18. C. Mackenzie *et al.*, *Annu. Rev. Microbiol.* **61**, 283 (2007).
19. A. Komeili, Z. Li, D. K. Newman, G. J. Jensen, *Science* **311**, 242 (2006).
20. We thank R. Gaudet, D. Rudner, C. Schmidt, L. Shapiro, R. Schekman, D. Weitz, and N. Wingreen for discussions and S. Lacefield, B. Scheid, and P. de Boer for advice. This work was supported by the Harvard Nanoscale Science and Engineering Center, a Fulbright grant to S.L., and NIH grant GM18568 to R.L.

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Materials and Methods
Figs. S1 to S3
Table S1
References

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A Kinase-START Gene Confers Temperature-Dependent Resistance to Wheat Stripe Rust

Daolin Fu,[1]*† Cristobal Uauy,[1]*‡ Assaf Distelfeld,[1,2]* Ann Blechl,[3] Lynn Epstein,[4] Xianming Chen,[5] Hanan Sela,[2] Tzion Fahima,[2] Jorge Dubcovsky[1]§

Stripe rust is a devastating fungal disease that afflicts wheat in many regions of the world. New races of *Puccinia striiformis*, the pathogen responsible for this disease, have overcome most of the known race-specific resistance genes. We report the map-based cloning of the gene *Yr36* (*WKS1*), which confers resistance to a broad spectrum of stripe rust races at relatively high temperatures (25° to 35°C). This gene includes a kinase and a putative START lipid-binding domain. Five independent mutations and transgenic complementation confirmed that both domains are necessary to confer resistance. *Yr36* is present in wild wheat but is absent in modern pasta and bread wheat varieties, and therefore it can now be used to improve resistance to stripe rust in a broad set of varieties.

Bread wheat (*Triticum aestivum* L.) provides ~20% of the calories consumed by humankind. The increasing world demand for cereals requires improved strategies to reduce yield losses due to pathogens. Wheat stripe rust, caused by the fungus *Puccinia striiformis* f. sp. *tritici* (PST, table S1), affects millions of hectares of wheat, and virulent races that have appeared within the past decade are causing large yield losses (*1*–*3*). Historically, resistant varieties have provided an economical and "environmentally friendly" method to control stripe rust. Numerous race-specific resistance genes have been deployed by breeders, but each has had limited durability, presumably because of rapid pathogen evolution. In contrast, partial resistance genes (i.e., "slow-rusting") offer a broader spectrum of resistance than race-specific genes; they are generally more effective at adult plant stages and usually confer more durable resistance (*1*). Unfortunately, our understanding of partial resistance to cereal rusts is limited because none of these genes has yet been cloned.

We report here the positional cloning of the high-temperature stripe rust resistance gene *Yr36*. This gene was first discovered in wild emmer wheat (*T. turgidum* ssp. *dicoccoides* accession FA15-3, henceforth DIC) (*4*). Analysis of *Yr36* isogenic lines in different genetic backgrounds confirmed that this gene confers partial resistance to PST under field conditions and is associated with significant yield increases when the pathogen is present. In controlled environments, plants with *Yr36* are resistant at relatively high temperatures (25° to 35°C) but susceptible at lower temperatures (e.g., 15°C) (*4*). *Yr36* resistance, originally discovered in adult plants, has some effectiveness in seedlings at high temperatures (fig. S1). Other high-temperature partial resistance genes have provided durable resistance to stripe rust and are used frequently in wheat breeding programs (*5*–*8*).

To clone *Yr36*, we crossed the susceptible durum wheat variety Langdon (LDN, Fig. 1A) with the resistant isogenic recombinant substitution line RSL65 (Fig. 1B), which carries *Yr36* in a LDN genetic background. We screened a population of 4500 F_2 plants using *Yr36* flanking markers *Xucw71* and *Xbarc136* (*4*) and identified 121 lines with recombination events between these two markers. On the basis of genes from the rice colinear region (*9*), nine polymerase chain reaction (PCR) markers were developed to construct a high-density map of *Yr36* (Fig. 1, C and D, and table S2). With the use of replicated field trials and controlled environment inoculations (tables S3 and S4 and figs. S2 and S3), *Yr36* was mapped to a 0.14-cM

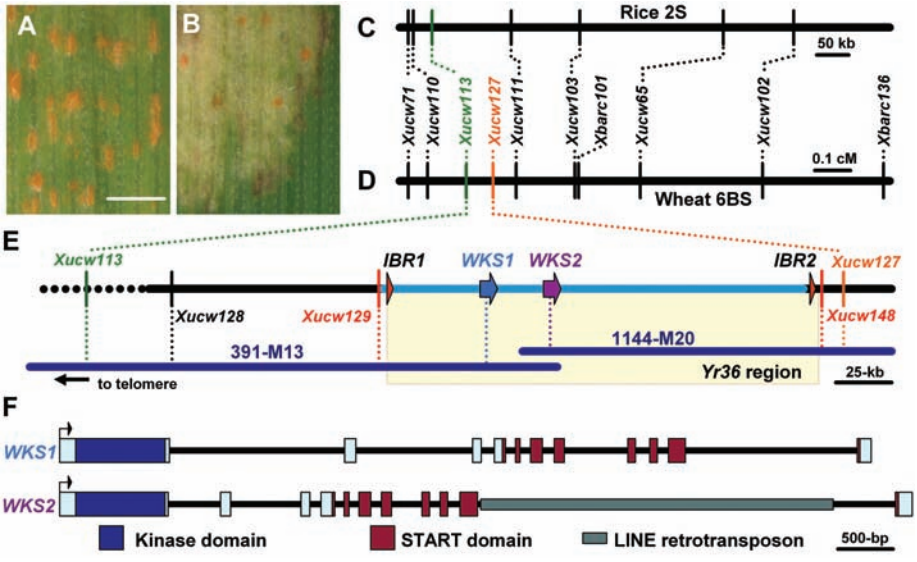

Fig. 1. Map-based cloning of *Yr36*. (**A** and **B**) Phenotype of susceptible parent Langdon with PST sporulation (A) and partially resistant parent RSL65 (B). Scale bar, 1 mm. (**C** and **D**) Genetic maps of colinear regions of rice chromosome 2 (C) and wheat chromosome 6B (D). (**E**) Physical map of the *Yr36* region. Genes are represented by colored arrows and the deleted region in Langdon by a light blue line. (**F**) Structure of the *WKS* genes. Exons are represented by rectangles, and the kinase and START domains are shown in different colors.

[1]Department of Plant Sciences, University of California, Davis, CA 95616, USA. [2]Department of Evolutionary and Environmental Biology, University of Haifa, Haifa 31905, Israel. [3]USDA-ARS, Western Regional Research Center, Albany, CA 94710, USA. [4]Department of Plant Pathology, University of California, Davis, CA 95616, USA. [5]USDA-ARS and Department of Plant Pathology, Washington State University, Pullman, WA 99164, USA.

*These authors contributed equally to this work.
†Present address: Department of Agronomy, Shandong Agricultural University, Tai'an, Shandong 271018, China.
‡Present address: John Innes Centre, Colney, Norwich NR4 7UH, UK.
§To whom correspondence should be addressed. E-mail: jdubcovsky@ucdavis.edu

interval delimited by markers *Xucw113* and *Xucw111* (Fig. 1D).

Screening the RSL65 bacterial artificial chromosome (BAC) library (*10*) with the distal marker *Xucw113* yielded six BACs (fig. S4). BAC ends were used to rescreen the library and extend the contig by chromosome walking. BAC-end marker *Xucw127* (table S2 and fig. S4) was mapped proximal to *Yr36*, thereby completing the physical map (Fig. 1E). BAC clones 391M13 and 1144M20 were sequenced and a contiguous 314-kb sequence including the flanking markers was annotated and deposited in GenBank (EU835198, fig. S5). New markers were developed from the sequence (table S2), and *Yr36* resistance (eight PST races, table S5) was mapped between *Xucw129* and *Xucw148* (0.02 cM).

This region has two pairs of duplicated genes (fig. S5). The first pair includes two short putative genes (*IBR1* and *IBR2*) with an "in between RING finger" domain (IBR, pfam01485). The two other duplicated genes, which we designated *WHEAT KINASE-START 1* and *2* (*WKS1* and *WKS2*, Fig. 1F), encode 86% identical proteins that have a predicted kinase domain followed by a predicted steroidogenic acute regulatory protein-related lipid transfer domain (START, pfam01852). *WKS1*, *WKS2*, and *IBR1* are deleted in the susceptible parent (Fig. 1E). The *WKS* genes were prioritized for functional characterization because their domains have been associated with plant responses to pathogens in other species (*11*–*13*).

Primers specific for *WKS1* and *WKS2* kinase and START domains (table S6) were used to screen a population of 1536 ethyl methanesulfonate (EMS)–mutagenized M_2 lines from the common wheat breeding line UC1041+*Yr36* (*14*). Of the 117 mutants found in the TILLING screen (*15*), we selected for functional characterization six mutants with changes in conserved amino acids in WKS1 (figs. S6 and S7A) and three with premature stop codons in *WKS2* (table S7).

Of the six *WKS1* mutants, five showed susceptible reactions similar to the susceptible UC1041 control line (Fig. 2, A to F, and figs. S8 and S9). In contrast, none of the *WKS2* truncation mutants was susceptible (fig. S8), which suggested that *WKS1* is *Yr36*. Both the kinase (fig. S8) and START domains (fig. S9) were necessary for the resistance response. Laser point scanning confocal microscopy showed that the T6-312 mutant had an unrestricted network of fungal growth, whereas the control line with a functional *WKS1* gene had a resistance response inside the leaf with reduced fungal growth delimited by autofluorescing plant cells (Fig. 2, G to J).

To confirm the identity between *WKS1* and *Yr36*, we transformed the susceptible wheat variety Bobwhite with a 12.2-kb genomic frag-

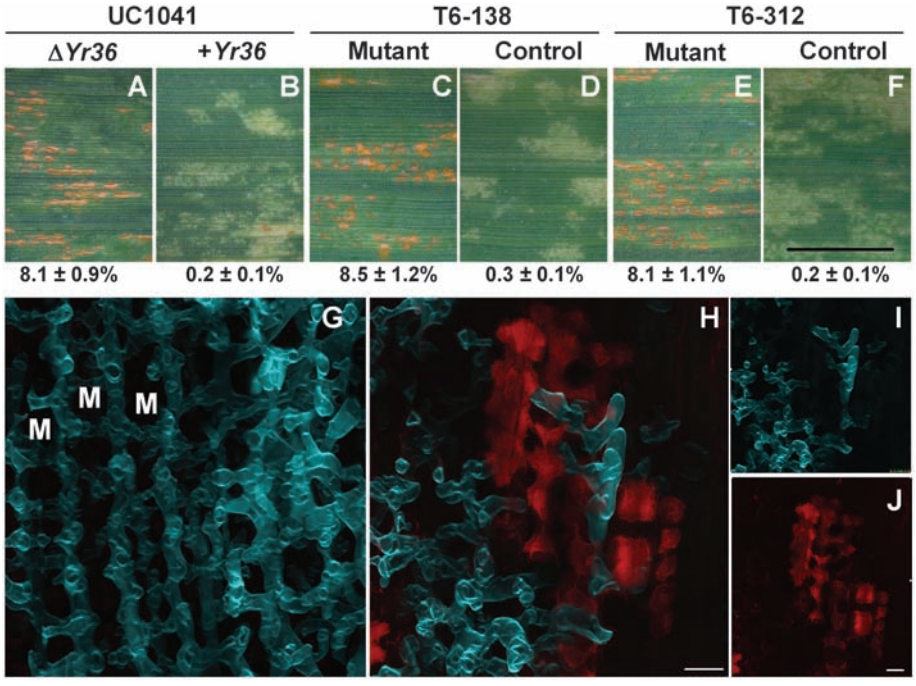

Fig. 2. Functional validation of *Yr36* by mutational analysis. (**A** to **F**) Leaf surfaces 11 days after PST inoculation. Scale bar, 5 mm. Numbers below leaves are average percent leaf area with pustules ± SEM (N = 8, fig. S2). An analysis of variance (ANOVA) of the log-transformed data showed significant differences ($P < 0.01$) between mutant and control lines. (A) UC1041 without *Yr36*. (B) UC1041+*Yr36* isogenic line used for mutagenesis. (C and E) Lines T6-138 and T6-312 with homozygous mutations in the *WKS1* kinase domain. (D and F) Sister lines without the mutations. These and additional mutant lines are described in table S7 and figs. S6 to S9. (**G** and **H**) A dual-channel, confocal microscopic z-series inside a wheat leaf 13 days after PST inoculation. Scale bar, 20 μm. The fungus stained with Uvitex 2B (Polysciences Inc., Warrington, PA; false-color blue) and autofluorescing wheat leaf cells (false-color red) are visible. (G) The susceptible T6-312 mutant has an extensive mycelial network in which each (invisible) plant mesophyll cell (selected cells shown as M) is encircled by a hypha. (H) The T6-312 control line has a poorly developed fungal network surrounded by autofluorescent mesophyll cells that presumably were involved in the resistance response. (**I** and **J**) Separate channels of (H). Scale bar, 20 μm.

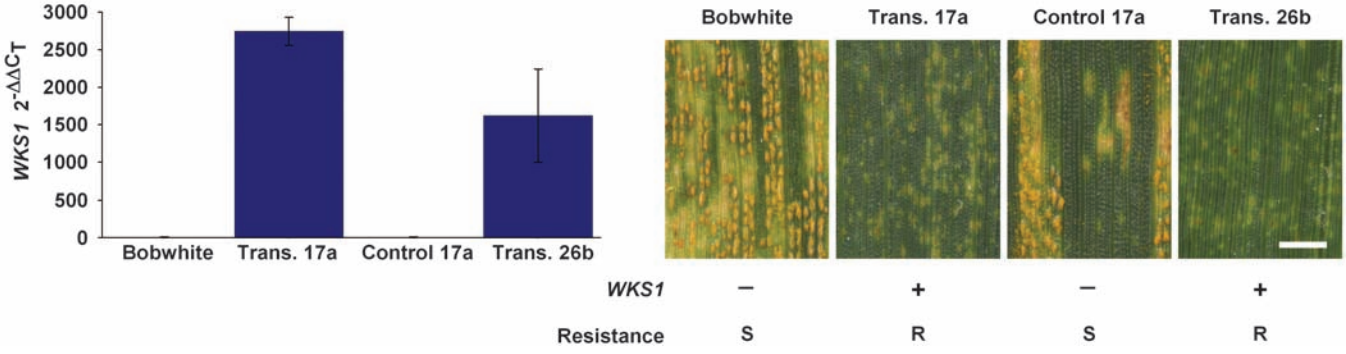

Fig. 3. *WKS1* transcript levels and resistance phenotype in transgenic wheat plants. Left panel: Average *WKS1* transcript levels (±SEM) in independent transgenic events 17a (five plants) and 26b (seven plants) were determined by quantitative reverse transcription (RT) PCR. Negative controls are the untransformed variety Bobwhite and the average of three T_1 sister lines of 17a without the transgene. Right panel: Leaf phenotypes (S, susceptible; R, resistant). Scale bar, 2 mm. Southern blots and transcription profiles of individual T_1 plants are shown in fig. S10.

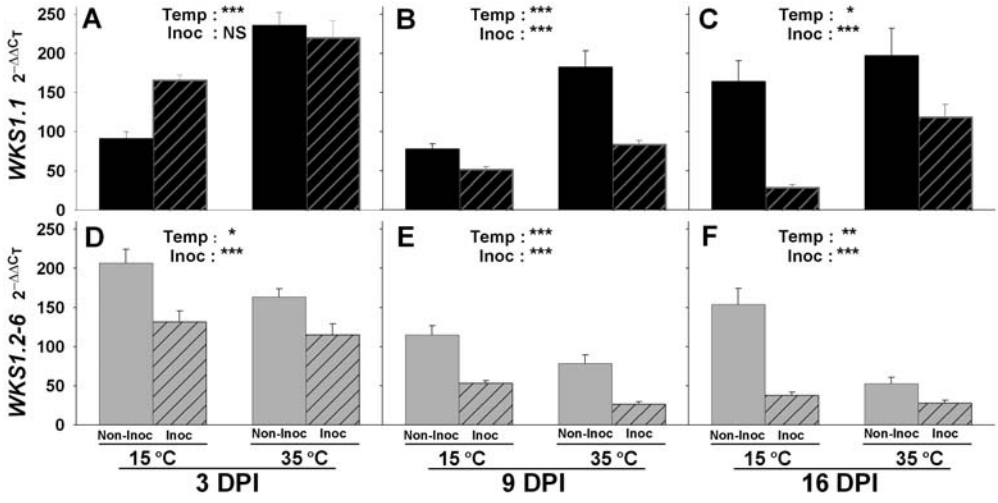

Fig. 4. (**A** to **F**) Effect of temperature and PST inoculation on transcript levels of *WKS1* transcript variants WKS1.1 and WKS1.2-6 in RSL65. Quantitative RT-PCR transcripts of WKS1.1 are indicated in black and those of WKS1.2-6 in gray. PST-inoculated plants are indicated by stripes and non-inoculated controls by solid colors. No significant interactions between temperature and inoculation were detected in the individual two-way ANOVAs, except for (A) (significant differences between inoculation classes only at low temperature). $*P < 0.05$, $**P < 0.01$, $***P < 0.001$; NS, not significant. Each data point is an average based on six replicates (±SEM). Overall ANOVAs are presented in table S8; comparisons between WKS1.1 and WKS1.2-6 transcript levels are shown in fig. S12.

ment that includes the complete *WKS1* coding and flanking regions (*14*). Only two of the nine independent T_1 transgenic lines had complete *WKS1* transcripts, and they were both resistant to stripe rust (Fig. 3 and fig. S10), thereby demonstrating that *WKS1* is *Yr36*.

The cloning and sequencing of 56 full-length *WKS1* cDNAs revealed six alternative transcript variants (WKS1.1 to 1.6, fig. S11). WKS1.1 encodes a complete WKS1 protein, whereas the other five (WKS1.2 to WKS1.6, henceforth WKS1.2-6) lack exon 11 and encode proteins with truncated START domains. Some of the missing amino acids are well conserved across the plant kingdom (fig. S7B). Quantitative PCR showed that even the lowest transcript levels of WKS1.1 and WKS1.2-6 are only one-third of those of *ACTIN*, indicating relatively high transcript levels. Overall, high temperature up-regulates WKS1.1 (Fig. 4, A to C, and fig. S12) and down-regulates WKS1.2-6 (Fig. 4, D to F, and fig. S12) ($P < 0.0001$, table S8).

PST inoculation consistently down-regulated WKS1.2-6 across temperature and time, but the effect on WKS1.1 transcript levels varied with sampling times (Fig. 4, A to C). Comparisons between WKS1.1 and WKS1.2-6 transcript levels in PST-inoculated plants (fig. S12, A to C) showed no significant differences at low temperature (susceptible response, $P > 0.55$) and significantly higher values of WKS1.1 relative to WKS1.2-6 at high temperature (resistant response, $P < 0.01$) for all 3 days.

The relative increase in transcript levels of the variant with the complete START domain (WKS1.1) at high temperature parallels the observed high-temperature resistance conferred by *Yr36*. START domain proteins in humans are known to play important roles in lipid trafficking, metabolism, and sensing; and their binding with sterols and ceramides results in protein conformational changes [reviewed in (*16*)]. If the putative WKS1 START domain has the ability to bind lipids from PST (or redirected by PST) at high temperature and change its conformation, this may cause the kinase domain to initiate a signaling cascade leading to the observed programmed cell death (Fig. 2 and fig. S8). The WKS1 serine-threonine kinase domain (pfam00069) was confirmed to have kinase activity (fig. S13).

The combination of the kinase and START domains in WKS1 apparently is the result of a novel domain shuffling, because these two domains are not found together in other organisms (*14*). The most similar protein in *Arabidopsis* to the putative WKS1 START domain is EDR2, a protein that negatively regulates plant defense to the powdery mildew pathogen *Golovinomyces cichoracearum* (*12*–*14*). EDR2 has PH (pfam00169) and DUF1336 (pfam07059) domains, which are absent in WKS1. The WKS1 kinase has high similarity to several *Arabidopsis* WAK-like kinases (fig. S6), but WKS1 lacks the additional domains characteristic of WAK-like kinases (*17*). The WKS1 kinase belongs to the non-RD kinases, which are frequently involved in the early steps of the innate immune response (*11*).

The appearance of this novel gene architecture preceded the origin of the Triticeae, because *WKS1* and *WKS2* were detected in several species from this tribe (table S9 and fig. S14). However, the presence of these two genes was rare among Triticeae species and varied across accessions within those species where they were detected. This suggests that *WKS1* and *WKS2* were lost repeatedly in several grass lineages, including the diploid donors of the A and D genomes of polyploid wheat (table S9). Among 131 wild and cultivated tetraploid wheat accessions, *WKS1* was detected only in wild wheat (24% of accessions), which suggests that *WKS1* was not incorporated into the initial domesticated forms. In hexaploid wheat, *WKS1* was present only in five accessions where the DIC segment was incorporated recently (table S10).

Introgression of *WKS1* in transgenic Bobwhite wheat and in susceptible varieties by backcrossing improved their resistance to stripe rust (*4*). This indicates either that WKS1 is sufficient to improve resistance, or that WKS1 can trigger intermediate genes still present in these varieties that initiate the hypersensitive response. Because *WKS1* is absent from almost all modern commercial varieties of pasta and bread wheat (table S10), the introgression of *Yr36* could have a broad impact in improving resistance to this pathogen. *Yr36* resistance has remained effective against the numerous stripe rust races present in California (2004–2008 field tests) and to all races tested so far in controlled environments (table S5). Moreover, *Yr36* has improved resistance in a variety carrying the partial resistance gene *Yr18* (*4*), which suggests that pyramiding appropriate combinations of partial resistance genes may provide adequate resistance against this pathogen. The discovery of different proteins and resistance mechanisms for the partial resistance genes *Yr36* and *Yr18/Lr34* (*18*) suggests that this type of resistance may involve a heterogeneous group of genes and mechanisms.

References and Notes

1. R. P. Singh, H. M. William, J. Huerta-Espino, G. Rosewarne, paper presented at the 4th International Crop Science Congress, Brisbane, Australia, 26 September to 1 October 2004 (www.cropscience.org.au/icsc2004/symposia/3/7/141_singhrp.htm).
2. X. M. Chen, *Aust. J. Agric. Res.* **58**, 648 (2007).
3. A. M. Wan, X. M. Chen, Z. H. He, *Aust. J. Agric. Res.* **58**, 605 (2007).
4. C. Uauy *et al.*, *Theor. Appl. Genet.* **112**, 97 (2005).
5. A. Qayoum, R. F. Line, *Phytopathology* **75**, 1121 (1985).
6. H. S. Shang, *Sci. Agri. Sin.* **31**, 46 (1998).
7. R. F. Line, *Annu. Rev. Phytopathol.* **40**, 75 (2002).
8. Q. Ma, H. S. Shang, *J. Plant Pathol.* **86**, 19 (2004).
9. A. Distelfeld *et al.*, *Funct. Integr. Genomics* **4**, 59 (2004).
10. A. Cenci *et al.*, *Theor. Appl. Genet.* **107**, 931 (2003).
11. C. Dardick, P. Ronald, *PLoS Pathog.* **2**, e2 (2006).
12. D. Tang, J. Ade, C. A. Frye, R. W. Innes, *Plant J.* **44**, 245 (2005).
13. S. Vorwerk *et al.*, *BMC Plant Biol.* **7**, 35 (2007).
14. See supporting material on *Science* Online.
15. C. M. McCallum, L. Comai, E. A. Greene, S. Henikoff, *Nat. Biotechnol.* **18**, 455 (2000).
16. F. Alpy, C. Tomasetto, *J. Cell Sci.* **118**, 2791 (2005).
17. J. A. Verica, Z. H. He, *Plant Physiol.* **129**, 455 (2002).

18. S. G. Krattinger *et al.*, *Science* **323**, 1360 (2009); published online 19 February 2009 (10.1126/science.1166453).
19. Supported by USDA-CSREES grants 2005-00975 and 2006-55606-16629 and by U.S. and Israel BARD Fund grant US-4024-07. We thank C. Li, F. Paraiso, L. Penman, K. L. Richardson, S. Bassein, M.R. Paddy, Q. Lam, and M. Jindal for technical assistance and R. Thilmony and M. Whalen for critical reading of the manuscript. Gene sequences have been deposited in GenBank with accession numbers EU835198-EU835200, FJ154103-FJ154118, and FJ155069-FJ155070.

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SOM Text
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Tables S1 to S10
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A Putative ABC Transporter Confers Durable Resistance to Multiple Fungal Pathogens in Wheat

Simon G. Krattinger,[1]* Evans S. Lagudah,[2]*† Wolfgang Spielmeyer,[2] Ravi P. Singh,[3] Julio Huerta-Espino,[4] Helen McFadden,[2]‡ Eligio Bossolini,[1]§ Liselotte L. Selter,[1] Beat Keller[1]†

Agricultural crops benefit from resistance to pathogens that endures over years and generations of both pest and crop. Durable disease resistance, which may be partial or complete, can be controlled by several genes. Some of the most devastating fungal pathogens in wheat are leaf rust, stripe rust, and powdery mildew. The wheat gene *Lr34* has supported resistance to these pathogens for more than 50 years. *Lr34* is now shared by wheat cultivars around the world. Here, we show that the LR34 protein resembles adenosine triphosphate–binding cassette transporters of the pleiotropic drug resistance subfamily. Alleles of *Lr34* conferring resistance or susceptibility differ by three genetic polymorphisms. The *Lr34* gene, which functions in the adult plant, stimulates senescence-like processes in the flag leaf tips and edges.

Improved control of fungal rust diseases in cereals through breeding varieties with durable rust resistance is critical for world food security. International attention has been recently drawn to the continuing major threat of fungal rust diseases of cereals, highlighting the need for effective and durable sources of rust resistance. The most profitable and environmentally friendly strategy for farmers to control wheat rusts in both the developing and the developed world is to grow genetically resistant wheat varieties.

The wheat gene *Lr34* is associated with resistance to two rust diseases of wheat, leaf rust (caused by *Puccinia triticina*) (Fig. 1, A and B), and stripe rust (*P. striiformis*) (*1*–*3*), as well as powdery mildew (*Blumeria graminis*) (*4*). *Lr34* provides an important source of partial resistance that is expressed in adult plants during the critical grain-filling stage and is most effective in the uppermost leaf, the so-called flag leaf. When deployed with other adult plant resistance genes, near-immunity can be achieved (*5*). Flag leaves of many wheat cultivars containing *Lr34* develop a necrotic leaf tip, a morphological marker described as leaf tip necrosis (Fig. 1C) (*6*, *7*). The gene was first documented in Canada by Dyck although *Lr34*-containing germplasm has been a part of wheat improvement since the early 20th century. Wheat cultivars containing *Lr34* occupy more than 26 million ha in various developing countries alone and contribute substantially to yield savings in epidemic years (*8*).

The *Lr34* gene has remained durable, and no evolution of increased virulence toward *Lr34* has been observed for more than 50 years. This is in contrast to many other rust resistance genes, the so-called gene-for-gene class, that provide resistance to some but not all strains of a rust species (*9*–*12*). Despite the importance of adult plant resistance genes (*13*), no such gene has been cloned to date. Understanding the molecular nature of this class of resistance has important implications for long-term control of rust diseases. Previous studies have localized the codominant gene *Lr34* on the short arm of chromosome 7D between the two markers gwm1220 and SWM10 (*14*, *15*). We further reduced the target interval in a map-based cloning approach based on three high-resolution populations (*16*) (table S1). High-resolution mapping revealed a 0.15-cM target interval for *Lr34* flanked by *XSWSNP3/XcsLVA1* and *XcsLVE17* (Fig. 1D). The 363-kb physical interval containing both flanking markers was fully sequenced in the *Lr34*-containing hexaploid wheat cultivar Chinese Spring (FJ436983). Sequence analysis revealed the presence of a gene-rich island containing eight open reading frames (Fig. 1E) predicted to encode proteins with homologies to a hexose carrier, an ATP-binding cassette (ABC) transporter, two cytochromes P450, two lectin receptor kinases, a cysteine proteinase, and a glycosyl transferase. The latter two genes were interrupted by repetitive elements and were excluded as candidates for *Lr34*. Molecular markers derived from the coding sequences resembling one of the two lectin receptor kinases (SWDEL3), the ABC transporter (SWDEL2/csLVD2), and the hexose carrier (SWDEL1) were cosegregating with *Lr34*.

To determine whether one of these cosegregating genes corresponds to *Lr34*, we examined for sequence differences in their coding regions from the three pairs of +/−*Lr34* parental lines of the mapping populations. Consistent sequence polymorphism between the alleles of all parental pairs was found only in the putative ABC transporter gene. Second, we sequenced locus-specific DNA fragments covering parts of the six candidate genes on two γ-irradiation (m19 and m21) and six sodium azide–induced *Lr34* mutants (2B, 2F, 2G, 3E, 4C, and 4E) that were selected for loss-of-function of the *Lr34* resistance. Each mutant showed sequence alterations in the putative ABC transporter gene (table S2), leading to either splice site mutations resulting in strongly reduced splicing efficiency or mis-splicing (fig. S1), amino acid exchanges, frame shifts, or premature stop codons (Fig. 1F). To test for the presence of additional mutations in the other genes cosegregating with *Lr34*, we sequenced DNA fragments covering 12 to 15 kb of the other five candidate genes and intergenic regions on the six mutants 2B, 3E, 4C, 4E, m19, and m21 without finding any sequence polymorphism. Hence, we can exclude the possibility that the eight independent mutations found in the putative ABC transporter gene are due to a generally very high mutation frequency in these lines, and we conclude that this gene is responsible for conferring the durable *Lr34* disease resistance.

Lr34 cosegregated with partial resistance to adult plant stripe rust (*Yr18*), powdery mildew (*Pm38*), as well as leaf tip necrosis (*Ltn1*). The mutants were more susceptible to leaf rust, stripe rust, and powdery mildew, and they did not show leaf tip necrosis. These observations, based on eight independent mutations within a single putative ABC transporter gene, strongly suggest that the same gene controls resistance based on

[1]Institute of Plant Biology, University of Zurich, Zollikerstrasse 107, 8008 Zurich, Switzerland. [2]Commonwealth Scientific and Industrial Research Organisation (CSIRO) Plant Industry, GPO Box 1600, Canberra, ACT, 2601, Australia. [3]International Maize and Wheat Improvement Center (CIMMYT), Apdo. Postal 6-641, 06600 Mexico DF, Mexico. [4]Campo Experimental Valle de Mexico INIFAP, Apdo. Postal 10, 56230 Chapingo, Edo de Mexico, Mexico.

*These authors contributed equally to this work.
†To whom correspondence should be addressed. E-mail: evans.lagudah@csiro.au (E.S.L.), bkeller@botinst.uzh.ch (B.K.)
‡Present address: Science and Research Division, Department of Innovation, Industry, Science and Research, Industry House Level 6, 10 Binara Street, Canberra, ACT, 2601, Australia.
§Present address: Institute of Plant Sciences, University of Bern, 3013 Bern, Switzerland.