mechanism of resistance and the basis of race specificity are due to gene-for-gene interactions. However, some disease resistance genes are thought to be durable, because they are not dependent on the recognition of a single *Avr* gene product from the pathogen by an R gene. The adult-plant resistance gene *Lr46* has provided non-race-specific resistance to leaf rust that has remained effective for more than 30 years. The gene also has a pleiotropic effect on resistance to stripe rust (*Yr29*). Using recombinant, chromosome substitution line populations, we previously located *Lr46* on the terminal region of the long arm of chromosome 1B that is syntenic to chromosome 5L of rice. To fine map the *Lr46* gene region, high resolution mapping (HRM) populations were developed that represent 4,100 gametes. The EST-derived, STS marker *XSTS3680* that co-segregated with *Lr46* in the original mapping populations was mapped 0.15 cM distal to *Lr46* in the HRM populations. A BAC contig of the *Lr46* region is being constructed. New SSR and SNP markers identified from the BAC clones and linked to *Lr46* have been evaluated on a set of diverse wheat lines to determine their usefulness for marker-assisted selection. Gene expression studies can complement map-based cloning efforts, because expression data can be used for identifying candidate genes, identifying expression markers, and for generating and testing hypotheses about genetic resistance mechanisms. To identify transcripts associated with *Lr46*-mediated adult-plant resistance, the Affymetrix Wheat GeneChip Microarray was used to identify transcriptional changes in isogenic lines with and without *Lr46*. Considering the increasing worldwide use of *Lr46* and other adult-plant genes for durable rust resistance, it is essential to obtain a greater understanding of their mechanisms of resistance. Also essential is obtaining the best possible markers for breeding for durable resistance.

Genetic analysis of host-toxin interactions in the wheat–Stagonospora nodorum pathosystem.

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Stagonospora nodorum, causal agent of Stagonospora nodorum blotch (SNB) in wheat, produces multiple necrosis-inducing host-selective toxins (HSTs) that interact with dominant host sensitivity genes to cause disease. Absence of either the toxin or the dominant host gene precludes recognition and results in an incompatible (resistant) response. Therefore, these host-toxin interactions are mirror images of classical gene-for-gene interactions. One of the first HSTs identified in this system was SnToxA, which was horizontally transferred from *S. nodorum* to the tan spot pathogen *Pyrenophora tritici-repentis* around 1941. This event is considered to have been significant for the establishment of tan spot as a pathogen. Sensitivity to SnToxA is governed by the *Tsn1* gene on the long arm of chromosome 5B. To date, eight additional toxins, designated SnTox1 through SnTox8, have been identified, and their corresponding host sensitivity genes, designated *Snn1* through *Snn8,* have been mapped to wheat chromosome arms 1BS, 2DS, 5BS, 4BL, 5BS, 6AL, 5DS, and 3DL, respectively. Genetic analysis of several host-toxin interactions indicates that they play important roles in the development of disease in adult plants as well as seedlings, and their effects are mostly additive. To gain a better understanding of compatible host-toxin interactions at the molecular level, we have embarked on the 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 4 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.