Developing sustainable strategies to protect wheat roots from take-all disease

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Q&A session

Presenter: **Javier Palma-Guerrero (Rothamsted Research, UK)**

The webinar recording is available on the IWGSC YouTube channel at https://youtu.be/25vkQUTkp3w

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Q: Thank you very much for this very interesting seminar. Very interesting CLSM and SEM studies! So you think the primary mode of action of Gh vs Gt is ISR via e.g. PR-1 or JA increase.

Our microscopic and transcriptomic results suggest that ISR is / may be involved during Gh infection resulting in fungal growth cessation in the outer cortex. We could detect upregulation of JA related genes during Gh infection, as well as genes involved in lignin production. We also found downregulation of genes involved in various aspects of cell wall synthesis, which has been shown to be related to a defense mechanism against infection by Fusarium in Arabidopsis roots resulting in JA and ethylene higher levels. We are now studying this in more detail by doing hormone analysis and confirming the different lignification response during Gh and Gt infection.
We also found PR1 upregulation during Gh infection, however, to my knowledge, PR1 activation is normally found during SAR and not during ISR. We haven't found upregulation of genes from the SA pathway during Gh infection, so we are waiting to obtain the hormone levels measurements results. It has been previously reported that roots respond differently to necrotrophic pathogens compared to the respond in aboveground tissue. It is possible that SA may play a more important role than JA in roots. No JA diagnostic genes were induction during Gt infection either. Therefore, it is possible that multiple mechanisms may be involved during Gh infection, but this need further investigation.

Q: Have you tested the levels of SA and JA levels from Gt and Gh inoculated root samples and controls at 2, 4 & 5 DAI.? If yes, how they are differing?

We are doing these measurements right now.

Q: Are the Gt strains crop specific or are can they affect several species e.g. wheat, barley, triticale?

G. tritici is quite specific to cereals, and it is limited to wheat, barley and triticale. It doesn't infect oats or triticale. Whereas T. avenae mainly infects oats.

Q: Have you found sources of tolerance/resistance in triticale that could be used to improve wheat resistance more easily than using crosses with T. monococcum?

We haven't found any resistance in the very limited number of commercial triticale cultivars so far explored. We have only found resistance in rye, but it is possible that some triticale lines may show resistance.

Q: Has Dr Mike Hammond Kosack also looked at FHB resistance?

Our core *T. monococcum* collection (n=50) was explored by the RRes team for FHB resistance through replicated , spray inoculated field trials in the mid-2000s. We did not observe any promising sources of FHB resistance.

Q: Have the natural antagonist candidates been tested in the field yet? Also, do you anticipate the antifungal compounds produced by the natural antagonists have a negative impact on the beneficial fungi in the field?

So far, we have only confirmed that Gh is not affecting wheat plants health in semi-field conditions assessing plants infecting by Gh at the adult stage. Gh didn't affect the plant measurements or grain yields, but we did this by using big pots with soil inoculated with Gh, not in field conditions.
One the RRes experimental farm we have several fields where Gh occurs naturally. When multiple cultivars have been tested in these fields, we have not observed any reductions in crop growth, development or final grain yield. This has been published - Osborne, S.-J., McMillan, V., White R. and Hammond-Kosack, K. E. (2018) Elite UK winter wheat cultivars differ in their ability to support the colonisation of beneficial root-infecting fungi Phialophora. Journal of Experimental Botany **69**, 3103-3115. Press release - https://www.rothamsted.ac.uk/news/crop-immunisation-can-root-out-take-all-fungus
We have also tested the natural antagonists against the beneficial Gh fungus during confrontations in petri dishes and we did not observe any growth inhibition, which suggests that antagonists and the beneficial Gh are compatible.

Q: Do you investigated how many candidate genes were identified from pathogens which are interactions with known target proteins of host?

I am not sure that I understand well this question. But in any case, we have not yet explored the fungal transcriptome, because the genome sequences for our strains will not be available until 2022. Therefore, we don't know yet what fungal genes are induced during root infection. We have not explored the *in-planta* Gt transcriptome studies recently published by other groups specifically to address this topic.

Q: Thank you for this interesting and informative seminar. Have major QTLs been described from controlled and field experiments in Triticum species? What is the heritability of the trait in controlled experiments? Thank you.

I am not sure that I understand well this question. But we don't have yet QTLs data for *T. monococcum*, we are currently genotyping the mapping population. We don't know yet about the heritability of the trait.

Q: Is the take-all disease enhanced in organic farming?

This is an interesting question. During organic farming, the crop rotation is usually more diverse and has a higher proportion of clover, and we know that higher residual N adversely affects take-all infection, so that could be beneficial against take-all. Not many organic farmers go in for rotations with consecutive winter wheats because of weed problems. They use a fallow or cover crop break and follow a winter cereal (wheat) with a spring cereal of barley. If they decide to go back to wheat after wheat then this could be a risk in terms of take-all disease.