Transcript of a talk given by
Dr. Dennis VanEnglesdrop talk at Medina Ohio

As Kim mentioned, I'm part of the Bee Informed membership and we conduct the loss survey every year. We've done that now for 9 years. Five years ago, we switched that survey and we started talking about not just winter loss but summer loss and annual loss. What you can see, which is quite troubling is that our winter loss we had just over 35% (the yellow), has come down a little bit over the peaks we saw earlier. We look at our summer loss we see that that has now surpassed our winter loss. So now we're in a situation where we lost more colonies between April and October than in between October and April. It's astonishing because that's the time when bees should thrive. There's lots of good nourishment out there. It's a great time for bees! So, it's really troubling that we're seeing these high losses in the summer. This is the annual loss of course. You could guess what the summer losses might be. We also ask beekeepers what they think is a reasonable loss over the last year. On average, beekeepers say that they should lose about 15% of their colonies. Could you imagine if a cow farmer said, "Oh yeah, I lost 15% of my cows over the course of the year and I'd be happy?" This is troubling because we've come to accept these very high rates of loss in our industry. I wonder whether we need to; if we need to think of what we consider acceptable rates of loss if they really are acceptable. Traditionally, before varroa mites, before the 1980s, people who lost 10% were too embarrassed to tell their neighbors and now we're happy with 15%, in some cases. Certainly, there has been a big shift. As beekeepers, you know that this shift and this whole idea of colonies dying has really captured the public's imagination. It's meant that we have a lot of people, not necessarily beekeepers, who have really clear ideas on what is causing these losses. Very strong and clear ideas. I'm reminded of a saying from a Baltimore critic, "For every complex problem, there is an answer that is clear; that is simple; that is wrong." The drivers of colony losses are complex. There are several different factors interplaying and playing off of each other and we have to remember that it's broader than one issue.

Although, I'm mandated in this talk to talk about varroa. I certainly think that varroa is a major contributor to colony losses. I'd like to bring up some of the caveats. The first one is is that I recognize when I say I think varroa mites are the major cause of losses in the US, many people get annoyed by this. I've been a beekeeper for 30 years and I'm approaching this whole idea from the point of view of an epidemiologist, not someone who studies individuals but as someone who studies a population. It's very true that people have lost colonies from poor nutrition and pesticides. I'm surprised at the level of varroa mite and how quickly we see varroa mite populations increase at the population level.

The other caveat is that a lot of the data I'm presenting is correlated data. And correlation is not the same as causation. If you want to find the best predictor of colony losses based on correlation, it's actually the number of people who are arrested for Marijuana possession. So, it's inversely proportionate. Now of course, when you see this statistic you all know there is nothing related and this is a fluke. This is not causation. There has to be a biological mechanism there. As sometimes things are co-

variates; they're co-related. For instance, there's a very strong correlation between shark attacks and ice cream sales. That's a very good, strong correlation again. But, that's only because you swim in the summer, which is when you eat most of the ice cream that that correlation exists. I say this because a lot of the data I'm about to show throughout this study is correlative data. Now, the preponderance of evidence — once you've seen correlations year after year, over different populations, and in different situations — just on their own, they're not experimental data; you need experimental data to justify it or you need a lot of other correlative studies to help. For instance, smoking. We have no experimental data that smoking's bad for you. It's all correlative.The reason for that is it would be unethical to cut this room in half and say "You guys smoke two packs a day and you guys don't" and see what happens. Sometimes things aren't set up to do good experiments with because they're unethical or too costly. Correlation has a value but it's not the be-all-end-all. There are four factors affecting colony health: parasites and pathogens, pesticides that farmers and beekeepers are applying to control varroa mite, poor nutrition because of monoculture habitat or the lack of good forage there, and management.

When we do the winter loss survey, we follow it up with a management survey. We ask beekeepers what they think is the major reason your colonies dies this past winter. Right away, the response differs between sub-groups of beekeepers: backyard beekeepers have very different responses than commercial beekeepers. Backyard beekeepers are people who have under 50 hives. Commercial beekeepers have over 500 hives. If we look at backyard beekeepers, they identify a poor winter as the major cause of loss, followed by starvation, and then weak colonies in the fall. These are all manageable conditions. However, if we look at commercial beekeepers, they identify queen failure, followed by varroa mite, and then pesticide as the leading three causes of losses. The queens and varroa mite interchange year by year, in terms of whether they're ranked first or second, but clearly, varroa mites are identified by commercial beekeepers as being a problem. These are guys who are actively managing and they still recognize the problem with varroa mite. This is the silent monster in our midst. It is a very amazingly well-adapted parasite for bees. It's really good at what it does, which is hang on to bees, move into root cells, and have more babies. We'll look at its anatomical features and respect why it's so good. The first thing you'll notice is that it has this really hard shell. It's a hard tic, in fact. Its hard shell keeps it from dehydrating. If you think of other mites, like honey bee tracheal mite, it has to live in the trachea of the bees and dries up really quickly and can't stay out in the environment for too long. However these mites have a very thick shell, its exoskeleton, which allows them to survive outside of the hive, on the bottom board of the hive, or outside of the food chamber for extended periods of time.

Varroa Mite Biology

The varroa mite has all these sensory hairs but they also have a line of hard and nail-like hairs on the outside and that helps deter other bees from chewing on that mite. It deters rooming and it's those sharp projectile spines that mites have. There's also a wax coating that absorbs the smell of the colony, which acts as a chemical camouflage. It smells like a colony, which are really dark and humid. So now, the bees would rely on

their sense of smell to get rid of intruders and now, it smells just like the colony and it adapts well to it because that wax is able to absorb the smell of the colony.

Looking at the feet of the mite, they're a little bit different in the fact that they still have these claws to grab onto things but you'll notice that they have all these little hairs here. Honeybees have a pad there, like a suction cup pad, which allows them to walk upside down. These mites use these hairs and in fact, these hairs are a lot like the hairs on a gecko's feet. It creates this really large surface area so, it's the attraction of molecules that keeps the mite able to walk on the vertical surface. It's causing this huge surface area and so it's molecular bonding occurs very weakly but because the mite is so light and because of that tremendous surface area, it's able to crawl upside down or up the cone or sideways. It's gotten very good at moving around both on the mite and away from the mite.

It also has a very special tube that allows it to breathe. One of the first things it does is when it enters a breed cell, it buries down into the brewed food, that pool of liquid food that the bees are breeding so this tube allows it breathe while it's submerged in that surface. It's adapted for that behavior as well. That really strong outer shell, its carcass, that exoskeleton is also really thin, which allows it slips between the plates on the abdomen of the bee and get right in there and feed between that soft tissue that connects the hard plates.

Varroa Treatments

Now, I'm going to talk about how to make your own varroa treatments so you can control mites. It's totally illegal and you can never repeat it! Of course, maybe a year or two ago if you were a commercial beekeeper and you were asking me what you should treat, I would tell you to call other commercial beekeepers. That's because there were lots of different products that were a lot cheaper to make yourself and apply to the hive. Those days are over now. The only two products you can use don't work anymore. Amitraz you can't buy in a way that you can make your own product anymore that doesn't have high levels of xylene in it or you have to smuggle it into the country. So there is no secret backyard recipe. What's going to happen next? There certainly is research out there that may be suggestive and may be helpful the fact is that most of those products are years away. We're stuck with what we have right now and we have to figure out how to use it most effectively and more efficiently in a way that's going to sustain until these new products come out. So here I'm going to come up with the last little quote that there is no silver bullet and frankly, you don't need one. It is far more important to "find the right kind of gun, load the gun, and most importantly, be able to figure out where the werewolf is." Really, what we are doing is hunting mites in our operation. If you are a hunter or fisherman, you know that the first thing you need to do is to figure out the behavior of the fish. One of the first people to work for me, Mike Andre, he was a trout fisherman who would go fly fishing and he would talk for hours about how he would select his fly. Sometimes they had to be tied green, sometimes brown, depending on what grasshoppers were jumping into the river. He had to know exactly how that fish behaved, what they were eating at that time of year to know how to

catch those trout. For us, we need to have a very comprehensive understanding of the mite and mite biology in order for us to treat mite problems.

For this talk, I want to break it up into three sections, first talking about the behavior of the mites. What makes a mite a mite, the behavior of that mite, the vulnerabilities, and how we get rid of it. We'll also talk about the life cycle of the varroa mite and where it belongs in the tree of life and that might give us some insight on how it behaves and what it does. Then we want to spend some time understanding the population dynamics of varroa mites, especially moving away from just thinking about varroa. Some would argue that mites aren't the things killing your colonies, but the viruses they transmit that are killing them. We'll conclude with some treatment options and strategies.

Mite Background

This is the tree of life in the euchareates, more advanced so it doesn't include some of the bacteria or archaebacteria, which live in thermal vents. If we look at the animal kingdom, we see the protists, which includes nosema as a protist that became the fungi. We can also see the vertebrates breaking off. The mollusks where we can see the crabs and crustaceans. We also see the arthropods. I can't say enough about them. 80% of life on earth is an arthropod. There are over 100 million ants for every single person on earth. Ants alone weigh more than all the mammals, which includes people, cattle, elephants, and all the wild life. Ants weigh more than all of those collectively. Arthropods are the most successful organisms on earth, and they include bees and mites. If we look at the extent, which means the existing or living arthropods on earth today, you can see it includes the spiders, or the chelicerates. That's where the mites are included. Recently, they thought the spiders and insects were really closely related but they've reanalyzed that now and spiders are very distantly related to the arthropod group. They're not as closely related. These of course are the centipedes and millipedes, crayfish and crabs, and then the insect. The mites are a part of the chelicerates. This class has a bunch of different organisms: scorpions true spiders, spider scorpions, daddy long legs, and also, the tic. There are over 100,000 of them and these mites are extremely diverse. They're defined by the fact they have six pairs of appendages — so they have twelve appendages. Four of those are legs, of course. In the blue section is the pedipalp and inside the mouth parts is called the chelicerates, of which they're named. They have simple eyes, not compound, which means they don't see very well. They might see some light but they certainly don't see images. They mostly feed on liquid external digestions, so they're digesting their food inside of their body and then sucking the nutrients in. If we look at the chelicerate, they are quite admirable. They are tipped with fangs so they're really good for horror movies, but what they're doing they're putting their fangs in and spit into the organism they've grabbed, liquify it, and then suck up the juices. That's how they get their food. Surrounding those chelicerae are pedipalps, which look like stunted legs. The male adapters actually have legs like this because they ejaculate into the pedipalp so they've modified that into their mating organ. It actually springs out so sometimes you can see the mated spider because you can see the pedipalp hanging out of her still. For most organisms, these pedipalps have hairs that are used to smell, to sense and walk around the environment.

I’m going to show a video of a deer tick in this process feeding. It tells us a little of how varroa mites might do it. You can see the two pedipalps getting pushed aside there and that chelicerae moving. It’s like a straw when it gets plugged; you have blow out. This tick does the same thing: it starts blowing out enzymes into the flesh of this mammal. Eventually it does that back and forth until it liquefies and the blood becomes easily accessible. It can then suck up that blood. This is a tic, which varroa mites are, and so mites do that same sort of behavior. They’re pedipalps so they are finding the right place and they’re using their chelicerae to cut through that exoskeleton of the bees in order to suck up the liquid. If we look at a close-up of a varroa mite, we can see little pedipalps here and chelicerae surrounding the mouth parts it’s going to use to suck up the juices of the bee.

There are over 50,000 species of mites and ticks. There are at least a million and an amazing diversity of these mites. There are millions and millions per acre of soil mites. There’s some evidence that suggests that tracheal mites started out as soil mites that transferred over onto bees and started living in the tracchia. When people are worried about having the new mite that’s killing all the bees, we’ll get samples and undoubtedly, those are soil mites that bees accidentally brought to the colony.

The hard tick, which the varroa mites are part of, is how they look for their host: they have their legs waiting in the air, waiting for the host to come by and they feed on the host. They have manath floor, which is the mouth part that gets drilled down between the chelicerae. When it’s fully engorged, the female will drop to ground and lay her eggs. In the case of deer ticks, the eggs hatch and they become larva. They’ll get on mammals, birds, or people and fall off again, become nymphs, get onto another host, fall off, get onto deer or other hosts, engorge, and lay eggs. When you see this pattern and see this same mite go to different hosts, that means that this is a great vector of disease because if any of those animals had a disease, then it could suck that disease into the blood and pass it on to the next feeding. When you see this sort of behavior — feeding on more than one host — you expect it be associated with by a vector of some disease in those populations, which is certainly true for varroa. Mites feed on multiple generations of bees and as a result, it’s a great vector of bee diseases, specifically viruses. Of course, in this case, it’s lyme disease. Beekeepers really need to be careful. It was a bad year for deer ticks.

In terms of bees, there are 86 different mites that live with bees. Very few of them are parasites. Most of those are scavenger mites. They live in the bottom board and are eating pollen. Sometimes, you’ll get heavy levels of these feeding mites in your pollen frames. If you’ve ever stored frames of pollen and it gets powdery and misty, it’s probably because you have a heavy pollen mite infestation in there. That also affects native bees, carpenter bees, and mason bees. You can see the pollen shredded up into dust. You have pollen mites and then mites that eat the pollen mites (predatory mites) and then you have hitchhiker mites that basically are using the bees as a bus to move from flower to flower to eat the pollen. We also have parasitic mites that feed directly on the bees and bee brood. One of those of course is the tracchia mite, which came about in the early 80s. This little mite changed our industry. They live in the tracheal trunk of the honey bee and make it difficult to breathe. But, something happened because

varroa mite came and the control products for varroa mite also helped control the tracchia mite or that the genetic changes needed by bees in order to keep mites low happened really quickly; a simple genetic switch. We very rarely find tracchia mites at all anymore and they're not an issue. We don't find them at high enough levels; we don't even monitor them anymore.

Varroa destructor

If we look at varroa mites right now, we'll see that it has a worldwide distribution. This map was made in 2010 and since then, Madagascar and most of Africa, have varroa mite but they don't seem to cause as much trouble there as other places. Hawaii has varroa mite now but there are only a small places that don't like the island of Newfoundland, islands in the Caribbean, but mostly, mites are everywhere we keep bees except for Australia.

Varroa destructor did not come from apis mellifera, the European honeybee. It originated with apis cerana, the Asian honeybee. In apis cerana, varroa caused very little damage. In fact, not all varroa mite in apis cerana are the same. There are these different haplotypes, or varieties, of varroa mite. One of those is the Japanese haplotype, the mite that was introduced into Paraguay and was the first North and South American variant. That variant is not very lethal; it's gentle. Unfortunately, there's the Korean haplotype, which predominates the world today. It's more aggressive, reproductive, and successful type of varroa mite.

In 1904, varroa jacobsoni was first described as apis cerana by scientists. When a scientist names something they have a voucher specimen but it turns out there were several types of species of varroa and the varroa we had originally was not varroa jacobsoni, which is where the new name varroa destructor because scientists found out it was a different variation. Both varroa jacobsoni and cerana live well on apis cerana. Apis cerana is the Asian honeybee; a much smaller bee with smaller colonies. In terms of honey production, it produces maybe 2 kilograms of honey a year, so it's not very productive. But, the bees live with mites very handily. One of the reasons they do is that the capping, the length of development time for these bees, only means that mites that go into droned brood will be reproductive. Otherwise, the workers develop so fast that even though the varroa mite will invade that cell, it won't be able to have any children. Also, if that drone brood has more than one invading mite, somehow the bees sense that and they'll cap that drone brood very heavily with a heavy capping and that prevents that drone from emerging (you'll see these distinctive little holes). So basically, they're exhuming that drone and the mites in the cell so then both drone and the varroa die. This apis cerana has developed this behavior which allow it to control the mite in its natural habitat. We have to be very careful about bringing stuff in; that people have done more to spread this mite than anything else. We started here in Indonesia in the 1900s then the Russian soldiers brought apis melliferae (the European honey bee) into Asia and the mite jumped species. The Russian soldiers brought some of these colonies back into Germany and we can actually base the spread of mites distributing across Europe because they're right beside a military base outside of Berlin. The Russians brought them over to Germany and it spread throughout Europe. We've had

introductions into Brazil and in the 80s, we saw introductions into the US and Canada, and Hawaii in 2007. We can see that it's spread internationally because of human trade.

So what causes it? We can see the shipments of queens in packages. We also know that moving colonies is a great way of moving things around the world. We're reliant, in this country and many others, on a moveable pollination force. Half the colonies in this country are in almonds. That means if there's any new disease or any new parasite that gets into the country and into the almond orchards, they're across the country in that next year. We have to be on the lookout for new diseases very vigilantly to stop it before it becomes a national issue.

We talk about trucking our bees over long periods of time, but to give it some perspective, us moving bees from Florida to California is the same thing as moving bees from South Korea to Bangladesh twice a year. Or going from Brussels to Moscow twice a year. This is a huge amount of distance of travel with our bees several times in the year. In our survey, we consistently show that people who are migratory beekeepers lose fewer bees than non-migratory beekeepers. So certainly, we think some of the spread, especially in Europe, can be traced along the beekeeper migratory rates. In Europe, there's a big migration into the Black forest to collect honey dew. The migratory nature is what makes the industry necessary. But even without people, mites are able to spread pretty quickly. There was a great study done in Germany where they had an infested colony and they watched colonies placed in several time points and distances from that hive to see when they would become infested. So within one month, you saw colonies within 100 meters infested; within two and half months, five hundred meters away were infested; within three months, 6 or 7 kilometers away. This is a huge ability to move very quickly across the landscape because a lot of those mites that were moving are on drones, who are famously unfaithful — they'll go to any colony they want.

How Varroa Mites Feed

Let's go into the biology of the varroa mite and how it feeds. We have the mites who live on the adult bee, the phoretic stage. When the bees are feeding the larva, they come in to smell, and when the smell is right they'll jump off, bury underneath that larvae, and live in that brood food until it gets capped. The mite will then crawl up and start to lay eggs. Eventually, all her daughter mites that are fully matured when their outer cover is solid and brown — otherwise it gets dehydrated and the exoskeleton is there to keep it wet — will come out.

Here we have the life stage of a worker bee: 3 days as an egg, a larval uncapped stage — a growth stage when the nurse bees are feeding them like crazy and they'll get fat. It's the only stage in which the bee grows — it gets capped and becomes the pupil stage — it spins a cocoon of that larva, it goes to the bathroom for the first time. It then undergoes this amazing transformation because all of its organs dissolve and becomes a bag of fat, which gets organized into legs, arms, hearts, organs, etc. It's a remarkable time for the pupil bee. The adult emerges somewhere around day 21. The female mites on the nurse bee enters the cell a day or two before capping. After capping, about 3 days later, it lays its first egg. It then lays an egg every 30 hours thereafter. However,

only those female mites that get sparitized will survive hatching out with the emerged worker bee. The first egg she lays is unfertilized, exactly like honey bees, where fertilized eggs become female and unfertilized eggs become male. So that male comes out and becomes a white color who will always remain that color. They will die after the adult bee emerges. The female eggs gets laid and they'll mature. They'll then mate with their brother as a result of that. Some of the mites don't get mated, so they're virgins. That means they could invade another cell, lay male eggs, and mate with their son. Generally, those are not successful. About 10% of mites haven't been mated and will lay duds; they'll just lay males. Are there ways that we can induce this sterility so the mites aren't mating? We have phoretic mites that feed on larval bees and if the larva smells right they'll stay in that cell, but if it doesn't, they'll leave with the worker and go on to another properly aged cell. Right after it gets capped, that fifth in-star larva releases a special scent and that scent allows the varroa's ovaries to activate. It starts to produce its first age. If it doesn't get that shot of smell in the first 24 hours, the ovaries don't develop and she'll never start laying eggs. That activation is a really important step.

You can see the male is much smaller than the female. He actually will court her and spends a lot of time figuring out where their sex organs meet. They have the transfer of sperm, she stores it, just like a queen bee does, to fertilize the eggs for the rest of his for life. The fact that it's the length of time under the cappings is what limits the number of mature mites that can develop. The longer you're under the cappings, the more your daughters are going to be fully sparitized. This explains why varroas prefer drone bees. If you can get under a drone cell that's capped for 13-16 days, you can have, on average, 2.2-2.6 kids that are fully sparitized and able to survive with the drone that emerges. The workers are only capped between 12-12.5 days, which means they have half as many kids that can successfully emerge. That's why drones are this great breeding ground and that's why varroa mite really do prefer drones. Varroa mites do invade queen cells, which aren't capped long enough to have any viable offspring.

Tools to Control

Let's talk about what types of tools we have to control the mites. The mites reproduce in the brood cells and cause damage at this level, critical for the winter, and they reproduce at a rate that doubles the colony every month. We've seen the national averages and that over the last five years we've done this, the average mite load in this country come August is in excess of what we think is causing damage to colonies. The average load in September or October is well above what we think doesn't cause damage.

Let's talk about the Bee Informed Management Survey results. You'll see on the graph whiskers that aren't what you usually see as error bars on the data. These are 95% confidence intervals. If I were to sample this population again and again, that average would fall between the two lines on the chart 95% of the time. If the two lines overlap, that means that the populations are not significantly different. They're exactly the same statistically. If they don't overlap, we consider them significantly different. The people

who reported using a known varroa mite control product lost a lot fewer colonies than those who didn't. What's worrying though is that 60-70% report not using a known varroa mite control product in the last 12 months. It's useful to look at the history of treatment in Germany because they've been dealing with this problem for longer than we have. In 1982 when they got the varroa mites, they used a product based on fluvalinate as a treatment once a year. It stopped working by 1987 and they started using 1-2 winter treatments of coumaphos, a different, active ingredient using it twice a year, five years after the introduction. In 1995, after that didn't work anymore, they had problems in the summer so they started using formic acid as a mid-season treatment and then using coumaphos in the winter. Today, they have to be much more aggressive with mite levels and treat more often. They use drum brood removal in the spring, two formic acids in the summer, and oxalic acid treatment in the winter. In Germany, their treatment regimes have changed dramatically.

We'll break this down and talk about each product. On the chart we have all the known varroa mite control products: the purple is for people who use this product, the yellow is someone who used the product but not the product in question. Amatraz lost fewer colonies than those who used another product and certainly more than others.

I'll go through each of these products and summarize them in terms of how to think about them. If we think about amatraz, cuomofos, and fluvalinate, these are our synthetic products; our hard chemicals we used first. They're fairly safe in terms of humans because they're lipifilic — they like fat, which means they don't migrate into honey, they migrate into wax. But, they build up there. Then you have softer, bio-based products, like apiguard, apilifevar. These are essential oil based so they're thimal and fumigants. We also have the organic acids: the formic acid and acelic acid. This is correlative data, which is not the same as causation. In 2013 and 2014, people who used cuomafos or fluvalinate lost the same number of colonies as those who reported not using anything at all because these two products have lost their efficacy. However, the next year 2014-2015, we did see the people who used the products lost fewer colonies than those who used nothing at all but it wasn't different than any of the products. Our real concern is that it builds up in the wax. The bee bread that we test, not the wax, have these known miticides in them. So we're feeding a miticides to young bees and that has negative effects on them.

Amatraz users lose fewer colonies than those who use nothing at all or use another known varroa mite control product. It's one of the newer products on the market that has lower colony losses. Commercial beekeepers used none at all and lost about 34%, used it once and lost 30%, used it twice and lost 24%, used it three times and lost 20%, used it four times and lost 10%. To use this product four times a year is a disaster waiting to happen. If you're going to use it more than once, you'll want to combine it with an organic acid or an essential oil based product. Even if you kill 99% of the mites, that 1% of mites who are resistant to amatraz can be knocked out by that other product. Just using one product consistently like this is in the long term is a short sided option.

We also do mite resistance asay so if you're part of a tech transfer team, you'll send in your kits and we'll check the mite levels and their susceptibility to certain products. We

also do this randomly across the country. Basically, we put a handful of bees in your yard with your mites as long as they have 5 or more mites per hundred. We'll put them in a jar with a little bit of the product, we'll wait 6 hours, we count how many mites died, then we kill all the mites to figure out what percentage of mites died in the presence of the product. If a mite population is considered susceptible, it means that 85% of the mites die within 6 hours. We haven't detected any populations that are truly susceptible to any of our main products, including flumethrin, which isn't registered in the product (it's used in Europe but it's our out-control). We have some level of resistance. They're not killing more than 85% of the mites. In fact true resistance is also rare. It occurs if more than 85% of the mites are surviving, we'd consider that resistant population. We have one or two colonies evidence of cuomafos or flavalinate. We have yet to find that in amatraz. However, there are a lot of beekeepers complaining about the product. They're putting the strips in, coming back, and finding high levels of mites because there's been a switch to a homemade formula versus these strips. The strips are much more expensive but it's a great way to insure that you're not getting out of the control and speed up resistance development. Use low doses of the strips. The product that stands out the most, with over 50% of detections, are the varroa sides. We're killing an arthropod already on an arthropod. There's no question that the presence of cuomafos, fluvalinate, and amatraz negatively affect the queens and workers. It's a lot like chemotherapy — you don't do it because it's good for you, but because it's better than the alternative. Fortunately, we do have softer, organic products like oxalic and formic acids. I would make sure that 40 days before I had the super, I would put in the finewall treatment if I had detectable levels (maybe apiguard or apiliphar). If you're honey flow ends early, I would do another check and if I have detectable levels of three or more mites by August, I would put on a formic acid pad. I would do three treatments: essential oil, formic acid, and an oxalic treatment. There have been some complaints about formic not working: if you're doing a mid-season formic acid when it's really hot out, what happens when you're putting those pads on that formic gets flashed out there, they start to work in the first 24 hours, but what happens is that it gets under the capping kills 80% of the varroa under capping. If you can damage them, that's great because you don't have to wait for them to come out, but it turns out that it doesn't kill them. But, it's damaging the hairs on it's pedipalps, which means that the mites aren't able to smell the right stage brood anymore. So often, your mite levels are three times higher than before you treated maybe because you have fewer percentage in the brood because you've damaged those adult varroa mites. Sometimes, that mite check after a formic treatment will give you a heart attack but will decrease 2 weeks after. My personal preference would be the thimble, formic, and drip oxalic when it's broodless.

We've talked about these organic acids and essential oils as if they're somehow better for the bees than the synthetics. They may be better but nothing is free. We wanted to treat colonies that didn't have any mites and then look at the expression of different stress bees to see if the treatments themselves had immeasurable effect on the stress of the bees. The answer yes. And that's true for cuomafos, amatraz, formic acid, and thimal. If we can slow the population growth down, it means we have more time for intervention. If we can get to three mites per hundred by October rather than September, it gives us more time because we want to keep our honey supers on, which means more honey, but we have to take them off to put the treatments on. There are

different methods to stretch that time period. One is this new product that prevents the invasion of mites into the colony from your neighbor. They'll be these varroa gates that the bees will have to crawl through. It'll be a treated strip.

Population Dynamics

Let's talk about population dynamics in terms of what happens with mites in your colonies over the year; at the population and colony level. We'll also look at the field data from five years of intensive survey through various efforts, including the national honey board and the bee informed partnership efforts.

We know this female mite, smells the right larva, and comes up when it's capped. What she does next is remarkable: she bites a hole in the developing larva, which is actually a feeding well. Immediately across from that well, she poops on the cell. That way, all of her offspring, who don't have the mouthparts as hard as they need to to cut through that larval bee, they crawl up and smell for that fecal matter and know that the food is exactly opposite. It's a remarkable communication structure. All those varroa are feeding over that same feeding well. Of course, that feeding well is also an opening so you can get bacterial infections there but it's also a way the bees can introduce viruses into the colonies. One of the best signs when you look at a cell that a worker bee has just emerged from, you'll see an immature mite because it's still white, but you'll often see a lot of fecal matter. In fact, if you have a dead-out in the spring or middle of the fall, those are probably your strongest colonies. Those are usually all varroa mite. The best way to know is if you look on the bottom and there are white dots, which are called fecal pellets. That's a good way of knowing you have high mite levels; you have a lot of varroa poop, you have a lot of varroa mites.

But of course in that feeding, we transmit a lot of bee viruses. One of the things my lab has been doing is the National Honey Bee Disease Survey. Every state that wants to participate gets funding to take 24 samples semi-randomly selected and they send us live bees in bee boxes. We do alcohol washes for varroa mites and nosema. We'll find the viruses and pesticides. Then we'll look at the association of the viruses and varroa mites. As your mite population goes up, the prevalence of the number of bees in that colony that have the viruses goes up. It's a close relationship. The more mites, the more viruses you'll have. That's not always true for all the viruses. For example, the Lake Sinai virus because it isn't transmitted by varroa mites. However, for DWV (Deformed Wing Virus), it's a very strong association. Right now, we have a hard time finding bee colonies in the country that don't have detectable levels of varroa mite. So 90% of colonies in the country have some level of DWV. But, it's not a very virulent virus; it doesn't kill colonies until it has very high titer counts, which is different than the acute paralysis viruses, like APC (Acute Paralysis Virus). We don't often find them but when we do we find them quickly. Not all viruses are the same. These viruses are driving a lot of these problems.

This next slide is the data from all the colonies we've sampled. Blue is for stationary and red is for migratory beekeepers with the National Honey Bee Disease Survey over the last five years. There's a huge variation and pattern in them; if you look at the green

section, that five mites per hundred, a critical number. We now think that when you hit over three mites per hundred, your bee population is in trouble. However, when you get over five mites per hundred, your bees are not only in trouble but even if you intervene, you should expect to see losses. Those thresholds are very different than the thresholds I would've suggested four years ago and much different than the thresholds twenty years ago. So we think that the change in thresholds are ten to twenty mites ten years ago. What's changed are the viruses. The viruses have been in the bees before varroa. Some would say that the viruses have a benefit for the bees. Japanese researches have shown that the bees that are more likely to be the guard bees are more likely to get viruses, so in fact viruses might benefit the colonies to protect it as long as it doesn't get too widely dispersed. As long as you have a situation where viruses aren't getting spread from sister to sister but only from mother to daughter, because that's the only way it'll get spread without varroa mite, you expect those viruses to be benign. If it killed the host, it would kill itself. However, when you start to see transmission horizontally (sister to sister), which varroa mites allow them to do then you expect to be much more virulent much quicker. For example, if I make a lot of copies of myself somehow then I'm more likely to get sucked up by a varroa mite so when my colony dies I move over with the bee and invade my neighbor. There's a lot of pressure for those viruses to mutate and become virulent. They're the ones who are going to survive in a world through horizontal transmission. It's not that the mites have changed but the viruses that the mites are transmitting have changed this story to low levels mites that are causing measurable damage to the cause.

If you mathematically model a population of mites, you can expect colonies to die every 1 in 3 years depending on where you are. If you're in the tropics and you have brood production all year round, you expect your colonies to die from the mites within one year. If you're in central Europe, you expect them to die every two years and in the temperate regions, every 3 to four years. That's why Northern states in Europe don't have high mortality rates than Southern ones, like Denmark has low winter mortality rates because the varroa aren't growing as aggressively. The population is doubling every month.

It's important to understand how we measure mites. We don't do a sugar roll but we collect the bees in the same way and do an alcohol wash in the lab, a much more efficient way of doing it if you're doing big samples.

The most important message from this video is that the need for a half cup measuring cup because that totals to about 320 bees. When you're taking samples to send in kits, you need the 300 bee count otherwise the sample is not as accurate. It's really important to recognize, no matter your method, it's number of mites per adult bee. We have to look at the population dynamics of adult bees and the brood in the colony relative to the mites. If we have an adult bee population of 30,000 or 10,000 that have the exact same number of mites in them, the mites per bees is going to be very different because you have more bees diluting. The bees that are most critical for winter survivorship start getting produced in August. This is when eggs get laid, larvae start to mature, and take you through the winter. If you'd like to have the healthiest bees possible, you need to try and make sure that the production window is as disease-free

as possible. If that bee is supposed to live 6 months over the winter and you lose 10% of your life, that explains why we lose so many bees in March.

In this graph, we see that 80% of mites are in the brood so if we take that percentage, it's about 4 mites per hundred. That's pretty close to the national average. Looking at the peak population, we have 30,000 bees, 400 mites, which totals 1.3 mites per hundred. If you're doing a sugar roll in your yard, for every 2 colonies, 1 will have a mite level of 1 per average, which is very low. You don't want to enter a new season with more than 1. Every other colony should not have detectable levels of mites. If you have higher than that, you're starting at a big disadvantage and will become very hard to recover. In October, we see that the population has decreased drastically, about 12,000 bees, with the brood decreasing rapidly. That's 33 mites per hundred times 80% that's 6.6 mites per hundred, which is in keeping but you'll have very little brood. This is a critical time for those colonies to survive. Untreated colonies will die between 1-3 years. Often, if [beekeepers] send us their debri from their winter, we'll find it's loaded with mites.

So, we have a level of 5 mites per 100, which has come from several different studies. A German study has shown that after 5 mites per 100 that even if you treat it, you'll have serious losses. Another Argentinian study came up with the same threshold. Our tech transfer team also said that when beekeepers suffer 5 mites or more, they'll start seeing heavy losses very quickly. For many independent sources globally, that 5 seems to be the stabilizing point where you start seeing critical problems. Anything that's less than 3 is grey and less than 5 is pink. Coming in August, you'll see that more than half of the colonies for the rest of the year until December, have levels that are higher than we think that even treatments will prevent losses from occurring in those situations. There's little question that nationally, on average, according to this national honey bee disease survey, we have mite populations that are exceeding what we think causes damage.

One of the methods we've been using is the centennial apiary program or "self-monitoring." You can do this as a group and send in samples from your apiary or you can do it individually. The yellow is personal results from a beekeeper in Maryland, the orange is everyone else's average mite levels who was part of the program that year, and the grey is the aphis national average. Yellow has sent his samples in for June and he has only a quarter of the mite levels of everyone else. In July, he's still low and by August he's still at half of where everyone else is. But in October, his levels go through the roof. What's happening is that mite problems are not your own but of your neighbor's. There are a lot of beekeepers that out of goodwill have decided not to treat their colonies. We think that this transmission of mites across the landscape goes into different colonies. Out in the environment, we have mite mines acting as untreated colonies; they blow up and spread their goodwill to all the colonies within 3 kilometers. These explosions are happening all the time. Some of this data is from the aphis survey but a lot of it is from our tech transfer team, which does several different things: we do our big national survey and we put people in with commercial beekeepers to help them monitor their colonies over the course of the year. They're actively taking samples. If you're a part of one of these teams, you get these reports every year that talks about

the number of samples taken within your operation. It also gives your average mite levels this year and last year. It also anonymously compares you and other people in the program within your region. Typically, when people join our tech transfer team, their mite levels exceed thresholds very frequently. But, if you tease this apart, you'll see the averages are a bit betraying. You'll see not just the average but the range of mites (each dot represents a colony). However, there are outliers that are waiting to explode and transfer to your neighbor. If you were going through 100 colonies, there is a reasonable error rate of 1-2% with a couple of these landmines blowing up in July and August, and their spreading to the neighbors, by September or October, you'll have 20 or 30 of these mines going off and spreading. It's important that all beekeepers have an active and vigilant monitoring program.

Pesticides

In the national honey bee disease survey, we regularly collect, test, and analyze bee bred for pesticides. How do you summarize all the products in there? So we calculated something called a hazard potent, which takes into account the toxicity of the product and how much of the product we found. We can then graph the average hazard quotient in colonies that had no detectable mites, less than 3, 5, 10, between 10-25, and 25. We often hear from beekeepers that something is happening in the environment, like the mites are eating that pesticide, having super-kids, and the population is growing like crazy. If you have a pesticide that locks out 30% of your worker force then you don't have more mites in the colony, you have the same number of mites and fewer bees. If you have certain colonies within the landscape that have had a high exposure to pesticide, it means you have another potential explosion.

Here's a project we just did this year that dealt with fungicides on blueberries in Maine and the results were not at all what we expected. We have the control, which is they went to get the blueberries from Florida at an organic farm and then the experimental was that they also went to a blueberry farm, but where the berries were sprayed with pesticides. It had slightly more mites but not that much more than the experimental. So, when we look at blueberries, it was the control not the experimental that grew much faster. Another possibility is when we have these outyards where we're dropping colonies into the landscape and pulling colonies out to spread into the system, we're creating mini-landmines that blow up in the landscape and spread into the neighboring colonies. It's both your neighbors and internals practices that explain these big spikes that we see in certain colonies that spread the mites.

We asked commercial beekeepers how they monitor, keeping in mind most of them lost 33%: 17 of them visually inspected mites, some did a drop where they stuck a sticky board in for treatment. It was only the people who started doing sugar rolls and alcohol washes that had noticeably lower loss rates the following winter. There's no real difference between the two methods in terms of accuracy but the people doing alcohol are spending more money doing and are listening more carefully. Something about doing mass sampling, sending them out, and getting back in a report makes that data more actionable than only doing sugar rolls.

How/when to do alcohol washes: I would monitor my population of mites two weeks after every pollination event. I would do it pre-supering to make sure that I don't have detectable mite levels. If I have more than one mite for every other colony, than I have a problem. It will manifest itself in August. Mid-season, if you can, I would check for levels that are well in excess of we think would cause damage. Two weeks after your fall treatment, you want to come in and make sure your mite treatment works.

The time of year you need to sample is the time of year when you don't have time to do it, so I suggest joining one of these groups. Within two weeks, you get results. If I could suggest anything, I would talk to other members to the tech transfer team, especially if you have over 200 colonies.

Drone brood pull is a great way to know if you have varroa mite, but you can't tell the exact infestation rate and whether it's important or not.