Stripe Rust Resistance in the Wheat Cultivar Jagger is Due to *Yr17* and a Novel Resistance Gene

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ABSTRACT

Yellow rust, also known as stripe rust, is caused by *Puccinia striiformis* Westend. f. sp. *tritici* Eriks (PST) and is one of the most common and persistent wheat (*Triticum aestivum* L.) diseases worldwide. A mapping population of recombinant inbred lines from the cross of 'Jagger' (moderately resistant) × '2174' (moderately susceptible) was tested at three sites in Washington where predominant races PST-114 and PST-116 naturally occurred, at Rossville, KS, where PST-100 was inoculated, and in Beijing, China, where a predominant Chinese stripe rust race CYR32 was inoculated on adult plants. A major quantitative trait locus for adult-plant stripe rust resistance was located on the short arm of chromosome 2A (*QYr.osu-2A*), where Jagger was found to carry markers for resistance gene *Yr17* from *Aegilops ventricosa* Tausch (syn. *Triticum ventricosum*). Therefore, *Yr17* is likely the resistance gene on chromosome 2A in Jagger. Markers for *Yr17* were found to occur frequently in cultivars from the southern Great Plains but only occasionally in cultivars from other U.S. wheat regions. A novel resistance gene was mapped on the long arm of chromosome 5A (*QYr.osu-5A*), for which the Jagger allele showed consistent resistance to multiple races of the stripe rust pathogen. A significant genetic effect of the resistance gene *Lr34/Yr18* from 2174 was detected only when the population was tested with CYR32 in China.

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Abbreviations: ARS, Agricultural Research Service; AUDPC, area under the disease progress curve; CAU, China Agricultural University; CIM, composite interval mapping; DS, disease severity; HRW, hard red winter; IT, infection type; LOD, logarithm of the odds; PCR, polymerase chain reaction; PST, *Puccinia striiformis* Westend. f. sp. *tritici* Eriks; QTL, quantitative trait loci; rAUDPC, relative area under the disease progress curve; RIL, recombinant inbred line; SSR, simple sequence repeat; WSU, Washington State University.

Global wheat (*Triticum aestivum* L.) production in 2009 was approximately 659.3 million t, compared with maize (*Zea mays* L.) (796.3 million t) and rice (*Oryza sativa* L.) (433.5 million t) (USDA-FAS, 2009). Several fungal diseases impact global wheat production, but in the past decade yellow rust (also known as stripe rust) caused by the obligate parasite *Puccinia striiformis* Westend. f. sp. *tritici* Eriks (PST) has caused large yield losses (Fu et al., 2009). Although fungicides are available to control this disease, exploitation of natural resistance genes in adapted cultivars has been the most economical and widely used approach (Fu et al., 2009; Krattinger et al., 2009).

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More than 70 genetic loci or quantitative trait loci (QTL) have been officially or provisionally designated as conferring resistance to stripe rust in cultivated wheat or its wild relatives (Chen, 2005; Carter et al., 2009). Many of these resistance genes have minor effects and are effective primarily in adult plants but are active against multiple races of the pathogen. Other genes have large effects and are effective in seedlings and throughout the plant life cycle but are race specific (Kolmer et al., 2009).

Since the gene pool of hexaploid common wheat (*Triticum aestivum* L.; $2n = 6x = 42$; AABBDD) is believed to have been heavily exploited, many efforts have been made to introduce resistance genes into hexaploid wheat from diploid and tetraploid progenitors or from other relatives of common wheat (Lin et al., 2009). For example, several resistance genes on a single fragment of chromosome 2N of *Aegilops ventricosa* Tausch (syn. *Triticum ventricosum*) ($2n = 4x = 28$; DDNN) were translocated onto the short arm of chromosome 2A of wheat line VPM1 (Maia, 1967; Bariana and McIntosh, 1993, 1994; Robert et al., 1999; Seah et al., 2000; Helguera et al., 2003). This chromosomal fragment contains a gene cluster including *Yr17* for stripe rust, as well as *Lr37* for leaf rust (*Puccinia triticina* Eriksss.) and *Sr38* for stem rust (*Puccinia graminis* Pers. f. sp. *tritici* Eriks. & E. Henn) and has been widely deployed in wheat breeding programs around the world. The epidemiological consequences of deployment of the two types (major and minor) of resistance genes, alone and in combination, are significant. The selection pressure induced by deployment of major race-specific genes has caused shifts in virulence patterns of *P. striiformis* according to the gene-for-gene theory of Flor (1971). Therefore, wheat breeders prefer to deploy genes of minor effect in combination with each other or in combination with race-specific genes to ensure durability of the resistance in the host plant (Kolmer et al., 2009). This strategy is difficult to achieve using phenotypic selection alone because the presence of an effective race-specific gene masks the presence or absence of genes of minor effect, but gene pyramids can be developed by selection using closely linked molecular markers (Lin and Chen, 2008, 2009).

Starting in 2000, when stripe rust began occurring regularly in the central and southern Great Plains region of the United States, the hard red winter (HRW) wheat cultivar Jagger (PI 593688) (Sears et al., 1997) and its relatives exhibited very high resistance to stripe rust across many environments. Jagger was used as a parent in many breeding programs in the Great Plains and in other parts of the world. This is evidenced by the continued preponderance of Jagger progeny grown commercially in the southern and central Great Plains of the United States. Jagger or its offspring accounted for, at minimum, 41% of the Kansas wheat acreage in 2011 (USDA-NASS, 2011) and 39% of the Oklahoma wheat acreage in 2010 (USDA-NASS, 2010). From those reports, the cultivar Jagger, at its peak, accounted for 40% of the Kansas wheat acreage from 2002 to 2004 and 38% of the Oklahoma wheat acreage from 2006 to 2008. Unfortunately, the genetic basis of stripe rust resistance in Jagger and its progeny remains unknown.

Because of its value to producers and breeders, Jagger was crossed with the HRW wheat cultivar 2174 (PI 602595) to create a recombinant inbred line (RIL) population highly suited to map genes conferring adaptation and disease resistance (Chen et al., 2009a, b, 2010b; Cao et al., 2010). As indicated by its pedigree of IL71-5662/PL145 (= Newton sib)//2165, 2174 provided a genetically divergent source of resistance to foliar diseases with wide adaptation to the southern Great Plains. Jagger had some of the highest resistance to stripe rust among HRW wheat cultivars while 2174 was moderately susceptible. This mapping population was included in the WheatCAP applied genomics project, which was partially funded by USDA-Cooperative State Research, Education, and Extension Service (CSREES) (USDA-CSREES, 2011). This present study was initiated in 2008 with the objective of determining the genetic architecture (number and location) of loci for stripe rust resistance in the Jagger × 2174 RIL population and identifying molecular markers linked to the resistance phenotype that could be used for selection. At the time we conducted this study our hypothesis was that the resistance was due to multiple genes of minor effect and that most of those would have been previously unreported in Jagger.

MATERIALS AND METHODS

Mapping Population and Resistance Evaluation

The two HRW wheat cultivars Jagger (pedigree KS84W485/'Stephens') and 2174 (pedigree IL71-5662/PL145 (= Newton sib)//2165) were crossed and progeny advanced through single seed descent to generate an F_5–derived RIL population with 282 total RIL progeny. All test environments in this study were planted with seed from the same set of RILs. The population has been deposited in the National Plant Germplasm System (Beltsville, MD).

Seedling reactions of the first 96 of the 282 RILs and two parents were evaluated in the greenhouse at Pullman, WA, using previously described methods (Chen and Line, 1992). Races PST-17, PST-37, PST-43, PST-45, PST-100, PST-114, PST-116, and PST-127 were used, which collectively cover all virulences identified so far in the United States (Chen, 2005; Chen et al., 2010a). PST-100 has been the most predominant race in the United States since 2005 and PST-127 is the most widely virulent race with virulence to 17 of the 20 differential genotypes (Chen et al., 2010a). As the seedling tests were qualitative, the test for each race was conducted once. The spring wheat genotype 'Lemhi' (CItr 11415) was included in each race test as a susceptible check.

About 10 seeds of each line were planted in plastic pots (5 by 5 by 5 cm) filled with a potting mixture (6 L peat moss, 2 L perlite, 3 L sand, 3 L potting soil mix, 4 L vermiculite with

lime, 0.35 L Osmocote (Scotts Miracle-Gro, Marysville, OH), and 2 L water). Inoculation was conducted at the two-leaf stage as described by Chen and Line (1992). Inoculated seedlings were kept in a dew chamber at 10°C for about 24 h without light and then grown in a growth chamber operating at 16 h light and 8 h dark with diurnal temperatures gradually changing from 4°C at 0200 h to 20°C at 1400 h. Infection type (IT) data were recorded 18 to 21 d after inoculation based on the zero-to-nine scale as described by Line and Qayoum (1991).

The population was evaluated in the field at four sites over 3 yr in the United States and China. The first 96 RILs were evaluated for resistance to stripe rust under natural infection at the Washington State University (WSU) Spillman Farm at Pullman, WA (2008 and 2009), and at the WSU-USDA-Agricultural Research Service (ARS) Plant Introduction Farm at Central Ferry, WA (2009). At both locations, the single-row plots were 1 m long and spaced 0.5 m apart. Three resistant cultivars (Madsen [PI 511673], Stephens [CItr 17596], and Coda [PI594372]) and two susceptible genotypes (Moro [CItr 113740] and the WA breeding line WA7821) were included as controls. Two replications were planted at each location and year. The plots were surrounded with seven rows of the susceptible line, WA7821, to act as spreader rows for the stripe rust pathogen.

The 96 RILs and their two parental lines were evaluated in 2009 and 2010 at the China Agricultural University (CAU) Farm in Beijing, China. Plots were arranged in the field in a completely randomized design with two replications for the RILs and six replications for parents. Each line was planted in two rows. The plants were inoculated with race CYR32 at the flag leaf stage (Zadoks stage 37) (Zadoks et al., 1974).

Those 96 RILs plus an additional 58 RILs from the population were tested in 2010 at the Kansas State University experimental station at Rossville, KS. The parents, two resistant cultivars, Fuller (PVP 200800130) and Wesley (PI 605742), and three susceptible cultivars or lines, KS89180B, McNair 701 (CItr 15288), and Trego (PI 61257), were used as controls. The experimental design was a randomized complete block with three replications. Single row plots were approximately 1.5 m long and 30 cm apart. Every third drill pass (1.5 m wide) was planted with the highly susceptible breeding line KS89180B to serve as spreader rows. Plants of the RILs were inoculated with stripe rust race PST-100 by first transplanting previously inoculated plants of the susceptible cultivar Morocco (CItr 6878) in the spreader rows of KS89180B in mid April followed by inoculation of KS89180B plants with an oil (Soltrol 170, Chevron Phillips Chemical Company LP, The Woodlands, TX) suspension of urediniospores using an ultralow volume sprayer weekly from early jointing to early boot stage (Zadoks 31–41) (Zadoks et al., 1974).

Data collection started when plots of the susceptible checks showed an infection severity of 10% and continued on a weekly interval until initiation of senescence in all tests. In Pullman, WA, in 2008, the stripe rust epidemic occurred late in the growing season due to dry weather. Ratings were conducted on 2, 9, 15, and 22 July, well after heading (Zadoks 50) (Zadoks et al., 1974), which occurred during the second week of June. In Pullman, WA, in 2009, heading occurred during the second week of June but the stripe rust epidemic began earlier and ratings were conducted on 19, 24, and 30 June and 7 and 14 July. In Central Ferry, WA, in 2009, rating began after heading when plant development ranged from Zadoks 55 to 70. Reaction to stripe rust was evaluated five times (28 May and 4, 6, 19, and 26 June). At CAU in 2009, the disease spread very quickly, and the phenotype was scored on 29 May and 2 and 7 June for replicate 1 and 26 May and 2 and 7 June for replicate 2. At CAU in 2010, the phenotype was scored only once, on 6 June, for both of two replicates at 25 d after heading. At Rossville, KS, in 2010, the phenotype was scored only once from 21 to 22 May at the early to medium milk stage (Zadoks 73–75).

Stripe rust symptoms were recorded as IT based on a zero-to-nine scale in Washington and Kansas (Line and Qayoum, 1991) and on a zero-to-four scale in China. In all locations, disease severity (DS) was scored as a percentage (0–100) of the upper leaf canopy infected. For all environments with multiple readings, the DS data were used to calculate area under the disease progress curve (AUDPC) as [(severity2 + severity1)/2] × (time2 − time1) summed over all time intervals (Chen and Line, 1995). The AUDPC values were reported on a relative (rAUDPC) basis by dividing all values within an environment by the highest AUDPC value in that environment (Chen and Line, 1995). The mean rAUDPC values, averaged over replications within environments, were used to determine allelic effects in the QTL analysis. The DS data obtained at Rossville and CAU in 2010 were not calculated as rAUDPC since the phenotype was scored only once.

Development of Additional Polymerase Chain Reaction Markers for the VPM1 Translocation

The VPM1 segment was translocated into wheat from chromosome 2N of *A. ventricosa* and carries several important resistance genes including *Sr38*, *Yr17*, and *Lr37* (Maia, 1967; Bariana and McIntosh, 1993; Robert et al., 1999; Seah et al., 2000; Helguera et al., 2003). We developed additional polymerase chain reaction (PCR) markers for this segment to more completely analyze recombinants in the Jagger × 2174 population. Typically, the VPM1 segment has been identified using the forward primer VENTRIUP with the reverse primer LN2 to amplify a PCR product specifically from chromosome 2N. Another forward primer URIC, combined with LN2, amplifies a product from chromosome 2A or 2N (Helguera et al., 2003). Originally a restriction fragment length polymorphic marker that was mapped on chromosome $2A^m$ of diploid wheat *T. monococcum* L. (Dubcovsky et al., 1996), *cMWG682*, was converted to a PCR marker to better distinguish between the VPM1 segment and the collinear region of chromosome 2A in hexaploid wheat. In this study, VENTRIUP, LN2, and URIC were used to isolate *cMWG682* simultaneously from either chromosome 2N or chromosome 2A in a one-shot PCR reaction. Polymerase chain reaction was performed in Quickload Master Mix (New England BioLabs, Ipswich, MA) under the following conditions: denature at 94°C for 5 min, amplification for 35 cycles (94°C for 30 s, 65°C for 30 s, and 72°C for 50 s per cycle), and final extension at 72°C for 10 min.

An *A. ventricosa* resistance gene analog, *Vrga1*, belongs to a nucleotide binding-leucine rich repeat gene family of five members derived from the VPM1 segment (Spielmeyer et al., 1998; Seah et al., 2000). This gene (AF158634) was colocalized with the rust resistance genes *Sr38*, *Yr17*, and *Lr37* present on the 2NS

chromosomal segment of *A. ventricosa* (Seah et al., 2000). A pair of primers (VRGA-F9, 5′-GATGCCGATGTCCCTGGT-3′, and VRGA-R3, 5′-AGTAAGAACGAAAGAAACAGGAGTGCT-3′) was designed to amplify *Vrga1* from Jagger and 2174. Several copies of the *Vrga1* gene were obtained, and a new pair of primers VRGA-F11 (5′-AATCCAAAGGTCAGCAATCC-3′) and VRGA-R5 (5′-GGAATCCAGGTCCTTGAGGAAC-3′) was used to detect polymorphisms of PCR products from the two parental lines. One copy of the *Vrga1* gene was mapped in the Jagger × 2174 population of 96 RILs. Since no crossover was detected in the targeted region, the remaining 186 RILs were genotyped to find recombinant events on chromosome 2A. Polymerase chain reaction was performed with a Taq polymerase, denatured at 94°C for 5 min and amplification for 40 cycles (94°C for 30 s, 55°C for 30 s, and 72°C for 30 s per cycle) with a final extension at 72°C for 10 min.

Genetic Mapping of Stripe Rust Resistance

A genetic map was constructed from a set of 310 simple sequence repeat (SSR) markers previously used to map a major QTL for powdery mildew reaction associated with *Pm3a* in the Jagger × 2174 RIL population (Chen et al., 2009b). Fifty SSR markers were added in this study to the previous linkage maps to increase map density. Genetic linkage groups were first constructed using MapMaker 3.0 (Lincoln et al., 1993). Centimorgan values were calculated based on the Kosambi mapping function. WinQTLCart 2.5 (Wang et al., 2006) was then used to detect QTL using composite interval mapping (CIM). The CIM model was conducted with a 10-cM window size and 0.5-cM walk speed. A QTL was claimed when the logarithm of the odds (LOD) score for a QTL exceeded the threshold of 2.5. The mean rAUDPC or DS value for each line from different environments was analyzed to determine significant effects of single markers on resistance using one-way ANOVA.

Twenty pairs of parental lines representing U.S. germplasm that were used to construct mapping populations in the WheatCAP applied genomics project (Chao et al., 2007), plus an additional 30 cultivars released in the central and southern Great Plains since 1994, were genotyped using the molecular marker *TaVrga-A1*. These cultivars were chosen due to their diversity in genetic background or known pedigree relationship to Jagger.

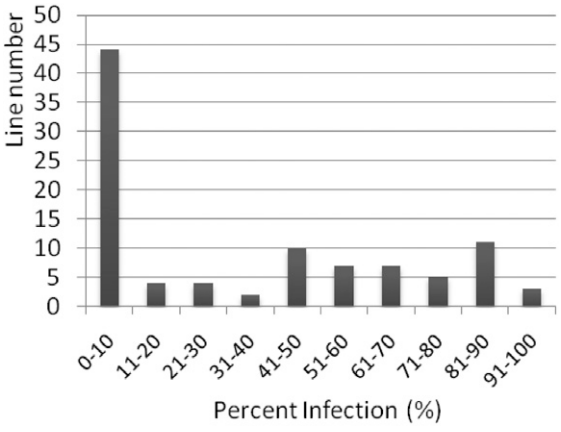

Figure 1. Frequency distribution of the 96 recombinant inbred lines from the Jagger × 2174 population for percent infection score averaged across environments.

RESULTS AND DISCUSSION

At the seedling stage under controlled greenhouse conditions, both Jagger and 2174 were moderately susceptible or susceptible (IT = 7 or 8) to races PST-17, PST-37, PST-45, PST-100, and PST-116 and Jagger was susceptible to PST-114 and PST-127 (2174 was not tested). The entire population was susceptible to PST-100 and PST-127 and moderately susceptible or susceptible to PST-43, PST-45, and PST-114 (data not shown), indicating that little or no seedling resistance was found to segregate in the Jagger × 2174 population in response to these stripe rust races.

Segregation of Stripe Rust Resistance in Field Environments

The Jagger × 2174 RIL population showed significant variation in reaction to stripe rust in multiple field environments. On the last scoring date in Pullman, WA, in 2008, IT values among the RILs of the population ranged from 2 to 8, with an average of 4.3, and DS ranged from 2 to 65%, with an average of 18%. In 2009, when stripe rust was more severe, average IT values were 5.0 and 5.2 in Pullman and Central Ferry, WA, respectively. The DS values in 2009 ranged from 2 to 90% with an average of 33% in Pullman and from 1 to 90% with an average of 37% in Central Ferry. At CAU in 2009, the average DS value was 26% but 28 RILs showed complete resistance (0% DS) and nine RILs showed extreme susceptibility (100% DS). At CAU in 2010, IT values ranged from 0 to 4 with an average of 1.1 and the average DS value was 19%. At Rossville, KS, IT values ranged from 1 to 9 with an average of 6.7 and the average DS value was 46%.

Based on relative resistance averaged across locations and years for those RILs (Fig. 1), approximately 44% of the RILs were considered resistant (0–10% of the highest DS value), suggesting that resistance in this population was conferred, in part, by a major gene. The remaining 56% of the RILs were considered intermediate to susceptible (11–100% of the highest DS value), but the variation within this subset indicated susceptibility was modified by additional minor gene(s).

At the Washington locations, the predominant races in 2008 were PST-114 (virulent on Lemhi [*Yr21*], Heines VII [*Yr2* and *YrHVII*], Moro [*Yr10* and *YrMor*], Produra [*YrPr1* and *YrPr2*], Yamhill [*Yr2*, *Yr4a*, and *YrYam*], Stephens [*Yr3a*, *YrS*, and *YrSte*], Lee [*Yr7*, *Yr22*, and *Yr23*], Fielder [*Yr6* and *Yr20*], Tres [*YrTr1* and *YrTr2*], Express, AvS*Yr8*NIL, AvS*Yr9*NIL, Clement [*Yr9* and *YrCle*], and Compair [*Yr8* and *Yr19*]) and PST-116 (virulence same as PST-114 plus Paha [*YrPa1*, *YrPa2*, and *YrPa3*]) with a low frequency of PST-138 (virulence same as PST-116 plus Hyak [*Yr17* and *YrTye*]) (Carter et al., 2009). In 2009, the predominant races were still PST-114 and PST-116 with an increased frequency of PST-138 and PST-127, which is the most virulent race identified to date in the United States. PST-127 is virulent on all 20 wheat differential genotypes except for Moro (*Yr10* and *YrMor*),

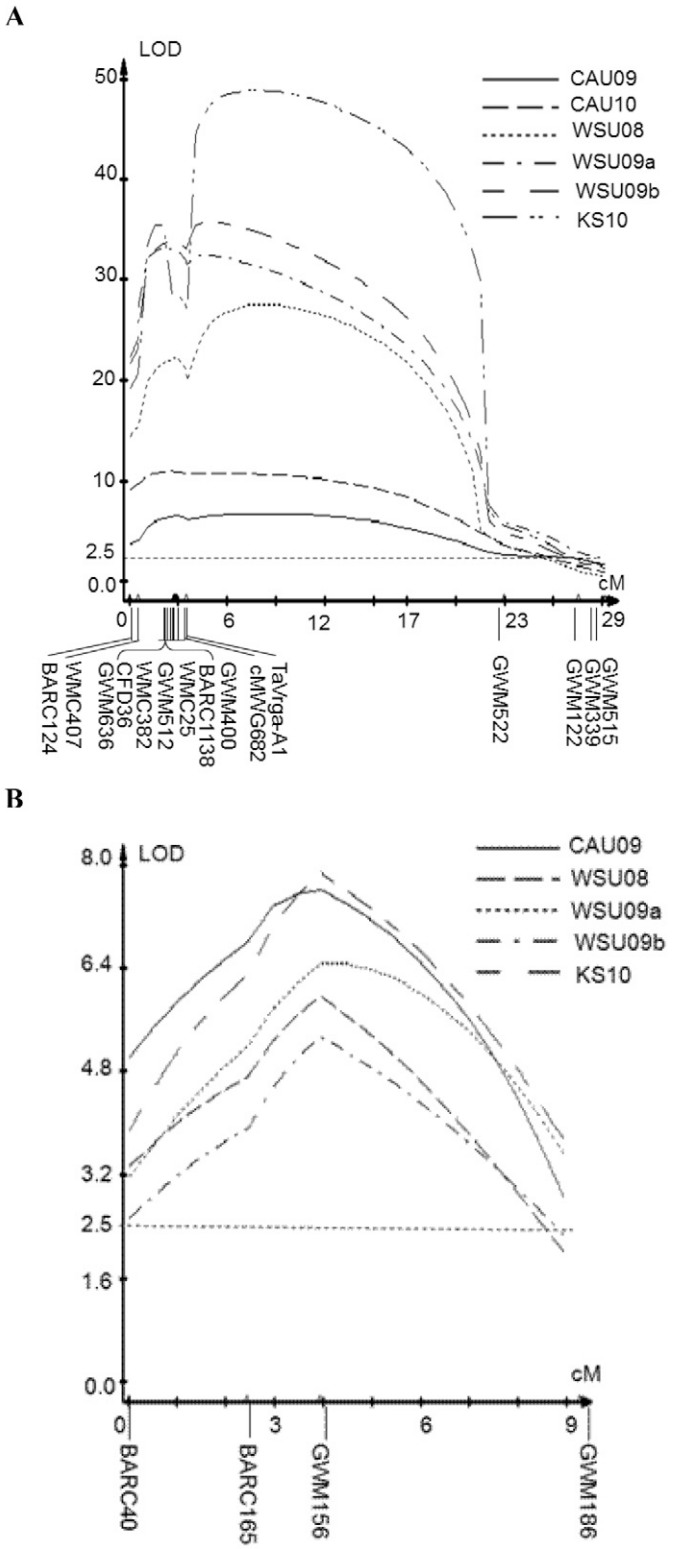

Figure 2. (right) Chromosomal location of quantitative trait loci (QTL) for reaction to stripe rust in the recombinant inbred lines (RILs) of the Jagger × 2174 population. A) A major QTL, *QYr.osu-2A*, on the short arm of chromosome 2A. B) A minor QTL, *QYr.osu-5A*, on the long arm of chromosome 5A. A and B) A solid line indicates a QTL characterized at China Agricultural University (CAU) in 2009 (CAU09), a dashed line indicates a QTL characterized at CAU in 2010 (CAU10), a dotted line indicates a QTL characterized in Pullman Research Station at Washington State University (WSU) in 2008 (WSU08), a dash-dotted line indicates a QTL characterized in Pullman Research Station at WSU in 2009 (WSU09a), a broken line indicates a QTL characterized in Central Ferry Research Station at WSU in 2009 (WSU09b), and a dash-double dotted line indicates a QTL characterized in Rossville Station in 2010 (KS10). Except for CAU10 and KS10 for which the disease response was reported as disease severity (DS), all other locations were based on relative area under the disease progress curve (rAUDPC). Molecular markers along the chromosome are placed as centimorgans on the horizontal axis. The horizontal dotted line represents a common threshold value of 2.5 logarithm of the odds (LOD).

AvsYr5NIL, and Tres (*YrTr1* and *YrTr2*). PST-127 has the combination of virulence on *Yr1*, *Yr2*, *Yr3a*, *Yr4a*, *Yr6*, *Yr7*, *Yr8*, *Yr9*, *Yr17*, *Yr19*, *Yr20*, *Yr21*, *Yr22*, *Yr23*, *YrExp1*, *YrExp2*, and several other resistance genes. In China, race CYR32 is virulent to 18 differential genotypes (*Yr1*, *Yr2*, *Yr3*, *Yr4*, *Yr6*, *Yr7*, *Yr9*, *Yr17*, *Yr22*, *Yr23*, *Yr27*, *YrA*, *YrCV1*, *YrCV2*, *YrCV3*, *YrG*, *YrSD*, and *YrSO*). CYR32 was first detected in 1991 on Red Abbondanza in Qinghai, China, and it is currently one of the predominant races. Eighty percent of commercial cultivars and germplasm in China were susceptible to CYR32 (Wan et al., 2003, 2004).

At Rossville, the experiment was inoculated with two isolates of race PST-100. PST-100 has the same virulence pattern on differentiates as PST-114 except that it is avirulent on Moro (*Yr10* and *YrMor*) and Tres (*YrTr1* and *YrTr2*). Although PST-100 is avirulent on the Hyak two-gene differential line containing *Yr17* and *YrTye*, these two isolates of PST-100 were partially virulent (IT = 7) on seedlings of Avocet-*Yr17*, a single-gene differential line containing *Yr17*.

Highly significant correlations ($p < 0.001$) were detected among all pairs of environments for AUDPC scores: Pullman in 2008 vs. Pullman in 2009, $r = 0.82$, and Pullman in 2009 vs. Central Ferry in 2009, $r = 0.90$. The r value exceeded 0.6 when comparing CAU in 2009 with the Washington environment within years. Highly significant correlations in DS were also detected between these environments and Rossville, KS. These results suggested common gene(s) may be responsible for adult-plant reactions in this population in different locations and years.

A Major Quantitative Trait Loci for Stripe Rust Resistance on 2A

A total of 360 molecular markers were used to create the linkage map in the Jagger × 2174 RIL population. We detected a major QTL near *Xgwm400* on the short arm of chromosome 2A for reaction to stripe rust in this population. Thirteen SSR markers constitute the same linkage group spanning approximately 30 cM in genetic distance (Fig. 2A). Two subgroups were separated by approximately 20 cM, one including nine markers tightly linked as a cluster (*Xbarc124*, *Xwmc407*, *Xgwm636*, *Xcfd36*, *Xwmc382*, *Xgwm512*, *Xwmc25*, *Xbarc1138*, and *Xgwm400*) and the other including four markers closely linked within

a 6-cM region (*Xgwm522*, *Xgwm122*, *Xgwm339*, and *Xgwm515*). In other mapping populations (Röder et al., 1998; Somers et al., 2004), *Xbarc1138* was mapped on the long arm of chromosome 2A (Song et al., 2005), *Xwmc25* was mapped in homeologous regions of chromosomes 2B and 2D (Somers et al., 2004), and all of the remaining 11 markers were mapped on the short arm of chromosome 2A (Somers et al., 2004), except for *Xgwm400* that had been previously mapped to chromosome 7B. These comparative maps indicated that the linkage group associated with the major QTL for stripe rust in the present study was located on the short arm of chromosome 2A. Therefore, this major QTL was designated *QYr.osu-2A*.

The major QTL *QYr.osu-2A* explained 80 to 93% of the total phenotypic variation in each of the U.S. environments (Fig. 2A). In contrast, the LOD values from the CAU environments for *QYr.osu-2A* at *Xgwm400* was 7.0 for rAUDPC in 2009 and 10.6 for IT scored at the same site in 2010. Only 36% of the total phenotypic variation was explained by the QTL in either year, and therefore only part of the adult-plant reaction to race CYR32 was controlled by the *QYr.osu-2A* locus. Overall, *QYr.osu-2A* explained up to 80% of the total phenotypic variation across years and locations, strongly suggesting that a major gene was responsible for resistance to multiple races of stripe rust, including PST-114 and PST-116 that frequently occur in Washington, PST-100 in the southern Great Plains, and race CYR32 in China.

The mean DS across all environments for those RILs carrying the Jagger allele at *QYr.osu-2A* was 6.1% whereas those carrying the 2174 allele had a mean value of 52.9%, indicating that Jagger carries a resistant allele and 2174 carries a susceptible allele for resistance to stripe rust. The genetic effect at *QYr.osu-2A* between the Jagger allele and the 2174 allele was highly significant in each environment (Fig. 3A).

Four stripe rust resistance genes or QTL were previously located on chromosome 2A, those being *Yr17* (Bariana and McIntosh, 1993; Seah et al., 2000; Helguera et al., 2003), *QYrtm.pau-2A* mapped to $2A^{m}L$ in diploid *T. monococcum* (Chhuneja et al., 2008), and the minor QTL *QYr.inra-2AL* and *QYr.inra-2AS1* from hexaploid wheat (Dedryver et al., 2009). Final mapping of two markers, *cMWG682* and *TaVrga-A1*, indicated that Jagger has the translocated VPM1 segment carrying *Yr17*, *Sr38*, and *Lr37* and that *QYr.osu-2A* is located within the VPM1 segment.

The *cMWG682* was specific to the VPM1 segment that was transferred into two breeding lines, UC1419 and UC1041. Jagger showed the same band pattern as UC1419 and UC1041 but 2174 showed a different band pattern than Jagger (Fig. 4A). All RILs showed the presence of PCR products either from chromosome 2N in Jagger or chromosome 2A in 2174, allowing direct mapping of *cMWG682* in the Jagger × 2174 population. When primers VRGA-F9 and VRGA-R3 were used to amplify *Vrga1* genes, one of three genes showed a polymorphism in band size (Fig. 4B). Jagger showed the same allele as UC1419 and UC1041 but 2174 showed a different band pattern from Jagger, facilitating direct mapping of one copy of *Vrga1* on chromosome 2A (designated *TaVrga-A1*).

A previous map in the diploid wheat *T. monococcum* showed *cMWG682* and *TaVrga-A1* to be 14.3 cM apart (Dubcovsky et al., 1996; Helguera et al., 2003). No crossover was detected between *TaVrga-A1* and *cMWG682* among the 96 RILs and, subsequently, the remaining 186 RILs of the Jagger × 2174 population. In addition, two SSR markers, *Xcfd36* and *Xwmc382*, in the linkage group including *cMWG682* and *TaVrga-A1* were reported to span approximately 16 cM (Somers et al., 2004) but no crossovers were detected in our population (Fig. 2A). The lack of crossovers at *QYr.osu-2A* in the Jagger × 2174 population is consistent with the expected nonpairing between the alien VPM1 segment in Jagger with the homologous wheat 2AS region in 2174. The peak of *QYr.osu-2A* was centered on *TaVrga-A1* and *cMWG682* (Fig. 2A). Therefore, we conclude that the resistance gene at *QYr.osu-2A* is likely *Yr17*.

The presence of the VPM1 segment in Jagger appears inconsistent with the reported pedigree by Sears et al. (1997). Jagger was developed by the Kansas Agricultural Experiment Station (Rossville, KS) and the USDA-ARS and reported to be from a cross between the breeding line 'KS82W418' and the soft white winter wheat cultivar Stephens (CI 17596) (Sears et al., 1997). Stephens does not have the marker allele for the VPM1 segment (Fig. 4) or the resistance phenotype for *Yr17* (Chen, 2005). KS82W418 could not be retrieved for this study. However, KS82W418 was the progeny of the cross between KS75216 and Plainsman V (PI 591702), which were both available for testing. KS75216 was a white-seeded sibling of the variety Newton. Neither Plainsman V nor KS75216 has the VPM1 marker allele (data not shown). Therefore, it is likely that the reported pedigree of Jagger is incorrect.

Among the 39 Coordinated Agriculture Project mapping population parents genotyped using *cMWG682* and *TaVrga-A1*, Jagger, Heyne, KS01HW163-4, and OR9900553 had the VPM1 segment or 10% of the total (Table 1). An additional 30 hard winter wheat cultivars from the Great Plains were genotyped, and approximately 33% of these cultivars were found to have the resistant allele at the *QYr.osu-2A* locus, a moderately high frequency. The presence of the VPM1 segment in these cultivars can be explained by their pedigrees as most of these cultivars have a close pedigree relationship with Jagger. For example, Jagalene is a cross of Jagger and Abilene (PI 511307). KS01HW163-4 is an experimental line from a cross between Trego and a sib of Betty (PI 612578), which was selected from the same cross as Jagger (Sears et al., 2001).

Yr17 has been described as conferring race-specific, all-stage (i.e., seedling and adult) resistance (Chen, 2005).

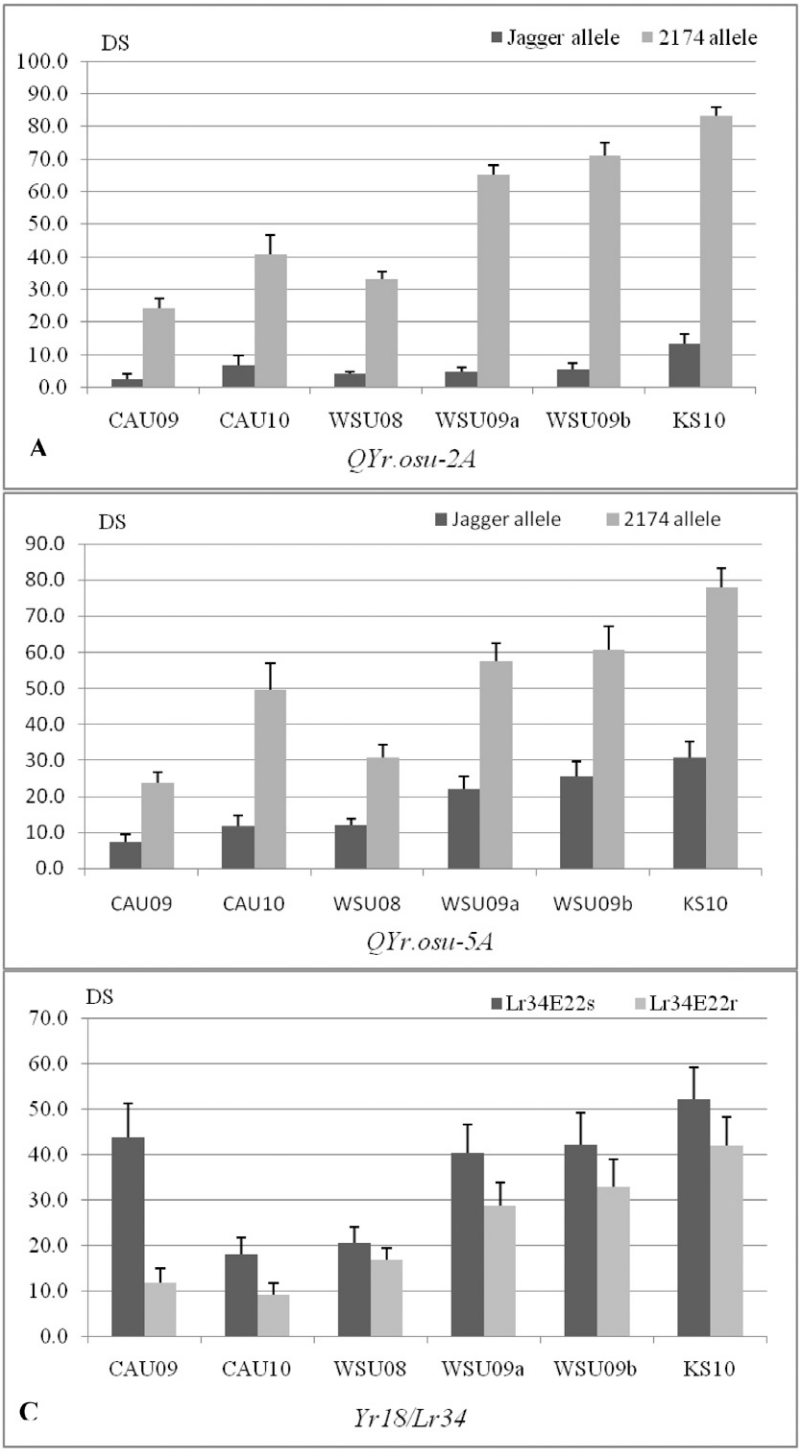

Figure 3. Comparison of reactions of genetic alleles at two quantitative trait loci (QTL) to stripe rust. The disease severity (DS) values for recombinant inbred lines (RILs) were analyzed to estimate allelic effects at China Agricultural University (CAU) in 2009 (CAU 2009), CAU in 2010 (CAU10), Pullman Research Station at Washington State University (WSU) in 2008 (WSU08) and 2009 (WSU09a), Central Ferry Research Station at WSU in 2009 (WSU09b), and at Rossville, KS, in 2010 (KS10). A) *TaVrga1* was analyzed to represent the *QYr.osu-2A* locus. B) *Xgwm156* was analyzed to represent the *QYr.osu-5A* locus. C) *Lr34E22* was the marker for the *Lr34/Yr18* gene, for which *Lr34E22s* is the Jagger allele and Lr34E22r is the 2174 allele. Bar indicates standard error.

However, in this study seedlings appeared moderately susceptible whereas adult plants were moderately resistant to the same races. It appears that *Yr17* can be considered a race-specific adult-plant resistance gene, at least for some isolates.

The genetic effect of *QYR.osu-2A* (*Yr17*) was highly significant in each location and year of this study (Fig. 3A). This is surprising since virulence to *Yr17* was previously reported in the United States and China (Chen, 2005). In Washington, races with virulence to *Yr17* had been first reported in

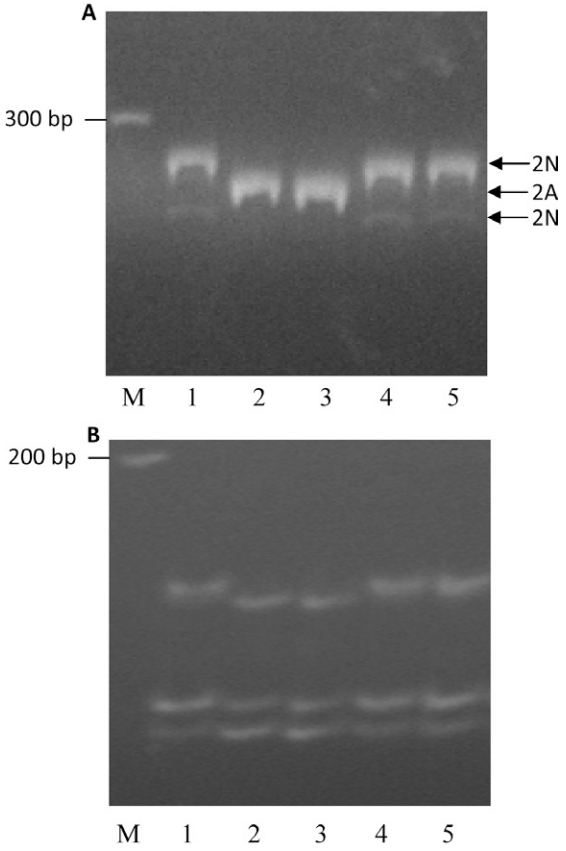

Figure 4. Allelic variation in two gene markers, *cMWG682* and *TaVrga-A1*. M. DNA fragment size marker. 1. Jagger. 2. 2174. 3. Stephens. 4. UC1419. 5. UC1041. UC1419 and UC1041 were confirmed to have the translocated VPM1 segment from *Aegilops ventricosa*. A) *cMWG682* marker. Polymerase chain reaction was performed using two forward primers, VENTRIUP and URIC, and one reverse primer, LN2. On a 6% acrylamide gel, primers VENTRIUP and LN2 amplified a 262-bp product from the translocated 2N segment and primers URIC and LN2 amplified a 275-bp product from chromosome 2A or 285-bp product from the translocated 2N segment. B) *TaVrga-A1* marker. Polymerase chain reaction was performed using primers VRGA-F11 and VRGA-R5. Polymerase chain reaction products were characterized on a 6% acrylamide gel.

the early 1990s but were not dominant during 2008 and 2009 when the Jagger × 2174 population was scored. PST-127 became the most prevalent race in the Pacific Northwest in 2010 and the field resistance of Jagger was consequently compromised. In China, CYR32 was reported to be virulent to *Yr17* and this may explain why the effect of *QYr.osu-2A* was smallest in China compared with experimental locations in the United States. At Rossville in 2010, *Yr17* provided very high resistance in adult plants against inoculated PST-100 isolates. However, we noted increased ITs and severity on Jagger derivatives at the end of the season. This was consistent with reports of increased severity on lines with *Yr17* throughout the Great Plains in 2010.

A Novel Quantitative Trait Loci for Stripe Rust Resistance on 5A

A previously undiscovered QTL for stripe rust reaction was consistently detected across years and locations (Fig. 2B). The four markers were mapped on the long arm of chromosome 5A based on their association with a group of markers including *VRN-A1*, which is responsible for stem elongation and winter dormancy release in this population (Chen et al., 2009a). Hence, this QTL was designated *QYr.osu-5A*.

Three QTL for stripe rust reaction were previously reported on chromosome 5AL, and all of them were located in the central region of the long arm of 5AL between markers *Xgwm126* and *Xbarc151* (Bariana et al., 2006; Chhuneja et al., 2008; Lan et al., 2010). However, *QYr.osu-5A* was located in a region close to the centromere of this chromosome and is not identical with the genes or QTL reported so far. Hence *QYr.osu-5A* is a novel stripe rust resistance locus.

QYr.osu-5A was centered on *Xgwm156*. The LOD value at *Xgwm156* was 6.0 and accounted for 25% of the total phenotypic variation in rAUDPC characterized in Pullman in 2008. The LOD value at *Xgwm156* was 6.5 and accounted for 28% of the total phenotypic variation in rAUDPC characterized in Pullman in 2009. The LOD value at *Xgwm156* was 5.4 and accounted for 22% of the total phenotypic variation in rAUDPC characterized in Central Ferry in 2009. The LOD value at *Xgwm156* was 7.6 and accounted for 30% of the total phenotypic variation in rAUDPC characterized at CAU in 2009. The LOD value at *Xgwm156* was 4.7 and accounted for 30% of the total phenotypic variation in IT characterized at CAU in 2010. The LOD value for *QYr.osu-5A* at *Xgwm156* was 7.9, which explained 31% of the total phenotypic variation at Rossville in 2010. Overall, *QYr.osu-5A* explained 26% of the total phenotypic variation. Reactions of the gene(s) at the *QYr.osu-5A* locus to stripe rust were stable and consistent across years and locations.

The mean DS across all environments for RILs carrying the Jagger allele at *QYr.osu-5A* was 18.3% whereas those carrying the 2174 allele had a mean value of 50.1%, indicating that Jagger also carries a resistant allele and 2174 carried a susceptible allele. The genetic effect at *QYr.osu-5A* between the Jagger allele and the 2174 allele was also highly significant in each environment (Fig. 3B).

Genetic Effect of the *Lr34/Yr18* Gene

Although 2174 carries a susceptible allele at each of *QYr.osu-2A* and *QYr.osu-5A*, its DS in China was 5 and 0% in 2009 and 2010, respectively, indicating high resistance to CYR32. These results indicated that 2174 has one or more resistance genes to stripe rust.

The cultivar 2174 is known to carry the *Lr34* gene for resistance to leaf rust. The *Lr34* locus also confers resistance to stripe rust and therefore this locus is known as

Table 1. Wheat cultivars or lines used for determining the frequency of the resistant or susceptible alleles at *QYr.osu-2A*.

Allele	Source†	Cultivar or line (with source of mapping population)
Resistant	CAP	Jagger (Oklahoma and Nebraska), OR9900553 (Oregon), and Heyne and KS01HW163-4 (Kansas)
Susceptible		2174 (Oklahoma), TAM105 (Nebraska), Stephens (Oregon), UC1110 and CIMMYT-2 (PI 610750) (California), Platte and CO940610 (Colorado), SS550 and PIONEER 26R46 (Georgia), Rio Blanco and IDO444 (Idaho), Zak and ID0556 (Idaho), P91193 and P92201 (Indiana), Harry and Wesley (Kansas), GRN*5/ND614-A and NY18/Clark's Cream 40-1 (Minnesota), McNeal and Thatcher (Montana), Rugby and Maier (North Dakota), Pio 25R26 and Foster (New York), Cayuga and Caledonia (New York), Weebill and Jupeteco (Texas, USG3209 and Jaypee (Virgina), Finch and Eltan (Washington), and Louise and Panawawa (Washington).
Resistant	SGP	Cutter, Danby, Doans, Fuller, Jagalene, Jagger, Neosho, OK Bullet, Overley, Protection, Santa Fe, Shocker.
Susceptible		Above, Centerfield, Custer, Deliver, Duster, Endurance, Fannin, Guymon, Hatcher, Intrada, JEI 110, Lakin, Okfield, Ok102, Ripper, TAM 110, TAM 111, TAM 112, Trego.

†CAP, Coordinated Agriculture Project (Chao et al., 2007); SGP, southern Great Plains.

Lr34/Yr18 (Krattinger et al., 2009). A PCR marker developed from the *Lr34* gene was mapped in the Jagger × 2174 population (Cao et al., 2010). At CAU in 2009, the mean DS value of those RILs carrying the Jagger *Lr34E22s* allele was 43.8% whereas the mean DS value of those RILs carrying the 2174 *Lr34E22r* allele was 11.8%, indicating a highly significant allelic effect of *Lr34/Yr18* on CYR32 tested in China ($p < 0.001$) (Fig. 3C). A similar effect was detected in 2010 ($p < 0.05$) (Fig. 3C). No significant genetic effect of *Lr34* on resistance to stripe rust races tested in Washington or Kansas could be detected, although the mean DS value of those RILs carrying the Jagger *Lr34E22s* allele was higher than that of those RILs carrying the 2174 *Lr34E22r* allele (Fig. 3C).

Hence *Lr34* in the 2174 parental line showed partial resistance to stripe rust in China. This gene alone did not provide a sufficient level of resistance to different races of stripe rust pathogens tested in the U.S. environments, as the *Lr34* genetic effect was apparently masked in the presence of the resistant alleles at the *QYr.osu-2A* and *QYr.osu-5A* loci. In China in 2010, the 2174 parental line still showed strong resistance even though the *Lr34* effect was decreased, suggesting 2174 might have additional gene(s) for protection against CYR32.

FUTURE PERSPECTIVES

We have identified one new QTL for stripe rust resistance and detected two previously reported genes for resistance in the cultivars Jagger and 2174. These results are critical to U.S. wheat breeders now that virulence to *Yr17* has increased in the United States. The presence of *Yr17* in many advanced breeding lines in the Great Plains indicates that the U.S. wheat crop is more vulnerable than previously thought and that additional resistance genes should be pyramided to protect the wheat crop from the threat of stripe rust.

Findings reported here invoke an important precautionary note on how resistance gene sources are perceived and therefore utilized. Often breeders perceive seedling-stage resistance as an immune reaction attributable to a single locus with race specificity and minimal environmental interactions whereas durable adult-plant (partial) resistance is attributable to multiple loci not subject to race-specific interactions but subject to environmental interactions. Resistance in Jagger indeed fit the adult-plant phenotype—durable for over a decade across a broad geography and subject to environmental interactions—yet can now be attributed in large part to a single major locus subject to race changes that eventually overcame resistance, at least partially. Other examples of single-gene adult-plant resistance to stripe rust can be cited, such as *Yr18* and *Yr36* (Fu et al., 2009; Krattinger et al., 2009). These examples, and that in Jagger, serve to remind breeders that a more complete knowledge of the genetic architecture of resistance sources may decrease unforeseen risks in their deployment.

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