sion. More than 15 genes for resistance have been identified and named in wheat, some of which have been shown to interact in a gene-for-gene relationship. However, the trigger for the switch from biotrophic to nectrotrophic growth of the pathogen and the mechanisms of resistance in the host are not known. To better understand the biology of this pathosystem, the genome of the pathogen was sequenced completely by filling in the gaps in an 8.9× draft sequence. The essentially finished sequence contains 18 chromosomes from telomere to telomere, plus five fragments. Four of the five fragments contain telomeres so they presumably make up two additional chromosomes for a total of 20. A comparative bioinformatics analysis of *M. graminicola* with seven other sequenced fungal genomes revealed that *M. graminicola* possessed fewer enzymes than expected for degrading plant cell walls. Analyses of grass-infecting pathogens *versus* those from other hosts indicated that the suites of cell wall-degrading enzymes were tailored to break down the cell wall compositions of their particular hosts. The frequency of transposable elements in the genome of *M. graminicola* was intermediate between those of other sequenced fungi. Many long (> 10 kb) retrotransposons were identified in the finished genome compared to the draft sequence, indicating the need for finishing of other fungal genomes. Availability of the finished genome for *M. graminicola* should greatly aid research on this organism and will help to understand its interaction with wheat.

Poster 27. Transcriptome analysis of high-temperature adult-plant resistance conditioned by Yr39 during the wheat-Puccinia striiformis f. sp. tritici interaction.

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Stripe rust (caused by *Puccinia striiformis* Westend. f. sp. *tritici* Eriks. (*Pst*)) is a destructive disease of wheat worldwide. High-temperature, adult-plant resistance (HTAP) to stripe rust is non-race-specific, inherited quantitatively, and is often more durable than race-specific resistance. Previously, we identified and mapped the single *Yr39* HTAP stripe rust resistance gene in the spring wheat cultivar Alpowa, which was identified on chromosome 7BL and accounted for 64.2% of the variation in HTAP resistance. To identify transcripts associated with *Yr39*-mediated HTAP resistance, we selected two recombinant inbred lines from an 'Alpowa/Avocet Susceptible' cross that differed at the *Yr39* locus to represent an incompatible (*Yr39*) and compatible (*yr39*) interaction with *Pst*. Using the Affymetrix Wheat GeneChip, we profiled the transcription changes occurring in flag leaves of these two lines over a time-course after treatment with *Pst* urediniospores and mock-inoculation. This time-course study identified 107 and 10 transcripts that were significantly induced and repressed during *Yr39*-mediated HTAP resistance, respectively. Only one transcript was induced during the compatible interaction. The temporal pattern of transcript accumulation showed a peak at 48 h after infection, which was supported by quantitative PCR assays that showed a rapid increase in fungal biomass after this time in the compatible interaction. Most (64%) of the annotated transcripts specifically induced during HTAP resistance were involved in defense and/or signal transduction, including transcripts associated with pathogenesis-related protein production, phenylpropanoid (lignin) and anthocyanin biosynthesis, and receptor-protein-kinase signalling. As expected for non-race-specific resistance, no transcripts associated with an oxidative burst and/or hypersensitive response were identified. This study represents the first transcript profiling of HTAP resistance to stripe rust in wheat, and we conclude that *Yr39*-mediated HTAP resistance involves substantial gene expression changes associated with known nonspecific defense mechanisms.

Poster 28. Interdisciplinary approaches to understanding the mechanisms of cereal crop–aphid pest interactions using a wheat–greenbug system.

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The greenbug, *Schizaphis graminum* (Rondani), is an important aphid pest of small grain crops, especially wheat and sorghum in the Southern Plains of the U.S. No host resistance gene against aphid pests in cereal crops has been cloned, and the mechanisms of host resistance against aphid feeding are not well understood. At the Texas Agricultural Experiment Station – Amarillo, we have a research program aimed at understanding the mechanisms of interaction between the

phloem-feeding aphid pests and cereal crop hosts using the wheat–greenbug as a model system. Recent results from our research on the following projects follow. (1) Toward map-based cloning of the *Ae. tauschii*-derived greenbug resistance gene *Gb3* in wheat. *Gb3* was mapped in the distal bin of wheat chromosome arm 7DL. Fine genetic mapping for *Gb3* is under way. Marker enrichment has identified over 30 *Gb3*-linked SSR, AFLP-, EST-, or RFLP-converted STS markers in the distal bin. Two STS markers flanking *Gb3* are being used to screen an *Ae. tauschii* BAC library to initiate chromosome walking. (2) Expression profiling of host defense responses against greenbug feeding. In a 2-genotype (bulked segregant R and S super pools), 3-time-point (0, 24, and 48 hours after infestation, hai), 3-replicate experiment, 18 Affymetrix GeneChips were used to investigate *Gb3*-mediated defense responses upon greenbug feeding. Of the 55K transcripts surveyed, 48 showed significant differences in constitutive expression between the R and S pools ($P = 0.05$). Among more than 6,000 transcripts with significant changes in expression level in both genotypes at 24hai, 165 were significantly up-regulated in the R pool as compared with those in the S pool at either 24 hai or 48 hai or both. Defense responses to greenbug feeding appear to be more similar to plant pathogens, in which the jasmonic signaling pathway seems to play important roles. (3) Development of cross-species transferable microsatellite markers for evaluation of biotypic diversity in the greenbug. Over 100 SSR markers were developed through database mining of the pea aphid and green peach aphid EST and genomic resources. Cross species transferability of these markers was high. Sixty SSRs were used to evaluate genetic diversities among six greenbug biotypes. Host-associated genotypic variation and geographical differentiation among these clones were revealed.

Poster 29. Haplotype structure and genetic diversity at Fusarium head blight resistance QTL in soft winter wheat germ plasm.

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Several quantitative trait loci for resistance to *Fusarium* head blight (FHB) have been mapped in wheat. Haplotyping strategies make use of previous QTL mapping and molecular marker information. We selected markers reported to be near FHB resistance QTL mapped in Sumai 3, Wuhan 1, and Ernie to haplotype a large set of Eastern soft winter wheat lines submitted by breeders. The objectives of this research were to (1) determine the genetic relationship among soft winter wheat lines with native and exotic sources of resistance using simple sequence repeat (SSR) marker data, (2) compare the SSR marker haplotypes of soft winter wheat lines with those of Sumai 3, Wuhan 1, and Ernie at known FHB-resistance QTL, and (3) identify lines with novel sources of FHB resistance. Reaction of the soft winter wheat entries evaluated was skewed toward resistance, with 59 lines classified as resistant, 116 moderately resistant, and 28 intermediate. Only 12 and 18 lines were considered moderately susceptible and susceptible, respectively. Of the resistant lines, 24 have exotic sources of resistance in the pedigree and the remaining resistant lines had only soft winter germ plasm in their pedigrees. Entries were grouped into 16 clusters that were generally based on breeding program or geographic origin of lines. The Chinese wheat cultivars having the *Fhb1* resistance gene were grouped separately from all other entries. The eight soft winter wheat entries in this study that have the *Fhb1*-resistance gene based on haplotype data were resistant in the field evaluation. The *Xsts3B-256* and *Xgwm533* markers can be clearly used to identify lines with the *Fhb1*-resistance gene. However, fine mapping is needed in other regions in which FHB resistance QTL have been located; particularly for resistance from Ernie, because allele sizes of Ernie for markers in the 5A and 4BL QTL intervals are common among Eastern soft wheat germ plasm. A number of soft winter wheat breeding lines did not share any haplotype at known QTL evaluated in this study. These lines likely carry novel sources of FHB resistance.