Poster 15. Wheat lectins: A key defense strategy against Hessian fly attack.

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The Hessian fly [*Mayetiola destructor* (Say)] is one of the most destructive pests of wheat (*Triticum aestivum* L.) worldwide. The wheat–Hessian fly interaction operates in a gene-for-gene manner and can be classified as exhibiting incompatible (resistant plant) or compatible (susceptible plant) interactions. Virulent larvae cause stunting and yield loss in susceptible plants, whereas avirulent larvae die within a few days of the infestation. The resistant plants show little sign of stress or yield loss, suggesting an energy-efficient active defense at the molecular and physiological levels. To unravel the molecular mechanisms operating during compatible and incompatible interactions, we employed a transcriptomics approach utilizing various tools such as differential display, Curagen Gene-Calling, and the Affymetrix GeneChip Wheat genome array to identify unique and novel genes that are differentially expressed in these interactions. Our studies revealed the accumulation of transcripts belonging to a prominent class of genes encoding lectins in the resistant plants. Plant lectins, also referred to as agglutinins, are a heterogenous group of proteins that are able to reversibly bind simple sugars and/or complex carbohydrates and have been implicated in defense against pests and pathogens. The lectins identified in the wheat plants could be grouped into several categories such as the jacalin-related lectins (*Hfr-1*, *Wci-1*, Horcolin), chitin-binding lectins (*Hfr-3*, WGA), and others. Quantitative, real-time PCR studies indicated a strong accumulation of transcripts of some lectin genes in the resistant plants and positively correlated with increased protein levels as assessed by immunodetection. Further functional characterization of one of the lectins, HFR-1, revealed its antifeedant and insecticidal properties leading to detrimental effects on related dipteran larvae. Hessian fly larval behavioral studies showed that avirulent Hessian fly larvae on resistant plants exhibited prolonged searching and writhing behaviors as they unsuccessfully attempted to establish feeding sites. The rapid accumulation of HFR-1 and other lectins indicates an early defense response to Hessian fly larval attack and correlates well with the behavior of the avirulent larvae on the wheat plants. The predominant mode of action seems to be contributing to conditions that starve the avirulent larvae, leading to antibiosis. Our results open up potential applications in engineering transgenic wheat plant lines over-expressing lectins that will confer resistance against this and other devastating insect pests.

Poster 16. Lesion mimic associates with adult-plant resistance to leaf rust in wheat.

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Lesion mimic (LM) symptoms that resemble plant disease symptoms in the absence of pathogen infection may confer enhanced disease resistance to a wide range of pathogens. Wheat line Ning7840 shows LM symptoms at heading that resemble flecking symptoms of initial leaf rust infection and adult-plant resistance (APR) to leaf rust. The gene responsible for LM, designated as *lm*, was a recessive gene from a natural mutation and was located on the proximal region of chromosome 1BL within deletion bin C1BL6-0.32 using a population of 179 recombinant inbred lines (RIL) derived from the cross 'Ning7840/Chokwang'. Ning7840 has the short arm of chromosome 1R from the rye T1B°·1R translocation, therefore, carries *Lr26*. To identify the gene for APR to leaf rust and understand the relationship between *lm* and APR, the RIL population was infected with the isolate PRTUS55, an isolate virulent to *Lr26*, at anthesis in greenhouse experiments. The result showed that *lm* was associated with APR to rust, and the lines with the LM phenotype had a significantly higher level of resistance than those non-LM lines across all experiments. Composite interval mapping consistently detected a QTL, *Qlr.pser.1B,* for APR on chromosome 1BL. *Qlr.pser.1B* co-segregated with *lm* and explained 61.0% of phenotypic variation for leaf rust resistance in two greenhouse experiments. An additional QTL was detected on chromosome 7DS and coincided with the marker for an APR gene *Lr34* (csLV-Lr34). A significant interaction was observed between *lm* and *Lr34*. A combination of the two genes significantly reduced both rust area and infection type. The gene *lm* may have pleiotropic effect on APR by limiting the growth and development of fungi in wheat leaf tissue.