

Strigolactones Negatively Regulate Mesocotyl Elongation in Rice during Germination and Growth in Darkness

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(Received March 18, 2010; Accepted May 17, 2010)

Strigolactones (SLs) are newly discovered plant hormones that regulate plant growth and development including shoot branching. They also stimulate symbiosis with arbuscular mycorrhizal fungi. Rice has at least three genes that are involved in SL synthesis (*D10*, *D17/HTD1* and *D27*) and at least two genes that are involved in SL signaling (*D3*) and SL signaling or downstream metabolism (*D14/D88/HTD2*). We observed that mesocotyl elongation in darkness was greater in rice mutants defective in these genes than in the wild type. Exogenous application of a synthetic SL analog, GR24, rescued the phenotype of mesocotyl elongation in the SL-deficient mutants, *d10-1*, *d17-1* and *d27-1*, in a dose-dependent manner, but did not affect mesocotyl lengths of the SL-insensitive mutants, *d3-1* and *d14-1*. No significant differences in cell length were found between the *d* mutants and the wild type, except for some cells on the lower half of the *d3-1* mesocotyl that were shortened. On the other hand, the number of cells in the mesocotyls was 3- to 6-fold greater in the *d* mutants than in the wild type. Treatment with GR24 reduced the number of cells in the *d10-1* mesocotyl to the wild-type level, but did not affect the number of cells in the *d3-1* and *d14-1* mesocotyls. These findings indicate that SLs negatively regulate cell division, but not cell elongation, in the mesocotyl during germination and growth of rice in darkness.

Keywords: Cell division • *dwarf* mutants • Mesocotyl • Rice • Strigolactones.

Abbreviations: ABA, abscisic acid; CCD, carotenoid cleavage dioxygenase; GA, gibberellin; JA, jasmonate; SL, strigolactone.

Introduction

Strigolactones (SLs), a group of terpenoid lactones, were initially isolated from root exudates, that triggered germination of seeds of parasitic plants of the genus *Striga* and stimulated plant symbiosis with arbuscular mycorrhizal fungi (Cook et al. 1972, Akiyama et al. 2005, Besserer et al. 2008, Yoneyama et al. 2008). They have recently been identified as a new hormonal signal that regulates shoot branching in plants (Gomez-Roldan et al. 2008, Umehara et al. 2008). The involvement of SLs in the regulation of shoot branching, particularly in inhibiting the outgrowth of axillary buds, was assessed using a series of enhanced shoot branching mutants, which include *more axillary growth (max)* of *Arabidopsis thaliana*, *ramosus (rms)* of pea (*Pisum sativum*), *decreased apical dominance (dad)* of petunia (*Petunia hybrida*) and *dwarf (d)* or *high-tillering dwarf (htd)* of rice (*Oryza sativa*) (Dun et al. 2009, Leyser 2009, Beveridge and Kyojuka 2010). SLs also probably have other roles in growth and development, including seed germination, root growth, flower development and leaf senescence (Bradlow et al. 1988, 1990, Woo et al. 2001, Snowden et al. 2005, Yan et al. 2007). Indeed, it was recently reported that SL biosynthesis/signaling genes were expressed in mature seeds and young seedlings as well as adult plants, suggesting that their gene products play roles in germination and early development of *Arabidopsis* (Mashiguchi et al. 2009).

In rice, five *DWARF (D)* genes have been demonstrated to be responsible for SL biosynthesis (*D10*, *D17/HTD1* and *D27*), SL signaling (*D3*) and SL signaling or downstream metabolism (*D14/D88/HTD2*) (Beveridge and Kyojuka 2010), and recessive mutants defective in these genes have reduced stature and

Plant Cell Physiol. 51(7): 1136–1142 (2010) doi:10.1093/pcp/pcq075, available online at www.pcp.oxfordjournals.org

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increased numbers of tillers (Ishikawa et al. 2005). Among the SL biosynthetic genes, *D10* encodes a protein that is a type of CAROTENOID CLEAVAGE DIOXYGENASE 8 (CCD8) (Arite et al. 2007), while *D17/HTD1* encodes CCD7, another subclass of CCDs (Zou et al. 2006, Umehara et al. 2008). *D10* and *D17/HTD1* appear to catalyze sequential carotenoid cleavage reactions of precursors of SL synthesis (Umehara et al. 2008). *D27* was recently shown to encode an iron-containing protein, which works downstream of steps mediated by CCD7 and CCD8 in plastids (Lin et al. 2009). On the other hand, *D3* is involved in SL signaling and *D14/D88/HTD2* is involved in SL signaling or downstream metabolism (Umehara et al. 2008). *D3* encodes an F-box leucine-rich repeat (LRR) protein, which possibly acts in the ubiquitin-mediated degradation of a key factor of the SL signaling pathway (Ishikawa et al. 2005). *D14/D88/HTD2* encodes a protein of the α/β -hydrolase superfamily, which is proposed to function downstream of SL synthesis, as a component of hormone signaling or as an enzyme that participates in the conversion of SLs to the bioactive form (Arite et al. 2009, Gao et al. 2009, Liu et al. 2009).

The mesocotyl is a tissue located between the coleoptilar node and a basal part of the seminal root in young seedlings (Hoshikawa 1989). Its growth is controlled not only by the lengthwise elongation of each cell, but also by an increase in the number of cells by cell division. Cell elongation begins with swelling from water absorption, and then cell division is promoted and the elongation and growth of each cell occurs (Hoshikawa 1989). Previous studies have suggested that elongation of the mesocotyl is controlled by multiple genetic, developmental and environmental signals (Jones et al. 1991, Mori et al. 2002, Sawers et al. 2002, Choi et al. 2003). In general, light absorption by phytochrome in plants inhibits mesocotyl growth (Loercher 1966, Vanderhoef and Briggs 1978). Moreover, gibberellins (GAs) are known as facilitators of mesocotyl elongation, and auxin has recently been shown to down-regulate expression of a gene encoding polyamine oxidase, which was tightly correlated with inhibition of cell extension in the outer tissues of maize mesocotyl, thereby possibly enhancing elongation of the mesocotyl (Jones et al. 1991, Laurenzi et al. 1999, Cona et al. 2003). On the other hand, mesocotyl elongation seems to be inhibited by endogenous abscisic acid (ABA) in maize (Saab et al. 1992) and by endogenous jasmonate (JA) in rice (Riemann et al. 2003).

In this study, we found that mesocotyl elongation in dark-grown rice seedlings was higher in the SL-related *d* mutants than in the wild type. To understand how SLs affect mesocotyl elongation in dark-grown rice seedlings, we examined (i) whether the phenotype of the mesocotyl elongation in the *d* mutants was rescued by treatment with GR24, a synthetic SL analog, and (ii) whether cell division and/or cell elongation during mesocotyl growth contributed to the enhanced mesocotyl elongation of the *d* mutants. Possible roles of SLs in mesocotyl elongation in dark-grown rice seedling are discussed.

Results

Phenotype of rice SL mutants

After growing in darkness for 8 d, mesocotyls of SL-deficient (*d10-1*, *d17-1* and *d27-1*) and SL-insensitive (*d3-1* and *d14-1*) mutants were at least 2- to 5-fold longer than those in the wild type (cv. Shikari) (Fig. 1A–C). Interestingly, the *d3-1* mesocotyls were ~2-fold longer than the mesocotyls of the other mutants (Fig. 1C). On the other hand, the lengths of coleoptiles were not significantly different between the mutants and the wild type, except for the *d3-1* coleoptile, which was only 40% as long as the other mutants and the wild type (Fig. 1D). When the seedlings were grown under light conditions, however, the mesocotyl and coleoptile lengths of the mutants were comparable to those of the wild type (data not shown), suggesting that the enhanced mesocotyl elongation in all the mutants and the reduced coleoptile

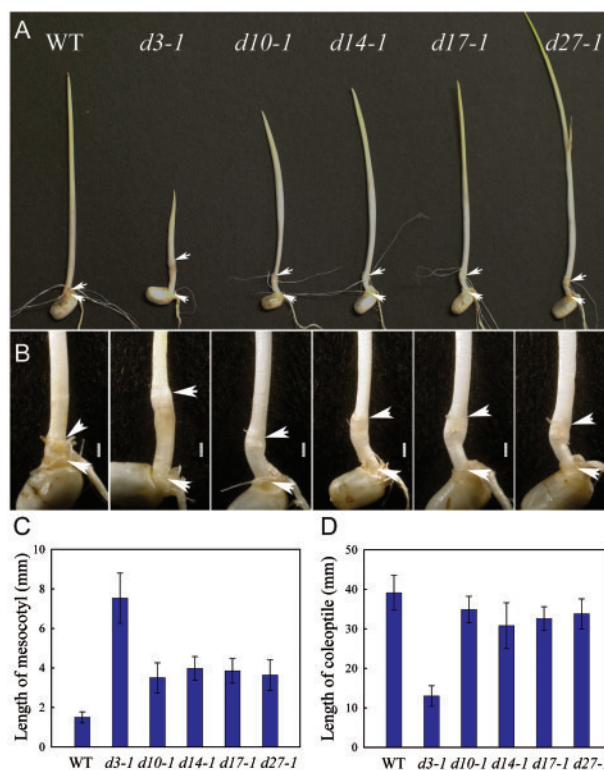


Fig. 1 Comparison of the morphology of *d* mutant and wild-type seedlings grown in the dark. (A) Phenotypes of seedlings of *d* mutants and the wild type (WT) when germinated and grown for 8 d under darkness. Upper arrows and lower arrows indicate the positions of the coleoptilar node and a basal part of the seminal root, respectively. The mesocotyl is the tissues between the two arrows. Bars = 1 cm. (B) Higher magnification of the mesocotyl regions in (A). Some roots were removed to clearly see the mesocotyls. Bars = 1 mm. (C) Length of mesocotyls of 8-day-old seedlings. Values are mean \pm SD, $n = 15$. (D) Length of coleoptiles of 8-day-old seedlings. Values are mean \pm SD, $n = 15$.

elongation in the *d3-1* mutant occur only under dark conditions.

Effect of SL on mesocotyl length of rice *d* mutants

To examine the role of SLs in mesocotyl elongation, seedlings were germinated and grown on agar plates containing different concentrations (0, 0.01, 0.1 and 1 μ M) of GR24, a synthetic SL analog, for 8 d in darkness. GR24 did not affect mesocotyl elongation of the wild type but decreased the lengths of mesocotyls of SL-deficient mutants (*d10-1*, *d17-1* and *d27-1*) in a dose-dependent manner (Fig. 2). At a GR24 concentration of 1 μ M, the lengths of mesocotyls were almost the same between the SL-deficient mutants and the wild type. On the other hand, the lengths of mesocotyls of SL-insensitive mutants (*d3-1* and *d14-1*) were not changed by treatment with GR24 (Fig. 2).

When growth was observed over time, the mesocotyls of mutant and wild-type seedlings began to elongate after 1 d and continued to elongate to 2 d at the same rate (Fig. 3, blue curves). Subsequently, mesocotyl elongation slowed and almost stopped in the wild type but continued in *d3-1*, *d10-1* and *d14-1* (Fig. 3, blue curves). GR24 (1 μ M) did not affect mesocotyl elongation of the wild type or the SL-insensitive mutants (*d3-1* and *d14-1*) but it decreased the mesocotyl length of SL-deficient *d10-1*, making it similar to the wild type (Fig. 3, red curves).

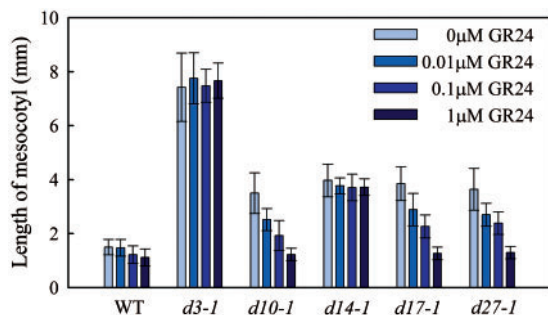


Fig. 2 Effect of GR24 on mesocotyl elongation of 8-day-old dark-grown *d* mutant and wild-type (WT) seedlings. Each value represents mean \pm SD, $n = 10-15$.

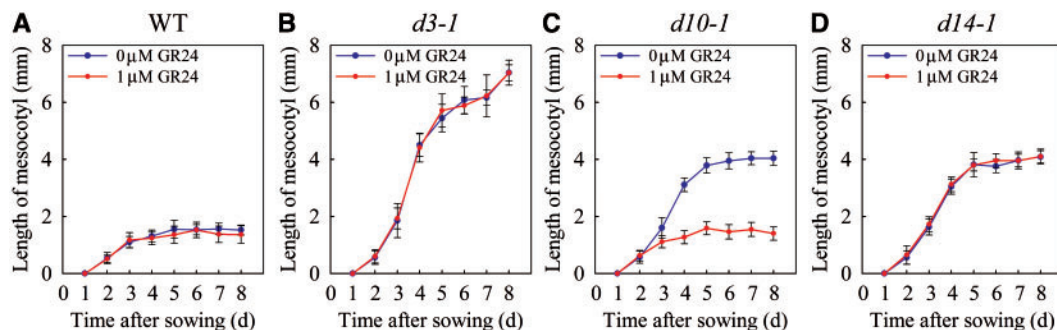


Fig. 3 Time-course of mesocotyl elongation of dark-grown *d* mutant and wild-type (WT) seedlings. Blue and red curves indicate untreated and GR24-treated seedlings, respectively. Each value represents mean \pm SD, $n = 10$.

Enhancement of cell division, but not cell elongation, in the mesocotyls of rice *d* mutants

Is the enhanced mesocotyl elongation of the *d* mutants the result of increased cell division or increased cell elongation? We measured cell length in vertical sections of the mesocotyls in *d3-1*, *d10-1*, *d14-1* and the wild type. During this investigation, we noticed that cell length differed between the upper half and the lower half of the *d3-1* mesocotyl, and thus we decided to separate the mesocotyls into two parts (upper part and lower part; Fig. 4A). Cell lengths in both the upper and lower parts of all the mesocotyls were $\sim 100 \mu$ m, except for *d3-1*, in which the cell lengths in the lower half of the mesocotyl were only $\sim 60 \mu$ m (Fig. 4B–C). Treatment with 1 μ M GR24 had no visible effect on cell length (Fig. 4B–C).

The cells in the mesocotyl are rectangular and elongated in the vertical direction as shown in Fig. 4C. The average number of cells from the bottom to the top of the mesocotyl was ~ 13 in the wild type, ~ 70 in *d3-1* and ~ 38 in *d10-1* and *d14-1* (Fig. 5, –GR24). When the seedlings were grown in the presence of 1 μ M GR24, the cell number in the *d10-1* mesocotyl was reduced to almost that of the wild type, whereas the cell numbers in *d3-1*, *d14-1* and wild-type mesocotyls were not changed (Fig. 5, +GR24). Taken together, these results indicate that SLs negatively regulate cell division, but not cell elongation, in the mesocotyl during germination and growth of rice in darkness.

Discussion

As mentioned above, SLs stimulate the growth of mycorrhizal fungi and control shoot branching, and probably affect seed germination, root growth, flower development and leaf senescence. Here we show that SLs or their downstream metabolites also negatively regulate mesocotyl elongation in rice when germinated and grown in darkness. This new role is demonstrated by our findings that rice mesocotyl elongation was enhanced in mutants deficient in or insensitive to SL (Fig. 1), and that a synthetic SL, GR24, rescued the phenotype of mesocotyl elongation in the SL-deficient mutants but not in the SL-insensitive mutants (Figs. 2, 3).

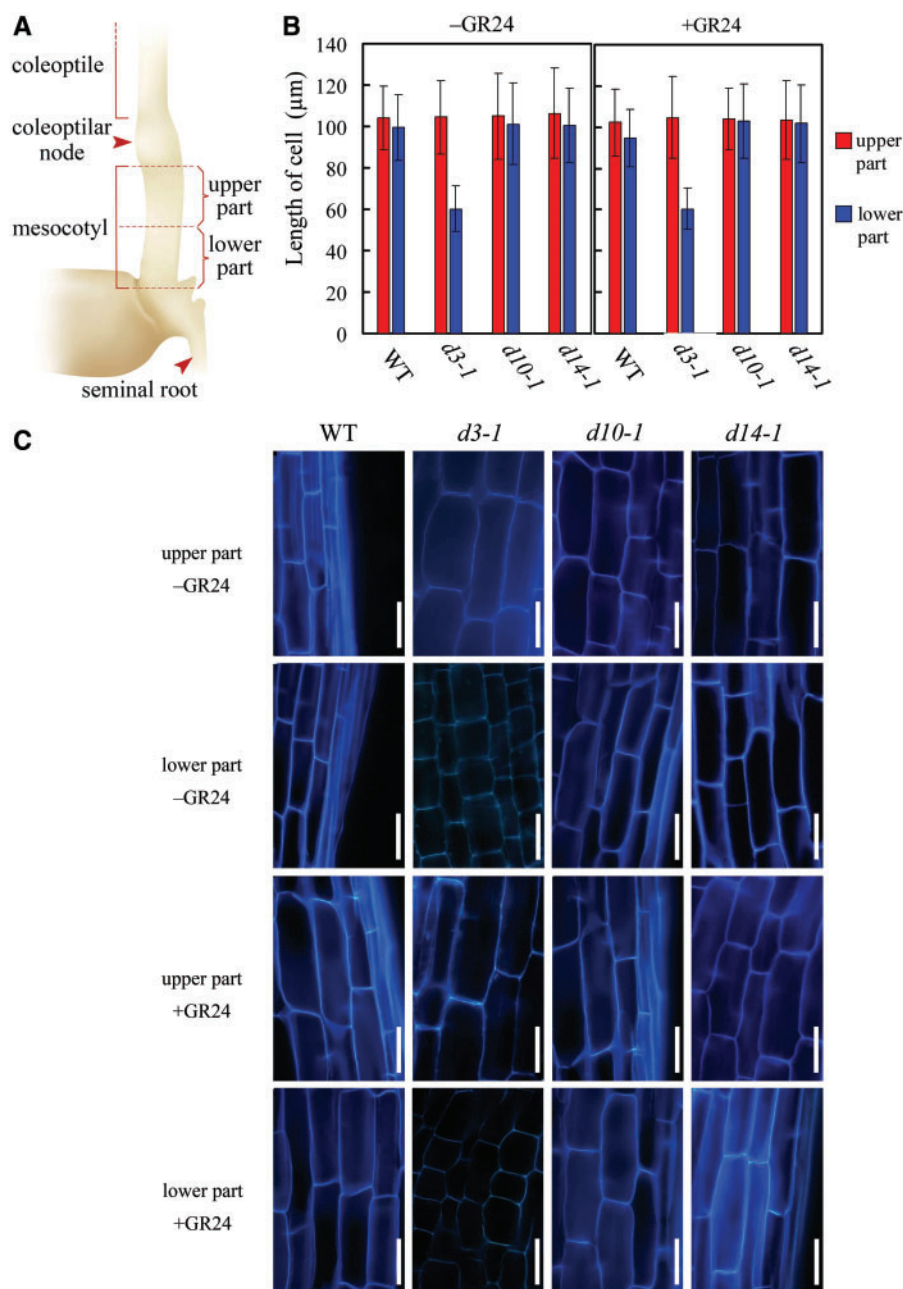


Fig. 4 Cell lengths in mesocotyls of dark-grown seedlings. (A) Definition of the upper part and the lower part of the mesocotyl. (B) Vertical length of cells in the upper and lower parts of mesocotyls in 8-day-old seedlings treated with (+GR24) or without (−GR24) 1 µM GR24. Each value represents mean ± SD, $n = 400\text{--}450$. (C) Representative tissue sections of mesocotyls from the wild type (WT), *d3-1*, *d10-1* and *d14-1*. Bars = 50 µm.

The results shown in **Figs. 4, 5** indicate that the enhancement of mesocotyl elongation in the *d* mutants is due to enhanced cell division, but not to enhanced cell elongation, in the mesocotyls, and that SLs or their downstream metabolites have an inhibitory effect on cell division in the dark-grown mesocotyls. In preliminary analyses, mRNA levels of some cell division-related genes were higher in the *d3-1* mesocotyl than in the wild-type mesocotyl a few days after sowing (data not shown). Together, these results raise the possibility that SLs also

negatively regulate cell division in the axillary buds, which might explain the observations that shoot branching of the *d* mutants is enhanced (Ishikawa et al. 2005). To understand the general roles of SLs in plant growth and development, it would be of interest to examine this possibility.

The *d3-1* seedlings, which are defective in SL signaling, have more severe phenotypes [longer mesocotyl and shorter coleoptile (**Fig. 1**)] than the other *d* mutants. Why? It may be that in the SL-related mutants other than *d3-1*, either (i) SLs could be

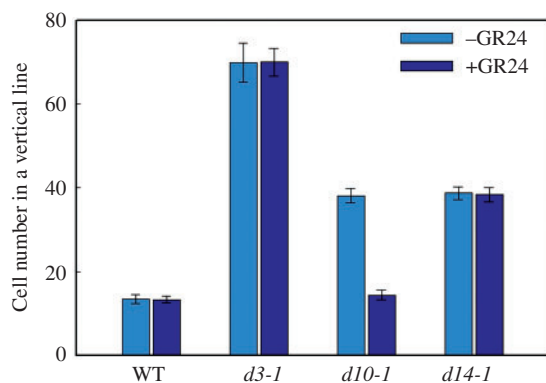


Fig. 5 Cell numbers and effect of GR24 on cell numbers in a vertical line in mesocotyls of dark-grown seedlings. Values indicate the numbers of cells between the coleoptilar node and the basal part of the seminal root in dark-grown 8-day-old seedlings of *d* mutants and the wild type (WT). GR24 concentration was 1 μ M. Each value represents mean \pm SD, $n = 30$.

produced by some other pathway or (ii) even in the absence of SLs, SCF^{D3} (an SCF-type ubiquitin E3 ligase) might still interact with its substrate, both of which would result in more moderate phenotypes. In the *d3-1* mutant, however, SL signaling would be severely affected, leading to more severe phenotypes. Another explanation for the more severe phenotypes of the *d3-1* mutants is that D3 may have roles in SL-independent processes.

These hypotheses may also explain differences in phenotypes that are observed in SL-related mutants of Arabidopsis. Arabidopsis has several SL-related genes (*MAX1*–*MAX4*) and mutants of these genes (*max1*–*max4*) show enhanced shoot branching. One of the *MAX* genes (*MAX2*) is very similar to *D3* in sequence, and like *D3* is involved in SL signaling. Also like the *d3-1* mutant, the *max2* mutant has more severe phenotypes (elongated hypocotyls when grown in the light and hyposensitivity of germination to red and far-red light) (Stirnberg et al. 2002, Shen et al. 2007) than do the other *max* mutants.

Recently, some progress has been made in elucidating the regulatory mechanisms of mesocotyl elongation through studies of some mutants affecting mesocotyl growth in rice. For instance, rice *phytochrome A* (*phyA*) and *coleoptile photomorphogenesis 1* (*cpm1*) impair the phytochrome-mediated inhibition of mesocotyl and coleoptile elongation (Takano et al. 2001, Biswas et al. 2003). The rice *phyA* mutant is insensitive to far-red light, which negatively affects the elongation of the mesocotyl and coleoptile (Takano et al. 2001). In the *cpm1* mutant, the phytochrome signaling that inhibits growth is impaired (Biswas et al. 2003). Interestingly, the mesocotyl and coleoptile elongate more in *cpm1* than in the wild type in darkness (Biswas et al. 2003). On the other hand, mesocotyl elongation in the rice JA-deficient mutant *hebiba* was enhanced when germinated and grown under dark conditions as well as under illumination with red light (Riemann et al. 2003), suggesting that JA is also involved in negative regulation of

mesocotyl elongation in rice. Rice genes (e.g. *OsAOS1* and *OsAOS4*) encoding allene oxide synthase, which is a key enzyme in JA synthesis, are up-regulated by red and far-red light in shoots (Haga and Iino 2004), and thus JA levels dramatically increase in rice seedlings in response to red light (Riemann et al. 2003). The induction of JA synthesis in rice seedlings under light conditions may explain why the phenotype of elongated mesocotyls in the *d* mutants was not observed when seedlings were germinated and grown under light conditions. In other words, the increased JA levels may mask the effect of a deficiency of SLs or impairment of SL signaling on mesocotyl elongation in light-grown seedlings of the *d* mutants. Indeed, in preliminary experiments, we found that exogenous application of JA to the *d* mutants during germination in darkness suppressed their elongated mesocotyl phenotype (data not shown). Further studies are needed to understand the relationship between SLs and JA and how it affects mesocotyl elongation in rice seedlings.

Materials and Methods

Plant materials and growth conditions

Rice (*Oryza sativa* L.) tillering dwarf mutants, *d3-1*, *d10-1*, *d14-1*, *d17-1* and *d27-1*, and their wild type (cv. Shiokari) were used in this study (Kinoshita and Takahashi 1991, Ishikawa et al. 2005).

Dehusked rice caryopses were sterilized in a 10% (v/v) sodium hypochlorite solution for 1 h. After washing with deionized water, seeds were kept in the water for 10 h at 4°C in darkness, and then ~15 seeds were sown on each 0.7% (w/v) agar plate. The agar plate was placed in a dark box made of black cardboard (Haga and Iino 2004) and then the dark box was covered by black cloths. The boxes were put in the growth chamber, and the seeds were germinated and grown at 28°C under completely dark conditions. For the time-course experiments, independent dark boxes were used for each time point.

SL treatment and analysis

GR24 was synthesized as described previously (Umehara et al. 2008). For SL treatment, 0.7% (w/v) agar was melted by autoclaving, and then GR24 (100 mM; dissolved in 100% acetone) was added to the melted agar (~50°C) to prepare agar plates with 0.01, 0.1 or 1 μ M GR24 (final concentration) (each plate contained 0.001% acetone). The deesterilized seeds were placed on the agar plates with 0.01, 0.1 or 1 μ M GR24 and were germinated and grown at 28°C under complete darkness. For negative controls, agar plates with 0.001% acetone were used.

Measurement of cell length and cell numbers in vertical sections of the mesocotyls

Six dark-grown 8-day-old seedlings from each mutant line and the wild type were selected for measuring and counting mesocotyl cells. The mesocotyls were embedded in 5% (w/v) agar and cut in 8- to 10- μ m-thick vertical sections with a microtome (MicroRom HM 650 V; Nikon, Tokyo, Japan). The sections were

observed with a fluorescence microscope (ECLIPSE E600; Nikon) and photographed with a Micro Color camera (DS-Ri1; Nikon). The cells could be clearly seen by the autofluorescence of their cell walls. The lengths of 50–100 cells in both the upper and lower parts of the mesocotyl (Fig. 4A) (a total of 400–450 cells for each part) were measured with NIS-Elements D (Nikon).

The numbers of cells in the outer layers (including the epidermis) of the mesocotyls from the coleoptilar node to the basal part of the seminal root in vertical sections were counted under a microscope. This number was counted for 30 columns of cells in each line of rice.

Acknowledgments

We thank Drs Moritoshi Iino and Ken Haga for instructions on how to grow rice seedlings under completely dark conditions and for stimulating discussions. We thank Drs Eiji Nambara, Yusuke Jikumaru, Mikiyoshi Umehara, Kiyooki Kato, Masao Watanabe, Yuhua Li, Shin-ichi Arimura for stimulating discussions. This work was partly supported by grants-in-aid from the Ministry of Education, Culture, Sports, Science and Technology of Japan, a grant from the Ministry of Agriculture, Forestry and Fisheries of Japan, and a grant from the Bio-oriented Technology Research Advancement Institution (Promotion of Basic Research Activities for Innovative Biosciences).

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