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SPEAKERS

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

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 and welcome to another segment of Two Bees in a Podcast. We are talking again, today, about that thing, Amythat you and I find ourselves talking a lot about, which is, which is Varroa. Right? Varroa is just a really big deal in honey bees. It kills lots of honey bees all around the world. We've had many guests on this podcast talk about Varroa from so many different angles, from biology to control, to their spread, etc. But, today, we get to talk about a really unique topic related to Varroa. And that's potentially the control of Varroa using genetic technologies. And we are fortunate today to be joined by Nicky Faber, who's a PhD student currently in the laboratory of genetics and the department of plant sciences at Wageningen University and research in the Netherlands. But, while Nicky was a master's student at the University of Edinburgh, after graduating with a master's, she segued to doing some research on honey bees. And it's that research that we're going to talk with Nicky about today. So Nicky, thank you so much for joining us on this podcast.

Guest 01:52
Yeah, thank you for having me.

Jamie 01:54
So you, you and your colleagues published a study in 2021, specifically about controlling Varroa, and I read the paper, and it's really predicting how Varroa can be controlled using some genetic technologies that are available. And we're going to make sure and link to this paper in our show notes. But, before we get down into the weeds, could you tell us a little bit about yourself, your education, how you ended up doing honey bee research on this particular topic?

Guest 02:22
Yeah, sure. So yeah, I'm Nicky. I'm in my second year of my PhD, and I've been doing my bachelor's and my master's in Wageningen University, as well as my PhD right now. And I got interested in gene drives during my master's because there was a symposium about them. And I, I got interested because I saw the potential of, of gene drives being able to be used for conservation for the control of invasive species. And so, I decided to look for an internship in my master's on gene drives, which is how I ended up in Edinburgh at the Roslin Institute. And there, I did a modeling project with grey squirrels, which are invasive in the UK. And then after that project was done, my supervisor had actually gone to the US for a conference where he met up with another PI, who was doing bee research, and they were kind of talking about the gene drives and the grey squirrels. And they, they thought of, like, what if we could use gene drives for controlling Varroa mites, which is how the project was sort of initiated. And then, because modeling is pretty similar, it doesn't really matter which species you're doing it for, I could pretty easily transition from the grey squirrels to the Varroa mites. And I did a modeling project on that. And then afterwards, I went back to Wageningen, and I'm now doing my PhD also on gene drive from a different angle.

Amy 03:53
So, you've mentioned gene drive a couple of times, and, you know, I'm not a genetics person. I don't really know much about genetics, and I know that you've kind of explored, so you said that you explored gene drive technology to control Varroa. So, I guess, you know, just taking a step back, can you explain what a gene drive is? And what does that have to do with, I guess, just the environment and what does that all mean?

Guest 04:18
Yeah, no problem. So, a gene drive is basically a selfish gene. So, instead of the inheritance being a 50-50 chance, it actually skews its own inheritance ratio to the point where it's inherited in almost 100% if it's very efficient of the offspring. So, it's inherited more than you would expect randomly. And this phenomenon occurs naturally in a lot of species. Yeah, in a lot of species. But then, some scientists thought, "What if we could actually use this phenomenon for population control?" And in 2013, when CRISPR/Cas9, which is a genetic editing technology, was discovered, this sort of made the -- sort of paved the way for, for gene drives to be more efficient and to be used for population control. And so, how this works is basically, with the CRISPR/Cas, the CRISPR/Cas works as a sort of copy and paste mechanism. So, we have the gene drive on one chromosome, and then it copy paste itself into the second chromosome. So, this is how a synthetic gene drive skews its own inheritance ratio. So, that is the first thing that it does, it's inherited in more than 50% of the offspring. But then, the second thing that the synthetic gene drives do is they impose a fitness cost on the, on the individual. So, the most efficient type of gene drive actually disrupts female fertility. So, at first, the gene drive is a pretty low frequency. And then, usually,the gene drive individuals made with wild types. So, that gene drive is heterozygous, which means there's only one copy of the gene drive. But then, once the gene drive reaches higher frequency in the population, you have higher chance that you get homozygous for the gene drive, so they have two copies. And then both copies of the female fertility gene are disrupted, so females become sterile. And at that point, the population starts declining in size, until it even could cause a complete population crash. So, that's kind of how a gene drive could be used for population control.

Amy 06:45
That's really interesting. So, is that done with the -- that's done in the plant world too, right? Because I have, like, heard of different plants having, like, unfertile plants versus the fertile plants, or some of those invasive species. So, is that kind of the same technology?

Guest 07:04
Not really, no. Gene drives don't really work that well in plants because the copy pasting mechanism that I mentioned, plants don't really do that, that much. They, usually, when there's a DNA break, they just paste the two strands back together, instead of using the copy paste, like the, they don't use the second strand of DNA as sort of a template to repair the broken strand with. So, implanted gene drive wouldn't really work.

Jamie 07:31
So, Nicky, I was listening to you explain it, and I think you explained it, like, perfectly. I completely understood it while you were going through the process. Like I said, I had read your paper and read the same thing. And it's really fascinating to me that it's this selfish gene that can perpetuate itself in the population more than occurs just randomly. So, I love this idea that it can ultimately skew towards 100% inheritance. So, in other words, it just shows up at every individual. So, if I get this, understand this correctly, essentially, you develop this, you push it into, I'll say, a lab reared individual or an individual that you've got some sort of control over, and then you release that into the population. And if it works well, it ultimately shows up throughout the population. And in the particular example you gave, you were talking about disrupting female fertility. So, in that particular case, the population would crash because females would become sterile. So, I'm curious before we get specifically into your project with Varroa, are there some examples of other organisms for which this has worked well? Things that have been controlled, or at least things that seem to be controlled well, at least in a laboratory setting?

Guest 08:43
Yeah, for sure. So, first, as a note, the gene drive type that I'm talking about here is the most, the most common one or the most talked about type of gene drive, because it's supposed to be the most efficient. But, there's a lot of other types of gene drives as well that are really different in terms of their properties. So, yeah, just as a side note. But, then, gene drives have been tested in a lot of -- or in a lot of model species. And most notably in mosquitoes, which is where also the first, yeah, the first thoughts of application started because of malaria, of course. And in mosquitoes, gene drives are very efficient, they can go over 99% inheritance for the gene drive, so that's much higher than 50%. And then, they've also been tested in yeast, where they're pretty efficient in Drosophila melanogaster, where they're less efficient but still pretty okay. And then, there have also been test with mice where it doesn't really work that well yet.

Jamie 09:49
So Nicky, this is absolutely fascinating stuff. So, you're talking about it kind of from this general perspective. So, now I want to mine down straight to what you guys did specifically with Varroa. So, tell us how you got, you know, from this idea of gene drive into specifically looking at Varroa.

Guest 10:09
Right. So, Varroa has a pretty, sort of, unique life histories. The lifecycle is pretty specific, which is why it's important to do the modeling first before you actually start building a gene drive in a lab or something. So, we built a model in a language called R. So, we model individual Varroa mites, and we also track their genetics and everything. And then, we just have the model go around in generations of the Varroa mites. And yeah, like you said, we introduce the gene drive into the model. And then, we just see what happens, basically, see if the gene drive can spread through the population or not. And we look at the outcomes.

Amy 10:52
Okay, Nicky, so I'm trying to wrap my mind around the model, and what does it mean, how does a model incorporating gene drive for Varroa work? Like what does the process look like?

Guest 11:05
So, the way you can think of it is as just a giant Excel table in which every row represents one Varroa mite. And so, we have this big table for Varroa that we're tracking, and we're just letting it go through the steps in the model. And then, we're sort of tracking its, its genome with a gene drive in it or not, as well. So, the first step in a model is brood infestation, so it's a stochastic model. So, we have everything that happens has a certain chance of happening. And so, every Varroa has certain chance of infesting a brood cell or not. And so, then, we sort of track those that do infest, and then we track the reproduction, etc. So, this is kind of how, how the model keeps going around. We had this model that mimics the Varroa life history as closely as possible. And then, what we did, we started with ten Varroa mites in a single honey bee colony, and then we would introduce a certain number of gene drive individuals into the colony as well. And then, from there, we would let the model go through the generations and see if the gene drive would spread or not. And what we see, or what we saw pretty quickly was that -- it, the gene drive didn't really spread very well at all. And this is because of the unique Varroa life history traits. So, Varroa has a lot of -- or does a lot of inbreeding, because it goes, usually, a single female goes into a brood cell. And there, she produces one male offspring, and then several female offspring, and then there's mating in that cell as well. So, usually, it's just full sibling mating. And so, the problem for the gene drive is that it's not able to sort of fly under the radar for long enough for it to spread because of the inbreeding. Individuals are becoming homozygous for the gene drive too quickly. And thus, they're becoming infertile too quickly. So, the gene drive doesn't really spread very well, at all, was our first conclusion. So, then, we looked at what if we just introduce more gene drives right off the bat and see if that improves things, and it does, but then, it's just higher -- the gene drive is just at higher frequency because you're introducing more. So, it's still not really spreading in that sense. So, our third idea was what if the, the beekeeper does certain management techniques to, sort of, increase outbreeding in the, in the Varroa population? So, one thing the beekeeper could do is actually reduce the amount of brood that's in the, in the honey bee colony. So, the Varroa mites have fewer cells to reproduce in, so then the chance would become higher that two female Varone mites would go in the same brood cell, and then you have a chance of outbreeding. So, we looked at that and it showed that this type of brood breaks, the introduction of brood breaks, doesn't really, well, it does increase outbreeding, but not to a meaningful extent. So, it's still not good enough to really spread the gene drive through the Varroa population. And then, finally, what we looked at is if a beekeeper just continues with a parasite treatment to keep the honey bee colony alive, maybe then the gene drive

would have enough time to spread, because what we saw in our model is that usually after two or three years of not doing any control of Varroa mites, and then starting with 10 individuals at the start, so that's, that's a pretty small amount. Then, within two or three years, the Varroa levels are just too high for the bee colony to deal with it, basically. So, the gene drive only has two or three years to spread through the population. So, we thought maybe with a parasite treatment, this could be a longer period, and maybe then the gene drive would have enough time to spread. But, but this strategy still didn't really help. So, our overall conclusion is, basically, that the life history of Varroa mites effectively doesn't allow any gene drive spread or any any significant amount of gene drive spreads. So, it's pretty unlikely that a gene drive will be a technique that could be used to control Varroa mites, unfortunately.

Jamie 15:46
So Nickyy, this is kind of crazy stuff. It's interesting to me to see, you know, oftentimes, when we talk about Varroa biology, we emphasize the beekeepers, this inbreeding that occurs when a single foundress goes into a single cell, and you're saying that in that particular case, the gene drive spreads too quickly because the Varroa instantly become infertile, so they don't come out of the cell and have an opportunity to mate with other, you know, produce offspring that outcross. And then, you talked about condensing the brood area so that you would get multiple Varroa going into the brood cell and essentially, your take home conclusion is the entire biology, entire life history of Varroa works against gene drive. So, it really makes me wonder, kind of on a greater scale, how does the life history of Varroa affect so many other things that beekeepers are having to battle? But, I'm curious, did you guys model an effect of season, because there's often natural expansion of Varroa populations and natural contraction of Varroa populations seasonally? Yeah, so we'll start there. That's my first follow up question is did you guys model seasonal effects?

Guest 17:04
Oh, yes, so our Varroa model is based on a certain amount of brood being available. So, during the winter, this value is pretty much just zero. And then, in the spring, this kind of starts to pick up, and then it's highest in the summer and it goes down again in the fall. So, this is kind of the amount of brood that we're giving for the Varroa to reproduce in. And we did see that at the beginning of the season in the spring, there are still very few Varroa, so there's not really a lot of outbreeding at all. And then during the summer, there's a lot of brood, so there's still not really enough outbreeding. And then in a fall is really when the outbreeding happens, because the brood number starts to decline and the Varroa numbers are extremely high. So then, at the end of the season, outbreeding does happen, sometimes.

Jamie 17:57
But you're arguing that it's not enough outbreeding to perpetuate the gene drive, though, in that particular case?

Guest 18:04
Yeah, no, it's not enough for the gene drive, because, remember that the gene drives sort of spreads over generations. So if, if there's no outbreeding, then the gene drive is just staying in its own little, like, lineage and not jumping to the whole population.

Jamie 18:22

This, this is really interesting. So, in this particular case, though, if I understand correctly, your model is predicting in two to three years, it could happen. But your argument is in two to three years, the colonies are dead because the Varroa populations are just too high. Right? That's, that's essentially the idea.

Guest 18:41
The gene drive after two or three years is still at quite low frequency.

Jamie 18:45
Oh, gosh.

Guest 18:45
So, even that is not enough.

Jamie 18:47
Okay. So, the take home message is just what you said. Essentially, it does not look like that this is a, potentially, a good strategy for Varroa control long-term.

Guest 18:57
Yeah, exactly.

Amy 18:58
Yeah. So, I mean, I guess that just means for beekeepers, that they continue to do what they're doing now by applying miticides and rotating active ingredients. And, you know, many beekeepers have that brood break, which, which tries to help minimize the amount of Varroa in their colonies as well. So, I guess my next question is really just, you know, what would need to be done next? Or are there any things that we could do to move forward with this? I mean, is it just one of those things where you've decided, you know, it's probably not going to happen, so we're not going to look at it anymore, and we're going to go back to working with squirrels or, you know, is there something that could be done next, but what do you think?

Guest 19:41
I think in terms of the gene drive for Varroa control, I don't think it's going to happen. But for gene drives, in general, there's still a lot of work to be done, of course, and one of the things that I'm looking into in my PhD is to see how genetic diversity impacts the efficiency of the gene drive, because most gene drives are still being tested in model organisms and in lab strains of those organisms. So, there's very few genetic diversity. And I'm trying to see, if you actually move the gene drive to the real world, if it's still as effective as it seems to be in some species. So, that's, that's the thing that I'm working on. But, I would say for the Varroas, probably, it's a dead end.

Jamie 20:27
That's really interesting to hear, because oftentimes, you know, these genetic technologies can be touted as the next. the next thing, and it's so fascinating to me that the Varroa life history just absolutely works against it. And I think that that probably, I just think about your skill set with modeling and your

genetic technology skill set. It just seems to me that there's so many other questions that can be answered for Varroa using these modeling strategies as well. I mean, I think about the development of pesticide resistance in a, in a Varroa population. How quickly does that gene spread through the population when a single mite becomes resistant to a particular treatment? How quickly might that show up in the population? And what about its life history perpetuates those genes or, or truncates, their perpetuation? It's really fascinating and your research, while it might have shown that maybe gene drive's not an appropriate technology for Varroa, it does to me, open a great world of how modeling in Varroa can answer so many of these other questions that beekeepers have, and it's really interesting that you took that approach and work with your colleagues, both at University of Edinburgh, here at Purdue University, and, and even others to study this. It's really fascinating.

Guest 21:47
Thank you. Yeah, it really underlines the importance of modeling things first, especially for gene drives, before you actually jump in and start by putting in the lab because it might not even really work in the first place.

Jamie 22:01
I'll have to ask that age old question, though. So, everything you found, while it is interesting, it's completely based on a model. Can you envision a scenario that you could still try it in the field, and it might work? Or do you think the model is conclusive enough to where this is just it, you've got the answer, and maybe shouldn't follow this any further?

Guest 22:22
I think you could try it but it might be a lot of work for the little promise it's showing now, because to move a gene drive, or to actually get a gene drive in practice, then you need to first make sure that CRISPR is working in your species, which is already not trivial. And then building a gene drive and optimizing it is also not easy at all. I think a lot of people who know about gene drives get a little scared. But actually, I think that's not completely necessary because it's really hard to get a gene drive to work. So, yeah, it might be a lot of work to try it, to just try it in the lab, even though the model already shows that it might not work. But, you could do it.

Jamie 23:10
So, no, yeah, I can hear in your voice that it probably shouldn't be done. I can hear that hesitation. You're, you're saying, "My model looks good. I want to make sure that it saves some people time." Nicky, this has all been fascinating stuff. Thank you so much for studying it. I really appreciate you spending some time with us discussing your research here on this podcast.

Guest 23:30
Yeah, thank you so much for having me.

Jamie 23:31
Absolutely. Good luck with your PhD work.

Guest 23:34

Thank you.

Amy 23:47
You know, Jamie, I just, every time we talk about Varroa, I just, like, think about how weird Varroa is, right? Like, they're just so strange, and we try to get rid of them. And we try these different methods of how to get rid of them. And it just is, like, maybe we will get rid of them one day, but for some reason, that last episode, I felt like she was like, "Okay, it's not going to work." And maybe that's a good thing, you know, because maybe we can focus on other efforts. And I think that's okay, sometimes in research to at least take a look and know that something doesn't work. And so, I wanted to kind of talk about just the modeling process in gene drives in general, because I hadn't ever really heard of a gene drive before today. I mean, I don't know, I just had not heard of it.

Jamie 24:31
Yeah, the whole topic is really interesting, right? And so it's this, this idea that you can put a gene into something that limits, in this case, she kept talking about fertility of the females, and that this gene is, quote unquote, selfish, so that it perpetuates in the population more, more than should just randomly occur, right? So, instead of a 50-50 inheritance, it's nearly a 99% inheritance. So, that means very quickly, it becomes the dominant allele of that particular gene throughout the population, and then you experience population growth. Absolutely fascinating. A lot of this is made possible by this newer, or new-ish, technology that a lot of -- it's hard not to hear about if you go to a research meeting, this CRISPR/Cas9 technology, which I really loved the way that Nicky described it, which is just a copy paste mechanism. You copy and paste something into the genome, and then from there, you can follow along how it goes. And Amy, you know, it was a little bit of a bummer through the process, that the ultimate conclusion is that this wouldn't work for Varroa. But it really, it really introduced us to the world and importance of modeling, right? So --

Amy 25:47
Yeah, I mean, so when I --

Jamie 25:48
I'll pause there and let you follow up.

Amy 25:51
So, when I first read the, when I first read the title, I was thinking that they were doing something, you know, in the laboratory. And then, as I started getting more into it, I realized that it was this model. And so, I was wondering if you could maybe just talk about, you know, modeling in the scientific world. You know, is that kind of like what people do before they go into the actual lab piece, before they go into the field piece? Like, I don't know, I'm learning about science right now. So, what's up with the modeling?

Jamie 26:16
Amy, it's probably what people should do before they go into the lab or to the field. But, so, modeling is a very specific discipline that's not a skill set that everyone has, right? So, I'm a biologist by training, and entomology, the study of insects, is just a sub-discipline of biology. And so, I tend to be an organism specialist. So, I understand how an organism functions. Well, within biology, there's also

modeling, there's also physiology, there's genetics, there's population ecology, and on and on and on. And so, what Nicky brought to the table is a bit of modeling expertise. And models are, are essentially mathematical ways of predicting how something will happen based on the influence of things that you write into the model. So, for example, if you know, if you start, she kept mentioning things like, "Well, we started with 10 Varroa at the beginning of the season, and we modeled in how brood increases over time, and we know the reproductive rate of Varroa. So, we can say if we start with 10 Varroa with this much brood, as the brood population grows throughout the year, we can predict that we'll get this many Varroa at this time, and this many." And the model is more complicated than that because then she said, "Well, now let's argue that we put in one modified individual or 50 modified individuals, and how quickly it would take. Now, we've also got a model in the fact that, you know, a good chunk of Varroa aren't outbreeding, they're only going into single cells by themselves. So, there's other -- no other Varroa in there to produce offspring with which their offspring can cross." And so, essentially, you're throwing in to this mathematical system. Everything that you can think of to predict the likelihood of whatever outcome you're interested in knowing. And so based on brood growth, based on the number of Varroa you start with, based on how heritable a trait is, all of these things can be put into the model. Now, models are just that. They're models. And we all know that the real world doesn't always do what models, say. For example, let's just pick on the one that everybody knows, which is weather. Weather forecasts are models.

Amy 28:32
Right.

Jamie 28:33
Yeah, that's all they are, is someone's put into calculation what the weather currently is, where the fronts are, all of this stuff, the barometric pressure, the humidity, all that stuff feeds into a model, then, to predict, oh, it's going to be 30 degrees outside today and sunny, and we're, 17% chance of rain. All of that is the output of a model. So, models are only as good as the amount of information that you're able to feed into them on the front end. So, Nicky kept saying, you know, it's possible that you could go into the field or the lab, and this might work, but our model is predicting that it's highly unlikely. And so, the beauty of models is they can save people lots of time, right, from the laboratory's perspective, or they can make predictions that, like weather that we use every day. So, it's not a skill set we all have, it's certainly not a skill set I have, but it's a very powerful tool. But, again, they're only as good is the information that you're able to feed into it on the front end. That's why we predict weather better today than we did 50 years ago, because we've got more information to feed into the model. Well, as we learn more about Varroa and bee biology, etc., our models will get better. But, it sounded to me, if I interpret what she said correctly, that her model nearly conclusively show that this isn't necessarily a route that should be pursued for Varroa control, which I still find fascinating.

Amy 29:58
Yeah, I do too. I just love that all the guests that we bring on to the podcast, it's just there's so many different perspectives, so many different researchers and specialists out there. And it's really fun to see everyone kind of playing their part in the scientific world.

Jamie 30:11

I agree completely, Amy. And for you listeners out there, I hope that you appreciate the diversity and the expertise variety that comes across from all the beekeepers and bee scientists that we have on our podcast. You know, there's a lot of stuff that I can do and a lot of stuff I can't do, a lot of stuff Amy can and can't do. And the fact that we're able to bring in folks with all of these, just, differing ideas, I really hope helps you enjoy bees and beekeeping better, makes you guys better beekeepers. So, Amy, ultimately, it's to help beekeepers. I hope you beekeeper listeners out there enjoyed being able to hear that interview with Nicky because I found what she talked about really, really, really interesting.

Stump The Chump 30:52
It's everybody's favorite game show, Stump the Chump.

Amy 31:02
Welcome back to the question and answer segment. Jamie, the first question we have, I'm going to ask this, and I'm not even quite sure how to ask this, but the first one is: Is there a difference between frames being perpendicular versus parallel to the entrance of the hive? And that's, that was not the difficult question that I was going to ask. I was going to ask is, someone was saying something about a warm way versus a cold way. So, I'm wondering what the heck that means.

Jamie 31:30
So, Amy, before I even answer that question, the benefit of doing a podcast where we've got thousands of listeners is if I'm wrong about this upcoming answer, someone will point it out to me. And we have had a couple of podcasts on thermoregulation and honey bee colonies, and I want to stress this is not my expertise. So, I'm going to answer with what I know, but if you're listening out there, and know a -- if I'm wrong or a more complete answer, or if I'm right, feel free to just email me and let me know. I'll be happy to change this answer in a future podcast. But this is an interesting question, because Chris Oster, our lab manager, came by and asked me this question yesterday. And then he went and did a little bit of a literature review on this topic. And anyway, I'll tell you what we shared. So, generally speaking, we say that this is the best management practice. I'm not even sure if it's necessary, but it's just what, quote, we say, beekeepers say. Generally speaking, the recommendation is to face colonies south, right? If you're in the northern hemisphere and your colony faces south, the sun moves, well, not the sun moves, but the way the Earth turns, it gives the appearance that the sun moves from east to west across our sky. And it takes a bit of a southern orbit when it does that. So, essentially, when the Earth is turning, if you're in the northern hemisphere, and you point your colony south, it maximizes sun exposure on the entrance of the colony, and I'm assuming if you're in the southern hemisphere, it might be pointing north may be the recommendation, but that may not be a safe assumption. So, I'll just throw that out there. Okay. The idea is maximum sun exposure on the colony entrance keeps the bee colonies active longer. I don't know that this has ever been tested. It's just one of those general recommendations. This specific question is interesting, because, frankly, I've never thought about it, which is if you orient -- so most colonies that we use here in the States, or most hives that we use here in the States, the frames run perpendicular to the nest entrance. So, if your nest is facing south, then your frames are running north south.

Amy 33:39
Yep.

Jamie 33:40
Okay. But, in Europe, some hive designs are more square than they are rectangular. So, you, you have the opportunity within the hive to run the frames perpendicular to the nest entrance or parallel to the nest entrance. So, this question is essentially saying, does it matter from a thermoregulation perspective, if the frames are running, you know, with the nest entrance or against the nest entrance? And I would argue maybe it's less important with the nest entrance because you can turn the hive. And maybe, maybe the question is more should frames run north south, or should they run east west? And I've never heard that it matters at all about thermoregulation. So, Chris actually went and looked this up yesterday and found, again, this is one paper, and I want to stress that there could be new research out there on this topic that we've overlooked, but it didn't matter from a thermoregulation perspective. He did find one paper, though, in bee world that was, I don't remember, 30, 40, 50 years old. It was an interesting thing, is that there was some super minor evidence at the time that bees might be orienting their comb from a magnetic --Earth's magnetic perspective. I don't want to chase this rabbit too far because I'm not sure I'm totally sold on this idea, but this idea that when colonies were put on combs that were running a certain direction, they would remove the bees from those hives, put them into a cylindrical hive that had a nest entrance in the very center bottom of the hive in a completely dark room with no other signals to the bees, they were more often than not building the combs in the same direction as they were built in the hive that they were pulled from originally. And this would suggest that they had, quote, remembered the orientation of their combs and realigned those when they were building it in a dark hive, absent other stimuli. And so, the authors had hypothesized that maybe they were able to do that from an Earth's magnetic memory perspective. "Hey, this is the way our cones were aligned, originally. Absent all other stimuli, we can align them this way now." But even that was a little new, was a little, I don't wanna use the word questionable, but it was kind of understudied and kind of just hypothesized. So, to make a long, long, long, long, long question short, sorry, answer short, we probably don't know with certainty. And if beekeepers do it, they probably do it more anecdotally. But, if you're an expert in thermoregulation, let me know. I do know, I do know, because I've seen it with my own eyes, is that colony clusters within a hive, during colder months, tend to migrate to the warm side of the box or the warm side of hive. For example, when my colonies are oriented in my backyard, I would usually face them east. Again, this is me not following my own best management practice. But, it's because that's the space that I had and the direction that I could point them. During November, you know, kind of moving into our cooler months, the clusters tended to be found on the southern side of the box, which further emphasizes that that's the side of the box that gets maximum sun exposure. So, we know clusters may move in a hive in response to the temperature they're feeling in the nest. And that's probably because our boxes are just naturally not very thermoregulatory sensitive. In other words, if the fact that they can detect the heat and migrate to one side of the box, it's not insulated appropriately. So, they can detect those things, but I don't know that it really matters at the end of the day if we orient our combs one way or another to maximize on this behavior, but if you're aware of other research suggesting otherwise, feel free to let me know.

Amy 37:51
All right, so the second question is not as hard as the first question.

Jamie 37:55

Thank goodness.

Amy 37:57
The second question is what is the melting temperature of beeswax?

Jamie 38:02
Well, if you're in the Fahrenheit world, that's, this is the answer for you Americans out there, it's around 140-145 degrees Fahrenheit, right? It depends on the adulterations in the wax, but it kind of hovers in there. If you're in the absolute rest of the world, the Celsius or centigrade using world, it's around 63. So 145 F or 63 C is the melting temperature of wax. Again, it all -- the wax part will melt at that temperature regardless, but if it's adulterated, it has a lot of debris or other stuff in it, it may appear to be melting slower than that or might need a slightly higher temperature. But, nevertheless, that's that's the target temperature.

Amy 38:41
And you never want to get it to, like, boiling, right?

Jamie 38:43
Exactly. Absolutely.

Amy 38:45
All right. So, the third question that we have. So, I don't know if our listeners have heard, but Varroa found its way to Australia recently, didn't it? And so, I'm just wanting to ask, you know, what's the situation with Varroa found in Australia?

Jamie 39:00
Yeah, so it's very unfortunate, Amy, just like what you mentioned. Varroa was found in Australia relatively recently. And the reason it's unfortunate is because if you've kept bees long enough and can remember the days of managing colonies without Varroa, those truly were the good old days. If you manage against Varroa, you know how difficult it is, you know how devastating it is to watch Varroa kill colonies. It's terrible. And fortunately, for Australian beekeepers, they were able to live for decades and decades and decades and manage bees under a paradigm that didn't include for Varroa, but now they have Varroa. It's terrible. We feel terrible for Australian beekeepers just because we know what it's going to produce in their industry. And of course, the government, being the regional governments and maybe even local governments, that are trying to figure out ways to get this under control, maybe slow the spread of Varroa, they've implemented a lot of strategies. I'm not an expert on it. So, I hate to dig myself into a hole talking about what they've done, but essentially, there's been a lot of things, which has included kind of no movement zones, quarantine zones, zones where if you keep bees in the zone, your colonies are inspected. I believe there's even been destruction of colonies that have been found to have Varroa in an early effort maybe to eradicate Varroa or to low the spread of Varroa. But it's, it's one of those things that truly is heartbreaking. I know the Australian beekeepers are trying to figure out what to do, the government's trying to figure out best how to help the beekeepers, I know it's created some conflict in some places in the industry there. This tends to be natural. I saw something very similar happened in Italy when small hive beetles were first found there. My heart goes out to the beekeepers

in Australia. And I hope out that all of their measurements to slow the spread of Varroa, or maybe even eradicate Varroa, are fruitful. Of course, when it, when it's established, it's very hard to eradicate. So, I kind of feel like, maybe, very soon there'll be moving into a new phase, which is, well how do we manage this thing? And it's just sad to see, but it's tough. We know that it's going to lead to the loss of colonies, and we hope for the best for them. But realistically speaking, we know they're entering a new world of managing beekeepers -- bee colonies -- in a way that they've never had to do before. And so, you know, my general hope and my general prayer is that we figure out, collectively, as a global community of beekeepers and bee scientists, we figure out how to manage Varroa better, that there are better control options on the table that we can be -- that this is something that doesn't have to consume our discussions in the bee world soon. And my heart goes out again to the beekeepers there. And I hope that, as a community, we bond, and figure out how to control this terrible, terrible thing that impacts bees, bee colonies around the world.

Amy 41:45
Yeah, I think that was really well said, Jamie. And you know, Varroa is just one day, we'll get it. We'll get it one day. My hopes are up. Alright, so if anyone has questions, we love getting the questions on social media. We are open to having questions on our email listserv. Jamie and I mentioned it in the past, but if we have not gotten to your question, please feel free to send us another question and, and we'll try to get it on the next episode. And so, thank you so much for listening to this Q&A segment.

Serra Sowers 42:16
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.