Poster 8. Saturation mapping of scab resistance QTL in Ernie and application to marker-assisted breeding.

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Fusarium head blight (FHB) is caused mainly by *Fusarium graminearum* in wheat and results in significant yield and quality losses in humid and warm areas of the world. QTL for scab resistance have been mapped in exotic and native sources. However, only a few QTL have been widely deployed in breeding programs using marker-assisted selection due to the lack of diagnostic and tightly linked markers for most QTL. Four major QTL for type-II resistance were previously mapped on chromosomes 5A, 4B, 3BS, and 2B of Ernie. A set of 243 'Ernie/MO94-317' RILs were evaluated in inoculated, mist-irrigated, scab nurseries at Columbia, MO, and Blacksburg, VA. The 4B QTL region was associated with field FHB severity (R^2=4.2%), index (R^2=4.4%), kernel quality assessed as 100-grain weight (R^2=8.0%), and Fusarium-damaged kernels (FDK, R^2=6.2%). The awn-inhibitor gene B_1 is associated with field FHB incidence (R^2=4.5%) and index (R^2=5.3%) in the Virginia test and with FHB severity (R^2=4.2%) in the Missouri test. Another QTL associated with 100-grain weight is on chromosome 2DS (R^2=12.4%). One minor QTL for FDK (R^2=4.3%) on chromosome 5A is separate from the major QTL for type-II resistance and the B_1 gene. Tightly linked markers are being used for marker-assisted selection in breeding populations for the four QTL in Ernie and the two major QTL on chromosomes 3BS and 6B of Sumai 3, which will facilitate the pyramiding of various QTL for FHB resistance using marker-assisted selection in cultivar development. This material is based upon work supported by the U.S. Department of Agriculture under Agreement No. 59-0790-4-102. This is a coöperative project with the U.S. Wheat & Barley Scab Initiative.

Poster 9. Map-based cloning of the fungal toxin sensitivity gene Tsn1 in wheat.

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The wheat *Tsn1* gene on wheat chromosome arm 5BL confers sensitivity to the host-selective proteinaceous toxins Ptr ToxA and SnToxA produced by the pathogenic fungi *Pyrenophora tritici-repentis* and *Stagonospora nodorum*, respectively. Compatible *Tsn1*-ToxA interactions lead to extensive cell death and disease susceptibility. We employed a map-based strategy combined with haplotype analysis of natural populations to delineate the *Tsn1* candidate region to a 120-kb segment containing five genes. Comparative sequence analysis of multiple independent EMS-induced ToxA-insensitive mutants revealed that *Tsn1* is a member of the NBS-LRR class of disease resistance genes but, in this case, it confers susceptibility. Evaluation of the level of microcolinearity between the orthologous regions of wheat chromosomes 5A and 5B, *Brachypodium*, and rice indicated that the 5A region, *Brachypodium*, and rice share a higher level of microcolinearity than the 5B region does due to the presence of numerous transpositions, deletions, and rearrangements present in the wheat 5B region. *Tsn1* lies on a 100-kb, chromosome 5B-specific insertion that is specific to ToxA-sensitive genotypes. Homoeoalleles of *Tsn1* do not exist on chromosomes 5A and 5D, and recessive *tsn1* alleles are rare because ToxA-insensitivity is usually conferred by the null allele on 5B. Phylogenetic analysis indicated that *Tsn1* is related to other NBS-LRR proteins encoded by toxin sensitivity genes and several rust resistance genes from other grasses. The isolation of *Tsn1* will allow us to decipher the molecular interactions and mechanisms associated with the wheat-*P. tritici-repentis* and wheat-*S. nodorum* pathosystems.