The concurrence of Stagonospora nodorum blotch resistance with host-selective toxin insensitivity in tetraploid wheat.

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Resistance to Stagonospora nodorum blotch (SNB) in hexaploid wheat (*Triticum aestivum* L.) is associated with insensitivity to host-selective toxins (HSTs) produced by the pathogen. In this research, we evaluated the association between HST insensitivity and SNB resistance in tetraploid wheat (*T. turgidum* L.). Two natural populations consisting of 172 wild emmer (*T. turgidum* subsp. *dicoccoides*) accessions and 206 cultivated tetraploid wheat accessions, including *T. turgidum* subsps. *carthlicum, polonicum*, *turgidum*, *dicoccum*, and *turanicum*, were inoculated with a mixture of three *S. nodorum* isolates and infiltrated with purified SnToxA and crude culture filtrate of isolate Sn2000KO6-1 (an isolate with a disrupted *ToxA* gene). The associations between SNB resistance and insensitivity to SnToxA and Sn2000KO6-1 were 43% and 30%, respectively. To further investigate the correlation between SNB resistance and insensitivity to specific HSTs, a doubled-haploid (DH) population consisting of 146 lines was developed from the cross between the SNB-susceptible durum cultivar Lebsock and the SNB-resistant *T. turgidum* subsp. *carthlicum* accession PI 94749. Genetic linkage maps constructed in this population spanned 2,036.7 cM and consisted of 283 markers that covered all 14 chromosomes. We inoculated the population with the *S. nodorum* isolates Sn2000 and LDNSn4 to evaluate the development of SNB. We also infiltrated the population with the purified HSTs SnToxA and SnTox3 and with crude culture filtrate from isolates LDNSn4 and Sn2000KO6-1 to evaluate reaction to the HSTs and identify the corresponding host genes conferring sensitivity. QTL analysis revealed that genomic regions on chromosome arms 2BS, 4BS, and 5BL governed resistance to isolate Sn2000, and loci on chromosome arms 2BS, 3AS, 5BS, and 5BL conferred resistance to isolate LDNSn4. The effects of the 5BS and 5BL QTL were due to the underlying toxin sensitivity loci *Snn3* and *Tsn1,* which confer sensitivity to the previously characterized toxins SnTox3 and SnToxA, respectively. No evidence for a host-toxin interaction associated with the 3AS QTL was found, but the effects of the 2BS and 4BS QTL were due to novel host-toxin interactions that have not been previously reported. Therefore, these results led to the identification of two new *S. nodorum* HSTs and their corresponding host sensitivity genes, and they demonstrate that these novel host-toxin interactions, along with previously characterized host-toxin interactions, play important roles in the development of SNB in this population. This research indicates that host-toxin interactions in the wheat-*S. nodorum* pathosystem are major disease conferring factors in tetraploid wheat, just as they are in hexaploid wheat.positional cloning of two host-sensitivity genes: *Tsn1* on 5BL and *Snn1* on 1BS. Toward the map-based cloning of *Tsn1* on chromosome 5B, we sequenced and assembled chromosome 5A and 5B BAC contigs spanning the gene. Evaluation of gene content and micro-colinearity between the orthologous regions of 5A, 5B, and rice chromosome 9 indicated the 5A region and rice share a higher level of micro-colinearity than the 5B region does with rice due to the presence of numerous transpositions, deletions, and rearrangements present in the wheat 5B region. In addition, the 5B *Tsn1* candidate region is nearly four times larger than the corresponding region of 5A due to the presence of additional genes and transposable elements. At least ten genes exist within the 350-kb *Tsn1* candidate-gene region, and they are currently being validated by comparative sequence analysis of *Tsn1*-disrupted mutants and virus-induced gene silencing. An important applied by-product of this research is the development of efficient PCR-based markers for *Tsn1*, which are being used to introgress SnToxA insensitivity into adapted germ plasm. Overall, this research demonstrates the potential of the wheat–*S. nodorum* pathosystem to be an excellent toxin-based inverse gene-for-gene model.