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INCREASED SENSITIVITY TO, AND REDUCED PRODUCTION OF, ETHYLENE IN AN ABA-OVERPRODUCING TOMATO MUTANT

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The single-gene male-sterile *7B-1* mutant in tomato (*Lycopersicon esculentum* Mill.) shows reduced de-etiolation of hypocotyl growth, has an elevated level of endogenous abscisic acid (ABA) and reduced amounts of growth-active gibberellins (GAs), is supersensitive to exogenous ABA, and is resistant to abiotic stresses in light but not in the dark. The existence of crosstalk between light signaling and plant hormones, and the interaction of ABA and GA biosynthetic pathways with ethylene, led us to investigate the possible role of ethylene in the *7B-1* mutant. In the dark, *7B-1* seedlings exhibited the normal triple response to 1-aminocyclopropane–1-carboxylic acid (ACC), an ethylene precursor, similar to the wild type. In light, however, hypocotyl growth of mutant seedlings was more sensitive to ACC but less sensitive to the inhibitors of ethylene's action, such as silver thiosulfate, than the wild type. The *7B-1* mutant seedlings produced reduced levels of ethylene, which could account for the increased sensitivity to exogenous ACC. The mutant leaf discs also contained higher chlorophyll content and showed greater chlorophyll degradation in response to ACC than the wild type, and these could also be related to low ethylene production by the mutant. Fluridone, an inhibitor of ABA biosynthesis, countered the ACC-induced inhibition of hypocotyl elongation, and it also restored the wild-type phenotype in *7B-1* plants. The results suggest that the reduced de-etiolation of hypocotyl growth and the increased sensitivity of the mutant to ethylene in light are due to reduced ethylene production, which in turn may be related to high endogenous ABA. The data presented support our earlier findings that the *7B-1* mutant has a defect in light perception which affects both hormonal sensitivity and endogenous levels, thereby affecting hypocotyl and shoot growth.

Key words: ABA, ACC, crosstalk, ethylene, growth, light, tomato mutant.

INTRODUCTION

Many of the developmental processes in plants occur as a result of interactions between light and internal factors, including hormones. Light is known to alter the levels of endogenous hormones – auxins, gibberellins, cytokinins, brassinosteroids, abscisic acid (ABA) and ethylene – in plant tissues (for review: Kraepiel and Miginiac, 1997; Khurana et al., 1998; Neff et al., 2000). Varying combinations of light and dark periods might thus directly affect plant development by altering the levels of hormones or sensitivity to them (Reid et al., 1991; Thomas and Vince-Prue, 1997).

Plant developmental processes seem to be affected by the interaction of several hormones rather than by a single hormone (Davies, 1995; Reid and Howell, 1995), so it is not surprising that many mutations in hormone signaling pathways are defective in response to more than one hormone. For example, *Arabidopsis* mutants resistant to auxin confer cross-resistance to ethylene, cytokinins, ABA, gibberellin, brassinosteroid and/or ethylene (Pickett et al., 1990; Wilson et al., 1990; Ephritikhine et al., 1999). Ethylene influences numer-

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Abbreviations: ABA – abscisic acid; ACC – 1-aminocyclopropane–1-carboxylic acid; BM – basal medium; DW – dry weight; FLU (fluridone) – 1-methyl–3-phenyl–5-[3-trifluoromethyl-(phenyl)]–4(1*H*)-pyridinone; FW – fresh weight; GAs – gibberellins; STS – silver thiosulfate ($Ag_2S_2O_3$); WT – wild type.

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ous growth and developmental processes, including shoot elongation and the prominent "triple response" in the dark which determines the shape and stature of germinating seedlings (Abeles et al., 1992; Davies, 1995). It has been shown that ethylene biosynthesis and tissue sensitivity to ethylene can be modified by other plant hormones. For example, ethylene production can be regulated by auxin, cytokinin and ABA (for review: Spollen et al., 2000), and ethylene signaling can interact with the action of auxin (Pickett et al. 1990), gibberellin (Shechter et al., 1989) or ABA (Ghassemian et al., 2000; Beaudoin et al., 2000). There are also reports on ethylene's positive and negative feedback on its own levels (for review: Fluhr and Mattoo, 1996).

ABA is generally regarded as an inhibitor of shoot growth (Pilet and Barlow, 1987), whereas GAs promote elongation (Hooley, 1994; Ross et al., 1997). In maize, however, the accumulation of ABA, induced by low water potential, did not affect root growth, and this was related to restricted ethylene production (Spollen et al., 2000). On the other hand, the impairment of shoot growth caused by ABA deficiency in two tomato mutants was in part related to increased ethylene evolution (Sharp et al., 2000; Sharp and LeNoble, 2002). There are also reports on the interaction of GAs and ABA biosynthetic pathways with ethylene biosynthesis or signaling (Kao and Yang, 1983; Shechter et al., 1989; Spollen et al., 2000; Hansen and Grossmann, 2000), and of cytokinin acting through ethylene production (Golan et al., 1996), indicating the existence of crosstalk between ethylene and other hormones. Light is also known to affect ethylene synthesis by reducing 1-aminocyclopropane–1-carboxylic acid (ACC) oxidase activity (Kao and Yang, 1982; Finlayson et al., 1998), and extension of the photoperiod promotes the conversion of ACC to ethylene (Weckx and Pouke, 1989; Jensen and Veierskov, 1998). Thus, the crosstalk between ethylene and other hormones such as ABA seems to be regulated by light.

The spontaneous recessive single gene *7B-1* mutant in tomato (*Lycopersicon esculentum*) is male-sterile under long days (LD) but fertile under short days (SD) (Sawhney, 2004). The mutant also shows reduced light-induced inhibition (i.e., de-etiolation) of hypocotyl growth, has an elevated level of endogenous ABA but reduced levels of growth-active GAs, and exhibits light-dependent supersensitivity to exogenous ABA (Fellner et al., 2001). In addition, seed germination in the *7B-1* mutant is resistant to ABA and osmotic stresses, and this resistance is exhibited only in light, specifically blue light (Fellner and Sawhney, 2001, 2002). This study addressed the following questions:

(1) Does the *7B-1* mutant exhibit differential growth response to exogenous ethylene and, if so, is this response affected by light and/or by ABA?
(2) Is ethylene evolution affected in the *7B-1* mutant?

MATERIALS AND METHODS

PLANT MATERIAL AND GROWTH CONDITIONS

The single-gene recessive *7B-1* mutant in tomato (*Lycopersicon esculentum* Mill. background cv. Rutgers) was isolated as a photoperiod-sensitive male-sterile mutant (Sawhney, 2004). For all experiments, *7B-1* and WT seeds were obtained from plants grown in SD (8 h photoperiod). For sterile culture, seeds were soaked in 50% (v/v) Javex solution (3% sodium hypochlorite) (Colgate-Palmolive Canada, Toronto, Canada) for 20 min, rinsed extensively with sterile distilled water, and sown on 0.7% (w/v) agar medium in Petri dishes (100 × 15 mm). The basal medium (BM) contained Murashige and Skoog (1962) salts, 1% (w/v) sucrose and 1 mM Mes (2-(*N*-morpholino)-ethanesulfonic acid) (pH adjusted to 6.1 by KOH). Petri dishes with germinated seeds were placed in an incubator under a temperature regime of 25˚C-light/23˚C-dark and a 16 h photoperiod, under white fluorescent tubes at illumination of 25–40 μE m^{-2} s^{-1}. Older plants were grown in soil (Sunshine #1 soil mix, Sun Gro Horticulture, U.S.A.) in a growth chamber under a temperature regime of 25˚C-light/18˚C-dark and a 16 h photoperiod, under fluorescent tubes and incandescent bulbs at illumination of 90 to 200 μE m^{-2} s^{-1}.

HYPOCOTYL ELONGATION

Seeds were germinated on BM containing various concentrations of ACC (Sigma, St. Louis, MO), silver thiosulfate (STS) (6 mM stock solution prepared by pouring 12 mM $AgNO_3$ into 96 mM sodium thiosulfate solution in a 1:1 ratio, with constant stirring) (Kumar et al., 1998), $CoCl_2$, $AgNO_3$, or a mixture of ACC with fluridone (FLU) (Eli Lilly, Indianapolis, IN). Hypocotyl lengths were measured after 6 days.

MEASUREMENT OF ETHYLENE PRODUCTION

Seeds were germinated in sterile culture as above, but Gelrite gellan gum (Sigma-Aldrich Canada, Oakville, ON) was used instead of agar, and all media were prepared 7 days prior to use in order to minimize ethylene production by the media. After 7 days, 6 germinated seeds were transferred to each 50 ml Falcon tube (Becton Dickinson Labware, NJ) filled to the 35 ml mark with BM made with Gelrite. To half of these tubes, 10 μM (±)-cis,trans ABA (Sigma-Aldrich Canada, Oakville, ON) was added to the media. Seedling height was measured at the time of transfer, and again after 1 and 7 days of growth.

Ethylene production was measured after 24 h and 7 days of growth. The tubes were capped at 9:00 a.m., and 6 h later 1 ml air samples were taken through a septum in the center of the cap. Air samples were injected into a gas chromatograph (Photovac 10Splus,

TABLE 1. Effects of ACC (10 μM) and STS (100 μM) alone, or in combination, on hypocotyl lengths of WT and *7B-1* seedlings grown in light and in dark. Hypocotyl lengths were measured after 6 days of seed sowing on Murashige and Skoog (1967) basal medium. The data represent the mean ± SE (n = 12) based on one experiment. Similar results were obtained in two independent experiments. Statistical significance was analyzed using Student's *t* test

		Hypocotyl length (mm)	
Genotype	Treatment	Light	Dark
WT	Control	20.0 ± 0.4	62.8 ± 1.4
	ACC	15.5 ± 0.5*	21.1 ± 1.0*
	STS	34.0 ± 1.5*+	72.2 ± 3.6*+
	ACC + STS	22.9 ± 0.5+	35.8 ± 2.2*+
7B-1	Control	26.1 ± 0.4	60.8 ± 1.4
	ACC	15.4 ± 0.3*	23.1 ± 0.9*
	STS	30.8 ± 1.4+	79.0 ± 2.6*+
	ACC + STS	26.0 ± 0.8+	31.6 ± 2.9*+

* significantly different ($P \leq 0.05$) from WT or *7B-1* control
+ significantly different ($P \leq 0.05$) from ACC-treated WT or *7B-1*.

Photovac, Markham, ON), and evolved ethylene was calculated based on an ethylene standard (Praxair, Danbury, CT), and plant fresh weight (FW). Plant FW at the 24 h measurement interval was estimated based on the relationship between plant height and FW (r^2 = 0.96). The ethylene concentration in each tube was taken as the average of two subsamples. Three tubes were used for each treatment for both *7B-1* and WT plants, and the experiment was replicated twice in a randomized complete block design.

MEASUREMENT OF CHLOROPHYLL CONTENT AND SENESCENCE IN LEAF DISCS

Leaf senescence was measured by the method of Aharoni (1989). Leaves from the second whorl of one-month-old *7B-1* and WT plants were surface-sterilized by soaking in 0.5% (v/v) Javex solution (3% sodium hypochlorite) for 20 sec, and then rinsed several times in sterile distilled water. Leaf discs 8 mm in diameter were punched from leaf blades. For each genotype, leaf discs were taken from 4 plants and 5 discs were floated with the abaxial surface in contact with either 10 ml water or ACC solution in sterile Petri dishes. There were 3 replicates (each with 5 discs) for each treatment. Leaf discs were incubated in the dark at 25°C for 4 days. Chlorophyll was extracted in *N*,*N*-dimethylformamide (JT Baker Chemical, Phillipsburg, NJ) (5 leaf discs in 7 ml solution) for 5 days in darkness at 4°C. The chlorophyll level was determined with a Beckman spectrophotometer (model DU® 7400, U.S.A.) at 664.5 nm (for Chl *a*) and 647 nm (for Chl *b*). The equation of Inskeep and Bloom (1985) was used for chlorophyll quantification, and then chlorophyll content was related to leaf disc FW.

FLURIDONE TREATMENT OF PLANTS IN VIVO

For each genotype, 4 to 5 42-day-old plants grown in the soil in a growth chamber were sprayed with 10 μM fluridone for one week at 48 h intervals. Fluridone was dissolved in absolute ethanol and then diluted to 0.001% ethanol with distilled water. Control plants were sprayed in the same way with 0.001% ethanol. Plant height was measured weekly over the next two months.

RESULTS

RESPONSIVENESS OF WT AND *7B-1* MUTANT TO ACC, STS, $AGNO_3$, OR $COCL_2$

7B-1 hypocotyls were longer than those of WT seedlings in light but not in the dark (Tab. 1; see also: Fellner et al., 2001). In both genotypes, exogenous ACC (10 μM), an ethylene precursor, inhibited hypocotyl growth in light as well as in the dark. In light, however, *7B-1* hypocotyls were more sensitive than the WT to the inhibitory effect of ACC, but in the dark the response was similar to WT (Tab. 1). Also, at low concentration (1 μM), ACC had no effect on the growth of WT hypocotyls, but was inhibitory to *7B-1* hypocotyls (Fig.1); at high concentration (100 μM), ACC had a similar inhibitory effect. STS (100 μM), an inhibitor of ethylene's action, significantly promoted hypocotyl elongation in WT (by 70%) in light, but in *7B-1* seedlings the stimulation was minimal (Tab. 1). In the dark, STS stimulated hypocotyl growth in both genotypes (Tab. 1). STS also completely overcame the inhibitory effect of ACC on hypocotyl growth in light, but was much less effective in the dark in both genotypes (Tab. 1). $AgNO_3$ (100 μM), another inhibitor of ethylene's action, had no effect on WT and mutant hypocotyl growth in light; however, like STS, it completely reversed ACC-induced hypocotyl growth inhibition in light but not in dark (data not shown). $CoCl_2$ (1–100 μM), an inhibitor of ACC oxidase, also had no marked effect on the hypocotyl growth of WT and mutant seedlings in light, and did not restore the ACC-induced inhibition of hypocotyl growth (data not shown).

In the dark, the ethylene-induced "triple response" includes the inhibition of root and hypocotyl elongation, promotion of hypocotyl swelling, formation of an exaggerated apical hook in the hypocotyl, and abnormal directional growth (Reid and Howell, 1995). The hypocotyl widths of etiolated *7B-1* and WT control seedlings did not differ. Likewise, ACC (100 μM) caused similar swelling below the hook region in both genotypes, and STS repressed ACC-induced swelling similarly in WT and *7B-1* (data not shown). Also, at 10 μM

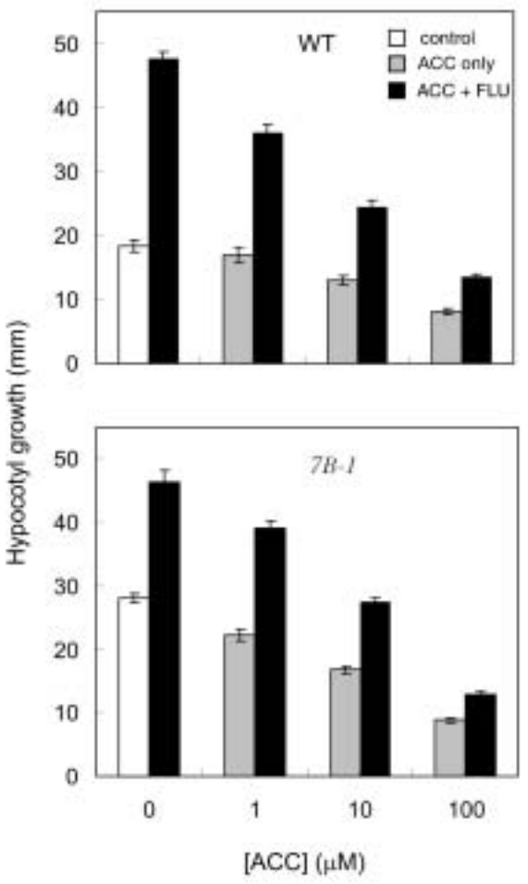

Fig. 1. The ability of fluridone (FLU) to affect ACC-induced inhibition of hypocotyl growth in 6-day-old WT and *7B-1* seedlings grown in light. Seedlings were grown on BM with or without FLU (1 μM) and/or ACC (1, 10 or 100 μM). Values represent the means ± SE of at least 12 seedlings from one representative experiment (some SE bars are not visible because they did not exceed 1%).

or 100 μM ACC, *7B-1* and WT hypocotyls exhibited similar agravitropic growth and formation of an exaggerated apical hook (data not shown). Thus, WT and *7B-1* seedlings, treated with ACC and STS or untreated, showed similar responses in the dark.

EFFECTS OF FLURIDONE ON HYPOCOTYL GROWTH

WT and *7B-1* hypocotyls were treated with fluridone (FLU), an inhibitor of ABA biosynthesis (Gamble and Mullet, 1986; Saab et al., 1990), either alone or in combination with ACC, to determine the possible cross-talk between ethylene and ABA. FLU alone (1 μM) strongly stimulated hypocotyl growth of WT seedlings by about 160%, but its effect on mutant hypocotyls was much less, about 65% (Fig. 1; see also: Fellner et al., 2001). When FLU was supplied together with ACC, hypocotyl growth stimulation decreased with increasing concentrations of ACC, and there was relatively greater growth inhibition in WT than in *7B-1* seedlings (Fig. 1).

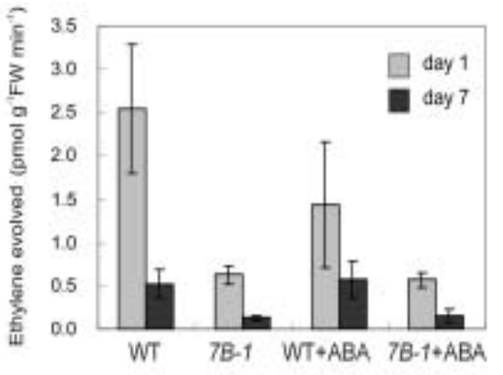

Fig. 2. Ethylene evolution from young WT and *7B-1* mutant seedlings. Germinated seeds were transferred to 50 ml tubes (6 germinated seeds per tube) filled with BM with or without ABA (10 μM). Ethylene production was measured after 1 day (24 h) and after 7 days of growth. Each value represents the mean ± SE of ethylene evolved in 6 tubes in two independent experiments.

ETHYLENE PRODUCTION IN WT AND *7B-1* MUTANT SEEDLINGS

After both 1 and 7 days of growth, *7B-1* seedlings produced significantly less ethylene than WT on a FW basis, although ethylene production was also considerably reduced in WT seedlings after 7 days (Fig. 2). The effect of exogenous ABA on ethylene production in WT and *7B-1* seedlings was not statistically significant, but there was a clear indication of reduced ethylene production in one-day-old WT seedlings, not in *7B-1*. After 7 days, ABA had no effect on ethylene production in either genotype (Fig. 2).

GROWTH OF WT AND *7B-1* MUTANT PLANTS IN VIVO IN RESPONSE TO FLURIDONE

Wild type and *7B-1* plants did not differ in height until ~65 days after seed sowing (Fig. 3a). However, at flower anthesis ~70 days after seed sowing and beyond, *7B-1* plants were significantly taller than WT plants (Figs. 3a, 4). The increased height of *7B-1* plants was due mainly to longer internodes (Fig. 3b). As in the case of hypocotyls (Fellner et al., 2001), FLU at 10 μM inhibited shoot growth of WT and *7B-1* plants; in WT the inhibition was weak, but in *7B-1* plants it reduced plant height to that of the control or of FLU-treated WT plants (Fig. 3a). As shown in Figure 3b, FLU-induced

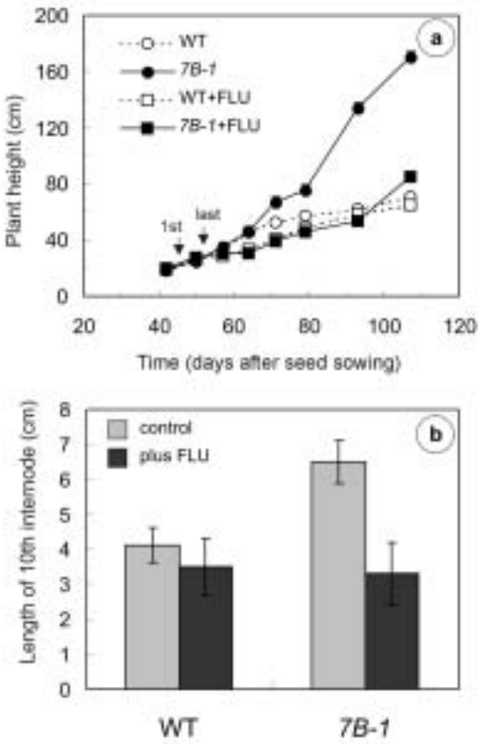

Fig. 3. Effect of fluridone (FLU) on the growth of WT and *7B-1* plants in vivo. **(a)** Time course of shoot growth of WT and *7B-1* plants grown in light in a growth chamber in the absence or presence of FLU (10 μM). 42-day-old plants were sprayed with FLU for one week at intervals of 48 h. Arrows with "1st" and "last" indicate times of first and last FLU treatment, **(b)** Effect of FLU on length of 10th internode of WT and *7B-1* plants 44 days after last treatment. Each value represents the mean ± SE of 4 or 5 plants of one experiment. Similar results were obtained for two independent experiments. SE bars in (a) are not visible because they did not exceed the dot diameter.

Fig. 4. Comparison of 3-month-old WT and *7B-1* plants grown in growth chamber under 16 h photoperiod.

inhibition was related to reduction in internodal length. However, the node number of treated *7B-1* plants was not affected (data not shown).

CHLOROPHYLL CONTENT AND DEGRADATION IN WT AND *7B-1* LEAVES

The leaves from the *7B-1* mutant grown in vivo had about 30% more chlorophyll (4997 ± 80 μg g^{-1} FW) than WT leaves (3728 ± 73 μg g^{-1} FW). In control leaf discs, the relative chlorophyll loss during 4 days of incubation was similar (~58%) in WT and the mutant. ACC (1–100 μM) caused a marked loss of chlorophyll in leaf discs; in WT the loss was similar at the three ACC concentrations tested, but in the *7B-1* mutant it increased with the increase in ACC concentrations (Fig. 5). In both genotypes, the chlorophyll *a*/*b* ratio was about 2.8 in fresh leaves, and about 3.0 in leaf discs incubated in the absence or presence of exogenous ACC.

DISCUSSION

Ethylene inhibits hypocotyl growth in numerous plant species (Neljubow, 1901; Jackson, 1979), and this response has been used to isolate a number of ethylene mutants (Ecker, 1995). In the wild type and *7B-1* mutant of tomato, exogenous ACC (10 μM) also inhibited hypocotyl growth in light and dark, but in light its effect on the mutant was significantly greater than its effect on WT (Tab. 1). Also, a low concentration (1 μM) of ACC had no effect on WT but inhibited the growth of *7B-1* hypocotyls (Fig. 1); STS, an inhibitor of ethylene's action, caused greater stimulation of hypocotyl growth in WT than in mutant seedlings (Tab. 1). These observations suggest a possible defect in ethylene production by the mutant, and the data on ethylene evolution support this view; the *7B-1* seedlings showed reduced ethylene evolution relative to WT (Fig. 2). *7B-1* leaves also had relatively greater chlorophyll content than WT leaves and, unlike WT, showed a greater loss of chlorophyll with increasing ACC concentration. These observations can also be explained on the basis of low ethylene production by mutant

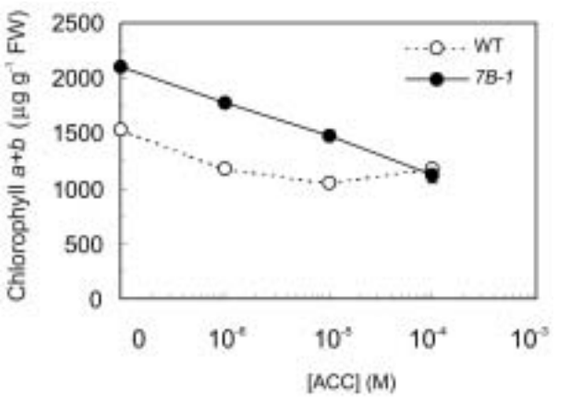

Fig. 5. Chlorophyll loss in leaf discs of *7B-1* and WT in the presence of various concentrations of ACC. Leaf discs were punched from 2nd leaf of one-month-old WT and *7B-1* mutant plants grown in LD. Discs were incubated in the dark with or without ACC, and after 4 days the chlorophyll content was determined. Values are the means ± SE of 3 replicates of one experiment (SE bars are not visible because they did not exceed 1%).

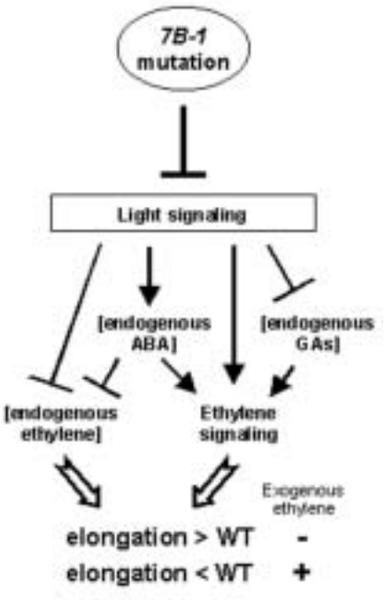

Fig. 6. Working model to show the effects of the *7B-1* mutation on light and hormone signaling during hypocotyl and shoot growth in tomato. The model suggests that the *7B-1* mutation causes a defect in light perception and signaling, which may cause a drop in ethylene evolution, directly or via ABA accumulation. At the same time, reduced light signaling leads to ABA accumulation and reduced levels of endogenous GAs, which may increase the responsiveness of the *7B-1* mutant to ethylene. The increased hypocotyl and shoot growth in the *7B-1* mutant is explained on the basis of low ethylene production, whereas reduced growth in the presence of exogenous ethylene is due to increased ethylene signaling. Arrows and T-bars represent positive and negative effects, respectively.

leaves, as the role of ethylene in the induction of leaf senescence is well known (e.g., Noodén and Leopold, 1988). Further, both STS and $AgNO_3$, inhibitors of ethylene's action, completely or partially overcame the inhibitory effect of ACC in WT and mutant hypocotyls, suggesting that ethylene has an important role in hypocotyl growth in tomato.

Increased sensitivity to ethylene and reduced production of ethylene by the *7B-1* mutant may not be the primary defects of this mutation, however, since the dark-grown *7B-1* and WT seedlings showed similar morphology and the *7B-1* seedlings exhibited the normal triple response. The ABA signaling pathway is known to interact antagonistically or synergistically, depending on the plant organ or tissue, with ethylene's action in plant growth and development (Abeles et al., 1992). *7B-1* mutant hypocotyls have high endogenous ABA and low levels of growth-active GAs, yet they show reduced de-etiolation in light (Fellner et al., 2001), a phenotype generally uncharacteristic of such hormonal changes. We have previously suggested that since the *7B-1* mutant is an ABA over-producer, there is likely a defect in the ABA signaling pathway which is responsible for the reduced de-etiolation of hypocotyls (Fellner et al., 2001; Fellner and Sawhney, 2002). However, since FLU reverses the growth inhibition caused by ACC (Fig. 1), it seems that the increased sensitivity to exogenous ethylene may be due to high ABA in the mutant. Also, although not significant, exogenous ABA tended to reduce ethylene production in WT seedlings after 1 day, but it had no effect on mutant seedlings. Thus it is possible that high endogenous ABA in the *7B-1* mutant affects ethylene production, leading to increased sensitivity to ethylene and reduced de-etiolation of hypocotyl growth. In addition, the tall stature of *7B-1* plants and increased inhibition of internodal growth of the mutant by FLU, relative to WT (Figs. 3a,b, 4), are also indicators that high ABA may affect reduced ethylene production in the mutant. Crosstalk between ABA and ethylene has been shown in other systems. For example, maize roots grown at low water potential had high ABA accumulation, and this was due to restriction of ethylene production (Spollen et al., 2000). Similarly, in the ABA-deficient tomato mutants *flacca* and *notabilis*, inhibition of shoot growth was via increased ethylene evolution (Sharp et al., 2000). Finally, since gibberellins have also been suggested to reduce tissue sensitivity to ethylene (Shechter et al., 1989), the low levels of endogenous growth-active GAs in *7B-1* hypocotyls (Fellner et al., 2001) may also affect tissue sensitivity to ethylene.

Another important aspect of this and our earlier studies is that the differential sensitivity of the *7B-1*

mutant to exogenous ethylene and ABA is observed only in light, not in dark. We have previously suggested that *7B-1* is likely a photomorphogenic mutant with reduced sensitivity to light, especially blue light, for hypocotyl growth and seed germination (Fellner and Sawhney, 2001; 2002). Since light promotes the conversion of ACC to ethylene (Weckx and van Pouke, 1989) and in tomato the extension of photoperiod leads to an increase in ethylene evolution (Jensen and Veierskov, 1998), a defect in light perception in the *7B-1* mutant could therefore potentially lead to reduced ethylene production

The model presented in Figure 6 suggests that the *7B-1* mutant has a defect in light perception and signaling, and that this defect leads to an increase in endogenous ABA and reduction of both ethylene evolution and endogenous GAs. The reduction of ethylene production is likely a consequence of the increase in endogenous ABA, although the direct effect of light signaling on ethylene evolution cannot be disregarded. The reduced ethylene production in the mutant leads to greater hypocotyl growth and shoot elongation compared to the WT, and both high endogenous ABA and reduced GAs may affect ethylene signaling. This study has shown the importance of light in regulating hormone sensitivity and hormone levels in a tissue, and the possible existence of cross-talk between ABA, ethylene and GAs during hypocotyl growth in tomato.

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