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SPEAKERS

Amy, Serra Sowers, Stump The Chump, Guest, Jamie

Jamie 00:10
Welcome to Two Bees in a Podcast brought to you by the Honey Bee Research Extension Laboratory at the University of Florida's Institute of Food and Agricultural Sciences. It is our goal to advance the understanding of honey bees and beekeeping, grow the beekeeping community and improve the health of honey bees everywhere. In this podcast, you'll hear research updates, beekeeping management practices discussed and advice on beekeeping from our resident experts, beekeepers, scientists and other program guests. Join us for today's program. And thank you for listening to Two Bees in a Podcast. Hello, everyone, welcome to another segment of Two Bees in a Podcast. Today, we are talking about a very specific issue related to honey bee viruses and honey bee ability to handle those viruses. In fact, we are talking about a manuscript that we'll make sure and link in the show notes. The manuscript is entitled, "Context-Dependent Viral Transgenerational Immune Priming in Honey Bees." That is a mouthful. But fortunately, we have one of the authors of that manuscript here today with us. He's actually showing up on our podcast a second time. I just want to welcome to our podcast, again, Dr. Mike Simone Finstrom. Mike, thank you so much for joining us on Two Bees in a Podcast.

Guest 01:30
Thanks for having me. It's a pleasure.

Jamie 01:31
Everyone, Mike is a Research Molecular Biologist at the USDA ARS Honey Bee Breeding and Physiology Laboratory in Baton Rouge, Louisiana. Mike, you and your colleagues published a very interesting paper that we really want to spend some time talking to you about. But before we get into that manuscript and talk about viruses and what transgenerational immune priming is in honey bees, could you remind the audience a little bit about yourself, how you got where you are?

Guest 01:58
Yeah, so, I'm a research scientist at Baton Rouge. I've been here since fall of 2015. I started off with Marla Spivak in Minnesota, studying propolis and honey bee behavior and doing everything from understanding how physiology impacts behavior, and then I went to work with Dave Tarpy at NC State

before coming here. So, I've really gone to research everything from really hardcore bee behavior to be genetics and queen quality. And since being here, I've really delved a lot of work into honey bee viruses. But all of my work really does encompass everything from colony health dynamics to individual bee behavior, and how all of those things really interplay. So I really like to cover everything from the bee physiology and bee health to really how that affects colony health, and really, then, the commercial beekeeping industry as a whole.

Amy 03:08
Well, I'm excited for the in-person conferences to start coming back so I can sit down with you and pick your brain a little bit about everything. So we invited you today to talk about your paper that recently came out. And it discusses something called transgenerational immune priming. I have no idea what that means. So would you be able to explain what that means for myself and for our listeners?

Guest 03:34
Yeah, so transgenerational immune priming is, in a way, it sounds like this really complicated process, and it can be, but it's really cool process that happens in humans, and it's amazing that it happens in insects as well. We're really understanding more and more about how common it can be in insects, but it's one of these processes, in terms of humans, how mothers can transfer some immune benefits as they're sort of breastfeeding the young children and transferring some immune properties through that process. So basically, it's this idea that one generation, so a parent, can transfer some immunity to their offspring. So that's transgenerational, so cross-generation immune priming. So that's one generation priming or alerting the immune system of the next generation. And so really, that's this process of the parental generation alerting the immune system and, basically, prepping the immune system of their offspring to prepare for any diseases that they might be exposed to in the future.

Jamie 05:15
So Mike, in my experience behind every good research project, there's a really good story. So I know our listeners are out there thinking, okay, I understand transgenerational immune priming. And so parental exposure to some sort of pathogen helps them pass some sort of immunity on to their offspring, potentially. But I'm curious how you and your colleagues came up with the idea for this project. I mean, were you sitting around drinking coffee one day and said, hey, let's look at transgenerational immune priming in honey bees. What prompted this study that we're talking about today?

Guest 05:49
Yeah, that's a great question. So this was really the work of Sarah Lang who couldn't be here today. So this is Sarah Lang's master's thesis project. We kind of have been talking about this idea for a long time, and this is all part of this sort of larger work that we were doing as part of this NIFA-funded grant where we were injecting tons of pupa with viruses to look at how different feedstocks, so Russian honey bees or pollen, honey bees, Italian, Carniolan, we were looking at how different stocks of bees responded to viral infection. So one of our questions that she was sort of coming up with was, well, if these queens are exposed to virus, does that then affect their offspring differently? And she came up into these questions as questions of transgenerational immune priming. There had been very few studies on transgenerational immune priming with respect to viruses in general, really. There had only

been a couple of studies on it in honey bees at that point at all. So we thought this is a really straightforward thing that we could do that would really be interesting and worthwhile, and could be a really cool and valuable question to answer. So, we're really excited by that question.

Amy 07:50
Yeah, so you said that there wasn't much about viruses. But in your paper, you also discussed the immune priming that had been shown for American foulbrood. And so can you elaborate on this a little bit?

Guest 08:04
Yeah. So the best evidence really, certainly in honey bees, and really excellent evidence for immune priming in insects, in terms of transgenerational immune priming, has been shown for American foulbrood, and this is done by this European group. They basically injected honey bee queens with heat-killed spores of American foulbrood. What they found was the queens that were exposed to these heat-killed spores of American foulbrood, their offspring showed really good resistance to future exposure to challenges with the bacteria. So you basically, in a way, are vaccinating these queens by injecting them with American foulbrood, and then you put them in a colony, have them lay eggs, and then they in vitro reared. So they raised larva in the lab and then give them live spores of American foulbrood. They found really strong reductions in whether or not those larva died from American foulbrood disease. So it's really cool. And then they actually found sort of how that happened, the mechanism of this. So they continued to do that work with American foulbrood, so continuing that worked on, and I believe they have some patterns and process so they're really continuing that work, which is really exciting. So I think there's some really cool work and efforts there. Now, it's interesting. There's been some other work with other diseases like European foulbrood that hasn't worked so well. So it's kind of interesting why it works so well with American foulbrood in that context, and not the EFB. I think even our study has shown some of this and that's why we have this long complicated title, right? But I think that there are some really interesting complications there that require some follow-up.

Amy 10:25
That is so cool.

Guest 10:26
Yeah, the American foulbrood story. And they've had several publications now. It's a really cool story.

Amy 10:34
So, I have a really dumb question. How do you vaccinate a queen?

Guest 10:39
Yeah, so what they did, at least in those initial studies, was they basically did inject the queens with heat-killed spores of American foulbrood. So, kind of similar to what we would do with like a flu vaccine. Essentially, you're being injected with some bit of a virus or a little bit of a pathogen itself that's not going to actually lead to an infection. So it's a similar sort of process in that way.

Jamie 11:21

So I was sitting there listening to you talk about American foulbrood, and I'm like, gosh, there are a lot of beekeepers listening to this podcast right now, they're going well, man, all we have to do is maybe get scientists to inject honey bee queens with dead versions of every disease that we can think of, and maybe the offspring will be resistant. And then you mentioned right after that, it's not working quite as well for European foulbrood. Now, I really kind of want to zero in on what you and your colleagues were looking at in the first place, which was specifically deformed wing virus. I think a lot of beekeepers know about deformed wing virus. It's transmitted by Varroa in a lot of things that it does, but can you tell us a little bit about this virus to provide background for your experiment and as well as what we know about how it is normally transmitted to bees?

Guest 12:08
Alright, we chose deformed wing virus because it is the most prevalent virus to which all honey bees are exposed. You're right that it is primarily transmitted through Varroa mites. And so when it's transmitted through Varroa, the Varroa are directly injecting it into bees, right? But queens aren't typically getting it from Varroa. If they are getting it from Varroa, the colonies are so sick that they're not going to survive, or the queens typically wouldn't survive. However, deformed wing virus, like many other viruses, are also spread orally. So bees feed it to each other. So a nurse bee can feed it to a queen and a nurse bee can feed it to other nurse bees, which can feed it to larva. And the queen can also give it to her eggs. So the queen can pass it on transovarially through her eggs. And then it can also be spread sexually, so drones can pass it on in their semen through the mating process. And that's one way that the queen then can also pass it through to her eggs. So it can spread through her colony through all of these different ways. It's infectious or causes symptoms at different degrees based on these sort of different infection routes. So if it's injected by Varroa, it's going to cause more symptoms at a lower dose. It takes a much higher dose to cause symptoms if it's orally transmitted.

Amy 12:22
So that's always been a question that we discussed within our lab and with the grad students, just discussing Varroa and how it's transmitted. And it just seems like, as you've said, it's like every single way possible it could be spread, it can be spread, and it will spread. So it just seems like a lot of different avenues of examining this. So can you tell us more about your methods for your project and how you conducted your experiment?

Guest 14:47
Yeah, so for this, it did involve several different steps. The first step was just trying to kind of get these sorts of dosing situations, right? But the main goal was to really replicate at least those two main ways that queens would be exposed to the virus sort of naturally. So we wanted to give the queen's virus orally, and then give the queen's virus sort of through the sexual infection route. So we exposed queens orally by mixing virus and sugar syrup, and then we hand fed them virus and then we mixed virus into semen, and then this virus was given to them in that semen during the artificial insemination process. And then all of these queens were artificially inseminated, with, basically, a big batch of a similar mix of semen. It was just some of that semen either had virus mixed in it or not. Again, because semen can have some virus mixed in it, we wanted to make sure that they were, essentially, exposed to the same thing. So we, basically collected semen from 100 drones, mix it all up, and then split it up and mixed an additional amount of virus in some of it and they gave that to some queen, so that we had

basically tweens that were basically orally spiked with virus to replicate those that were given that oral dose of virus, and then ones that were sexually exposed to a virus. And then, we put all of those queens, essentially, into colonies. And then, we followed them over time. Then, we collected those offspring, or we collected offspring from these colonies. We essentially exposed those offspring to virus later by injecting them with known amounts of virus. And then we looked at their offspring's response to viral infection. The paper that was published that we're talking about now is basically the offspring response to virus. Coming up, we're finally getting to submit the corresponding paper that's looking at the field colony response, so something else to look forward to that's gonna come up later.

Jamie 18:10
So Mike, you were talking in your discussion about methods, exactly about something that scientists would pick up on. Deformed wing virus is so widespread. Te offspring might have already been exposed to it, the queens that you use could have been exposed to it, you mentioned the oral exposure through sugar water, that's pretty good. But I liked this comment that you made about the fact that drone semen probably has DWV in it already. So even your negative controls that you weren't spiking with virus possibly could have had DWV in there. So how did you guys handle that? Did you look for DWP in your queens beforehand? Was there evidence of them already being exposed to it? I mean, I know that's why we have controls in the first place. But how did you guys handle that experimentally?

Guest 19:03
Yeah, so basically, what we do is we made everything relative to the controls. We did use colonies that had low mites to begin with. So, they have less background DWV. It's sort of the best-case scenario that we can do and we did screen colonies at the beginning to try to select for colonies that did have low background DWV infection. So we kind of tried to control it as best that we can, but there's only so much that you can do with background levels of DWV. And so you can make everything relative to that control group so that you're eliminating as much of that background noise as you can.

Amy 19:58
Yeah, we know with honey bee research there's a lot of background noise.

Guest 20:01
Exactly.

Amy 20:03
So can you tell us the results from your study? I'm eager to hear what you all found out from your research?

Guest 20:11
Yeah, so the main finding really was that we did find that, at least, in one of these groups. So we had multiple queen sources that we tested, which is important to say that I didn't include sort of in this method. So we had actually three different queen sources that we tested that weren't affected, or that were exposed to these viruses. And, again, this sort of highlights this context dependency of this viral transgenerational immune priming. So we did find this effect of transgenerational immune priming, particularly in one of these queen sources. So when, particularly in one of these queen sources, we

found that offspring that were exposed in one of these conditions that they did seem to resist infection more effectively and showed reduced clinical symptoms. And it was pretty cool to see that. It's interesting, again, because there's so many different things happening. There are so many layers here that are happening because there's this genetic component between the different queen sources but then there's also the sort of oral versus sexual transmission route of infection that's happening. It seems like one was more effective than the other for this queen source. So there's just a lot of different things to follow-up here that provide interesting avenues to study.

Jamie 22:10
Which method of priming actually made the biggest difference? Was it the oral priming or the instrumental insemination priming?

Guest 22:18
Yeah, so it appeared, at least, in the one queen source, the sexual exposure did knock down the amount of virus or the offspring had reduced virus in the one queen source compared to the oral infection. So, again, I think it's important to know this is based on the conditions that we used in this experiment. So there are so many different things that need to be worked out, I think, for these sort of treatments, if they could be effective. I think this highlights, this is kind of the first step for this kind of work. I think, it's kind of true with the American foulbrood work, and they've been working on this for years, and they still are, that there's so much work to be done in terms of the dose and the delivery. In terms of the delivery, for the American foulbrood work, they started out with injection, but as kind of you mentioned, Jamie, does it make sense to just be injecting all these sort of dead or alive pathogens into our queens, right? Not really. So how is it going to be best to deliver vaccines to our queens? What's the way that makes the most sense if this is going to be a tenable solution. And in reality, we've learned so much about vaccines from a sort of a public viewpoint in the last two and a half years, and we know so much, again, just how much the dose matters, right? And we've really used, in terms of this experiment, one dose. In our timing of exposure and then our test, we did basically one time point, we saw these effects. So what would have happened if we had done multiple time points and multiple doses? Experiments with queens take so much effort and so much time. Queens are sort of a hard resource to work with, for some of these kinds of experiments that you can only do so much. But this result alone was super exciting. I think it really just shows how much more work we need to do in terms of all of these nuanced information to really figure out some of these seemingly minor information. But that's sort of the important information that we need to know to see how effective this really could be.

Jamie 25:23
So Mike, you guys made us, I'd call it a side comment in the manuscript, that was really interesting to us. As we were reading the paper, you guys stated that all pupa, the resulting offspring from the queens that were exposed, all the pupa were inoculated equally, but only 60% showed clinical signs of infection. So does that mean that in a normal situation, 40% can withstand or are resistant to the virus? And is this what others have shown similarly, in the literature?

Guest 25:57
Yeah. So the tolerance and resistance question is a good one. So I would say that range is probably within the scope of what's been shown in the literature, but honestly, it ranges really greatly across

colonies. So certainly, it's going to range, it can really range from 0 to 100% with clinical science, particularly, depending on how susceptible colonies are. Sometimes, you can see resistance, and sometimes you can see tolerance, I think, from some of the work that I've done. Now, more recently, with viral work, I think we're skewing a lot to seeing, more commonly, tolerance of virus infection. So what tolerance means is that bees are able to basically handle a high viral load, so they can live with a higher viral infection, whereas, resistance means that they are able to actually combat the infection and reduce that viral load. So I do think we tend to see a little bit more tolerance than resistance. That we can see. But I do think it's common to see, especially at the disinfection dose that we use, you can see the sort of 60% range showing clinical signs would be normal. That was a dose that we wanted, so that we would see a variation. Now, if you're gonna inject them with a high dose of virus, you're gonna see 100% clinical science, so it kind of does depend on the dose. If you're gonna pick a high dose to inject them with, you're gonna get clinical science or death in all the bees.

Amy 28:29
Even within the past couple of years and hearing about viruses and different clinical signs and symptoms in people and in honey bees, it's just really fascinating to hear some of the research that's been going on for a while, and I think a lot of us understand a little bit more about vaccines and viruses and ways that they can be transmitted. So I feel like this has just been a really great article and a really great conversation in general about that topic. So I have the last question, and it is, what does this all mean for beekeepers? So, as a beekeeper, what should we be considering? Or is there anything that we can do?

Guest 29:11
Yeah, I think in terms of the larger picture, and one of these things that I'm sure that I probably ended the last time I talked about this, or last time I talked with you, so forgive me if this is my soapbox, but I do think, to me, the most important thing that we can do is really help support bees in the ways that they sort of support themselves and support their natural defenses. And, to me, the most exciting thing about bees is the fact that they have all of these amazing traits and all of these amazing defenses to fight off all of their pathogen parasites, and we have so much more work to do to understand these things. I mean, who knew that they're able to pass on some of these immune defenses from queen through to the eggs, right? If this is just one of their tools in their toolkit or something they have in their arsenal, but that there is a sort of context dependency or maybe there's some genetic component to how they express this trait that we need to explore, or that we can support, either passively or actively, then we need to pursue that. But trying to figure out sort of these nuances, I think, is important. But really trying to figure out the small picture, but also think of the big picture and really just working to support our healthy bees, and seeing why these colonies are the healthy survivor ones versus the other ones, I think, is important. But, really, I think, really supporting those behaviors as physiological traits, I think, to me is really the key to sustainable and healthy beekeeping. It's not the only thing. But I think it's a long way to go so we can really help the bees help themselves.

Jamie 31:40
So Mike, that was great. I know our listeners are going to want to know a lot more about this research project. So I want to tell all of you out there that we're making sure to link this manuscript in our show

notes. Mike, thank you so much for joining us. I really appreciate the time you spent telling us about your research.

Guest 31:57
Thank you so much. It was a pleasure to be here.

Jamie 31:59
Everyone, that was Dr. Mike Simone Finstrom, who's a Research Molecular Biologist at the USDA ARS Honey Bee Breeding Laboratory and Physiology Laboratory in Baton Rouge, Louisiana. Thank you for listening to this segment of Two Bees in a Podcast.

Amy 32:29
Jamie, I feel like I'm still just trying to comprehend viruses being transmitted throughout the colony, like the different ways of exposures, the different routes of exposures.

Jamie 32:39
It's really kind of complicated and scary. When we talk about DWV, we think, well, Varroa is the primary mover of this virus. But he said himself that there's oral, there's sexually transmitted disease, right? Drones can pass this to queens, and so queens can pass it to their eggs. It's just crazy to think about how once a virus gets into a colony, it can show up everywhere. And the neat thing that he said, too, about that, is that the way that it's passed seems to matter. So, if the Varroa is doing it, it really makes DWV nasty. If some of these other routes are doing it, then maybe it's not quite as bad.

Amy 33:16
It's almost like the different routes of exposures have different dilutions, like the orally versus the instrumentally inseminated. I thought that was so cool that they're able to add viruses into it, but it still affect and have different results based on those different routes.

Jamie 33:32
Yeah, so this idea of transgenerational immune priming, basically, it means the immune system is being primed to address something. And in this case, it's not you being exposed directly to it, it's your queen mother being exposed to it that benefits you. And it was interesting, just like you said, that if she was exposed orally, like, she consumed the virus versus if she received it through artificial insemination, which is essentially trying to replicate how she would receive it during copulation, so if she received it orally or during copulation, it mattered downstream how resistant to or tolerant of the virus these offspring were. It's really fascinating.

Amy 34:18
I mean, is this commonly done? I mean, who else studies this? Like, is this common in insects? Or is this like just a honey bee thing?

Jamie 34:26
Yeah, one of the things that he mentioned earlier in the interview was really intriguing to me. People sort of look at this with humans, it's gaining traction, but with insects, they know so much less about

transgenerational immune priming. He gave us that great example of American foulbrood and how it was discovered in the first place. So, now, at this point, it's just like, well, what about to European foulbrood? No? Okay. Well, what about the forming virus? Yes. Well, what about Israeli acute paralysis virus or chronic bee paralysis virus or Nosema disease? So there's really a lot of ways that this can go. And hey, the way he concluded his comment for beekeepers is we need to figure out what bees do to fight these things naturally and see if we can enhance it. And his comment is like, hey, this is a way that bees potentially fight DWV. If we can understand the system better, maybe we can enhance it to the benefit of bee health.

Amy 35:19
Yeah. The other thing that's really interesting is that I've heard you give a talk about the different stressors of honey bees and how queens are really blamed for a lot of what goes on in the colony, right? You've got a bad layer, or your queen is not laying the way that you want her to be laying, or, or or. There are lots of things and, in this case, she really could be benefiting some of the other offspring, right? Potentially.

Jamie 35:44
It's really intriguing to me, this idea that a parent's exposure to a pathogen benefits the offspring through this method. So in this case, the queen can be benefiting her workers just because she's had previous exposure, prior exposure to this pathogen. You're right, queens are blamed for a lot of things. But here's a really neat example of how to flip that script where she's actually benefiting her offspring. The whole time I'm sitting here thinking, I was like, well, queen exposure can transgenerationally -- gah, that's such a hard word -- transgenerationally prime the immune system of our offspring, but what about the contributing father? What if drone exposure to these things also makes a difference? So this sounds like one of those areas of research that's just wide open.

Amy 36:34
Yep. I can't wait to see what he publishes down the road.

Stump The Chump 36:43
It's everybody's favorite game show, Stump The Chump.

Amy 36:52
Welcome back to the question and answer time. Jamie, one of our listeners has a question about mini mating nucs. They were saying mini mating nucs seem to be all the rage now. But some beekeepers are saying that it's not possible to get a quality queen from such a small nuc. They're specifically talking about that tiny three-frame, polystyrene mating nuc. So do you have any thoughts on this? Is there any research? I think it's a really common practice to have mini mating nucs.

Jamie 37:21
So this is an interesting question. And I don't know if I'm reading between the lines appropriately to answer it, but all mating nucs, to me, are very small. And I think they're talking about a specific brand of styrofoam that's maybe particularly small, but to me, they're all kind of super tiny anyway. I don't know that there's a big enough range between the smallest of mini mating nucs and the largest of mini mating

nucs for it to really make that big of a deal in the queen meeting world. Let me just elaborate just briefly on this for the benefit of the listener. We use the word nuc to describe a nucleus colony, but a nuc can accommodate full-size frames that would otherwise go on a full-size hive. So basically, a nuc is just a smaller version than a full-size hive, only in the sense that it accommodates fewer frames than a full-size hive does. Whereas, a mating nuc doesn't accommodate the same size frames as a full-size hive at all. You can't take frames from a full-size hive and put into a mating nuc because the mating nucs don't accommodate them. They're genuinely smaller boxes. The reason queen breeders use these is that it's easier for queen breeders to have hundreds or thousands of these things to put queen cells into than it is for them to have full-size nucs or full-size hives. It just takes less equipment, less space, it's easier to manage, etc. So these mating nucs, by default, are very small. There's not much standardization across queen breeders with regard to mating nucs because they tend to make their own. The ones that are available in the equipment catalogs aren't even standardized across companies or across styles. Whereas, I can buy a nuc from company A and company B and Company C and they're going to all accommodate the same size frames. You can't do that with mating nucs because there's just a general lack of standardization. So they're small by default. They're small by design. There is some scaling within the mating nuc world from smaller mini meeting nucs to larger mini mating nucs. But I think from a functionality standpoint, you're going to be almost equally successful producing queens in all of them. I know that there are some folks who say the queens are better if they're produced in full-size hives. And I could see a little bit of an argument for that. But if that were true, our entire industry would have to change because queen breeders almost always use these mini mating nucs. So I'd have to see some data on that to support the idea that queens are worse coming from the smaller end of those mini mating nucs versus the larger end, because from my perspective, they're all kind of small. Right? And so, it's hard for me to see that argument.

Amy 37:26
But you also kind of wonder with the queens whether or not they need a certain amount of space, right? I mean, not only like to just emerge from and be in, but then also coming back. I mean, are they using up that much space? And are they going to swarm as soon as they get back?

Jamie 40:31
Honestly, Amy, what I saw in the research is less about the size of the hive, but more about how long the queen is allowed to lay post-mating in that mating nuc. I'm scared to speculate because I don't remember the paper exactly, but there is a paper out there that showed the queens who are allowed to remain in mating nucs X amount of time are much better than those that are allowed to remain in mating nucs a shorter period of time, and I forget what it is. But something like if you put a queen cell into a mating nuc, generally the queen is going to emerge within a week. She goes out and mates the next two weeks, and then she lays eggs. Some folks will say you don't just need to grab a queen from a mating nuc the moment you see eggs in cells. You need to give her a few weeks after that because research has shown when she crosses that few weeks threshold, that she's going to be better as a queen than if you just yanked her right out of that colony the moment you saw an egg. I forget what it is. That's why I'm scared to put a number on it. But it seems like it was something like 35 days. After 35 days, you've maximized how good they can be, and then you'll take them out at that point versus something like two days after you see her. So again, I hesitate to say that it was 35 days, but there is definitely research to show that there seems to be a week that matters versus a size that matters.

Amy 42:00
Right. Okay. So for the second question, so the second question, I actually received this question pretty often. It has to do with population control for a backyard beekeeper. So this beekeeper wants to maintain three to five colonies. How do we keep this population somewhat constant? Right? So you're always kind of growing and losing and growing and shrinking. And that's just kind of how the beekeeping world works. I mean, could this person make splits? Make nucs? Give them/sell them away? Is that your recommendation? Is culling splits hive an acceptable best practice? Or is that something that's kind of frowned upon? How do we stick to the number that we want to stick to?

Jamie 42:43
Yeah, I mean, this is a super important question because honey bees are addictive. What you'll find, especially if you live in an area that's very productive, bees make bees. So what you're going to do is you're going to have three colonies, and they're going to grow super massive. If you're in a nectivorous area, you're going to produce a ton of honey, and you're gonna say, "Oh, gosh, my bees are so strong, the honey flows over, but my bees are so strong. What do I do? I don't want more hives, how do I stop where I am?" Well, remember, there are a couple of things that are worth knowing here. Number one, generally speaking, in order to make honey, you're going to have to practice swarm control. So you can just keep practicing swarm control and a colony population is naturally going to dwindle over spring, sorry, over summer and over fall. They're going to carry the right number of bees with them through winter. So essentially, you could just do nothing, and the population will naturally shrink on its own. That's harder to do if you live in a subdivision and you're looking out the back window, and you see these colonies that have massive beards on the colonies, and it's early summer and you're like, "Oh my gosh, I've got so many bees, I'm not going to make any more honey, what should I do with all these bees?" Well, you could still do nothing and the population is going to naturally dwindle on its own. But some folks have that kind of entrepreneurial spirit, and they'll go, "Hmm, I'm not going to make any more honey in summer. But I've got all these bees, let me make a nuc off of all three of those colonies." So three nucs and maybe sell them or give them away, that's another option. I actually faced the same issue. When I was keeping bees in my backyard up where I live now, coming out of spring, the colony populations were very strong. And so what I did is I would just shake queenless packages and donate those queenless packages here to the bee lab just to beef up some of our research colonies. I'm not suggesting that all of you guys out there listening should do that.

Amy 44:33
We're going to have so many donating.

Jamie 44:35
But, what I was saying is queenless packages could be sold to people to beef up their colony strength. So you've got a few options. You could do nothing and the bee populations are gonna go through the natural cycle and they'll dwindle when they need to dwindle. You can split and make nucs and sell those nucs, or you can split and increase your colonies. But a lot of people just want to make sure that they stay at that one colony level, in which case, if you split, you've got to sell. And really, those are kind of your options. It's hard because, as a beekeeper, if everything's going well, you've got that just natural desire to make more bees, but you just have to fight it off and just know that if you do nothing at

all, you manage diseases and food, of course, but if you do nothing otherwise, there'll be natural population control in the hive as the colony goes through summer and fall.

Amy 45:23
Alright, so the third question is, this person's wondering if they can feed their bees without a super, for example, in early spring, and then later, add the supers during the nectar flow. So they're wondering if the syrup that's stored from what they fed could be moved around the hive or eventually end up in their honey? Is it possible, I guess do bees go and take sugar water, put it in a comb and then eat it again and move it around?

Jamie 45:48
So there is ample anecdotal, at least, evidence that honey bees will move nectar and/or honey around the hive. So I suppose this questioner is worried that if they're feeding bees sugar syrup in a single, say, deep hive body with no other supers on, and then they stop, and when they add supers and bees start making honey and filling into those supers, is there any chance that that sugar water that they fed will end up in those supers. So, of course, there's a chance, right? I mean, there's a chance that it will be blended into the honey that's coming in by the bees themselves. But honestly, that's something that's really negligible and not something that I worry about really at all. The general recommendation is to not feed bees when bees are actively making marketable honey, when you have honey supers on that exist for the purpose of collecting and harvesting the honey. So what I would say to this is feeding outside of the honey flow and stopping before the honey flow gives you a pretty negligible residue, I'll call it a residue, a negligible amount of sugar water or corn syrup that would end up in your marketable honey. It will be in there, bees can move it around, but it's negligible and not something about what you would worry. I mean, commercial beekeepers do this all the time and don't worry. So the key is just don't feed during an active nectar flow. And honestly, if you're feeding in advance of the major nectar flow, the bees are turning a lot of that sugar water into energy to prepare for the coming nectar flow. They're converting it into new bees, they're building new comb. And I just really think the movement of nectar, the movement of sugar water around into your honey supers is going to be so negligible that you shouldn't really worry about it much at all. The key is just don't feed while you have supers on that bees are going to be putting honey into.

Amy 45:49
Is it true that they would prefer the natural pollen and nectar sources out there versus the supplemental?

Jamie 45:53
Yeah, I mean, you see this time and time and time again. If you're feeding bees, when the major nectar flow starts, you'll often see bee use of that sugar water that you're feeding them plummet. And by that, I mean, they take it out of the jars or the feeders but much slower than they would if there is no nectar coming in at all. They tend to shift. I mean, it's not a complete elimination. They will still take that sugar that's in their hive or in their feeder but it is a significant reduction. There seems to be some sort of biological trigger that's satisfied in bees when they go collect the nectar themselves rather than being fed directly with sugar syrup. So that definitely seems to be an impact.

Amy 48:36
Alright, so those are the questions. If you all have any more questions, please feel free to email us or send us a message on our social media pages. Thank you.

Serra Sowers 48:48
Thank you for listening to Two Bees in a Podcast. For more information and resources on today's episode, check out the Honey Bee Research Lab website at UFhoneybee.com. If you have questions you want answered on air, email them to us at honeybee@ifas.ufl.edu or message us on social media at UF honey bee lab on Instagram, Facebook and Twitter. This episode was hosted by Jamie Ellis and Amy Vu. This podcast is produced and edited by Amy Vu and Serra Sowers. Thanks for listening and see you next week.