using the expression s_k to convey the meaning m_l. Similarly, $Q_{kl} = \mu(s_k, m_l)/\Sigma_j \mu(s_k, m_j) = \mu(m_l, s_k)$ is the probability of interpreting the expression s_k to mean m_l. The need to communicate meanings is related to events in the shared world of the linguistic community. Therefore, one can define a measure σ on the set of possible meanings (Σ_2^*) that speakers and hearers might wish to communicate with each other. Given this, we can define $a_{ij} = tr[P^{(i)}\Lambda(Q^{(j)})^T]$, where Λ is a diagonal matrix such that $\Lambda_{ii} = \sigma(m_i)$. This is the probability that an event occurs and is successfully communicated from a user of G_i to a user of G_j. $F(G_i,G_i)$ is the probability that users of G_i will have a successful communication with each other. Communication might break down in one of two ways: (i) poverty: an event happens whose meaning cannot be encoded by G_i, and (ii) ambiguity: an event happens whose meaning has an ambiguous encoding in G_i leading to a possibility of misunderstanding. Thus, $F(G_i,G_i)$ is a number between 0 and 1 and denotes the fitness of G_i. Maximum fitness, $F(G_i,G_i) = 1$, is achieved by grammars that can express every possible meaning (zero poverty) and have no ambiguities.

33. To study the effect of finite (small) population sizes, the deterministic Eq. 1 is replaced by a stochastic process. In this case, we observe that the population adopts one of the candidate grammars (that admits a stable equilibrium) for some time and then jumps to another equilibrium. If the candidate grammars differ in their fitness, then the stochastic process performs an evolutionary optimization on the space of all grammars.
34. Denote by x_i the fraction of individuals who use G_i of universal grammar U_1; denote by y_i the fraction of individuals who use G_i of universal grammar U_2. U_1 and U_2 contain, respectively, n_1 and n_2 candidate grammars. Some of the candidate grammars can be part of both universal grammars. The universal grammars, U_1 and U_2, can also differ in the number of sample sentences, b_1 and b_2, that are being considered. Therefore, we have to take into account the rate of producing offspring with grammatical communication; this rate is given by the declining function $r(b)$. An alternative interpretation is that $r(b)$ describes the cost that is associated with learning. The dynamics are described by

$$\dot{x}_i = r(b_1)\sum_{j=1}^{n_1} x_j f_j^{(1)} Q_{ji}^{(1)} - \phi x_i \qquad i = 1, \ldots, n_1$$

$$\dot{y}_i = r(b_2)\sum_{j=1}^{n_2} y_j^{(2)} f_j^{(2)} Q_{ji}^{(2)} - \phi y_i \qquad i = 1, \ldots, n_2$$

We have $f_i^{(m)} = \Sigma_{j=1}^{n_1} x_j F(G_i^{(m)}, G_j^{(1)}) + \Sigma_{j=1}^{n_2} y_j F(G_i^{(m)}, G_j^{(2)})$, $m \in \{1, 2\}$, and $\phi = \Sigma_{i=1}^{n_1} f_i^{(1)} x_i r(b_1) + \Sigma_{i=1}^{n_2} f_i^{(2)} y_i r(b_2)$, where the superscripts 1 and 2 refer to U_1 and U_2, respectively.

35. In general, it is advantageous to reduce the size of the search space, because a smaller n leads to a larger accuracy of grammar acquisition. The situation is more complex, however. Consider two universal grammars U_1 and U_2 with $n_1 > n_2$. Suppose U_1 is resident and U_2 is an invading mutant. If n_1 exceeds the coherence threshold, then U_2 will always outcompete U_1. If n_1 is below the coherence threshold, then U_2 can only invade if the specific grammar adopted by the population of U_1 speakers is also part of U_2; otherwise U_1 can resist invasion by U_2. The selective difference between U_1 and U_2 is small if both n_1 and n_2 values are either well above or well below the coherence threshold. Hence, selection is strongest close to the coherence threshold (if $n_1 \approx n_2$).
36. This problem has been solved before, in a different context. How many words, N, can be stably maintained in a population if each child hears b words during its language acquisition period and has a probability, ρ, to memorize a new word after one encounter? The answer is $N < b\rho$ [M. A. Nowak, J. B. Plotkin, V. A. A. Jansen, *Nature* **404**, 495 (2000)].
37. The implicit assumption here is, of course, that the rule-based grammars can generate at least these N sentence types. In a principles and parameters framework, condition 4 implies that the number of parameters k has to be less than N.
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Broad-Spectrum Mildew Resistance in *Arabidopsis thaliana* Mediated by *RPW8*

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Plant disease resistance (*R*) genes control the recognition of specific pathogens and activate subsequent defense responses. We show that the *Arabidopsis thaliana* locus *RESISTANCE TO POWDERY MILDEW8* (*RPW8*) contains two naturally polymorphic, dominant *R* genes, *RPW8.1* and *RPW8.2*, which individually control resistance to a broad range of powdery mildew pathogens. Although the predicted RPW8.1 and RPW8.2 proteins are different from the previously characterized *R* proteins, they induce localized, salicylic acid–dependent defenses similar to those induced by *R* genes that control specific resistance. Apparently, broad-spectrum resistance mediated by *RPW8* uses the same mechanisms as specific resistance.

The majority of characterized *R* genes participate in gene-for-gene interactions, in which the *R* product appears to act as a receptor that recognizes a product of the corresponding avirulence (*Avr*) gene from the pathogen, inducing defense responses. The *R* gene–mediated defenses typically involve a rapid, localized necrosis, or hypersensitive response (HR), at the site of infection, and the localized formation of antimicrobial chemicals and proteins that restrict growth of the pathogen (*1, 2*). Many crops rely on *R* genes for resistance to specific pathogens, but resistance fails in the presence of strains of the pathogen that lack the corresponding *Avr* genes. Broad-spectrum disease resistance is therefore desirable, and has been achieved through the use of recessive mutations (*3*); a challenge is to develop broad-spectrum resistance with dominant *R* genes (*4*). More than 20 of the *R* genes that confer specific resistance have been characterized and they form five classes of protein with differing combinations of five conserved structural motifs. With the exception of Hm1, a toxin reductase, and Pto, a protein kinase, the characterized *R* proteins contain a leucine-rich repeat (LRR) motif believed to specify recognition (*1, 5, 6*). All of the characterized *A. thaliana* *R* genes encode proteins with motifs for a nucleotide-binding site (NBS) and an LRR, and similar *R* genes have been isolated from other plant species (*1*). Probes for conserved sequences in NBS-LRR motifs have detected numerous homologs in the genomes of crop plants (*7*), and more than 100 in the genome of *A. thaliana* (*8*). The *A. thaliana* loci *RPW7* and *RPW8* from accession Ms-0 map to the same interval on chromosome 3, and confer resistance to the powdery mildew pathogens *Erysiphe cruciferarum* UEA1 and *E. cichoracearum* UCSC1, respectively (*9*). During the mapping of *RPW8*, we discovered that this locus segregated from the mapped NBS-LRR *R*-gene homologs (*8*), suggesting the presence of a different type of resistance gene, which we characterize here.

We genetically mapped *RPW8* to a fragment of genomic DNA from Ms-0 in cosmid B6 (Fig. 1A) (*10*), and confirmed that Col-0 plants containing the B6 transgene (T-B6) were resistant to *E. cichoracearum* UCSC1 (Fig. 2A) (*10*). The B6 DNA sequence (*11*) revealed three open reading frames (ORFs) (Fig. 1A) encoding a serine/threonine protein kinase 2 (*SPK-2*) (GenBank accession number S56718) and two uncharacterized genes, which we named *MSC1* and *MSC2*. Subclones of B6 in a plant transformation vector were introduced into Col-0 plants by *Agrobacterium*-mediated transformation (*11, 12*), and those that contained either *MSC1* or *MSC2*, or both of these ORFs, conferred resistance to *E. cichoracearum* UCSC1 (Fig. 1A). This indicated that *RPW8* comprises two independently acting genes, *MSC1* and *MSC2*, which we therefore renamed *RPW8.1* and *RPW8.2*, respectively. Ms-0 cDNAs for *RPW8.1* and *RPW8.2* (*13*) were introduced

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into accession Col-0 plants under control of the highly active cauliflower mosaic virus *35S* promoter (*11, 12*), and transgenic plants T-*35S::RPW8.1* and T-*35S::RPW8.2*, homozygous for the corresponding transgene, were resistant to *E. cichoracearum* UCSC1, whereas T-*35S::SKP-2* plants used as controls were susceptible (Fig. 1B) (*10*).

The characterized *R* genes normally control resistance to only a narrow range of isolates of a species of pathogen (*1*). To examine the range of pathogens controlled by *RPW8*, we inoculated *A. thaliana* Col-0 plants transgenic for *RPW8* with a selection of pathogens virulent on accession Col-0. Transgenic plants T-B6, T-*35s::RPW8.1*, and T-*35s::RPW8.2* were resistant to all of the tested powdery mildew pathogens of *Arabidopsis*. These included 15 isolates of *E. cichoracearum*, *E. cruciferarum* isolate UEA1 [indicating that *RPW7* (*9*) is also determined by *RPW8.1* and *RPW8.2*], *E. orontii* isolate MGH, representing three distinct species (*14*), and *Oidium lycopersici* isolate Oxford. A feature of these powdery mildew pathogens is that they each have a very wide host range. For example, *E. cichoracearum* UCSC1 also infects members of the Cucurbitaceae (*15*), *E. cruciferarum* UEA1 also infects members of the Brassicaceae (*9, 15*), and *O. lycopersici* Oxford attacks members of the Cucurbitaceae and the Solanaceae (*16*). T-B6 plants were susceptible to the other pathogens tested, including *Peronospora parasitica* Noco2 (*10*), the cauliflower mosaic virus, and the bacterium *Pseudomonas syringae* pv *tomato* DC3000 (*17*).

The genomic sequence of *RPW8.1* contained a single 197-nucleotide (nt) intron and produced a 711-nt transcript with 444 nt of predicted coding sequence, and the genomic sequence of *RPW8.2* contained a 128-nt intron and produced a 798-nt transcript with 522 nt of predicted coding sequence (GenBank accession number AF273059). Northern analysis indicated that *RPW8.1* and *RPW8.2* were expressed constitutively, but increased after infection with *E. cichoracearum* UCSC1 (*10*). The predicted proteins RPW8.1 and RPW8.2 had 45.2% sequence identity (*10*), and were relatively small (molecular weights 17,000 and 19,973, respectively) and basic (isoelectric points of 9.46 and 10.05, respectively). Their sequences predicted an NH_2-terminal transmembrane (TM) domain, or possibly a signal peptide, and a coiled-coil (CC) domain (*10*). Although RPW8.1 and RPW8.2 had no significant homology to any characterized proteins, they had 22 and 33% identity, and 51 and 62% similarity, respectively, to a 130 amino acid sequence forming the NH_2-terminus of the predicted product of resistance protein–like gene BAB08633, which has an NBS and an LRR motif at residues 171 to 751.

We compared the DNA sequences of the *RPW8.1* and *RPW8.2* alleles from seven *A. thaliana* accessions resistant and susceptible to *E. cichoracearum* UCSC1 (*11, 15*). Resistance of accessions Kas-1 and Wa-1 to *E. cichoracearum* UCSC1 is controlled by loci *RPW10* and *RPW13*, respectively, both of which map to the *RPW8* locus (*18*). The DNA sequences of the *RPW8.1* and *RPW8.2* alleles from Kas-1 and from Wa-1 were identical to those from Ms-0. Accessions Ler, Nd-0, and Ws-0 were moderately susceptible, and the DNA sequences of their *RPW8.1* and *RPW8.2* alleles were predicted to encode proteins with 90.5 to 98.3% amino acid sequence identity to those encoded by the Ms-0 alleles. There is an insertion at the 3′ end of the allele for *RPW8.1* in Nd-0, and a premature stop at the 3′ end of the alleles for *RPW8.2* in Ws-0 and Sy-0. Other differences from Ms-0 were

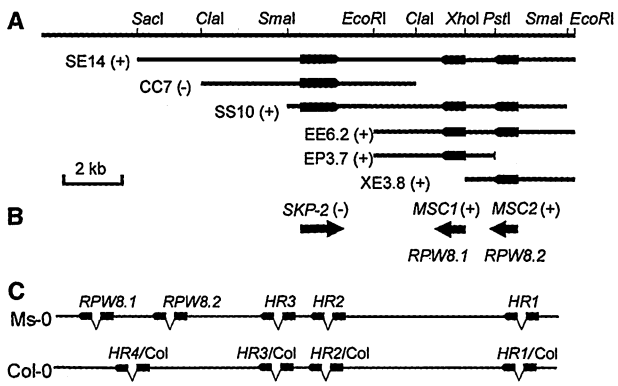

Fig. 1. Isolation of the *RPW8* locus. (**A**) Restriction sites in cosmid B6 used for making the indicated subclones (*11*). Subclones introduced into Col-0 by *Agrobacterium*-mediated transformation are marked (+) if they conferred resistance to *E. cichoracearum* UCSC1, and (–) if they did not. ORFs detected in the B6 sequence (*11*) are shown as thick lines. Only subclones containing either or both *MSC1* and *MSC2* conferred resistance. (**B**) cDNAs for *RPW8.1* and *RPW8.2* (*13*), and for *SKP-2* were cloned in vector pKMB (*26*) under control of the highly active viral *35S* promoter, and introduced into *A. thaliana* Col-0 by *Agrobacterium*-mediated transformation (*11, 12*). Plants containing the *35S::RPW8.1* or *35S::RPW8.2* transgenes were resistant (*10*), indicated by (+), and plants containing the *35S::SKP-2* transgene were susceptible, indicated by (–), to *E. cichoracearum* UCSC1. (**C**) Organization of *RPW8.1* and *RPW8.2* and homologs in accessions Ms-0 and Col-0. Exons are indicated as thick lines; each gene has a single intron indicated by a gap.

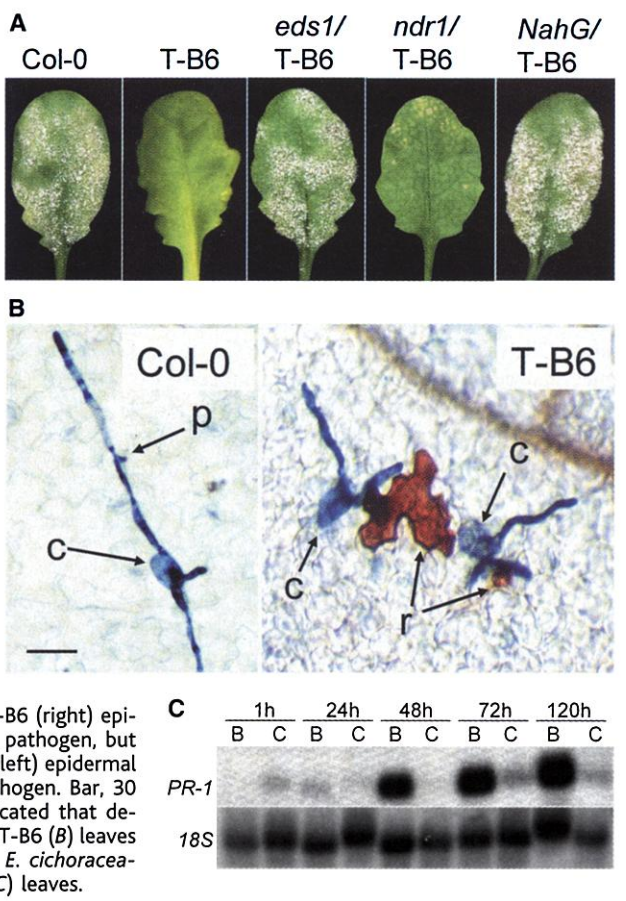

Fig. 2. Analysis of *RPW8*-mediated resistance of *A. thaliana* to *E. cichoracearum*. (**A**) Ten days after plants were inoculated with conidia from *E. cichoracearum* UCSC1 (*27*), (left to right) white mycelium and vegetative spore masses of the pathogen had developed on leaves of accession Col-0, which was susceptible, but not on leaves of Col-0 plants containing the *RPW8* transgene, T-B6, which was resistant. T-B6 plants in the *eds1-2/eds1-2* background (*20*) were susceptible, T-B6 plants in the *ndr1-1/ndr1-1* mutant background (*20*) were resistant, and T-B6 plants containing the *NahG* transgene encoding salicylate hydroxylase were susceptible. (**B**) Germinated conidia (c, blue) on surfaces of leaves 30 hours after inoculation. Hydrogen peroxide formation was detected with diamino benzidine (*28*) as a brown reaction product (r) in T-B6 (right) epidermal cells penetrated by the pathogen, but could not be detected in Col-0 (left) epidermal cells penetrated (p) by the pathogen. Bar, 30 μm. (**C**) Northern analysis indicated that defense gene *PR-1* was induced in T-B6 (*B*) leaves 48 hours after inoculation with *E. cichoracearum* UCSC1, but not in Col-0 (*C*) leaves.

conserved: for example, in RPW8.2, two (T64S and D116G) of the five amino acid differences occurred in all four susceptible accessions (*10*). Accession Col-0 is extremely susceptible to mildew and we could not detect alleles of either *RPW8.1* or *RPW8.2* by Southern analysis (*10*). However, the corresponding sequence in Col-0 [bacterial artificial chromosome (BAC) T20E23] revealed a single gene, CAB62477, predicted to encode a protein with 52 and 33% amino acid sequence identity to RPW8.1 and RPW8.2, respectively.

Some *R*-gene loci consist of clusters of homologs that represent sites of rapidly evolving *R*-gene specificity (*19*). Analysis of the nucleotide sequence of cosmid J4-2 from Ms-0 (*10*) revealed that *RPW8* was linked to three ORFs with 62.6 to 82.3% DNA sequence identity to *RPW8.1* and *RPW8.2*. These three paralogs were named Homologous to *RPW8* 1 (*HR1*), *HR2*, and *HR3* (GenBank accession number AF273059). They are closely related (99.4 to 99.9% DNA sequence identity) to their orthologs CAB 62474, CAB62475, and CAB62476, respectively, in BAC T20E23 from accession Col-0 (*10*); we named CAB62477 as *HR4* (Fig. 1C). A recombination breakpoint between *RPW8.2* and *HR3*, detected with marker 3B3-L (*10*) indicated that *HR1*, *HR2*, and *HR3* did not confer resistance to the powdery mildews. The *RPW8* locus of Ms-0 therefore contained five *RPW8* paralogs, and the locus in Col-0 contained four (Fig. 1C).

A majority of the characterized *A. thaliana R* genes mediate resistance mechanisms that involve an HR, induced H_2O_2 formation and expression of the pathogenesis-related (PR) genes, and require salicylic acid (*1*) and the signal pathway genes *EDS1* or *NDR1* (*20*). We examined whether these features occur in *RPW8*-mediated resistance. Defense responses in T-B6 plants could be detected 30 hours after inoculation with *E. cichoracearum* UCSC1, as the formation of H_2O_2 in epidermal cells penetrated by the pathogen (Fig. 2B); the penetrated cells subsequently collapsed, forming microscopic lesions characteristic of the HR and there was no further growth of the pathogen (Fig. 2A) (*9*). By contrast there was no evidence of cellular H_2O_2 when Col-0 epidermal cells were penetrated by the pathogen (Fig. 2B), which grew to form masses of white mycelia and conidia on the leaf surface in 10 days (Fig. 2A). *PR-1* transcripts accumulated in T-B6 plants 48 hours after inoculation, but not in inoculated Col-0 plants (Fig. 2B). Resistance was abolished in T-B6 plants containing salicylate hydroxylase, which converts salicylic acid to catechol (Fig. 2A). Resistance was also abolished in plants containing the *RPW8* transgene from T-B6 crossed into the *eds1-2/eds1-2* mutant background, but not in the *ndr1-1/ndr1-1* mutant background (Fig. 2A), indicating that *EDS1*, but not *NDR1*, was required for resistance.

RPW8.1 and *RPW8.2* are similar to many of the characterized *R* genes in that they are naturally occurring, polymorphic, dominant alleles (*1*), occur in a gene cluster (*1, 19, 21*), and induce defense response associated with HR. Unlike other characterized *R* genes, *RPW8.1* and *RPW8.2* conferred resistance to a wide range of powdery mildew diseases of *A. thaliana*, reminiscent of that conferred by recessive alleles at the barley *MLO* locus (*3*). However, *mlo*-based resistance involves a spontaneous-lesion phenotype and cell wall apposition in epidermal tissues before pathogen attack (*3*), which is different from the HR phenotype triggered by *R* genes (*1, 9*). None of the tested powdery mildew pathogens could overcome *RPW8*-mediated resistance, indicating that *RPW8*-mediated resistance does not involve a gene-for-gene interaction (*2, 21, 22*), or possibly that each of these pathogens has an *Avr* gene that interacts with *RPW8*. The NH_2-termini of NBS-LRR *R* proteins play a role in the regulation of cell death (*23*), defense pathway-signaling (*20*), and in the determination of specificity (*24*). RPW8.1 and RPW8.2 had limited homology to the predicted NH_2-terminus of an NBS-LRR *R*-like protein. *RPW8.1* and *RPW8.2* may therefore have some NBS-LRR *R* gene functions, to recognize powdery mildew pathogens, or to recruit NBS-LRR *R* gene–mediated defenses for resistance. Alternatively RPW8.1 and RPW8.2 may interact with NBS-LRR *R* proteins to initiate resistance responses, possibly as targets for pathogen virulence factors that are guarded by NBS-LRR *R* proteins as has been proposed for Pto (*25*).

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