead control colony on March 1st, 2013. The volume of dead bees was estimated to be 3.5 l using 1-L graduate cylinder using Atkins (1986) method.

might be due to the fact that the daily average temperature was lower in 63 of 91 days in the winter of 2010/2011 than of 2012/2013. The overall average temperature in the winter months was −3.8 °C (25 °F) in 2010/2011, approximately 2.78 °C (5 °F) lower than in 2012/2013.

Second and perhaps the foremost; why do honey bees vanish from neonicotinoid-treated colonies during the winter? It is striking and perplexing to observe the empty neonicotinoid-treated colonies because honey bees normally do not abandon their hives during the winter. This observation may suggest the impairment of honey bee neurological functions, specifically memory, cognition, or behavior, as the results from the chronic sublethal neonicotinoid exposure. Although the failure to initiate brood rearing and the vanishing of the worker caste in the neonicotinoid-treated colonies might be governed by completely different mechanisms, they suggest the possible involvement of cascading events prior to the occurrence of CCD. The findings from this study could be used to elucidate mechanisms by which sublethal neonicotinoid exposure impairs honey bees' ability to over winter with symptoms consistent with CCD.

We conclude that when honey bees were exposed to either imidacloprid or clothianidin at a dose of 0.73 ng/bee/day for 13 consecutive weeks from July to September 2012, six of twelve previously healthy neonicotinoid-treated colonies died and all progressed to exhibit CCD symptoms during the winter months. The survival of control colonies and the absence of CCD-like symptoms in the only dead control colony not only augment this conclusion but also support the finding that chronic

sub-lethal neonicotinoid exposure do not appear to compromise honey bees' immunity toward pathogen infection. The mechanisms by which sub-lethal neonicotinoid exposure caused honey bees to vanish from their hives during the winter months needs to be elucidated.

Acknowledgements

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Fewer Honeybees Died Over the Winter, a Report Says

Honeybees could be on their way back, according to a new federal report.

The collapse of bee populations around the country in recent years has led to warnings of a crisis in foods grown with the help of pollination. Over the past eight years, beekeepers have reported losses over the winter of nearly 30 percent of their bees on average.

The new survey, published on Thursday, found that the loss of managed honeybee colonies from all causes has dropped to 23.2 percent nationwide over the winter that just ended, down from 30.5 percent the year before. Losses reported by some individual beekeepers were even higher. Colony losses reached a peak of 36 percent in 2007 to 2008.

The survey of thousands of beekeepers was conducted by the Department of Agriculture and the Bee Informed Partnership, an organization that studies apian health and management.

"It's better than some of the years we've suffered," said Dennis vanEngelsdorp, a director of the partnership and an entomologist at the University of Maryland. Still, he noted, a 23 percent loss "is not a good number." He continued, "We've gone from horrible to bad."

He said there was no way to say at this point why the bees did better this year.

Jeff Pettis, the co-author of the survey who heads the federal government's bee research laboratory in Beltsville, Md., warned that "one year does not make a trend."

While much attention has been paid to colony collapse disorder, in which masses of bees disappear from hives, that phenomenon has not been encountered

in the field in the past three years, Dr. vanEngelsdorp said. Instead, what has emerged is a complex set of pressures on managed and wild bee populations that includes disease, a parasite known as the varroa mite, pesticides, extreme weather and poor nutrition tied to a loss of forage plants.

Treating colonies for the varroa mite, an Asian parasite that first reached the United States in 1987, seems to have the most direct effect on stemming losses, Dr. vanEngelsdorp said. “The beekeepers that are treating for varroa mites lose significantly fewer colonies than beekeepers that are not treating colonies for varroa mites,” and those who treat them four or five times a year do better than those who only treat them once or twice, he said.

The new report will not satisfy those who argue that the loss of bees can be traced to a class of pesticides known as neonicotinoids, especially one manufactured by Bayer.

Those views are supported by papers such as one published this month in the journal Bulletin of Insectology that found that six of 12 previously healthy colonies exposed to the pesticides died and all exhibited symptoms of colony collapse disorder in the winter.

Bayer attacked that study, saying that the lead author, Dr. Chensheng Lu of the Harvard School of Public Health, “greatly misdiagnosed colony collapse disorder” in the colonies he studied, and that he used dosages of the pesticide 10 times greater than what bees might encounter in the wild.

In an interview, Dr. Lu said that Bayer should reveal what it believes an “environmentally relevant” level of the pesticide should be.

Dr. vanEngelsdorp said that Dr. Lu and his colleagues gave the bees doses far beyond what they would encounter in nature, and over longer periods of time, so the new study only shows that “high doses of ‘neonics’ kill bees — which is not surprising.”

Rather than looking for a single chemical or class of chemicals, Dr. Pettis said, it is important to assess the interplay of parasites, illness, food sources and pesticides. “Nobody likes that kind of complicated story, but year to year, all those factors could play into colony health,” he added.

Eric Mussen of the University of California, Davis, said colony collapse disorder and other pressures have made beekeepers focus more intently on maintenance of their colonies than in the past.

"People are being forced now to look more carefully at their bees," he said. "If you don't take care of them, you lose them."

Journeys Over a Hot Stove

Humorous stories/anecdotes from my travels around the country, with simple, delicious recipes.

The tick-borne Powassan virus – you don't want it

No food or comedy this week. I felt this story was important enough to tell it straight. Also, this blog has a mind of its own… it does what it does.

Beyond Lyme – a nightmare tick virus

The Centers for Disease Control and Prevention list 14 diseases that people can get from tick bites. Many of them are regional, like Heartland virus in the Midwest and Colorado tick fever in the Rockies, but there are six that are fairly widespread and can infect people here in Maine. Lyme heads the list for its prevalence (it's estimated that Lyme is carried by half of all deerticks, which are also known as blacklegged ticks) and for being at "top of mind" among people and the medical community here.

Lyme, a bacterial disease, has had plenty of attention among some of my fellow bloggers and also in the Bangor Daily News, and rightly so. It can make you very sick in the short term and spawn a host of health problems down the road. But the bacterium responds to aggressive antibiotic therapy, so many people make a full recovery after initial symptoms.

The tick-borne Powassan virus, on the other hand, doesn't respond to *anything*.

Lyn Snow's case

We were friends and neighbors of Lyn Snow and her husband Jack, whose house is just three doors up the road, and got together several times for dinner and other things, until Lyn took ill last November. She'd been out on a walk with a mutual friend, Jerry, and returned, unbeknownst to her, with a deertick lodged in her scapula. Two days later, she was sick enough to go to PenBay Medical Center, which found the tick, suspected Lyme and started intravenous antibiotics. She got progressively worse over the next few days, then became unconscious, and when her kidneys shut down she was transported to Maine Medical Center. She remained in a

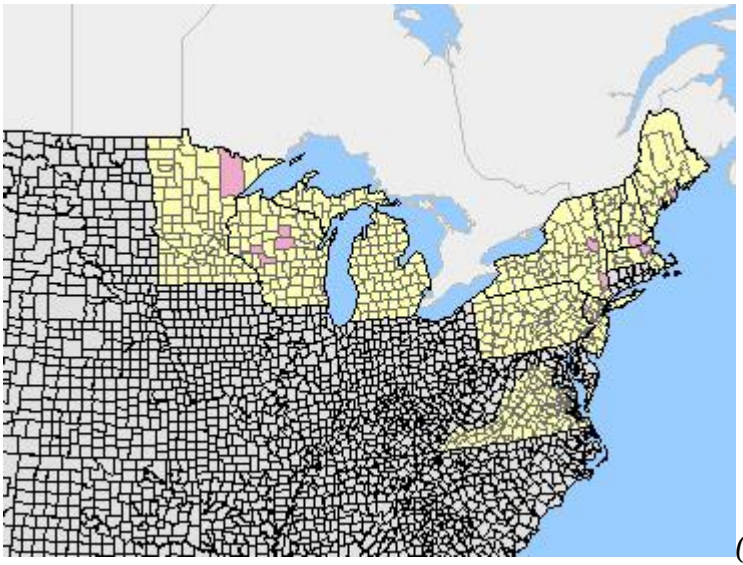
coma and was unresponsive to any stimuli. An EEG was performed, showing minimal brain activity.

About that same time – several weeks after the bite, her doctors sent blood samples to the labs at CDC's Division of Vector-Borne Diseases in Ft. Collins, Colorado. Tests indicated the pathogen was either Powassan or a variant strain of it. Case closed. There was nothing to be done.

Some five weeks after being bitten, life support was discontinued and Lyn died the following day. She was one of only 50 people in the U.S. (and the fifth in Maine) ever to contract Powassan, and only the fifth to die from it. Her story was covered widely in the media statewide, including this newspaper.

The virus and its surveillance

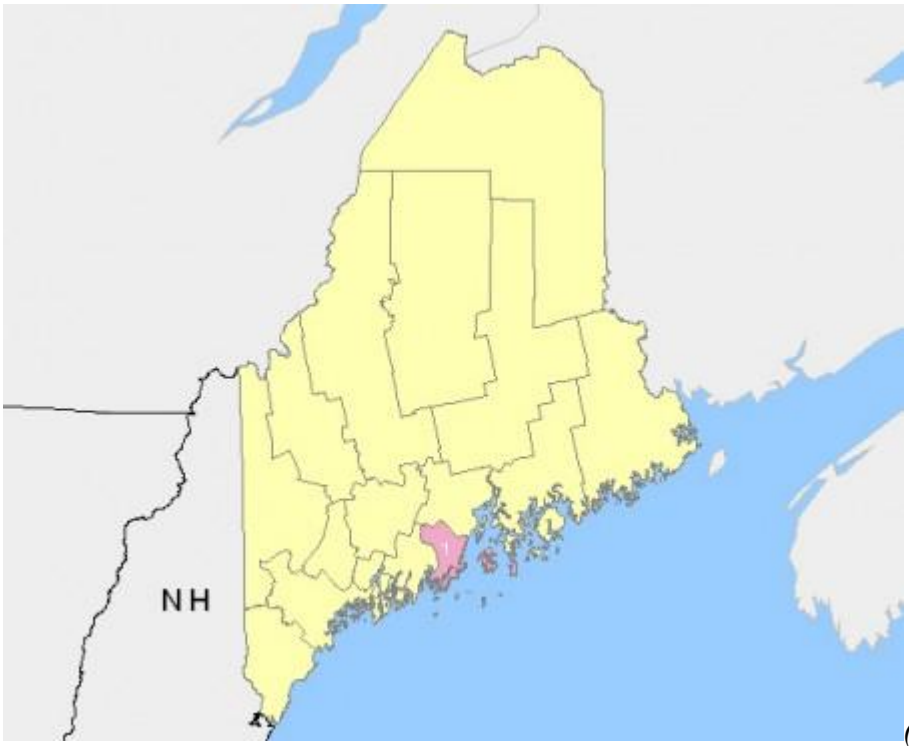
Powassan virus, named after the town of Powassan, Ontario where the first case was identified in the late 1950s (a young boy who died after a tick bite), can cause symptoms similar to encephalitis, including seizures, brain swelling, and loss of consciousness, and in 10% of the cases, death. Half the patients who survive suffer permanent and debilitatingly severe neurological damage.



(Cases testing positive for Powassan, 2013. Two fatal – in Poughkeepsie, NY, and here in So. Thomaston, ME)

As rare as it is, this is one very terrifying germ. It's almost certainly in the woods around us, but it's impossible to know just how prevalent it is. I spoke with Dr. Sheila Pinette, Director of Maine Centers for Disease Control in Augusta, and she added some important information: though the Powassan's host (or vector) is the deertick, it's believed the ticks come more frequently from woodchucks, mice, and squirrels than deer. Others have said the tick moves from the woodchuck to the deer, and then to humans. (See Maine CDC's Powassan page here). What makes it all so sinister is that, unlike with Lyme-bearing ticks, there is *no delay* between the bite and the transmission of the virus. Once it bites you, you have the virus. Maine Medical Center has an excellent web page on this virus and other tick-borne pathogens.

Maine CDC and the University of Maine seek more information about ticks, and to do this they want your ticks in the mail! The better the surveillance, the more we know about the risk level in our area.



(Lyn's case on a Powassan-positive map, 2013. Both maps from U.S. Geological Survey and Centers for Disease Control and Prevention)

I also spoke with Dr. Marc Fischer, epidemiologist at CDC's facility in Ft. Collins. His specialty is arboviruses, a name derived from **AR**thropod-**BO**rne viruses (arthropods include insects, arachnids, and crustaceans – animals with an exoskeleton and a segmented body). Dr. Fischer also directs ArboNET, a CDC-sponsored national surveillance system for arboviral diseases in the U.S. ArboNET was established in 1999 to respond to the spread of West Nile Virus, but has since expanded to surveil any disease-causing virus from insects and arachnids (which includes ticks), like Powassan. But ArboNET is a *passive* surveillance operation: they don't go out and collect ticks. They keep track of all arbovirus diseases reported to them by the medical community nationwide.

Dr. Fischer knew about Lyn Snow's case with Powassan. He indicated that surveillance of tick-borne diseases – especially those that mimic the symptoms of encephalitis – is catch-as-catch-can. Some clinicians don't find a tick bite and misdiagnose the disease. Others fail to report. But so far, it may be the best data collection effort out there.

Permethrin beats DEET

What to do? Any and everything possible to avoid tick bites. My wife and I bought several bottles of a permethrin clothing treatment, which is odorless and colorless: mix the solution in a

large plastic bag, add four or five pieces of tightly rolled-up clothing you wear a lot in summer, seal the bag, and massage the liquid throughout the clothing. After it soaks for two hours, you can remove the clothing and run it through the dryer. Permethrin-treated clothes can be washed and dried several times without diluting the chemical's effectiveness. It lasts a month or more. Then do it again! (The brand we bought is TickBlock, from Massachusetts).

Permethrin is hugely more effective than DEET at repelling and killing ticks. If a tick lights on permethrin-treated clothing, it will curl up, fall off, and die. Permethrin's toxicity to humans is also very low: it's used as an insecticide on fruits and vegetables, so we eat it all the time.

So that's about it. Ticks are here in abundance, and we don't know what they've got in them.

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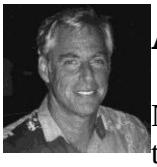
I want to thank Dr. Sheila Pinette, Director of Maine CDC in Augusta, and Dr. Marc Fischer, epidemiologist at CDC's (federal) Division of Vector-Borne Diseases in Fort Collins, Colorado, for their help in preparing this piece.



About Ned White

Ned White is an author, photographer, crossword constructor, humorist, traveler through 49 states, and an avid cook. He lives with his wife in South Thomaston.

http://hotstove.bangordailynews.com/2014/05/30/home/the-tick-borne-powassan-virus-you-dont-want-it/#.U4qVGELVMjA.twitter

Source: Bangor Daily News, May 30, 2014

Environmentalists Fight New Pesticide

By RYAN ABBOTT

WASHINGTON (CN) - Without consulting with expert biologists, the U.S. Environmental Protection Agency authorized a new pesticide that could affect as many as 1,377 federally protected species, environmental groups claim in court.

The Center for Biological Diversity, the Center for Food Safety and Defenders of Wildlife sued the EPA in Federal Court, challenging its authorization for widespread use of cyantraniliprole (CTP), a new pesticide that the agency expects to be used on a wide variety of agricultural crops, as well as golf courses, lawns, ornamental plants, fly bait and public health pests.

According to the lawsuit, CTP is a broad-spectrum systemic insecticide absorbed throughout plants.

"It kills by causing unregulated activation of ryanodine receptors, which results in unregulated muscle contraction, paralysis, and death," according to the complaint.

It adds: "Based on data showing the concentrations or amounts of CTP that cause direct effects, EPA classified the chemical as 'slightly to moderately toxic to freshwater fish; slightly toxic to estuarine/marine fish; slightly to very highly toxic to freshwater invertebrates; moderately to highly toxic to estuarine/marine invertebrates, highly toxic to benthic invertebrates; highly to very highly toxic to terrestrial insects' from acute exposures."

The groups claim that though the agency found that CTP was not acutely toxic to birds and mammals, their offspring showed "some impacts to weight and effects to thyroid and liver from chronic exposures."

Their complaint accuses the EPA of lacking information for specific species before performing a risk analysis.

"While EPA admitted that it lacked the ability to make determinations about the specific species and habitats affected by CTP, it 'identified a total of 1,377 listed species that overlap at the county-level with areas where cyantraniliprole is proposed to be used ... This preliminary analysis indicates that there is a potential for cyantraniliprole use to overlap with listed species and that a more refined assessment is warranted,'" the complaint states. "Based on these risks, several entities, including some of the plaintiffs here, urged EPA to consult with the Services before authorizing uses of cyantraniliprole."

The Services refers to the U.S. Fish and Wildlife Service and/or the National Marine Fisheries Service.

The EPA registered the pesticide in January, including product mixtures containing other pesticides such as thiamethoxam.

"Despite specific comments urging EPA to consult and EPA's conclusions in the Risk Assessment that additional analyses and cooperation with the Services were necessary, EPA finalized its CTP Registration Decision without consulting with NMFS or FWS as required by Section 7 of the ESA [Endangered Species Act]," states the complaint.

The groups say the government's authorization of the pesticide violates the Endangered Species Act. They want a court order vacating the EPA authorization of CTP use.

The groups' lead counsel is Patti Goldman, with Earthjustice, of Seattle.

Toxin-Resistant Corn Pest Thwarts Genetic Engineering Efforts

Posted By Matthew Heller On June 5, 2014 @ 5:00 am In Front Page: Health & Lifestyle,Health & Lifestyle,National,News,Top Stories | No Comments

Larry Hasheider walking along one of his corn fields on his farm in Okawville, Ill. (AP/Jeff Roberson)

LOS ANGELES — In a case of evolution outfoxing agricultural biotechnology, a voracious rootworm that was supposed to be poisoned by genetically-modified corn has become resistant to the toxins produced by the plant.

Populations of western corn rootworms, which used to cause billions of dollars in damage to U.S. crops, plummeted across the Midwest after Bt corn engineered to produce insecticidal toxins from the bacterium Bacillus thuringiensis was first planted in 1996. The toxins killed the worms and reduced the use of conventional pesticides, helping to make Bt corn so popular that it now accounts for three-quarters of the U.S. corn crop.

But according to a new study by entomologists at Iowa State University, the rootworm has rapidly developed a resistance to the toxins, with potentially disastrous consequences for farmers and the environment.

The Western Corn Rootworm

“Unless management practices change, it’s only going to get worse,” Aaron Gassmann, a co-author of the study, told Wired. “There needs to be a fundamental change in how the technology is used.”

Gassmann has been studying Bt-resistant worms since 2009, when he looked into reports of extensive rootworm damage in Bt cornfields in northeast Iowa. Resistance has also been reported in parts of Illinois, Minnesota, Nebraska and South Dakota. The new paper describes further incidents of resistance in other parts of Iowa.

“These first cases of resistance by western corn rootworm highlight the vulnerability of Bt maize to further evolution of resistance from this pest,” the study said.

Entomologists told Wired that the resistance is the result of farmers failing to set aside refuges where non-Bt corn could be planted, and biotech companies not enforcing the planting of refuges.

Within the non-Bt fields, Wired noted, rootworms would remain susceptible to the Bt toxin. By mating with any Bt-resistant worms that chanced to evolve in neighboring fields, they would prevent resistance from building up in the gene pool.

“Biotech companies have successfully lobbied [the U.S. Environmental Protection Agency] for major reductions in refuge

requirements," said entomologist Bruce Tabashnik of the University of Arizona. He and other scientists have pushed the EPA to double the current requirements, but so far without success.

Farmers likely won't stop using Bt corn because it is still effective against other pests. But as rootworms become more resistant, Gassmann said, farmers will turn to insecticides, increasing their costs and forfeiting the ecological benefits originally gained by using Bt corn.

The results of the new study "illustrate that Bt crops producing less than a high dose of toxin against target pests may select for resistance rapidly; consequently, current approaches for managing Bt resistance should be reexamined," the Iowa State researchers concluded.

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Print Page

By Colleen Quinn
State House News Service

June 05. 2014 5:27PM

House agrees to bill aimed at keeping mosquito population down

BOSTON -- Municipal public works employees and seasonal workers would be given back the authority to drop non-toxic pesticide pellets into storm drains in an effort to prevent an outbreak of mosquito-borne diseases, under a bill that has cleared the Massachusetts House.

Cases of West Nile virus and Eastern Equine Encephalitis (EEE) surged in the last few years, and in 2012 two Massachusetts residents' deaths were attributed to the mosquito-borne diseases.

State public health officials believe this year could bring another high-risk season.

Mosquitoes with West Nile live in small containers of standing water, and transfer the disease to humans. EEE is carried by birds and spread bird-to-bird by mosquitoes.

The legislation (H 3568) would restore emergency authority to public works employees to use pesticides in catch basins.

Tom Philbin, a legislative analyst for the Massachusetts Municipal Association, said allowing DPW workers to apply pesticides in storm drains will help wipe out breeding grounds for mosquitoes.

"It could save lives, and it doesn't cost communities any money," he said Thursday.

Public works employees were allowed to drop pesticides in catch basins from 2001 to 2009, but in 2010 the Department of Agricultural Resources decided against renewing employees' ability to use the pesticides. State law reverted back to allowing only licensed professional pesticide applicators, leaving the job mainly to those who work at local mosquito control commissions.

Mosquito control board members have urged lawmakers to once again allow municipal employees to drop the pesticides, saying it would help them do their job to stop the spread of diseases.

Municipal officials have tried for years to get the authority restored. The House last July advanced an earlier version of the bill (H 757) which was amended Thursday and passed on to the Senate.

http://www.metrowestdailynews.com/article/20140605/NEWS/140607686

Print Page

'Bee-safe' Biopesticide Could Be Neonicotinoid Alternative

UK researchers find new biopesticide that has limited effect on honeybees

Published on: **Jun 9, 2014**

A novel bio-pesticide created using spider venom and a plant protein has been found to be safe for honeybees, despite being highly toxic to a number of key insect pests, according to a study by UK-based Newcastle University.

The new research tested the insect-specific Hv1a/GNA fusion protein bio-pesticide – a combination of a natural toxin from the venom of an Australian funnel web spider and snowdrop lectin.

Feeding acute and chronic doses to honeybees – beyond the levels they would ever experience in the field – the team found it had only a very slight effect on the bees' survival and no measurable effect at all on their learning and memory.

Publishing their findings last week, the authors say the insect-specific compound has huge potential as an environmentally-benign, 'bee-safe' bio-pesticide and an alternative to the chemical neonicotinoid pesticides.

UK researchers find new biopesticide that has limited effect on honeybees

Though research prepared by the USDA and the U.S. EPA have determined there are several causes for declines in bee and pollinator populations, some studies have linked neonicotinoids to the declines. The EU has already authorized a ban on neonicotinoid seed treatments.

Related: NRCS Pledges $3 Million for Honey Bee Health

By pollinating some key crop species, honeybees make a vital contribution to food security. The decline of these insects raises significant concerns about farmers' ability to feed a growing population.

Newcastle professor Angharad Gatehouse, one of the supervisors on the project, says the study's findings suggest that Hv1a/GNA is unlikely to cause any detrimental effects on honeybees.

"Previous studies have already shown that it is safe for higher animals, which means it has real potential as a pesticide and offers us a safe alternative to some of those currently on the market," he says.

During the study, the bees were exposed to varying concentrations of the spider/snowdrop bio-pesticide over a period of seven days. Throughout the study period, the team carried out a series of memory tests and recorded any changes in behavior.

Related: House Subcommittee Reviews Bee Health Research

Honeybees naturally perform sophisticated behaviors while foraging that require them to learn and remember floral traits associated with food. Disruption to this important function has profound implications for honeybee colony survival, because bees that cannot learn will not be able to find food and return to their hives.

"This is an oral pesticide so unlike some that get absorbed through the exoskeleton, the spider/snowdrop recombinant protein has to be ingested by the insects," explained research lead Erich Nakasu, a PhD student at Newcastle University.

Unlike other pesticides, Hv1a/GNA affects an underexplored insecticidal target, calcium channels. These are more diverse than commonly-targeted insecticide receptors, such as sodium channels, and therefore offer the potential for more species-specific pesticides.

"Calcium channels are linked to learning and memory in bees so it's vital that any pesticide targeting them does not interfere with this process," Nakasu says.

Related: **Bee Health Campaign Suggests Neonicotinoid Treatments Unnecessary**

"Although Hv1a/GNA was carried to the brain of the honeybee, it had no effect on the insect which suggests the highly selective spider-venom toxin does not interact with the calcium channels in the bee."

The larvae were also unaffected by the Hv1a/GNA, as they were able to break it down in their gut.

"Around 90% of the world's plants are directly or indirectly reliant on pollinators to survive," says Dr. Geraldine Wright, one of the authors on the paper.

"If we destroy the biodiversity of pollinators then it will be irrelevant how effective our pesticides are because we won't have any crops to protect."

This research was funded by the UK'sTechnology Strategy Board.

Source: Newcastle

THE CONVERSATION

5 June 2014, 6.06am BST

Iconic monarch butterflies under threat from rising herbicide use

AUTHOR

John Pleasants
Adjunct Assistant Professor of Ecology, Evolution and Organismal Biology at Iowa State University

Monarch butterfly: not scared of wearing bold colours. Dean Morley, CC BY-ND

Monarch butterflies are known for their striking flame-orange and black appearance, and especially for their mass migration in their millions to spend winters in the mountain forests of Mexico. But despite growing problems with deforestation in Mexico, their struggle begins at home in the United States and Canada.

The butterflies that fly to Mexico are the great-great-great grandchildren of the monarchs that were in Mexico the previous winter. In 2013 the overwintering population in Mexico covered 0.67 hectares of fir forest (about 44 million butterflies) the lowest since counts began in 1994. Since 1999 their numbers have declined 82%.

What could have caused this? During their larval stage monarchs, which can be found from the US central states to the east coast and into southern Canada, feed exclusively on milkweed plants. We observed in 2001 that many monarchs were feeding on milkweeds in agricultural fields – more than 80% of monarchs from the Midwest. Since then, milkweeds in and around agricultural crop fields have gradually been eliminated, through a combination of spraying with Roundup (glyphosate) herbicide and increased planting of corn and soybeans genetically modified to be resistant to the herbicide.

In previous studies, we've shown that the magnitude of monarch population decline in Mexico matched the magnitude of the decline in the abundance of milkweeds in butterflies' prime breeding habitat – the Corn Belt region of the US. This strongly suggests that milkweed loss is the primary factor in the species' decline.

In a recently published paper, Tyler Flockhart and colleagues in Canada and Australia

examined the monarch population decline and tried to gauge the relative roles of all the factors that may lie behind it. Besides the loss of milkweeds in the breeding area, these include climate change, the loss of forests at the overwintering sites, and occasional catastrophic weather events.

The research team developed a population model that incorporated information about birth and death rates for each of the roughly four generations that comprise the monarch annual migration cycle. A number of pieces of information necessary to generate such a population model are unknown or poorly known, so assumptions had to be made. With that caveat, they were able to confirm that the primary driving force behind the population decline was loss of milkweeds in the breeding area, with the other factors playing a minor role.

With population models, not only can you examine the past but also extrapolate into the future. The good news is that the population model projects the population to decline by only a further 14% over the next century – much less than the precipitous drop in numbers over the last 20 years. The bad news is that the new, much lower population level makes the species more vulnerable to events such as catastrophic heatwaves or severe winters that can wipe out millions at a stroke. The model puts the chance of population extinction in the next century at a low but distinctly non-trivial 5%.

The monarch has equally fetching attire as a caterpillar.
Singer Ron/USFWS, CC BY

The chief reason why future declines will not be as pronounced is because milkweeds have been virtually eliminated from farmland, meaning that losses stemming from that habitat have already been accounted for. At present there are two main habitats that provide milkweeds for monarch butterflies.

The Conservation Reserve Program (CRP) provides incentives to farmers to set aside land from growing crops. This land is typically planted with grasses to prevent erosion but often has milkweeds. I have estimated that there are 1.4m hectares (3.6m acres) of CRP land in the Midwest providing suitable milkweed habitat – if there were incentives for farmers to plant milkweed on their CRP land, this could prevent further declines and even promote a population comeback. Unfortunately at present the high demand for corn means farmers are tempted to convert CRP land back to crops.

The second important habitat is roadsides. Transportation Department officials need to be informed about the impact of roadside management practices, such as spraying with herbicide and mowing, on monarchs.

Incentives to plant other flowering species besides milkweed in CRP land and roadsides would

help. Pollinators such as bees and butterflies are in serious decline, largely due to loss of habitat. Other threats to pollinators such as the widespread use of insecticides such as neonicotinoids could also be eliminated.

NOAA NATIONAL OCEANIC AND ATMOSPHERIC ADMINISTRATION
UNITED STATES DEPARTMENT OF COMMERCE

NOAA scientists find mosquito control pesticide use in coastal areas poses low risk to juvenile oysters, hard clams

Climate stressors, however, increase risk to shellfish

June 9, 2014

Four of the most common mosquito pesticides used along the east and Gulf coasts show little risk to juvenile hard clams and oysters, according to a NOAA study.

However, the study, published in the on-line journal ***Archives of Environmental Contamination and Toxicology***, also determined that lower oxygen levels in the water, known as hypoxia, and increased acidification actually increased how toxic some of the pesticides were. Such climate variables should be considered when using these pesticides in the coastal zone, the study concluded.

"What we found is that larval oysters and hard clams can withstand low levels of pesticide use, but they are more sensitive to pesticides if their ecosystem is suffering from local climate stressors like hypoxia and acidification," said the study's lead author, Marie DeLorenzo, Ph.D., NOAA environmental physiology and microbiology program lead with **NOAA's Centers for Coastal Ocean Science**. "Hopefully these data will benefit both shellfish mariculture operations and environmental resource agencies as they manage the use of mosquito control pesticides near their coastal ecosystems."

Juvenile clams used in toxicity testing. (Credit: NOAA)

Commercial shellfishing has a large economic national impact. **NOAA Fisheries** estimated that U.S. oyster and hard clam landings for 2010 were worth nearly $118 million and $41 million, respectively. Shellfish growers, however, are concerned that pesticide spraying near the coastlines may contaminate both their hatcheries and source waters. This is compounded by a lack of data on the toxicity of mosquito insecticides for these shellfish.

These ecologically and economically important species inhabit tidal marsh habitats along the U.S. Atlantic and Gulf of Mexico coastlines. Clams and oysters are also important for the coastal ecosystem because they filter water, improving water quality, and serve as habitat and food sources for other estuarine species.

Approximately 200 mosquito species live in the United States. In addition to causing painful itchy bumps to people, mosquito bites can transmit serious diseases such as malaria, dengue fever, and West Nile virus. One approach to controlling mosquitoes is to apply pesticides by spraying from planes or trucks over a large area. However, to effectively control mosquitoes, the pesticides must target species which live in aquatic habitats that are also home to sensitive estuarine species. This may pose a risk to coastal environments. Also, since many residential communities where the pesticides may be used are near these coastal aquatic habitats, the potential for direct overspray or unintentional drift into these waters is increased.

The study sought to address a lack of toxicity data for mosquito control pesticide effects on shellfish early life stages. The research team examined the toxicity of four mosquito control pesticides (naled, resmethrin, permethrin, and methoprene) to larval and juvenile life stages of hard clams (*Mercenaria mercenaria*) and Eastern oysters (*Crassostrea virginica*).

Lethal thresholds were determined for the four pesticides, and differences in sensitivity were found between chemicals, species, and life stages tested. Overall, clams were more susceptible to mosquito control pesticides than oysters. Naled, an organophosphate chemical, was the most toxic compound in oyster larvae, while resmethrin was the most toxic compound in clam larvae. Decreased swimming activity was observed after four days in larval oysters and decreased growth was found in juvenile clams and oysters after 21 days.

Using a hazard assessment, which compared the toxicity thresholds to concentrations expected in the environment, the researchers calculated a low-level of risk to clams and oysters from application of these pesticides for mosquito control.

The researchers also tested the pesticides' toxicity under climate stress conditions. The more extreme climate conditions caused increased pesticide toxicity.

The study did not address the impacts of the pesticides on other shellfish such as shrimp or lobsters.

NOAA's mission is to understand and predict changes in the Earth's environment, from the depths of the ocean to the surface of the sun, and to conserve and manage our coastal and marine resources. Join us on **Facebook**, **Twitter**, **Instagram** and our other **social media channels**.

BBC NEWS

SCIENCE & ENVIRONMENT

10 June 2014 Last updated at 12:38 ET

GM lab mosquitoes may aid malaria fight

By Jonathan Webb
Science reporter, BBC News

Scientists have created mosquitoes that produce 95% male offspring, with the aim of helping control malaria.

Flooding cages of normal mosquitoes with the new strain caused a shortage of females and a population crash.

The system works by shredding the X chromosome during sperm production, leaving very few X-carrying sperm to produce female embryos.

In the wild it could slash numbers of malaria-spreading mosquitoes, reports the journal **Nature Communications**.

Although probably several years away from field trials, other researchers say this marks an important step forward in the effort to produce a genetic control strategy.

Malaria is transmitted exclusively by mosquitoes. Despite reductions brought about by measures such as nets or spraying homes with insecticides, it continues to kill hundreds of thousands of people annually, mostly in sub-Saharan Africa.

The idea of using a "sex-distorting" genetic defect to control pest populations was proposed over 60 years ago, but this is the first time it has been practically demonstrated.

The researchers, led by Prof Andrea Crisanti and Dr Nikolai Windbichler of Imperial College London, transferred a gene from a slime mould into the African malaria mosquito *Anopheles gambiae*. This gene produces an enzyme called an "endonuclease" which chops up DNA when it recognises a particular sequence.

Prof Crisanti said his team exploited a "fortuitous coincidence": the target sequence of that endonuclease is found specifically - and abundantly - on the mosquito's X chromosome. "In *Anopheles gambiae*, all 350 copies are together, side-by-side on the X chromosome," he told BBC News.

When sperm are produced normally, in mosquitoes or in humans, 50% contain an X chromosome and 50% a Y chromosome. When they fuse with an egg these produce female and male embryos, respectively.

In the new mosquitoes, the X-attacking endonuclease is turned on specifically during sperm formation. As a result, the males produce almost no X-containing sperm - or female offspring. More than 95% of their progeny are male.

Breaking the cycle

Importantly this change is heritable, so that male mosquitoes pass it on to about half their male progeny. This means if the artificial strain is released into a population - in the lab or in the wild - the trait can spread until *most* males are only producing male offspring, perhaps eradicating the population altogether.

"It can be a self-sustaining effect," said Dr Windbichler.

Indeed, in five test cages that started with 50 males and 50 females, when the team introduced 150 of their new sex-distorter males, the number of females plummeted within four generations. After another couple of generations, in four out of five cages, the population died out entirely.

Both these effects are beneficial, Prof Crisanti explained, because only female mosquitoes bite humans and spread malaria. So a drop in female numbers might slow its spread, while a population crash could "break the cycle" of malaria transmission.

Dr Luke Alphey founded the company Oxitec to develop genetic control strategies for harmful insects and has pioneered the use of GM mosquitoes to **help control dengue fever**. He told the BBC the new research was exciting, but suggested that if used in the wild, this particular sex-distorter strain might not spread indefinitely and would need to be "topped up".

For a really successful, spreading system to eradicate malaria mosquitoes, "You'd have to get such a system expressed on the Y chromosome," Dr Alphey said.

The new study's authors agree this would be much more powerful. "You'd need to release fewer individuals, because all males will inherit the gene from their fathers and pass it on to all their sons - so the effect would not be diluted," said Dr Windbichler.

"Theoretically, if you have it on the Y," Prof Crisanti added, "one single individual could knock out an entire population."

In fact, Dr Windbichler and Prof Crisanti showed in **another recent paper** that this type of gene insertion on the mosquito Y chromosome is perfectly achievable.

"They haven't yet put it all together," Dr Alphey commented, "but all the pieces are in place."

Dr Alphey also commented that the power of that proposed technique would pose additional questions for researchers and regulators. "In principle, what you get is extinction," he said.

"Humans have undoubtedly driven a very large number of species to extinction - but we've only deliberately done it with two: smallpox and rinderpest. Would we want to do that with *Anopheles gambiae*?"

Dr Alphey's answer to his own question appears to be "maybe".

"If this species were to suffer a population crash, it's hard to see how significant negative side-effects might arise," he explained. "The mosquitoes are not keystone species in their ecosystems. And this technique only affects one species, *Anopheles gambiae*, among more than 3,000 known species of mosquitoes."

"If we rely instead on pesticide control we would likely kill non-malarial mosquitoes and many other insects besides. The genetic approach is much more precise."

Crisanti and Windbichler think that extinction is unlikely, even with the proposed Y chromosome-driven system, but agree that caution is warranted. "There are a lot of tests to run through," Dr Windbichler said.

"We are still a couple of years from this being applied in the field. It's very promising but there's still a long way to go."

Dr Michael Bonsall, a reader in zoology at the University of Oxford, said the new research was "super cool" and demonstrated "just how important these sorts of GM technologies are at reducing insect vector population sizes."

"This has important implications for limiting the spread of malaria," Dr Bonsall said, though he also noted that it was "a long way from being deployed."

To begin testing the safety and efficacy of the sex-distorter strains on a bigger scale, Prof Crisanti's team has built a large facility in Italy. "We have big, contained cages in which we can reproduce a tropical environment - and we can test several hypotheses on a

very large scale."

Meanwhile, he and his colleagues are pleased to have developed such a promising genetic weapon against malaria using the elusive sex-distortion mechanism, proposed many years ago.

"One of the first people to suggest it was the famous British biologist Bill Hamilton, while he was actually here at Imperial as a lecturer for a while," commented Dr Windbichler. "So it was theorised 60 years ago, but never put in practice."

The Life Scientific, broadcast at 9am on Tuesday 10th June, featured malaria researcher Professor Janet Hemingway.

News

FOR IMMEDIATE RELEASE
June 10, 2014

Connecticut Department of Public Health
Contact: William Gerrish
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State Mosquito Program Begins Mosquito Testing for West Nile and Eastern Equine Encephalitis Viruses

First case of EEE identified in the state during 2013; public, clinicians reminded of EEE risk

Hartford – The State of Connecticut Mosquito Management Program today announced it is monitoring mosquitoes for the presence of viruses that can cause illness in people including West Nile virus (WNV) and eastern equine encephalitis virus (EEE). The mosquito trapping and testing program, coordinated by the Connecticut Agricultural Experiment Station (CAES), began June 2nd. Test results to date are negative.

The program also released surveillance results from the 2013 season. The results include the first confirmed human case of eastern equine encephalitis (EEE) in a Connecticut resident. In April, the Centers for Disease Control and Prevention (CDC) confirmed that an adult resident of eastern Connecticut who had been hospitalized with encephalitis and died in the Fall, was positive for EEE.

The resident had been tested for WNV, but was not tested for EEE before death. Fortunately, through the astute actions of a physician at a local hospital, further testing was initiated that lead to the post mortem diagnosis of EEE infection.

“While rare, EEE is serious and underscores the importance of taking personal precautions to avoid mosquito bites,” said DPH Commissioner Dr. Jewel Mullen. “The presence of this virus in Connecticut should also remind clinicians to include EEE, along with WNV, among their possible diagnoses so that appropriate tests can be done.”

Dr. Mullen said that DPH is preparing an advisory to Connecticut clinicians regarding the current epidemiology of EEE and WNV as well as testing options for diagnosing mosquito-borne diseases.

EEE Surveillance
Mosquitoes infected with EEE virus transmit the virus by biting humans. EEE is a rare illness in humans, and only a few cases are reported in the United States each year. Most cases occur in the Atlantic and Gulf Coast states.

Last season there was significant EEE virus circulation in eastern Connecticut, including infected mosquitoes, a horse, and pheasant flocks. Mosquitoes with EEE virus were identified in five

Connecticut towns: Haddam, Hampton, North Stonington, Plainfield and Voluntown. A horse stabled in Griswold died from EEE-associated illness during the second week of September. In early to mid-September, pheasants in a farm flock in Killingly and a flock in Sprague died from EEE infections.

The numbers and types of mosquitoes with EEE identified in the Pachaug State Forest in Voluntown prompted the Department of Energy and Environmental Protection to temporarily close part of the forest to recreational activities and to conduct ultra-low volume ground spraying to reduce the number of mosquitoes.

WNV Surveillance
Last season, the Connecticut Agricultural Experiment Station (CAES) identified WNV-positive mosquitoes at trap sites in 22 towns: Branford, Bridgeport, Darien, East Haven, Fairfield, Glastonbury, Greenwich, Groton, Manchester, New Haven, North Branford, Norwalk, Plainfield, Stafford, Stamford, Stratford, Voluntown, Wallingford, Waterford, West Haven, Westport and Wilton. Four Connecticut residents from the towns of Stamford, Stratford, and Bridgeport were diagnosed with WNV-associated illnesses. There were no fatalities.

Connecticut Mosquito Management program
The management of mosquitoes in Connecticut is a collaborative effort involving the Department of Energy and Environmental Protection (DEEP), the Connecticut Agricultural Experiment Station (CAES) and the Department of Public Health (DPH), together with the Department of Agriculture and the Department of Pathobiology at the University of Connecticut (UCONN). These agencies are responsible for monitoring and managing the state's mosquito population levels to reduce the potential public health threat of mosquito-borne diseases.

The CAES maintains a network of 91 mosquito-trapping stations in 72 municipalities throughout the state. CAES began its mosquito trapping and testing season last week. Mosquito pools that test positive for WNV and EEE, as well as human cases of these illnesses, will be posted on the Connecticut Mosquito Management Program website.

For information on West Nile virus and EEE, including what you can do to prevent getting bitten by mosquitoes and the latest mosquito test results, visit the Connecticut Mosquito Management Program Web site at www.ct.gov/mosquito.

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Print

War on Cornfield Pest Sparks Clash Over Insecticide

JUNE 12, 2014
By: **Bloomberg**

Pesticide use is surging among U.S. farmers who are worried about insect resistance to Bt corn. But scientists warn that overuse of chemicals may create a worse problem down the road.

Pesticide use is surging among U.S. corn farmers who are worried that some insects have become resistant to genetically modified versions of the crop.

That's an unexpected reversal since one of the promises of engineered corn when it was introduced 17 years ago was its ability to kill pests. The use of soil insecticides for the crop plunged 90 percent through 2010, according to the U.S. Department of Agriculture.

RELATED STORIES

- **Pros and Cons of Soil Insecticide Use for Corn Rootworm Protection**
- **Four States Confirm Rootworm Resistance**

Whether the return to pesticide use makes sense, or is simply spurred by a chemical industry marketing campaign, is at the center of one of the biggest debates in the Corn Belt this spring. At the heart of the controversy is whether snuffing out pests in the short term with chemicals may create a worse problem down the road.

Farmers say they need to do whatever it takes now to control the western corn rootworm, the most damaging U.S. corn pest. Although Monsanto Co. designed its corn to kill the worms, resistant bugs have been found in four states and growers say pesticides are needed again to protect their crops.

It would be "financial suicide" to plant rootworm-killing corn without a soil insecticide as a secondary way to control the larvae, said Illinois farmer Mike Jenks, echoing the views of growers across the Midwest.

That view is driving up profit for pesticide makers like FMC Corp. and American Vanguard Corp. They're marketing corn insecticides as a kind of insurance policy that costs $12 to $25 an acre. Root-eating larvae of the flying insect has historically cost U.S. farmers $1 billion in expenses and lost harvest, according to USDA estimates.

Pesticides Unnecessary

Some scientists are skeptical that a return to pesticide use is in the long-term interests of farmers. Soil insecticides don't improve root health or yields when the corn is already producing its own insecticide, according to a paper by University of Illinois scientists published online April 25 by the Journal of Applied Entomology. Iowa State University researchers reached a similar conclusion last year.

Chemical insecticides are simply redundant, said Michael Gray, a University of Illinois entomologist.

"It's pretty clear where the science and the scientific community is on this point," Gray said. "It really does not add much."

Entomologists also warn that the additional insecticide may exacerbate the resistance problem that farmers fear. That's because pairing pesticides with engineered corn exposes insects to extra toxins, delaying maturity. That leads to increased mating between resistant worms, hastening the evolution of rootworms that aren't vulnerable to GMO corn.

Insurance Approach

An EPA panel of scientific advisers warned in March that "the use of a soil insecticide with a Bt hybrid should not be done." The report refers to GMO corn as Bt because it includes a gene from Bacillus thuringiensis, a soil bacterium widely used to control insects.

That echoes a 2012 warning to the EPA from 22 corn entomologists that "an insurance-based approach" to insecticides "will only increase insect resistance."

That concern over resistant rootworms is driving up sales for insecticide makers including American Vanguard, FMC and Syngenta AG, which dispute the scientists' findings.

There haven't been any studies confirming that soil insecticides are speeding the evolution of resistant rootworms, said Peter Porpiglia, head of product developing at Newport Beach, California-based American Vanguard. "It's pretty much conjecture."

Surging Demand

American Vanguard may have the most at stake. Net income jumped 56 percent from 2011 through 2013 and soil insecticides for corn have been "a very material driver," said William Kuser, a spokesman. More than a quarter of its sales come from soil insecticides such as Smart Choice 5G and Aztec, used on about 5 million acres (2 million hectares) of corn.

Farmers' concerns will boost demand for soil insecticides through at least 2015, American Vanguard Chairman and Chief Executive Officer Eric Glenn Wintemute told analysts on a May 1 conference call.

"We will continue to push our message," Wintemute said on the call. "It's a good return on investment to make a soil insecticide application."

FMC runs ads in farming magazines showing insects gorging on corn roots, warning that Bt "protection isn't enough." The Philadelphia-based company promises to boost yields by as much as 11 bushels an acre with its Capture LFR insecticide.

The campaign seems to be working. Profit in its agriculture unit jumped 55 percent from 2011 through 2013, driven by rising North American pesticide sales.

FMC CEO Pierre Brondeau expects sales to increase this year, in part due to "concern over corn rootworm," he said on a May 7 conference call.

Resistance Rare

The company dismisses concerns that promoting Capture for use with Bt corn may accelerate resistance.

"That's not a theory we necessarily agree with," said David Wheeler, FMC's technical business manager for North American crops.

FMC dropped 0.3 percent to $77.99 at the close in New York while American Vanguard declined 1.1 percent to $14.25 and Syngenta depositary receipts also fell 1.1 percent.

Insecticides are effective in controlling grubs, wire worms and other lesser pests, and are appropriate when resistant rootworms are are known to be in a field.

Monsanto says resistant rootworms are rare. Only 0.2 percent of the 37 million acres with Monsanto's rootworm-protected corn have had "unexpected damage" from the insects, said Jeffrey Neu, a spokesman for the St. Louis-based company.

Toxin Overexposure

Grey, the entomologist, said resistance is probably more common. Resistant worms have been found in six Illinois counties and they may be about as common in other states, he said.

Rootworm resistance can occur when farmers plant GMO corn on the same plot year after year, overexposing bugs to toxins. Other growers fail to plant a so-called refuge of conventional corn to deter resistance.

Similar overuse of Monsanto's Roundup herbicide led to the evolution of superweeds that aren't vulnerable to the world's best-selling weedkiller.

Rotating crops, planting a refuge and using a variety of Bt corn types are the best ways to control rootworms, not insecticides, said Gray.

"When these Bt hybrids came into the marketplace, one of the biggest selling features was that producers were going to eliminate the use of soil insecticides," Gray said. "Right now we are basically throwing everything at this insect. It's not a sustainable approach."

See Comments

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Safer Streams

By Anne Russell Gregory, www.defendersblog.org | June 16, 2014 | Last Updated: June 16, 2014 10:33 am

Last week, Defenders and our allies chalked up another courtroom victory – this time, to protect endangered salmon and steelhead from five deadly pesticides in the Pacific Northwest. Although the five pesticides – carbaryl, chlorpyrifos, diazinon, malathion, and methomyl – were designed to kill insects, they either end up killing the endangered fish, destroying their habitat, inhibiting their reproduction, or killing the insects the fish prey on to survive. Now, the Environmental Protection Agency (EPA) has agreed to put new rules in place to keep these pesticides from being used along streams, where the chemicals can leach into the water and cause so much damage.

The National Marine Fisheries Service (NMFS) raised concerns about these pesticides in 2008 and 2009, when their research found that by allowing these substances to be used near streams, the EPA was putting endangered species in further danger of extinction – in direct violation of the Endangered Species Act. NMFS determined that the pesticides would not only put nearly 30 species of salmon and steelhead at risk, but it would also damage the very streams they rely on for survival. NMFS did their part by planting buffer zones along the streams to minimize the damage, but that step alone would not be enough to keep the streams and their inhabitants safe. NMFS ordered the EPA to protect the fish and the streams by prohibiting any aerial spray of the pesticides within 600-1,000 feet of salmon waters, and any ground application of the pesticides within 50-600 feet.

Despite NMFS' order, the EPA refused to implement the recommendation, opting instead for less protective measures. NMFS even wrote to the EPA, explaining that the agency's inferior protections would end up killing juvenile salmon and their prey. However, the EPA continued to ignore the urgent warnings, allowing pesticides to bypass the inadequate buffers and continue to run into the streams. In November 2010, a year after the mandate from NMFS, the EPA still hadn't put any new protections in place. It was a stalemate. NMFS had used all its authority to demand the right protections, but they didn't have the power to force the EPA to obey. So Defenders and our allies stepped in – we filed suit to force EPA to act, and to ensure that pesticides will not jeopardize the survival and recovery of endangered salmon and steelhead. Last week's long-awaited agreement is the result: a necessary step in protecting the endangered salmon, steelhead, and streams in the Pacific Northwest

Under this new agreement, the EPA will prohibit aerial spraying of the pesticides within 300 feet of salmon waters, and ground application within 60 feet. These "no-spray buffers" will remain in place until the EPA works with NMFS to review and analyze the complete impacts the pesticides can have

on the fish and their streams. EPA also agreed to notify pesticide users, state and local governments, and federal agencies about the reinstated buffers, highlighting the applicable streams. This way, farmers will know how and where they can protect salmon and steelhead from pesticides, and can work to develop alternatives.

As Jason Rylander, our senior staff attorney on the case, said "It's kind of a no-brainer that salmon and pesticides don't mix. Today's agreement will go a long way towards ensuring that these highly toxic chemicals stay out of rivers and streams and out of the food chain." This agreement is not only important for salmon and steelhead, but also serves as a wakeup call to EPA that it cannot ignore orders to protect endangered species.

This article was originally published and writted by Anne Russell Gregory, a contributing writer for www.defendersblog.org. For the original story and more information, please click HERE.

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Print Page

By Brian Fraga

June 12. 2014 6:48PM

Two Fall River DPW workers hospitalized after being exposed to pesticide at landfill

State police hazmat team called to examine dump truck carrying toxic substance

FALL RIVER — Two Department of Community Maintenance workers were hospitalized Thursday after being exposed to a toxic pesticide at the Fall River Industrial Park landfill.

Just before 2 p.m., the workers were behind another trash truck when it dumped its load, agitating a white powder that got into the men's faces and immediately caused their eyes to start burning, Fall River Fire Chief Robert Viveiros said.

The DCM workers drove to the Fall River Fire Department's headquarters on Commerce Drive, where medical rescue personnel helped decontaminate and wash out the men's eyes before transporting them to Charlton Memorial Hospital for observation.

Viveiros contacted the Massachusetts state hazardous materials team to investigate and to begin decontamination operations. Meanwhile, the state sent another hazmat team to track down the garbage truck that had dumped the pesticide, Viveiros said.

The driver of that truck, which was located near Fort Devens, also began experiencing his eyes burning, and he was taken to a local hospital for treatment, Viveiros said.

Hazmat officials determined the chemical to be oxybisphenoxarsine, an anti-microbial pesticide that Viveiros said should have been incinerated instead of being taken to the landfill.

PHOTO/ HERALD NEWS PHOTO | JACK FOLEY

Members of a hazardous materials team from the Massachusetts Department of Fire Services look over the city trash truck where two workers were affected by an irritating powder dumped by a private trash hauling truck Thursday at the landfill.

The Massachusetts Department of Environmental Protection also responded to the Fall River Fire Department and accompanied the hazmat team to the landfill, Viveiros said.

Hazmat officials recommended that the two Fall River DCM employees — who had been treated and released — be readmitted to the hospital. Viveiros said they returned to the hospital Thursday evening to be kept for observation.

Meanwhile, the DCM truck remained parked across the street from the Fall River Fire Department headquarters late Thursday.

"We can't do anything with it until the determination is made on how to decontaminate it," Viveiros said.

http://fall-river.wickedlocal.com/article/20140612/NEWS/140618158

Print Page

UPDATE | Wadley woman ID'd after dying from insecticide exposure

Updated: Thu 6:15 PM, Jun 12, 2014 *By: Staff Email*

More Video...


Woman dies from pesticides


Insecticide Death

Thursday, June 12, 2014

JEFFERSON COUNTY, Ga. (WRDW) -- Deputies are investigating the death of a Wadley woman who inhaled insecticide.

Deputies were called Farm Street where someone told 911 that 58-year-old Rosa B. Gilmore-Green was weak an unable to move just before 8 p.m. The Jefferson County Coroner says shed died just before 1 this morning.

Gilmore-Green's 12-year-old grandson lives with her and was admitted into GRU Medical Center Thursday morning, Robert Chalker of the Jefferson County Sheriff's Office said. His condition is unknown at this time.

Gilmore-Green's two grandaughters were also sent to GRU this morning, were examined and released, Chalker said. They were not at Gilmore-Green's residence when she was exposed to the poisonous gas, but at another residence where she was transported before going to Jefferson ER.

The initial incident happened on North Martin Luther King Blvd and she was picked up by EMS from Farm Street.

An investigation showed a family member distributed an agriculture insecticide in her residence earlier in the day, Jim Anderson with Jefferson County Emergency Services said.

The chemical was identified as Aluminum phosphide (Fumitoxin), Anderson said.

"Aluminum phosphide (AlP) is a cheap, effective and commonly used pesticide. However, unfortunately, it is now one of the most common causes of poisoning among agricultural pesticides," **according to the US National Library of Medicine.**

A haz-mat team was called out to decontaminate everyone who was exposed to the woman and the juveniles, including the emergency room and first responders. The emergency room was reopened overnight. No other part of the hospital was exposed to chemical residue.

An autopsy will be performed at the GBI Crime Lab in Atlanta, Ga. to determine the cause of death.

Have information or an opinion about this story? Click here to contact the newsroom.