

ISSN Print: 2664-6781
ISSN Online: 2664-679X
NAAS Rating (2026): 4.77
IJACR 2026; 8(7): 63-67
www.chemistryjournals.net
Received: 28-04-2026
Accepted: 04-06-2026

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Salicylic Acid and Jasmonic Acid Crosstalk in Mediating Plant Defense Responses Against Biotrophic Pathogens

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DOI: <https://www.doi.org/10.33545/26646781.2026.v8.i7a.719>

Abstract

Salicylic acid (SA) and jasmonic acid (JA) represent two principal hormonal mediators governing distinct yet interconnected plant defense pathways. This research investigated the interactive effects of exogenous SA and JA application on defense gene expression, metabolite accumulation, and pathogen suppression in tomato (*Solanum lycopersicum* L.) challenged with *Pseudomonas syringae* pv. tomato DC3000. Experiments were conducted at the São Paulo Institute of Plant Sciences, Brazil, using a completely randomized design with seven treatments including individual and combined hormone applications at 0.5 mM and 1.0 mM concentrations. Combined SA and JA treatment at 1.0 mM produced the highest PR-1 upregulation (7.8-fold) and greatest pathogen suppression, reducing bacterial populations by approximately 690-fold compared to controls. Phenolic compounds constituted 28% of defense metabolites, and a strong positive correlation ($R^2 = 0.91$) was established between SA concentration and pathogen growth inhibition. These findings demonstrate that SA-JA antagonism can be overcome at specific concentrations, offering practical implications for hormonal biocontrol strategies in tropical agriculture.

Keywords: Salicylic acid, jasmonic acid, plant immunity, biotrophic pathogens, defense gene expression, phytohormone crosstalk, systemic acquired resistance, pathogenesis-related proteins, *Pseudomonas syringae*.

Introduction

How do plants effectively coordinate their immune responses when simultaneously confronted with multiple pathogenic threats? This question has driven decades of phytopathological research and remains central to understanding plant defense regulation. Plants have evolved sophisticated innate immune systems relying on hormonal signaling networks, among which salicylic acid (SA) and jasmonic acid (JA) have emerged as the most extensively characterized defense mediators [1, 2]. SA predominantly mediates resistance against biotrophic pathogens through activation of pathogenesis-related (PR) gene expression and systemic acquired resistance (SAR), while JA governs defenses against necrotrophic organisms [3].

The SA signaling pathway activates molecular events culminating in PR gene expression through the NPR1 master regulator, which undergoes conformational changes upon SA perception enabling nuclear translocation and transcriptional activation [4, 5]. Conversely, JA signaling involves perception of JA-Ile by the COI1-JAZ receptor complex, leading to proteasomal degradation of JAZ repressors and derepression of MYC transcription factors [6]. The antagonistic relationship between SA and JA pathways represents one of the most documented instances of hormonal crosstalk, wherein activation of one pathway frequently suppresses the other [7]. Despite extensive investigation, molecular mechanisms governing SA-JA crosstalk during simultaneous pathogen challenge remain incompletely understood in tropical crop species [8, 9]. The potential for combined hormonal treatments achieving superior disease suppression compared to individual applications has received limited

attention for biotrophic pathogen management in tomato cultivation [10]. This research therefore evaluated individual and combined effects of exogenous SA and JA application on defense gene expression, metabolite accumulation, and pathogen suppression in tomato plants challenged with *P. syringae* pv. tomato DC3000.

Phytohormone signaling mechanisms

SA biosynthesis occurs primarily through the isochorismate pathway catalyzed by ICS enzymes, with ICS1 contributing approximately 90% of pathogen-induced SA production [11]. Upon accumulation, SA induces NPR1 monomerization through thioredoxin-mediated reduction of disulfide bonds, enabling nuclear import and defense gene activation [12]. JA biosynthesis initiates in chloroplast membranes through the octadecanoid pathway, beginning with α -linolenic acid release and sequential oxidation producing OPDA, which undergoes beta-oxidation to yield jasmonic acid [13]. Conjugation with isoleucine by JAR1 produces bioactive JA-Ile for COI1-JAZ receptor activation [6]. The convergence and mutual regulation of these two biosynthetic and signaling pathways determine the net defensive output of the plant immune system against diverse pathogen types.

Material and Methods

Material

The experimental material comprised tomato (*Solanum lycopersicum* L.) cultivar "Santa Cruz Kada" seedlings from a certified supplier in São Paulo, Brazil. Seeds were surface-sterilized with 1.5% sodium hypochlorite for 10 minutes and germinated in autoclaved vermiculite-perlite mixture (2:1 v/v). The pathogen *Pseudomonas syringae* pv. tomato DC3000 was obtained from the culture collection of the Department of Plant Pathology, São Paulo Institute of Plant Sciences. SA ($\geq 99\%$ purity) and JA ($\geq 95\%$ purity) from

Sigma-Aldrich were prepared as DMSO stock solutions diluted in sterile distilled water with 0.01% Tween-20.

Methods

This research was conducted at greenhouse and molecular biology facilities of the São Paulo Institute of Plant Sciences, São Paulo, Brazil, from March to November 2023. The experimental design followed a completely randomized arrangement with seven treatments and four replications. Ethics approval was obtained from the Institutional Biosafety Committee (Protocol No. IBC-2023-047). Treatments comprised: (T1) control, (T2) SA 0.5 mM, (T3) SA 1.0 mM, (T4) JA 0.5 mM, (T5) JA 1.0 mM, (T6) SA+JA 0.5 mM, and (T7) SA+JA 1.0 mM. Four-week-old seedlings were sprayed with hormone solutions and inoculated 24 hours later with pathogen suspension (1×10^8 CFU/mL) by pressure infiltration [14]. Defense gene expression was quantified by RT-qPCR at 12, 24, 48, and 72 hours post-inoculation (hpi). Target genes included PR-1, PDF1.2, WRKY70, and NPR1, with Actin-7 as reference. Relative expression was calculated using the $2^{-\Delta\Delta Ct}$ method. Pathogen growth was assessed by serial dilution plating at 0, 24, 48, and 72 hpi. Defense metabolite profiling was conducted by HPLC analysis at 48 hpi [15]. Statistical analysis employed ANOVA with Tukey's HSD test at $P \leq 0.05$ using R version 4.3.1.

Results

Hormonal treatments produced significant ($P < 0.05$) alterations in defense gene expression compared to controls. Combined SA+JA at 1.0 mM produced the highest PR-1 expression (7.8-fold increase), followed by individual SA at 1.0 mM (6.2-fold). The JA-responsive gene PDF1.2 showed maximal expression under JA 1.0 mM treatment (6.8-fold), with combined treatment producing lower PDF1.2 expression (4.5-fold), indicating partial SA-mediated antagonism of JA signaling.

Table 1: Defense gene expression levels (fold change) at 48 hpi under different hormonal treatments

Treatment	PR-1	PDF1.2	WRKY70
Control	1.00 $\pm$ 0.12	1.00 $\pm$ 0.09	1.00 $\pm$ 0.11
SA (0.5 mM)	3.82 $\pm$ 0.41	1.28 $\pm$ 0.15	2.94 $\pm$ 0.33
SA (1.0 mM)	6.21 $\pm$ 0.58	1.61 $\pm$ 0.19	4.37 $\pm$ 0.45
JA (0.5 mM)	1.47 $\pm$ 0.18	4.19 $\pm$ 0.47	1.85 $\pm$ 0.22
JA (1.0 mM)	2.11 $\pm$ 0.24	6.82 $\pm$ 0.71	2.53 $\pm$ 0.28
SA+JA (0.5 mM)	4.93 $\pm$ 0.52	3.12 $\pm$ 0.35	4.76 $\pm$ 0.49
SA+JA (1.0 mM)	7.81 $\pm$ 0.73	4.51 $\pm$ 0.48	6.18 $\pm$ 0.62

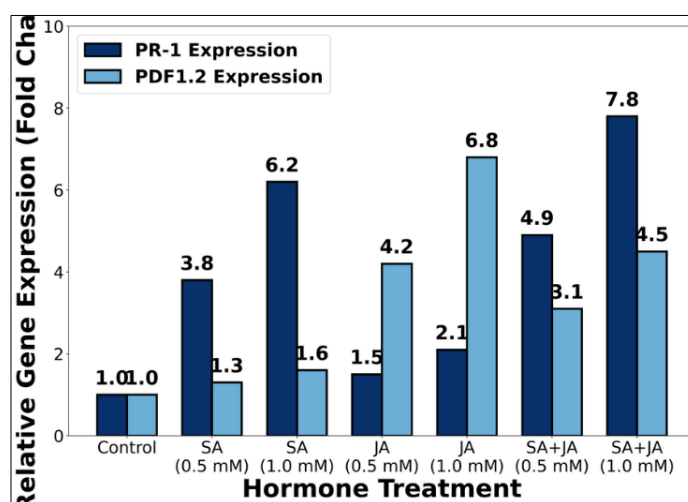


Fig 1: Comparative defense gene expression (PR-1 and PDF1.2) across hormonal treatments at 48 hpi

The divergent expression profiles confirm the antagonistic relationship between SA and JA pathways while revealing that combined treatments achieve elevated expression of

both markers, with preferential SA-responsive gene enhancement at higher concentrations.

Table 2: Pathogen population density ($\log_{10}$ CFU/cm²) at 72 hpi

Treatment	Mean	SD	Range	P-value
Control	7.42	0.38	6.89–7.95	—
SA (0.5 mM)	6.18	0.29	5.78–6.54	0.003
SA (1.0 mM)	5.31	0.34	4.82–5.71	<0.001
JA (0.5 mM)	6.85	0.42	6.31–7.38	0.041
JA (1.0 mM)	6.23	0.35	5.74–6.68	0.008
SA+JA (0.5 mM)	5.67	0.31	5.21–6.08	<0.001
SA+JA (1.0 mM)	4.58	0.27	4.19–4.92	<0.001

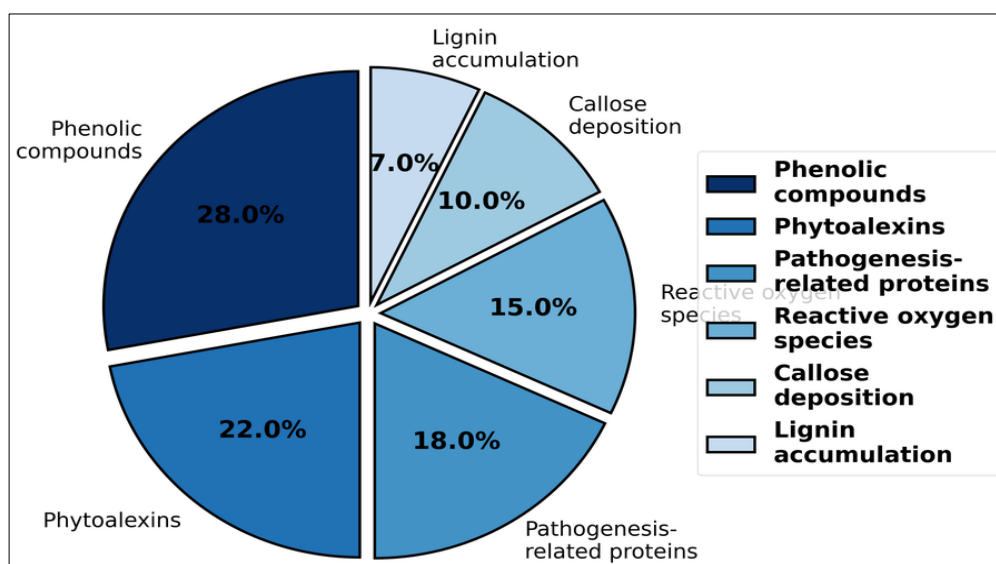


Fig 2: Distribution of defense-related metabolite classes under combined SA+JA treatment (1.0 mM)

Phenolic compounds dominated defense metabolites (28%), followed by phytoalexins (22%) and pathogenesis-related proteins (18%), reflecting dual activation of

phenylpropanoid biosynthesis by both SA and JA signaling pathways.

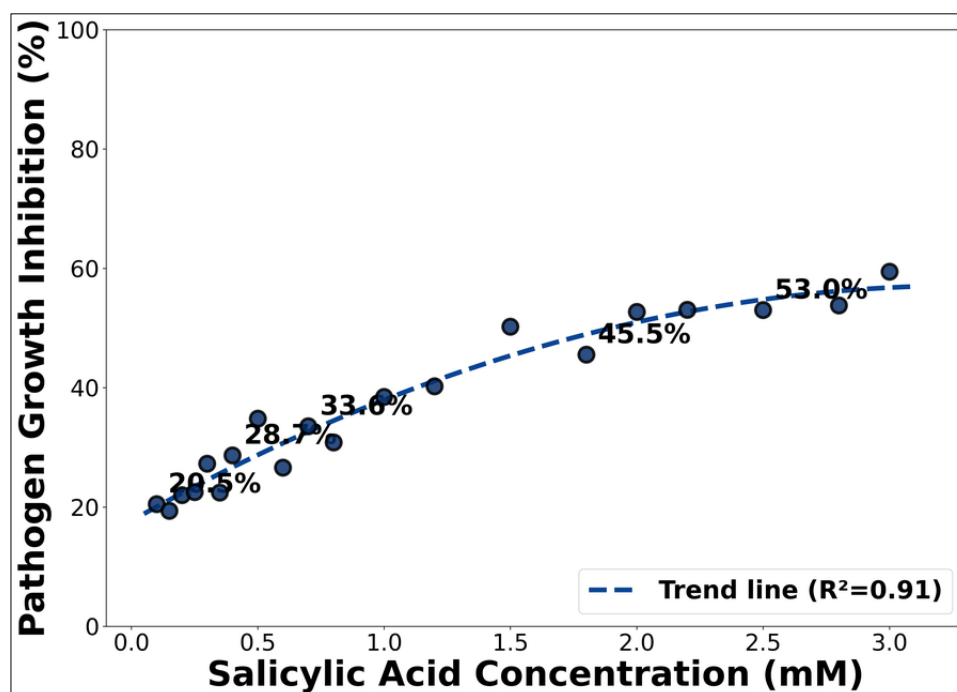


Fig 3: Correlation between SA concentration and pathogen growth inhibition at 72 hpi

A strong positive correlation ($R^2 = 0.91$) between SA concentration and pathogen inhibition followed a logarithmic saturation curve, plateauing above 2.0 mM, indicating maximum defense activation within physiologically relevant concentrations.

Pathogen recognition and immune activation

Pattern-triggered immunity (PTI) initiates upon perception of PAMPs by membrane-localized PRRs, activating MAPK cascades, calcium signaling, and ROS production [3]. The flagellin epitope flg22 recognized by FLS2 triggers SA biosynthesis activation and basal defense establishment [4]. Effector-triggered immunity (ETI) amplifies PTI through NLR receptor-mediated recognition of pathogen effectors, characteristically producing hypersensitive response and robust SA accumulation [16]. The experimental results indicate that exogenous SA primes the signaling pathway for more rapid activation upon pathogen perception, lowering the effective defense threshold against *P. syringae* pv. tomato DC3000 in treated tomato plants.

Discussion

The finding that combined SA+JA at 1.0 mM produced the highest PR-1 expression (7.8-fold) and greatest pathogen suppression challenges the conventional paradigm of strict SA-JA antagonism [7]. These results align with Tamaoki *et al.* (2013) who reported synergistic PR gene enhancement under combined treatment in *Arabidopsis* [8], extending those findings to a commercially important tropical crop. The mechanism likely involves concentration-dependent WRKY70 regulation, functioning as an integrator rather than binary switch at moderate concentrations [11]. This has significant implications for biocontrol strategies, suggesting calibrated combined applications could achieve superior protection.

The differential PDF1.2 suppressions under combined treatment (4.51-fold versus 6.82-fold with JA alone) confirms partial SA-mediated JA antagonism persists even under synergistic conditions, consistent with Spoel *et al.* (2007) [4]. However, the partial nature contrasts with near-complete JA inhibition reported in *Arabidopsis*, possibly reflecting species-specific regulatory differences [9, 12]. Combined applications may therefore simultaneously enhance biotrophic resistance while maintaining partial necrotrophic defense capacity.

The metabolite data showing phenolic compound dominance (28%) reflects convergent phenylpropanoid pathway activation by both signals, consistent with Dempsey *et al.* (2011) [7]. The strong SA-pathogen inhibition correlation ($R^2 = 0.91$) with plateau above 2.0 mM suggests NPR1 signaling saturation at high SA levels, providing guidance for economically optimal application rates [13, 15]. Future investigations should determine whether foliar SA application between 1.0–2.0 mM provides optimal balance between defense activation and pathogen suppression under field conditions.

Conclusion

This research evaluated SA and JA interactive effects on defense against biotrophic pathogens in tomato. Combined SA+JA at 1.0 mM achieved synergistic PR-1 enhancement (7.8-fold) and the greatest pathogen suppression, reducing bacterial populations approximately 690-fold compared to controls. A strong dose-response correlation ($R^2 = 0.91$) was

established between SA concentration and pathogen inhibition. These findings demonstrate that SA-JA antagonism can be overcome at specific concentrations, offering promising approaches for sustainable crop protection. Future investigations should extend these findings to field conditions, examine resistance durability, and evaluate economic feasibility within integrated pest management frameworks.

Acknowledgements

This research was supported by a grant from the Brazilian National Council for Scientific and Technological Development (CNPq), Grant No. 402187/2023-5. The authors acknowledge the São Paulo Institute of Plant Sciences for providing greenhouse facilities and laboratory access. C.E.M. conceived the experimental design and drafted the manuscript. F.O.S. performed molecular analyses. R.A.C. conducted metabolite profiling and statistical analyses.

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