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Genetics and genomics of wheat domestication-driven evolution

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ABSTRACT

The cereal crops wheat, rice, maize, and sorghum show conservation of large syntenic blocks in spite of more than 40-fold variation in genome and 20-fold variation in chromosome size. It has been proposed that independent mutations at orthologous loci in traits such as shattering, tough fruiting cases (glumes, in the case of wheat), and threshing may have led to domestication-driven convergent evolution. A different picture is emerging from comparative mapping and cloning of these genes in different cereal crops. It appears that these spike traits are controlled by multiple genetic pathways, and mutations at different loci have been selected during domestication-driven evolution.

Keywords: crop domestication, wheat, tenacious glume, brittle rachis, *Q* gene

INTRODUCTION

Mutations affecting spike traits, namely shattering (also called brittle rachis controlled by genes *Br1* and *Br2*), tough glume (controlled by genes *Tg* and *Sog*), and speltoid spike (*q*, non-free-threshing), were largely responsible for the domestication of wheat. We studied the molecular and comparative genetics of *Tg* and *Br* genes in 2×, 4×, and 6× wheat genotypes and other grasses, which revealed surprising results including the discovery of a new locus *Br2*. We also cloned the *Q* gene, which allowed us to unveil the structural and functional nature of the free-threshing character and other early domestication events. These studies will be briefly reviewed in relation to Vavilov's ideas of homologous series of variation, and the Paterson et al. (1995) hypothesis of convergent evolution by independent mutations at corresponding genetic loci during the domestication of cereal crops.

THE TEMPO OF GENOME EVOLUTION IN GRASSES

The cereal crops wheat, maize, sorghum, and rice and the new small-genome model *Brachypodium* (Draper et al., 2001) share 65 million years of evolutionary history. Although they maintain large syntenic blocks or, in some cases, whole chromosome homoeologies, this has occurred against the background of tremendous changes in the genomic landscapes of these plants. These changes have been brought about by aneuploidy (changes in basic chromosome number), polyploidy (changes in multiples of basic sets of chromosomes), and by the tremendous activity of transposable elements that have contributed to huge changes in genome size. The changes in size and basic chromosome number can

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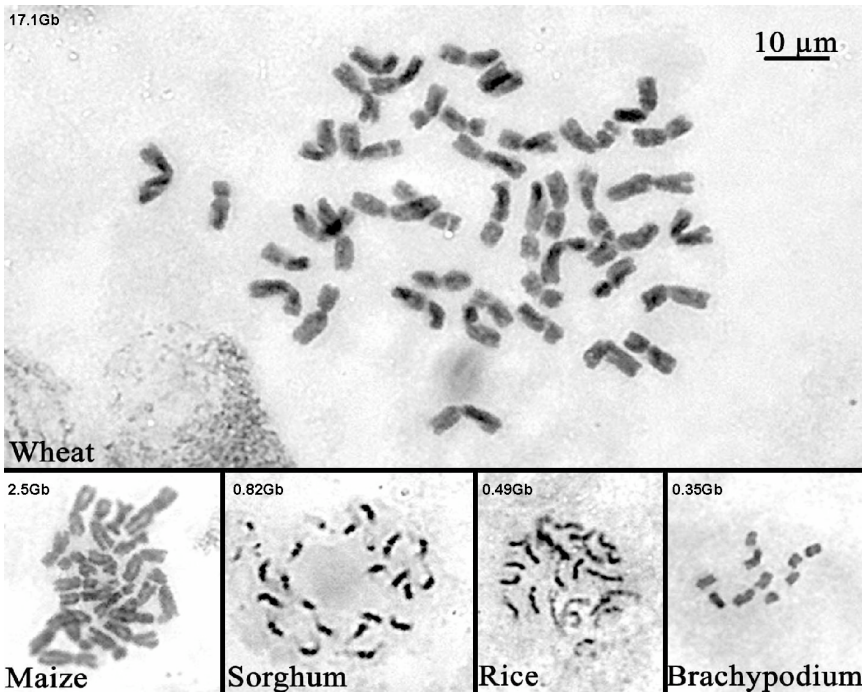

Fig. 1. Comparative chromosome number and morphology of wheat (*Triticum aestivum*, 2n = 6× = 42), maize (*Zea mays*, 2n = 2× = 20), sorghum (*Sorghum bicolor*, 2n = 2× = 20), rice (*Oryza sativa*, 2n = 2× = 24), and the new grass model *Brachypodium distachyon* (2n = 2× = 10). The pictures were taken at the same magnification. The basic chromosome number (1×) varies from 1× = 5, 7, 10, to 12; chromosome size can vary more than 20-fold; and genome size varies by about 50-fold.

be seen in Fig. 1. The genome size varies from 390 Mb in *Brachypodium distachyon* to 17 Gb in wheat. Aneuploidy has brought about changes in basic chromosome numbers: 1× = 5 in *Brachypodium*, 1× = 7 in wheat and the Triticeae, 1× = 10 in maize and sorghum, and 1× = 12 in rice. Paleoploidy may have contributed to chromosome number changes in maize (from 2n = 10 to 20) and more recent polyploidization has produced 2×, 4×, and 6x (1× = 7) wheat species (Fig. 2). Genomic sequencing has uncovered additional segmental genome duplications. Changes in DNA content have been brought about by the activity of transposable elements (discussed in Li et al., 2004). With this picture of genome dynamics as a backdrop, we will briefly discuss the genome mapping and evolution of domestication genes.

Mutations that led to the domestication of different ploidy wheats

The evolutionary relationships among the different ploidy levels in wheat species and their genotypic constitution in relation to domestication genes are shown in Fig. 2. All the wild diploid and wild tetraploid progenitor species have the genotypic constitution of *BrBrTgTgqq*. The domesticated diploid, tetraploid, and hexaploid wheat species and subspecies vary in their genotypic constitution. Independent mutations transformed a brittle spike into a tough spike, which led to the domestication of *Triticum monococcum* subsp. *monococcum*, *T. timopheevii* subsp. *timopheevi*, and *T. turgidum* subsp. *dicoccum*. Because the hexaploid wheat species originated under domestication, they shared these mutations from their respective progenitor species. Mutations to produce soft glumes occurred independently in *T. monococcum* and *T. turgidum*. *Triticum aestivum* inherited the soft glume mutation from *T. turgidum* at the A and B genome loci. Independent mutation at the D-genome *Tg* locus occurred following the origin of *T. aestivum*. We now know that mutation at *q* to produce *Q* occurred only once and is shared between *T. turgidum* and *T. aestivum* (Simons et al., 2006). We are now beginning to understand that independent mutations controlling shattering and tough glume traits may not be at the same locus in different wheat species.

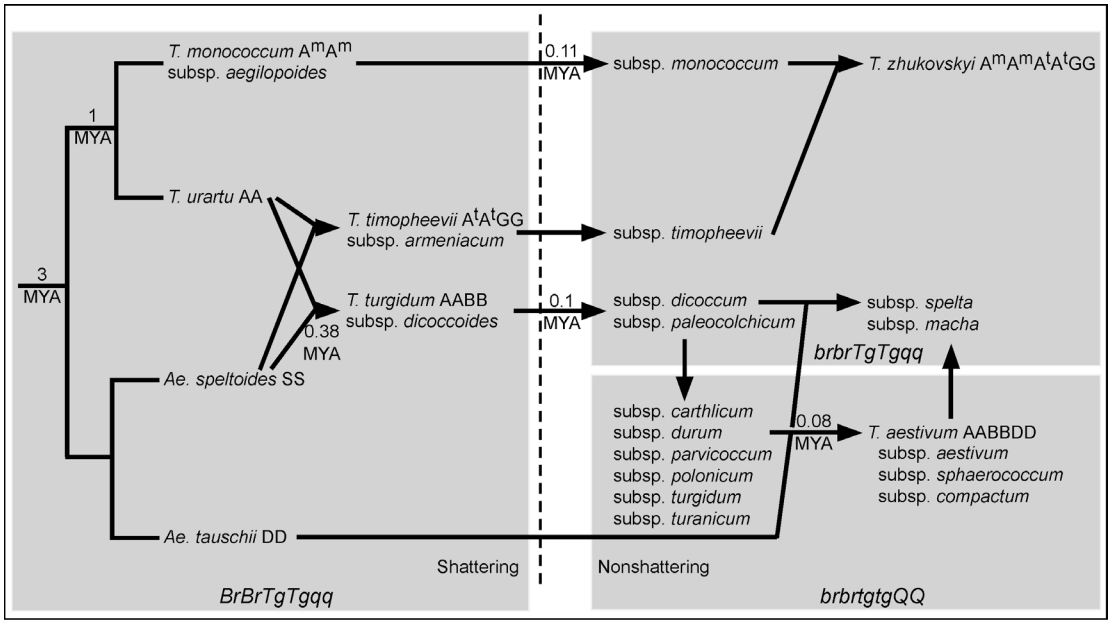

Fig. 2. Wild and domesticated diploid, tetraploid, and hexaploid wheat species, their phylogeny, timeline of evolution, and genotypic constitution with respect to domestication genes for shattering (*Br1*, *Br2* = brittle rachis; *br1*, *br2* = non-brittle rachis), tough glume (*Tg* or *Sog* = tough glume; *tg* or *sog* = soft glume), and threshing (*q* = speltoid and non-free-threshing; *Q* = square-headed and free-threshing). Modified from Li and Gill (2006).

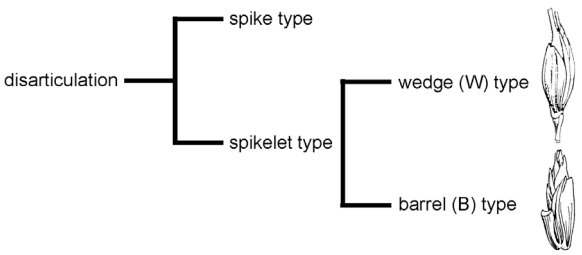

Fig. 3. The spike is a dispersal unit in spike-type disarticulation and the spikelet is the dispersal unit in W-type or B-type disarticulation. The *Br1* gene controls spike- or W-type disarticulation in most *Triticum* and *Aegilops* species; *Br2* controls B-type disarticulation in *Ae. tauschii*. Non-allelic recessive genes on 5A and 7A control W-type disarticulation in *T. monococum*.

COMPARATIVE GENETICS OF TOUGH GLUME

The tough glume gene *Tg* was first mapped on the chromosome arm 2DS in *T. aestivum* (Kerber and Rowland, 1974). Mutations at the same locus occurred in 2A and 2B as well (Simonetti et al., 1999; Taenzler et al., 2002). A soft glume (*Sog*) mutant in *T. monococcum* does not seem to be allelic to *Tg* and occupies a more proximal (towards the centromere) location (Sood et al., 2007). This mutation may be allelic to the spontaneous soft glume trait in *T. monococcum* subsp. *sinskajae*. Therefore it appears that two different genes in the wheat group have been recruited for the soft glume trait.

COMPARATIVE GENETICS OF BRITTLE RACHIS

There is a long history of genetic research on the brittle rachis locus in wheat. Two basic types of disarticulation are found in wheat, spike type and spikelet type (Fig. 3). In spike-type disarticulation, the spike breaks at the base and the whole spike is the dispersal unit. Spikelet-type disarticulation is further subdivided into wedge-type (W-type) and barrel-type (B-type). The W-type is more common, the B-type mainly occurring in *Ae. tauschii* and the D-genome cluster of polyploid species. In *Ae. speltoides*, var. *speltoides* has spike-type and var. *ligustica* has W-type disarticulation. The spike-type and W-type disarticulation are allelic, and the W-type is dominant (reviewed in Li and Gill, 2006). Cultivated *T. turgidum* has a tough spike controlled by recessive alleles *br-A1* and *br-B1*; subsp. *dicoccoides* has wild-type alleles at the *Br1* locus (*Br-A1* and *Br-B1*) that control W-type disarticulation.

T. timopheevii subsp. *armeniacum* has W-type disarticulation. The F_2 of subsp. *timopheevii* (tough spike) and *armeniacum* (W-type disarticulation) gave a 3:1 ratio for W-type vs. tough-spike trait. The W-type disarticulation gene was mapped on 3AS arm (Li and

Fig. 4. A tough-spike spontaneous mutant (left) of *Ae. tauschii* collected in Afghanistan by Bob Metzger was used to map and discover the *Br2* gene that controls B-type disarticulation (see Li and Gill, 2006, for more details).

Gill, 2006) and, hence, subsp. *armeniacum* has *Br-A1* derived from *T. urartu* (W-type). From these genetic data, we infer that both *T. timopheevii* subsp. *timopheevii* and *armeniacum* have *br-G1* on 3G, derived from *Ae. speltoides* var. *speltoides* with spike-type disarticulation. These data also demonstrate that *Ae. speltoides* var. *speltoides* contributed the G genome to *T. timopheevii,* whereas var. *ligustica* contributed the B genome to *T. turgidum* (Li and Gill, 2006).

A W-type gene was mapped to the 3DS arm in a hexaploid (*T. aestivum*), Tibetan semi-wild wheat (SWW) (Chen et al., 1998; Watanabe et al., 2002). F_1 plants from crosses of SWW with Chinese Spring displayed mostly W-type or, rarely, B-type disarticulation (Tsunewaki et al., 1990).

A spontaneous tough-spike mutant of *Ae. tauschii*, collected by Bob Metzger in Afghanistan (Fig. 4), enabled the genetic mapping of the B-type locus of *Ae. tauschii*. This mutant was recessive and gave a 3:1 ratio in crosses with a B-type *Ae. tauschii* accession. Li and Gill (2006) hypothesized that this mutant was allelic to *Br-D1* on 3DS. However, none of the *Br1* region markers showed tight linkage with the B-type allele of *Ae. tauschii*. Instead, the mutant was mapped to the long arm of 3D and given the designation *Br2*. A high-resolution map of *Br2* was constructed and compared to shattering QTLs in rice and maize (Fig. 5). No loci corresponding to *Br1* or *Br2* were detected in rice or maize. Moreover, the rice shattering QTL was different from the maize shattering QTL (Li and Gill, 2006).

In *T. monococcum*, W-type disarticulation is controlled by two recessive genes (Sharma and Waines, 1980). These genes have been mapped on chromosomes 5A and 7A (Rao and Dhaliwal HS, unpublished results).

WHAT HAVE WE LEARNED FROM CLONING THE *Q* GENE?

Although mutations at the shattering and tough glume loci led to the domestication of a wheat that produced a decent harvest, it still had a wild-type speltoid spike and plant habit, and the seed was hulled. The master switch was the mutation at *q* to *Q*, which produced the modern robust plant habit with square-headed and free-threshing spike (Fig. 6). For this reason, *Q* has been referred to as a super-gene, and a 100-year-long history of research on this gene was reviewed by Faris et al. (2005). The mutation from *q* to *Q* occurred only once and, most likely, in a plant similar to the tetraploid wheat *T. turgidum* subsp. *dicoccum*, which has a tough spike and a soft glume phenotype with a speltoid spike (*br1br1tgtgqq*).

The *Q* gene belongs to the AP2 family of transcription factors, which have been implicated in flower and seed development in *Arabidopsis* (reviewed in Riechmann and Meyerowitz, 1998). In wheat, this gene has been recruited for domestication. The *q* and *Q* alleles differ by a single amino acid at position 329; an isoleucine in *Q* promotes homodimer formation that presumably binds more efficiently to the promoter region of the *Q* gene and may lead to up-regulation of transcription in developing spikes and other plant tissues. This may account for the pleiotropic effect of the *Q* gene on many domestication-related traits, including glume shape and tenacity, rachis fragility, spike length, plant height, tiller number, seed size, and spike emergence time (Fig. 6) (Simons et al., 2006). Although the *Q* gene located in 5AL has the major effect, *q* homologs on 5B and 5D also are transcribed and contribute to the *Q* syndrome (Zhang et al., 2006, and unpublished results).

Evidence exists for an interaction between *Q* and other genes affecting spike morphology. Normally *Tg* is epistatic to *Q* and genotypes with the *TgTgQQ* allelic constitution are non-free-threshing. All synthetic hexaploids from crosses between naturally occurring tetraploid wheats (*Q* or *q*) and *Ae. tauschii* (*Tg*) are non-free-threshing, except for those containing *T. turgidum* subsp. *carthlicum* as the source of the A and B genomes. The free-threshing nature of *T. turgidum* subsp. *carthlicum*-derived synthetic hexaploids is presumed to be

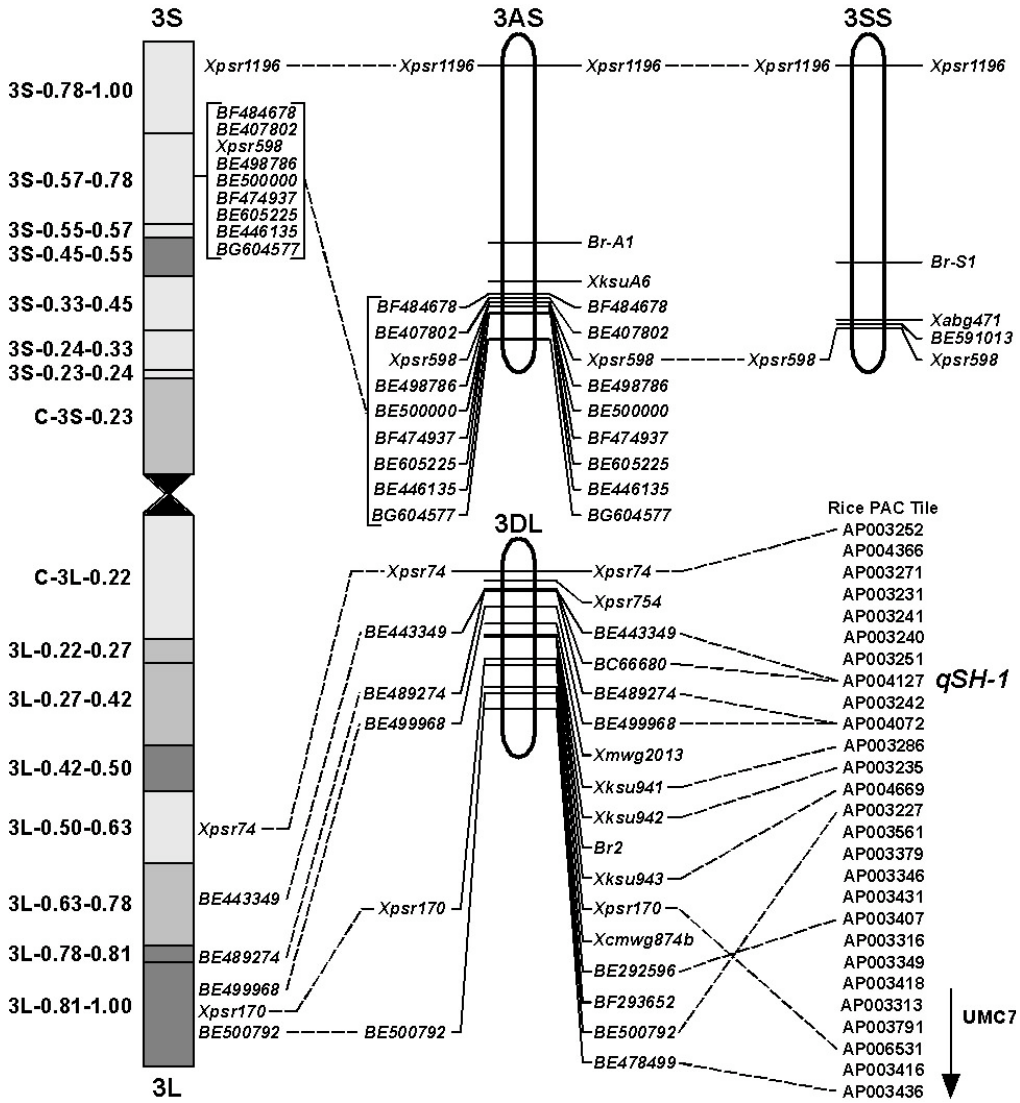

Fig. 5. Comparative mapping of *Br1* and *Br2* genes in wheat, rice, and maize (adapted from Li and Gill, 2006). The deletion bin-based consensus physical map of group-3 chromosomes is shown on the left. Based on tightly linked molecular markers, *Br1* was allocated to bin 3S-0.57–0.78 and *Br2* to bin 3L-0.81–1.00. The genetic maps of 3AS of *T. timopheevii*, 3SS of *Ae. speltoides*, and 3DL of *Ae. tauschii* were anchored to the deletion bin map. The 3DL markers were anchored to the rice PAC tile physical map. The maize shattering QTL was anchored to the rice physical map using the tightly linked probe UMC71/117. The data show that all shattering genes are non-allelic except *Br1-A1* and *Br-S1*.

due to the presence of *Q* (Kerber and Rowland, 1974). Therefore, it appears that *Q* and *Tg* may interact in a different fashion in different genetic backgrounds.

MULTIPLE GENETIC PATHWAYS FOR SEED SHATTERING IN GRASSES

The A-, B-, and D-genome diploid species (see Fig. 2) diverged from a common ancestor ca. 3 million years ago (MYA) (Huang et al., 2002). *T. monococcum* and *T. urartu* diverged from a common ancestor ca. 1 MYA. All four closely related species either seem to have different types of disarticulation or, even if it is the same type, seem to be controlled by different sets of genes. *Ae. speltoides* has spike- and W-type, and *T. urartu* has W-type disarticulation that are controlled by orthologous *Br1* genes on homoeologous group-3 short arms. In *T. monococcum*, W-type disarticulation is controlled by two recessive genes on 5A and 7A. In *Ae. tauschii*, B-type disarticulation is controlled by *Br2* on 3DL. However, *Ae. tauschii* may have genes orthologous to *Br1* because *Br-D1* was mapped on 3DS in SWW.

Fig. 6. Comparison of the *Q* gene and an EMS-induced *Q*-knockout mutant phenotype in the hexaploid wheat cultivar Bobwhite. In each of the panels, the *Q* phenotype is to the right and a *Q*-knockout mutant is to the left. The figures demonstrate that the *Q* gene influences plant height and spike emergence time (A), spike length and shape (B), glume shape and tenacity (C), seed size, shape, and color (D), and rachis disarticulation and seed threshability (E).

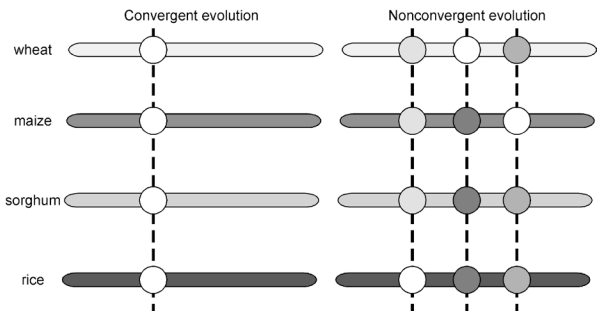

Fig. 7. The model of convergent evolution (left) postulates that mutations at corresponding genetic loci led to the domestication of cereals (after Paterson et al., 1995). The model of non-convergent evolution (right) proposes that multiple genetic pathways and mutations at a number of independent genes contributed to the domestication of cereals (after Li and Gill, 2006).

Glume softness is controlled by different genes (*Sog* or *Tg*) in *T. monococcum* and polyploid wheat species. The *Q* gene phenotype is not observed in other grass crops. These data indicate that multiple genetic pathways control seed shattering and other domestication traits (such as glume toughness and threshability) in grasses. Therefore, similar traits, such as non-shattering fruiting bodies, may superficially show the homologous series of variation postulated by Vavilov (1951) and are controlled by corresponding homoeoloci, as supported by Paterson et al. (1995). However, molecular cloning and fine mapping are now indicating that non-orthologous loci control the same trait in different species and grass crops (Li and Gill, 2006). In defense of Vavilov, unlike Paterson et al. (1995), he was perhaps thinking of genes with similar functions.

The hypotheses of convergent and non-convergent evolution of domestication are illustrated in Fig. 7. In the convergent model, mutations for non-shattering fruting bodies at orthologous loci led to domestication of cereals. In the non-convergent model, the shattering pathway may be shared among grasses, but mutations at different loci were selected in different species for non-shattering fruiting bodies, indicating that mutations in different genes can give rise to traits with similar function. Similar models apply for toughness and threshability of fruiting bodies and would explain why different genes have been selected for the same trait in different species. In fact, chance mutations at the same locus occurring in independent lineages and selected for during domestication is improbable. The spontaneous mutation for tough spike, which was collected in a wild population of *Ae. tauschii* in Afghanistan, turned out to be a different gene (*Br2*) than the more common *Br1*.

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REFERENCES

Chen, Q.-F., Yen, C., Yang, L. 1998. Chromosome location of the gene for brittle rachis in the Tibetan weedrace of common wheat. Genet. Resour. Crop Evol. 45: 407–410.

Draper, J., Mur, L.A.J., Jenkins, G., Ghosh-Biswas, G.C., Bablak, P., Hasterok, R., Routledge, A.P.M. 2001. *Brachypodium distachyon*, a new model system for functional genomics in grasses. Plant Physiol. 127: 1539–1555.

Faris, J.D., Simons, K.J., Zhang, Z., Gill, B.S. 2005. The wheat super domestication gene *Q*. Wheat Inf. Serv. 100: 129–148 (Memorial Issue: Frontiers of Wheat Biosciences).

Huang, S., Sirikhachornkit, A., Su, X., Faris, J., Gill, B.S., Haselkorn, R., Gornicki, P. 2002. Genes encoding plastid acetyl-CoA carboxylase and 3-phosphoglycerate kinase of the *Triticum/Aegilops* complex and the evolutionary history of polyploid wheat. Proc. Natl. Acad. Sci. USA 99: 8133–8138.

Kerber, E.R., Rowland, G.G. 1974. Origin of free-threshing character in hexaploid wheat. Can. J. Genet. Cytol. 16: 145–154.

Li, W., Gill, B.S. 2006. Multiple genetic pathways for seed shattering in the grasses. Funct. Integ. Genomics 6: 300–309.

Li, W., Zhang, P., Fellers, J.P., Friebe, B., Gill, B.S. 2004. Sequence composition, organization, and evolution of the core Triticeae genome. Plant J. 40: 500–511.

Paterson, A.H., Lin, Y.R., Li, Z., Schertz, K.F., Doebley, J.F., et al. 1995. Convergent domestication of cereal crops by independent mutations at corresponding genetic loci. Science 269: 1714–1718.

Riechmann, J.L., Meyerowitz, E.M. 1998. The AP2/EREBP family of plant transcription factors. Biol. Chem. 379: 633–646.

Sharma, H.C., Waines, J.G. 1980. Inheritance of tough rachis in crosses of *Triticum monococcum* and *T. boeoticum*. J. Hered. 71(2): 214–216.

Simonetti, M.C., Bellomo, M.P., Laghetti, G., Perrino, P., Simeone, R., Blanco, A. 1999. Quantitative trait loci influencing free-threshing habit in tetraploid wheats. Genet. Res. Crop Evol. 46: 267–271.

Simons, K.J., Fellers, J.P., Trick, H.N., Zhang, Z., Tai, Y-S., Gill, B.S., Faris, J.D. 2006. Molecular characterization of the major wheat domestication gene Q. Genetics 172: 547–555.

Sood, S., Kuraparthy, V., Bai, G., Dhaliwal, H.S., Gill, B.S. 2007. Molecular mapping of soft glume (Sog) gene in diploid wheat. PAG XV Abstracts, P282, p. 172.

Taenzler, B., Esposti, R.F., Voccino, P., Brandolini, A., Effgen, S., Heun, M., Schafer-Pregl, R., Borghi, B., Salamini, F. 2002. Molecular linkage map of einkorn wheat: mapping storage-protein and soft-glume genes and bread making quality QTLs. Genet. Res. Camb. 80: 131–143.

Tsunewaki, K., Yamada, S., Mori, N. 1990. Genetical studies on a Tibetan semi-wild wheat, *Triticum aestivum* subsp. tibetanum. Jpn. J. Genet. 65: 353–365.

Vavilov, N.I. 1951. The origin, variation, immunity and breeding of cultivated plants. Chronica Botanica 13(1/6):1–366, Ronald Press, New York.

Watanabe, N., Sugiyama, K., Yamagishi, Y., Sakata, Y. 2002. Comparative telosomic mapping of homoeologous genes for brittle rachis in tetraploid and hexaploid wheats. Hereditas 137: 180–185.

Zhang, Z., Tai, Y-S., Simons, K.J., Fellers, J.P., Gill, B.S., Faris, J.D. 2006. Functional analysis of the domestication gene Q in wheat. PAG XIV Abstracts, P828, p. 307.