

The Taproot podcast

Season 3, Episode 2 : The world according to Dan: truth, stats, and SNPs

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Guest: Dan Kliebenstein

Transcribed by Joe Stormer

[Instrumental theme music]

Ivan Baxter: Hello, Taproot listeners. Should we call you Taprooters? Tapsters? Tapenades? Tapas? I'm Ivan Baxter, and today we have another episode in our busting myths series.

Liz Haswell: And I'm Liz Haswell. Over the last decade, plant biologists have become more aware of the importance of carefully considered statistics – not just throwing the old t-test at things. Today's guest, Dan Kliebenstein, has been part of a movement to bust the myth that your data are true as long as p is less than 0.05. Recently he published an opinion piece in *Plant Cell* entitled “Reassess the t Test: Interact with All Your Data via ANOVA”. Today we're talking about another one of the papers from his lab.

Ivan: For those of you who may not be completely steeped in the population genetics world, a few terms for you: GWAS refers to *genome-wide association* mapping, a method for looking at the association between genetic markers (which we'll call SNPs in this discussion) and phenotypic traits in diverse populations. So with that, let's get on with the episode.

[Instrumental theme music]

Ivan: It is really my pleasure to introduce our guest today. He is . He got his PhD at Cornell with Rob Last and did a post-doc at the Max Planck Institute with Dr Thomas Mitchell-Olds and Jonathan Gershenzon. And then he moved on to the University of California-Davis, where he has progressed through the ranks and is now a full professor. And he's also a DG visiting professor at the University

of Copenhagen, where he's had an appointment for many years as well. Dan is an editor at four different journals including Plant Cell and eLife, which I think is just crazy [sic] and I don't know how he has the time. And he has published so many papers that I couldn't actually get a count from his CV. And he comes at things from a very different approach. I've always had very fascinating conversation with Dan and I know we're gonna have another one today. So he has a very active Twitter account where he spouts many of these interesting ideas. So Dan Kliebenstein, welcome to The Taproot.

Dan Kliebenstein: Thanks for having me.

Liz: Dan, it's so awesome to have you here. Like Ivan said, I always really enjoy following you on Twitter, reading your papers and seeing your seminars. Okay, so today's paper is a pretty recent paper to come out of Dan's lab called "The Quantitative Basis of the *Arabidopsis* Innate Immune System to Endemic Pathogens Depends on Pathogen Genetics". The first author is Jason Corwin and it came out in 2016 in PLOS Genetics. So Dan, you wanna just give us a brief overview of what this paper was about?

Dan: Sure. I have to work on titles, don't I?

Liz: That was a long one!

Ivan: We all have to work on titles.

Dan: Yeah, so this was like the semi-culmination of about ten years of work that started when Katherine Denby actually did a sabbatical in the lab and then through Heather Rowe, a grad student, and then Jason. And so he was interested in basically he wanted to get at just how many genes actually control a host-pathogen interaction and what might their identity be. And so most of the field was using specially biotrophs like *Pseudomonas* or something like that. So of course we had to say, "Let's just do the complete opposite and use a general necrotroph that basically eats anything that has a chloroplast." This was *Botrytis cinerea*. And Jason and Heather had been playing around and they're been noticing that there's tons of genetic variation in the pathogen itself, almost more

than in most plant species even. And so Jason added a third question in addition to how many genes and what they are. He said, "How does the pathogen actually influence the answer you find from the plant." And so he took these four diverse *Botrytis* isolates, put them in a hundred or so *Arabidopsis* successions and basically did GWAS. There were some specific genes we found, but we felt more comfortable in sort of the general conclusions that it says that there is a hundred to a thousand genes in the plant that's actually naturally variable in affecting the interactions. Which pathogen you actually use shifts the specific host genes you find and it's mostly genes that are not in the classical ETI amp-pamp map kianase pathways. It's actually the cell wall genes, the plant toxin genes, the things that are actually doing the resistance and not signaling the resistance, which is kind of different from the biotrophic view and the general of the committee that the important stuff was the signaling and the downstream stuff was kind of the boring worker ants. At least in this case, it looked like the downstream stuff was actually playing more of a role than the upstream stuff in natural variation.

Liz: So what, why do you think you found genes associated with downstream responses rather than the known signaling pathways? Is it the difference between looking for gene-expression patterns versus all the other people were doing genetic screens?

Dan: I don't think it's the mutant-screen aspect, cuz in Jane Glazebrook's old phytoalexin-deficient mutant screens, it was about half signaling and half actual camalexin biosynthesis. I think it's more if you want to get a big paper, you don't want boring old enzyme. You kind of want this transcription factor and then of course you call it a master regulator, right? But I think it's more what we consider exciting or interesting.

Ivan: So it's a choice of what to follow up on, in some ways?

Dan: Yeah, it's a choice of what to follow up on. Especially if you get a cell wall modifying enzyme, they are really hard to figure out what they exactly do and why they exactly have an effect. Whereas if you have a transcription factor you at least have EMSAs and all sorts of specific assays that you can do that'll walk

you towards a clearer answer.

Ivan: There was a great paper that came out two weeks ago in PLOS Biology looking at human genetics showing how there's a huge bias in which genes we study in humans towards a couple thousand already known genes.

Dan: A similar one about six years ago in yeast where they showed that the same thing was true in yeast. And I think they might have done the same work in *E. coli*. So it's not just a bias in humans. It looks like it's an issue in every organism that's studied.

Ivan: No, I don't think that it's a bias in studying humans; I think it's a human bias in how we study organism.

Dan: Yeah.

Ivan: You did all this complicated genetics and I know that you love complicated interactions. Does it bother you that you were only able to do four isolates of the fungus and not be able to do genetics of the fungus versus the genetics of the plant?

Dan: It bothered me but the reason that we did the four was when we sat down and did the math we realized we could do the hundred by hundred matrix, but we'd have no power for anything. And so then we focused on just the four to get an answer on that little aspect of the interaction. And then we flipped it to a hundred isolates with three plants to get a little view of that. And then the reason we only did the four was that we weren't sure if doing it in *Arabidopsis* was the right place to do it. That's why we went to these other dicots to try to ask like if we just start putting in all the plant phylogeny, where should we have asked the question?

Ivan: Yeah, super exciting. There's something that I wanted to follow up on. You mentioned that you were more comfortable with this paper saying about how many genes, not as much the specific identity of the genes.

Dan: Once you get to the specific identity, basically what we found was that

started becoming really isolate-specific, and then a masters student took Jason's image analysis and just said, "Let's not use lesion size as our measurement of whether a mutant has an effect; let's use the shape of the lesion or the color of the lesion." And all the sudden that started saying we went from a fifty percent success rate to like eighty percent of the mutants were having an effect on different aspects of the lesion.

Ivan: One question that I have have for you is you have these very kind of complicated set of experiments. There's a bunch of assumptions you made about what it would take for you to say this gene is one of the set involved in this process. And you definitely went in and tested some of those assumptions by modifying parameters and seeing if it changed the result, and in general it didn't do much. But there's a lot of sort of judgement calls that you have to make here so there's ambiguity and how do you deal with that ambiguity when you're trying to pull a paper like this together and try to publish it?

Dan: My sister's a lawyer and she's comfortable with the idea that you make an argument based on like hundreds to – at least tens to hundreds of legal cases' precedents before that and often times in science it feels like we've kind of lost that use of the existing literature. And so for example in this paper a lot of the ambiguous choices (like two of more SNPs per gene) actually came from previous papers on glucosinolates where we had previously cloned genes via QTL mapping for the past decade. And so then we went to GWAS with glucosinolates we had this collection of like ten-to-twenty empirically validated genes we knew we had to be there so we had this positive control set. And so then we used those genes with glucosinolates to ask how would we adjust the parameters to maximize our ability to see those genes. And then we took that and translated it to the *Botrytis*. And so there, probably the biggest ambiguous choice was we all assumed glucosinolates and *Botrytis* GWAS hits would have some sort of similar genetic architecture; and that part we didn't have the ability to test.

Liz: But what if you're starting out without that background? You have to decide for yourself on that sliding scale how many possible false negatives you're willing

to tolerate and how many false positives you will tolerate and then - I don't know - you're choosing statistics to try to find your way through a bunch of data.

Dan: Then you have to figure out who *you* are.

Liz: I thought you just have to make sure that p was less than 0.05, and then you were done. Right?

Dan: No, you have to think about how much effort you want to put in. Like do you want to test forty T-DNA mutants or do you want to test the most interesting T-DNA mutants? Cuz like often times, I mean unfortunately I don't get like an Ivan Baxter bionomics massive single three hit GWAS hits ever.

Ivan: I never get those anymore anyway.

Dan: Yeah, and I always had this sort of hundred-to-a-thousand genes just sort of at the threshold. So we kind of had to ask, "So we don't have any reason to say that one gene is any more interesting or less interesting than another." And then it became, "How much effort did Jason actually want to put in?" Could I get him to do a hundred T-DNA mutants? So then that became, "Okay, let's settle on twenty." So then we did make a choice there and said, "Let's focus on genes that have never been associated with the pathogen." So we actually took GWAS hits that were in known genes and didn't test them, cuz we figured the goal was to find new pathways. And then we just said, "We're gonna have some false negatives and we're gonna have some false positives, and we'll just throw the whole ugly mess out there and people can take with it what they want."

Ivan: And in this case it seems that the genes you tested had a phenotype, and you mentioned that if you get more creative with your phenotyping you get like 80%?

Dan: Yeah, it goes up and there's also a background effect because these are T-DNA mutants only in Columbia. So Columbia might actually be the wrong background for some genes to even have a visible phenotype or a detectable phenotype. And then that would get to sort of Liz's question about negative controls. We honestly don't know what would happen if we took a hundred

random (like single gene specific knock out mutants with nothing else in the background) and then tested them for resistance to anything.

Liz: Right. That's very interesting.

Dan: So we don't really have that sort of true absolute negative idea of "Do we have genes that we know don't affect a phenotype?" Cuz if you go to the circadian clock, you can start playing around and you start realizing that all these peripheral things have influences of like thirty minutes, twenty minutes, an hour, flowering time. There was a recent paper showing that they can find tons of mutants that were affecting flowering time. So then you start to get to the point where, "Is pleiotropy really rampant or are we all just doing the experiments wrong?"

Liz: How should people get around that? How does the field handle this issue of, "Pleiotropy is bad; you should have one phenotype", versus "Pleiotropy is on the other end of the scale; pleiotropy is inevitable, because that's the way organisms evolve." Dan, what's the way forward?

Dan: The way forward would probably be getting both sides to agree to an experiment that they would accept.

Ivan: So a registered report?

Liz: Yeah. Well let's zoom out a little bit and just talk a little bit more about statistics. You do tons of statistics in all of your papers; this is where your heart and soul is. What's your philosophy about all that.

Dan: It's kinda like we have a big oenology wine department here and they have kind of an argument, "Junk grapes, junk wine." SO if you think of the wine as the statistics, the grapes would be the experimental design. So for us, we worry as much about the experimental design as we do about the statistics. And so we will have – for *Botrytis* we'll have like eight replications per experiment and then we'll try to do three separate experiments at three separate times. And the benefit of that is you can look at the amount of difference between your genotypes, and then you can look at the amount of difference between your different experiments

and that gives you an idea of like, "Was the experiment variation really strong and the genotype was significant but it wasn't quite as strong." And that lets you say, "At least that lets us feel more comfortable in saying "Yes, this gene probably has an effect but it's not very big." And so then we work less about the p-value; the p-value is more like a guideline that lets us get the reviewers to buy into us and then we look at more where the variance or effect size is. That really depends on the reviewer community we're also expecting.

Liz: Yeah, but why is everybody so fixated on p less than 0.05. Why is that the cut-off and how has our field getting to the point where (I'm not saying that WE do this but) you can see people continuing to do experiments until they reach that point.

Dan: That part I don't know about. Every once in a while what we have is like if we have $p=0.05$, and we're like [tentatively], "Ehhh, I don't know." We'll actually repeat the whole thing over and if there's a real signal the p-value should go down but if there's no signal and we're just randomly sampling them we should get the p-value to go up. Because the idea that if you do more experiments the p-value keeps going down and down and down, it's only true if there is a signal.

Liz: Yeah, right, I get that. I guess I'm just trying to ask a more philosophical question about: I feel like people rely on p-values because we're uncomfortable with small differences and because there's so much inherent variability in biological systems as it is. We're really looking for ways to distinguish variability from meaningful difference. That make sense?

Dan: Well, right, it's the same things as why do you do a transcriptomics and you say $p\text{-value}=0.05$ and a two-fold difference. And so that's because in the early microarray experiments they didn't do stats so they just relied on the two-fold threshold. And then they were like, "Oh, that's kind of arbitrary," and then they said, "Let's put a p-value in."

Ivan: Yeah, and that was also because you could only do single microarrays for what we would now pay for a full RNA-seq experiment.

Dan: Or hundreds of them if you pool them, yeah. So I think it gets more to that idea of: there's so much variance out there that we want to use a threshold that's a little blanket for Linus to make us feel comfortable.

Ivan: Yeah. I think that when we make that the bar to publication and are not willing to let people have ambiguity at the end of their papers, it makes it harder to just keep moving the field forward.

Dan: When I was growing up and reading the first issues of Plant Cell, the last paragraph of papers used to be like, "What haven't we shown or what questions have we raised, and what needs to be done in the future?" That used to be in the 70s and 80s and the early 90s that papers were written. "What did we find? What do we have no clue about? What's confusing? And what do we do next?" Now it seems like discussions are like, "What did we find? Boom." And so then having absolute p-values to make absolute claims, it kind of fits into that. And often times if I've seen papers that use a multi-prong argument step like, "We have Point A, B, C, and D in these independent experiments and they support this claim," often times readers nowadays don't seem to grab onto that. They want a single figure that makes that claim. And then once you have a single figure desire then you have a single p-value desire, and a single effects value desire. And sort of that more complicated, developing an argument seems to have disappeared, or is at least not as received anymore. I don't know if that makes sense.

Liz: Absolutely, I know exactly what you mean. We've written papers where we say, you know, "We're gonna test this hypothesis. Here's evidence number one that it's correct, but of course it could also be that. So here's evidence number two that it could be correct, but it could also be this. But here's evidence three but it could also be that. But taken together . . ." [Laughs] The Venn diagram of all the possible explanations for all of these observations is our original hypothesis. Those are hard papers to write and hard papers to convince people to publish, for sure.

Dan: I know as an editor, there's the p-value issue and it's kind of related to a

more mechanistic detail in papers. So as an editor when a reviewer comes back and says, "This one point hasn't been fully proven," I've actually started asking myself and asking the reviewer often times, "Alright, what if you pull that entirely out of the paper. Does the story really fall apart?"

Liz: Right.

Dan: And most of the time the entire story doesn't fall apart if that one thing that's sort of bugging somebody is taken out or caveated or stuff. I have to do it as an editor paper-by-paper and reviewer-by-reviewer and have that discussion with everybody time-by-time. Because we've all had that review come at us, and so once you've had that review come at you like ten, twenty, thirty times, it's just natural human nature to turn around and kind of do that back (even if it wasn't the person doing it to you). It sort of trains you of what a review is suppose to be.

Liz: Exactly! You've got this sort of like reviewer responses in your pocket. One is, "Well, they've described a phenomenon but they haven't really made a molecular explanation or mechanism for it," or "Their statistics don't match $p=0.05$." It's like a list. It's a short cut.

Ivan: And people use it as – I think Dan's approach is saying, "What if you take this one of the four points out. Does it change the core message of the paper?" "Well, no, but it makes it less novel."

Dan: Yeah, I've heard that one. That one's usually like, then I'll turn back and say, "Okay, why is it less novel if a chair is still a chair." And usually it doesn't matter. It seems to be that the more you can get the reviewers into a discussion or the authors into a discussion, the easier it is to address all these things we hate about peer review. Or statistics, or mechanistic detail, or . . .

Ivan: So if I can change the direction of the conversation slightly. One of the things when you're thinking about the mythical p-value, the sort of the flip side is when people use it to argue that, "If the p-value is not 0.05, you know that they're the same." Which I think is even scarier.

Liz: Yeah.

Dan: I've never quite understood that one, right? At least I know that at the last lab at Cornell we used to have like two-and-a-half, three hour lab meetings, and Rob would always beat into us that the absence of evidence is not the evidence of absence. So that one I've never grasped. Why if it's like 0.20 p-value, people say there's no significant difference. They're not significantly different but it doesn't mean that if you did another environment or if you did the experiment a thousand times instead of ten times - maybe it's a really small difference. And I know in our lab (just the last couple of weeks), if there's not a significant difference, we've started to saying, "They're similar." And the word *similar* sort of has that fuzziness that allows you in the future to say, "Oh, if we did it in salt stress, then of course they changed. And in that experiment they're similar, and in this experiment they're different." And *similar* has just a little bit of that softer language connotation. I've used something like that but I usually say "indistinguishable in this set of experiments". I mean, part of this came about like I did a sabbatical as a grad student in Jeff Dangl's lab and I was work on superoxide dismutase antibodies in his old LSD1 mutant. He had three growth chambers that were from the same supplier and I would grow plants in all three of them and they would have a completely different superoxide dismutase profile, even though it was the same manufacturer and the same growth chamber, and the same soil and the same pots and the same seed batch. So I realized at that point that things that replicate between two labs are actually quite shocking to me sometime. Sometimes we'll actually do our experiments across four growth chambers intentionally, just so we can get a feed for how stable the mutant is versus how sensitive it is to those environmental changes that we think are small but can actually be quite big to the plant.

Liz: Yeah, I mean this is such an interesting conversation to think about whether what we're publishing is (I'm gonna do the air-quotes again) "truth". Cuz Dan, you're sort of harkening back to the olden days when it really felt like people would do some experiments and they would write up the results and then they

would say, "Here's what we think is happening; here's an argument that that might now be happening; and here's the future directions." And I feel like that whole sort of, "It's okay for this to be the state of science or the state of our understanding, but it's also okay for that to be mutable and for us to be the next you know do another set of experiments that might change our mind." I feel like that doesn't happen anymore, it like it really has to be a ladder on the rung of knowledge. But, I mean, maybe I shouldn't be talking like, "I'm some old time-y person." It feels like often papers are written to move us forward only exclusively toward truth. Do you know what I'm saying?

Dan: I guess it depends. I mean I've heard that statement like, "Is what we're publishing *the truth*," but only in the last couple of years."

Liz: Oh, that's interesting.

Dan: If I think back to like, like when I interviewed here, I interviewed and had to talk to two of the guys who basically founded plant chemistry and enzymology. If I had used the word *the truth* with them, they would have just annihilated. Right? Because *the truth* has sort of this singular connotation that there's ONE truth. I try to think of it more as, "Are the papers we're publishing *true* in the way we did them." It doesn't mean they're the *universal truth* that will apply in every environment and every growth chamber and every species. But is what we put out there true based on how we did it. Which I think to me is a little bit different than "Is what we're publishing *the truth*?" Given all the genotype by environment and species variation in animals and plants, there is no *the truth* really. But is the paper that was either we're publishing or put in front of me as an editor or reviewer; is it internally true – honest with reporting it, honest in preparing them, and honest in the materials and methods so people know what was and was not done. The truth gets to sort of the idea of the *master regulator*, gets to sort of the idea of the *important*. Are those terms hindering us in terms of moving forward?

Ivan: To come back to this idea of which is more important: is the paper done and written in a truthful manner or is it novel? There's a lot of pressure to say,

"This is novel," and what's novel about it. The idea of saying, "This is what we think our results say; this is what we don't know; this is what we have to do again to be sure about this," is just opening the door for somebody to say, "Well once you've done that next experiment, you'll have shown it and it'll be novel. So come back and you'll have done the next experiment and we'll all want to actually just publish this paper. Then we can worry about that."

Dan: Yeah, novelty is the toughest. I know I think about novelty very differently since I had Rob was a yeast geneticist, Tom was an evolutionary quant geneticist, and Jonathan was a biochemist. And so to me, a single enzyme or a single metabolite or a single transcription factor in the absence of any higher-order biology, I would say struggle finding novelty in that. While for me, host-pathogen interactions (ten genes or a thousand genes) for me is novel but it's only novel for a year or two and then, "Now we have a thousand, what's novel?" I know as an editor novelty is the hardest thing. Sometimes the really novel papers are easy because you're like, "I would have never, ever thought of that." But I do think we maybe don't allow people to write those of we punish them for trying to write them cuz there's so many question out there that we don't even contemplate asking because we're afraid that people are going to say, "That's just boring." So often times some of the most novel stuff I see is in the smaller sort of thematic journals where people are describing phenomena that we've never seen that before, ever. Those often times end up having the more novel aspects in twenty or thirty years, while the specific mechanistic novelty papers we're all excited about now sometimes fade away a little bit.

Liz: Yeah, well I think we'd both be interested in hearing what you would advise a young person sort of starting out and trying to feel their way around these kinds of issues – novelty and significance. What does a young person do? Do they chase the term *novelty*?

Dan: I don't know; am I the right person to say that?

Ivan: [Chuckling] Well, Dan, you are the editor of four journals and highly-esteemed community member.

Dan: But equally I've had lots of people tell me, "You have no Science, Nature, of Cell paper, so you're a failure."

Ivan: Pardon my French, but [bleep] them.

Dan: Right? The path I have taken is I've said what you just said. I think there are these interesting questions and I'm going to bash my head against the NSF funding wall until I get money for what I want to do, and hopefully people will find that exciting or interesting. It did take me (what was it?) seven years before I got my first fully-funded NSF grant, and I've only been in the job seventeen years. And I've only had four or five fully-funded grants myself. I would have reviews say, "Oh, Nobody in their right mind could clone a metabolite QTL. Nobody can use co-expression to find enzymes. UVR8 is definitely not the UVB photoreceptor. And so forth." I don't know honestly if I would suggest an early career researcher to follow that path nowadays. That is not a fun path and it can be very hard. To me, my personality was such that if I got money to do something I was bored with, I was not going to do it well. And so I had to fight to do something that I was really excited about. So I think for an early career researcher (or even a mid or late career researcher) you have to ask, "Who are you and what interests you?" And realize there are some paths that are going to be much more of a fight, and if that's the way you want to go, prepare for it and hunker down and go for it. And there's other ways that aren't quite so much a fight. I mean there'll be fights in a different way. And so for me it's: figure out what really interests you; think about whether or not it interests you because it interests everybody else or because you think it's the way the community should work or move forward; and constantly ask yourself if you're being nuts. I know I ask myself on a daily basis, "How crazy [sic]?" Like if we find something new like in this GWAS paper we've been talking about. All the caveats and tests were basically Jason and I going, "How did we screw this up? What could we have done wrong that is creating this pattern?" So that's the way I've kind of operated. And there are other ways to do it.

Ivan: Well I do think that's a very good way to think about some of these things

where you're not able to do the simple mutant wild type (the simple answer to a simple question), but trying to think through all the ways you could be wrong. If you can't account for them, at least be clear about why you can't account for them. Looking at your publication record, there is a market for those because you've been able to publish a lot of these papers. I know Dan is very passionate about asking for people who are evaluating publication records to take into account the actual papers and not where they're published, which I heartily endorse.

Dan: Whenever I'm chair of a search committee, I actually try to force the first round to be based on their writing about future teaching and future research solely, and not even look at their CV or publication record.

Liz: I can't even imagine that happening. It's sort of the same thing as I was talking about for p-values or anything else. It's like the shorthand for quality. We'll always looking for the fast route to a decision – the quick way to know, “This person is good, this person is not good.” And the trifecta of Science-Nature-Cell is just such an easy way to just make an evaluation without really thinking.

Ivan: Although as we have discussed on a previous podcast, that's not the best way to do that.

Liz: I'm just trying to understand why we feel driven to these short-hand and quick routes to evaluating anything – each other or publications or ideas or data. It just seems to be that the common theme is speed.

Dan: Well, speed and if you have a threshold and you're wrong, you can say, “Oh, it was just an experimental or technical error that caused it to cut that threshold,” whether it be the number of Science/Nature/Cell papers for somebody who's hired, a p-value for an experimental detail, or whatever threshold you're using. It's kind of that (at some deep, cynical, biting level) a threshold and a quantitative number is kind of an easy way to accept the chance that you might be wrong, but it's the threshold's fault. If you have to evaluate it on the content,

then it's subjective, and if there's an error it is you and the way you thought about it.

Ivan: Well, I can say with high confidence, a p-value of WAY less than 0.05, that this has been a really interesting and exciting discussion, and I really appreciate you taking the time to talk with us, Dan. If people have follow-up questions or thoughts they want to share with you, how can they get a hold of you?

Dan: So the Twitter account is @SpicyBotrytis . Otherwise, all you have to do is Google search my last name (however you feel like spelling it) and it's find me. There are no other Dan Kliebensteins in the world.

Ivan: Alright. Liz, I know there are other Liz Haswells, but how can people reach this particular one?

Liz: This particular one can be found: she has a very highly novel Twitter feed, @EHaswell.

Ivan: And you can find my somewhat-novel, somewhat-quantitative, somewhat-significant Twitter presence, @BaxterTwi. And with that: thank you all for listening and thank you again, Dan, for joining us.

Liz: Thanks, Dan.

Dan: Thank you.

[Instrumental theme music]

Ivan: The Taproot is brought to you by the American Society of Plant Biologists and the Plantae website. It is cohosted and edited by Ivan Baxter and Liz Haswell, and produced by Mary Williams and Melanie Binder. We get editing help by ASPB Convirons scholar Juniper Kiss, and social media and blog post writing posting help by ASPB intern Katie Rogers. If you like this episode, tell your friends and colleagues and be sure to subscribe on your podcast player of choice. Thanks for listening, and we'll bring you another story behind the science next week.

[Instrumental theme music]