the OH features have not yet participated in the general shear motion of the gas cloud surrounding the remnant. In this scenario, the OH spots are the sites where the gas has most recently been shocked to form masers. The time scale for the shear to be effective is about 5×10^5 years, which is much larger than the typical lifetime of maser clouds.

The distribution of 1720-MHz OH masers along the SNR–CO-cloud interface suggests that there is an association between the three components and that the OH masers are being generated behind a shock. However, questions regarding the striking kinematic differences between the OH and CO gas need to be addressed in more detail. Future observations of CS to search for low-velocity molecular gas associated with the G359.1-0.5 SNR, as well as a search for the 1665- and 1667-MHz OH lines, are needed to confirm the nature of the OH 1720-MHz maser pumping mechanism and the nature of the association between the nonthermal remnant and its surrounding ring of molecular gas.

REFERENCES AND NOTES

1. D. A. Frail, W. M. Goss, V. I. Slysh, *Astrophys. J.* **424**, L111 (1994).
2. K. I. Uchida, M. Morris, M. Yusef-Zadeh, *Astron. J.* **104**, 1533 (1992).
3. A. D. Gray, L. E. Cram, R. D. Ekers, W. M. Goss, *Nature* **353**, 237 (1991).
4. F. Yusef-Zadeh, M. Morris, D. Chance, *ibid.* **310**, 557 (1984); H. Liszt, *Astrophys. J.* **293**, L65 (1985).
5. K. I. Uchida, M. Morris, J. Bally, M. Pound, F. Yusef-Zadeh, *Astrophys. J.* **398**, 128 (1992).
6. J. Bally, A. A. Stark, R. W. Wilson, C. Henkel, *ibid.* **324**, 223 (1988).
7. B. E. Turner, *ibid.* **255**, L33 (1982).
8. D. Hollenbach, and C. F. McKee, *ibid.* **342**, 306 (1989); M. Wardle, *Mon. Not. R. Astron. Soc.* **251**, 119 (1991).
9. M. Elitzur, *Astrophys. J.* **203**, 124 (1976).

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A Receptor Kinase–Like Protein Encoded by the Rice Disease Resistance Gene, *Xa21*

Wen-Yuan Song,* Guo-Liang Wang,* Li-Li Chen, Han-Suk Kim, Li-Ya Pi, Tom Holsten, J. Gardner, Bei Wang,† Wen-Xue Zhai, Li-Huang Zhu, Claude Fauquet, Pamela Ronald‡

The rice *Xa21* gene, which confers resistance to *Xanthomonas oryzae* pv. *oryzae* race 6, was isolated by positional cloning. Fifty transgenic rice plants carrying the cloned *Xa21* gene display high levels of resistance to the pathogen. The sequence of the predicted protein, which carries both a leucine-rich repeat motif and a serine-threonine kinase–like domain, suggests a role in cell surface recognition of a pathogen ligand and subsequent activation of an intracellular defense response. Characterization of *Xa21* should facilitate understanding of plant disease resistance and lead to engineered resistance in rice.

Receptor protein kinases mediate cellular signaling processes in diverse biological systems. In many animal systems, the binding of ligands to the extracellular receptor domain of these proteins activates a cytoplasmic tyrosine kinase domain. This interaction produces a variety of cellular responses, such as cell proliferation, differentiation, and survival (*1*). The few plant receptor kinase–like proteins that have been studied to date carry serine-threonine specificity in the kinase domain (*2*). One of these proteins, the S-receptor kinase (SRK), has been postulated to mediate self-recognition between pollen and stigma during pollination, although transformation experiments have not yet conclusively demonstrated such a role (*3*). The biological function of the other plant receptor kinase–like proteins remains unclear.

One possible role for these proteins in plants is in mediating disease resistance. Cellular signaling processes appear to be of central importance to the mechanisms by which plants resist viral, bacterial, and fungal pathogens. Evidence for this model comes from the recent characterization of several disease resistance genes from dicotyledonous species. The deduced amino acid sequence of five of these genes demonstrates the presence of either a serine-threonine kinase (STK) or a leucine-rich repeat (LRR) domain, suggesting a role in protein phosphorylation or protein-protein interactions (*4*).

Three members of the monocotyledonous family Poaceae (maize, rice, and wheat) provide most of the calories consumed by humans. Despite their agronomic importance, molecular genetic studies of monocots have been hindered by the large genome size of most of these plants. Rice provides an amenable system for positional cloning in monocots because of its small genome size, extensive genetic map, and ease of transformation.

Here we report positional cloning of the rice gene *Xa21*, which confers resistance against the bacterial pathogen *Xanthomonas oryzae* pv. *oryzae* race 6 (*Xoo*) (*5*). The de-

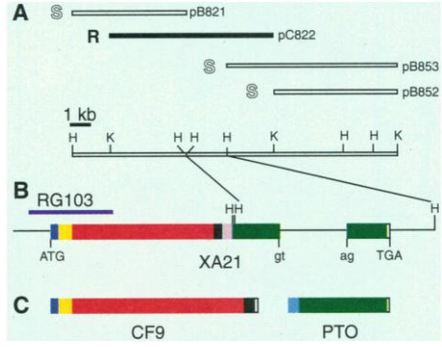

Fig. 1. (**A**) Partial restriction enzyme map and rice complementation analysis of cosmid 116. Transformation of Taipei 309 with genomic subclones pB821, pC822, pB852, and pB853 produced plants with resistance (R) or susceptibility (S) to *Xoo* race 6 strain PX099Az. The 9.6-kb Kpn I DNA fragment of cosmid 116 was cloned into plasmid pTA818 (*6*) to generate pC822. Hind III (H), Kpn I (K), and Hind III–Kpn I DNA fragments of cosmid 116 were ligated to pBluescript SK$^+$ (Stratagene) to generate pB821, pB852, and pB853, respectively. Scale is indicated by a bar. (**B**) The XA21-coding region and deduced amino acid sequence. The ATG and TGA codons, the RG103-hybridizing region, and 5′ and 3′ splice junctions corresponding to the consensus sequences of eukaryotic mRNAs are marked. The intron is designated by a horizontal bar. (**C**) The tomato CF9 and PTO deduced amino acid sequences. Domains of the protein are indicated in (B) and (C) as follows: blue, presumed signal peptide; yellow, unknown; red, LRR; black, transmembrane; pink, juxtamembrane; green, kinase; turquoise, nonhomologous region; and white, COOH-terminal tail.

W.-Y. Song, G.-L. Wang, L.-Y. Pi, T. Holsten, B. Wang, P. Ronald, Department of Plant Pathology, University of California, Davis, CA 95616, USA.
L.-L. Chen and C. Fauquet, International Laboratory for Tropical Agricultural Biotechnology/The Scripps Research Institute–Institut Francais de Recherche Scientifique et Technique pour le Developpement en Cooperation (ORSTOM), Scripps Institute, Plant Division MRC7, 10666 North Torrey Pines Road, La Jolla, CA 92037, USA.
H.-S. Kim and J. Gardner, Center for Engineering Plants for Resistance Against Pathogens, University of California, Davis, CA 95616, USA.
W.-X. Zhai and L.-H. Zhu, Institute of Genetics, Academic Sinica, Beijing, China 100101.

*These authors contributed equally to this work.
†Present address: Department of Molecular Microbiology, School of Medicine, Washington University, St. Louis, MO 63110, USA.
‡To whom correspondence should be addressed.

rived amino acid sequence of the *Xa21* gene product is similar to that of animal receptor kinases. We use transformation experiments to demonstrate that the plant receptor kinase–like protein, XA21, has a specific function, namely pathogen recognition and response. Compared with previously cloned genes, the structure of *Xa21* represents an as yet uncharacterized class of plant disease resistance genes and supports a role for cellular signaling in plant disease resistance.

We previously identified three chromosome 11 markers linked to the *Xa21* locus (6). One of these markers, RG103, hybridized with eight genomic DNA fragments in the resistant *Oryza sativa* ssp. Indica line, IRBB21, and the *Xa21* donor species, *O. longistaminata*. At least seven of these fragments cosegregated with the *Xa21* locus in 386 F_2 progeny (6). The deduced amino acid sequence of RG103 showed 20 to 30% identity to diverse proteins carrying LRR motifs.

The analysis described above suggested that the RG103-hybridizing DNA fragments in line IRBB21 may include the *Xa21* gene. To test this hypothesis, we used RG103 as a hybridization probe to isolate clones from bacterial artificial chromosome (BAC) and cosmid libraries (7, 8). Sixteen partially overlapping subclones representing seven of the RG103-hybridizing genomic fragments were transformed into the normally susceptible line, *O. sativa* ssp. Japonica var. Taipei 309, by particle bombardment (9). For each subclone, ~15 independently transformed lines were generated. On average, six plants were subsequently propagated clonally from each independently transformed line. Four months after bombardment, a total of 1500 transgenic plants carrying 16 different subclones were inoculated with *Xoo* to test for resistance. Fifty plants, arising from nine independently transformed lines containing a 9.6-kb Kpn I subclone, showed a reduction in lesion length as compared with susceptible controls (Figs. 1A and 2, A and B). Bacterial growth curve analysis demonstrated that growth of the pathogen was reduced by a factor of 10 and 100 in transgenic line 106-22 carrying the 9.6-kb Kpn I subclone as compared with IRBB21 and the recipient line Taipei 309, respectively (Fig. 2C). These results indicate that the transgenic lines containing the 9.6-kb subclone gained resistance to *Xoo*. Because *Xa21* is the only known chromosome 11 locus that confers resistance to *Xoo* race 6, we have designated

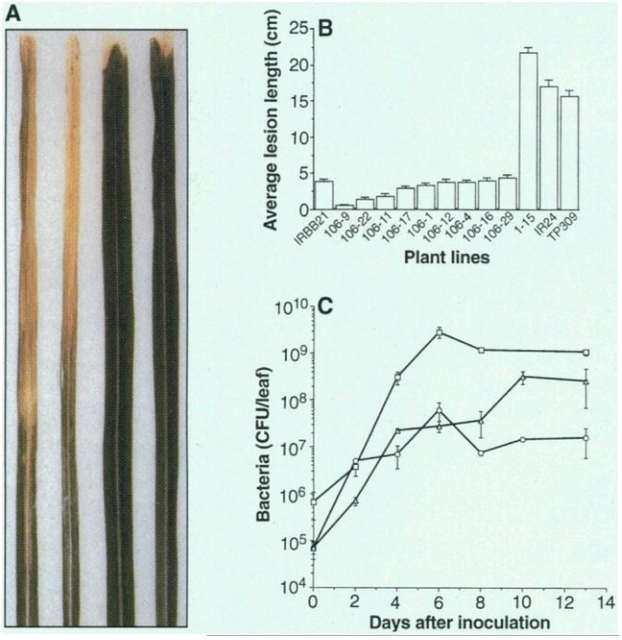

Fig. 2 (left). Resistance to *Xoo* strain PX099Az is conferred by the cloned *Xa21* gene. (**A**) Photograph taken 11 days after inoculation with *Xoo* race 6 strain PX099Az. Leaf 1 and 2, Taipei 309; leaf 3 and 4, transgenic line 106-22 containing the cloned *Xa21* gene. (**B**) Lesion length data of nine *Xa21*-containing (designated 106-1 through 106-29) and vector-containing (1-15) transgenic lines. Each data point represents two to nine clonal individuals with three to six inoculations per individual. (**C**) Growth of *Xoo* race 6 strain PX099Az in transgenic and control lines. Three clonal individuals from transgenic line 106-22 were inoculated 4 months after transformation. For each time point, the bacterial populations were determined by grinding three leaves separately, plating on potato sucrose agar media containing 200 μM azacytidine, and counting colony-forming units (CFUs). Squares, Taipei 309; circles, transgenic line 106-22 carrying the cloned *Xa21* gene; triangles, resistant *O. sativa* ssp. Indica line, IRBB21. Bars in (B) and (C), SEM. Inoculation was carried out as described (*24*). **Fig. 3 (right).** Predicted amino acid sequence of the *Xa21* gene product. The deduced protein domains are indicated as follows: (**A**) Potential signal sequence; (**B**) unknown function; (**C**) LRR; (**D** and **F**) charged; (**E**) transmembrane; (**G**) juxtamembrane; (**H**) serine-threonine kinase; and (**I**) COOH-terminal tail. Highly conserved amino acids in the LRR are shown in red. The 15 amino acids that are invariant among protein kinases are shown in green (*16*). Residues that indicate serine-threonine specificity are in large uppercase letters (*16*). N-glycosylation sites are underlined. The location of the intron (marked by a slash) was confirmed by sequencing cDNAs from IRBB21 and the transgenic 106-17 line (*11*). The GenBank accession number for XA21 genomic and cDNA sequences is U37133. Single-letter abbreviations for the amino acid residues are as follows: A, Ala; C, Cys; D, Asp; E, Glu; F, Phe; G, Gly; H, His; I, Ile; K, Lys; L, Leu; M, Met; N, Asn; P, Pro; Q, Gln; R, Arg; S, Ser; T, Thr; V, Val; W, Trp; and Y, Tyr.

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A MISLPLLLFVLLFSALLLCPSSS                                   23

B DDDGDAAGDELALLSFKSSLLYQGGQSLASWN                          55
  TSGHGQHCTWVGVVCGRRRRRHPHR                                 80

C       VVK LLLRSSN LSGIISPS                                98
  LGNLSFLRE LDLGDNY LSGEIPPE                               122
  LSRLSRLQL LELSDNS IQGSIPAA                               146
  IGACTKLTS LDLSHNQ LRGMIPREI                              171
  GASLKHLSN LYLYKNG LSGEIPSA                               195
  LGNLTSLQE FDLSFNR LSGAIPSS                               219
  LGQLSSLLT MNLGQNN LSGMIPNS                               243
  IWNLSSLRA FSVRENK LGGMIPTNA                              268
  FKTLHLLEV IDMGTNR FHGKIPAS                               292
  VANASHLTV IQIYGNL FSGIITSG                               316
  FGRLRNLTE LYLWRNL FQTREQDDWGFISD                         346
  LTNCSKLQT LNLGENN LGGVLPNSF                              371
  SNLSTSLSF LALELNK ITGSIPKD                               395
  IGNLIGLQH LYLCNNN FRGSLPSS                               419
  LGRLKNLGI LLAYENN LSGSIPLA                               443
  IGNLTELNI LLLGTNK FSGWIPYT                               467
  LSNLTNLLS LGLSTNN LSGPIPSE                               491
  LFNIQTLSIMINVSKNN LEGSIPQE                               516
  IGHLKNLVE FHAESNR LSGKIPNT                               540
  LGDCQLLRY LYLQNNL LSGSIPSA                               564
  LGQLKGLET LDLSSNN LSGQIPTS                               588
  LADITMLHS LNLSFNS FVGEVPT                                611
  IGAFAAASG ISIQGNAKLCGGIP                                 634

D DLHLPRCCPLLENRKH                                         650

E FPVLPISVSLAAALAILSSLYLLITW                               676

F HKRTKK                                                   682

G GAPSRTSMKGHPLVSYSQLVKATDG                                707

H FAPTNLLGSGSFGSVYKGKLNIQDHVAVKVLKLENPKALKSFTA             751
  ECEALRNMRHRNLVKIVTICSSIDNRGNDFKAIVYDFMPNGSLE             795
  DWIHPETNDQADQRHLNLHRRVTILLDVACALDYLHRHGPEPVV             839
  HCDIKSSNVLLDSDMVAHVGDFGLARILVDGTSLIQQSTS                 879
  SMGFIGTIGYAAPEYGVGLIASTHGDIYSYGILVLEI                    916
  VTGKRPTDSTFRPDLGLRQYVELGLHGRVTDVVDTKLILDSENW             960
  LNSTNNSPCRRITECIVWLLRLGLSCSQELPSSRTPTGDIIDEL            1004

I NAIKQNLSGLFPVCEGGSLEF                                   1025
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the gene contained on the 9.6-kb subclone *Xa21* (*5*).

To localize *Xa21* on the 9.6-kb Kpn I subclone, we carried out additional transformations with four partially overlapping DNA subclones from this region. These experiments delineated a 2.3-kb Hind III DNA fragment required for full resistance activity (Fig. 1, A and B). Sequencing of the 9.6-kb Kpn I genomic fragment containing the 2.3-kb Hind III fragments, revealed a single, large open reading frame (ORF) of 3075 base pairs, interrupted by one intron of 843 base pairs (Fig. 1B). Sequencing of complementary DNAs (cDNAs) indicates that the intron is processed as predicted in both IRBB21 and the transgenic line 106-17 (*10*, *11*). In RNA blot experiments of IRBB21, four bands hybridize with RG103 (*12*). The largest band of ~3.1 kb is consistent with the size of the full-length cDNA isolated from line 106-17 (*11*).

Analysis of the derived 1025–amino acid (aa) sequence of XA21 revealed several regions with similarity to known protein domains (Fig. 3). The NH_2-terminus encodes 23 hydrophobic residues characteristic of a signal peptide (*13*). Amino acids 81 to 634 consist of 23 imperfect copies of a 24-aa LRR. The conserved glycine is characteristic of extracellular LRR-containing proteins (*14*). Amino acids 651 to 676 encode a 26-aa hydrophobic stretch that is likely to form a membrane-spanning helix. As is typical for type Ia integral membrane proteins, five of the six amino acids that follow this hydrophobic region (aa 677 to 682) are basic (*15*). The sequence encoded by aa 708 to 1025 contains a putative intracellular protein kinase catalytic domain. This region carries the 11 subdomains and all 15 invariant amino acids diagnostic of protein kinases. XA21 contains sequences in subdomains VI (consensus DLKPEN) and VIII [consensus G(T/S)XX(Y/F)XAPE] that are indicative of serine-threonine (as opposed to tyrosine) specificity (*16*).

For identification of proteins similar to XA21, a database search with the predicted XA21 polypeptide sequence was performed (*17*). XA21 showed greatest similarity to RLK5 (54.7% similarity; 35.5% identity) and TMK1 (53.1%; 29.8%), *Arabidopsis* receptor–like kinase proteins of unknown function—showing conservation with both the LRR domain and the STK domain (*18*). The putative extracellular domain revealed similarity to the tomato resistance gene *Cf9* (54.9%; 32.5%), to wheat proteins of unknown function (53.1%; 33.3%) (Fig. 1C), to the bean polygalacturonase–inhibiting protein (53.1%; 29.5%), and to the *Antirrhinum* FIL2 protein (55.7%; 35.8%) (*14*, *19*). The STK domain of XA21 is most similar to that of the SRK-related proteins (56.3%; 33.7% to SRK-29) and to the tomato *Pto* resistance gene product (56.5%; 30.6%) (*3*, *20*) (Fig. 1C).

We have observed that *Xa21*-hybridizing sequences are clustered at the *Xa21* locus (*6*, *21*). Similarly, the *Pto*, *M*, *Cf9*, and *N* resistance genes are all members of clustered gene families (*4*). In addition to the *Xa21* gene family, there are at least seven major genes and one quantitative trait locus encoding resistance to viral, bacterial, and fungal pathogens clustered within 30 cM on chromosome 11 (*22*). Sequence comparison of the members of the *Xa21* gene family and linked disease resistance genes may lead to clues regarding evolution of plant disease resistance.

Ultimately, a better knowledge of the signal transduction pathway in plants should facilitate the design of new approaches to disease control. For instance, intra- and intergeneric transfer of disease resistance genes may provide an additional tool for breeders in combatting plant disease. Our results indicate that *Xa21* functions in diverse rice species and subspecies to reduce infection, and that genes encoding subsequent signal transduction components are conserved. Results from other laboratories suggest that intergeneric transfer of disease resistance genes may provide resistance to pathogens. For example, the tomato *Pto* and *Cf9* resistance genes function in the closely related genus tobacco to confer a defense response to strains of *Pseudomonas syringae* pv. *tabaci* and to the AVR9 peptide, respectively (*23*). Because the phytobacterial genus *Xanthomonas* infects virtually every crop species worldwide, future engineering and transfer of the *Xa21* resistance gene may help reduce loss resulting from *Xanthomonas* infection in recipient species.

REFERENCES AND NOTES

1. W. J. Fantl, D. E. Johnson, L. T. Williams, *Annu. Rev. Biochem.* **62**, 453 (1993).
2. J. C. Walker, *Plant Mol. Biol.* **26**, 363 (1994).
3. J. C. Stein, B. Howlett, D. C. Boyes, M. E. Nasrallah, J. B. Nasrallah, *Proc. Natl. Acad. Sci. U.S.A.* **88**, 8816 (1991).
4. B. J. Staskawicz, F. M. Ausubel, B. J. Baker, J. G. Ellis, J. D. G. Jones, *Science* **268**, 661 (1995).
5. G. S. Khush, D. J. Mackill, G. S. Sidhu, in *Bacterial Blight of Rice* (International Rice Research Institute, Manila, Philippines, 1989), p. 207.
6. P. Ronald *et al.*, *Mol. Gen. Genet.* **236**, 113 (1992).
7. G.-L. Wang, T. E. Holsten, W.-Y. Song, H.-P. Wang, P. C. Ronald, *Plant J.* **7**, 525 (1995).
8. Partial fill-in reactions were performed on Sal I–digested cosmid vector pHC80 and Sau 3AI partially digested DNA from the resistant *O. sativa* ssp. Indica line, IRBB21. After ligation, the clones were in vitro packaged with GigapackII and transfected into competent *Escherichia coli* NM554 (Stratagene). High-density filters containing 60,000 clones were screened with RG103 as described (*7*).
9. Transformation was essentially as described [S. Zhang *et al.*, *Plant Cell Rep.*, in press] except that embryogenic calli or suspension cells (or both) were used.
10. DNA and cDNA sequencing were done with the Sequitherm Long Read Cycle Sequencing Kit (Epicentre Technologies) in combination with the LI-COR Model 4000L Automated Sequencer (Li-COR). Fifteen overlapping subclones were constructed for sequencing the 9.6-kb Kpn I DNA fragment.
11. For cDNA analysis, poly(A) RNA was isolated as described (*12*). Reverse transcriptase–polymerase chain reaction (RT- PCR; Stratagene) reactions were carried out according to the manufacturer's instructions with the following modification: 35 PCR cycles were used with denaturing at 94°C for 1 min, annealing at 56°C for 2 min, and extension at 72°C for 4 min. A 5′ primer corresponding to a 24-bp sequence precisely preceding the start codon and a 3′ primer corresponding to a 24-bp sequence located 23 bp downstream of the stop codon were used to amplify the cDNA. The 3.1-kb RT-PCR product was cloned into the PCRII vector with the use of the TA cloning kit (Invitrogen). The sequence of the full-length cDNA corresponded precisely to the pC822 deduced ORF and to the deduced ORF of a PCR product amplified from the transgenic line 106-17 with the same primer pair. There is an in-frame stop codon 14 amino acids upstream from the XA21 start codon. There are no other start codons in this 14-aa sequence. In addition to this full-length cDNA isolated from the transgenic plant, three partial cDNAs corresponding to the *Xa21* gene were isolated from IRBB21. A lambda ZapII cDNA library was constructed by the random priming method (Stratagene). Plaques (1.5×10^6) were screened with DNA fragments amplified from the *Xa21* LRR and kinase regions.
12. Six-week-old IRBB21 greenhouse-grown plants were inoculated with *Xoo* race 6 strain PXO99Az. Five to 8 cM of leaf tip tissue harvested 0, 3, 6, 12, 24, 48, 72, 96, 120, 144, and 168 hours after inoculation were pooled. Ten micrograms of polyadenylated RNA were prepared by oligo(dT) cellulose chromatography, separated on a 1.4% formaldehyde gel, transferred to Hybond N membranes (Amersham), and hybridized with RG103.
13. G. von Heijne, *Mol. Biol.* **184,** 99 (1985).
14. D. A. Jones, C. M. Thomas, K. E. Hammond-Kosack, P. J. Balint-Kurti, J. D. G. Jones, *Science* **266**, 789 (1994).
15. J. N. Weinstein, R. Blumnethal, J. van Renswoude, C. Kempf, R. D. Klausner, *J. Membr. Biol.* **66**, 203 (1982).
16. S. K. Hanks, A. M. Quinn, T. Hunter, *Sciençe* **241**, 42 (1988).
17. The SwissProt, PIR(R), GenPept, and GenPept updates databases were searched with the BlastP algorithm [S. F. Altschul, G.Warren, W. Miller, E. W. Myers, D. J. Lipman, *J. Mol. Biol.* **215**, 403 (1990)]. The Wisconsin sequence analysis program GAP was used to calculate the percent identity and similarity of proteins.
18. C. Chang *et al.*, *Plant Cell* **4,** 1263 (1992); J. C. Walker, *Plant J.* **3**, 451 (1993).
19. GenBank numbers s49302, s49299, s49301, s49300; M. Steinmayr, P. Motte, H. Sommer, H. Saedler, A. Schwarz-Sommer, *Plant J.* **5**, 459 (1994); P. Toubart *et al.*, *ibid.* **2**, 367 (1992).
20. GenBank number Z30211; G. B. Martin *et al.*, *Science* **262**, 1432 (1993).
21. J. Jiang, B. S. Gill, G. Wang, P. C. Ronald, D. C. Ward, *Proc. Natl. Acad. Sci. U.S.A.* **92**, 4487 (1995).
22. D. Mackill and M. Bonman *Phytopathology* **82**, 746 (1992); Y. Hayano-Saito, paper presented at the 15th International Botanical Congress, Yokohama, Japan, 28 August 1993; G. Wang *et al.*, *Genetics* **136**, 1421 (1994); A. Yoshimura, T. W. Mew, G. S. Khush, T. Omura, *Phytopathology* **73**, 1409 (1983).
23. B. Staskawicz, J. Jones, G. Martin, personal communication.
24. H. E. Kauffman, A. P. K. Reddy, S. P. Y. Hsieh, S. D. Merca, *Plant Dis. Rep.* **57**, 537 (1973).

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