Webinar Q&A

Genetic dissection of disease resistance mechanisms hijacked by a necrotrophic pathogen of wheat

11 March 2021

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The webinar recording is available on the Phytobiomes Alliance YouTube channel at https://youtu.be/wmCU_lo0OSE

Q: It appears CS and Cadenza have several Snn genes. I wonder whether this makes these cultivars more susceptible to Stago?
Answer Given (timestamp 50:00)

Q: Are there circadian rhythms in the secretion of toxins during infection?
Answer Given (timestamp 51:20)

Q: In the ""Genetic dissection of host gene-NE interactions"" slide, could you please give us a few more details about the ""Inoculate mapping population"" experiment and the QTL analysis?
In addition, what distinguishes the NE-producing isolates from normal isolates? Are they engineered in expressing NEs highly?
Answer Given (timestamp 52:25)

Q: Is ToxA the only suspected case of horizontal gene transfer in these systems?
Answer Given (timestamp 55:35)

Q:I am working with the biotrophic pathogen causing powdery mildew and found it quite striking that Tsn1 has the typical NLR structure of R genes. Now I was wondering whether you know of any disease that Tsn1 causes resistance to? It would be interesting to test this, especially regarding that depending on the allele (sometimes minor changes) of a gene we find resistance or susceptibility.
Answer Given (timestamp 55:55)

Q: knocking out the Tsn1 e Snn1genes, does it cause any changes in the host besides the possible greater resistance to the host?
Answer Given (timestamp 57:22)

Q: Phylloplane interactions - would you expect a similar number & complexity of interactions amongst some compatible or incompatible members of the phylloplane microbiome?
Answer Given (timestamp 59:25)

Q: Kindly explain about three gene expression again and it's outcome?

The three genes Tsn1, Snn1 and Snn3 are all regulated by light. Transcription of Tsn1 and Snn1 declines through the daylight hours into early night, and then begins to increase and reaches maximum abundance at about dawn. Snn3 is also regulated by light, but its pattern is opposite of Tsn1 and Snn1. These oscillations are repeated every 24 hours.

Q: Do you have any ideas/evidence of what these genes may be involved in besides susceptibility for them to keep these genes evolutionary?
They are possibly defeated resistance genes that are still present in some germplasm. It is possible that some also serve other functions that we have not yet identified.

Q: Did you tried the edited mutants with other pathogens?
Not the edited mutants, but we have tested EMS-induced mutants for reaction to other pathogens. In these cases, there was no change compared to the wild types.

Q: Why are the Snn genes expression light dependent? do you have a theory ?
If Tsn1 and Snn1 once acted as R genes, they may have evolved to reach peak expression at dawn because that is the time of day when pathogens are most likely to infect. However, that is speculation.

Q: If all Snn genes activate identical signaling pathways, wouldn't it be more efficient to manipulate those to achieve resistance than knocking out many Snn genes?
Yes, that indeed would be a good strategy, but we would first need to determine if all, or which ones, activate the same pathways.

Q: Do you think you have identified at this stage all of the molecular interactions for this host-pathogen recognition system?
Most certainly not. We have data from infiltrations of crude culture filtrates of various isolates that indicates more S gene-NE interactions are involved. It is also possible that there are other types of interactions involved in this system that we have yet to discover.

Q: Great work!! Questions: 1- How did the snn/tsn genes evolve, since the plants are better off without having these genes to escape pathogen recognition??, 2- Are resistance genes to biotrophs more primitive than susceptibility genes to necrotrophs?
Your second question answers your first. Yes, biotrophs are more primitive than necrotrophs so R genes evolved first. Therefore, many Tsn/Snn genes are likely defeated R genes that are still hanging around in some germplasm.

Q: Did you observe any disagreement between toxin screening and pathogen screening of your mapping population??
Yes, NE screening does not always predict resistance/susceptibility to disease. For example, in durum wheat, the Tsn1-ToxA interaction plays no role what-so-ever in tan spot susceptibility. That is an extreme example, but we have yet to learn the reason for this. Other interactions might play minor to major roles in disease depending on the host genotype, what other interactions are also present, and other factors.

Q: I would like to ask if Tsn1 is the only gene involved in the production of TOXA in spot blotch? Can you please explain more about TOXA in Bipolaris sorokiniana.
I do not work on the spot blotch system, so I cannot answer if ToxA is the only NE produced. However, because spot blotch is a necrotroph, I would suggest that it is likely that it produces additional NEs.

Q: If breeders remove the susceptible genes for necrotrophic infection, will that not increase susceptibility for biotrophic infection?

Not unless those susceptibility genes are directly involved in the recognition of the infecting biotrophs. We have not observed this to be the case for any of these genes so far. All of the biotroph R genes and the necrotroph S genes known so far in wheat are different.