The IPM Practitioner
Monitoring the Field of Pest Management
Volume XXXIV, Number 5/6 (Published August 2014)

What Are You Eating? Labeling Genetically Engineered Food (GMOs)

By William Quarles

Photo courtesy of Jennifer Bates

Genetically engineered sweet corn contains systemic insecticides from *Bacillus thuringiensis* (BT) and residues of the systemic herbicide glyphosate. It may be mixed in with the conventional corn you buy at the local supermarket. Mixing is permitted because GMOs are not labeled.

Genetically modified (GM) foods are not labeled, despite the fact that 90% of Americans support labeling (Acres 2012). Consumers are exposed to these new genetic creations and their systemic pesticides without their knowledge. The effects of longterm, widespread exposure to these products have not been fully investigated, and most of the studies supporting their safety have been produced by industry (Domingo and Bordonaba 2011).

Genetically modified organisms (GMOs) are labeled in Europe, but the political process in the U.S. has been paralyzed due to vigorous lobbying by major corporations. Currently, there are labeling laws only in Maine, Connecticut, and Vermont (Caldwell 2013).

When GMOs were introduced, regulators had to decide whether to treat them like food or like drugs and pesticides. New drugs are evaluated through toxicology tests in rats, then clinical tests in humans. Even if these tests prove them safe, adverse reactions can occur as the drug is marketed. For instance, problems with Vioxx® surfaced only after millions of people had taken the drug. Adverse reactions could be identified because the product was labeled, and postmarket surveillance identified the source of the problem (Karha and Topol 2004; Freese 2007).

Conflicting Toxicity Tests

GMOs should be labeled because they are novel foods, containing genes and proteins not found in nature. They should be labeled because short term toxicity tests have given conflicting results, and longterm tests in rodents are few, flawed and hard to interpret. There is vigorous disagreement among researchers, and clinical toxicology tests in humans have never been conducted (Snell et al. 2012; Domingo and Bordonaba 2011; Dona and Arvanitoyannis 2009).

GMOs should be labeled because consumers are exposed to systemic insecticides from *Bacillus thuringiensis* (BT) and systemic herbicides such as glyphosate (Roundup®). Concentrations of glyphosate are often higher in Roundup Ready® crops than in unmodified plants (Bohn et al. 2014). Though it has a generally benign toxicological profile, some studies have shown glyphosate or its formulations can cause birth

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The IPM Practitioner
Monitoring the Field of Pest Management

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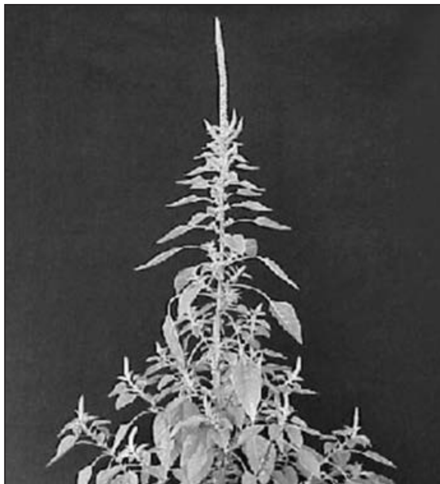

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GM glyphosate resistant crops have led to creation of superweeds such as Palmer amaranth, *Amaranthus palmeri.*

defects and endocrine disruption in animals (Richard et al. 2005; Paganelli et al. 2010; Dallegrave et al. 2003). BT corn can affect the immune system of mice, and it has caused elevated leucocyte and lymphocyte levels in pigs (Finnamore et al. 2008; Walsh et al. 2012). We will probably be exposed to these products throughout our lifetimes.

Environmental Concerns

GMOs should be labeled because many people are concerned about their environmental effects. Critical habitat of the monarch butterfly is being destroyed by aerial sprays of glyphosate associated with Roundup Ready® crops (Pleasants and Oberhauser 2012). Overuse of Roundup is polluting air and water (Chang et al. 2011; Battaglin et al. 2005). Due to glyphosate resistant superweeds, new crops tolerant to 2,4-D and other herbicides are in the pipeline. The result will be increased environmental pollution and more concentrated herbicide residues in food (Green et al. 2008; Quarles 2012; Bohn et al. 2014). GM crops are causing genetic pollution of heirloom plants and organic crops (Mallory-Smith and Zappiola 2008; Duke and Powles 2009). Because insects are becoming resistant to BT crops, GMOs are treated with systemic neonicotinoids that may be causing problems with bees (Hopwood et al. 2013, Quarles 2011; 2014; Goulson 2013).

GMOs should be labeled because that would force segregation of crop products. Strict crop segregation would make it easier to avoid contamination of the food supply with plants engineered to produce drugs. Problems of this type have already occurred. Starlink® corn was meant to be animal feed only, but it got mixed in with the human food supply. The mixup cost industry about $1 billion (MacIlwain 2005).

The Right to Know

GMOs should be labeled because consumers should know what they are eating. Labeling is required for food allergens and food additives. An increasing number of people have food related health problems such as celiac disease (see below), and they should be able to choose what they eat (Armenakas and A.-Armenakas 2013). Food choices are inherent to our genetics. Some things we like, others we don't. Some we are allergic to, some are hard to digest. We are genetically adapted to our food, and an unexpected change of diet can have unforeseen consequences.

GMOs should be labeled because some people may need choices for religious or philosophical reasons. For instance, a Hindu might want to avoid food containing bovine genes. The courts have determined that the way money is spent is speech, perhaps the ability to choose the food we eat is protected

Photo courtesy of Glenda Denniston

Aerial sprays of glyphosate on Roundup Ready® crops destroy the milkweed habitat of the monarch butterfly, *Danaus plexippus.*

speech. According to one scholar, "food choice is important for a number of reasons beyond safety, including its impact on health, its importance to religion, its value in cultural identity, and its importance as self-expression and a form of speech" (Rencher 2012).

Regulation Overwhelmed

GMOs should be labeled because the technology is proliferating and intelligent regulation is being challenged. First generation GMOs were mostly incorporated into animal feed and processed foods. But fresh food is now being commercially developed, including sweet corn, papayas, squash and zucchini; and genetically modified animals such as salmon with growth hormone are in the pipeline. New technology is being explored, hundreds of plants are being transformed, and the resulting flood may overwhelm responsible regulation. Industry is pressuring for no regulation at all since they believe GMOs have been proven safe (Herman and Price 2013; Saurabh et al. 2014; Caldwell 2013).

GMOs should be labeled because our knowledge of genetics is incomplete. There has been an explosion of new knowledge. Only recently have we discovered that many humans are not genetically homogenous. We are actually mosaics, and the genomic content can vary from cell to cell (Zimmer 2013). Exposure to pesticides and toxins can cause inheritable epigenetic changes such as obesity in rats and perhaps people (Skinner 2014). We are finding that gene expression and gene regulation is much more complex than we thought, allowing the development of gene editing and gene silencing technology. Small RNAs can play an important role (Gorman and Maron 2014). (See Box A)

Box A. Do Plant Micro-RNAs Help Regulate Human Genes?

Small RNAs such as micro RNA (miRNA) regulate many plant genes, and newer genetic engineering techniques involve introducing miRNA into plants to silence genes. These techniques have been used to create tobacco without nicotine, coffee without caffeine, corn toxic to the corn rootworm and many others (Saurabh et al. 2014; Parrott et al. 2010).

But does the engineered RNA from these plants produce adverse biological activity in humans? Small RNAs in humans regulate up to one-third of human genes. Dysregulation can lead to cancer and other health problems (Ivashuta et al. 2009; Mitchell et al. 2008; Zhang et al. 2012). Industry researchers in 2010 believed that absorption of plant-produced small RNAs "from food or feed by humans would be an extremely infrequent event" (Parrott et al. 2010).

But in 2012 a paper was published showing that miRNAs from plants are stable to digestion and cooking and can be absorbed by mammals, appearing in blood and organs (Zhang et al. 2012). This fact is not too surprising, as miRNAs are only 20-24 nucleotides (nt) long, and short fragments of DNA are known to be absorbed by mammals (Rizzi et al. 2012). MicroRNAs are stable in the mammalian bloodstream, and have potential as biomarkers for cancer (Mitchell et al. 2008; Chen et al. 2008). They have also been found in breast milk (Jiang et al. 2012; Vaucheret and Chupeau 2012). Another publication found that exogenous RNAs from corn, rice, wheat, microbes, and insects were present in human plasma (Wang et al. 2012).

Zhang et al. (2012) showed that about 5% of miRNAs in humans are of plant origin, and that MIR168a from rice was able to regulate mammalian gene expression, increasing blood LDL levels. The paper generated a firestorm of controversy because if cross kingdom gene regulation from plants to animals occurs, GMO safety assessments will have to consider changes in plant RNA (Petrick et al. 2013; Jiang et al. 2012). Many miRNAs in soybeans, rice and other plants have perfect sequence complementarity to human genes (Ivashuta et al. 2009).

So far, the results of Zhang et al. (2012) have not been confirmed (Dickinson et al. 2013; Chen et al. 2013). But the experiment is testing the limits of our knowledge and has stimulated new research (Witwer and Hirshi 2014).

RNA technology may have been rushed to commercialization, since there is much yet to learn, and research is active. As commercialization increases, regulatory failures may occur due to incomplete knowledge. Labeling will help identify problems.

Scalpel-like Insertions a Myth

Genetic engineering is not a precise science. The intended gene along with a marker and promoter gene is introduced at random into the plant genome by *Agrobacterium tumefacians*, a particle gun, or by other methods (Rizzi et al. 2012). Active plant genes may be silenced, new allergens may be created, mutations and pleiotropic effects may occur (Antoniou et al. 2012; Wilson et al. 2006; Hartung and Schiemann 2014; Parrott et al. 2010).

Pleiotropic effects occur when one gene interacts with another elsewhere on the genome. Changing one gene can cause changes in regulatory genes and gene expression. "Depending on the place of insertion, the introduced gene can alter metabolic pathways or induce the expression of previously silent genes, possibly resulting in increased expression of toxins, antinutrients, or allergens or reduced levels of essential nutrients" (Borchers et al. 2010). For instance, when the gene for bean *alpha*-amylase inhibitor (alphaAI1) was inserted into peas, the pea plant modified the protein and produced an allergen. At least 30 proteins unrelated to the insertion changed concentration, and some of these became more allergenic (Chen et al. 2009).

Few Plants Converted

Only a small percentage of plants are successfully converted, and those converted may be crippled, with poor growth characteristics due to pleiotropic effects. Most GMOs are eliminated in premarket screening, and this may be the most important part of the safety assurance protocol. But not all adverse or unintended genetic changes can be identified at this stage (Antoniou et al. 2012; Domingo and Bordonaba 2011).

An adverse new gene may not be expressed until triggered by an environmental stress. For instance, 8,000 genes changed expression in both GMO and wild type plants, when *Arabidopsis* was drought stressed (Ricroch et al. 2011). Roundup Ready plants show clear changes after glyphosate treatment. Industry researchers admit that unintended changes occur and are expected in GM crops. They also insist that such changes occur with conventional breeding (Herman and Price 2013).

Substantial Equivalence

GMOs are regulated by the principle of substantial equivalence, and "if a new food is found to be substantially equivalent in composition and nutritional characteristics to an existing food, it can be regarded as being as safe as the conventional food" (Domingo and Bordonaba 2011). In the best case, the transformed product is compared with the parental variety grown under the same conditions. Problems include choosing what to compare; ideally it should be everything of biological relevance including nutrients, antinutrients, allergens, and possible toxins (Magana-Gomez and de la Barca 2009). (Antinutrients are enzyme inhibitors or other plant materials that prevent breakdown and absorption of nutrients).

Substantial equivalence tests cannot identify all sources of toxicity.

Significant differences may be found between the GMO and its isogenic equivalent. For instance, the first version of Roundup Ready soybeans had lower levels of isoflavones (Antoniou et al. 2012). But regulators allow comparisons to other cultivars grown over a wide range of conditions. There are wide standard deviations, and nutrients of GM soybeans, for instance, are usually substantially equivalent to soybeans grown somewhere, sometime (Antoniou et al. 2012; Parrott et al. 2010). The FDA has found all of the 148 transgenic events it has evaluated meet the standards of substantial equivalence (Herman and Price 2013).

Unintended Effects

Substantial equivalence works by comparing known gene products, and has a problem finding unintended effects. Finding a new toxin by compositional analysis is difficult. How do you find a needle in a haystack, if you do not know what a needle looks like? So substantial equivalence cannot really identify all sources of toxicity (Magana-Gomez and de la Barca 2009).

New techniques such as proteomics and metabolomics, where large arrays of plant products are analyzed, can help with the evaluation, but these techniques also have weaknesses, including systematic errors, statistical problems, and lack of reproducibility. Even with these techniques, only 50-100 metabolites can be assessed (Chassy 2010; Ricroch et al. 2011).

Lamarck's Revenge and Epigenetic Changes

Lamarck believed that environmental factors could cause inherited changes, and this turns out to be true. Environmental events can cause DNA to be tagged with methyl groups leading to inherited modifications of gene expression called epigenetic changes. Where and how plants are grown can lead to inherited changes in gene expression. These changes allow plants to adapt to local conditions (Batista and Oliveira 2010). Tissue culture techniques used to produce GMOs cause mutations, genomic and epigenetic changes called somaclonal variations independent of transgene events. These changes can complicate the determination of substantial equivalence (Neelakandan and Wang 2012; Smulders and de Klerk 2011; Rodriguez-Enriquez et al. 2011).

Toxicology Tests Flawed

Though many tests have confirmed safety, feeding tests in rats have given conflicting results. There are disturbing disagreements. For instance, industry tests of BT corn (MON 863) showed substantial equivalence and little evidence of toxicological effects (Hammond et al. 2006). A reanalysis of the same data found statistically significant elevation of white blood cell counts and evidence of hepatorenal toxicity (Seralini et al. 2007). Criticisms of

Feeding tests in rats are often flawed and give conflicting results.

Drawing by Diane Kuhn

the reanalysis were that the observed significant differences were either "unrelated to treatment or of no biological or clinical importance" (Doull et al. 2007; Domingo and Bordonaba 2011).

One review (Dona and Arvanitoyannis 2009) concludes that GM foods, "may cause hepatic, pancreatic, renal, and reproductive effects and may alter hematological, biochemical, and immunologic parameters the significance of which remains unknown." Criticism of that research was that the reviewers were biased and emphasized experiments of doubtful validity (Rickard et al. 2010; Domingo and Bordonaba 2011).

The problem is that there are relatively few studies and "no trials have been carried out twice in the same conditions by different research teams" (Snell et al. 2012). To be valid, agreed upon protocols have to be rigorously followed, and there are few longterm studies. One review identified only nine longterm feeding experiments involving rats and mice, and found flaws with all of them. Some of the experiments confirmed safety, others found possible adverse effects (Snell et al. 2012).

The technology is being pushed to the limits, with discussions of "statistically significant but not biologically relevant" and "biologically relevant but not statistically significant" (Bartholomaeus et al. 2013). Bizarre things keep happening that require evaluation by expert panels (Hardisty et al. 2013; Delaney et al. 2013).

Corn and soy GMOs appear in many processed foods such as corn chips, tofu, cereal and other products. Without labeling you will not know where these are.

Photo courtesy of Jennifer Bates

Little Relation to Reality

Most of the animal toxicity tests for GMOs use short term tests under the best case conditions. Toxicology tests rely on carefully chosen strains of healthy rats that are not predisposed to cancer or disease. Only the GMO is tested, not the GMO plus pesticide residues (Snell et al. 2012). Products actually being sold on the consumer market are never tested.

Some industry researchers believe that whole food animal tests are not useful or relevant, and this may be true (Herman and Eckmay 2014). America is not a country populated by healthy rats. We have a wide range of health conditions, including diabetes, food allergies, celiac disease, asthma, organ transplants, infections, cancers, heart disease, and immune deficiency. Old people, children, babies, and pregnant women are more sensitive to toxic exposures. They are unwittingly exposed both to GMOs and the toxic pesticides used to grow them (NRC 1993; US 2012).

Horizontal Gene Transfer

Some people are concerned that the novel genes of GMOs will be integrated into their bodies. In fact, "uptake of DNA fragments obtained from food is a normal physiological process in many species, including humans" (Rizzi et al. 2012). Humans ingest up to one gram of dietary DNA a day. In mice 95% of dietary DNA is degraded beyond detection. But fragments have been detected in the gastrointestinal tract, blood cells, and organs, such as the liver (Rizzi et al. 2012). Transgenic 35S DNA from cauliflower mosaic virus promoter has been found in leucocytes, kidneys, and muscles of rainbow trout fed GM soybean (Parrott et al. 2010).

In one of the few human feeding trials, gene transfer from GM soy to intestinal microflora was detected in some subjects before the start of the experiment, reflecting "long term consumption of GM foods." The transgene survived passage in the small intestine, but was degraded in the large intestine. The researchers did not believe the gene transfer events posed a risk to human health (Netherwood et al. 2006).

Short 300 base pair (bp) segments of the glyphosate tolerance transgene CP4 EPSP have been internalized by human intestinal cells in vitro. So short DNA fragments may survive digestion and reach human blood and organs. But there is no evidence of active transcription or stable nuclear integration in any mammalian tissues or cells examined so far. What effects celiac disease and other intestinal problems have on the situation are unknown (Rizzi et al. 2012; Mansueto et al. 2014).

Fragments of proteins produced by GMO genes may survive digestion. When the blood of 69 Canadian women was analysed, Cry1Ab protein from BT was found in 93% of pregnant women, 80% of fetuses, and 69% of non-pregnant women tested. Herbicides associated with GMOs were also detected. The researchers believed the protein and herbicides were of dietary origin. This experiment should be repeated with better analytic techniques to confirm the results (Aris and Leblanc 2011).

Antibiotic Resistance Genes

Transgenes often contain marker antibiotic resistance genes (ARGs) as part of the construct. Transfer of these resistance genes to bacteria in the human intestine is possible, but is probably a low frequency event (Borchers et al. 2010; Parrott et al. 2010). However, Americans eat about one billion meals a day, and that may increase the odds. Other possible sources of ARGs include feedlots, supermarket meat, drinking water, and medical use of antibiotics (Rizzi et al. 2012; Penders et al. 2013; Peng et al. 2013).

A new allergen in a GM crop such as peas can make unrelated proteins such as ovalbumin, and possibly gluten, more allergenic.

Allergies

Many foods contain toxins or allergens. For instance, kidney beans contain lectins that are toxic until cooked (Herman and Ekmay 2013). Soybeans contain allergenic proteins (Herman 2003). New foods can cause allergies in people that have never been exposed to the allergen before. A case in point is Kiwi fruit; there was a population predisposed to allergy, and no one knew until it was introduced (Borchers et al. 2010).

Transgenic food has to be screened carefully. As mentioned earlier, transgenic expression of a bean protein in peas caused the protein to become an allergen. It also enhanced the immunogenicity of several other pea seed proteins and made the unrelated protein ovalbumin more allergenic (Borchers et al. 2010; Chen et al. 2009). When a Brazil nut allergen was transferred to soybeans, the soybeans caused Brazil nut allergies. GMOs can be allergenic if new proteins produced are cross reactive with known allergens, if they are themselves allergenic, or if they cause increased production of an endogenous allergen (Nordlee et al. 1996; Borchers et al. 2010; Fernandez et al. 2013; Goodman et al. 2013).

Screening a protein for allergy is difficult. One can guess from sequence homology to known allergens, ease of digestion, and serum screening (Crevel 2005; Spok et al. 2005; Goodman et al. 2013). For instance, BT Cry9c from Starlink corn was identified as a possible allergen because it resisted digestion. But not all proteins produced by a GMO can be screened, and only with clinical tests can you know for sure. Even then the sample size would have to be large. Common food allergies involve about four percent of the population, but rare allergies exist (Borchers et al. 2010; Leung et al. 2014).

Processed Food

First generation GMOs appear mostly in processed foods and animal feed. GMO proteins such as BT (Cry1Ab) and glyphosate tolerance enzyme (CP4 EPSPS) are probably denatured by cooking (Hammond and Jez 2011). Cooking partially degrades DNA into smaller fragments. Corn chips and other processed foods contain undegraded DNA fragments of about 200-400 bp. But allergens may not be destroyed by processing (Rizzi et al. 2012; Hammond and Jez 2011). Newer GMOs are being developed that will be eaten without cooking or processing (Saurabh et al. 2014; Parrott et al. 2010).

Problems with GMO Food?

Organizers of a recent American Chemical Society symposium stated, "The safety of transgenic crops, effects on human and animal health, and impact on the environment (such as changes in weed communities, gene flow, and evolution of resistance to pests) remain concerns. Long term effects of using transgenic crops are still not entirely clear, although no scientifically documented health problems have arisen after almost 20 years of consumption of transgenic products" (Rimando and Duke 2013).

But how do we really know about health problems, since the GMOs are not labeled, so no epidemiological studies can be done? If there are problems with GMOs, what should we look for? We should look for problems that exist now that did not exist before they were introduced in 1994. Any kind of dramatic change in the health of the population should be noted. Taking the animal experiments as a clue, we should be alert for gastrointestinal problems. Because new proteins are being ingested, we should look for allergies.

One strange new disease is eosinophilic esophagitis. The esophagous becomes inflamed from food ingestion, leading to strictures. Before the 1990s, it was unknown, and "it is not entirely clear what the pathogenesis might be." Treatment is steroids, proton pump inhibitors, and marked changes in diet (Leung et al. 2014).

An Epidemic of Food Related Diseases

In fact, America is in the midst of an epidemic of food related diseases. The percentage of the population with gastric upset requiring medical attention has increased 5-fold in the last 22 years (US 2012). The percentage of adults with diagnosed type 2 diabetes has nearly doubled in 15 yrs (CDC 2012). The percentage of children with food allergies has increased 50% in 14 years, fatty liver in children has increased nearly 3-fold in 22 years, and the percentage of asthmatics in the general population has increased 15% in 9 years (Jackson et al. 2013; Welsh et al. 2013; Dabelea et al. 2014; Akinbami et al. 2012). Celiac disease has increased four fold in 50 years, gluten sensitivity has increased from negligible numbers to as much as 6% of the population over the last 10 years (Ludvigsson et al. 2013; Mansueto et al. 2014; Leung et al. 2014).

Diseases possibly related to food ingestion and pesticide exposure such as autism, ADHD, and clinical depression have skyrocketed (Shelton et al. 2014; Beseler et al. 2008; Bouchard et al. 2010). Incidence of autism in children 8 years old has more than doubled in 10 years (CDC 2014), and the percentage of children diagnosed with ADHD has increased 42% in 8 years (Visser et al. 2014). The percentage of the population with prescriptions for antidepressants has increased about 5-fold in 20 years, and about 25% of the adult population has a mental illness—mostly anxiety, depression, and mood disorders (US 2012; Reeves et al. 2011).

One theory is that the diseases are caused by changes in our intestinal microbes due to overuse of antibiotics (Blaser 2014). Another theory is that obesity effects spring from epigenetic changes triggered by exposure to toxins and pesticides (Skinner 2014). There are other ideas, such as the hygiene hypothesis (Leung et al. 2014). Certainly, overconsumption of sugar and processed foods could be a factor.

Are GMOs contributing to these problems? We do not know because the food is not labeled, and it is impossible to do retrospective studies. GMOs should also be labeled so that people suffering from these health problems have all the knowledge they need to adjust their diet and lifestyle.

There are large increases in gastric upset, allergies, celiac disease, and sensitivity to wheat and gluten.

Conclusion

GMOs can cause longterm exposures to systemic pesticides with uncertain consequences. Toxicity tests in animals have given conflicting results. Feeding tests in rats are often flawed and hard to interpret. Substantial equivalence tests cannot identify rare toxins or allergens. No clinical trials have ever been done.

There has been a steep increase in food related diseases, and those afflicted have a right to know what they are eating. Labeling is needed to allow epidemiological studies to trace allergies and rare diseases. Labeling is needed because food choice is a part of protected speech.

Genetic engineering technology is profound and promises to feed the multitudes and turn the earth into a new Garden of Eden. But maybe the residents of the new Eden need to know what is in the apple.

Acknowledgement

The author wishes to thank Erin Berg for helping with the research on this article.

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References

Acres. 2012. Americans want GMO-food labeling. *Acres USA* 42(5):10.

Akinbami, L.J., J.E. Moorman, C.S. Bailey et al. 2012. Trends in asthma prevalence, health care use, and mortality in the United States, 2001-2010. CDC, *NCHS Brief No. 94*, May 2012.

Antoniou, M., C. Robinson and J. Fagan. 2012. *GMO Myths and Truths*. Earth Open Source, 123 pp. www.earthopensource.org

Aris, A. and S. Leblanc. 2011. Maternal and fetal exposure to pesticides associated to genetically modified foods in eastern townships of Quebec, Canada. *Reproductive Toxicol.* 31:528-533.

Armenakas, S. and M.A.-Armenakas. 2013. Genetically modified organisms in United States agriculture: mandate for food labeling. *Food Nutri. Sci.* 4:807-811.

Bartholomaeus, A., W. Parrott, G. Bondy et al. 2013. The use of whole food animal studies in the safety assessment of genetically modified crops: limitations and recommendations. *Crit. Rev. Toxicol.* 43(52):1-24.

Batista, R. and M. Oliveira. 2010. Plant natural variability may affect safety assessment data. *Reg. Toxicol. Pharm.* 58:S8-S12.

Battaglin, W.A., D.W. Kolpin, E.A. Scribner et al. 2005. Glyphosate, other herbicides, and transformation products in Midwestern streams, 2002. *J. Amer. Water Resources Assoc.* 41(2):323-332.

Beseler, C.L., L. Stallones, J.A. Hoppin et al. 2008. Depression and pesticide exposures among private pesticide applicators enrolled in the Agricultural Health Study. *Environ. Health Perspectives* Online Sept. 1, 2008.

Blaser, M.J. 2014. *Missing Microbes*. Henry Holt, New York. 273 pp.

Bohn, T., M. Cuhra, T. Traavik et al. 2014. Compositional differences in soybeans on the market: glyphosate accumulates in Roundup Ready GM soybeans. *Food Chem.* 153:207-215.

Borchers, A., S.S. Teuber, C.L. Keen et al. 2010. Food safety. *Clin. Rev. Allergy Immunol.* 39(2):95-141.

Bouchard, M.F., D.C. Bellinger, R.O. Wright et al. 2010. Attention deficit/hyperactivity disorder and urinary metabolites of organophosphate pesticides. *Pediatrics* May 17, 2010.

Update

Caldwell, M. 2013. 5 surprising genetically modified foods. *Mother Jones*, August 5, 2013. www.motherjones.com

CDC (Centers for Disease Control). 2012. Increasing prevalence of diagnosed diabetes—United States and Puerto Rico, 1995-2010. *MMWR Surveillance Summaries*, Vol. 61, No 45, pp. 918-921.

CDC (Centers for Disease Control). 2014. Prevalence of autism spectrum disorder in children aged 8 years—Autism and Developmental Disabilities Monitoring Network, 11 Sites, United States 2010. *MMWR Surveillance Summaries*, Vol. 63 (2):1-13. March 28, 2014.

Chang, F-C., M.F. Simcik and P.D. Capel. 2011. Occurrence and fate of the herbicide glyphosate and its degradate aminomethylphosphonic acid in the atmosphere. *Environ. Toxicol. Chem.* 30(3):548-55.

Chassy, B.M. 2010. Can –omics inform a food safety assessment? *Reg. Tox. Pharmacol.* 58:562-570.

Chen, H., G. Bodulovic, P.J. Hall et al. 2009. Unintended changes in protein expression revealed by proteomic analysis of seeds from transgenic pea expressing a bean *alpha*-amylase inhibitor gene. *Proteomics* 9:4406-4415.

Chen, X., K. Zen and C.Y. Zhang. 2013. Reply to "lack of detectable oral availability of plant microRNAs after feeding in mice." *Nat. Biotechnol.* 31(11):967-969.

Chen, X., Y. Ba, L. Ma et al. 2008. Characterization of microRNAs in serum: a novel class of biomarkers for diagnosis of cancer and other diseases. *Cell Res.* 18:997-1006.

Crevel, R. 2005. Industrial dimensions of food allergy. *Proc. Nutr. Soc.* 64:470-474.

Dabelea, D., E.J. Mayer-Davis, S. Saydah et al. 2014. Prevalence of type 1 and type 2 diabetes among children and adolescents from 2001 to 2009. *JAMA* 311(17):1778-1786.

Dallegrave, E., F.D. Mantese, R.S. Coelho et al. 2003. The teratogenic potential of the herbicide glyphosate-Roundup in Wistar rats. *Toxicol. Lett.* 142:45-52.

Delaney, B., S. Karaman, J. Roper et al. 2013. Thirteen week rodent feeding study with grain from molecular stacked trait lepidopteran and coleopteran protected (DP-004114-3) maize. *Food Chem. Toxicol.* 53:417-427.

Dickinson, B., Y. Zhang, J.S. Petrick et al. 2013. Lack of detectable oral availability of plant microRNAs after feeding in mice. *Nat. Biotechnol.* 31(11):965-967.

Domingo, J.L. and J.G. Bordonaba. 2011. A literature review on the safety assessment of genetically modified plants. *Environ. International* 37:734-742.

Dona, A. and I.S. Arvanitoyannis. 2009. Health risks of genetically modified foods. *Crit. Rev. Food Sci. Nutr.* 49:164-175.

Doull, J., D. Gaylor, H.A. Greim et al. 2007. Report of an expert panel on the reanalysis by Seralini et al. (2007) of a 90-day study conducted by Monsanto in support of the safety of a genetically modified corn variety (MON 863). *Food Chem. Toxicol.* 45:2073-2085.

Duke, S.O. and S.B. Powles. 2009. Glyphosate resistant crops and weeds: now and in the future. *AgBioForum* 12(3/4):346-357.

Fernandez, A., E.N.C. Mills, M. Lovik et al. 2013. Endogenous allergens and compositional analysis in the allergenicity assessment of genetically modified plants. *Food Chem. Toxicol.* 62:1-6.

Finnamore, A. M. Roselli, S. Britta et al. 2008. Intestinal and peripheral immune response to MON810 maize ingestion in weaning and old mice. *J. Agric. Food Chem.* 56:11533-11539.

Freese, W. 2007. Regulating transgenic crops: is government up to the task? *FDLI Update*:14-17. www.fdli.org

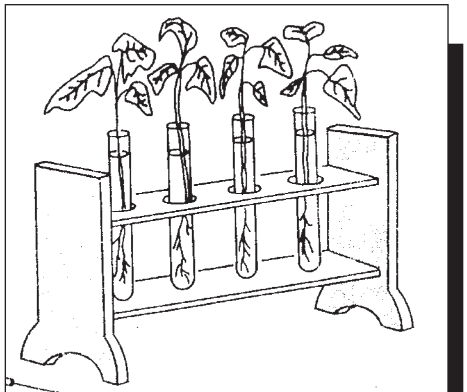
Goodman, R.E., R. Panda, H. Ariyarathna. 2013. Evaluation of endogenous allergens for the safety evaluation of genetically engineered food crops: review of potential risks, test methods, examples and relevance. *J. Agric. Food Chem.* 61:8317-8332.

Gorman, C. and D.F. Maron. 2014. The RNA revolution. *Sci. Amer.* 310(4):53-59.

Goulson, D. 2013. An overview of the environmental risks posed by neonicotinoid insecticides. *J. Appl. Ecol.* 50:977-987

Green, J.M., C.B. Hazel, D.R. Forney and L.M. Pugh. 2008. New multiple-herbicide crop resistance and formulation technology to augment the utility of glyphosate. *Pest Manag. Sci.* 64:332-339.

Hammond, B., J. Lemen, R. Dudek et al. 2006. Results of a 90-day safety assurance study with rats fed grain from corn rootworm protected corn. *Food Chem. Toxicol.* 44:147-160.



Tissue culture causes mutations.

Hammond, B.G. and J.M. Jez. 2011. Impact of food processing on the safety assessment for proteins introduced into biotechnology-derived soybean and corn crops. *Food Chem. Toxicol.* 49:711-721.

Hardisty, J.F., D.A. Banas, C. Gopinath et al. 2013. Spontaneous renal tumors in two rats from a thirteen week rodent feeding study with grain from molecular stacked trait lepidopteran and coleopteran resistant (DP-004114-3) maize. *Food Chem. Toxicol.* 53:428-431.

Hartung, F. and J. Schiemann. 2014. Precise plant breeding using new genome editing techniques: opportunities, safety, and regulation in the EU. *The Plant J.* 78:742-752.

Herman, E.M. 2003. Genetically modified soybeans and food allergies. *J. Exp. Biol.* 54(386):1317-1319.

Herman, R.A. and R. Ekmay. 2014. Do whole food animal feeding studies have any value in the safety assessment of GM crops? *Reg. Toxicol. Pharmacol.* 68:171-174.

Herman, R.A. and W.D. Price. 2013. Unintended compositional changes in genetically modified (GM) crops: 20 years of research. *J. Agric. Food Chem.* 61:11695-11701.

Hopwood, J., M. Vaughan, M. Shepherd et al. 2012. *Are Neonicotinoids Killing Bees?* The Xerces Society for Invertebrate Conservation. Online. 33 pp.

Ivashuta, S.I., J.S. Petrick, S.E. Heisel et al. 2009. Endogenous small RNAs in grain: semi-quantification and sequence homology to human and animal genes. *Food Chem. Toxicol.* 47:353-360.

Jackson, K.D., L.D. Howie and L.J. Akinbami. 2013. Trends in allergic conditions among children: United States, 1997-2011. *CDC NCHS Data Brief No. 121*, May 2013.

Jiang, M., X. Sang and Z. Hong. 2012. Beyond nutrients: food derived microRNAs provide cross kingdom regulation. *Bioessays* 34:280-284.

Karha, J. and E.J. Topol. 2004. The sad story of Vioxx, and what we should learn from it. *Cleveland Clin. J. Med.* 71(12):933-939.

Leung, P.S.C., S-A. Shu and C. Chang. 2014. The changing geoepidemiology of food allergies. *Clin. Rev. Allerg. Immunol.* 46:169-179.

Ludvigsson, J.F., A. Rubio-Tapia, C.T. van Dyke et al. 2013. Increasing incidence of celiac disease in a North American population. *Am. J. Gastroenterol.* 108:818-824.

MacIlwain, C. 2005. US launches probe into sales of unapproved transgenic corn. *Nature* 434:423.

Magana-Gomez, J.A. and A.M.C. de la Barca. 2009. Risk assessment of genetically modified crops for nutrition and health. *Nutr. Rev.* 67(1):1-16.

Mallory-Smith, C. and M. Zapiola. 2008. Gene flow from glyphosate resistant crops. *Pest Manag. Sci.* 64:428-440.

Mansueto, P., A. Seidita, A. D'Alcamo et al. 2014. Non-celiac gluten sensitivity: literature review. *J. Am. Coll. Nutr.* 33(1):39-54.

Mitchell, P.S., R.K. Parkin, E.M. Kroh et al. 2008. Circulating microRNAs as stable blood based markers for cancer detection. *PNAS* 105(30):10513-10518.

Neelakandan, A.K. and K. Wang. 2012. Recent progress in the understanding of tissue culture induced genome level changes in plants and potential applications. *Plant Cell Rep.* 31:597-620.

Netherwood, T., S.M. Martin-Orue and A.G. O'Donnell. 2004. Assessing the survival of transgenic plant DNA in the human gastrointestinal tract. *Nat. Biotechnol.* 22(2):204-209.

Nordlee, J.A., S.L. Taylor, J.A. Townsend et al. 1996. Identification of a Brazil nut allergen in transgenic soybeans. *New Engl. J. Med.* 334:688-692.

NRC (National Research Council). 1993. *Pesticides in the Diets of Infants and Children.* Natl. Academy Press, Washington, DC. 386 pp.

Paganelli, A., V. Gnazzo, H. Acosta et al. 2010. Glyphosate based herbicides produce teratogenic effects in vertebrates by impairing retinoic acid signalling. *Chem. Res. Toxicol.* 23:1586-1595.

Parrott, W., B. Chassy, J. Ligon et al. 2010. Application of food and feed safety assessment principles to evaluate transgenic approaches to gene modulation in crops. *Food Chem. Toxicol.* 48:1773-1790.

Penders, J., E.E. Stobberingh, P.H.M. Savelkoul et al. 2013. The human microbiome as a reservoir of antimicrobial resistance. *Frontiers Microbiol.* 4:1-4.

Update

Peng, S., J.S. Yu, Z.X. Xiang et al. 2013. Metagenomic insights into chlorination effects on microbial antibiotic resistance in drinking water. *Water Res.* 47(1):111-120.

Petrick, J.S., B. Brower-Toland, A.L. Jackson et al. 2013. Safety assessment of food and feed from biotechnology derived crops employing RNA mediated gene regulation to achieve desired traits: a scientific review. *Reg. Toxicol. Pharmacol.* 66:167-176.

Pleasants, J.M. and K.S. Oberhauser. 2012. Milkweed loss in agricultural fields because of herbicide use: effect on the monarch butterfly population. *Insect Conserv. Diversity*, March, 2012. 10 pp.

Quarles, W. 2011. Pesticides and honey bee death and decline. *IPM Practitioner* 33(1/2):1-8.

Quarles, W. 2012. Brave new world—systemic pesticides and genetically engineered crops. *IPM Practitioner* 33(3/4):1-9.

Quarles, W. 2014. Neonicotinoids, bees, birds, and beneficial insects. *Common Sense Pest Control Quarterly* 28(1-4):3-19.

Reeves, W.C., T.W. Strine, L.A. Pratt et al. 2011. Mental illness surveillance among adults in the United States. Centers for Disease Control and Prevention, *MMWR Morbidity and Mortality Weekly Report*, Vol. 60, Supplement. September 2, 2011. 11 pp.

Rencher, K.L.. 2012. Food choice and fundamental rights: a piece of cake or pie in the sky? *Nevada Law J.* 12:418-442.

Richard, S., S. Molemi, H. Sipahutar et al. 2005. Differential effects of glyphosate and Roundup on human placental cells and aromatase. *Environ. Health Perspectives* 113(6):716-720.

Rickard, C. 2010. Response to "health risks of genetically modified foods." *Crit. Rev. Food Sci. Nutri.* 50(1):85-91.

Ricroch, A.E., J.B. Berge and M. Kuntz. 2011. Evaluation of genetically engineered crops using transcriptomic, proteomic, and metabolomic profiling techniques. *Plant Physiol.* 155:1752-1761.

Rimando, A.M. and S.O. Duke. 2013. Human health and transgenic crops symposium introduction. *J. Agric. Food Chem.* 61:11693-11694.

Rizzi, A., N. Raddadi, C. Sorlini et al. 2012. The stability and degradation of dietary DNA in the gastrointestinal tract of mammals: implications for horizontal gene transfer and the biosafety of GMOs. *Crit. Rev. Food Sci. Nutr.* 52(2):142-161.

Rodriguez-Enriquez, J., H.G. Dickinson, R.T. Grant-Downton et al. 2011. MicroRNA misregulation: an overlooked factor generating somaclonal variation? *Trends Plant Sci.* 16(5):242-248.

Saurabh, S., A.S. Vidyarthi and D. Prasad. 2014. RNA interference: concept to reality in crop improvement. *Planta* 239:543-564.

Seralini, G.E., D. Cellier and J.S. de Vendomois. 2007. New analysis of a rat feeding study with a genetically modified maize reveals signs of hepatorenal toxicity. *Arch. Environ. Contam. Toxicol.* 52:596-602.

Shelton, J.F., E.M. Geraghty, D.J. Tancredi et al. 2014. Neurodevelopment disorders and prenatal residential proximity to agricultural pesticides: the CHARGE study. *Environ. Health Perspectives*, June 23, 2014.

Skinner, M.K. 2014. A new kind of inheritance. *Sci. Amer.* 311(2):45-51.

Smulders, M.J.M. and G.J. de Klerk. 2011. Epigenetics in plant tissue culture. *Plant Growth Reg.* 63:137-146.

Snell, C., A. Bernheim, J.-B. BergŽ et al. 2012. Assessment of the health impact of GM plant diets in longterm and multigenerational animal feeding trials: a literature review. *Food Chem. Toxicol.* 50:1134-1148.

Spok, A., H. Gaugitsch, S. Laffer et al. 2005. Suggestions for the assessment of the allergic potential of genetically modified organisms. *Intl. Arch. Allergy Immunol.* 137:167-180.

U.S. Dept. Health and Human Resources, CDC, Natl. Center for Health Statistics. 2012. *Health, United States, 2012.* With special feature on emergency care. 489 pp.

Vaucheret, H. and Y. Chupeau. 2012. Ingested plant miRNAs regulate gene expression in animals. *Cell Res.* 22:3-5.

Visser, S.N., M.L. Danielson, R.H. Bitsko et al. 2013. Trends in the parent report of health care provider-diagnosed and medicated attention-deficit/hyperactivity disorder: United States, 2003–2011. *J. Am. Acad. Child and Adolescent Psychiatry*, 19th November 2013.

Walsh, M.C., S.G. Buzolanu, M.C. Rea et al. 2012. Effects of feeding Bt MON810 maize to pigs for 110 days on peripheral immune response and digestive fate of the Cry1Ab gene and truncated Bt toxin. *PLoS ONE* 7(5):e36141.

Wang, K., H. Li, Y. Yuan et al. 2012. The complex exogenous RNA spectra in human plasma: an interface with human gut biota? *PLoS ONE* 7(12):e51009.

Welsh, J.A., S. Karpen and M.B. Vos. 2013. Increasing prevalence of non-alcoholic fatty liver disease among United States adolescents, 1988-1994 to 2007-2010. *J. Pediatrics* 162(3):496-500.

Wilson, A.K., J.R. Latham and R.A. Steinbrecher. 2006. Transformation induced mutations in transgenic plants: analysis and biosafety implications. *Biotechnol. Genet. Eng. Rev.* 23:209-237.

Witwer, K.W. and K.D. Hirschi. 2014. Transfer and functional consequences of dietary microRNAs in vertebrates: concepts in search of corroboration. 2014. *BioEssays* 36:394-406.

Zhang, L., D. Hou, X. Chen et al. 2012. Exogenous plant MIR168a specifically targets mammalian LDLRAP1: evidence of cross-kingdom regulation by microRNA. *Cell Res.* 22:107-126.

Zimmer, C. 2013. Mosaics: DNA doubletake. *New York Times*, Sept. 16, 2013.

GMO's may be present in many processed foods.

Photo courtesy of Jennifer Bates

2013 ESA Conference Notes

By Joel Grossman

These Conference Highlights are from the Nov. 10-13, 2013, Entomological Society of America (ESA) annual meeting in Austin, Texas. ESA's next annual meeting is November 16-19, 2014, in Portland, Oregon. For more information contact the ESA (3 Park Place, Suite 307, Annapolis, MD 21401; 301/731-4535;http://www.entsoc.org.

Pesticide Treadmills Forever

In the Great Plains and eastern USA Corn Belt states, western corn rootworm, *Diabrotica virgifera virgifera*, is resistant to soil insecticides, corn-soybean crop rotations, and BT proteins engineered into hybrid corn, said Michael Gray (Univ of Illinois, N-305 Turner Hall, Urbana, IL 61801; megray@illinois.edu). Growers seem reluctant to remember that pesticide overuse leads to insect resistance. For a time, corn-soybean crop rotations and BT corn allowed a reduction of soil insecticides, and scouting was used to monitor rootworm populations. But the rootworm is now resistant to rotations and BT corn, so prophylactic soil insecticide has returned.

Rotation resistant western corn rootworms, first detected in the mid-1990s, were a big entomological surprise. Previously, soybean crop rotations were sufficient to break the western corn rootworm life cycle in corn fields. Then perplexed corn growers in Northeastern Iowa and parts of Illinois began finding large numbers of "rotation resistant" western corn rootworms in soybean fields carrying over to corn crops.

At first, western corn rootworms developing in soybean fields had to move back into corn and feed on corn pollen to mature their eggs; and were unable to lay eggs in soybeans. But the rotation resistance traits began to spread, and over time the rootworms became even better adapted to soybeans. Joe Spencer of the University of Illinois showed that elevated levels of a protease inhibitor enable rotation resistant corn rootworms to better tolerate soybeans.

Photo courtesy Peggy Greb USDA

Western corn rootworm, *Diabrotica virgifera virgifera*, is resistant to BT and to crop rotation.

Soil Chemicals

Applications of soil chemicals were the mainstay strategy for many years until BT corn hybrids were developed. With BT resistance, soil chemicals are back in fashion again. Indeed, 47% of Illinois corn growers are once again using soil insecticides; even though costly hybrid BT corn seed, which is also supposed to be preventative, is still being sown. Unfortunately, another soil insecticide lesson from the 1990s is also being ignored; namely, since the 1990s it has been known that soil insecticides increase adult corn rootworm emergence.

Perhaps there is an economic logic to this seeming ignorance of the biological and ecological lessons of the past: Grower seed costs have doubled since 2003, with stacked hybrids containing other traits such as herbicide resistance; but savings from growing hybrids is $7 billion. Thus, BT corn acreage has expanded to where 92% of Illinois farmers surveyed are now using BT corn; and 53% use a 20% structured refuge (non-BT corn) in a mostly futile attempt to prevent resistance. The 20% structured refuge does not work, because of rootworm behavior and mating patterns. Refuge in a bag, the mixing of corn varieties in fields, works a little bit better. Though growers might consider just going back to a non-BT hybrid plus a soil insecticide for continuous corn.

Resistance to BT

The Cry3Bb1 protein, the major trait in BT seeds, marketed primarily by Monsanto, worked well when it started out. But the high pest mortality produced by BT contained the seeds of its own failure. Predictably, continuous exposure to high dose BT proteins since the 1990s has led to the development of resistant corn rootworm populations.

Indeed, in 2013, "significant western corn rootworm larval injury in first-year cornfields" planted to BT hybrids (Cry3Bb1 protein) was confirmed in Illinois. Even "pyramided" BT corn (multiple insecticidal proteins besides Cry3Bb1) plus crop rotations is no longer a panacea, with some areas still sustaining high crop injury. Since "there is no rescue treatment" for corn rootworm, and yield losses can be 15%, soil insecticides are back as a "prophylactic insurance approach." Whether corn growers will actually relearn the lessons of the past *and* do things differently remains an open question; though so far, the answer is an emphatic No.

Conference Notes

Baiting Glucose Averse German Cockroaches

Glucose aversion, which is believed to be inheritable, has rendered ineffective standard German cockroach, *Blattella germanica*, baiting strategies that relied upon mixing a toxicant with glucose, said Alexander Ko (North Carolina State Univ, Raleigh, NC 27695; ko.e.alexander@gmail.com). German cockroaches have apparently adapted to toxic glucose baits via a neural mechanism whereby glucose stimulates deterrent receptor neurons and suppresses gustatory neurons.

Alternative nutrient bait formulations are needed to compete with environmental attractants such as food and dirty dishes in kitchens. Thus, German cockroach glucose responses were compared with responses to alternative sugars such as fructose. High protein and high carbohydrate baits with hydramethylnon as the toxicant were evaluated. Alternative baiting strategies such as alternating protein and carbohydrate baits were also tested.

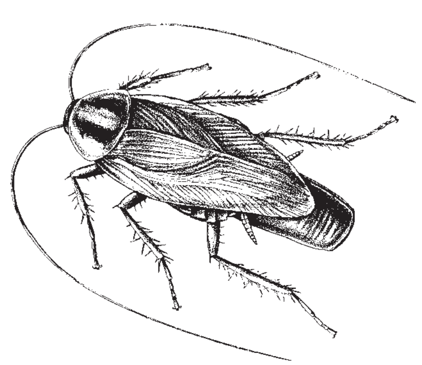
A good IPM strategy is to expose German cockroaches to baits without toxicants for at least three days before introducing the toxicant. A high carbohydrate bait, consisting of three parts carbohydrate and one part protein was considered ideal for glucose averse German cockroaches. Exposing German cockroaches to a nontoxic protein diet before introducing a carbohydrate bait with a toxicant produced the highest mortality. Bait research is continuing, as German cockroach nymphs and adult females have different nutrient needs than males.

Bad Vibrations

"It has been known for decades that plants respond to sound and vibration, but the ecological significance of these responses is unclear," said Heidi Appel (Univ of Missouri, 371 Bond Life Sci Center, Columbia, MO 65211; AppelH@missouri.edu). One source of acoustic energy with particular relevance to plant fitness is the activity of insect herbivores. Indeed, the "bad vibrations" of caterpillar chewing can trigger plant biochemical defense systems.

Arabidopsis thaliana, a mustard widely used in botany experiments, was pretreated with the vibrations caused by caterpillar feeding. Treated plants had higher levels of glucosinolate and phenolic defenses when subsequently attacked by caterpillars of imported cabbageworm, *Pieris rapae*, than did untreated plants or plants treated with the vibrations of wind or other insect sounds.

"Although the way in which plants perceive mechanical vibrations is not well understood, a vibration signaling pathway would complement the known signaling pathways that rely on airborne volatiles or phloem borne signals," said Appel. "We suggest that vibration represents a new long distance signaling mechanism in plants responsible for systemic induction of chemical defenses."



German cockroach, *Blattella germanica*

"Aphids and other insects drop to the ground when they sense the heat and humidity of the mammalian herbivore's breath," said Matan Ben-Ari (Univ of Haifa, Haifa 31905, Israel; matbenari@gmail.com). "But aphids can also use other cues: plant vibration, air movement and visual movement detection. These cues, however, are ambiguous and unreliable as they might stem from environmental conditions such as winds...Since dropping to the ground exposes aphids to unfavorable conditions on the ground, discerning the exact origin of unreliable cues is highly important."

In laboratory experiments, pea aphids, *Acyrthosiphon pisum*, were exposed to varying durations, intensities and synchronizations of reliable cues, such as mammalian breath, and unreliable cues, such as vibrations from wind. "Aphids can differentiate between the possible origins of the unreliable by linking them to a reliable cue," said Ben-Ari. "Asynchronous cues are taken to originate from winds or other environmental perturbations. By carefully noting the cue's sensory source, timing, duration and intensity, aphids reach optimal decisions using limited and sometimes unreliable information" such as plant vibrations or wind gusts.

Neonicotinoid Seed Stimulates Soy Mites

Neonicotinoid insecticide "use as seed treatments for soybean and corn is ubiquitous," said Karly Henry (South Dakota State Univ, 244 AGH Box 2207A, Brookings, SD 57007; karly.henry@sdstate.edu). Spider mites (Tetranychidae) are not susceptible to neonicotinoids, leading to outbreaks after neonicotinoids are applied to diverse plants. Because spider mite outbreaks can cause up to 60% yield loss on soybean, wide use of neonicotinoid seed treatments may have negative impact on soybean production, especially in suboptimal growing conditions.

In South Dakota greenhouse experiments, soybean seeds were treated with a neonicotinoid (CruiserMaxx®, thiamethoxam) or planted untreated; a second set of experiments included short periods of water stress. "When plants were grown in optimal conditions, spider mite abundance was similar between the two treatments," said Henry. Spider mite abundance was significantly higher on neonicotinoid treated plants that were stressed.

Conference Notes

Best Soils for Biocontrol Nematodes

Since "90% of insects have a soil-dwelling stage in their life cycle, entomopathogenic nematodes (EPN) could be an effective biocontrol for soil-dwelling agricultural pests," said Suzanne Yocom (Millersville Univ, Millersville, PA 17551; suzyocom@gmail.com). "EPN perform better in soil textures with higher field capacity," such as sand-clay rather than clay.

"Soil moisture, which facilitates nematode mobility, may be more important than the interstitial space provided by varying soil texture," said Yocom, who used *Galleria mellonella* larvae as experimental hosts for *Heterorhabditis bacteriophora* nematodes. "High organic matter, such as peat, may provide both the interstitial space and field capacity to increase EPN mobility."

Native California Hedgerows Aid Stink Bug Biocontrol

"Hedgerows of native California shrubs and perennial grasses bordering rotational field crops were examined for the abundance of beneficial and pest insects compared to fields with semi-managed weedy field margins," said Rachael Long (Univ of California, 70 Cottonwood St, Woodland, CA 95695; rflong@ucdavis.edu). "During two years of sampling in the Sacramento Valley (2009-10), hedgerows attracted more beneficial insects than pests, resulting in slightly higher biocontrol of stink bug egg masses in processing tomato fields. We conclude that replacing weedy areas on field crop edges with managed hedgerow plantings will increase beneficial insects rather than pest insects on farms and enhance biocontrol of pests in adjacent crops."

According to Long, "our study provides evidence that field edge plantings of native California shrubs and perennial grasses can reduce pest numbers and enhance parasitoid wasps, leading to slightly better biocontrol of stink bugs in processing tomatoes, compared with semi-managed weedy field margins. A cost-benefit model of these benefits, along with pollination benefits from native bees attracted to hedgerows and adjacent crops, is a starting point for valuing the economic benefit of multiple ecosystem services resulting from hedgerows restoration in intensive agricultural landscapes."

Alfalfa Perimeter Traps Immigrating Lygus

"Alfalfa trap crops interplanted in strawberries can suppress the strawberry pest *Lygus hesperus* and reduce associated damage in summer strawberries (Swezey et al. 2007)," said Diego Nieto (Univ of California, 1156 High St, Santa Cruz, CA 95064; dnieto@ucsc.edu). "However, using the same trap crops to interfere with early spring migration of lygus bugs into strawberry fields from weedy borders could add another layer of prevention, further improving yields."

Flowering alfalfa, *Medicago sativa*, is a trap crop for Lygus bugs.

"Typically, lygus bug adults migrate to strawberry fields from surrounding natural or weedy areas during the spring," said Nieto. Due to the strong attraction of alfalfa to *L. hesperus* adults, spring trap crops have the potential to intercept these immigrants, preventing them from immediately establishing populations in strawberries. Alfalfa trap crops can then be selectively treated with insecticides or tractor mounted vacuums to remove these immigrants from the system.

In wild flowering vegetation adjacent to a strawberry field edge in Prunedale, California lygus bugs were marked with an egg white solution so that an ELISA test could distinguish marked immigrants. "Immigrating *L. hesperus* adults were concentrated in alfalfa trap crops relative to adjacent strawberry plants during the two-week study," said Nieto. "Of the marked *L. hesperus* immigrant adults captured, 92% and 85% were collected from alfalfa in 2009 and 2010 respectively. These results show that alfalfa trap crops can be useful in managing pest pressure from the first generation during the spring and early summer."

Fatal Fungal Attraction and Mosquito IPM

Anopheles stephensi, a major Asian mosquito vector of human malaria, is highly attracted to spores of insecticidal fungi such as *Beauveria bassiana* and *Metarhizium anisopliae*, said Thomas Baker (Pennsylvania State Univ, 105 Chem Ecol Lab, University Park, PA 16802; tcb10@psu.edu). In Y-tube choice tests with clean air and different fungal spores, unfed (for 24 hours) female *An. stephensi* avoid *Penicillium* and choose lethal *B. bassiana*.

Nonanol and other attractants released by the fungi are likely involved. But the question of *why* mosquitoes are attracted to a soil fungus (in nature) that is both lethal to them and which they usually never encounter was the more interesting question for Baker. In 1969, Harris et al. writing in the journals *Nature* and *Science* reported that mosquitoes were attracted to and fed on caterpillars; and after feeding were able to lay fertile eggs.

Conference Notes

In 1979, Waage hypothesized that in the Mesozoic era, perhaps 65 million years ago, mosquitoes were feeding on other insect larvae. Then with the appearance of mammals on the Earth's surface, mosquitoes switched hosts from insects to mammals. In 2011, Martel et al. (*PLOS*; "Mosquito Feeding Affects Larval Behaviour and Development in a Moth") reported that yellow fever mosquitoes, *Aedes aegypti*, use Egyptian cotton leaf worms, *Spodoptera littoralis*, as a "host of last resort" when vertebrate hosts are unavailable. Caterpillar biocontrol by mosquitoes proved measurable: 1) slower caterpillar development, 2) lower pupal weight, and 3) caterpillar larvae leaving cotton plants in the lab when mosquitoes were around.

In winter, *B. bassiana* is seen infesting dead flies; and mosquitoes such as *An. stephensi* are attracted to dead compatriots killed by sporulating fungi. In Y-tube choice tests, *An. stephensi* females were more attracted to dead *Heliothis* and *Manduca* caterpillar larvae infected with *B. bassiana* than to healthy caterpillar larvae.

When fungus-killed caterpillars are frozen and then warmed back up to room temperature, it is evident something in the *B. bassiana* spores rather than in the caterpillars themselves attracts female mosquitoes. Mosquitoes prefer dead caterpillars infected with sporulating fungi over uninfected dead caterpillars. Similarly, fungal infected live caterpillars are preferred over uninfected live caterpillars.

These facts have IPM implications. If the proper dose of *B. bassiana* is oil sprayed onto a cloth, more *An. stephensi* are attracted and die, compared to a blank cloth. "Fungal pathogens infect through contact and so applications of spores to surfaces such as walls, nets, or other resting sites provide possible routes to infect mosquitoes in and around domestic dwellings," said Baker. Thus, biocontrol fungi like *B. bassiana* can be used as "novel biopesticides against mosquito vectors that transmit malaria."

Spiraling Whitefly IPM in Florida

Rugose spiraling whitefly (RSW), *Aleurodicus rugioperculatus*, spread from Guatemala to Miami-Dade County in 2009, and is now damaging ornamentals in 17 Florida counties. "Because control of this new invasive species using chemical insecticides in the urban locations is challenging, we evaluated the efficacy of two ecofriendly insecticides, a biocontrol fungus, *Isaria fumosorosea* (PFR 97®) and an insect growth regulator (IGR) (Talus®), which are safe to use in the urban settings," said Vivek Kumar (Univ of Florida, 2725 S. Binion Rd, Apopka, FL 32703; vivekiari@ufl.edu).

Alone or in combination the IGR and the biocontrol fungus were significantly more effective than the unsprayed control. But the biocontrol fungus caused higher mortality than the IGR. By day 10, the biocontrol fungus produced 100% mortality. "Preliminary data from leaf disk bioassays suggests that *Isaria fumosorosea* holds potential for control of RSW populations on host plants," said Kumar.

Horticultural Oil for Asian Citrus Psyllid

"Low volume (LV) aerial and ground sprays have become an important method of application in Florida citrus," said Moneen Jones (Univ of Florida, 2685 SR 29 North, Immokalee, FL 34142; mmjones2@ufl.edu). Standard grower treatment for Asian citrus psyllid (ACP), *Diaphorina citri*, is six sprays of varied insecticides (see *IPMP* Volume 34 December 2013). Horticultural mineral oil can suffocate ACP nymphs, reduce egg-laying, increase adult mortality and decrease incidence of huanglongbing (HLB) or citrus greening disease.

"Our results demonstrate that applications of low volume oil are as effective in controlling ACP as select insecticides when using a threshold approach, and that there is no difference in juice quality or yield," said Jones, who used stem tapping to sample ACP.

Calendar

August 8-10, 2014. NOFA Summer Conference. Umass, Amherst, MA. Contact: Christine Rainville, 508/572-0816; www.nofasummerconference.org

August 9-13, 2014. Annual Conference American Phytopathological Society (APS). Minneapolis, MN. Contact: APS, 3340 Pilot Knob Rd., St. Paul, MN 55121; 651-454-7250; aps@scisoc.org

August 10-15, 2014. 99th Annual Conference Ecological Society of America. Sacramento, CA. Contact: www.esa.org

August 24-27, 2014. Annual Meeting, Structural Pest Control Reg. Officials (ASPCRO). Missoula, MT. www.aspcro.org

September 21, 2014. Bird Conservation Alliance Meeting. St. Louis, MO. Contact: Steve Holmer, Bird Conservation Alliance, 202-88-7490; sholmer@abcbirds.org

September 30, 2014. Deadline Application Ecological Horticulture, Center for Agroecology, Santa Cruz, CA. Contact: CASFS, UC Santa Cruz, 831-459-3240; casfs@icsc.edu

October 4-5, 2014. Hoes Down Festival. Full Belly Farm, Guinda, CA. Contact: Ecological Farming Association, www.hoesdown.org

October 21-24, 2014. PestWorld, NPMA Annual Meeting. Orlando, FL. Contact: NPMA, 10460 North St., Fairfax, VA 22030; 800-678-6722; www.npmapestworld.org

November 16-19, 2014. Annual ESA Meeting. Portland, OR. Contact: ESA, 10001 Derekwood Lane, Suite 100, Lanham, MD 20706; 301/731-4535; http://www.entsoc.org

January 21-24, 2015. 34th Annual EcoFarm Conference. Asilomar, Pacific Grove, CA. Contact: Ecological Farming Association, 831/763-2111; info@eco-farm.org

January 23-25, 2015. NOFA 33rd Annual Organic Farming and Gardening Conf. Saratoga Springs, NY. Contact: NOFA, 585/271-1979; www.nofany.org

January 30-February 3, 2015. Annual Conference, Association Applied Insect Ecologists, Napa, CA. Contact: www.aaie.net

February, 2015. Annual Meeting Weed Science Society of America. Lexington, KY. Contact: www.wssa.net

February, 2015. 26th Annual Moses Organic Farm Conference. La Crosse, WI. Contact: Moses, PO Box 339, Spring Valley, WI 54767; 715/778-5775; www.mosesorganic.org

March 2015. California Small Farm Conference. Contact: www.californiafarmconference.com

March 24-26, 2015. 8th Intl. IPM Symposium. Salt Lake City, UT. Contact: Elaine Wolff, Wolff1@illinois.edu

Conference Notes

Kaolin for Asian Citrus Psyllid

Kaolin clay particle films are certified organic (e.g. OMRI) and do not harm parasitoids providing Asian citrus psyllid biocontrol, said Ki Kim (Univ of Florida, 700 Experiment Stn Rd. Lake Alfred, FL 33850; kidkim@ufl.edu). Choice bioassays, electrical penetration grasp (EPG) and scanning electron microscopy show kaolin particle attachment to insect leg parts, which inhibits leaf-grasping. Plus there is reduced "stylet pathway and xylem ingestion behaviors on kaolin-treated leaf tissue" and increased "time spent in non-probing behaviors." All of which means less potential for ACP to transmit HLB (huanglongbing) or citrus greening disease.

"Choice-tests showed *D. citri* preferentially settled on non-kaolin treated leaves," said Kim. "This demonstrates the potential utility of kaolin in HLB management as a feeding deterrent to reduce transmission of the *Candidatus Liberibacter asiaticus*, a putative causal agent of HLB."

"The level of kaolin required to repel psyllids was 1.2% wt/vol, at which level, the dislodgeable kaolin residue was 56 $\mu g/cm^2$ (361 $\mu g/in^2$)," said Kim. "In the field, cumulative rainfall of 6.6 cm (2.4 in) reduced kaolin residues below the level shown to deter *D. citri* from colonizing and feeding on treated plants."

Turnip Trap Crop for Organic Cabbage

"The yellowmargined leaf beetle, *Microtheca ochroloma*, is arguably the most damaging pest in organic crucifer production in the southern United States," said Rammohan Balusu (Auburn Univ, 301 Funchess Hall, Auburn, AL 36849; balusrr@auburn.edu). "Both adults and larvae feed on the foliage of crucifer crops such as turnip, mustard, radish, napa cabbage, cabbage, collards and watercress, with the potential for major economic loss." However, the beetles show a strong preference for turnip and napa cabbage, which were tested as trap crops to protect cabbage from yellowmargined leaf beetle.

"Perimeter trap cropping tactic was evaluated by using turnip (var. 'Purple top white globe') as trap crop and cabbage (var. 'Farao') as cash crop," said Balusu. Trap crop was planted two weeks prior to cash crop in seven rows of 40 ft (12 m) long, 2.5 ft (0.8 m) wide beds with plant spacing of 3.5 ft (1.1 m) between and 1 ft (0.3 m) within the row.

"Significantly lower densities of *M. ochroloma* adults and larvae were recorded in the cabbage plots bordered by turnip compared to control plots on most sampling dates, which resulted in significantly lower damage ratings," said Balusu. "High densities of *M. ochroloma* were recorded in the turnip border, suggesting that the turnip trap crop was attractive enough to divert *M. ochroloma* away from cash crop. Interestingly, the turnip trap crop had to be treated only once with Entrust® WP (organic formulation of spinosad) to prevent population spill-over."

Missouri Organic Farms Mass Trap Japanese Beetles

"Mass trapping is an effective strategy to manage Japanese beetles, *Popillia japonica*, in blueberry and elderberry plantings" on organic farms in central Missouri, said Jacob Wilson (Lincoln Univ, 900 Chestnut St, Jefferson City, MO 65101; wilsonj@lincolnu.edu). On three organic farms, "an initial survey was done early in the summer to determine the average number of beetles per plant and what part of the field was experiencing the highest insect pressure."

"Traps were placed around the perimeter of the fields at least 5 meters (16.4 ft) from the crop and at least 5 meters (16.4 ft) from each other," said Wilson. "Traps were concentrated in the areas with higher pest pressures. Traps were emptied three times per week and the number of beetles was quantified and recorded. Plant damage was estimated during the study and trapping was continued until beetles were no longer consistently caught in every trap."

In five weeks, with 12-16 traps per farm, 1.12 million to 1.55 million Japanese beetles were trapped per farm. One farm had 2.5% damage in elderberry. The blueberry farm had minimal Japanese beetle damage; less than 1% damage in some areas, versus a still acceptable 9.5% damage level in areas not in close proximity to the traps.

"Further research needs to be conducted in order to determine the optimum number of traps per planted acre for each crop, as well as to ascertain the optimal distance to place traps from the crop and from each other," said Wilson.

Japanese beetle, *Popillia japonica*, is susceptible to mass trapping.

Sunn Hemp Intercrop Benefits Squash

Cover crops add diversity to fields of zucchini and other cucurbits, potentially increasing biocontrol of striped cucumber beetle, *Acalymma vittatum*; spotted cucumber beetle, *Diabrotica undecimpunctata howardi*; weeds, plant pathogens and nematode species in the soil, said Cerruti Hooks (Univ of Maryland, 4112 Plant Sci Bldg, College Park, MD 20742; crrhooks@umd.edu). In Hawaii, sunn hemp, *Crotalaria juncea*, interplanted with zucchini reduced pestiferous soil nematodes, squash leaf diseases and aphid virus transmission.

In Maryland, sunn hemp intercropped with zucchini was handclipped to 45 cm (18 in) and the clippings were allowed to fall between the rows as a mulch. Every other row was strip-tilled, and zucchini was planted into the strip tilled rows; with sunn hemp planted every 2-4 ft (0.6 to 1.2 m).

At 28 days after planting, sunn hemp plots had fewer cucumber beetles and more spiders. At the end of the season, cucumber beetle populations were higher on sunn hemp plants than on the cucurbit crop. Sunn hemp soils also had more bacteria and fungus-eating nematodes, as well as omnivores. Marketable zucchini yields were significantly higher with sunn hemp, even without fertilizer inputs. Hence, sunn hemp intercrops in vegetables are multifunctional. Hooks is also evaluating weed control with sunn hemp.

Beauveria Biopriming Reduces Aphids

Both *Beauveria bassiana* and *Metarhizium anisopliae* have been applied experimentally in Africa to inoculate and "bioprime" faba bean seeds in an effort to protect growing plants from pea aphid, *Acyrthosiphon pisum*; black bean aphid, *Aphis fabae*; and bean leafminer, *Liriomiza* spp. said Juliet Akello (Makerere Univ, Kampala, Uganda; akello@uni-bonn.de).

B. bassiana survives up to four months inside bean plants. On faba beans bioprimed with *B. bassiana*, pea and black bean aphids show reduced development, delayed reproductive onset and a lower fecundity rate; 100% of the untreated faba bean plants were killed by aphids, versus only 30% of plants grown from seed treated with *B. bassiana*.

The inoculation or biopriming involves soaking bean seeds in a fungal spore suspension for four hours. In the aphid experiments, the bioprimed bean plants were given a "boost" 21 days later by adding *B. bassiana* to the root zone. The biopriming and boosting can be done before giving plants to farmers, or farmers can add the boost to the bioprimed seeds after planting.

Bee Protection Heats Up

On June 20, 2014 President Obama published a Presidential Memorandum creating a Pollinator Task Force. The Task Force includes the EPA, the USDA, and several other government agencies. Within 180 days from the date of the memorandum, the Task Force is supposed to develop a National Pollinator Health Strategy, including explicit goals, milestones, and metrics to measure progress. The strategy involves research into a wide range of adverse practices, including pesticide exposures. It includes restoration of pollinator habitat by government agencies. These plans may include: "facility landscaping, including easements; land management; policies with respect to road and other rights-of-way; educational gardens; use of integrated vegetation and pest management; increased native vegetation; and application of pollinator-friendly best management practices and seed mixes."

Following closely on the Memorandum was an EPA Assessment Document, "Guidance for Assessing Pesticide Risk to Bees," published June 23, 2014. This document provides a protocol for testing pesticides that might cause problems for bees.

Worldwide Assessment of 800 Studies

An international group of 29 scientists has produced a massive meta analysis of 800 neonicotinoid studies. The analysis, known as the Worldwide Integrated Assessment (WIA) will be published in the peer-reviewed *Journal of Environmental Science and Pollution Research*. It was undertaken by the Task Force on Systemic Pesticides, a group of scientists affiliated with the International Union for Conservation of Nature (IUCN).

Conclusions from the research were released in a press conference, June 24, 2014. Their conclusions were similar to those in the BIRC publication, "Neonicotinoids, Bees, Birds and Beneficial Insects," published this April in *Common Sense Pest Control Quarterly*. Neonicotinoids are persistent, have serious sublethal effects, are toxic through both acute and chronic exposure, and contaminate soil and water. "In bees, field realistic concentrations adversely affect individual navigation, learning, food collection, longevity, resistance to disease and fecundity. For bumblebees, irrefutable colony level effects have been found, with exposed colonies growing more slowly and producing significantly fewer queens." For further information on the meta analysis, contact Madeleine Chagnon, madeleine.chagnon@gmail.com

Neonic Ban in Europe

On December 1, 2013 a two year European ban on the three neonicotinoids: imidacloprid, chlothianidin, and thiamethoxam was implemented. The ban is on seed treatments, soil application of granules, and foliar application to bee friendly plants. The EPA is currently re-evaluating neonicotinoids in the U.S., but they are moving very slowly. http://www.epa.gov/pesticides/about/intheworks/ccd-european-ban.html

Honey Bee Improvement in Europe?

According to an August 12, 2014 press release from Bayer Crop Science, "New field data from nearly 400,000 bee colonies from 21 countries in Europe and the Mediterranean show that overwintering losses of honey bee colonies – a leading indicator of general bee health – are at their lowest level in years. The non-profit honey bee research association COLOSS (prevention of honey bee COlony LOSSes), which comprises more than 360 scientific professionals from 60 countries, has published new data showing that the overall mortality rate of bees in the 2013/2014 winter was nine percent – losses below 10 percent are considered to be normal." If this information is confirmed, it is certainly good news.

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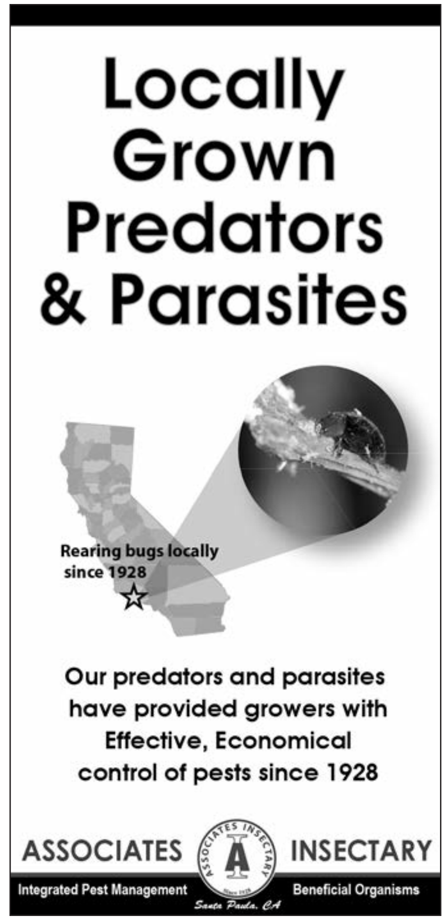
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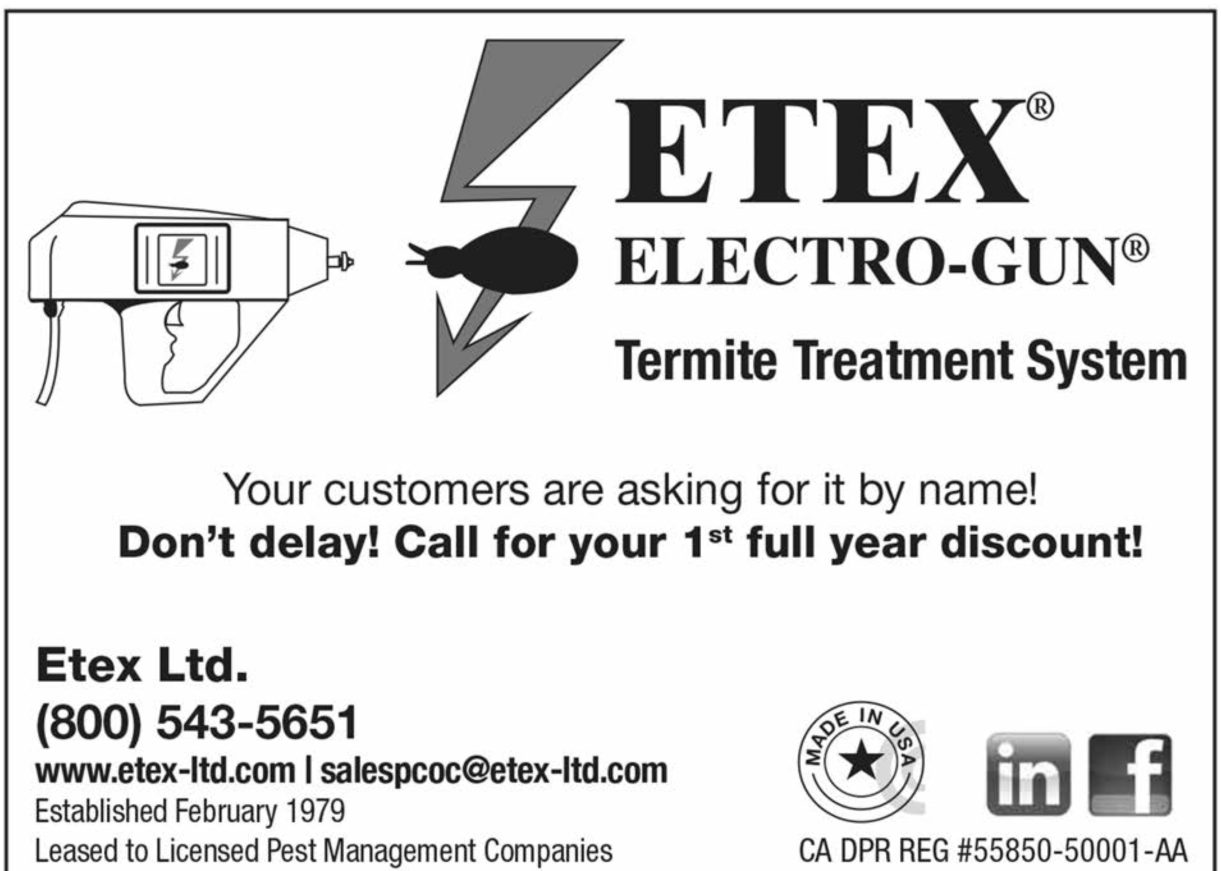
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