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MOS2, a Protein Containing G-Patch and KOW Motifs, Is Essential for Innate Immunity in *Arabidopsis thaliana*

Yuelin Zhang,[1,2] Yu Ti Cheng,[1] Dongling Bi,[2] Kristoffer Palma,[1] and Xin Li[1,3,*]

[1]Michael Smith Laboratories
University of British Columbia
2185 East Mall
Room 301
Vancouver, BC V6T 1Z4
Canada

[2]National Institute of Biological Sciences
Zhongguancun Life Science Park
7 Science Park Road
Beijing 102206
People's Republic of China

[3]Department of Botany
University of British Columbia
6270 University Blvd.
Room 3529
Vancouver, BC V6T 1Z4
Canada

Summary

Innate immunity is critical for sensing and defending against microbial infections in multicellular organisms. In plants, disease resistance genes (*R* genes) play central roles in recognizing pathogens and initiating downstream defense cascades [1]. *Arabidopsis SNC1* encodes a TIR-NBS-LRR-type R protein with a similar structure to nucleotide binding oligomerization domain (Nod) proteins in animals [2, 3]. A point mutation in the region between the NBS and LRR of SNC1 results in constitutive activation of defense responses in the *snc1* mutant. Here, we report the identification and characterization of *mos2-1*, a mutant suppressing the constitutive defense responses in *snc1*. Analysis of *mos2* single mutants indicated that it is not only required for resistance specified by multiple *R* genes, but also for basal resistance. Map-based cloning of *MOS2* revealed that it encodes a novel nuclear protein that contains one G-patch and two KOW domains and has homologs across the animal kingdom. The presence of both G-patch and KOW domains in the MOS2 protein suggests that it probably functions as an RNA binding protein critical for plant innate immunity [4, 5]. Our discovery on the biological functions of MOS2 will shed light on functions of the MOS2 homologs in animals, where they may also play important roles in innate immunity.

Results

Identification of *mos2-1*

Mutations in *Arabidopsis NPR1* block the induction of pathogenesis-related (*PR*) genes and pathogen resistance by salicylic acid (SA) [6]. Originally identified as a suppressor of *npr1*, *snc1* (*suppressor of npr1-1, constitutive 1*) mutant plants constitutively express *PR* genes, accumulate high levels of SA, and are resistant to the virulent bacterial pathogen *Pseudomonas syringae maculicola* ES4326 (*P.s.m.* ES4326) and the oomycete pathogen *Peronospora parasitica* Noco2 (*P.p.* Noco2) [7]. The mutant plants also have reduced stature and constitutively express the *pBGL2-GUS* reporter gene [8]. To dissect the signaling pathways activated by *snc1*, we identified 15 loci that are required for the constitutive disease resistance in *snc1* [9]. *snc1 npr1-1* plants carrying a suppressor mutation, designated *mos2-1*, were identified by their intermediate size (Figure S1A, available in the Supplemental Data available with this article online). The constitutive *pBGL2-GUS* expression is completely abolished in *mos2-1 snc1 npr1-1* plants (Supplemental Figure S1B), and suppression of the constitutive *PR-2* expression in *mos2-1 snc1 npr1-1* was further confirmed by RT-PCR (Figure 1A).

The *mos2-1 snc1 npr1-1* triple mutant was backcrossed with *snc1 npr1-1* to determine the genetic nature of the *mos2-1* mutation. The F1 plants had the morphology and size of *snc1 npr1-1* plants, indicating that *mos2-1* is recessive. Progeny from the selfed F1 plants were plated on MS medium, and the *pBGL2-GUS* expression was examined on 20-day-old seedlings. Among 19 plants, 13 had the typical *snc1*-like GUS staining (data not shown), indicating that *mos2-1* is a single recessive mutation (expected ratio 3:1, χ^2 = 0.44; $p > 0.1$).

mos2-1 Suppresses the Constitutive Disease Resistance to Pathogens in *snc1 npr1-1*

To determine if the *mos2-1* mutation affects resistance in *snc1 npr1-1* plants, the *mos2-1 snc1 npr1-1* mutant plants were inoculated with the virulent pathogens *P.s.m.* ES4326 and *P.p.* Noco2. As shown in Figure 1B, *snc1 npr1-1* plants were resistant to *P.p.* Noco2, whereas *mos2-1 snc1 npr1-1* plants were as susceptible as *npr1-1*. Similarly, *snc1 npr1-1* plants showed enhanced resistance against *P.s.m.* ES4326, whereas *mos2-1 snc1 npr1-1* plants were as susceptible as *npr1-1* (Figure 1C). Thus, *mos2-1* completely suppresses the constitutive disease resistance in *snc1*.

mos2-1 Partially Suppresses the High SA Level in *snc1 npr1-1*

Because *snc1* accumulates high SA, the endogenous SA level in the *mos2-1 snc1 npr1-1* plants was determined to check whether the SA pathway is affected in the triple mutant. As shown in Figures 1D and 1E, *mos2-1* dramatically reduced the SA level in *snc1 npr1-1*, but the SA level was still higher than that in *npr1-1*, indicating that *mos2-1 snc1 npr1-1* plants are capable of synthesizing SA.

To determine whether the suppression of the constitutive *PR-2* expression is due to reduced SA accumulation, *mos2-1 snc1 npr1-1* plants grown on MS medium, with or without the SA analog 2,6-dichloroisonicotinic acid (INA), were analyzed for *PR-2* expression. As

*Correspondence: xinli@interchange.ubc.ca

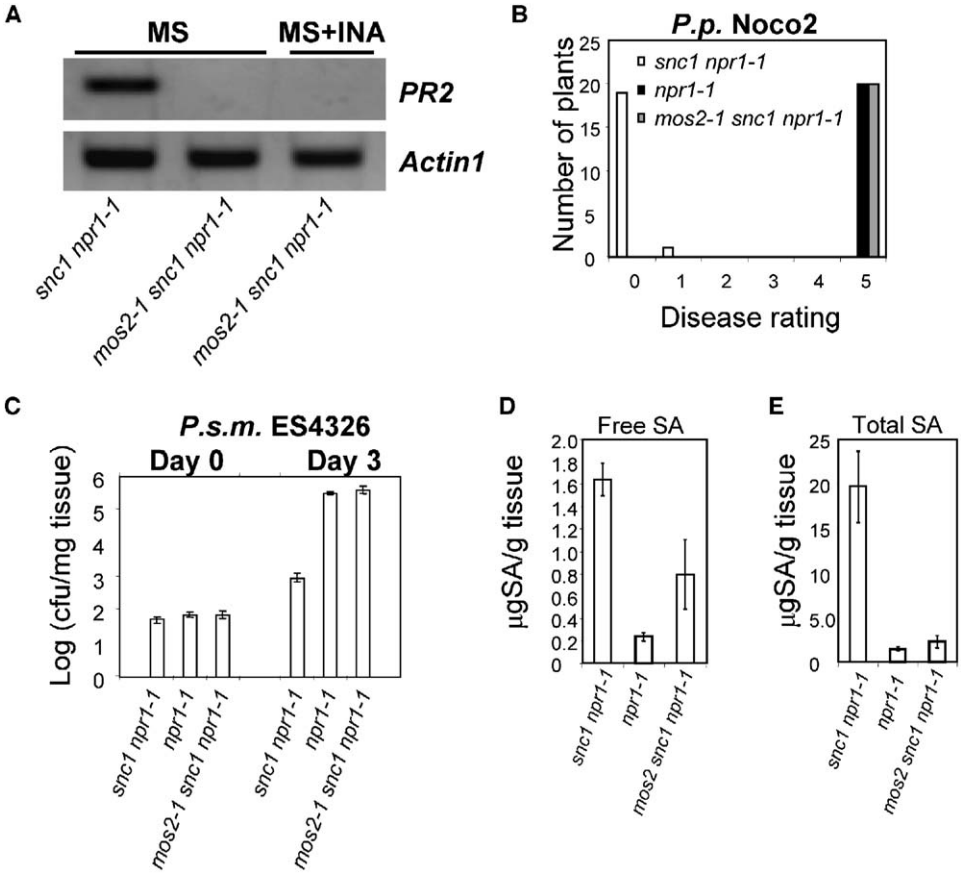

Figure 1. Characterization of *mos2-1 snc1 npr1-1*

(A) *mos2-1* suppresses constitutive expression of *PR-2* in *snc1 npr1-1*. *PR-2* expression in *snc1 npr1-1* and *mos2-1 snc1 npr1-1* plants grown on MS, and *mos2 snc1 npr1-1* plants grown on MS supplemented with 0.05 mM INA. RNAs were extracted from 20-day-old plants and reverse transcribed to obtain cDNA. The cDNA samples were normalized by real-time PCR with the *Actin1* probe. *PR2* and *Actin1* were amplified by 35 cycles of PCR using equal amounts of total cDNA, and the PCR products were analyzed by agarose gel electrophoresis and ethidium bromide staining.

(B) Growth of *P.p.* Noco2 on *snc1 npr1-1*, *npr1-1*, and *mos2-1 snc1 npr1-1*. Two-week-old seedlings were sprayed with *P.p.* Noco2 at a concentration of 50,000 spores per ml of water. The infection was scored 7 days after inoculation with the following disease rating: 0, no conidiophores on the plants; 1, no more than five conidiophores per infected leaf; 2, 6–20 conidiophores on a few of the infected leaves; 3, 6–20 conidiophores on most of the infected leaves; 4, five or more conidiophores on all infected leaves; 5, 20 or more conidiophores on all infected leaves.

(C) Growth of *P.s.m.* ES4326 on *snc1 npr1-1*, *npr1-1*, and *mos2-1 snc1 npr1-1* plants. Leaves of four-week-old plants were infiltrated with a bacterial suspension at OD_{600} = 0.0001. At days 0 and 3, leaf discs within the infiltrated area were taken to measure the bacterial growth in the leaves. The values presented are averages of four replicates ± standard deviations. Abbreviation: cfu, colony forming units.

Free (D) and total (E) SA level in *snc1 npr1-1*, *npr1-1*, and *mos2-1 snc1 npr1-1* plants.

Plants were grown on soil and leaf tissue was collected for SA extraction. SA levels were measured with high-pressure liquid chromatography using a procedure previously described [21]. The values presented are averages of four replicates ± standard deviations. All experiments were repeated at least twice with similar results.

shown in Figure 1A, INA-treatment cannot restore *PR-2* expression in *mos2-1 snc1 npr1-1* plants.

Mutations in genes that are required for SA synthesis such as *EDS5* and *SID2* render plants more susceptible to both virulent and avirulent pathogens [10–12]. Although *eds5-3* completely blocks the increased SA synthesis in the *snc1 npr1-1* double mutant, it only has a marginal effect on *snc1 npr1-1* morphology and has no effect on constitutive *PR-2* expression. This suggests that an SA-independent pathway is activated in *snc1 npr1-1* and that constitutive expression of *PR-2* in *snc1 npr1-1* is a hallmark of the activation of this pathway [3]. Because *PR-2* expression in *snc1 npr1-1* is completely blocked by the *mos2-1* mutation and the SA level is only partially affected by *mos2-1* mutation in *snc1 npr1-1*, MOS2 is most likely a component controlling the SA-independent pathway. Other preliminary results indicate that at least one other *MOS* gene is also required for the SA-independent pathway (K.P. and X.L., unpublished data). The failure of INA to restore *PR-2* expression further suggests that suppression of constitutive *PR-2* expression by *mos2-1* is due to its effect on the SA-independent pathway rather than reduced SA accumulation.

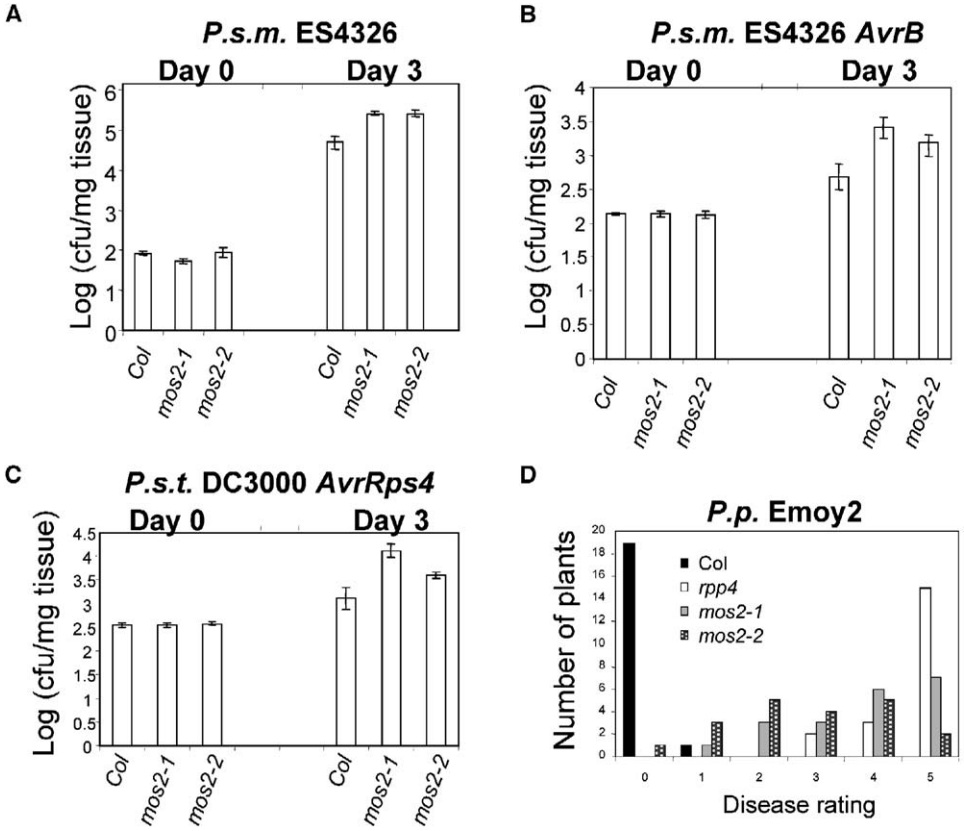

Figure 2. Both Basal Defense and *R* Gene-Mediated Resistance Are Compromised by Mutations in *MOS2*

All experiments were repeated at least once with similar results.

(A) Growth of *P.s.m.* ES4326 on wild-type Col, *mos2-1* and *mos2-2* plants. Leaves of four-week-old plants were infiltrated with a bacterial suspension at OD_{600} = 0.0001. The bacterial growth was determined as described in Figure 1.

(B) Growth of *P.s.m.* ES4326 *AvrB* on wild-type Col, *mos2-1*, and *mos2-2* plants.

(C) Growth of *P.s.t.* DC3000 *AvrRps4* on wild-type Col, *mos2-1*, and *mos2-2* plants. For (B) and (C), Leaves of four-week-old plants were infiltrated with a bacterial suspension at OD_{600} = 0.001. The bacterial growth was determined as described in Figure 1.

(D) Growth of *P.p*. Emoy2 on Col wild-type, *rpp4*, *mos2-1*, and *mos2-2* plants. *rpp4* is a mutant with the complete *RPP4* cluster deleted in Col which was isolated as one of the revertant alleles of *snc1* [3]. Two-week-old seedlings were sprayed with *P.p.* Emoy2 at a concentration of 100,000 spores per ml of water. The infection was scored 7 days after inoculation per the disease rating as described in Figure 1.

Error bars indicate the standard deviation from the average.

MOS2 Is Required for Basal Resistance against *P.s.m.* ES4326

To identify the *mos2-1* single mutant, *mos2-1 snc1 npr1-1* was crossed with wild-type plants. In the F2, allele-specific primers were used to find individuals that were *mos2-1* homozygous but wild-type at the *SNC1* and *NPR1* loci. Lines homozygous for *mos2-1* with no *snc1* and *npr1-1* mutation were kept as *mos2-1* single mutants. To check whether *MOS2* is important for the basal defense response, *mos2-1* and *mos2-2* plants were inoculated with *P.s.m.* ES4326 at a density of OD_{600} = 0.0001. Although *P.s.m.* ES4326 normally does not cause disease symptoms on wild-type plants at this low dose, disease symptoms were observed on the *mos2* single mutants. When the bacterial titers in the plants were determined, approximately 10-fold more bacteria accumulated in both *mos2-1* and *mos2-2* plants than in wild-type plants (Figure 2A), suggesting that *MOS2* is required for basal resistance.

MOS2 Is Required for *R* Gene-Mediated Resistance specified by *RPM1*, *RPS4*, and *RPP4*

When *mos2-1* plants were infiltrated with *P.s.m.* ES4326 *AvrB* and *P.s.t.* DC3000 *AvrRps4*, which carry Avr genes corresponding to the resistance genes *RPM1* and *RPS4* in the Columbia ecotype, respectively, the bacteria accumulated to approximately one order of magnitude higher level than in wild-type (Figures 2B and 2C). This indicates that *MOS2* is required for resistance mediated by *RPM1* and *RPS4*. However, mutations in *MOS2* only partially block the resistance mediated by *RPM1* and *RPS4*, since bacterial growth in mutants such as *rar1* and *eds1* that completely block *R* gene-mediated resistance is normally 100- to 1000-fold higher than that in the wild-type plants [13, 14].

When *mos2-1* plants were challenged with *P.p.* Emoy2, which is specifically recognized by *RPP4*, the resistance was abolished by *mos2-1* (Figure 2D). The infected leaves also exhibited trailing necrosis, a phe-

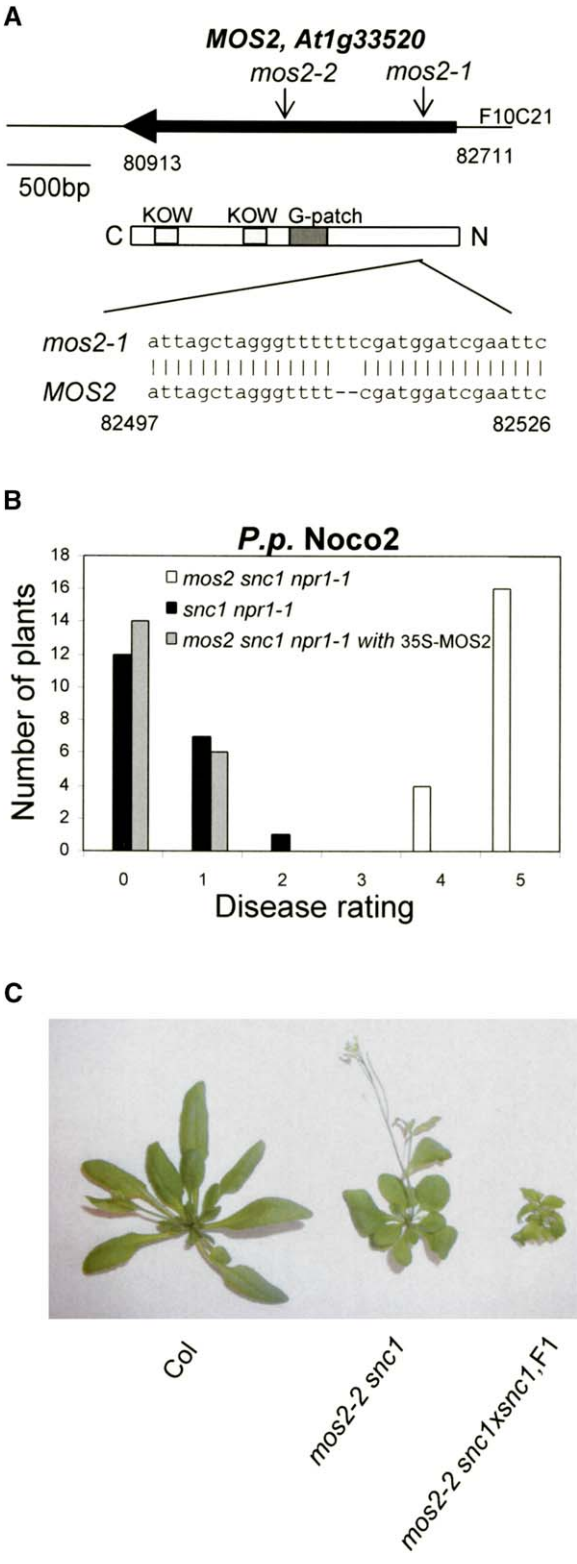

Figure 3. Map-Based Cloning of *MOS2*

(A) Gene structure of *MOS2* on F10C21 and mutation positions in *mos2-1* (2 bp insertion) and *mos2-2* (T-DNA insertion). The G-patch and KOW motifs are depicted in the schematic representation of the protein below. Abbreviations: N, N terminus; C, C terminus.

(B) Complementation of *mos2-1* by *MOS2* (*At1g33520*) cDNA. Growth of *P.p.* Noco2 on *mos2-1 snc1 npr1-1* and *mos2-1 snc1 npr1-1* transformed with *35S-MOS2*.

(C) Suppression of *snc1* morphology by *mos2-2*.

nomenon typically observed when *R* genes mediated resistance is compromised (Supplemental Figure S2) [14]. Thus, *MOS2* is also required for *RPP4* mediated resistance to *P.p.* Emoy2. As expected, resistance to *P.s.m.* ES4326 *AvrB*, *P.s.t.* DC3000 *AvrRps4*, and *P.p.* Emoy2 was also compromised in *mos2-2* plants (Figures 2B–2D).

Map-Based Cloning of *mos2*

To map the *mos2-1* mutation, *mos2-1 snc1 npr1-1* was crossed with Ler-*snc1*, a line with the *snc1* mutation introgressed into Ler [9]. Crude mapping located *mos2-1* between BAC F17F8 and F28J9 on chromosome 1. Fine mapping of *mos2-1* showed that the mutation is in a region between the markers T16O9 and T1E4 (Figure S3). The interval between these two markers is about 120 kb.

To identify the molecular lesion in *mos2-1*, a set of PCR fragments covering the region between marker T16O9 and T1E4 were amplified from *mos2-1 snc1 npr1-1* and sequenced. Comparing the sequences from the mutant with the *Arabidopsis* genome sequence revealed a two base-pair insertion in *At1g33520* (Figure 3A). The mutation in *mos2-1* causes an early frameshift and most likely abolishes the activity of the protein completely. When the expression level of *MOS2* was compared between Col wild-type and *snc1* plants, no significant changes in transcript level were observed (data not shown).

Two approaches were taken to confirm that the insertion found in *mos2-1* causes the suppression of the *snc1* phenotype. *MOS2* cDNA was first cloned under the control of CaMV 35S promoter and transformed into *mos2-1 snc1 npr1-1*. Nine out of 10 transformants obtained showed the morphology of *snc1* mutants (data not shown). As shown in Figure 3B, the progeny of the transformants also were resistant to *P.p*. Noco2, indicating that the *MOS2* cDNA complemented the mutation caused by *mos2-1*. A complementation test between *mos2-1 snc1 npr1-1* and *mos2-2*, an insertion allele of *MOS2* (SALK_033856) [15], was also carried out. When the two mutants were crossed, the F1 plants had the *mos2-1* morphology. Among 200 F2 plants analyzed, none displayed *snc1* morphology, indicating that *mos2-2* did not complement *mos2-1*. Furthermore, we identified *snc1 mos2-2* double mutants from the F2 plants and the *snc1* morphological phenotypes were also suppressed in these plants. When *snc1 mos2-2* was crossed with *snc1*, the F1 plants displayed *snc1* morphology (Figure 3C), indicating that *mos2-2* is a recessive mutation like *mos2-1*. Taken together, we concluded that *MOS2* is *At1g33520*.

To determine the subcellular localization of the MOS2 protein, MOS2 was fused to the N terminus of GFP under the control of its own promoter and transformed into *mos2-1* mutant plants. GFP fluorescence was de-

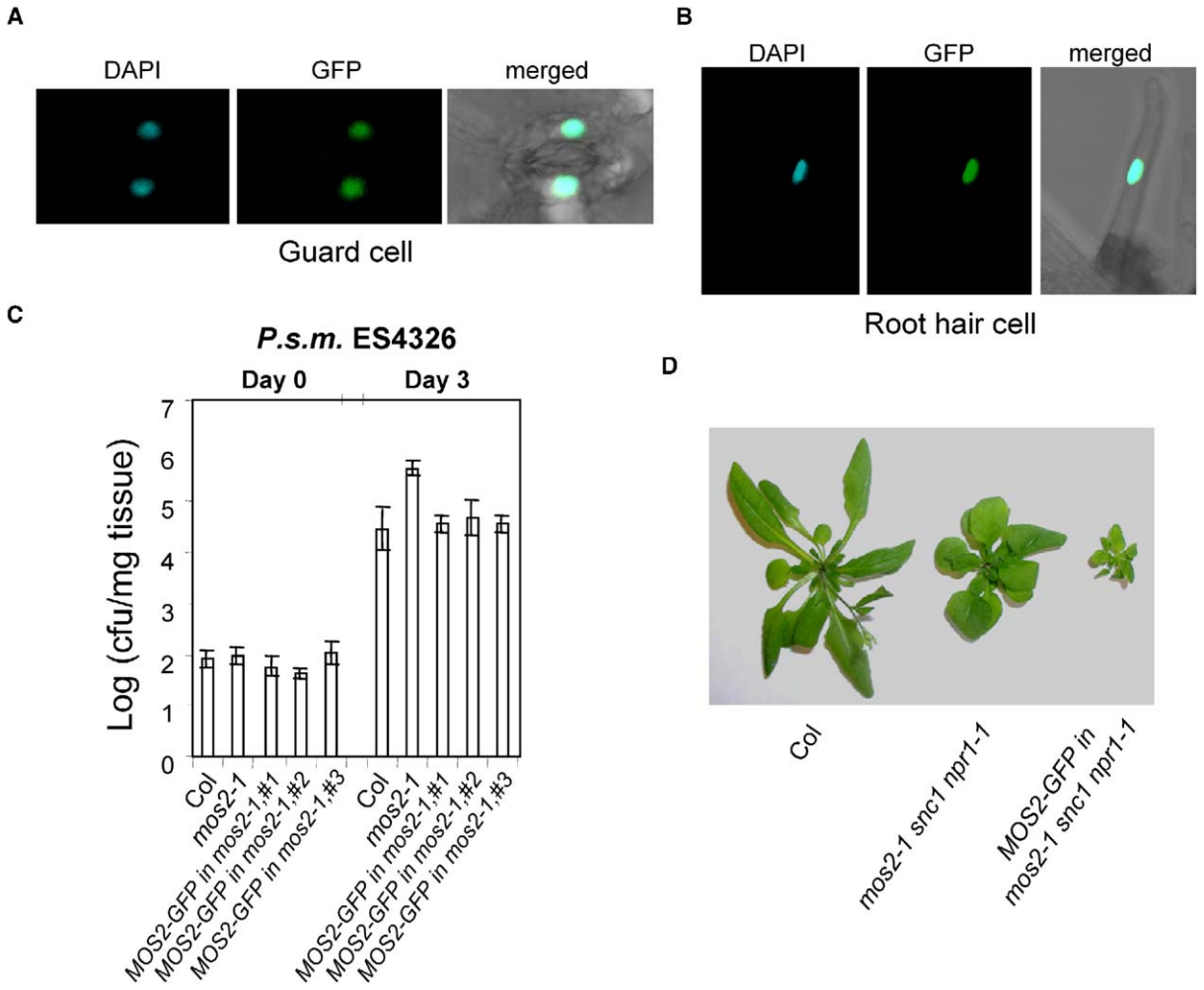

Figure 4. Analysis of Transgenic Plants Expressing MOS2-GFP under Its Own Promoter

(A) Guard cell and (B) root hair cell of two-week-old Col wild-type seedlings expressing the MOS2-GFP fusion protein were examined with confocal microscopy as described previously [22]. The picture shows a representative view of multiple transformants. DAPI staining was carried simultaneously to show the nuclear compartment.

(C) Growth of *P.s.m.* ES4326 on *mos2-1* and three independent *mos2-1* lines transformed with MOS2-GFP.

(D) Morphology of Col wild-type, *mos2-1 snc1 npr1-1*, and *mos2-1 snc1 npr1-1* transformed with *MOS2-GFP*.

Error bars indicate the standard deviation from the average.

tected mainly in the nucleus in transgenic plants expressing the MOS2-GFP fusion protein (Figures 4A and 4B). Not only *mos2-1* plants expressing the *MOS2-GFP* lost the enhanced disease susceptibility phenotype (Figure 4C), *mos2-1 snc1 npr1-1* transformed with *MOS2-GFP* also reverted to *snc1* morphology (Figure 4D), indicating that the fusion protein is functional in plants and MOS2 is most likely a nuclear protein. Analysis of the MOS2 protein with PSORT (http://psort.nibb.ac.jp/) revealed one putative nuclear localization motif in MOS2 (between residues 291–309).

MOS2 encodes a protein with unknown function but containing three recognizable signatures, one G-patch domain (amino acids 156–200) near the center, and two KOW motifs at the C terminus (amino acids 232–264 and 402–435). BLAST analysis showed that MOS2 has a single homolog in *Arabidopsis* and also homologs in human, mouse, and *C. elegans* (Figure S4). It is interesting to note that the G-patch and the KOW motifs are clearly conserved among these proteins. Genome-wide RNAi studies in *C. elegans* indicate that silencing of the *C. elegans MOS2* homolog causes an embryo lethal phenotype, suggesting that this gene is essential for the worm [16].

G-patch is a conserved domain found in type D retroviral polyproteins and eukaryotic RNA-processing proteins such as the 45-KDa splicing factor (SPF45) [4], suggesting that this domain mediates RNA-protein interactions. The G-patch domain located within the C-terminal extension of betaretroviral aspartic proteinase was recently shown to bind single-stranded nucleic acids [17]. The KOW motif was originally identified based on its conservation among three families of ribosomal proteins and the microbial transcriptional modu-

lator NusG [5]. Analysis of the crystal structure of NusG not only provided direct evidence that the KOW motif binds to RNA, but it also revealed that the KOW module is a close structural homolog of the tudor protein-protein interaction motif [18]. The putative protein binding sites differ from the area implicated in nucleic acid interactions, suggesting that the KOW motif may also facilitate protein-protein interactions.

In *Arabidopsis*, *PAD4* and *EDS1* are required for resistance specified by TIR-NBS-LRR-type *R* genes, whereas NDR1 is required for resistance specified by CC-NBS-LRR-type *R* genes [19, 20]. Mutations in *PAD4* and *EDS1* completely suppress the elevated SA level and the constitutive pathogen resistance in *snc1* [3, 7]. In contrast, *mos2-1* only partially affects the SA accumulation in *snc1 npr1-1*. We also found that *mos2-1 snc1* is less susceptible to *P.s.m.* ES4326 than *mos2-1* (data not shown), suggesting that resistance pathways independent of *MOS2* remain active in the *mos2-1 snc1* double mutant and contribute to the residual resistance. Unlike PAD4 and EDS1, MOS2 is not only required for resistance mediated by TIR-NBS-LRR-type *R* genes such as *RPP4* and *RPS4*, it is also required for resistance specified by *RPM1*, a CC-NBS-LRR-type *R* gene. Thus MOS2 appears to act downstream of *NDR1*, *PAD4*, and *EDS1* and is a critical component for a resistance pathway shared between TIR-NBS-LRR and CC-NBS-LRR-type *R* genes.

RNA binding proteins are emerging as important players in the regulation of various biological processes. The presence of both G-patch and KOW domains in MOS2 suggests that it may function as an RNA binding protein. Coupled with our previous finding that MOS3, which encodes a putative nucleoporin similar to human Nup96 that is important for mRNA export, is also required for the constitutive *PR* gene expression and disease resistance in *snc1* [9], we conclude that RNA processing may play important roles in the regulation of innate immunity in plants. In the future, it would be interesting to determine whether MOS2 homologs play similar roles in innate immunity in animals.

Supplemental Data

Supplemental Data include four figures and one table and are available with this article online at http://www.current-biology.com/cgi/content/full/15/21/1936/DC1/.

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GenBank Accession Numbers

Sequence data from this article have been deposited with the EMBL/GenBank data libraries under accession number DQ202264.