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

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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 Kyozuka 2010). SLs also probably have other roles in growth and development, including seed germination, root growth, flower development and leaf senescence (Bradow 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 Kyozuka 2010), and recessive mutants defective in these genes have reduced stature and
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 \( \alpha/\beta \)-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 developmental and environmental signals (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. Shiokari) (Fig. 1A–C). Interestingly, the \( d3-1 \) mesocotyls were \( \sim \)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](image-url)
elagination 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 µM) 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 µM, 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 µM) 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).

**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 ~100 µm, except for d3-1, in which the cell lengths in the lower half of the mesocotyl were only ~60 µm (Fig. 4B–C). Treatment with 1 µM 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 µM 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. 2** Effect of GR24 on mesocotyl elongation of 8-day-old dark-grown d mutant and wild-type (WT) seedlings. Each value represents mean ± 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 ± SD, n = 10.
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
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 pheno-
types 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 hyposensi-
tivity 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 pho-
tomorphogenesis 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 lino 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 defi-
cency 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 applica-
tion 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
deonized 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 lino 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 experi-
ments, 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 auto-
claving, 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 μM GR24 (final concentration) (each plate
contained 0.001% acetone). The desterilized seeds were
planted on the agar plates with 0.01, 0.1 or 1 μM 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 meso-
cotyl cells. The mesocotyls were embedded in 5% (w/v) agar
and cut in 8- to 10-μm-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.

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