Septoria tritici blotch (STB, caused by the fungus *Zymoseptoria tritici*) presents a significant threat to European wheat production, requiring substantial inputs of fungicide to exert adequate control and preserve yields for farmers. Within the EU, over 70% of annual EU fungicide usage in wheat is targeted towards STB, supporting a fungicide market worth ~€1.2bn per annum. In the absence of adequate control measures, STB can cause yield losses of up to 50% when susceptible wheat varieties are grown under weather conditions conducive to STB development. Unfortunately, our typical Irish climate is highly supportive of STB epidemics. Coupled with this is the loss of several chemistries due to the ability of *Z. tritici* to evolve resistance/tolerance to fungicides in a relatively short period.

**A tough opponent**

To date, three separate classes of fungicide have lost robust field efficacy against STB. In addition, the EU has recently removed the broad-acting fungicide chlorothalonil (CTL) from the registered list due to environmental concerns. CTL was a cornerstone of disease control strategies by mitigating the emergence of fungicide-resistant strains. As a result, Teagasc assessments indicate that the loss of CTL alone could reduce average net margins by up to 50% in wheat, with growers achieving national average yields at or just above break-even. In the short term, winter wheat production will only be a viable option for those with the lowest costs of production and with high-yielding sites. In the longer term, the future is bleaker because in the absence of CTL, a more rapid loss of efficacy of the remaining fungicides is expected due to high disease pressure. It is clear that if winter wheat production is to remain a viable enterprise in the Irish tillage sector, the generation of novel varieties with durable genetic resistance to STB is the only viable option remaining. When integrated into an appropriate integrated pest management (IPM) strategy, such resistant material would have the potential to support profitable returns and maintain wheat within current rotations. Delivering such resistant material is, however, challenging, primarily due to the knowledge deficit that exists surrounding the STB-wheat interaction and specifically with regard to the genetic response to STB through the infection cycle. As part of VICCI, we have made significant strides in addressing the knowledge gap by elucidating the infection cycle and identifying signature genetic networks and genes that are the first response of the plant to STB infection.

**A blotch on wheat production**

Understanding wheat’s response to septoria tritici blotch disease, the most destructive wheat disease in Western Europe.
A lot learned

*Z. tritici* spores quickly germinate upon landing on a leaf surface and gain entry into the leaf tissues by penetrating through the stomata (pores) in the leaf surface. However, for a period of time (up to 15 days) after this, there are no visible symptoms on the leaf surface, in spite of *Z. tritici* infection having occurred. Through research completed in VICCI, we now know that this ‘latent period’ is a critical aspect of the infection process. Indeed, extending the latent period will deliver a significant decrease in symptom development, and overall disease within the crop, which in turn reduces the necessity for high inputs of fungicide.

In parallel, we have also begun to interpret the genetic response of wheat to STB infection through the latent period. Using sequencing technology, we have identified the key host and pathogen genes involved in the early development of STB disease. Genome studies and cognate studies of the wheat metabolome (the metabolites produced in response to disease) have also given us great insights into the way that the disease evolves to attack new wheat varieties. Separately, we have identified new pathways involved in wheat’s defence against disease. This includes novel rapidly evolving genes, termed ‘orphan genes’ that are responsive to STB disease. Our findings also contradict some earlier studies, which suggested that the pathogen is relatively inactive during the early stages of disease: our genome studies suggest that there is significant cross-talk between the host and pathogen even as early as six hours after the pathogen infects the plant.

**Identifying resistant genes**

As part of these genome studies in VICCI, we have shown that the pathogen produces specific proteins that enable it to attack wheat varieties and, in parallel, wheat has evolved resistance proteins to help defend itself against this same attack. Different strains of the pathogen carry different attack genes, so called effectors. And wheat varieties differ in the resistance genes they carry and thus in the spectrum of pathogen strains that they can combat. Knowledge regarding the effectors present in Irish STB strains and the resistance genes present in the wheat varieties grown here is critical if we are to help breeders to tailor wheat varieties for the Irish tillage sector.

Breeders invest a lot of time and effort into introgressing resistance genes into new wheat varieties and thus it is very important that they have evidence that the selected genes have a significant quantitative effect on disease resistance. In VICCI, using gene silencing technology, we have validated the role of specific genes in STB disease resistance. VICCI has highlighted these genes as important targets for breeding STB-resistant wheat varieties.

STB disease has a very narrow host range and we hypothesised that this was because wheat carries genes that make it, unlike its relatives, susceptible to the disease. Through a collaboration with the John Innes Centre in the UK, we have identified two wheat lines with enhanced STB resistance and we are working with industry to map this resistance and introgress it into prebreeding material. While more time is required, it is clear that, armed with this new knowledge regarding the dynamic between wheat and STB disease, we are now in a much more informed position to help breeders develop wheat adapted to Irish disease pressures.

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