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Glyphosate Toxicity—Smoke or Fire?

By William Quarles

About 280 million pounds of glyphosate were applied in the U.S. in 2013. Much of the glyphosate is used for weed control in Roundup Ready® genetically modified (GMO) crops, and for weed control in many urban, suburban, and agricultural situations. It is also applied as a desiccant to wheatfields (USGS 2015; Duke and Powles 2009; Cessna et al. 1994).

Exposure is widespread, and is increasing. A private laboratory in the U.S. has found glyphosate in 93% of the people tested (Detox 2016). Glyphosate is present in air, water, soil, and food (Bohn et al. 2014, Darwent et al. 1994, Battaglin et al. 2005; Wagner et al. 2013). Systemic glyphosate cannot be washed off, and residues cannot be destroyed by cooking (Kruger et al. 2014; EFSA 2009). Glyphosate treated crops may be fed to animals, leading to residues of glyphosate in meat. Applicators can be exposed to glyphosate sprays, and others may be exposed through pesticide drift (Williams et al 2000; Kruger et al. 2014). When the new Roundup Ready GMO turfgrass is commercialized, dramatic increases in exposures may occur (Myers et al. 2016).

Glyphosate is overused, and as a result many weeds have become resistant. Sprays have also caused environmental problems, such as destruction of wildlife habitat. Human exposures are likely too high, but good data on actual exposure levels are lacking (Duke and Powles 2009, Myers et al. 2016).

Photo by Ken Hammond courtesy of the USDA

Aerial sprays of glyphosate formulations such as Roundup are applied to genetically modified (GMO) crops for weed control. There is exposure from pesticide drift and from residues in the food supply.

A Troubled Past

Glyphosate has had a troubled regulatory past, marked by changes in classification and interpretation of experimental results. Some of the original toxicology tests were fraudulent. Industrial Biotest Labs (IBT) and Craven Laboratories fabricated data for glyphosate and other pesticides in the 1970s, 1980s, and 1990s. Many toxicology tests had to be repeated (Cox 1995a; Myers et al. 2016).

Glyphosate was first classified as a possible human carcinogen due to increased kidney cancers in mice. There were also increased tumors of thyroid and pancreas in rats. Reanalysis of these experiments and later tests led to an EPA classification of "evidence of non-carcinogenicity for humans" (Cox 1995a; Cox 2004; Dykstra

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Update

and Ghali 1991). Reanalysis of the same data in 2015, led the International Association for Research on Cancer (IARC) to find glyphosate a probable human carcinogen (Guyton et al. 2015).

Smoke or Fire?

EPA evaluations during the 1990s led to the general perception that glyphosate is relatively benign, compared to other pesticides on the market (Dykstra and Ghali 1991). Reviews written by Monsanto employees or consultants show that glyphosate has low toxicity and is not likely to cause human health problems (Mink et al. 2011, Mink et al. 2012; Williams et al. 2000, Williams et al. 2012; Sorahan 2015; Greim et al. 2015; Kier and Kirkland 2013; Kier 2015). Other researchers have concluded glyphosate is a probable human carcinogen (Guyton et al. 2015), is toxic to human cells (Gasnier et al. 2009), a likely endocrine disruptor (Gasnier et al. 2009; Richard et al. 2005; Romano et al. 2010), causes breast cancer cells to proliferate (Thongprakalsang et al 2013), may cause birth defects (Dallegrave et al. 2003; 2007; Paganelli et al. 2010; Garry et al. 2002), may damage DNA and chromosomes (Guyton et al. 2015), may cause neurological damage (Gallegos et al. 2016), and may cause liver and kidney damage (Mesnage 2015ac). Some of these potential problems occurred at high doses, and some at very low doses.

These differences have left the public trying to figure out which of these allegations are just smoke, and which refer to a potentially serious toxic fire. Here we will do a quick review of the problem and try to help clarify the situation.

Biologically Active

Glyphosate is the N-phosphonomethyl derivative of the amino acid glycine. It is an acid, and is usually sold in the form of a salt. Glyphosate is biologically active in many living systems. It is an antibiotic, an enzyme inhibitor, and a metal chelator (Shehata et al. 2013; Mesnage et al. 2015a). Its herbicidal activity is based on inhibition of a key plant enzyme. But it can also inhibit liver enzymes that are responsible for pesticide detoxification in mammals, and can lead to reduced concentrations of those enzymes (Abass et al. 2009; Larsen et al. 2014; Samsel and Senhoff 2013a). In mammals, it can cause oxidative damage to tissues by interference with enzymes in the mitochondrial respiratory chain (Mesnage 2015a; Larsen et al. 2012). Vitamin C in orange juice may help protect against oxidative damage of glyphosate (Youness et al. 2016).

Can Be Toxic

Glyphosate formulations can be toxic if you ingest them. About 4,000 exposures a year are reported to the U.S. poison control centers. Ingestion of the pesticide can lead to gastrointestinal distress, cardiac problems including hypotension, arrhythmias, and cardiac arrest, swelling of the lungs and pneumonitis, oliguria, kidney damage, liver dysfunction, central effects such as seizures, sedation, coma, and death. Death has occurred at blood glyphosate levels of 734 microgram/ml (734 ppm). Peak levels occur at 4-6 hrs, elimination half life is 3-4 hrs, but it can still be present in serum after 5 days (Roberts et al. 2010). Toxicity increases with the volume of surfactant in the formulation (Seok et al. 2011).

In California in the 1980s, glyphosate formulations were the most frequent cause of pesticide illness among landscape workers, and were the third most problematic among agricultural workers. Frequent adverse effects occur partly because it is used often (Cox 1995b; Williams et al. 2000). Occupational exposure can cause eye and skin irritation, rapid heart rate, high blood pressure, nausea, and vomiting (Temple and Smith 1992). In Sri Lanka, field exposure to sprays of glyphosate formulations correlated with chronic kidney damage. However, the results were confounded by contaminated wells that were a co-factor (Mesnage 2015a).

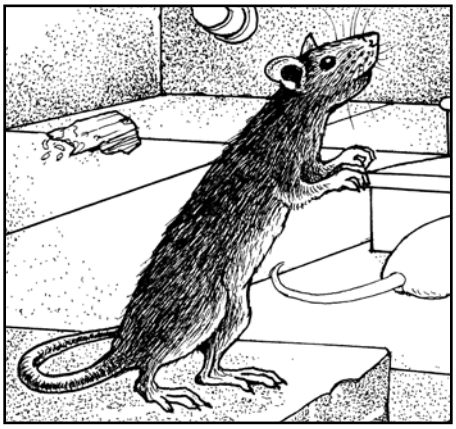
Tests Done on Glyphosate

Many regulatory toxicology tests are done on glyphosate, but the public is actually exposed to formulations containing glyphosate plus other toxic ingredients. Formulations do not receive the same regulatory scrutiny as the active ingredient (Myers et al. 2016).

Glyphosate used in pesticide formulations is about 96% pure and has toxic impurities such as the possible carcinogen N-nitrosoglyphosate. Surfactants in formulations may be contaminated with 1,4-dioxane, which is a likely carcinogen (Williams et al. 2000; Mesnage et al. 2015a).

The preferred regulatory test is oral administration of the active ingredient to rats, but only 20-33% is absorbed. That means glyphosate is 3-5 times more toxic to rats than the oral toxicology tests suggest (Williams et al. 2000). Dermal absorption of glyphosate is likely small (<2%), but dermal absorption of the toxic surfactant polyethoxylated tallow amine (POEA) may be 10% or more. Inhalation toxicity of glyphosate is 10x that of oral doses (Cox 1995a, Williams et al. 2000).

In rats glyphosate is excreted mostly unchanged (97.5%) in urine and feces. Much of it is excreted quickly (6 hrs), but there is a slow second elimination phase (100 hrs). Ingestion of glyphosate every day leads to maximum body concentrations after about 6 days. Glyphosate tends to concentrate in liver, kidney, and bone (Williams et al. 2000).



Drawing by Diane Kuhn

Most toxicology tests are done on glyphosate, not on formulations. Formulations are more toxic.

Formulations More Toxic

Formulations are much more toxic than glyphosate. Roundup is about 125 times more toxic to human cells than glyphosate (Mesnage et al. 2014). Original Roundup is about 400 times more toxic to frogs than glyphosate (Wagner et al. 2013). The acute oral toxicity in rats of the Roundup surfactant polyethoxylated tallow amine (POEA) is about 4x that of glyphosate (Williams et al. 2000). POEA is likely 10,000 times more toxic to human cells than glyphosate (Mesnage et al. 2013). Toxicity in humans of deliberately ingested formulations correlate with the volume of surfactant (Seok et al. 2011). Due to the toxicity of POEA, food containing glyphosate residues should also be tested for POEA.

Applicators are exposed to glyphosate formulations, not pure glyphosate. When formulations are sprayed onto crops, both glyphosate and co-formulants are absorbed by the plant, and presumably both occur in food (Sherrick et al. 1986; Bohn et al. 2014). POEA is routinely found in soil (Tush and Meyer 2016), and degradation products of polyethoxylate surfactants have been found in corn, soybeans, and other foods (She et al. 2012).

Mixtures of glyphosate with other pesticides can have synergistic effects. For instance, glyphosate plus cypermethrin has 4-9 times synergistic toxicity to tadpoles of *Rhinella arenarum* (Brodeur et al. 2014).

Some studies show that co-formulants of Roundup are potential endocrine disruptors (Defarge et al. 2016), and that glyphosate formulations may be associated with adverse developmental effects in rats (Beuret et al. 2004; Dallegrave et al. 2003; Daruich et al. 2001). These studies have been criticized by Williams et al. (2012).

The European Food Safety Authority (EFSA) has determined that glyphosate is not genotoxic. But EFSA believes "genotoxic effects observed in some formulations may be due to other constituents or co-formulants" (EFSA 2016). Europe has banned the use of POEA, and restricted the use of glyphosate formulations in public parks and other areas (EcoWatch 2016).

Probably Carcinogenic

Monsanto consultants have found glyphosate is not a likely carcinogen (Mink et al. 2012; Greim et al. 2015). IARC has found it to be a probable human carcinogen (Guyton et al. 2015). According to IARC, glyphosate produces a dose related increase of a rare kidney carcinoma in mice. It causes an increased incidence of pancreatic adenoma in rats, and blood vessel sarcoma and skin tumor promotion in mice. According to IARC, these studies are sufficient evidence that glyphosate causes cancer in animals (Guyton et al. 2015).

According to IARC, other effects of glyphosate toxicity include induced DNA and chromosome damage in mammals and in human and animal cells in vitro. Human populations exposed to sprays of glyphosate formulations showed increases in blood markers of chromosome damage (Guyton et al. 2015).

According to IARC, case control studies of occupational exposure in the USA, Canada and Sweden show increased risk of non-Hodgkin's lymphoma. These studies count as "limited evidence" of cancer in humans. Evidence was limited because another occupational study, the Agricultural Health Study (AHS), found no association of glyphosate with non-Hodgkin's lymphoma (Guyton et al. 2015). After the IARC review, a study was published showing occupational exposure to glyphosate formulations can lead to an increased risk of melanoma (Fortes et al. 2016).

Current Regulatory Exposure Standards

Though according to IARC, glyphosate will probably cause cancer if sufficient exposure occurs, controversy exists about how much glyphosate is too much.

Through research on toxic doses, regulatory agencies have developed thresholds of glyphosate exposures below which harm is unlikely. This threshold is sometimes called the Reference Dose (RfD) and sometimes the Accepted Daily Intake (ADI). The threshold is usually calculated by finding the lowest dose that causes no observed adverse effect, then dividing by 100.

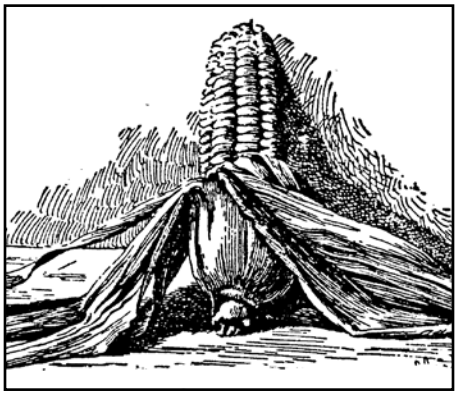
The U.S. EPA RfD for glyphosate is 1.75 mg/kg (1750 ppb) [ppb is parts-per-billion] based on developmental toxicity in rabbits. The European acute reference dose or ADI is 0.5 mg/kg (500 ppb). The Acceptable Operator Exposure Level (AOEL) is 0.1 mg/kg (100 ppb). The U.S. drinking water standard is 0.7 mg/liter (700 ppb) (EFSA 2016, Mesnage 2015a). There are no standards for glyphosate formulations.

A joint WHO/FAO committee has found that anticipated levels of dietary exposure to glyphosate are "unlikely to be genotoxic," and that dietary exposures are "unlikely to pose a carcinogenic risk to humans." The ADI for glyphosate plus its metabolites was 1.0 mg/kg (1000 ppb). The committee emphasized in vivo feeding experiments in mammals (FAO/WHO 2016).

According to the European Food Safety Agency (EFSA) glyphosate is "unlikely to be genotoxic or to pose a carcinogenic threat to humans." They considered only glyphosate, not formulations. The EFSA ADI is 0.5 mg/kg/day (500 ppb), and Acceptable Operator Exposure Levels (AOEL) are below 0.1 mg/kg/day (100 ppb) (EFSA 2016).

Chronic Diseases

A number of studies have been published showing correlations between glyphosate use and a number of chronic diseases, including chronic kidney disease. The papers have also established possible mechanistic links such as oxidative damage and enzyme inhibition that would explain how glyphosate could cause these diseases (Swanson et al. 2014; Samsel and Senhoff 2013ab). These correlations should be tested through a rigorous measurement of glyphosate exposures.

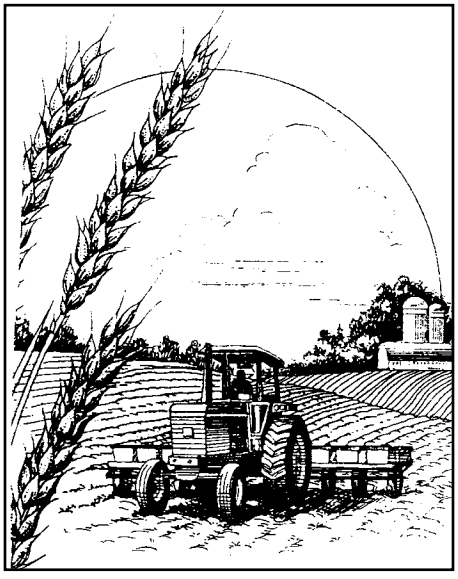


Tolerances for glyphosate residues on corn have increased 50 fold.

But the USDA does not test for glyphosate residues, and the FDA has only started testing this year. Since residue information is not generally available, best estimates are measurement of concentration in urine, or calculations based on probable exposures (Niemann et al. 2015; Williams et al. 2000).

Urinary Studies

Glyphosate has been found in the urine of applicators and the general public. A private testing laboratory has found glyphosate in 93% of people tested in the U.S. (Detox 2016). In Europe 44% of those tested were positive for glyphosate (FOEE 2014). Those who eat organic food have less, and sick people have larger amounts in their urine (Kruger et al. 2014). One experiment showed applicators had average urine concentrations of about 3 ppb and a maximum of 233 ppb. Operator exposures estimated from this experiment were below the European regulatory threshold (Acquavella et al. 2004, Niemann et al. 2015).



Tolerances for glyphosate residues on wheat have increased six fold.

Current Exposure Levels

To separate the smoke from the fire, much more data is needed on exposure levels. The public can be exposed to glyphosate through the diet, by drinking water, by bathing in contaminated water, and by exposure to pesticide drift from sprays, especially aerial sprays. In addition, applicators are vulnerable to spills during mixing, dermal absorption, inhalation, and accidental ingestion of the formulation (Williams et al. 2000).

Estimation of human exposures to glyphosate in the U.S. was published by Monsanto consultants in 2000 (Williams et al. 2000). Dietary exposures were calculated from crop tolerances, or maximum residues allowed on commodities. Since 1999, tolerances have increased from 0.1 to 5 ppm (50x) for corn, from 0.1 to 30 ppm (300x) for oats, from 5 to 30 ppm (6x) for wheat. The tolerance for soybeans has stayed at 20 ppm, but those for other oilseeds except canola have increased to 40 ppm (Benbrook 2016; US Code 2015).

Glyphosate application to U.S. crops was 17,260,209 lbs in 1995, 47,674,779 lbs in 1998, and 249,906,307 lbs in 2014. Glyphosate application rates on crops have increased about 15 fold since 1995 and about 5 fold since 1998 (Benbrook 2016).

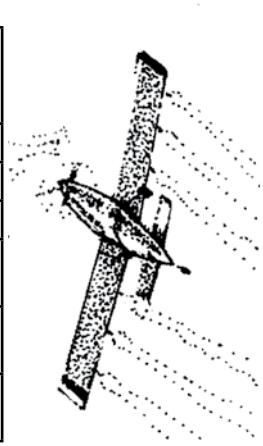
Dietary Exposure

According to Williams et al. (2000), worst case estimated daily exposures to glyphosate through diet in the U.S. were 24 ppb for adults and 52 ppb for children. (See Table 1.) The glyphosate exposures of 1998-1999 should be multiplied by at least 5 to reflect today's situation. Even so, current

Table 1. Estimate of U.S. Glyphosate Exposures. Estimated Exposures in 2016 Reflect a 5-Fold Increase in the Annual Application Rate.*

Type of Exposure	1999 Exposure ppb/day	European Standard ppb/day	Above European Standard?	2016 Exposure ppb/day	Above European Standard?	Above U.S. 1750 ppb/day?
Diet Adult	24	500	no	120	no	no
Diet Child	52	500	no	260	no	no
Acute Child	97	500	no	485	near	no
Acute Child + POEA	188	500	no	940	yes	no
Acute Operator	125	100	yes	625	yes	no
Acute Operator + POEA	288	100	yes	1440	yes	no

*From Benbrook 2016 and Williams et al. 2000



exposures through diet are likely less than the European ADI of 500 ppb (EFSA 2016).

Williams et al. (2000) believed there is no significant dietary exposure to POEA and other co-formulants. But apparently no one has ever actually looked for these residues in food. When formulations are sprayed onto crops, both glyphosate and co-formulants are absorbed by the plant (Sherrick et al. 1986; Bohn et al. 2014), and degradation products of polyethoxylates have been found in corn and soybeans purchased from the supermarket (She et al. 2012). Roundup contains about 50% co-formulants (MSDS 2006).

Acute Exposures

Acute estimated exposures in children for glyphosate were 97 ppb/day. Exposure to glyphosate plus the co-formulant polyethoxylated tallow amine (POEA) were an estimated 188 ppb/day (Williams et al. 2000). Many experiments show that POEA is more toxic than glyphosate (Williams et al. 2000; Mesnage 2015a). Since application rates have increased more than five fold, current acute glyphosate exposures for children may be near the European ADI, and exposure to glyphosate plus POEA could be nearly double the European ADI (EFSA 2016).

Acute aggregate glyphosate exposure to applicators was estimated at 125 ppb/day. Acute aggregate exposures to glyphosate plus POEA were 288 ppb/day (Williams et al. 2000). So acute exposures for applicators in 1998-1999 were likely above the current European regulatory standard of 100 ppb/day. Current exposures for glyphosate may be nearly 6x higher than the European standard. Exposures of glyphosate plus POEA may be 14x above the standard (EFSA 2016). But all estimated exposures are still below the U.S. RfD of 1750 ppb/day. (See Table 1.)

Toxicity Below Regulatory Thresholds

But are the regulatory standards sufficiently protective? Adverse effects used to calculate thresholds are often observation of gross morphology such as birth defects, organ damage, and tumors. But there can be adverse effects on metabolism and the endocrine system that occur at lower levels. Larsen et al. (2012) and Larsen et al. (2014) found signs of oxidative stress and drastically reduced liver cytochrome levels in rats at doses of 90 µg/kg/day (90 ppb). [A microgram, µg, is one-millionth of a gram.] So oxidative stress in rats was seen at doses near current estimated dietary exposures. These doses are also below current regulatory standards (See Table 1.)

Longterm Effects

And adverse effect levels are often set on the basis of short term feeding experiments. But the concern with glyphosate is chronic longterm exposures to low levels. Longterm feeding experiments in rats with doses of 0.1 ppb (0.1 µg/liter) Roundup in drinking water showed toxic effects on liver and kidneys. This amount of Roundup is equivalent to 0.05 ppb (0.05 µg/liter) of glyphosate. The U.S. drinking water standard for glyphosate is 0.7 mg/liter or 700 ppb. Concentrations of glyphosate 14,000 times lower than the U.S. drinking water standard caused measurable toxic effects in rats (Mesnage et al. 2015c).

From water containing 0.05 ppb of glyphosate, rats received a dose of 0.004 µg/kg bw/day (4 ng/kg/day) (Mesnage et al. 2015c). [A nanogram, ng, is one-billionth of a gram.] This dose of glyphosate is 125,000 times lower than the European ADI (Serralini et al. 2014; Mesnage et al. 2015c). This important experiment has been met with a storm of controversy. It has been retracted and republished, and it is now being repeated with technical modifications by two different research groups. If confirmed, current regulatory standards may not be sufficiently protective (Fagan et al. 2016).

Who Do You Believe?

Reviews written by Monsanto consultants have found glyphosate to have low toxicity and probable exposures not likely to cause cancer, endocrine disruption, birth defects, or organ damage. These reviews cover epidemiology (Mink et al. 2011; Mink et al. 2012; Sorahan 2015), genotoxicity (Kier 2015; Kier and Kirkland 2013), developmental effects (Williams et al. 2012), and chronic rat feeding experiments that emphasize glyphosate, not glyphosate formulations (Williams et al. 2000; Greim et al. 2015).

Experiments by independent researchers conducted with cell cultures (Thongprakalsang et al 2013; Gasnier et al. 2009; Richard et al. 2005), or with glyphosate formulations in rats (Bolognesi et al. 1997; Paganelli et al. 2010; Dallegrave et al. 2003; Mesnage et al. 2015c) give toxic findings such as genotoxicity, birth defects, endocrine disruption, cancer cell proliferation, liver and kidney damage.

Lack of agreement may be due to the difference in toxicity between glyphosate and its formulations. For instance, findings of genotoxicity for glyphosate formulations may be due to co-formulants, not glyphosate (Kier and Kirkland 2013, Williams et al. 2012; EFSA 2016).

Disagreements have led to highly technical discussions about who is right and who is wrong. For example, in the case of developmental and reproductive toxicology, possible technical deficiencies in experiments are identified (Williams et al. 2012) and refuted (Defarge et al. 2012; Belle et al. 2012) and rebutted (DeSesso et al. 2012ab). In highly technical squabbles like this, it often boils down to, who do you believe?

Conclusions

IARC has declared glyphosate a probable human carcinogen. But WHO has found that anticipated dietary exposures are not likely to cause cancer.

Rough estimates of probable exposure show some cause for applicator concern, since acute glyphosate exposure to applicators may be above current European regulatory standards. Acute exposures in children may also be above some regulatory thresholds.

If estimated exposures are correct, human dietary exposures to glyphosate are likely below regulatory limits. But some rat experiments have shown that exposure to glyphosate near estimated human dietary levels can cause oxidative stress, and changes in liver enzyme levels.

Glyphosate formulations are more toxic than glyphosate, and public exposure is to formulations. Formulation components persist in the environment, and research is needed to see if they are present in food.

Some studies implicate glyphosate in a number of chronic diseases, but exposures are not documented. To clarify the situation, more work is needed to measure actual human exposures to glyphosate and its formulations.

Whether glyphosate toxicity is identified as smoke or fire may depend on the nature of the experiment. In vivo glyphosate feeding trials in mammals tend to find fewer problems than experiments with formulations or cell cultures.

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William Quarles, Ph.D. is an IPM Specialist, Managing Editor of the IPM Practitioner, and Executive Director of the Bio-Integral Resource Center (BIRC). He can be reached by email at birc@igc.org.

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GMO Labeling Law Passes

More than 90% of the U.S. public wants genetically modified foods (GMOs) to be labeled. Vermont's historical GMO labeling law became effective July 1, 2016. This event increased the pressure on agribusiness to get a national GMO labeling law. A failed effort in the House of Representatives called by opponents, The DARK Act, would have prohibited labeling. After much lobbying, a bill that required labeling was passed by the Senate on July 7, and by the House of Representatives on July 14. President Obama signed it into law on July 29, 2016.

The law allows products to be labeled as GMO's by simple text, by a symbol yet to be determined, or by Quick Response (QR) codes that can be read by smart phones. The QR scan leads to a website that explains GMOs. The law will probably make it harder for those without cell phones to obtain the information. The law also allows the USDA to define GMO, and gene edited food may not have to be labeled.

A Cure for Pierce's Disease

Pierce's disease of grapevines is caused by the bacterium *Xylella fastidiosa*. The bacterium is transmitted to a plant's xylem by insects, usually a leafhopper, such as the glassywinged sharpshooter, *Homalodisca vitripennis*. The growing bacteria form a biofilm that plugs xylem vessels of the plant, preventing the transport of nutrients. As nutrients are not available, the plant starts to decline and die.

Up to now there has not been a cure. Treatments include systemic neonicotinoids that kill leafhoppers, but these have environmental problems, and may impact pollinators.

Pierce's disease was successfully cured by injecting an infected plant with a mixture of 4 naturally occurring bacterial phages. Injection of phages into uninfected plants also protected them against infection. Infections were monitored twice weekly over a 12-week period. Grapevine extracts were monitored for *Xylella* and phages by polymerase chain reaction (PCR).

Xylella spp. bacteria afflict a number of crops including grape, almond, oleander, and coffee. *Xylella* causes olive quick decline that is now killing olive trees in Italy. This phage method might also be used in those cases.

Huanglongbing or citrus greening disease is very similar to Pierce's disease. Huanglongbing is caused by the bacterium *Candidatus* Liberibacter asiaticus that plugs citrus xylem vessels as it grows. A phage has been isolated from citrus trees (Fu et al. 2015. *Plant Dis.* 99(3):320-324), and development of a phage treatment for huanglongbing should be explored.

Das, M., T.S. Bhowmick, S.J. Ahern et al. 2015. Control of Pierce's disease by phage. *PLoS ONE* 10(6):e0128902.

Atrazine Exceeds Levels of Concern

The herbicide atrazine is water soluble, mobile and persistent in the environment. It is a potential endocrine disruptor in mammals and amphibians. It is applied widely, especially in corn growing areas.

The EPA has just published a review of its ecological effects. The EPA found levels of concern for chronic risk were exceeded by 22 times for birds, 198 times for mammals, and 62 times for fish. The EPA also concludes that amphibians are at risk. Concentrations of 5 ppb can lead to reproductive problems in fish and 3.4 ppb will probably impact productivity, structure and function of aquatic plants. These are environmentally relevant concentrations. Also, terrestrial plant biodiversity is likely to be impacted by exposures due to runoff and spray drift.

Because of its potential effect on the endocrine system and ground water, atrazine has been banned in Europe. California recently added it as a reproductive toxicant to the Proposition 65 list.

EPA (Environmental Protection Agency). 2016. *Refined Ecological Risk Assessment for Atrazine.* Environmental Risk Branch III, Office of Pesticide Programs, USEPA, April 12, 2016. 518 pp.

Brassica Seed Meal Protects Apples

Adding organic matter to soil can change the microbial distribution in the rhizosphere and can help protect against plant diseases. Addition of brassica seed meal to soil was just as effective as preplant fumigation with the toxic fumigant 1,3-dichloropropene (Telone® 17) in protecting new apple trees against soil pathogens. Seed meals used were either a mixture of *Brassica juncea* and *Sinapis alba* or a mixture of *B. juncea* and *B. napus*.

After one year, effects on tree health of seed meals were similar to fumigation, but after four years, tree growth and yields were superior in amended soils compared to annual fumigation. Best results were obtained when meals were added to soil in autumn before a spring tree planting. Addition of seed meals near planting dates led to phytotoxicity unless the soil contained large amounts of organic matter.

Overall microbial diversity was not increased, and so increased diversity cannot explain the protective effect. Though there is an initial chemical effect due to glucosinolates from the brassicas, the longterm protective effect was likely due to a change in the microbial populations of the rhizosphere.

Increases were seen in soil microbials such as *Burkholderia* spp., Actinobacteria, sulfur oxidizing bacteria, and bacteria involved in nitrogen recycling. The soil also had larger concentrations of bacteria that metabolize pollutants such as chlorinated hydrocarbons, pyridine, and chlorophenol.

Soil amendments are effective alternatives to toxic soil fumigants for protection against soilborne pathogens.

Mazzola, M., S.S. Hewavitharana, and S.L. Strauss. 2015. Brassica seed meal soil amendments transform the rhizosphere microbiome and improve apple production through resistance to pathogen reinfestation. *Phytopathol.* 105:460-469.

Honey Bees and Fungicides

Fungicides have low acute toxicity to adult bees, and are thus often applied for disease control in flowering crops. However, pollen contaminated with fungicide can poison bee larvae. USDA researchers have found that field relevant levels of the fungicide Pristine® (boscalid and pyraclostrobin) can affect young adult worker bees (3-7 days old). Exposure to fungicide leads to lower rates of pollen consumption, reduced protein digestion, and lower energy (ATP) levels. Bees exposed to the fungicide also had higher levels of viruses.

These effects are similar to what is expected from poor bee nutrition. Exposure to fungicides can weaken a colony, making bees more susceptible to diseases. When fungicides are combined with exposure to neonicotinoids and other insecticides, it is not surprising that the annual honey bee colony loss rate is now about 44%.

Degrandi-Hoffman, G., Y. Chen, E.W. DeJong et al. 2015. Effects of oral exposure to fungicides on honey bee nutrition and virus levels. *J. Econ. Entomol.* 108(6):2518-2528.

Toxic Rat Chow

Regulatory assessments of pesticide toxicology rely on oral feeding experiments in rats. Control rats are fed standard rat chow, and then a pesticide is assayed for toxicity by adding it to the rat chow of test rats. This approach is reasonable as long as the rat chow is not contaminated. French researchers have now shown that standard rat chow is often contaminated with pesticides, heavy metals, and PCBs. All samples were contaminated with pesticide residues, including organophosphates, pyrethroids, fungicides, and herbicides. The most common contaminant was glyphosate, with concentrations up to 370 ppb.

These exposures could explain why test rats often get spontaneous diseases and have such a wide spread in mortality rates (38-83%) over two years. Test rats are often compared not only with internal controls, but with "historical controls." Historical controls could be confounded by variable contamination of rat chow.

Most of the rat chows analyzed contained GMOs. Since rat chow can contain varying amounts of GMOs, contaminated rat chow could explain why GMO toxicology tests often give conflicting results.

Mesnage, R., N. Defarge, L.M. Rocque et al. 2015. Laboratory rodent diets contain toxic levels of environmental contaminants: implications for regulatory tests. *PLoS ONE* 10(7):e0128429.

Monitoring Brown Marmorated Stink Bugs

The brown marmorated stink bug, *Halyomorpha halys*, is an invasive pest that attacks a wide range of crops. Insecticides are often used to control damage, but good monitoring techniques are needed to reduce the number of pesticides applied.

A standard pheromone trap is a black pyramid, 1.22 m (48 in) high, made of plywood that is deployed on the ground. This is baited by pheromones that attract the bugs. In a search for a better trap, USDA tried a coroplast trap with similar dimensions, a smaller ground based coroplast pyramid (29 cm; 11.4 in), hanging pyramid traps, and a semi-pyramid called Rescue.

The coroplast pyramid was the most sensitive, capturing more adults than all other trap designs. Smaller pyramids caught as many adults as the standard trap, but hanging traps caught fewer nymphs.

Morrison, W.R., III, J.P. Cullum and T.C. Leskey. 2015. Evaluation of trap designs and deployment strategies for capturing *Halyomorpha halys*. *J. Econ. Entomol.* 108(4):1683-1692.

Zika Virus Arrives in the U.S.

By William Quarles

The first cases of local mosquito borne Zika in the U.S. were discovered near Miami on July 28, 2016. This discovery has triggered a travel advisory and a restriction on blood collection in that area. There are about 1,600 travel related cases of Zika in the U.S., and any of those could start a mosquito borne epidemic in areas where the yellow fever mosquito, *Aedes aegypti*, or possibly the Asian tiger mosquito, *Aedes albopictus* is present. Travel related cases are likely to increase from U.S. attendance at the Olympics in Brazil.

There is no vaccine for Zika. The virus is transmitted by mosquitoes, blood transfusions, and sexual activity. The virus appears in body fluids such as saliva and urine. Infected men can transmit it sexually to women, and infected women can transmit to men. Most people infected with Zika do not even know they have it. Symptoms are fever, rash, headache and back pain (CDC 2015ab). (See *IPMP* 35(3/4):9, Feb. 2016.)

Zika and Microcephaly

Pregnant women infected with Zika can produce microcephalic babies. Zika can cross the placental barrier, and it targets developing nerve tissue. One of the mysteries is how an infection leads to microcephaly in some cases, and not others. Timing of the infection, cofactors such as toxic exposures, and genetic differences are all possible. Zika might also cause nerve damage that does not lead to microcephaly (Rasmussen et al. 2016).

Zika is present in 20 countries, and it is spreading quickly because exposed populations have no immunity. The World Health Organization declared an international health emergency on February 1, 2016. About 320 pregnant women in the U.S. are infected with Zika, and their progress is being monitored by the CDC (Simeone et al. 2016).

***Aedes aegypti*, shown here, and other *Aedes* mosquito species are able to transmit Zika virus.**

The microcephalic rate apparently varies from country to country. The original infections in Africa in 1947 and on Yap Island in Micronesia in 2007 were not associated with microcephaly (Duffy et al. 2009; CDC 2015a). A study in French Polynesia estimates the birth defect rate at 1% in pregnant women exposed to Zika in the first trimester (Cauchemez et al. 2016).

So far, Brazil has been hit the hardest. Original cases may have been overestimated, but the effect is substantial (Butler 2015). To estimate the birth defect rate, 88 women with a rash were tested for Zika virus, and the PCR test showed 72 had Zika virus in their urine, blood, or both. Infections were identified in week 6 to 35 of pregnancy. Of the 72, 2 miscarried, 42 were tested with ultrasound, and 28 declined. Of the 42 tested, 29% had a fetal abnormality, mostly due to restricted fetal growth. About 75% of the abnormalities resulted from exposure in first trimester (Brasil et al. 2016). Another study has estimated that the risk of microcephaly is about 1-13% if infection occurs in the first trimester (Johansson et al. 2016; Victoria et al. 2016).

Mosquitoes Involved

Zika in Brazil is being vectored by the yellow fever mosquito, *Aedes aegypti*. In the U.S., this species has been found in the Southeast, southern Texas and Arizona, and the San Francisco Bay Area. Another U.S. mosquito, the Asian tiger mosquito, *Aedes albopictus*, might also carry the infection. *A. albopictus* has a similar southern range, but can also be found further north in states such as Pennsylvania and Illinois (CDC 2015b).

These mosquitoes breed in containers around dwellings and bite in the daytime. Discarded automobile tires are a favorite breeding spot. They can be controlled by reducing breeding sources, larval control programs, and by the use of repellents.

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Butler, D. 2016. Microcephaly surge in doubt. *Nature* 530:13-15.

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Duffy, M.R. T.H. Chen, W.T. Hancock et al. 2009. Zika virus outbreak on Yap Island, Federated States of Micronesia. *New Engl. J. Med.* 360 (24):2536-2543.

Johansson, M.A., L.M.Y.T. Romero, J. Reefhuis et al. 2016. Zika and the risk of microcephaly. *New Engl. J. Med.* 375:1-4. July 7, 2016

Rasmussen, S.A., D.J. Jamieson, M.A. Honein and L.R. Petersen. 2016. Zika virus and birth defects—reviewing the evidence for causality. *New Engl. J. Med.* 374:1981-1987. May 19, 2016.

Simeone, R.M., D.M. Delman, E.E. Petersen et al. 2016. MMWR. Possible Zika virus infection among pregnant women United States and territories, May 2016. *MMWR* 60(20), May 27, 2016.

Victoria, C.G., L. Schuler-Faccini, 2016. Microcephaly in Brazil: how to interpret the reported numbers? *The Lancet* 387:621-624.

Special Pheromone Report—2015 ESA Conference

By Joel Grossman

These Conference Highlights were selected from "Synergy in Science," the Minneapolis, Minnesota (Nov. 15-18, 2015) co-meeting of the Entomological Society of America (ESA), the American Society of Agronomy, the Crop Science Society of America, and the Soil Science Society of America. The next ESA annual meeting in Orlando, Florida, Sept. 25-30, 2016, is a joint meeting with the International Congress of Entomology (ICE). For more information contact the ESA (3 Park Place, Suite 307, Annapolis, MD 21401; 301/731-4535; http://www.entsoc.org).

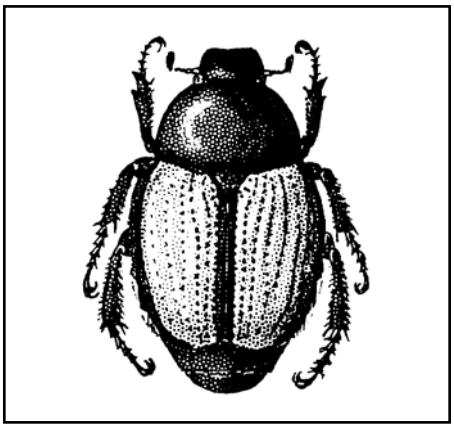
Japanese Beetle Dual-Lure Attract-and-Kill

"Our research evaluated a novel attract and kill device for Japanese beetle (JB), *Popillia japonica*," a pest whose annual cost in 2004 was estimated at $460 million, said Michael Mueller (Univ Michigan, CIPS, East Lansing, MI 48824; muell192@hotmail.com). "Attract and kill is a method of insect management that employs an attractant such as a pheromone and a killing agent. Traditional methods of JB management have relied on mechanical trapping and direct application of insecticides to crops and soil. Disadvantages of these approaches include the need to empty traps daily and exposure of workers, the crop, and beneficial organisms to insecticides."

Vineyard attract-and-kill devices were 10 cm x 10 cm (3.94 x 3.94 in) nylon pouches treated with deltamethrin and containing Trece® JB dual lures. A video camera linked to a DVR recorded duration and numbers of JB approaches and contacts with control devices with and without the deltamethrin toxicant. "The lack of repellency of deltamethrin, high proportion of device contacts and high retention times suggest that the attract and kill device is a promising new JB management tool," said Mueller. Larger field trials are needed to optimize JB pheromone lure release rates and duration, and numbers of control devices per vineyard or unit area. A 3 month lure duration is considered ideal. Alternative toxicants also need evaluation, as hyper-excitation is a sublethal pyrethroid effect.

Pheromones and Sweet Alyssum

Pheromone lures inside deltamethrin-treated pouches that attract and kill Japanese beetles, and interplanted floral resources such as sweet alyssum to nourish asparagus miner natural enemies, are



Japanese beetle, *Popillia japonica*

useful IPM tools for mid-Michigan asparagus fields, said Amanda Buchanan (Michigan State Univ, 349 Food Safety & Toxicol, East Lansing MI 48824; alynn@msu.edu). Japanese beetles, which scar asparagus ferns and reduce yields, contact the kill pouches for 5 seconds and die within 3 hours. The asparagus miner, a specialist miner whose larvae tunnel through the stems and spread pathogens, is attacked by parasitoids responsive to nearby or interplanted flowering plants.

Populations of Japanese beetles were monitored weekly for 9 weeks with pheromone-baited traps and a 25-point grid count survey. "Weekly vacuum samples measured the abundances of asparagus miner adults, parasitoid wasps, predators and herbivores in the floral canopy," said Buchanan. Floral resources planted with asparagus included sweet alyssum, partridge pea, buckwheat, and the control (weeds). Sweet alyssum was the best choice as a companion plant to attract asparagus miner parasitoids, attracting significantly more parasitoid wasps than the other interplants.

Effective Bed Bug Aggregation Pheromone

Alvaro Romero (New Mexico State Univ, 945 College Ave, Las Cruces, NM 88011; aromero2@nmsu.edu reported on bed bug aggregation pheromones. Earlier in 2015, Gries et al. (Simon Fraser Univ, Brit Columbia, Canada) identified a 6-component bed bug aggregation pheromone blend. The five volatile components attracting bed bugs to "safe shelters" are: 1) dimethyl disulfide; 2) dimethyl trisulfide; 3) (*E*)-2-hexenal; 4) (*E*)-2-octenal; 5) 2-hexanone. The sixth pheromone component, histamine, is less volatile and causes arrestment upon contact.

According to Gries et al. (2015), "a blend of all six components is highly effective at luring bed bugs into traps. The trapping of juvenile and adult bed bugs, with or without recent blood meals, provides strong evidence that this unique pheromone bait could become an effective and inexpensive tool for bed bug detection and potentially their control."

"Obnoxious sweetness" was the term used by Kemper in 1926 to describe the smell from bed bug nymphal glands chemically identified in the 1960s as (*E*)-2-hexenal and (*E*)-2-octenal, said Dong-Hwan Choe (Univ California, Entom 382,

Riverside, CA 92521; donghwan.choe@ucr.edu). These two aldehyde chemicals, now known to be part of the bed bug pheromone blend, are also produced defensively after attacks by pharaoh ants, bats and other predators.

Low doses of (*E*)-2-hexenal and (*E*)-2-octenal are also released continuously by bed bugs, and can be extracted from filter paper exposed to bed bugs, said Choe. Most likely (*E*)-2-hexenal and (*E*)-2-octenal are evaporating from a pouch-like dorsal scent gland opening. The health impact on humans from continuous bed bug release of (*E*)-2-hexenal and (*E*)-2-octenal is unknown.

Monitoring Bed Bugs

"Monitoring is the first step of an IPM program," providing knowledge of how many pests and where they are located, said Changlu Wang (Rutgers, Thompson Hall, New Brunswick, NJ 08901; cwang@aesop.rutgers.edu). Monitoring tools for bed bugs have to detect low population levels, both initially and after treatment.

Wang's research group has developed several innovations. They have found bed bugs are attracted to textured surfaces, and they use a special fabric tape. Adding fabric tape to ClimbUp® Interceptor traps increased bed bug capture compared to plastic surfaces. They have also found that bed bugs are attracted to black or red versus white or other colors. Bed bugs are more attracted to vertical shapes than horizontal ones.

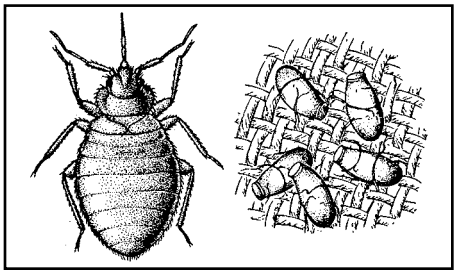
Wang's group has developed inexpensive bed bug traps using dry ice or a yeast mixture as a carbon dioxide source. Costing under $20, the sugar–yeast monitor is basically a bucket into which is mixed 750 grams (26.5 oz) of sugar, 150 grams (5.3 oz) of yeast and 3 liters (6.3 pints) of warm water. Stirring activates the mixture.

A lure combining nonanal, L-lactic acid, 1-octen-3-ol and spearmint oil boosted bed bug capture over 700%; working equally well with dry ice traps and sugar-yeast monitors. A combination of the sugar-yeast monitor and chemical lure (nonanal, L-lactic acid, 1-octen-3-ol and spearmint oil) was affordable and effective for monitoring bed bugs, especially in the absence of human hosts.

Ambrosia Beetle Lures Available

"Recently, the invasive ambrosia beetles in the *Euwallacea fornicatus* species complex, pests of avocado and other woody trees, were discovered in California and Florida," said Allard Cossé (USDA-ARS, 1815 N University St, Peoria, IL 61604; allard.cosse@ars.usda.gov). These ambrosia beetles, carry the Fusarium dieback pathogen. The species in Los Angeles, referred to as the Polyphagous Shot Hole Borer (PSHB), has a wide host range and attacks over 300 tree species, the top eight of which make up 25% of all street trees in southern California.

A kairomone, p-menth-2-en-1-ol has been identified. Enantiomeric mixtures of the kairomone were attractive to the pest in the laboratory and in California avocado orchards. Commercial lures containing p-menth-2-en-1-ol are available for monitoring this serious invasive insect pest.



Bed bug, *Cimex lectularius*, and eggs

Tridecane Attracts Pirate Bug

"Natural enemies such as generalist insect predators in the Chrysopidae (green lacewings), Reduviidae (assassin bugs), Lygaidae (bigeyed bugs), and Anthocoridae (pirate bugs) families utilize brown marmorated stink bug (BMSB), *Halyomorpha halys*, as prey," said Diego Fragal (Rutgers, 96 Lipman Dr, New Brunswick, NJ 08901). "However, the chemical cues used by BMSB predators in host location are largely unknown."

"We observed attraction of adults of the minute pirate bug, *Orius insidiosus,* to green bean pods previously infested with BMSB," said Fragal. "Using Gas Chromatography-Mass Spectrometry (GC-MS) analysis, we identified the alkane hydrocarbon tridecane as a major volatile associated with BMSB-infested bean pods." In field trials, *Orius* spp. were attracted to plants and traps baited with tridecane.

Stink Bug Pheromone Isomer Blends

Known for their stinking "allomonal volatiles, the Pentatomidae or stink bugs include 900 genera and over 5,000 sometimes brightly colored species ranging from beneficial predators such as spined soldier bugs, *Podisus maculiventris*, to pestiferous brown marmorated stink bugs, *Halyomorpha halys*, and harlequin bugs, *Murgantia histrionica*," said Donald Weber (USDA-ARS, Beltsville, MD 20705; don.weber@ars.usda.gov). Male stink bugs from 21 genera produce an even mix of sex pheromones to attract females and aggregation pheromones attracting males, females and nymphs. The pheromone blend mixtures and stereochemistry get quite complex, oftentimes attracting multiple stink bug species.

Murgantiol, the harlequin bug male-produced aggregation pheromone, also attracts brown marmorated stink bugs (BMSB). The key to harlequin bug pheromone attraction is a blend of 16 stereoisomers with the two pheromone isomers SSRS- and SSRR–10,11–epoxy–1–bisabolen–3–ol in about a 1.4:1 ratio. In 2-way field bioassays, all 16 stereoisomers were tested with field-collected wild harlequin bug nymphs and adults. A lure containing 8 isomers (the 2 active isomers plus 6 inactive isomers) was as attractive as the two active isomers alone, and it cost less to manufacture. A lure with all 16 stereoisomers also worked well, provided the two active compounds were present at a 1:1 ratio.

Conference Notes

Stink Bug Pheromones and Trap Crops

Pheromone dependence upon or interaction with green plants was evaluated using "7-way field choice bioassays" combining mixed isomer pheromone lures with collard trap crops and a non-host crop plant, soybean. Wild harlequin bug nymphs and adults showed much greater attraction when aggregation pheromone was combined with green plants. In 2-way choice tests, collards plus pheromone was much more attractive than soybean. However, pheromone lures tricked some bugs into going to the non-host, soybean. But collards plus pheromones was more attractive than soybean plus pheromones.

Glucosinolates and their isothiocyanate breakdown products are key harlequin bug attractants in the plant families Brassicaceae (mustard) and Capparaceae (caper). In a 7-day, 8-way choice test, combinations of the standard pheromone lure with benzyl isothiocyanate and allyl isothiocyanate attracted the most harlequin bugs. Pheromone alone was much more attractive than isothiocyanates alone; but the combination was by far the most effective.

"Host plants greatly increase the attractiveness of pheromone lures," said Weber. "This attractiveness is likely based on distinctive mustard plant volatiles, which presents the opportunity for traps, trap plants and trap crops with greatly enhanced attraction for monitoring and management of harlequin bug."

Fruit Fly Attract-and-Kill

Anamed® (ISCA Tech), a non-toxic, biodegradable emulsion containing oils, waxes, hydrolyzed protein and sugars can be combined with spinosad and "also dispensed on the crop in discrete drops, not the fine mist typically associated with pesticides," said Rodrigo Oliveira da Silva (ISCA, BR 285-Km 336, CEP 98700-000 Ijui- RS, Brazil; Rodrigo.silva@iscatech.com). In lab assays and Hawaii field tests from fall 2014 to spring 2015, Anamed was "highly attractive" to Mediterranean fruit fly, *Ceratitis capitata*; Oriental fruit fly, *Bactrocera dorsalis*; Malaysian fruit fly, *Bactrocera latifrons*; and melon fly, *Bactrocera cucurbitae*.

"Anamed's wax/oil composition provides long term rain and UV protection for the active ingredients, being active for at least 14 days after application," said da Silva. "Anamed doesn't target beneficial insects and also has the ability to reduce fungal issues associated with use of hydrolyzed protein type attractants."

Green Lacewing Pheromones

Lacewing larvae feed on aphids and other pests, and lacewing semiochemicals have pest control potential. According to Laura Breitkreuz (Univ Kansas, 1345 Jayhawk Blvd, Lawrence, KS 66045; l-breitkreuz@ku.edu), "green lacewings (Chrysopidae) number about 1,000 species, and adults have varied glands throughout their bodies secreting semiochemicals diverse in terms of their origins, chemical composition, and function."

"Many males are attracted in large numbers by naturally occurring semiochemicals. One example is *Anklopteryx (Sencera) anomala* in which males are found in aggregations on odor-omitting orchid flowers. This attractance can be tested in field experiments using traps baited with semiochemicals, such as methyl eugenol.

"Available data on semiochemicals come from one of three tribes (Chrysopini), and only 5 of 77 genera in this tribe have been analyzed, although attractance to semiochemicals has been shown in some other groups," said Breitkreuz. Thus far, 26 distinct semiochemicals secreted by lacewings have been identified, and several taxa are attracted to semiochemicals in field experiments. An attractant for *Chrysopa septempunctata* is being commercially developed.

Temperature Affects Pheromone Trap Data

"Early in the season under cool conditions many grape berry moth, *Paralobesia viteana*, males are captured in pheromone traps, but very little damage is observed," said Laura Bizzari (Michigan State Univ,

Calendar

June 23-25, 2016. Annual Meeting, Pest Control Operators CA, Honolulu, HI. Contact: PCOC, 3031, Beacon Blvd, W. Sacramento, CA 95691; www.pcoc.org

July 30-August 3, 2016. American Phytopathological Society Conference, Tampa, FL. Contact: APS, 3340 Pilot Knob Road, St. Paul, MN 55121; 651-454-7250; aps@scisoc.org

August 7-12, 2016. 101th Annual Conference, Ecological Society of America, Ft. Lauderdale, FL. Contact: ESA, www.esa.org

September 15-16, 2016. Fall Meeting BPIA. Arlington, VA. Contact: www.biopesticideindustry.org

September 25-30, 2016. Annual Meeting, Entomological Society of America, Orlando, FL. Contact: ESA, 9301 Annapolis Rd., Lanham, MD 20706; www.entsoc.org

October 18-21, 2016. NPMA Pest World, Seattle, WA. Contact: NPMA, www.npmapestworld.org

November 6-9, 2016. Annual Meeting, Soil Science Society of America. Phoenix, AZ. Contact: www.soils.org

November 6-9, 2016. Annual Meeting, Crop Science Society of America. Phoenix, AZ. Contact: https://www.crops.org

November 6-9, 2016. Annual Meeting, American Society of Agronomy. https://www.acsmeetings.org

January 25-28, 2017. 35th Annual EcoFarm Conference. Asilomar, Pacific Grove, CA. Contact: Ecological Farming Association, 831/763-2111; info@ecofarm.org

January 20-22, 2017. NOFA Winter Organic Farming and Gardening Conf. Saratoga Springs, NY. Contact: NOFA, 585/271-1979; www.nofany.org

February 2017. Annual Conference, Association Applied Insect Ecologists, Napa, CA. Contact: www.aaie.net

February 6-9, 2017. Annual Meeting Weed Science Society of America. Lexington, KY. Contact: www.wssa.net

February 23-25, 2017. 28th Annual Moses Organic Farm Conference. La Crosse, WI. Contact: Moses, PO Box 339, Spring Valley, WI 54767; 715/778-5775; www.mosesorganic.org

March 2017. California Small Farm Conference. Contact: www.californiafarmconference.com

202 CIPS, East Lansing, MI 48824; bizzari@msu.edu). "This pattern is reversed in later generations when temperatures are warmer, few *P. viteana* males are trapped, and damage is a problem at harvest."

Lab assays show grape berry moths live three times longer at 10°C (50°F) than at 28°C (82°F). Also, moth egg laying and mating is very low at 10°C (50°F) but high at 28°C (82°F). This temperature effect can explain why low damage is observed in cool springs when pheromone traps capture many moths. In other words, with many moths living longer at cooler temperatures there may simply be more time for moths to encounter pheromone traps. Later in the season when it is warmer, mating frequency and egg laying is accelerated. Pheromone trap catch data alone does not reflect these biological factors.

Monitoring Root Maggots

"Various plant-derived volatile chemicals attract phytophagous insects, but may also attract their natural enemies," which prompted "screening of such volatile compounds for attraction to natural enemies," said Louis Hesler (USDA-ARS, 2923 Medary Ave, Brookings, SD 57006; Louis.Hesler@ars.usda.gov). While conducting the experiment, there were catches of target flies such as *Delia* root maggots. "2-phenylethanol (2PE) is a component of decomposing onion pulp that attracts onion flies and seedcorn flies," two economically important pest species in the genus *Delia*, said Hesler. This compound can be used to monitor populations of *Delia* pests.

Volatile chemicals such as methyl salicylate or 2-phenylethanol were applied singly to traps either as stock or solvent-based solutions on a cotton dental roll (100 mg ca.) or as a commercial lure (CL; AgBio, Inc). Traps were deployed on 1-meter (3.3 ft) stakes above wheat and soybean canopies and about 2-meters (6.6 ft) high in corn plots.

In the field testing, there were also large catches of non-target fly species. Capture of non-target flies occurred on attractant-baited, yellow sticky traps (Pherocon® AM, Trece, Adair, OK) that were deployed 10- to 30-meters (33-98 ft) apart for various 2-day periods during summer. The biological reason for the attraction is unclear.

Orchard Pheromone Traps and Biocontrol

Pheromone traps are useful for monitoring plum curculio, *Conotracheus nenuphar*, Oriental fruit moth (OFM), *Grapholita molesta,* and codling moth, *Cydia pomonella*, in apples, cherries, peaches, plums and other Eastern USA orchard crops, said Jason Schmidt (Univ Georgia, 2360 Rainwater Rd, Tifton, GA 31793; jschmid2@uga.edu). "Until recently, organophosphate insecticides have been the primary control tactic for plum curculio in tree fruit production." Newer, more expensive chemistries include insect growth regulators (IGRs), oxadazines, and neonicotinoids. But neonicotinoids are under scrutiny for non-target impacts. Hence, the need for alternatives such as biocontrol.

Pheromone traps can be useful in studying biocontrols. Pheromone traps showed cyclical fluctuations in OFM populations during the season. Trap data correlated low plum curculio populations with 9.6% of fruit damage. Vacuum suction within a frame was useful for sampling soil-dwelling predators and potential prey. Predators were hand collected, including 8 types of spiders, true bugs (*Podisus maculiventris* and *Nabis* spp.), rove beetles, tiger beetles, lady beetles and ground beetles. Molecular gut content analysis indicated that predators consumed more plum curculio than OFM.

Varroa Miticides Impact Queen Pheromones

There are "shockingly high and damaging levels of coumaphos (Checkmite®; organophosphate) and fluvalinate (Apistan®; pyrethroid) in honey bee colonies," as beekeepers wage war against the Varroa mite, *Varroa destructor,* with off-label usage of higher doses, said Elizabeth Walsh (Texas A&M, Heep Center, College Station, TX 77843; walshe@tamu.edu). "Sublethal in-hive levels of these miticides have been shown to cause colony-wide health problems," a concern because honey bees are worth $17 billion to USA agriculture, mostly via pollination services.

Walsh's experiments showed that honey bee queens raised in beeswax containing miticides laid fewer eggs than those in miticide free environments. Miticides also affected pheromone concentrations, leading to smaller numbers of workers attending the queen.

According to Walsh, "our results indicate that exposure to miticides during queen development severely alters retinue behavior by impacting the queens' pheromones, which are what the queens use to attract a retinue." One IPM strategy is replacing old combs, as the lipophilic comb wax accumulates miticides.

Drosophila Egg-Laying Repellent

"An aversive odor, 1-octen-3-ol, was evaluated in lab choice and no-choice tests and found to repel female spotted wing Drosophila (SWD), *Drosophila suzukii*, but not males, from an attractive trap," said Anna Wallingford (Cornell Univ, 427 Barton Lab, Geneva, NY 14456; akw52@cornell.edu). High tunnel raspberry field tests were conducted under optimal conditions.

Green raspberry fruit was protectively bagged in the high tunnels to prevent SWD attack until 3 days prior to ripening. SWD egg laying was elevated at dusk, from 5-8 p.m. Thus, Wallingford concluded that dusk is the critical time for using an SWD egg laying repellent such as 1-octen-3-ol.

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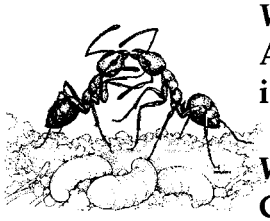

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