Suppressing Green Mold Decay in Grapefruit with Postharvest Jasmonate Application

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ABSTRACT. Jasmonic acid (JA) and methyl jasmonate (MJ), collectively referred to as jasmonates, are naturally occurring plant growth regulators involved in various aspects of plant development and responses to biotic and abiotic stresses. In this study, we found that postharvest application of jasmonates reduced decay caused by the green mold Penicillium digitatum (Pers.: Fr.) Sacc. after either natural or artificial inoculation of grapefruit (Citrus paradisi ‘Marsh Seedless’). These treatments also effectively reduced chilling injury incidence after cold storage. The most effective concentration of jasmonates for reducing decay in cold-stored fruit or after artificial inoculation of wounded fruit at 24 °C was 10 μmol·L−1. Higher and lower jasmonate concentrations were less effective at both temperatures. MJ at 10 μmol·L−1 also most effectively reduced the percentage of fruit displaying chilling injury symptoms after 6 weeks of storage at 2 °C and 4 additional d at 20 °C. When tested in vitro, neither JA nor MJ had any direct antifungal effect on P. digitatum spore germination or germ tube elongation. Therefore, it is suggested that jasmonates probably reduced green mold decay in grapefruit indirectly by enhancing the natural resistance of the fruit to P. digitatum at high and low temperatures.

Materials and Methods

PLANT MATERIAL AND FUNGAL CULTURES. Grapefruit (Citrus paradisi ‘Marsh Seedless’) were obtained from a local orchard and were used on the day of harvest. Before treatments, fruit were thoroughly washed with water and then air dried. Experiments were repeated two to four times, with 140 fruit replications for each postharvest treatment and six fruit per treatment for the artificial inoculation experiments. P. digitatum was obtained from an infected grapefruit and cultured on Potato Dextrose Agar (Difco, Detroit, Mich.). Spore suspensions were prepared by removing the spores with a bacteriological loop from the sporulating edges of a 2- or 3-week-old culture, and suspending them in sterile distilled water.
was 90% in both storage temperatures. Fruit treated at 2°C were air-dried in the shade. Control fruit were stored at 2°C for 6 weeks, and subsequently decay incidence was performed at the end of the 6-week cold storage and after an additional 4 d at 20°C. Grapefruit CI symptoms were manifested as rusty surface pitting. The severity of CI symptoms was assessed visually according to a four-stage scale, as follows: 1 = no pitting; 2 = a few scattered pits; 3 = pitting covering up to 30% of the fruit surface; and 4 = extensive pitting covering >30% of the fruit surface. The CI index was calculated according to the severity of symptoms (S) and the number of fruit affected (N) (Eq. [1]).

$$\frac{\sum_{i=1}^{N} (N_i \times S_i)}{\sum_{i=1}^{N} N_i}$$  \[1\]

The percentage of injured fruit was defined as the percentage of grapefruit with moderate to severe CI index (stages 3 and 4); such fruit were considered unacceptable for sale and consumption.

**In vivo assays with wounded grapefruit.** Grapefruit rinsed under tap water were dried and wounded with a desiccating needle (1 to 2 mm deep) at three sites around the stem end, and aliquots of 25 µL of jasmonate solutions were pipetted into each wound. Wounds of control fruit were treated similarly with sterile-distilled water instead of jasmonates. Wounded sites were inoculated with 25 µL of spore suspension of *P. digitatum* (*5 × 10^{4}* spores/mL) after 24 h incubation at 24°C. When dry, grapefruit were kept at 24°C in plastic trays under humid conditions for 4 to 7 d before determining the percent of infected sites. Six fruit were used per treatment (total of 18 wounds), and each experiment was performed four times with JA and twice with MJ.

**Effect of jasmonates on *P. digitatum* in vitro.** Spores of *P. digitatum* were suspended in 100 g·L^{-1} potato dextrose broth (Difco, Detroit, Mich.) at a final concentration of 2 × 10^{4} spores/mL. Aliquots (450 µL) of spore suspensions were transferred to wells of tissue culture clusters (Corning Costar Corporation, Cambridge, Mass.), and 50-µL aliquots of the various jasmonate solutions were added to give final concentrations of 1 to 1000 µmol·L^{-1}. Samples of the various test solutions (30-µL drops), were placed on ethanol-washed microscope slides (three drops per slide) kept in petri dishes padded with moistened filter paper, and incubated for 24 h at 25°C in darkness. Spore germination and germ tube elongation were measured in three microscope fields, each containing 40 to 50 spores, under a light microscope.

**Statistical analysis.** Results were analyzed with the SigmaStat statistical software (Jandel Scientific Software, San Rafael, Calif.). Two-factor analysis of variance (ANOVA) and Student-Newman-Keuls multiple range tests were performed where indicated. Unless noted otherwise, only results significant at *P* ≤ 0.05 are discussed.

**Results**

**Effect of postharvest dip treatments with MJ.** To examine the efficacy of MJ as a potential postharvest treatment to reduce decay in citrus fruit, we dipped grapefruit in MJ solutions of various concentrations (1 to 50 µmol·L^{-1}) and evaluated decay development and CI symptoms after 6 weeks of storage at 2°C and 4 and 21 d of simulated shelf life at 20°C. The results show that MJ appeared to reduce green mold decay that developed naturally immediately after cold storage and after 4 d of shelf life in a dose-dependent manner (Fig. 1A). However, after the prolonged simulation of shelf life (21 d), dipping in the highest concentration of MJ (50 µmol·L^{-1}) appeared to be less effective than dipping in 10 µmol·L^{-1} MJ (Fig. 1A). In fruit treated with 10 µmol·L^{-1} MJ, the decay percentage. Evaluation of CI was performed at the end of the 6-week cold storage and after an additional 4 d at 20°C. Grapefruit CI symptoms were manifested as rusty surface pitting. The severity of CI symptoms was assessed visually according to a four-stage scale, as follows: 1 = no pitting; 2 = a few scattered pits; 3 = pitting covering up to 30% of the fruit surface; and 4 = extensive pitting covering >30% of the fruit surface. The CI index was calculated according to the severity of symptoms (S) and the number of fruit affected (N) (Eq. [1]).

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incidences of decay during cold storage (2 °C) and after 4 or 21 d of shelf life were only 0.9%, 1.9%, and 2.8%, respectively, compared with 4%, 7%, and 12% in untreated control fruit.

The statistical analysis, summarized in Table 1, indicated that this 10-µmol·L⁻¹ MJ dose was also the most effective concentration in protecting grapefruit against CI (expressed as percentage of pitted fruit) after cold storage and 4 d of shelf life (Fig. 1B). In this treatment, the percentage of fruit exhibiting CI during cold storage was only 3%, compared with 16% in the untreated control fruit. After 4 d of shelf life, the incidence of CI in 10-µmol·L⁻¹ MJ-treated fruit was 8.4%, compared with 31% in control fruit (Fig. 1B). The severity of CI, expressed as CI index, was also similarly reduced by 10-µmol·L⁻¹ MJ (Table 1), as previously reported (Meir et al., 1996). The statistical analysis (Table 1) shows that MJ may affect decay during cold storage in a different manner from the way it affects CI incidence. However, depending on the shelf-life duration, 10-µmol·L⁻¹ MJ seems to be the most effective concentration to reduce both parameters.

Effect of Jasmonates on Artificially Inoculated Fruit. To examine the possible effect of jasmonates on decay caused by artificial inoculation with the green mold, *P. digitatum*, in the absence of CI, we pretreated the fruit with several exponential concentrations of jasmonates applied through wounds, and monitored decay development at 24 °C after inoculation with the fungus. The results (Fig. 2) show that JA effectively reduced the amount of green mold decay that developed in the inoculated wounds in a concentration-dependent manner, with a peak of efficacy at 10-µmol·L⁻¹. Thus, at 10-µmol·L⁻¹, JA reduced decay to 47% of the amount that developed in nontreated control fruit, whereas 1 and 100-µmol·L⁻¹ JA reduced decay to only 59% and 78%, respectively. Higher or lower concentrations (0.1 or 1000-µmol·L⁻¹ JA) were completely ineffective in reducing decay (Fig. 2). Similar results of reduction of green mold rot after artificial inoculation of ‘Marsh Seedless’ grapefruit with *P. digitatum* were also obtained when MJ instead of JA was applied in the same concentration range (data not shown).

**Table 1.** Summary of statistical analysis of the effects of methyl jasmonate (MJ) concentration and shelf time at 20 °C on decay development and chilling injury (CI) in grapefruit after 6 weeks of storage at 2 °C. Mean values were derived from the data of Fig. 1.

| Analysis | Decay (%) | CI index (1–4) | Fruit with CI (%) |
|----------|-----------|----------------|------------------|
| MJ concn (µmol·L⁻¹) |             |                |                  |
| 0 (control) | 7.6 a¹ | 1.77 a | 23.8 a |
| 1 | 5.4 b | 1.92 a | 24.2 a |
| 10 | 1.9 c | 1.37 b | 5.7 c |
| 50 | 2.3 c | 1.87 a | 15.7 b |
| Time at 20 °C (d) |             |                |                  |
| 0 | 1.7 b | 1.56 b | 10.2 b |
| 4 | 3.2 b | 1.91 a | 24.5 a |
| 21 | 8.0 a | ND² | ND |
| Two-factor analysis of variance |             |                |                  |
| MJ concn (C) | *** | *** | *** |
| Time at 20 °C (T) | *** | *** | *** |
| C × T | NS | ** | NS |

¹Means within each column, followed by different letters are significantly different at *P* = 0.05 according to the Student-Newman-Keuls multiple range test.

²ND = not determined.

³NS, **, *** Nonsignificant or significant at *P* = 0.01 or 0.001, respectively.

**Effect of Jasmonates on *P. digitatum* in Vitro.** To examine whether the effects of JA and MJ in reducing decay caused by the green mold, *P. digitatum*, were due to their direct antifungal activity, we incubated a *P. digitatum* spore suspension in a growth medium containing various concentrations of jasmonates. We found that neither JA nor MJ, at all concentrations tested (1 to 1000 µmol·L⁻¹), had any inhibitory effect on *P. digitatum* spor germination and germ tube elongation (data not shown). Moreover, the high concentrations of JA or MJ even enhanced slightly germ tube elongation of the pathogen.

**Discussion**

Jasmonates are naturally occurring plant growth regulators, known to be involved in various aspects of plant development and responses to biotic and abiotic stresses (Creelman and Mullet, 1995, 1997). In the present study, we found that postharvest application of jasmonates reduced decay caused by the green mold, *P. digitatum*, after either natural (Fig. 1A) or artificial (Fig. 2) inoculation of ‘Marsh Seedless’ grapefruit.

There are only a few reports of exogenously applied jasmonates actually protecting plants against disease development: in potato (*Solanum tuberosum* L. ‘Alpha’) and tomato (*Lycopersicon esculentum* Mill. ‘Baby’) plants, jasmonates protected against *Phytophthora infestans* (Cohen et al., 1993); in barley (*Hordeum vulgare* L. ‘Golden Promise’) plants JA protected against *Erysiphe graminis* (Schweizer et al., 1993); in cut roses (*Rosa hybrida* L. ‘Mercedes’) and strawberries (*Fragaria ×ananassa* Duch. ‘Delmarvel’) MJ protected against *Botrytis cinerea* (Meir et al., 1998; Moline et al., 1997); and in fresh-cut celery (*Apium graveolens* L.) and peppers (*Capsicum annuum*) MJ reduced microbial contamination (Buta and Moline, 1998).

In a similar manner to our findings in grapefruit (Figs. 1A and 2), 10-µmol·L⁻¹ JA or MJ was the most effective concentration in reducing decay also in strawberries (Moline et al., 1997) and in fresh-cut celery and peppers (Buta and Moline, 1998). However,
in the other plant systems examined, much higher jasmonate concentrations were required. For example, the most effective concentrations for reducing decay were 200 µmol·L⁻¹ MJ in cut roses (Meir et al., 1998), 300 µmol·L⁻¹ jasmonates in potato and tomato plants (Cohen et al., 1993), and 4.8 µmol·L⁻¹ 1A in barley plants (Schweizer et al., 1993). Similar to our present findings in grapefruit (Figs. 1A and 2), MJ is most effective in cut roses in reducing decay when applied in the pulsing solution at an optimal concentration (200 µmol·L⁻¹), but is less effective at either higher or lower concentrations (Meir et al., 1998).

We have demonstrated previously that postharvest application of MJ could significantly reduce CI after cold storage of various chilling-susceptible fruit, such as avocado (Persea americana Mill. ‘Hass’), grapefruit and bell pepper (Meir et al., 1996). A similar MJ protection against CI was reported previously for zucchini squash (Cucurbita pepo L.) also (Wang and Bata, 1994). The results of the present study further confirm that 10 µmol·L⁻¹ MJ was the optimal concentration for reducing CI in grapefruit after 6 weeks of cold storage and 4 additional d of shelf life (Fig. 1B, Table 1). It is well known that low-temperature stresses render the commodity to become more susceptible to postharvest pathogens (Bramlage and Meir, 1990). Indeed, storage of grapefruit at temperatures lower than 11 °C has resulted in CI, expressed as peel pitting of the fruit, increased attack by mold pathogens and stem end rots (Chalutz et al., 1981). In addition, the present statistical analysis clearly shows that the effects of MJ on CI and decay development depend on concentration and duration of the shelf life (Table 1). Therefore, it could be speculated that the inhibitory effect of MJ on the decay of cold-stored fruit (Fig. 1A) may be a result of its inhibitory effect on CI (Fig. 1B), which enhances fruit susceptibility to fungal attack. However, our findings strongly suggest that this is not the case. Firstly, there was no strict correlation between the MJ dose dependency of the two processes: 50 µmol·L⁻¹ MJ, which was the most effective concentration for decay reduction after 6 weeks of cold storage and 4 d of shelf life, was much less effective for CI reduction during this period (Fig. 1 and Table 1). On the other hand, 10 µmol·L⁻¹ MJ was the optimal concentration for decay reduction after 21 d of shelf life (Fig. 1A).

At this time, CI could not be evaluated because of the high percentage of decayed fruit, and because the sporulated fungus has completely covered the fruit surface. Secondly, this 10 µmol·L⁻¹ MJ concentration was also the most effective dose to protect grapefruit against decay caused by artificial inoculation with P. digitatum at 24 °C (Fig. 2). It appears therefore, that this dose of jasmonates is the optimal one to induce fruit resistance against decay in general, regardless of storage temperature.

MJ dipping of grapefruit seems to provide systemic protection against the green mold rot, P. digitatum, by eliciting resistance responses in the treated fruit. This conclusion is based on the following findings of the present study. a) Postharvest application of MJ to grapefruit significantly reduced the natural development of green mold decay during cold storage and subsequent shelf life at 20 °C (Fig. 1A, Table 1). b) The optimal dipping dose of MJ obtained for rot inhibition in these fruit was 10 µmol·L⁻¹ (Fig 1A and Table 1). c) This 10 µmol·L⁻¹ MJ (or 1A) concentration was also the optimal effective dose in reducing disease incidence after artificial inoculation of grapefruit with P. digitatum at 24 °C (Fig. 2). d) Jasmonates had no inhibitory effect on P. digitatum in vitro, even at high concentrations. It appears therefore, that the effects of jasmonates in reducing decay development in grapefruit were indirect, and operated through enhancement of the fruit natural defenses.

Indeed, in the literature, it is well established that jasmonates induce disease resistance and related responses in a wide variety of plants and cell cultures (Andresen et al., 1992; Chaudhry et al., 1994; Creelman et al., 1992; Creelman and Mullet, 1997; Doares et al., 1995; Farmer and Ryan, 1990; Gundlach et al., 1992; Nojiri et al., 1996; Xu et al., 1994). However, the mode of action of jasmonates in reducing disease development seems to differ among the various plants and pathogens examined. As in grapefruit, jasmonates reduce Phytophthora infestans in tomato and potato plants at much lower concentrations (300 µmol·L⁻¹) than those required (3.5 to 5 µmol·L⁻¹) for direct inhibition of the fungus (Cohen et al., 1993), and it was, therefore, suggested that jasmonates might protect the plants through the induction of host resistance responses (Cohen et al., 1993). In barley plants, however, JA reduces Erysiphe graminis only at high concentrations that inhibit appressoria formation and therefore, probably directly inhibit the fungus growth (Schweizer et al., 1993). On the other hand, in cut roses we previously showed that low MJ concentrations (200 µmol·L⁻¹) provide systemic protection against Botrytis cinerea by enhancing petal resistance, whereas 400 µmol·L⁻¹ MJ completely inhibits spor germination and germ-tube elongation of the fungus in vitro (Meir et al., 1998). Overall, it seems that the effects and mode of action of jasmonates in reducing disease development differ among the various crops and pathogens examined.

Another possible explanation for the mode of action of jasmonates in inducing disease resistance, may be by their induction of ethylene synthesis (Porat et al., 1993; Saniewski and Czapski, 1985). Especially, since it is known that ethylene induces the resistance of citrus fruit to P. digitatum (El-Kazzaz et al., 1983). However, there are also other cases in which jasmonates inhibit ethylene production (Nojavan-Aghari and Ishizawa, 1998). In cut roses, we found that jasmonate-induced resistance to pathogens is not ethylene mediated, since the MJ treatment was assayed in the presence of an ethylene antagonist (Meir et al., 1998). The possible effects of jasmonates on ethylene production in grapefruit were not evaluated in the current study.

In summary, our results clearly suggest that, when applied at low concentrations, jasmonates are potential postharvest treatments to enhance natural resistance and to reduce decay and CI in grapefruit. Since they are naturally occurring compounds and are given in low doses, jasmonates may provide a more environmentally friendly means to reduce the current massive use of chemical fungicides to control postharvest decay of grapefruit.

**Literature Cited**

Andresen, I., W. Becker, K. Schluter, J. Burges, B. Parthier, and K. Apel. 1992. The identification of leaf thionin as one of the main jasmonate-induced proteins in barley (Hordeum vulgare). Plant Mol. Biol. 19:193–204.

Bramlage, W.J. and S. Meir. 1990. Chilling injury of crops of temperate origin, p. 37–49. In: C.Y. Wang (ed.). Chilling injury of horticultural crops. CRC Press Inc., Boca Raton, Fla.

Buta, J.G. and H.E. Moline. 1998. Methyl jasmonate extends shelf life and reduces microbial contamination of fresh-cut celery and peppers. J. Agr. Food Chem. 46:1253–1256.

Chalutz, E., Y. Waks, and M. Schiffman-Nadel. 1981. The different induced proteins in barley (Hordeum vulgare). Plant Mol. Biol. 19:193–204.

Chaudhry, B., F. Muller-Uri, V. Cameron-Mills, S. Gough, D. Simpson, K. Skriver, and J. Mundy. 1994. The barley 60 kDa jasmonate-induced protein (JIP60) is a novel ribosome-inactivating protein. Plant J. 6:815–824.

Chen, E. 1988. Commercial use of long term storage of lemon with intermittent warming. HortScience 23:400–403.

Cohen, Y., U. Gisi, and T. Niderman. 1993. Local and systemic protection
against *Phytophthora infestans* induced in potato and tomato plants by jasmonic acid and jasmonic methyl ester. Phytopathology 83:1054–1062.

Creelman, R.A. and J.E. Mullet. 1995. Jasmonic acid distribution and action in plants: Regulation during development and responses to biotic and abiotic stress. Proc. Natl. Acad. Sci. USA 92:4114–4119.

Creelman, R.A. and J.E. Mullet. 1997. Biosynthesis and action of jasmonates in plants. Annu. Rev. Plant Physiol. Plant Mol. Biol. 48:355–381.

Creelman, R.A., M.L. Tierney, and J.E. Mullet. 1992. Jasmonic acid/methyl jasmonate accumulate in wounded soybean hypocotyls and modulate gene expression. Proc. Natl. Acad. Sci. USA 89:4938–4941.

Creelman, R.A., M.L. Tierney, and J.E. Mullet. 1992. Jasmonic acid/methyl jasmonate accumulate in wounded soybean hypocotyls and modulate gene expression. Proc. Natl. Acad. Sci. USA 89:4938–4941.

Doares, S.H., T. Syrovets, E.W. Weiler, and C.A. Ryan. 1995. Oligogalacturonides and chitosan activate plant defense genes through the octadecanoid pathway. Proc. Natl. Acad. Sci. USA 92:4095–4098.

Eckert, J.W. and G.E. Brown. 1986. Postharvest citrus diseases and their control. p. 315–360. In: W.F. Wardowski, S. Nagy, and W. Grierson (eds.). Fresh citrus fruits. AVI Publishing Co. Inc., Westport, Conn.

Eckert, J.W. and J.M. Ogawa. 1985. The chemical control of postharvest diseases: subtropical and tropical fruits. Annu. Rev. Phytopathol. 23:421–454.

El-Kazzaz, M.K., A. Chordas, and A.A. Kader. 1983. Physiological and compositional changes in orange fruit in relation to modification of their susceptibility to *Penicillium digitatum* by ethylene treatments. J. Amer. Soc. Hort. Sci. 108:618–621.

Farmer, E.E. and C.A. Ryan. 1990. Interplant communication: Airborne methyl jasmonate induces synthesis of proteinase inhibitors in plant leaves. Proc. Natl. Acad. Sci. USA 87:7713–7716.

Gundlach, H., M.J. Muller, T.M. Kutchan, and M.H. Zenk. 1992. Jasmonic acid is a signal transducer in elicitor-induced plant cell cultures. Proc. Natl. Acad. Sci. USA 89:2389–2393.

Meir, S., S. Philosoph-Hadas, S. Lurie, S. Droby, M. Akerman, G. Zauberman, B. Shapiro, E. Cohen, and Y. Fuchs. 1996. Reduction of chilling injury in stored avocado, grapefruit, and bell pepper by methyl jasmonate. Can. J. Bot. 74:870–874.

Meir, S., S. Droby, H. Davidson, S. Alsevia, L. Cohen, B. Horev, and S. Philosoph-Hadas. 1998. Suppression of Botrytis rot in cut rose flowers by postharvest application of methyl jasmonate. Postharvest Biol. Technol. 13:235–243.

Moline, H.E., J.G. Buta, R.A. Saftner, and J.L. Maas. 1997. Comparison of three volatile natural products for the reduction of postharvest decay in strawberries. Adv. Strawberry Res. 16:43–48.

Nojavan-Asgjari, M. and K. Ishizawa. 1998. Inhibitory effects of methyl jasmonate on germination and ethylene production in cocklebur seeds. J. Plant Growth Regul. 17:13–18.

Nojiri, H., M. Sugimori, H. Yamane, Y. Nishimura, A. Yamada, N. Shibuya, O. Kodama, N. Murofushi, and T. Omori. 1996. Involvement of jasmonic acid in elicitor-induced phytoalexin production in suspension-cultured rice cells. Plant Physiol. 110:387–392.

Porat, R., A. Borochov, and A.H. Halevy. 1993. Enhancement of petunia and dendrobium flower senescence by jasmonic acid methyl ester is via the promotion of ethylene production. Plant Growth Regul. 13:297–301.

Rodov, V., S. Ben-Yehoshua, R. Albagli, and D.Q. Fang. 1995. Reducing chilling injury and decay of stored citrus fruit by hot water dips. Postharvest Biol. Technol. 5:119–127.

Saniewski, M. and J. Czapski. 1985. Stimulatory effect of methyl jasmonate on the ethylene production in tomato fruits. Experientia 41:256–257.

Schiffmann-Nadel, M., E. Chalutz, J. Waks, and M. Dagan. 1975. Reduction of chilling injury in grapefruit by thiabendazole and benomyl during long term storage. J. Amer. Soc. Hort. Sci. 100:270–272.

Schweizer P., R. Gees, and E. Mosinger. 1993. Effect of jasmonic acid on the interaction of barley (*Hordeum vulgare* L.) with the powdery mildew *Erysiphe graminis* f.sp. *hordei*. Plant Physiol. 102:503–511.

Sembdner, G. and B. Parthier. 1993. The biochemistry and the physiological and molecular actions of jasmonates. Annu. Rev. Plant. Physiol. Plant Mol. Biol. 44:569–589.

Wang, C.Y. 1993. Approaches to reduce chilling injury of fruits and vegetables. Hort. Rev. 15:83–95.

Wang, C.Y. and J.G. Buta. 1994. Methyl jasmonate reduces chilling injury in *Cucurbita pepo* through its regulation of abscisic acid and polyamine levels. Environ. Expt. Bot. 34:427–432.

Xu, Y., P.L. Chang, D. Liu, M.L. Narasimhan, K.G. Raghothama, P.M. Hasegawa, and R.A. Bressan. 1994. Plant defense genes are synergistically induced by ethylene and methyl jasmonate. Plant Cell 6:1077–1085.