ITEMS FROM AUSTRALIA

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Identifying resistance to root-lesion nematodes (Pratylenchus thornei & P. neglectus) in wild relatives of wheat from the genera Triticum and Aegilops.

Common or bread wheat is an allohexaploid comprised of three genetically related genomes (A, B, and D) that originated as a hybrid of emmer wheat (BBA^uA^u) and *Ae. tauschii* (DD). *Pratylenchus thornei* and *P. neglectus* are migratory root-endoparasitic nematodes that feed and reproduce in the cortex of wheat and can reduce yield by up to 50% in intolerant wheat cultivars. Although wheat is their preferred host, they attack a range of crops including chickpea (*Cicer arietinum*), mungbean (*Vigna radiata*), and sorghum (*Sorghum bicolor*). The estimated annual value of wheat production lost in the northern Australian grain region from *P. thornei* and *P. neglectus* is up to \$46 and \$23 x 10^6, respectively.

The first aim of this research was to test the A, BA, and closely related progenitors of wheat to determine if resistance to *P. thornei* is present on these genomes. The second aim was to screen Chinese Spring–*Ae. speltoides* and related *Aegilops* species addition lines for resistance to both root-lesion nematodes (RLN).

To achieve the first aim, 148 wild wheat accessions obtained from Kansas State University via the Australian Winter Cereals Collection in Tamworth were tested for resistance over 2 years to *P. thornei*. This group of wild relatives included *Ae. speltoides* (S genome), *T. urartu* (A^u genome), *T. monococcum* (A^m genome), *T. timopheevii* (GA^u genomes), and *T. turgidum* (BA^u genomes).

Generally, all of the *Ae. speltoides* accessions that were tested were found to be resistant or partially so. Eight accessions (AUS26952, AUS26983, AUS26957, AUS26948, AUS26984, AUS26954, AUS26955, and AUS26951), however, were more resistant (produce lower *P. thornei* multiplication) than the current common wheat resistance standard GS50a in both experiments. None of these accessions were significantly better over both years, but AUS26948, AUS26952, and AUS26983 were significantly ($P < 0.05$) more resistant in Experiment 1.

Of the *T. urartu* accessions, nine (AUS26978, AUS26979, AUS26935, AUS26946, AUS26947, AUS26937, AUS27033, AUS26941, and AUS26932) performed consistently better than GS50a. Although none of the accessions were significantly better than GS50a over both years, AUS26935 was significantly ($P < 0.05$) more resistant in Experiment 1.

Twenty-two accessions of *T. monococcum* subsp. *aegilopoides* and one of *T. monococcum* subsp. *monococcum* were screened during this research. None of the accessions were found to be particularly susceptible in either experiment, but eight accessions (AUS27049, AUS27037, AUS27036, AUS27090, AUS27041, AUS27050, AUS27046, and AUS27091) produced lower *P. thornei* populations than GS50a in both experiments.

AUS27081 was the only *T. timopheevii* subsp. *armeniacum* accession in Experiment 1 to produce fewer *P. thornei* than GS50a. Unfortunately, none of the accessions tested in both Experiments 1 and 2 were able to out-perform GS50a over both years of testing.

Thirty accessions of *T. turgidum* subsp. *carthlicum*, 25 accessions of *T. turgidum* subsp. *dicoccoides* and one accession of *T. turgidum* subsp. *turanicum* were tested for resistance in Experiments 1 and 2. None of the *T. turgidum* subsp. *carthlicum* or *T. turgidum* subsp. *turanicum* accessions were found to be resistant. In fact, the majority of accessions were quite susceptible with only a few producing *P. thornei* populations similar to the resistant durum Yallaroi. The *T. turgidum* subsp. *dicoccoides* accessions also produced a wide range of results from quite susceptible through

to resistant. A number of accessions appeared to be moderately resistant with AUS27025 proving to be as resistant as GS50a over both years of testing.

In all, 148 wild wheat accessions were screened with 134 (91%) of these able to be screened over two years. Of the 134 accessions, 26 (19%) proved to be more resistant than the current best source of resistance, GS50a. Interestingly, 25 (96%) of the 26 elite accessions were from the diploid relatives of wheat.

Because resistant accessions were found among both *T. urartu* and *T. monococcum*, we have confirmed that there are one or more resistance genes on the A genome. A number of resistant accessions were also found among the *Ae. speltoides* accessions. Although *Ae. speltoides* is an S-genome diploid, it is thought to be the B-genome donor of modern bread or common wheat and, therefore, it is reasonable to hypothesize that resistance genes found on the S genome could be introduced into the B genome of domestic wheat. Thompson and Haak (1997) also have identified *P. thornei*-resistant accessions of the D-genome donor to wheat, *Ae. tauschii*. Theoretically resistance genes could be introduced into all three genomes (A, B, and D) of domestic bread wheat and combined to produce a higher level of resistance.

To achieve the second aim, two experiments determined the resistance of seven Chinese Spring–*Ae. speltoides* disomic addition (DA) lines, their parents, and four parental lines of other addition populations to the RLNs *P. thornei* and *P. neglectus*. *Pratylenchus thornei* multiplied more readily than *P. neglectus*, but statistically significant differences between resistant and susceptible checks were observed in both experiments. *Aegilops speltoides* (TA2780; S genome) was significantly more resistant to both RLN than Chinese Spring (TA3008). Resistance to *P. thornei* resistance statistically equal to that of *Ae. speltoides* was observed in TA7694 (DA 6B) and TA7693 (DA 5B). Additionally, TA7693 (5B), TA7690 (2B), and TA7695 (7B) for *P. neglectus*. Additionally, TA7692 (DA 4B), TA7690 (DA 2B), and TA7691 (DA 3B) were significantly more resistant to *P. thornei* than Chinese Spring but more susceptible than *Ae. speltoides*, indicating the presence of minor resistance genes. *Pratylenchus neglectus* resistance statistically equal to *Ae. speltoides* was identified in TA7963 (DA 5B), TA7690 (DA 2B), and TA7695 (DA 7B). *Aegilops searsii* (TA2355, S^s genome) and *Ae. biuncialis* (TA2782, UM genomes) were resistant to both RLN, whereas *Ae. longissima* (TA1910, S^l genome) was resistant to *P. neglectus* and moderately susceptible to *P. thornei*. *Triticum turgidum* subsp. *dicoccoides* (TA106) was susceptible to both species of RLN. Resistance to *P. thornei* has been reported on chromosomes 2B (Schmidt et al. 2005; Thompson et al. 1999; Toktay et al. 2006; Zwart et al. 2005, 2006) and 3B (Schmidt et al. 2005; Toktay et al. 2006), but these *Aegilops* accessions appear to possess several novel resistances for both *P. thornei* and *P. neglectus*, making them valuable for wheat breeding.

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Publications.

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Research interests.

1. Biochemistry and genetic control of factors that cause deterioration of wheat quality prior to harvest (preharvest sprouting and tolerance to preharvest sprouting, grain dormancy, late maturity α-amylase, and black point).
2. Biochemical and genetic control of color and color stability in Asian noodles (grain and flour constituents involved in color of wheat flour and color and color stability in Asian noodles - xanthophylls, flavonoids, polyphenol oxidase, peroxidase, lipoxygenase, and nutritive aspects of cereal xanthophylls - lutein and lutein esters).
3. Durum germ plasm with tolerance to hostile soils and root diseases and better adaptation to southern Australia.

Dormancy in white-grained wheat: mechanisms and genetic control.

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Grain dormancy is a major component of resistance to PHS resistance in red- and white-grained wheat. A QTL on chromosome 4A of both types has been associated with a component of this dormancy that is reflected in sensitivity of the embryo to ABA. Genetic studies involving reciprocal F_1s and doubled haploids suggest that two or more genes are involved in dormancy in white-grained wheat and that at least one is expressed in the seed coat. By analogy, it is tempting to suggest that the seed coat effect in white-grained wheats may be similar to that in red wheat and be controlled by a gene(s) on one of the group-3 chromosomes. A doubled-haploid population involving parents that both contain the 4A QTL but vary in dormancy phenotype was analyzed, and a new QTL was located on chromosome 3B close to the likely position of *R-B1a*. This QTL appeared to be linked to increased expression of genes controlling key enzymes in the flavonoid pathway and a significantly greater accumulation of soluble flavonoids. Interaction between a factor produced by the dormant seed coat and the ABA-sensitive embryo during early imbibition would appear to explain a significant part of dormancy in white-grained wheat and be consistent with the evolution of white wheat.

Pathway for water movement into dormant and nondormant wheat grain.

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The movement of water into harvest-ripe grains of dormant and nondormant genotypes of wheat was investigated using magnetic resonance micro-imaging (MRMI). Neither the rate of increase in water content nor the pattern of water distribution within the grain was significantly different in closely related dormant and nondormant genotypes during the first 18 h. Water entered the grain through the micropyle. By 2 h, water was clearly evident in the micropyle channel, embryo, and scutellum. After 12 h, embryo structures such as the coleoptile and radicle were clearly visible and water had accumulated between the inner and outer layers of the seed coat as well as in the crease. Varying the point of access