INTRODUCTION

As sessile organisms, plants are presented with numerous biotic challenges such as herbivory and pathogen attack. Plants initiate responses to these challenges by harnessing tightly regulated phytohormone networks. Salicylic acid (SA) levels increase in plants following pathogen infection and SA is critical for the development of systemic acquired resistance (SAR; Métraux et al., 1990; Rasmussen et al., 1991). There are two enzymatic pathways for the generation of SA: one via phenylalanine ammonia lyase and the other via isochorismate synthase (ICS). In tomato (Solanum lycopersicum), most pathogen-induced SA appears to be synthesized via the ICS pathway (Wildermuth et al., 2001; Uppalapati et al., 2003). SAR induction by biotic agents coincides with increases in SA levels (Iwai et al., 2007) and/or by acting on downstream targets of SA (Vernooij et al., 1995; Durrant and Dong, 2004). For example, the plant activator, probenazole, effective against bacterial, fungal, and oomycete diseases, stimulates SAR by increasing SA accumulation (Lawton et al., 1996). Tiadinil [TDL; N-(3-chloro-4-methylphenyl)-4-methyl-1,2,3-thiadiazole-5-carboxamide, TDL] is an effective for control of rice blast disease caused by Magnaporthe oryzae (Yasuda et al., 2006) and appears to induce resistance in a manner similar to BTH by acting on downstream targets of SA (Lawton et al., 1996; Yasuda et al., 2004). The TDL metabolite, N-(3-chloro-4-methylphenyl)-4-methyl-1,2,3-thiadiazole-5-carboxamide-3-methyl-ester (BTH), sold under the trade name, Actigard, is a SAR activator that was registered in Japan in 2003 under the trade name, V-GET. TDL was developed for disease management of SA-deficient transgenic plants expressing a bacterial salicylate hydroxylase (NahG; Gaffney et al., 1993) or ICS mutants like sid2 (Wildermuth et al., 2001), and mutants in downstream targets of SA such as npr1 (Mou et al., 2003). SAR induction by biotic agents coincides with increases in SA levels and a systemic transcriptional reprogramming that primes the plant to respond rapidly to minimize the spread or severity of further infections (Malamy et al., 1990; Métraux et al., 1990; Rasmussen et al., 1991; Vlot et al., 2009). This transcriptional reprogramming includes the expression of pathogenesis-related (PR) genes and deployment of peroxidases and other defense factors. In addition to induction by biotic agents, SAR responses are induced by exogenous application of SA to the foliage or roots (Ward et al., 1991).

Plant activators are chemicals that have no direct antimicrobial activity but induce disease resistance (Kessmann et al., 1994; Louws et al., 2001). A number of synthetic compounds have been developed that induce SAR by increasing SA accumulation (Iwai et al., 2007) and/or by acting on downstream targets of SA (Vernooij et al., 1995; Durrant and Dong, 2004). For example, the plant activator, probenazole, effective against bacterial, fungal, and oomycete diseases, stimulates SAR by increasing SA levels (Iwai et al., 2007). Tiadinil [TDL; N-(3-chloro-4-methylphenyl)-4-methyl-1,2,3-thiadiazole-5-carboxamide] is a plant activator that was registered in Japan in 2003 under the trade name, Actigard, stimulates SAR in many plant species without inducing SA accumulation (Lawton et al., 1996). Tiadinil is highly effective for control of rice blast disease caused by Magnaporthe oryzae (Yasuda et al., 2006) and appears to induce resistance in a manner similar to BTH by acting on downstream targets of SA (Lawton et al., 1996; Yasuda et al., 2004). The TDL metabolite, N-(3-chloro-4-methylphenyl)-4-methyl-1,2,3-thiadiazole-5-carboxamide-3-methyl-ester (BTH), sold under the trade name, Actigard, is a SAR activator that was registered in Japan in 2003 under the trade name, V-GET. TDL was developed for disease management of SA-deficient transgenic plants expressing a bacterial salicylate hydroxylase (NahG; Gaffney et al., 1993) or ICS mutants like sid2 (Wildermuth et al., 2001), and mutants in downstream targets of SA such as npr1 (Mou et al., 2003). SAR induction by biotic agents coincides with increases in SA levels and a systemic transcriptional reprogramming that primes the plant to respond rapidly to minimize the spread or severity of further infections (Malamy et al., 1990; Métraux et al., 1990; Rasmussen et al., 1991; Vlot et al., 2009). This transcriptional reprogramming includes the expression of.
ABA is well documented in relation to plant defense responses generated in the “New Yorker” background, similar to the method Ruhm” (Tal and Nevo, 1973), and seeds for these were obtained compared with its isogenic, wild-type (WT) background, “Rheinlands” roots from Phytophthora capsici to the soilborne oomycete pathogen USA). The homozygous ABA-deficient mutant Tomato plants (MATERIALS AND METHODS activators do not reverse the salt-induced increment in disease stressed and salt-stressed plants by chemically induced SAR, plant accumulation and, although overall disease is less in both non-stress. The results show that TDL applied to roots strongly pro-
BTH on ABA accumulation during a predisposing episode of salt stress. The objective of this study was to determine the effect of pretreatment of tomato seedlings with TDL and BTH in Arabidopsis and tobacco (Yasuda et al., 2008; Kusajima et al., 2010). However, it is not known if plant activators that target SA signaling impact the ABA-mediated susceptibility to root pathogens that occur following predisposing root stress in tomato. Because of the potential for unwanted tradeoffs and signaling conflicts in plants exposed to different stresses, as can occur in the field, we investigated how predisposing root stress impacts chem-
cally induced resistance in tomato. The objective of this study was to determine the effect of pretreatment of tomato seedlings with TDL and BTH on salt-induced predisposition to the foliar bacterial pathogen Pseudomonas syringae pv. tomato (Pst) and to the soilborne oomycete pathogen Phytophthora capsici. TDL is of particular interest in the context of soilborne pathogens such as Phytophthora capsici because it is often applied to plants as a root dip. We also determined the impact of SA, TDL, and BTH on ABA accumulation during a predisposing episode of salt stress. The results show that TDL applied to roots strongly pro-
tects the leaves from disease caused by Pst in both non-stressed and salt-stressed plants. In contrast, neither TDL nor BTH pro-
tects roots from Phytophthora capsici. The protection induced by plant activators against Pst does not result from reduced ABA accumulation and, although overall disease is less in both non-stressed and salt-stressed plants by chemically induced SAR, plant activators do not reverse the salt-induced increment in disease severity. MATE
RIALS AND METHODS PLANT MATERIAL AND GROWTH CONDITIONS Tomato plants (Solanum lycopersicum) of cultivars “New Yorker” or “Rheinlands Ruhm” and mutants within these backgrounds were used in experiments. “New Yorker” seeds were obtained from a commercial source (Totally Tomatoes, Randolph, WI, USA). The homozygous ABA-deficient mutant sitiens was com-
pared with its isogenic, wild-type (WT) background, “Rheinlands Ruhm” (Tal and Nevo, 1973), and seeds for these were obtained from the C.M. Rick Tomato Genetics Resource Center at the University of California, Davis. NahG transgenic plants were gen-
erated in the “New Yorker” background, similar to the method used by Guffey et al. (1993). The nahG construct containing the transgene salicylate hydroxylase under control of the CaMV 35S promoter in the binary vector pCIB200 was a gift of Syngenta Crop Protection, Inc. Tomato plants were grown in a hydroponic format. Prior to use, tomato seeds were surface sterilized with the following proto-
col: 50% HCl (10 min) and rinsed with sterile deionized H2O, 10% trisodium phosphate (15 min) and rinsed (3 ×) in sterile deionized H2O, 70% ethanol (10 min), and rinsed (3 ×) with sterile deionized H2O, and 50% commercial bleach (3% sodium hypochlorite; 20 min) followed by sterile deionized H2O rinse (3 ×). Follow-
ing surface-sterilization, seeds were placed on sterile germination paper in beakers containing sterile deionized H2O, transferred after 1 week to trimmed 5 ml polypropylene pipette tips, secured with foam test tube plugs, and placed into aerated hydroponic containers filled with 4 L of aerated, 0.5 × Hoagland’s solution. Seedlings were grown for an additional 2 weeks in a growth cham-
ber (150 μ mol m⁻² s⁻¹, 16 h photoperiod, 22°C, 70% RH) until at least two true leaves had developed on each plant. SA treatment. Plant activator treatment, salt treatments, and inoculation Four-week-old hydroponically grown tomato plants were immersed in 50 ml of 0.5 × Hoagland’s solution containing 10 ppm (37 μM) TDL (Nihon Nohyaku Co., Ltd), 10 ppm (47 μM) BTH (Syngenta Crop Protection, Inc.), 10 ppm (62 μM) salicylic acid-
sodium salt (SA; Sigma-Aldrich), or water for 7 days prior to salt stress and inoculation with a pepper isolate of Phytophthora capsici (from Volo County, CA; also pathogenic on tomato) or Pst (isolate B-64, gift of D. Cooksey). Pre-inoculation salt treatments consisted of exposing the roots to saline solution (0.2 M NaCl + 0.02 M CaCl₂) for 18 h. All seedlings collapsed within 10 min of expo-
sure to saline solution and regained full turgor within 2 hr of salt removal. Shoots were dip inoculated with 2-day-old Pst cultures adjusted to 1 × 10⁶ cfu ml⁻¹ in 1 L of 10 mM MgCl₂ with 80 μM Silwet L77. Roots were inoculated with 2 ml of zoospore suspension to achieve a final concentration of 1 × 10⁶ zoospores ml⁻¹. Pst and Phytophthora capsici disease analyses Four days post-inoculation (dpi) Pst-infected leaflets were surface sterilized with 70% EtOH for 10 s, rinsed in sterile H2O, and blotted dry. Samples were excised with a #3 hole punch (5 mm diameter) and ground in 200 μl 5 mM MgCl₂. A series of 10-fold dilutions were plated on King’s B medium; colonies were counted after 2 days of growth at 28°C. The relationship of disease and Phytophthora capsici DNA content was determined by quantitative polymerase chain reaction (qPCR; DiLeo et al., 2010). To correct for variability across samples, a similar amount of hypocotyl and root tissue was extracted for each sample and the qPCR analyses were performed on DNA extracts adjusted for total DNA content as measured with a Nanodrop spectrophotometer model ND-1000 (Thermo Fisher Scientific Inc., Wilmington, DE, USA). ABA analyses To determine the effect of SA on ABA accumulation during salt stress, ABA levels were measured in WT plants pre-treated with SA, TDL, or BTH. Following salt stress treatment for 18 h, roots and shoots were collected and immediately frozen in liquid N₂.
The tissues were lyophilized and placed at −20°C until extraction. The lyophilized tissue was ground in liquid N₂ to a fine powder with a mortar and pestle. 50–100 mg samples were collected, and each sample transferred to a microfuge tube. Cold 80% methanol (1.2 ml) containing butylated hydroxytoluene at 10 μg ml⁻¹ was added to each tube, which was then vortexed. The extracts were placed on ice and agitated occasionally for 30 min. The tubes were centrifuged for 5 min at 10,000 × g, and the supernatants collected. The pellet was extracted with 0.5 ml of 80% methanol and centrifuged to collect the supernatant. This step was repeated, all three supernatants were combined, and the methanol concentration of the extract adjusted to 70%. The extracts were applied to pre-wetted Sep-pak C18 columns (Waters, Inc., Milford, MA, USA) and eluted with 5 ml of 70% methanol. The eluate (∼7.5 ml) containing ABA was concentrated to near dryness at 37°C under vacuum and the volume adjusted to 300 μl with deionized water. The samples were analyzed by competitive immunoassay with an ABA immunoassay kit according to the manufacturer’s directions.
sums test or analysis was performed on all data sets. Log transformation was performed five times. SA accumulation was measured in one experiment. Experiments measuring ABA accumulation were three times with five replicates for each treatment within each experiment. Shoots from WT and NahG plants were processed using the same software (version 10.0; SAS Inc.) as indicated.

The Pst TDL did not prevent the proportional increase in both non-stressed and salt-treated seedlings (plants. Pretreatment with TDL at 10 ppm significantly reduced higher titer of pathogen (Figure 2) than non-stressed, inoculated plants. Pretreatment with TDL at 10 ppm significantly reduced Pst colonization and symptom severity in “New Yorker” plants in both non-stressed and salt-treated seedlings (Figure 2). However, TDL did not prevent the proportional increase in Pst colonization observed in salt-stressed plants relative to the non-stressed controls. Since TDL harnesses SA-mediated defenses, we treated SA-deficient NahG plants to see if TDL induces resistance under the different stress regimes in this highly susceptible background. As expected, NahG plants were more susceptible to Pst (Figure 2) and accumulated significantly less SA following Pst infection (data not shown) than the WT background “New Yorker.” However, TDL provided strong protection in the NahG plants and mitigated the predisposing effect of salt-stress on bacterial speck disease.

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**RESULTS**

**TDL PROTECTS TOMATO AGAINST THE BACTERIAL SPECK PATHOGEN**

***Pst IN NON-STRESSED AND SALT-STRESSED SEEDLINGS***

To determine if plant activators induce resistance to Pst under different stress regimes in our experimental format, roots of hydroponically grown seedlings of cv. “New Yorker” were treated with TDL and then either not salt-stressed or exposed to 0.2 M NaCl for 18 h prior to inoculation. In preliminary experiments, several concentrations of TDL were evaluated for phytotoxicity and for efficacy against bacterial speck disease with 10 ppm (37 µM) TDL selected as this concentration provided an optimal response. Concentrations higher than 10 ppm of TDL caused a slight bronzing of the roots and depressed growth of the seedlings, suggesting a mild phytotoxicity of the chemical in our experimental format at these higher levels. Inoculated salt-stressed seedlings (cv. “New Yorker” and NahG) were treated in the same format and stress regimes as above. TDL significantly reduced Pst colonization and symptom severity in “New Yorker” plants in both non-stressed and salt-treated seedlings (Figure 2). However, TDL did not prevent the proportional increase in Pst colonization observed in salt-stressed plants relative to the non-stressed controls.
reduced symptoms (Figure 3) and colonization by the pathogen than the background “Rheinlands Ruhm” (Figure 4). Nonetheless, TDL pretreatment of sitiens provided further protection against Pst (Figure 4).

**TDL AND BTH DO NOT REDUCE Phytophthora capsici DISEASE SEVERITY**

To determine if plant activators protect tomato roots and crowns against the oomycete pathogen, *Phytophthora capsici*, and predisposing root stress, tomato seedlings were treated with TDL or BTH (10 ppm), not stressed or salt-stressed as above, and then inoculated. There was no protection provided by the plant activators against disease caused by *Phytophthora capsici* in either the control or salt-treated plants, as reflected in symptom severity (not shown) and pathogen colonization (Figure 5).

**IMPACT OF SALINITY STRESS AND PLANT ACTIVATORS ON ROOT AND SHOOT ABA LEVELS**

Because elevated levels of ABA in tomato can enhance susceptibility to *Pst* (Mohr and Cahill, 2007) and *Phytophthora capsici*
not reduce ABA content relative to untreated plants (DiLeo et al., 2010). The effect of SA, TDL, and BTH on ABA levels was determined in roots and shoots. ABA concentrations in either shoots or roots at the time selected for inoculation in our treatment sequence were not altered by SA (Figure 6). However, a trend of increasing ABA accumulation was observed in TDL- and BTH-treated “New Yorker” plants relative to the corresponding control plants (Figure 7). Although the increase inABA accumulation in the plants treated with these plant activators is not statistically significant at a P ≤ 0.05, it can be said that SA, TDL, and BTH do not reduce ABA content relative to untreated plants (Figure 7). In addition, salt stress did not further increase the levels of ABA in plants that had been pretreated with TDL or BTH, which were similar to the salt stressed controls.

**DISCUSSION**

In a previous study, we demonstrated the predisposing effect of salt stress and a role for ABA as a determinative factor in predisposition in the tomato–Phytophthora capsici interaction (DiLeo et al., 2010). The present study is the first report of salt-induced predisposition to the bacterial speck pathogen, Pst, in tomato. Furthermore, the results with the ABA-deficient sitiens mutant are consistent with the salt-induced susceptibility to Pst being mediated by ABA (Figure 4). These results conform to studies in Arabidopsis where ABA has been reported to promote susceptibility to Pst (de Torres-Zabala et al., 2007; Yasuda et al., 2008).

Because SA has been shown to protect tomato against salt stress, possibly by an ABA-dependent mechanism (Sassies et al., 2009), plant activators that operate via the SA pathway were evaluated for effect on salt-induced predisposition. Protection of tomato against bacterial speck disease by BTH is well documented (Louws et al., 2001), and TDL has previously been shown to reduce the severity of bacterial and fungal infections without inducing SA accumulation (Yasuda et al., 2004, 2006). Here, TDL was shown to protect against Pst in both non-stressed and salt-stressed tomato plants. TDL pretreatment strongly reduced disease and colonization by Pst in both “New Yorker” and SA-deficient NahG plants. TDL, or more likely its biologically active metabolite, SV-05, presumably allows the NahG plants to mount an SAR response to Pst infection in the absence of SA accumulation (Figure 2). TDL provided protection in both non-stressed and salt-stressed plants, but did not reverse the predisposing effect of salt stress. An increase in Pst colonization was observed in the salt-stressed, TDL-pretreated plants of both genotypes, with comparable percentage increases relative to the corresponding non-stressed controls in “New Yorker” and NahG plants. This indicates that TDL does not reverse the salt-stress effect on disease, per se, and likely targets stress network signaling independently of an ABA-mediated process that conditions the salt-induced susceptibility observed in this system (Figures 2 and 4).

“Rheinlands Ruhm” also displayed salt-induced predisposition to Pst. Pretreatment with TDL significantly reduced Pst colonization in both “Rheinlands Ruhm” and sitiens (Figure 4). Similarly, TDL provided protection in both non-stressed and salt-stressed plants, but did not reverse the predisposing effect of salt stress in “Rheinlands Ruhm” plants. The salt-induced increment in colonization by the pathogen was comparable in both the untreated and TDL-pretreated plants (Figure 4). The ABA-deficient mutant, sitiens, is considerably less susceptible to Pst than its background “Rheinlands Ruhm,” and does not exhibit salt-induced predisposition (Figures 3 and 4).

![Figure 4](image-url)
Protection by plant activators against foliar pathogens is well established (Louws et al., 2001; Yasuda et al., 2004). However, relatively few studies have examined these compounds against soilborne pathogens and so TDL and BTH were evaluated for protection against root infection by Phytophthora capsici. Neither TDL nor BTH induced resistance or impacted salt-induced predisposition to Phytophthora capsici (Figure 5). Phytophthora capsici is an aggressive root and crown pathogen with a hemibiotrophic parasitic habit (Lamour et al., 2012) that triggers both SA- and jasmonic acid-mediated responses during infection of tomato (unpublished data). The results suggest that SA responses in tomato play a less important role in defense against Phytophthora capsici than to Pst.

The impact of SA and plant activators on ABA accumulation was measured in tomato roots and shoots. SA treatment and SA-deficiency conferred by NahG did not significantly impact ABA levels (Figure 6). However, ABA accumulation in non-stressed TDL and BTH treatments trended higher than those observed in salt-stressed plants that did not receive a plant activator treatment (Figure 7). Protection by TDL against Pst is likely the result of a triggered SAR response and not the result of an antagonistic effect on ABA levels. The efficacy of plant activators depends on the specific diseases targeted and the environmental context, which may present additional stressors to confound defense network signaling in the plant. A challenge for successful deployment of plant activators in the field is to manage the allocation, ecological and fitness costs that are associated with induced defenses (Heil, 2001; Heil and Baldwin, 2002; Heil and Bostock, 2002; Berger et al., 2007). These costs can be manifested by reduced growth and reproduction, vulnerability to other forms of attack, and potential interference with beneficial associations (Bostock, 2005). It would seem that the severity of these costs is conditioned in part by the milieu of abiotic stressors operative at any given time. Reactive oxygen species (ROS) contribute to the initiation of SAR (Alvarez et al., 1998), are induced by SA and BTH (Fitzgerald et al., 2004; van der Merwe and Dubery, 2006), and are essential co-substrates for induced defense responses such as lignin synthesis (Hammerschmidt and Kuc, 1982). ROS also are important in modulating abiotic stress networks, for example in ABA signaling and response (Cho et al., 2009). The potential compounding effect of ROS generated from multiple stressors presents a dilemma in that the plant
must reconcile these to adapt or else suffer the negative consequences of oxidative damage for failure to do so (Foyer and Noctor, 2003) (Paradoxically, SA and BTH also are reported to protect plants against paraquat toxicity, which involves ROS generation for its herbicidal action (Silverman et al., 2003). How plants balance ROS's signaling roles and destructive effects within multiple stress contexts is unresolved and a critically important area of plant biology with relevance for optimizing induced resistance strategies in crop protection (Van Breusegem et al., 2008; Foyer and Noctor, 2009). Although our experiments were conducted under highly controlled conditions, the results with TDI are encouraging and show that chemically induced resistance to bacterial speck disease occurs in both salt-stressed and non-stressed plants and in plants severely compromised in SA accumulation. Future research with plant activators should consider their use within different abiotic stress contexts to fully assess outcomes in disease and pest protection.

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