SESSION 5:

GENE DISCOVERY AND ENGINEERING RESISTANCE

Chairperson: Steve Scofield

QUANTITATIVE TRAIT LOCI MAPPING OF FUSARIUM HEAD BLIGHT RESISTANCE IN ADVANCED BACK CROSS POPULATION (BC1F6) DERIVED FROM TUN 34 × LEBSOCK TETRAPLOID WHEAT

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ABSTRACT

Fusarium head blight (FHB) caused by *Fusarium graminearum* has resulted in significant reduction in grain yield, quality and farm income in durum wheat growing areas of North America. There are a few known sources of resistance for FHB mostly derived from the Chinese hexaploid sources like Sumai3 and Wangshuibai. Distinct sources of resistance were identified by NDSU in Tunisian pedigrees. It has been shown that Tunisian lines have no relation to Chinese genotypes and therefore can be used to enhance the resistance in durum wheat by pyramiding resistant genes from different genetic backgrounds. To expedite identification of durum lines carrying these resistant alleles in earlier generations, use of molecular markers associated with FHB is essential.

Genomic regions associated with FHB were examined in 168 progenies of tetraploid Tun34 × Lebsock advanced back cross (BC_1F_6) population. To construct the genetic map, a total of 2300 DArT markers were tested for polymorphism between parents. The polymorphic markers were assembled into linkage groups at likelihood ratio statistic (LOD) greater or equal to three and followed by assembly of a consensus map using Kosambi mapping function.

Of the total DArT markers screened for polymorphism between parent lines Tun34 and Lebsock, 379 clones (15.1%) were polymorphic. Segregation ratios were compared to expected ratios for all markers using chi-square goodness of fit test. Results indicate segregation distortion of 5.2% ($P<0.01$) for this population. Of 379 markers, 359 were assigned ($LOD\geq3.0$) into 44 linkage groups with the minimum number of three markers. Following the grouping, genetic maps were constructed. Almost all of the linkage groups except two could be assigned to durum wheat chromosomes by alignment to previous published maps.

Genomic scan using Kruskal–Wallis rank-sum test identified significant ($P\leq0.001$) putative QTL associated with FHB on chromosomes 5B, 2A, 6B, 7A and 7B. A region on chromosome arm 5BL (4cM interval) showed the highest K score and an increase in resistance to FHB due to alleles of Lebsock parent. Composite interval mapping confirmed the presence of this significant (LOD=6.1) QTL explaining 14.7% of phenotypic variation for FHB. Since this population was phenotyped at two different seasons in replicated experiments, information from these QTL can be used in marker assisted selection (MAS) to control FHB.

ACKNOWLEDGEMENT AND DISCLAIMER

This material is based upon work supported by the U.S. Department of Agriculture, under Agreement No. 59-0790-4-109. This is a cooperative project with the U.S. Wheat & Barley Scab Initiative. Any opinions, findings, conclusions, or recommendations expressed in this publication are those of the authors and do not necessarily reflect the view of the U.S. Department of Agriculture.

TESTING TRANSGENIC SPRING WHEAT AND BARLEY LINES FOR REACTION TO FUSARIUM HEAD BLIGHT: 2009 FIELD NURSERY REPORT

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ABSTRACT

The 2009 field screening nursery, with 128 wheat and 208 barley plots was located at UMore Park, Rosemount MN. Trial entries and untransformed controls were submitted by the University of Minnesota (19+1 wheat), the Donald Danforth Plant Science Center (4+2 wheat) and USDA (48+1 barley). Lines with known reactions to Fusarium head blight (FHB) were also included as checks. The wheat checks used were the moderately resistant Alsen and Tom, the moderately susceptible 2375 and the susceptible cultivars Wheaton and Roblin. The barley checks were the moderately resistant line M122 and the susceptible cultivars Conlon (2-rowed), Robust and Stander. The experimental design was a randomized block with four replicates. Plots were 2.4 m long single rows. The trial was planted on May 6, 2009. All plots, except a non-inoculated Wheaton check, were inoculated twice. The first inoculation was applied at anthesis for wheat and at head emergence for barley. The second inoculation was applied three days after the initial inoculation (dai) for each plot. The inoculum was a composite of 50 *F. graminearum* isolates at a concentration of 200,000 macroconidia.ml^{-1} with Tween 20 (polysorbate) added at 2.5 ml.L^{-1} as a wetting agent. The inoculum was applied using a CO_2-powered backpack sprayer fitted with a SS8003 TeeJet spray nozzle with an output of 10ml.sec^{-1} at a working pressure of 275 kPa. Mist-irrigation was applied from the first inoculation on June 26 till July 26 to facilitate FHB development. FHB incidence and severity were assessed visually 20-21 d.a.i. for wheat and 13-14 d.a.i. for barley on 20 arbitrarily selected spikes per plot. FHB incidence was determined by the percentage of spikes with visually symptomatic spikelets of the 20 spikes observed. FHB severity was determined as the percentage symptomatic spikelets of the total of all spikelets observed in these 20 spikes. Plots were harvested at maturity on August 14 (barley) and 24 (wheat). The harvested seed from each plot was split to obtain a 25 g sub-sample, which was then cleaned by hand. The wheat subsamples were used to estimate the percentage of visually scabby kernels (VSK) and then all samples (wheat and barley) were ground and submitted for deoxynivalenol (DON) analysis. The data indicated that resistance was expressed in some of the transformed lines.

ACKNOWLEDGEMENT AND DISCLAIMER

This material is based upon work supported by the U.S. Department of Agriculture, under Agreement No. 59-0790-4-096. This is a cooperative project with the U.S. Wheat & Barley Scab Initiative. Any opinions, findings, conclusions, or recommendations expressed in this publication are those of the authors and do not necessarily reflect the view of the U.S. Department of Agriculture.

MOLECULAR AND GENETIC STUDIES ON FUSARIUM EAR BLIGHT DISEASE OF WHEAT

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ABSTRACT

In the UK, Fusarium Ear Blight (FEB) disease of cereal crops is sporadic and epidemic years are difficult to predict. Wheat ear infections are predominantly caused by two species, *F. graminearum* and *F. culmorum*. The former now predominates in areas where maize is frequently grown in the rotation as well as near major shipping ports. Infection by these species leads to the contamination of grain with mycotoxins. Since 2008, prior to arrival at the flour mill, each lorry load of wheat grain has to be tested for mycotoxin contamination, which is adding to farm costs.

Despite intensive investigation over the past 15 years on the molecular basis underpinning susceptibility and resistance to FEB in wheat, our knowledge on this pathosystem remains fragmentary. However, several key features have so far emerged: When infecting wheat ears, most *Fusarium* isolates produce a range of trichothecene mycotoxins including deoxynivalenol (DON) and its acetyl derivatives. Removal of DON producing ability from toxigenic *Fusarium* isolates causes reduced virulence. In wheat ears, natural resistance is (a) primarily effective post infection, (b) known to be QTL based and (c) *Fusarium* species non-specific. So far the only well characterised natural resistance mechanism is that associated with the major 3BS QTL derived from Sumai-3, which confers upon the plant the ability to convert DON to a DON-O-3 glycoside with reduced toxicity.

In this presentation, three research topics will be addressed. Firstly, we have dissected the hyphal infection process from the initial infected spikelet, through the rachis and into the adjoining spikelets of a susceptible wheat genotype. This has included a comprehensive microscopic study to locate hyphae and to characterise the responses of the neighbouring wheat cells (Brown et al., (2009) submitted). This study is now permitting the recovery of specific cell types by laser capture microscopy and tissue dissection for gene expression analysis by RT-PCR, Affymetrix microarray and 2nd generation sequencing analysis. Secondly, we are using the model *Arabidopsis* floral *Fusarium* - pathosystem (Urban et al., 2002), to identify both the pathogen and host components which either restrict or support the *Fusarium* infection process. Through a reverse genetics approach we have discovered that both NPR1 and EDS11 are independently required for floral resistance against *Fusarium*, whereas the salicylic acid (SA) and ethylene (ET) signalling pathways are either not required or have only a minimal effect on the interaction outcome (Cuzick et al., 2008, 2009). Previously, EDS11 had only been reported to be required for basal defence against virulent bacteria (Volko et al., 1998). The results arising from these *Arabidopsis* mutant analyses are in agreement with the results obtained from the wheat ear microscopic study. Thirdly, we are in the final stages of establishing at Rothamsted a Category 3 biological containment facility that combines controlled growth rooms and laboratories. This purpose built facility, will permit us to use the virus induced gene silencing (VIGS) technology based around the Barley stripe mosaic virus vector in combination with transgenic *Fusarium* strains and/or wild-type *Fusarium* isolates

of non-UK origin (none of which must be allowed to escape into the environment). The VIGS experiments done in this facility will explore the biological relevance of the genes discovered at the wheat host-pathogen interface and in the *Arabidopsis* pathosystem to *Fusarium* infection of wheat ears and leaves in susceptible and resistant genotypes.

ACKNOWLEDGEMENT

Rothamsted Research receives grant-aided support from the Biotechnology and Biological Sciences Research Council (BBSRC) of the UK. This research has also received support from a BBSRC responsive mode grant. AB and NB are supported by BBSRC studentships with Syngenta as the CASE partner.

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IDENTIFICATION OF TRICHOTHECENE TARGETS: NOVEL GENES FOR SCAB RESISTANCE IN BARLEY AND WHEAT

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ABSTRACT

The molecular mechanisms that control trichothecene mycotoxin sensitivity in plants are not well understood. To identify functional eukaryotic targets we screened the yeast non-essential deletion library (~4700 genes) with trichothecin (Tcin) and identified >100 trichothecene targets that enhance resistance when deleted from the genome. For the top resistant yeast mutants, the single gene knock-out conferred resistance to six fold higher concentration than the lethal dose for the isogenic parental strain. The largest group of resistant strains affected mitochondrial function, suggesting a role for fully active mitochondria in trichothecene toxicity. Tcin inhibited mitochondrial translation in the wild type strain to a greater extent than in the most resistant strains, implicating mitochondrial translation as a previously unrecognized site of action. The Tcin-resistant strains were cross-resistant to anisomycin and chloramphenicol, suggesting that Tcin targets the peptidyltransferase center of mitochondrial ribosomes. Tcin induced cell death was partially rescued by mutants that regulate mitochondrial fusion and maintenance of the tubular morphology of mitochondria. Treatment of yeast cells with Tcin led to the fragmentation of the tubular mitochondrial network, supporting a role for Tcin in disruption of mitochondrial membrane morphology. These results provided genome-wide insight into the mode of action of trichothecene mycotoxins and uncovered a critical role for mitochondrial translation and membrane maintenance in their toxicity. Our goal is to use the information from the yeast screen to identify mechanisms that contribute to trichothecene resistance in plants. *Arabidopsis* orthologs of the genes identified in yeast have been cataloged and scored based on both protein sequence homology and functional characterization. The genes represent a broad array of functional gene classes, including factors that influence translation, sterol synthesis, stress response, mitochondrial genome maintenance, mitochondrial morphology, lipid metabolism, ubiquitination, the unfolded protein response (UPR) pathway, mitochondrial ribosome function, and sphingolipid metabolism. Homozyous *Arabidopsis* knockout lines (T-DNA insertions) were identified using the *Arabidopsis* Information Resource (TAIR) database. These plants are currently being tested for response to mycotoxin exposure (DON and Tcin). In addition, the parental *Arabidopsis* strain (Columbia) with GFP-labeled mitochondria, endoplasmic reticulum, and chloroplast are being used to visualize the *in vivo* effect of the mycotoxins on organelle morphology and function.

CHARACTERIZATION OF FUSARIUM HEAD BLIGHT-RESPONSIVE GENES IN DIVERSE WILD AND CULTIVATED BARLEY

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ABSTRACT

Wild (*Hordeum vulgare* subsp. *spontaneum*) and cultivated barley (*Hordeum vulgare*) accessions offer varying degrees of resistance to Fusarium head blight (FHB). Integration of resistance from multiple diverse sources has the potential to extend resistance durability, ultimately helping barley producers control FHB. To identify genetically diverse barley lines carrying FHB resistance, DArT markers were used to genotype 102 wild or cultivated barley lines (80 FHB-resistant and 22 FHB-susceptible). Two major clades were identified: one comprised entirely of resistant, wild barley, the other containing resistant or susceptible, wild and cultivated barley. Multiple wild and cultivated lines, including parents of mapping populations, were selected from across these major clades for haplotype analysis. Previous GeneChip experiments have identified over 100 barley genes with significantly up-regulated transcript levels in response to treatment of *Fusarium graminearum* or DON. Forty-four of these genes, including those implicated in defense responses such as P450s, glutathione-S-transferases, and UDP-glucosyltransferases, are being sequenced from the diverse barley lines and analyzed for haplotype differences. Initial screens suggest the lack of a "golden ticket" haplotype associated with resistance. For example, analysis of a member of the UDP-glucosyltransferase gene class responsible for DON detoxification reveals multiple haplotypes, with no haplotype solely associated with all resistant or all susceptible lines.

UNRAVELING THE TRITICEAE-*FUSARIUM GRAMINEARUM* INTERACTION

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ABSTRACT

Little is known about the wheat/barley-*Fusarium graminearum* interaction and the genes and mechanisms that exhibit host plant resistance/susceptibility. We used RNA profiling to examine gene expression patterns in barley and *F. graminearum* during infection, the differential responses between resistant and susceptible barley and wheat genotypes during infection, and during the accumulation of trichothecenes in barley. Our results revealed a complex interaction between the hosts and *F. graminearum* and provided an opportunity to develop models for the interactions and identify genes that may play a role in resistance/susceptibility. We proposed that barley responds to trichothecene accumulation through two responses: one that increases the susceptibility of barley to infection through the induction of cell death responses, and another that provides increased resistance through induction of genes encoding trichothecene detoxification and transport processes. Recently, we have begun to examine the interaction between barley and the trichothecene deoxynivalenol (DON). Our results showed that DON is transported from the site of inoculation and is converted into DON-3-O-glucoside to reduce toxicity. Using a set of RNA profiling data of barley inoculated with DON, we identified a set of barley UDP-glucosyltransferases that we are functionally characterizing. Overexpression of one of the UDP-glucosyltransferases in *Arabidopsis* increased tolerance to DON.

HOST FACTORS CONTRIBUTING TO RESISTANCE/ SUSCEPTIBILITY TO *FUSARIUM GRAMINEARUM*

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ABSTRACT

Fusarium head blight (FHB)/scab caused by the fungus *Fusarium graminearum* is a destructive disease of wheat and barley. Activation of salicylic acid (SA) signaling enhances resistance to *F. graminearum* in *Arabidopsis*, and constitutive overexpression of the *Arabidopsis NPR1* gene, which is a key regulator of SA signaling, enhances disease resistance in transgenic *Arabidopsis* and wheat (Makandar et al., 2006). In *Arabidopsis*, the *PAD4* and *WRKY18* genes are two other important components of SA signaling. Furthermore, constitutive overexpression of *PAD4* and *WRKY18* enhances resistance against *F. graminearum* in transgenic *Arabidopsis*. To determine if *Arabidopsis* PAD4 and WRKY18 could be utilized to enhance FHB resistance, we have generated transgenic wheat plants which express AtPAD4 from the maize Ubiquitin (Ubi) gene promoter, and have transformed wheat with a Ubi:At*WRKY18* construct to express At*WRKY18*. Silencing factors that contribute to host susceptibility to *F. graminearum* is another approach that we have taken for enhancing FHB resistance. For example, in *Arabidopsis*, a lipoxygenase (LOX) involved in the synthesis of oxidized lipids (oxylipins) was found to contribute to susceptibility to *F. graminearum*. Experiments are underway to transform wheat with RNAi constructs to silence expression of three wheat LOX genes that exhibit homology to the *Arabidopsis* LOX. Progress on these experiments will be presented.

ACKNOWLEDGEMENT AND DISCLAIMER

This material is based upon work supported by the U.S. Department of Agriculture, under Agreement No. 59-0790-067. This is a cooperative project with the U.S. Wheat & Barley Scab Initiative. Any opinions, findings, conclusions, or recommendations expressed in this publication are those of the author(s) and do not necessarily reflect the view of the U.S. Department of Agriculture.

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MAPPING OF MRP GENE AS A CANDIDATE FOR QTL '*QFHS.KIBR-2DS'* TO REDUCE DON ACCUMULATION IN WHEAT GRAINS

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ABSTRACT

The QTL '*Qfhs.kibr-2DS'*, which reduces *Fusarium* DON accumulation in wheat grains was reported on 2DS chromosome of bread wheat cv. Gamenya and Nobeokabouzu-komugi. The gene for multi drug resistance-associated protein (MRP) located on 2DS was highly expressed during FHB spreading, and it could explain the low level of DON accumulation for the QTL. The MRP was tracked down as a candidate gene constituting the *Qfhs.kibr-2DS* (Handa et. al 2008). The wheat BAC clones of Chinese Spring (CS) were screened for the MRP genes on homoeologous chromosome 2A, 2B and 2D, and their genomic sequences were analysed. Based on the ORF sequences, the full length cDNA for Gamenya (*MRP-D.g*) and Sumai 3 (*MRP-D.s*) were isolated from FHB infected spikes by PCR. The specific primer set for 4.7kb *MRP-D.g* was designed to confirm chromosomal location of the cDNA clone by using 118 lines of DH population (Sumai 3×Gamenya). It was mapped on the expected position of *Qfhs. kibr-2DS* on the chromosome 2DS as an MRP allele. Then, seven sets of 2D genome specific primers for the *MRP-D* were designed to examine their allelic variation among wheat germplasms. So far two types of the MRP alleles were identified; Chinese wheat type (ex. Sumai 3 and CS) and the others (ex. Gamenya and Nobeokabouzu-komugi). The *MRP-D.g* isolated from Gamenya which has the QTL to reduce DON accumulation was confirmed to be located on the locus of *Qfhs.kibr-2DS*.

ACKNOWLEDGEMENT

This work was supported by a grant from the Ministry of Agriculture, Forestry and Fisheries of Japan (Genomics for Agricultural Innovation, TRC-1005).

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GENETIC MANIPULATION OF SUSCEPTIBILITY TO *FUSARIUM* HEAD BLIGHT

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ABSTRACT

Site-specific mutation of genes in the model plant *Physcomitrella patens* has identified several mechanisms responsible for susceptibility to *Fusarium graminearum* (the causal agent of Fusarium Head Blight or FHB) and to *Fusarium*-derived toxins, such as DON, DAS and ZON. A major point of control is the programmed cell death (PCD) pathway. Infection with *F. graminearum* or treatment with DON or DAS causes a stereotypical PCD that is associated with the production of reactive oxygen species (ROS), nuclear condensation and DNA fragmentation, and the induction of protease and nuclease enzyme activity and gene expression. Inhibition of PCD through the disruption of genes required for PCD or through the overexpression of anti-PCD genes suppresses sensitivity to toxins and inhibits susceptibility to *Fusarium*. This suggests that PCD is an important target for the pathogen and for pathogen-derived toxins and indicates that inhibition of PCD in the host plant may be a useful strategy for controlling FHB and DON contamination. A second effective approach to reducing infection by *Fusarium* is to induce immunity in the plant. Pre-treatment of *Physcomitrella* or wheat plants with chitosan induces a resistance response that is effective against FHB. The importance and utility of this response has been demonstrated in *Physcomitrella*, where overexpression of individual components of the induced response (particularly nucleases and peroxidases) confers enhanced resistance to FHB infection. Moreover, gene knockouts of the CEBiP chitosan receptor are no longer able to mount an effective induced response, suggesting a key role for this receptor in mediating chitosan-induced immunity. A recent screen for yeast mutants with altered sensitivity to tricothecene has identified several novel cellular targets for this toxin. We are currently creating the corresponding knockouts for these genes in *Physcomitrella* so that their contribution to FHB susceptibility and infection can be assessed *in planta*. Finally, we have recently shown that exposure to toxins, as well as FHB infection, is associated with ER-stress and the Unfolded Protein Response (UPR). Suppression of this stress pathway through genetic or chemical means provides an additional and potentially useful approach to controlling this disease.

ACKNOWLEDGEMENT AND DISCLAIMER

This material is based upon work supported by the U.S. Department of Agriculture, under Agreement No. 59-0790-6-063. This is a cooperative project with the U.S. Wheat & Barley Scab Initiative. Any opinions, findings, conclusions, or recommendations expressed in this publication are those of the author(s) and do not necessarily reflect the view of the U.S. Department of Agriculture.

IDENTIFYING AND CHARACTERIZING BARLEY GENES THAT PROTECT AGAINST TRICHOTHECENES

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ABSTRACT`

Our overall goal is to identify genes that play role in resistance to Fusarium Head Blight (FHB) and to develop and test transgenic wheat carrying these genes. In particular, we are interested in identifying genes that protect barley and wheat from the effects of trichothecenes. Previously, we conducted a large array of RNA profiling experiments during *Fusarium graminearum* infection of barley and inoculation with the trichothecene deoxynivalenol (DON). We identified a set of potential resistance genes that respond to trichothecene accumulation. The potential resistance genes encode a proline-rich like protein, a Bowman-Birk type trypsin inhibitor, a NB-ARC domain containing protein, a cysteine synthase, a NF-X1 zinc finger protein, and UDP-glucosyltransferases. We are using virus-induced gene silencing assays to functionally test these genes for their role in FHB resistance/susceptibility in wheat. The NF-X1 gene functions as a negative regulator of trichothecene-induced defense response in *Arabidopsis*. Wheaton and Bobwhite inoculated with VIGS-NF-X1 constructs exhibited statistically significant reduction in disease severity during the early stages of disease development compared to the empty vector VIGS control lines ($P<0.05$). From our RNA profiling experiments, we identified nine barley UDP-glucosyltransferases and cloned five full-length cDNAs for testing in yeast. We identified a barley UDP-glucosyltransferase gene that exhibits DON resistance based on the yeast assay. As a proof of concept, we generated transgenic *Arabidopsis* over expressing the barley UDP-glucosyltransferase and tested these plants for their ability to grow on media containing DON. After 4 weeks of growth on DON-containing media, the wild-type seedlings were albino and had ceased growing. Shoot and root growth were not inhibited in the UDP-glucosyltransferase overexpression lines grown on media containing 10, 15 and 20 ppm of DON, demonstrating that overexpression of UDP-glucosyltransferase in transgenic *Arabidopsis* protects plants from the deleterious effects of DON. Currently, we are developing transgenic wheat plants upregulating this UDP-glucosyltransferase gene.

EFFORTS TOWARD DISSECTING 2H- FHB QTL WITH TRANSPOSONS IN BARLEY

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ABSTRACT

Breeding of barley varieties resistant to FHB has been given a high priority in many areas where wheat and barley are grown, but it represents a daunting challenge for breeders due to the complex nature of resistance. It is known that the genetic basis of resistance to FHB is quantitatively inherited. Molecular mapping studies in barley indicate that two QTLs on chromosome 2H and 6H have a large effect on low kernel discoloration and could be used for marker-assisted selection for FHB resistance. It has been observed that lines having the QTL on 2H had 40% less head blight than lines that lacked this QTL, therefore warranting its detailed characterization. The maize *Ac/Ds* transposon system is an effective approach for gene identification and cloning in heterologous species. Using this system, single-copy *Ds* insertion lines (TNPs) were generated in barley to identify, tag, and determine genes and their function. Our recent successful demonstration in barley of *Ds* transposition at significant frequencies over multiple generations in addition to the preference of *Ds* to re-insert near the original site of excision and into genic regions facilitates saturation mutagenesis. Plants with single *Ds* insertions (TNPs), mapping near genes of interest, are important vehicles for gene identification through re-activation and transposition of *Ds*. Mapping and bioinformatics analysis of *Ds* flanking sequences indicate that the vast majority of *Ds* insertions (88%) are in genic regions. Our data indicate that "transposon walking", the sequential re-activation of *Ds,* can be used to identify QTLs and members of clustered gene families. We are saturating FHB related QTL regions with maize *Ds* elements to facilitate identification and characterization of genes associated with FHB resistance in the 2H-QTL. *Ds* elements in TNP lines mapped on chromosome 2H were re-activated by crossing with *Ac*TPase-expressing plants. New *Ds* transpositions have been identified by Southern blotting and *Ds* tagged genes are being cloned using inverse PCR. This effort of saturation mutagenesis with *Ds* transposons will lead to a better understanding of FHB resistance and the candidate genes that display this quantitative variation.

ASSOCIATION MAPPING OF QTL FOR FUSARIUM HEAD BLIGHT RESISTANCE IN WHEAT

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ABSTRACT

Wheat Fusarium head blight (FHB) is an important wheat disease worldwide. To identify new quantitative trait loci and validate previously reported QTLs for wheat resistance to FHB spread within a spike (type II), association mapping was conducted using a collection of 149 Asian accessions from China (120), Japan (26) and Korea (3) and 213 US elite breeding lines from three major hard wheat nurseries (SRPN, NPRN, ORPN) and two soft wheat nurseries (UESRWWN and USSRWWN) and elite breeding lines from Oklahoma State University, OK. FHB was evaluated by injecting 1000 conidiospores into a central spikelet of a spike and measuring the proportion of symptomatic spikelets (PSS) in a greenhouse of Kansas State University, Manhattan, KS. In general, Asian accessions had a relatively higher type II resistance than that of U.S. accessions. A total of 261 genome-wide SSR markers including these linked to known QTL for FHB resistance were used to analyze the population. Structure analysis clearly separated the Asian and US accessions into two groups. Separated analysis on each group identified three (Asian group) and four subgroups (US group). Simulation tests selected mixed model and K model for association computation of Asian group and U.S. groups, respectively. Eighteen markers/alleles showed significant association with FHB resistance in Asian population. Three previously reported QTLs on 3BS, 3BSc, and 5AS were validated in Asian population. Four marker alleles for 5AS QTL linked to FHB susceptibility in the Asian group suggested most of Asian accessions in this study may lack the resistance allele on 5A. Marker Xgwm276 on 7A was significant associated with FHB resistance in the Asian group, which has not been reported previously. Twelve accessions (8% in Asian group) with the Xgwm276-110 allele had a mean PSS of 0.14 that is lower than these accessions with Xgwm533-159 allele (PSS= 0.21). In the U.S.A. population, 18 alleles from 17 markers were associated with FHB resistance. Two previously reported QTLs on 3BS (Xgwm493, Xbac102) and 4D (Xbarc98, Xwmc473, Xgwm608) were validated. However, Xgwm493 and Xbarc102 showed FHB susceptible effect. Among all 17 significant markers, two markers Xcfa2263-140 (2A) and Xgwm320 -274 (2D) showed the largest effect on FHB resistance with a mean of PSS of 0.38. Therefore, the QTL on 2A and 2D are likely new QTL for FHB resistance in U.S. accessions. The results not only validated previously reported important QTL, but also discovered some new QTL. Some QTL in US lines may be different from Asian sources. Therefore, association mapping is an effective approach to study FHB resistance in wheat.

ACKNOWLEDGEMENT AND DISCLAIMER

This material is based upon work supported by the U.S. Department of Agriculture. This is a cooperative project with the U.S. Wheat & Barley Scab Initiative. Any opinions, findings, conclusions, or recommendations expressed in this publication are those of the author(s) and do not necessarily reflect the view of the U.S. Department of Agriculture.