Supporting Information

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SI Materials and Methods

Plant Material and Growth Conditions. All plants were of *Arabidopsis thaliana* Columbia (Col-0) background except for *della* quintuple mutant in Landsberg *erecta* (*Ler*). Origins of mutant and transgenic lines are as follows: *scl3-1* (SALK_002516), *gai* (SALK_082622), and *rga* (SALK_089146), the SALK T-DNA lines (http://signal.salk.edu) (1, 2); *ga1-3* (3, 4); *scr-5* and *shr-6* (SALK_002744) (1, 5, 6); QC25 (7); pSHR::SHR-GFP (8); pSCR::GFP-SCR (7, 9); pCO2::H_2B-YFP (10); *gai rga rgl1 rgl2 rgl3* (*della* quintuple mutant) (11); pSCR::gai-GR-YFP (12); pCYCB1::GUS (13). PCR-based genotyping was performed to confirm homozygous plants from genetic crosses. Sequence information of PCR primers used for genotyping is listed in Table S1. Seeds were surface sterilized in 5% sodium hypochlorite and 0.15% Tween-20 for 3 min, rinsed in sterile water, imbibed at 4 °C in the dark in sterile water for 3 d, planted onto 0.5× MS agar plates (0.5× Murashige-Skoog salt mixture; 0.5 mM 2-(N-morpholino) ethanesulfonic acid (MES), pH 5.7–5.8; 1% sucrose; 1% agar). Plates were incubated vertically under continuous light at 22 °C as previously described (1, 6). When gibberellin (GA)-deficient mutant background was used (*ga1-3*, *scl3 ga1-3*, and *35S*::*SCL3 ga1-3*), surface-sterilized seeds were imbibed in 100 μM of GA_3 (Duchefa) for 5 d, and rinsed thoroughly in sterile water before planting. For adult plants, seedlings grown under continuous light were transferred to soil and grown under long-day conditions (16-h light/8-h dark cycles). Plants in the GA-deficient *ga1-3* background (*ga1-3*, *scl3 ga1-3*, and *35S*::*SCL3 ga1-3*) were sprayed with 100 μM of GA_3 (Duchefa) three times a week.

Treatment and Root Assay. For root growth analysis, ~36–40 h postgermination (hpg) seeds were individually transferred to new MS agar plates, as described previously (14), supplemented with GA_3 (stock concentration 100 mM in ethanol, Duchefa), PAC (stock concentration 100 mM in ethanol, Duchefa), or DEX (stock concentration 10 mM in ethanol, Sigma). On the day of analysis, individually harvested seedling roots were cleared with ethanol and mounted as described below in GUS clearing method (15). The cell number in the root meristem was obtained by counting cortex cells from the quiescent center (QC) to the cell showing no signs of rapid elongation, and the elongation/differentiation zone (EDZ) was specified from the first cortex cell that exited from the meristem as described previously (14, 16). Root length was measured from digital images of the plates using ImageJ software (http://rsb.info.nih.gov/ij). Experiments were repeated three times independently, and data were analyzed using the Excel statistical package (Microsoft).

For analysis of formative ground tissue divisions, as mentioned above, ~36–40 hpg seeds were individually transferred to new MS agar plates, supplemented with either GA_3 (10 μM in final concentration), or PAC (1 μM in final concentration). For control experiments, the same batch of 36–40 hpg seeds were individually transferred to new MS agar plates. On the day of analysis, seedling roots in a given population ($n > 50$) were cleared and analyzed for additional periclinal divisions as described below.

Molecular Cloning and Transgenic Plants. To generate transcriptional and translational fusions, and overexpressors, the Gateway recombination cloning technology (Invitrogen) was used as described previously (1, 6) with minor modifications. For the *pSCL3::GUS* transcriptional fusion, ~2.5-kb upstream region from the start codon of the *SCL3* gene was cloned into the pMDC162 vector (17) by recombination reactions according to the manufacturer's instructions (Invitrogen). For the *pSCL3*::*SCL3-GFP* translational fusion, ~5-kb genomic fragment (promoter and ORF) was subcloned into pDONR221 (Invitrogen) by recombination reaction, and subsequently transferred into pMDC107 for C-terminal GFP fusion (17).

To overexpress the *SCL3* gene under the control of 35S promoter, a full length of *SCL3* ORF was amplified from Col-0 genomic DNA using Phusion high-fidelity polymerase (Finnzymes) with a pair of gene-specific primers for the *SCL3* gene. The PCR fragment was subcloned into pENTR Directional TOPO vector and subsequently transferred into the pEarleyGate100 gateway-comparable overexpression vector (18) according to the manufacturer's instructions (Invitrogen).

To generate the *pSCR::rga-GR-YFP*, the NOS terminator and the coding region of *GR-YFP* were amplified by PCR using genomic DNA from *pSCR::gai-GR-YFP* seedlings (12). The NOS terminator was subcloned into pENTR11-dual selection vector (Invitrogen), and subsequently the *GR-YFP* fragment was placed into the 5′ end of NOS terminator to create *pE11-GR-YFP*. Subsequently, a 2.5-kb *SCR* promoter fragment was placed into the 5′ end of GR-YFP to create *pE11-pSCR::GR-YFP*. The rga coding region placed downstream of the *SCR* promoter in frame with the 5′ end of GR-YFP to create a final entry vector *pE11-pSCR::rga-GR-YFP*. Sequence information of PCR primers used for plasmid construction is listed in Table S2.

The floral dip method (19) was conducted using either Col-0 or *scl3* for production of transgenic plants. The T_1 plants were selected, and subsequent homozygous T_2 plants were obtained through confirmation in the T_3 generation.

Expression Analysis. For qRT-PCR, seedlings were grown on filter paper stripe (~5 mm wide) in MS agar plates for 7 d and transferred to new MS agar plates supplemented with 10 μM of PAC or 10 μM of GA_3 for 6 h. Only roots were harvested for RNA isolation, and total RNA was isolated using an RNeasy Plant Mini kit according to the manufacturer's instructions (Qiagen). After RNA extraction, we treated the samples with RQ1 RNase free-DNase (Promega) to eliminate potential contamination of genomic DNA. The quality and quantity of the isolated RNA were inspected by both gel electrophoresis and spectrophotometry as previously described (1, 6). SYBR Premix ExTaq reagents (Takara) were used for qRT-PCR with the Mx3000P QPCR System (Stratagene). For the internal reference, the gene-specific primers for the *GAPC* gene (At3g04120) were used (20). Each experiment was conducted independently at least three times with biological replicates. The mean values of triplicates were calculated, and SDs (± SD) were indicated.

For mRNA in situ hybridization, a 961-bp fragment of *SCL3* was amplified and subcloned into pCR4 Blunt-TOPO vector (Invitrogen), designated as *pCR4-SCL3*. To generate the antisense probe, the *pCR4-SCL3* construct was linearized with the restriction enzyme SpeI, and DIG-labeled *SCL3* riboprobe was generated by in vitro transcription using DIG RNA labeling kit (Roche) according to the manufacturer's instructions. Tissue fixation and RNA hybridization was conducted using 4- to 5-d-old wild-type roots as described previously (21–23). Sequence information of PCR primers used for expression analysis is listed in Table S3.

Histology and Microscopy. To monitor the activity of GUS reporter, all GUS lines were immersed in GUS staining solution in the dark

at 37 °C to activate β-glucrunidase activity as described previously (1, 6). To restrict the diffusion of GUS blue staining, 5 mM of $K_3Fe(CN)_6$ and $K_4Fe(CN)_6$ were added, and tissues were immersed in 70% ethanol for 2 d as described previously (15). For clearing, dehydration procedure was followed through sequential ethanol series as described previously (15) with minor modifications. At the final step of dehydration, 0.7% (wt/vol) NaOH/60% (vol/vol) ethanol was supplemented for 10 min, and samples were sequentially exposed to 10% (vol/vol) glycerol/50% (vol/vol) ethanol, and 30% (vol/vol) glycerol/30% (vol/vol) ethanol. Seedlings were then mounted in 0.05% (vol/vol) Triton X-100/50% (vol/vol) glycerol.

For root sections, seedlings and GUS-stained seedlings were fixed overnight in FAA solution (10% (vol/vol) formaldehyde, 5% (vol/vol) acetic acid, and 50% (vol/vol) ethanol), and those fixed roots were embedded into 1% agarose and dehydrated by ethanol series as described previously (11). After progressing through the ethanol series (50, 70, 80, and 100% for 30 min each), agarose gel blocks with root samples were transferred to Peel-A-Way disposable embedding molds (Polysciences) as previously described (6). Plastic resin blocks were made with Technovit 7100 according to the manufacturer's instructions (Heraeus Kulzer). Serial sections (5 μm each) were generated with an HM 355S microtome (Microm). To visualize the Casparian stripe, suberin staining was conducted with root sections as described previously (23). Except for root sections with suberin staining (observed in FITC filter), all of the cleared roots were observed in differential interference contrast (DIC) optics, and images were obtained using an AxioCam MRc5 digital camera equipped with an Axio Imager.A1 microscope (Carl Zeiss). For confocal laser scanning microscopy, roots were mounted in distilled water with 10 μM of propidium iodide (Sigma), and images were obtained using Fluoview FV300 (Olympus) as described previously (6).

1. Lee M-H, et al. (2008) Large-scale analysis of the GRAS gene family in *Arabidopsis thaliana*. *Plant Mol Biol* 67:659–670.
2. Alonso JM, et al. (2003) Genome-wide insertional mutagenesis of *Arabidopsis thaliana*. *Science* 301:653–657.
3. Tyler L, et al. (2004) DELLA proteins and gibberellin-regulated seed germination and floral development in *Arabidopsis*. *Plant Physiol* 135:1008–1019.
4. Sun T-p, Kamiya Y (1994) The *Arabidopsis GA1* locus encodes the cyclase *ent*-kaurene synthetase A of gibberellin biosynthesis. *Plant Cell* 6:1509–1518.
5. Paquette AJ, Benfey PN (2005) Maturation of the ground tissue of the root is regulated by gibberellin and SCARECROW and requires SHORT-ROOT. *Plant Physiol* 138:636–640.
6. Yu N-I, et al. (2010) Characterization of SHORT-ROOT function in the *Arabidopsis* root vascular system. *Mol Cells* 30:113–119.
7. Sabatini S, Heidstra R, Wildwater M, Scheres B (2003) SCARECROW is involved in positioning the stem cell niche in the *Arabidopsis* root meristem. *Genes Dev* 17:354–358.
8. Nakajima K, Sena G, Nawy T, Benfey PN (2001) Intercellular movement of the putative transcription factor SHR in root patterning. *Nature* 413:307–311.
9. Gallagher KL, Paquette AJ, Nakajima K, Benfey PN (2004) Mechanisms regulating SHORT-ROOT intercellular movement. *Curr Biol* 14:1847–1851.
10. Heidstra R, Welch D, Scheres B (2004) Mosaic analyses using marked activation and deletion clones dissect *Arabidopsis* SCARECROW action in asymmetric cell division. *Genes Dev* 18:1964–1969.
11. Feng S, et al. (2008) Coordinated regulation of *Arabidopsis thaliana* development by light and gibberellins. *Nature* 451:475–479.
12. Ubeda-Tomás S, et al. (2008) Root growth in *Arabidopsis* requires gibberellin/DELLA signalling in the endodermis. *Nat Cell Biol* 10:625–628.
13. Donnelly PM, Bonetta D, Tsukaya H, Dengler RE, Dengler NG (1999) Cell cycling and cell enlargement in developing leaves of *Arabidopsis*. *Dev Biol* 215:407–419.
14. Ubeda-Tomás S, et al. (2009) Gibberellin signaling in the endodermis controls *Arabidopsis* root meristem size. *Curr Biol* 19:1194–1199.
15. Laplaze L, et al. (2007) Cytokinins act directly on lateral root founder cells to inhibit root initiation. *Plant Cell* 19:3889–3900.
16. Dello Ioio R, et al. (2007) Cytokinins determine *Arabidopsis* root meristem size by controlling cell differentiation. *Curr Biol* 17:678–682.
17. Curtis MD, Grossniklaus U (2003) A gateway cloning vector set for high-throughput functional analysis of genes in planta. *Plant Physiol* 133:462–469.
18. Earley KW, et al. (2006) Gateway-compatible vectors for plant functional genomics and proteomics. *Plant J* 45:616–629.
19. Clough SJ, Bent AF (1998) Floral dip: A simplified method for Agrobacterium-mediated transformation of *Arabidopsis thaliana*. *Plant J* 16:735–743.
20. Dill A, Thomas SG, Hu J, Steber CM, Sun T-p (2004) The *Arabidopsis* F-box protein SLEEPY1 targets gibberellin signaling repressors for gibberellin-induced degradation. *Plant Cell* 16:1392–1405.
21. Di Laurenzio L, et al. (1996) The *SCARECROW* gene regulates an asymmetric cell division that is essential for generating the radial organization of the *Arabidopsis* root. *Cell* 86:423–433.
22. Helariutta Y, et al. (2000) The *SHORT-ROOT* gene controls radial patterning of the *Arabidopsis* root through radial signaling. *Cell* 101:555–567.
23. Lim J, et al. (2005) Conservation and diversification of SCARECROW in maize. *Plant Mol Biol* 59:619–630.
24. Silverstone AL, Ciampaglio CN, Sun T-p (1998) The *Arabidopsis RGA* gene encodes a transcriptional regulator repressing the gibberellin signal transduction pathway. *Plant Cell* 10:155–169.
25. Frigerio M, et al. (2006) Transcriptional regulation of gibberellin metabolism genes by auxin signaling in *Arabidopsis*. *Plant Physiol* 142:553–563.

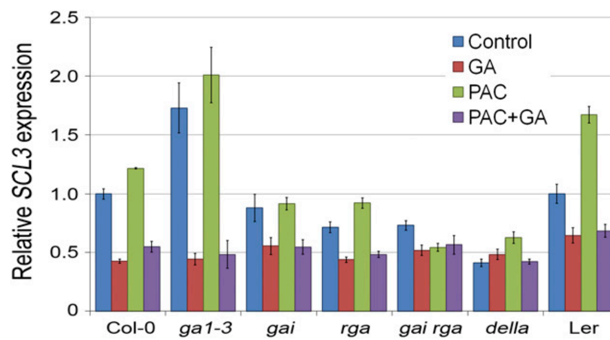

Fig. S1. Modulation of *SCL3* expression by the GA/DELLA pathway. qRT-PCR of *SCL3* transcripts in GA biosynthesis (*ga1-3*) and GA signaling (*gai*, *rga*, *gai rga*, and *della*) mutant roots in the absence or presence of exogenous GA_3 or PAC. The *SCL3* mRNA level in Col-0 grown in MS agar plates is arbitrarily set to 1. Error bars indicate SD from three biological replicates.

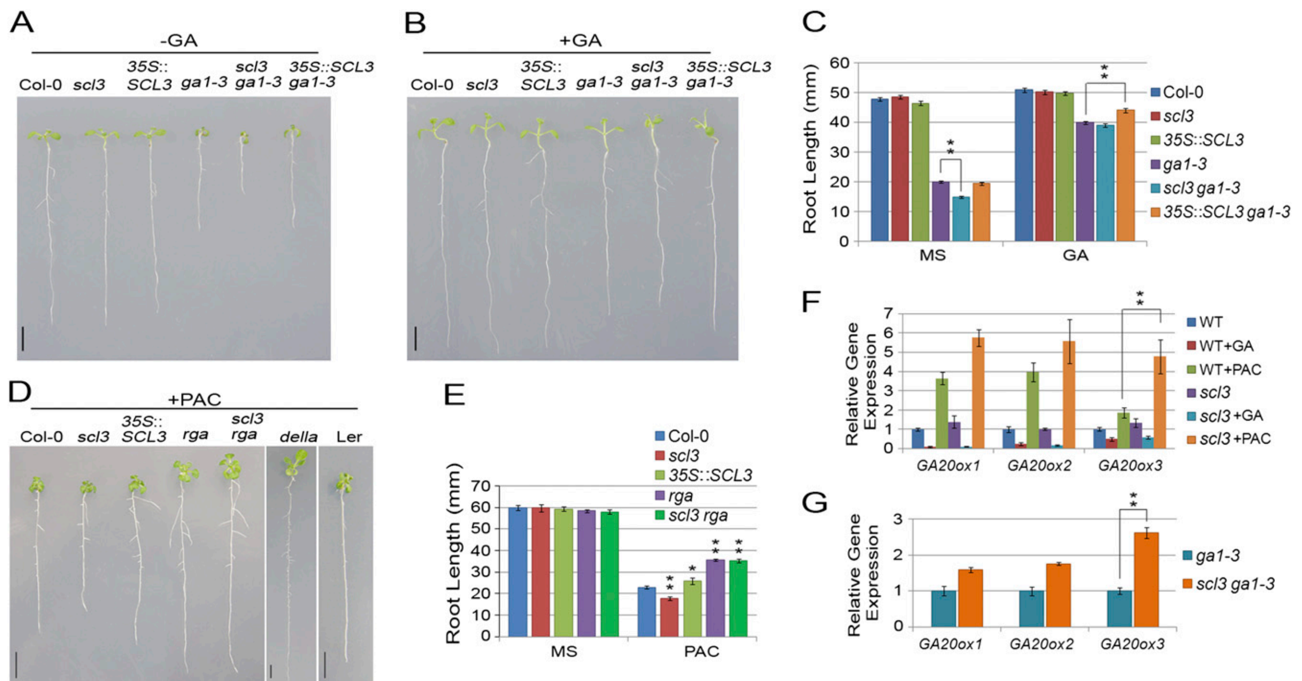

Fig. S2. SCL3 as a positive regulator in the GA pathway. (*A*–*E*) Root growth assay in the absence (*A* and *C*) or presence (*B* and *C*) of exogenous GA_3 (10 μM) or in the presence of 1 μM of PAC (*D* and *E*). (*A* and *C*) The *scl3* seedlings are indistinguishable from WT, whereas *scl3 ga1-3* double mutants show a more dwarf phenotype than *ga1-3* in the absence of exogenous GA_3. (*B* and *C*) In the presence of exogenous GA_3 (10 μM), both *scl3* and *35::SCL3* are indistinguishable from WT seedlings. (*D* and *E*) In the presence of 1 μM of PAC, root growth of *scl3* is more inhibited, whereas *35::SCL3* roots are more tolerant to PAC than WT seedlings. The *scl3 rga* double mutants are PAC resistant in a manner similar to *rga*. (*F* and *G*) Transcript levels of the GA biosynthesis genes *GA20ox1*, *GA20ox2*, and *GA20ox3* in the presence of exogenous GA_3 or PAC (*F*) and in the *ga1-3* background (*G*). In GA-deficient conditions, the expression levels of *GA20ox3* are significantly up-regulated in the *scl3* background. Statistical significance of differences was determined by Student's *t* test (*$P < 0.05$, **$P < 0.01$). (Scale bars in *A*, *B*, and *D*, 10 mm.)

Fig. S3. GA-mediated proliferative divisions in the root meristem are independent of SCL3 function. (*A*) Schematic zones of the *Arabidopsis* root along the longitudinal axis. The EDZ is defined from the boundary of the meristem zone (MZ) to the root-hypocotyl junction. (*B* and *C*) Root meristem size in the absence or presence of 1 μM of PAC at 12 dpg ($n > 20$). (*D–F*) The *CYCB1::GUS* expression in WT and *scl3* roots in the absence (*D*) or presence (*E*) of 1 μM of PAC at 7 dpg. Black arrowheads indicate the zone of prominently *CYCB1::GUS* expressing cells. (*F*) Length of the *CYCB1::GUS* expression zone. (*G* and *H*) Root meristem size in the GA-deficient *ga1-3* background at 10 dpg ($n > 20$). Error bars indicate SD from three biological replicates. (Scale bar, 30 μm.)

Fig. S4. Control of root cell elongation by SCL3 in the EDZ. (*A–D*) Root cell elongation in GA-deficient conditions by 1 μM of PAC at 12 dpg ($n > 20$) (*A* and *C*) or *ga1-3* at 10 dpg ($n > 20$) (*B* and *D*). In the presence of PAC, root cell elongation of *rga* and *scl3 rga* is resistant to PAC, indicating that *SCL3* acts downstream of *RGA* (*A* and *C*). In the GA-deficient *ga1-3* background, loss and gain of SCL3 function has opposing effects on root cell elongation (*B* and *D*). Red arrowheads demarcate a single cortex cell in the EDZ. Statistical significance of differences was determined by Student's *t* test (**$P < 0.01$). Error bars indicate SD from three biological replicates. (Scale bar, 10 μm.)

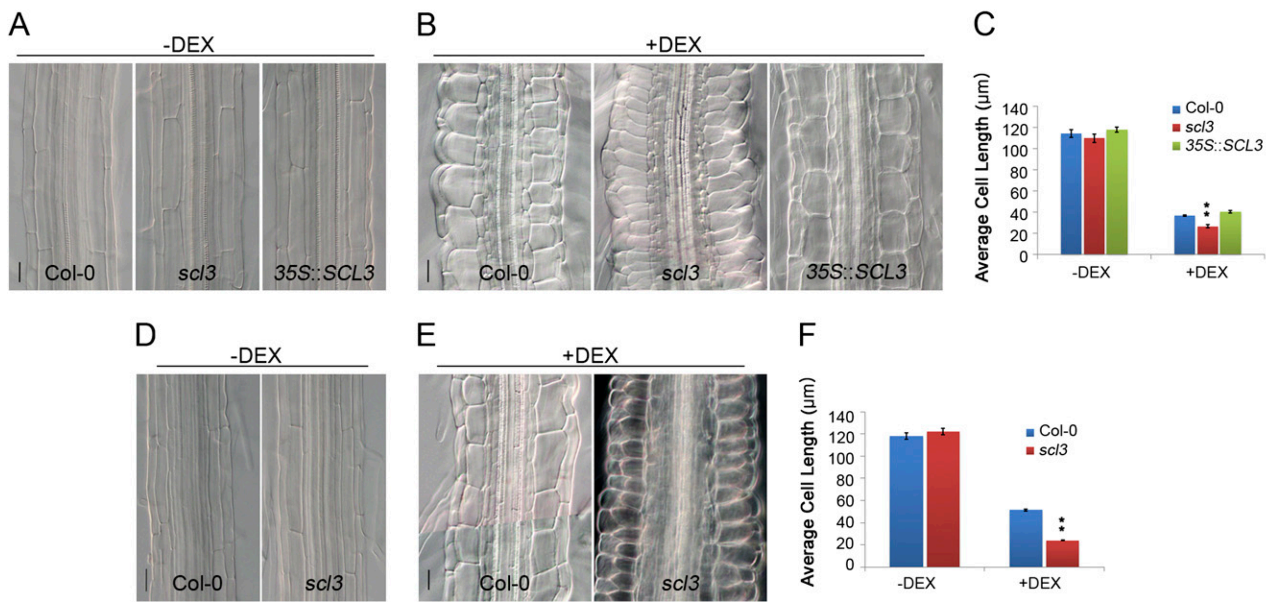

Fig. S5. SCL3–DELLA interaction controls root cell elongation in the EDZ. The *pSCR::gai-GR-YFP* seedlings in Col-0, *scl3*, and *35S::SCL3* were grown in MS agar plates for 4 d, transferred to MS agar plates supplemented with or without 10 μM of DEX, and incubated for another 3 d (*A* and *B*). In the absence of DEX (−DEX), no inhibition of cell elongation was observed (*A* and *C*). In the presence of 10 μM of DEX (+DEX), *pSCR::gai-GR-YFP* seedlings in Col-0 showed inhibition of cell elongation (*B*). Moreover, the surface of *pSCR::gai-GR-YFP* roots in *scl3* appeared bulged because the direction of cortex cell elongation was shifted perpendicularly to that of the untreated roots (*B*). In the presence of DEX, due to the inhibition of cell elongation, the average length of individual cells in the EDZ was significantly reduced in *scl3* compared with that in the WT background (*C*). Interestingly, *pSCR::gai-GR-YFP* in *35::SCL3* suppressed the inhibition of root cell elongation. Similarly, the *pSCR::rga-GR-YFP* seedlings in Col-0 and *scl3* were grown in MS agar plates for 4 d, transferred to MS agar plates supplemented with or without 10 μM of DEX, and incubated for another 3 d (*D* and *E*). In the presence of DEX, *pSCR::rga-GR-YFP* seedlings in *scl3* were more severely inhibited in cell elongation compared with *pSCR::rga-GR-YFP* seedlings in Col-0 (*E*). The image of *pSCR::rga-GR-YFP* seedlings in Col-0 was a composite of two consecutive pictures along the longitudinal root axis. The average length of individual cells in the EDZ was significantly reduced in *scl3* compared with that in the WT background (*F*). (Scale bar, 30 μm.) Statistical significance of differences was determined by Student's *t* test (error bars: SE, $^{**}P < 0.01$).

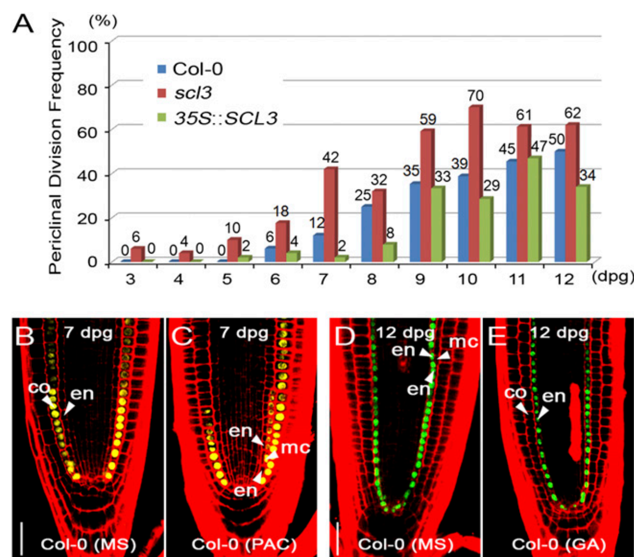

Fig. S6. SCL3 and bioactive GA levels modulate the formative division for ground tissue maturation. (*A*) Quantitative evaluation of the timing and extent of MC formation in loss and gain of SCL3 function under standard conditions. Frequency of the formative division gradually increases as the roots mature. As roots had matured, *scl3* seedlings had already undergone the periclinal division compared with WT roots, whereas *35S::SCL3* delayed the division. Thus, loss and gain of SCL3 function modulate the timing and extent of the formative ground tissue divisions. Intriguingly, a decreased frequency of MC formation conferred by *35S::SCL3* had diminished from 9 dpg onward. (*B* and *C*) Occurrence of the formative division in WT roots in the absence (*B*) or presence (*C*) of 1 μM of PAC. The PAC treatment promotes the formative division for MC formation marked by *pCO2::H$_2$B-YFP*. (*D* and *E*) The MC formation in WT roots in the absence (*D*) or presence (*E*) of 10 μM of GA$_3$. Exogenous GA$_3$ suppresses the formative division for MC formation marked by *pSCR::GFP-SCR*. (Scale bar, 30 μm.)

Fig. S7. Control of ground tissue maturation by the SCL3 and SHR/SCR modules. (*A–H*) Confocal images of *shr-6* roots in the absence (*C*, *D*, *G*, and *H*) or presence (*A*, *B*, *E*, and *F*) of SCL3 function. In the presence of 1 μM of PAC (*B* and *D*) or 10 μM of GA_3 (*F* and *H*), neither *shr-6* nor *scl3 shr* showed any formative division for MC formation in the cortex layer, confirming that SHR is required for the formative division. (*I*) Quantitative evaluation of the occurrence of MC formation in the *scr* background with loss or gain of SCL3 function. The *scl3 scr* double mutants showed an increase of the formative division, whereas *35::SCL3* in *scr* reduced the occurrence of the formative division up to 5 dpg. In addition, no effect of *35S::SCL3* on the delay in the MC formation were observed from 6 dpg onward, indicating that SCL3 regulates its own expression. (Scale bar, 30 μm.)

Fig. S8. Simplified models of SCL3–DELLA interaction in the control of GA-mediated root growth during postembryonic development. In the root, SCL3, attenuating the DELLA repressors, serves as a positive regulator to maintain GA homeostasis and thus a functional GA signaling pathway. (*A*) In the EDZ, the maintenance of GA signaling by SCL3–DELLA interaction primarily controls root cell elongation in the endodermis (red arrows), and in turn, coordinates cell elongation of other cell types. (*B*) In the MZ, the SCL3–DELLA signaling circuit for the maintenance of the GA pathway employs the SHR/SCR module to control the timing and extent of the formative ground tissue division. Bars indicate negative regulation, whereas arrows indicate positive regulation.

Table S1. Sequence information of PCR primers used for genotyping

Purpose	Name		Sequence (5′–3′)	Reference
Genotyping	scl3-1	R	ATGGTGGCTATGTTTCAAGAAG	(1)
		F_1	CACCAGTGATTCTCAAATGAG	
		F_2	TCACTTCCTGCATCTCCAAGC	
	scr-5	wt F	CTCCTCCTCCGATTCAGC	(5)
		mt F	CTCCTCCTCCGATTCAGT	
		R	TTGAGTAATCTCGCTGACA	
	shr-6	LP	TCCACCAAACCCATTCTCTAC	(6)
		RP	TCGTTGACAAACTTGTTGGCC	
	rga	R	GCGGAGGTTGCTTTGAAACTC	(1)
		F_1	AAAGCTTCGAGAATCGCTTGG	
		F_2	TAAGCGCGTGGACTAAACGAA	
	gai	R	CGACCGAAGCCAAACTAAATC	(1)
		F_1	GCCCTCGTGCCCTTTTTATAC	
		F_2	CGTAAACCAACTTCAATTGCTGTCA	
	ga1-3	wt F	TTTGGCCCAACACACAAACAAACCTT	(24)
		wt R	AAGCTTCGAACTCAAGGTTCTA	
		mt F	TGTATGCACGTTAACGATCAAT	
		mt R	TTTCTTCATACCACCTGCGTTC	
	T-DNA	LB1	GGCAATCAGCTGTTGCCCGTCTCACTGGTG	http://signal.salk.edu

Table S2. Sequence information of PCR primers used for plasmid construction

Purpose	Name		Sequence (5′–3′)	Reference
35S::SCL3	SCL3 pENTR	F	*CACC*ATGGTGGCTATGTTTCAAGAAG	This study
		R	TCACTTCCTGCATCTCCAAGC	
pSCL3::GUS	pSCL3 pENTR	F	*CACC*TTGTAACGAAGTCTGTTGTTCTC	This study
		R	TGAAGGCCAAAAGCTTGATTTTG	
pSCL3::SCL3-GFP	SCL3 pDONR	F	*GGGGACAAGTTTGTACAAAAAAGCAGGCTTC* TTGTAACGAAGTCTGTTGTTCTC	This study
		R	*GGGGACCACTTTGTACAAGAAAGCTGGGTC* CTTCCTGCATCTCCAAGCTGA	
pSCR::rga-GR-YFP	NOS Ter	F	(Xho1)CTCGAGTCCCCGATCGTTCAAACATTTGGCA	(12)
		R	(Xba1)TCTAGACGTTGATGAAGCTAATTCCCGATCT	
	GR-YFP	GR F	(Kpn1)GGTACCGCATGCGAAGCTCGAAAAACAAAGAAAAAAATC	
		GR R	(Xho1)CTCGAGTCATTTTTGATGAAACAGAAGCTTTTTGATATTTCC	
		YFP R	(Xho1)CTCGAGTTACTTGTACAGCTCGTCCATGCCG	
	pSCR	F	(Sal1)GTCGACCCTGGAAGTCCGATTGAGAGGAGAGG	
		R	(Kpn1-Not1)GGTACCGGTGCGGCCGCCCGTATCTAAGTCGTCTTCC	
	rga	F	(Not1)GCGGCCGCATGAAGAGAGATCATCACC	
		R	(Kpn1)GGTACCGTACGCCGCCGTCGAGAGTTTCC	
In situ hybridization	SCL3 Probe	F	GATTGATCTCGATGCTTCTG	This study
		R	TCACTTCCTGCATCTCCAAGC	

Gateway-compatible sequence is indicated by italics, and restriction enzyme sites are indicated by underlines.

Table S3. Sequence information of primers used for qRT-PCR

Purpose	Name		Sequence (5′–3′)	Reference
qRT-PCR	GAPC	F_2	AGCTGCTACCTACGATG	(20)
		R_2	CACACGGGAACTGTAAC	
	AtGA20ox1	F	CTTCCATCAACGTTCTCGAGC	(25)
		R	GGTTTTGAAGGTCGATGAGAGG	
	AtGA20ox2	F	AGAAACCTTCCATTGACATTCCA	
		R	AGAGATCGATGAACGGGACG	
	AtGA20ox3	F	ACTCGTCTCAAAGGCTGCAAC	
		R	GAGGCTCTCATCGACACCATG	
	RTSCL3	F	TGGCTGGTTTTGGGAATGTTCC	This study
		R	ATTACTGCGCACCCGCTCTC	