Poster 51. Genomic analysis and fine-mapping of two homoeologous wheat genes conferring susceptibility to Stagonospora nodorum blotch.

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The necrotrophic fungal pathogen Stagonospora nodorum produces multiple necrotrophic effectors (NEs), also known as host-selective toxins, which interact with corresponding wheat genes in an inverse gene-for-gene manner to cause the disease Stagonospora nodorum blotch (SNB). In previous research, we showed that the homoeologous wheat genes Sn3-B1 and Sn3-D1, located on wheat chromosome arms 5BS and 5DS, respectively, both recognize the NE SnTox3 to confer effector-triggered susceptibility. Here, we describe genome analysis and mapping results from ongoing efforts to clone the two Sn3 genes. Saturation mapping of the genes in relatively small F₂ populations using SSRs and EST-derived markers followed by comparative analysis with the rice and Brachypodium genomes revealed that both the Sn3-B1 and Sn3-D1 regions were highly conserved with regions of rice chromosome 12 and Brachypodium chromosome 4. This colinearity allowed us to develop numerous additional markers to further saturate the Sn3-B1 and Sn3-D1 regions. Subsequent fine-mapping of both genes in large F₂ populations resolved some co-segregating markers and delinated the genes to small intervals. BAC contigs identified with flanking markers were anchored to the Sn3-D1 genetic map. The ratio of physical to genetic distance in the Sn3-D1 region was estimated to be 500–800 kb/cM. Because these two NE sensitivity genes are homoeologous, we can work towards cloning them in parallel and, once cloned, we can study their evolutionary history and investigate their functional roles in mediating recognition of SnTox3.

Poster 52. Characterization of natural variation in the Tsn1 gene in Aegilops speltoides.

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The Tsn1 gene confers sensitivity to the necrotrophic effector (NE) ToxA, which is produced by the pathogens that cause tan spot and Stagonospora nodorum blotch on wheat. Although Tsn1 is a susceptibility gene, it contains resistance gene-like features such as protein kinase, nucleotide binding (NB), and leucine-rich repeat (LRR) domains. Previous research indicated that Tsn1 arose in the diploid B-genome progenitor of polyploid wheat. However, nucleotide variation in Tsn1 is nearly nonexistent among polyploids. Here, accessions of Aegilops speltoides (SS genome), a close relative of the B-genome progenitor, were studied to further characterize the structure, function, evolution, and diversity of Tsn1. Multiple plants from each of 123 accessions were evaluated for reaction to ToxA and genotyped for presence of Tsn1. A total of 95 accessions were insensitive to ToxA and null for Tsn1, whereas the remaining 28 harbored Tsn1 alleles and were either sensitive or insensitive to ToxA. Comparative sequence analysis of the 4,473-bp coding region from 15 sensitive Ae. speltoides plants revealed numerous single nucleotide polymorphisms (SNPs) compared to the Tsn1 allele in the durum wheat variety Langdon. Among Ae. speltoides accessions, there were approximately the same number of nonsynonymous and synonymous mutations, but none of the nonsynonymous changes occurred within the protein kinase, NB, or LRR domains indicating the importance of these domains for Tsn1 function. The diversity in Ae. speltoides allowed us to gain a better understanding of the evolution of Tsn1, and further studies will enhance our understanding of Tsn1-ToxA interactions.