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Salicylic Acid Induces Resistance in Potatoes Against *Rhizoctonia solani*, the Cause of Black Scurf and Stem Canker

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Abstract: Trials were conducted at the Potato Development Centre, Wicklow, New Brunswick, Canada to study the effect of Salicylic Acid (SA) in suppressing black scurf (*Rhizoctonia solani* Kuhn, AG-3) disease in potatoes (cv. Atlantic) under screenhouse conditions. The trials were designed as a completely randomized block and comprised of eight replicated treatments: Untreated, uninoculated control (CTH); untreated control inoculated with *R. solani* (CTD); healthy seed treated with SA (STH); seeds inoculated with *R. solani* and treated with SA (STD); healthy seeds with SA applied foliarly (FAH); seeds inoculated with *R. solani* and SA applied foliarly (FAD); healthy seeds with SA applied as soil drench (SDH); seeds inoculated with *R. solani* and SA applied as soil drench (SDD). Seedling emergence, plant canopy, disease severity of black scurf and total tuber weights were recorded. Plant canopy was significantly higher in plants inoculated with *R. solani* and treated with SA compared to uninoculated treatments. All SA treatments significantly reduced black scurf disease severity in stems compared to the untreated, inoculated controls. Compared to the CTD, black scurf disease severity in stems was reduced by 89.6 and 88.8% when FAH and SDH treatments, respectively, were used. Treatments inoculated with *R. solani* and treated with SA significantly increased potato tuber weights compared to the uninoculated controls. Present findings indicate that SA has the potential to be used as an alternative tool in managing black scurf disease in potatoes.

Key words: *Rhizoctonia solani*, acetyl salicylic acid, *Solanum tuberosum*, systemic acquired resistance

INTRODUCTION

Black scurf (*Rhizoctonia solani* Kuhn, AG-3) is a common disease of potatoes which is capable of causing significant reductions in tuber quality and yield worldwide (Anderson, 1982; Little *et al.*, 1988; Carling *et al.*, 1989; Powelson *et al.*, 1993; Jeger *et al.*, 1996; Tsrer *et al.*, 1996). The disease is more prevalent under cool and moist environmental conditions (Bandy *et al.*, 1988; Carling *et al.*, 1989; Banville, 1989) and may not occur in drier and warmer conditions (Davis, 1978; Weinhold *et al.*, 1982). Typical disease symptoms include black sclerotia formation on tubers and the presence of sunken necrotic lesions on roots, stolons and subterranean portions of the main stem (Carling and Leiner, 1986). The severity of black dot is not always associated with yield reduction. However, the formation of tuber-borne sclerotia downgrades quality of tubers, resulting in malformed tubers and an alteration in target size and tuber numbers (Anderson, 1982; Carling *et al.*, 1989; Jeger *et al.*, 1996). Infection of potato plants with *R. solani* can be caused by soil borne inoculum, tuber-borne inoculum, or via infected seed tubers (Banville, 1978; Platt, 1989; Powelson *et al.*, 1993; Platt *et al.*, 1993). Studies have shown that both tuber-borne and soil-borne inocula are important in the development of black scurf disease in potatoes (Frank and Leach, 1980; Tsrer and Peretz-Alon, 2005). It is also suggested that the control of tuber-borne inoculum is essential for the integrated management of *R. solani* in potatoes

(Frank and Leach, 1980). Losses caused by this pathogen can be minimized by using disease free seed tubers, planting seed near the soil surface and following a rotation scheme with two or more years between potato crops. In some cases, an increase in black scurf of potato was observed following crop rotation (Celetti *et al.*, 1990; Errampalli *et al.*, 1999). In addition, fungicides effective against soil-borne fungal pathogens of potato, including *R. solani*, are very limited (Powelson *et al.*, 1993; Wicks *et al.*, 1995; Johnston, 1995).

The phenomenon by which a plant exhibits an increased level of resistance to infection by a pathogen after appropriate stimulation is called induced disease resistance. The two forms of induced resistance that are clearly defined are Systemic Acquired Resistance (SAR) and Induced Systemic Resistance (ISR). These two forms of resistance can be differentiated from each other on the basis of the nature of elicitor and the regulatory pathways involved (Knoester *et al.*, 1999; Maleck *et al.*, 2000; Schenk *et al.*, 2000; van Wees *et al.*, 2000; Yan *et al.*, 2002). In SAR, a plant's own defense mechanisms are triggered by prior treatment with either a biological agent or chemical agent. Salicylic acid is an endogenous signaling molecule required for the induction of SAR (Klessig and Malamy, 1994). SAR is associated with induction of a group of Pathogenesis-Related (PR) genes and their corresponding proteins (Pieterse *et al.*, 1996; Delaney, 2000). In ISR, the resistance is activated by biotic and abiotic inducing agents and is dependent on the host plant's physical or chemical barriers (Kloepper *et al.*, 1992; Kessmann *et al.*, 1994). Contrary to SAR, ISR does not involve accumulation of PR proteins but relies on pathways regulated by jasmonate and ethylene (Pieterse *et al.*, 1998; Knoester *et al.*, 1999; Yan *et al.*, 2002). ISR is brought about by certain group of plant growth promoting rhizobacteria, which includes several species of *Pseudomonas*, without causing any damage to the plant's root system (van Loon *et al.*, 1998). It is believed that different antimicrobial proteins are induced in the two pathways, but the distinction of terminology is somewhat confused due to the possibility of crosstalk between SAR and ISR (Dong, 2001). Plants successfully resisting a pathogen during SAR have the capability to become highly resistant to subsequent infection not only by the original pathogen but also by other pathogens (Coquoz *et al.*, 1995; Lawton *et al.*, 1996). Acetyl Salicylic Acid (ASA; the active ingredient in aspirin) is a product known to provoke the SAR in plants, therefore raising their natural defense against potential diseases (Gaffney *et al.*, 1993; Lawton *et al.*, 1995; Dempsey *et al.*, 1999; Verberne *et al.*, 2000; Rairdan and Delaney, 2002). SA applied as seed treatment, spray and soil drench, reduced the number of lesions, lesion size and the percentage of necrotic leaf area following infection by *Phytophthora palmivora* in cacao (Okey and Sreenivan, 1996). However, SA has not been tested against potato diseases including black scurf. The objective of the present study was to assess the effect of SA in reducing the severity of black scurf under screenhouse conditions.

MATERIALS AND METHODS

Screenhouse Experiments

Screenhouse experiments were conducted at the Potato Development Centre, Wicklow, New Brunswick, Canada. Disease-free seed potato tubers (cv. Atlantic) were washed, air dried and used in the experiments. A solution of salicylic acid (SA) [$\text{HOOC}_6\text{H}_4\text{COOH}$, crystals, FW 138.12, EM Science, USA] was prepared by mixing 400 mg of SA in 600 μL of tween 80 and 200 mL of sterile distilled water (Bokshi *et al.*, 2003). This solution was diluted with distilled water to obtain a final volume of 6 L.

Experimental Set up

The experiment was designed as a one way completely randomized block and consisted of 8 treatments which were replicated four times. Each replicate consisted of 5 potato plants

(cv. Atlantic). The eight treatments were: 1) untreated, uninoculated control (CTH); 2) untreated control inoculated with *R. solani* (CTD); 3) healthy seed treated with SA (STH); 4) seed inoculated with *R. solani* and treated with SA (STD); 5) healthy seed with SA applied as foliar spray (FAH); 6) seed inoculated with *R. solani* with SA applied as foliar spray (FAD); 7) healthy seed with SA applied as soil drench (SDH); 8) seed inoculated with *R. solani* with SA applied as soil drench (SDD). The experiment (8 treatments x 4 replicates x 5 plants) was repeated twice. Pots were filled up to half with professional sphagnum peat-based growing medium (Premier Pro-Mix®, Premier Horticulture Ltée, Rivière-du-Loup, Québec) and used for the trial. All purpose plant food fertilizer was applied once every two weeks (Greenleaf Shur-Gro®, Greenleaf Products Inc., Burnaby, BC, N-P-K 20-20-20 applied at the rate of 5 mL in 4 L of water).

Rhizoctonia solani inoculum was prepared by culturing a naturally infected piece of tuber on Potato Dextrose Agar (PDA). The culture obtained was purified and multiplied on fresh PDA plates and allowed to grow for seven days at room temperature. Organic rye seeds were soaked in water for 24 h and then autoclaved at 121°C for 15 min. Each bag of rye seed (1 kg) received six plates of *R. solani* culture cut up into 1 inch cubes under aseptic conditions. The bags were then stored at room temperature for 60 days and were shaken weekly. The autoclaved, organically-grown rye seed inoculated with *R. solani* served as inoculum for the screenhouse studies. For treatments where inoculation was required, 50 g of *R. solani* infected rye seed was added on and around each potato seed piece at planting.

Seedling emergence was recorded once every 2 days starting 21 days after planting and until all plants emerged. A foliar spray and soil drench was carried out after full emergence, 5 weeks after planting. The solution used in foliar spray and soil drench was the same as the one used in seed treatment. It consisted of a mixture of 448 mg SA, 112 µL of Tween 80 and 1.12 L of water. The tween was used in order to make SA more soluble in water. The concentration needed was 0.01% of the final volume of the solution. The final concentration of SA was 400 mg L⁻¹.

The amount of spray applied was based on the standard pesticide application rate of 101 L acre⁻¹. This amounted for 14 mL of solution per plant. The soil drench was applied using a disposable pipette. However, for the foliar application, a spray bottle was calibrated in order to determine the number of sprays needed to deliver 14 mL. The tubers were harvested and were later assessed for disease severity on a scale of 0-100% (Cruickshank *et al.*, 1982; Dorrance and Inglis, 1997). Data were analyzed using CoStat (CoHort Software, Monterey, CA, USA) and the means were separated using LSD test at $p = 0.05$.

RESULTS

Seedling emergence was significantly higher in the uninoculated treatments compared to the inoculated ones in the initial stages. As the days progressed, the emergence in *R. solani* inoculated treatments did not differ significantly compared to uninoculated treatments (Fig. 1). In the initial stages significant differences in emergence were observed between FAH (25%), SDH (25%) and CTH (25%) over FAD (0%), SDD (0%), CTD (0%) and STD (0%). Over time, the significant differences in emergence were observed between all uninoculated treatments and CTD and STD (Fig. 1).

Plant canopy was significantly higher in plants inoculated with *R. solani* and STD (12.19 L plant⁻¹), FAD (13.48 L plant⁻¹) and SDD (12.31 L plant⁻¹) treatments over the uninoculated treatments (Fig. 2). The highest canopy was for FAD (13.48 L plant⁻¹) followed by SDD (12.31 L plant⁻¹), STD (12.19 L plant⁻¹) and CTD (9.37 L plant⁻¹). Canopies of all SA treated plants were significantly higher than that for CTD.

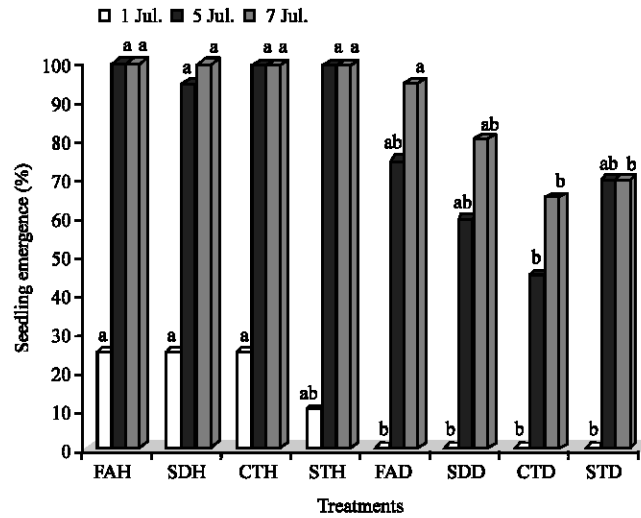


Fig. 1: Seedling emergence of potato tubers inoculated with *Rhizoctonia solani* and treated with salicylic acid. CTH = uninoculated untreated control; STH = healthy seed treated with salicylic acid; FAH = healthy seeds with salicylic acid applied as a foliar spray; SDH = healthy seeds with salicylic acid applied as a soil drench; CTD = inoculated untreated control; STD = seeds infected with *R. solani* and treated with salicylic acid; FAD = seeds infected with *R. solani* with salicylic acid applied as a foliar spray and SDD = seeds infected with *R. solani* with salicylic acid applied as a soil drench. Bars followed by the same letters are not significantly different from each other at $p = 0.05$

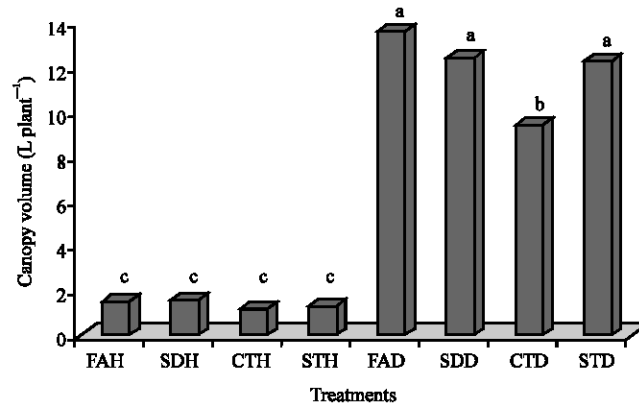


Fig. 2: Plant canopy measurements of potato tubers inoculated with *Rhizoctonia solani* and treated with salicylic acid. CTH = uninoculated untreated control; STH = healthy seed treated with salicylic acid; FAH = healthy seeds with salicylic acid applied as a foliar spray; SDH = healthy seeds with salicylic acid applied as a soil drench; CTD = inoculated untreated control; STD = seeds infected with *R. solani* and treated with salicylic acid; FAD = seeds infected with *R. solani* with salicylic acid applied as a foliar spray and SDD = seeds infected with *R. solani* with salicylic acid applied as a soil drench. Bars followed by the same letters are not significantly different from each other at $p = 0.05$

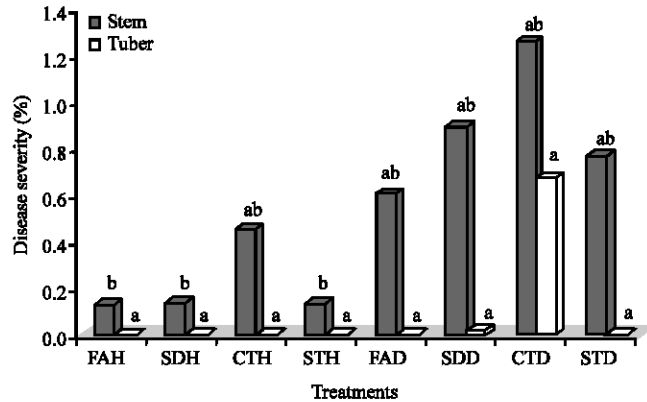


Fig. 3: Disease severity of black scurf in potato tubers inoculated with *Rhizoctonia solani* and treated with salicylic acid. CTH = uninoculated untreated control; STH = healthy seed treated with salicylic acid; FAH = healthy seeds with salicylic acid applied as a foliar spray; SDH = healthy seeds with salicylic acid applied as a soil drench; CTD = inoculated untreated control; STD = seeds infected with *R. solani* and treated with salicylic acid; FAD = seeds infected with *R. solani* with salicylic acid applied as a foliar spray; and SDD = seeds infected with *R. solani* with salicylic acid applied as a soil drench. Bars followed by the same letters are not significantly different from each other at $p = 0.05$

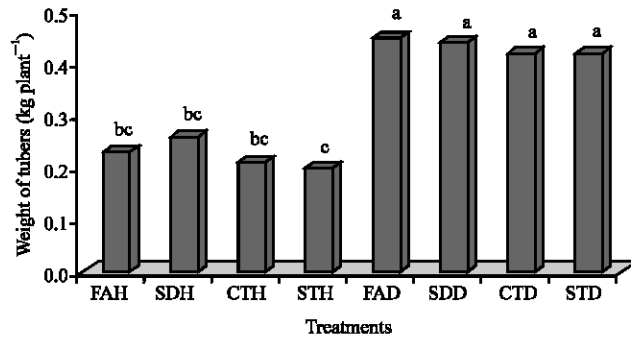


Fig. 4: Total weight of tubers inoculated with *Rhizoctonia solani* and treated with salicylic acid. CTH = uninoculated untreated control; STH = healthy seed treated with salicylic acid; FAH = healthy seeds with salicylic acid applied as a foliar spray; SDH = healthy seeds with salicylic acid applied as a soil drench; CTD = inoculated untreated control; STD = seeds infected with *R. solani* and treated with salicylic acid; FAD = seeds infected with *R. solani* with salicylic acid applied as a foliar spray and SDD = seeds infected with *R. solani* with salicylic acid applied as a soil drench. Bars followed by the same letters are not significantly different from each other at $p = 0.05$

Compared to the inoculated, untreated control (CTD), black scurf disease severity was reduced by 88.8 and 89.6% in stems of plants receiving SDH and FAH treatments, respectively. The highest disease severity of *R. solani* in stems (1.25%) and tubers (0.67%) was observed in the CTD treatment (Fig. 3). CTD (1.25%) differed significantly from STH (0.14%), SDH (0.14%) and FAH (0.13%) with regard to disease severity in the stems. All treatments tested did not differ significantly among themselves with respect to disease severity in tubers (Fig. 3).

All treatments that were inoculated with *R. solani* and treated with SA yielded significantly higher tuber weights than the remaining treatments (Fig. 4). The highest tuber weight (yield) was recorded in FAD (0.45 kg plant⁻¹) followed by SDD (0.44 kg plant⁻¹), CTD (0.42 kg plant⁻¹) and STD (0.42 kg plant⁻¹) (Fig. 4).

DISCUSSION

Black scurf of potatoes caused by *Rhizoctonia solani* is an economically important disease resulting in poor quality tubers and reduced yields (Anderson, 1982; Little *et al.*, 1988; Powelson *et al.*, 1993; Tsrer *et al.*, 1996). Fungicides that are effective in managing soil-borne fungal pathogens including *R. solani* are scarce (Powelson *et al.*, 1993; Johnston, 1995; Wicks *et al.*, 1995). In addition, crop rotations followed to reduce incidence of black scurf have not been very successful (Celetti *et al.*, 1990; Errampalli *et al.*, 1999). There is a need to look for alternative methods to control black scurf. Use of SA and other derivatives have been shown to be involved in the suppression of several pathogens (Okey and Sreenivan, 1996; Romero *et al.*, 2001; Pérez *et al.*, 2003). In the present study, the effect of SA in reducing the severity of black scurf was studied under screenhouse conditions.

The treatments which received SA in the form of seed treatment, foliar spray or soil drench improved the germination of seed tubers and plant canopy compared to the control. In addition, all SA treatments increased plant canopy in a significant manner compared to the uninoculated treatments. The SA treatments reduced the severity of *R. solani* considerably in both stems and tubers, ultimately improving tuber yield. In another study, lesion number, lesion area and percentage of necrotic leaf area of two cacao clones infected with *Phytophthora palmivora* were reduced when SA was applied as seed treatment, spray and soil drench (Okey and Sreenivan, 1996). Foliar application of synthetic analogs of SA such as 2,6-dichloroisonicotinic acid (INA) also reduced fire blight symptoms by 45% when compared to uninoculated controls (Kessmann *et al.*, 1994). In chickpea, exogenously supplied SA stimulated systemic resistance against Fusarium wilt and reduced the disease severity by 23 and 43% in plants which were treated with 40 and 80 µg mL⁻¹ of SA through root application (Saikia *et al.*, 2003). Pre-harvest treatment of sweet cherry with 2 mM SA significantly reduced lesion diameters caused by *Monilinia fructicola* compared to control post-harvest treatments (Yao and Tian, 2005). Similarly, treating sweet cherry with SA at 0.5 mM for 10 min reduced the incidence of decay and lesion size caused by *Penicillium expansum* (Chan and Tian, 2006).

In plants, exposure to pathogens triggers the activation of resistance mechanisms which prevents infection, aids in recovery from disease and prevents further infection. This phenomenon by which a plant's own defense mechanisms are induced due to prior treatment with either biological or chemical agent is known as Systemic Acquired Resistance (SAR) (Ryals *et al.*, 1996; Sticher *et al.*, 1997). SAR-protected plants when infected with a pathogen exhibit morphological and biochemical changes such as faster lignification response which corresponds to an increase in peroxidase activity (Ajilan and Potter, 1992) increased glucose and fructose concentrations in systemic tissue (Chandra and Bhatt, 1998); accumulation of fungitoxic β-ionone derivatives (Wyatt and Kuc, 1992); induction of lipoxygenase (Staub *et al.*, 1992), antimicrobial fatty acid derivatives (Namai *et al.*, 1993), phenylalanine ammonialysase, phytoalexins (Elliston *et al.*, 1977) and hydroxyproline-rich glycoprotein (Raggi, 1998). When activated, SAR can prevent infection from a wide range of pathogens (Bowling *et al.*, 1994; Coquoz *et al.*, 1995; Lawton *et al.*, 1996). It is associated with the induction of a suite of pathogenesis-related (PR) genes including acidic and basic β-1-3-glucanases, chitinases and a wide array of other genes of unknown function (Linthorst, 1991; Ward *et al.*, 1991). The pathways in induced resistance mechanisms are regulated by important signal molecules such as SA, jasmonate and ethylene, all of which bring about substantial alterations in gene expression and are

involved in complex crosstalk (Maleck *et al.*, 2000; Glazebrook *et al.*, 2003; Spoel *et al.*, 2003). SA is a naturally occurring, endogenously produced compound which is required for the induction of SAR (Gaffney *et al.*, 1993; Rairdan and Delaney, 2002). Treatment of plants with SA or its synthetic analogs 2,6-dichloroisonicotinic acid (INA) or benzo (1,2,3)thiadiazole-7-carbothioic acid S-methyl ester (BTH) results in induction of PR gene expression and resistance.

The importance of SA in SAR was illustrated by removing SA through the ectopic expression of salicylate hydroxylase (NahG) which ultimately blocked the onset of SAR (Gaffney *et al.*, 1993). The product of salicylate hydroxylase gene catalyzes conversion of SA into catechol which is an inactive compound. In NahG plants, the production of SA and induction of SAR was not observed and the NahG plants are hyper-susceptible to a wide range of pathogens (Delaney *et al.*, 1994; Navarre and Mayo, 2004). Similarly, increasing SA concentrations through endogenous synthesis or exogenous application played a role in the induction of SAR (White, 1979; Métraux *et al.*, 1990; Rasmussen *et al.*, 1991; Malamy *et al.*, 1992; Enyedi *et al.*, 1992; Verberne *et al.*, 2000). SA-mediated signaling is well characterized in Arabidopsis, tobacco and cucumber compared to potato and rice which produce high levels of basal SA (Hammerschmidt and Kuc, 1982; Raskin *et al.*, 1990; Coquoz *et al.*, 1995; Dempsey *et al.*, 1999; Navarre and Mayo, 2004).

The precise mechanism by which induction of SAR leads to resistance is not completely understood while the several PR genes expressed during resistance development were antagonistic to pathogens (Alexander *et al.*, 1993; Cao *et al.*, 1994; Sticher *et al.*, 1997). So far, no single PR gene has been shown to be essential for SAR indicating the necessity of coordinated expression of these genes for production of resistance (Rairdan *et al.*, 2001). In plants, SA levels increase following pathogen infection in proximal and distal ends of the tissue (Malamy *et al.*, 1990; Rasmussen *et al.*, 1991) which mediates the development of SAR causing substantial changes in gene expression including in genes encoding pathogenesis-related proteins. Such scenario operates well where there is no endogenous production of SA but raises several questions on the operation of SA-mediated signaling and triggering of SAR in plants which have high constitutive SA levels. It is not clear whether such plants respond positively to the increase in inherent high levels of SA or the higher levels of SA become a hindrance and make the cells less competent in perceiving or transducing the SA signal as is the norm with other plant hormones (Weyers and Paterson, 2001). One school of thought believes that potato might have poor SA signal perception based on a study wherein SA could not induce resistance to *Phytophthora infestans* while arachidonic acid was able to induce the resistance (Yu *et al.*, 1997). There are others who believe that some mutants of Arabidopsis with high basal levels of SA constitutively express SAR (Bowling *et al.*, 1994) and there are suggestions that the presence of high SA levels makes potato more resistant to diseases (Vleeshouwers *et al.*, 2000). Although potato has high amounts of total SA, it is capable of tightly regulating the amount of free SA which will allow the tissue to remain responsive to free SA (Navarre and Mayo, 2004).

There are also reports which state that SA-mediated resistance are restricted to effects in the treated tissue and when applied exogenously it does not translocate efficiently throughout the plants (Enyedi and Raskin, 1993). In addition, SA deposited on leaf surface is rapidly broken down and brings about a short term response. The safety margin that separates the rates at which SA is effective and the rate at which it becomes strongly phytotoxic is very narrow (Romero *et al.*, 2001). These limitations have prevented the consideration of SA as a practical solution for disease control. SAR has not been adopted successfully for disease control in potato and other crops but it has the capabilities of becoming a viable strategy for disease control with the rapid increase in characterization of SA-mediated signaling in plants. Since the SA treatments improved emergence and canopy, reduced disease severity and improved tuber weight, their importance in plant disease cannot be completely ruled out. Further field trials and analysis of SA levels prior to planting and after harvest will give a better understanding about the role of SA in plant disease control.

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