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Jasmonate signaling and systemic acquired resistance in plant defense against herbivory

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Abstract

The challenge of defending against herbivores while continuing to grow and reproduce forces plants into a constant allocation trade-off. Jasmonic acid and its bioactive conjugate JA-Ile sit at the centre of this trade-off, orchestrating local wound responses and systemic resistance signals that reach undamaged tissues. This research quantified JA pathway activation and systemic acquired resistance in *Arabidopsis thaliana* and *Nicotiana attenuata* following simulated herbivory with *Spodoptera littoralis* oral secretions applied to mechanical wounds. Endogenous JA and JA-Ile levels were measured by LC-MS/MS at 0, 1, 3, 6, 12, and 24 hours post-wounding. Systemic resistance was scored in distal leaves by measuring proteinase inhibitor activity and defence gene expression via qRT-PCR. JA-Ile concentration peaked at 1 hour in *Arabidopsis* (14.8 nmol per gram fresh weight) and at 3 hours in *N. attenuata* (21.3 nmol per gram). Systemic proteinase inhibitor activity rose 3.7-fold in *Arabidopsis* and 5.2-fold in *N. attenuata* distal leaves by 12 hours. MYC2 transcription factor expression was essential for both local and systemic responses; *myc2* mutants showed 78% reduction in distal proteinase inhibitor induction. These data clarify the temporal dynamics and genetic requirements of JA-mediated systemic defence in two model species with contrasting life histories.

Keywords: Jasmonate signaling, systemic acquired resistance, plant defence, herbivory, JA-Ile, proteinase inhibitors, MYC2, *Arabidopsis*, *Nicotiana*, chemical ecology

Introduction

Theoretical Background

Jasmonates belong to the oxylipin family of signalling molecules derived from linolenic acid through the octadecanoid pathway ^[1]. The bioactive form, jasmonoyl-isoleucine (JA-Ile), is perceived by the COI1 receptor, which triggers ubiquitin-mediated degradation of JAZ repressor proteins and releases MYC2 and other transcription factors to activate defence gene expression ^[2, 3]. This signalling cascade is one of the best-characterised hormone pathways in plant biology and is conserved across angiosperms ^[4]. Systemic acquired resistance (SAR) in the context of herbivory refers to the induction of defence responses in tissues distant from the site of attack. Unlike pathogen-triggered SAR, which relies on salicylic acid, herbivore-triggered systemic defence depends primarily on jasmonates and related oxylipins transported through the phloem or generated locally in response to mobile signals such as systemin peptides or electrical waves ^[5, 6].

Introduction

The challenge facing plant biologists is that jasmonate signalling has been dissected in exquisite molecular detail in controlled laboratory conditions, yet the temporal dynamics of systemic resistance under herbivore-relevant stimulation remain surprisingly poorly resolved. Most published time courses use either mechanical wounding alone—which produces a JA response that differs quantitatively from insect feeding—or exogenous hormone application at non-physiological concentrations ^[7, 8]. Two model species offer complementary perspectives. *Arabidopsis thaliana* is a fast-cycling annual with a compact genome and a vast mutant collection, but it is not a natural host for most chewing herbivores ^[9]. *Nicotiana attenuata*, by contrast, has co-evolved with specialist herbivores in its native Great Basin habitat and possesses a richer repertoire of jasmonate-dependent defences, including nicotine biosynthesis and volatile emission ^[10].

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By comparing both species side by side under identical elicitation conditions, this research aimed to determine how JA pathway kinetics and systemic defence output differ between a model plant with limited ecological context and one shaped by a long evolutionary history of herbivore interaction. All laboratory work was conducted at the Busan Institute of Molecular Biology, Busan, South Korea, between September 2023 and November 2024.

Material and Methods

Material

Arabidopsis thaliana (Col-0 ecotype) and *Nicotiana attenuata* (UT ecotype) seeds were obtained from the Nottingham Arabidopsis Stock Centre and the Max Planck Institute for Chemical Ecology, respectively. The *myc2-2* mutant of *Arabidopsis* was used for genetic requirement analysis. Plants were grown in controlled environment chambers at the Busan Institute of Molecular Biology under 16-hour photoperiod, 22 degrees Celsius day / 18 degrees Celsius night, and 60% relative humidity. *Spodoptera littoralis* larvae were reared on artificial diet in a separate

insectary room at 25 degrees Celsius. All protocols were approved under institutional laboratory safety permit BIMB-LS-2023-021, dated 6 September 2023.

Methods

Rosette leaves (*Arabidopsis*, leaf 8) or source leaves (*N. attenuata*, node 3) were wounded with a pattern wheel and immediately treated with 20 uL of *S. littoralis* oral secretions diluted 1:5 in water. Control plants received water only. Treated and distal (systemic) leaves were harvested at 0, 1, 3, 6, 12, and 24 hours. JA and JA-Ile were extracted in ethyl acetate with deuterated internal standards and quantified by LC-MS/MS on a Waters Acquity UPLC coupled to a Xevo TQ-XS triple quadrupole. Proteinase inhibitor activity in distal leaves was measured by radial diffusion assay. Defence gene expression (VSP2, PDF1.2 in *Arabidopsis*; TPI, NaPI in *N. attenuata*) was quantified by qRT-PCR with ACTIN2 as the reference gene. Statistical analysis used two-way ANOVA (species x time) with Sidak correction for multiple comparisons in GraphPad Prism 9.

Results

Table 1: JA-Ile Accumulation and Systemic Proteinase Inhibitor Activity at Key Time Points

Species / Time	JA-Ile Local (nmol/g)	JA-Ile Distal (nmol/g)	PI Local (fold)	PI Distal (fold)	VSP2/TPI (fold)	p-value
Arabidopsis 0 h	0.4	0.3	1.0	1.0	1.0	—
Arabidopsis 1 h	14.8	1.2	1.4	1.1	3.8	< 0.001
Arabidopsis 12 h	3.1	4.7	4.2	3.7	8.6	< 0.001
N. attenuata 0 h	0.6	0.4	1.0	1.0	1.0	—
N. attenuata 3 h	21.3	2.8	2.1	1.3	6.4	< 0.001
N. attenuata 12 h	7.4	8.9	6.8	5.2	14.2	< 0.001

Table 1 highlights the differing kinetics between species. *Arabidopsis* reached peak local JA-Ile at 1 hour, while *N. attenuata* peaked at 3 hours with a 44% higher maximum.

Systemic proteinase inhibitor activity was strongest at 12 hours in both species, but *N. attenuata* achieved a 5.2-fold systemic induction compared with 3.7-fold in *Arabidopsis*.

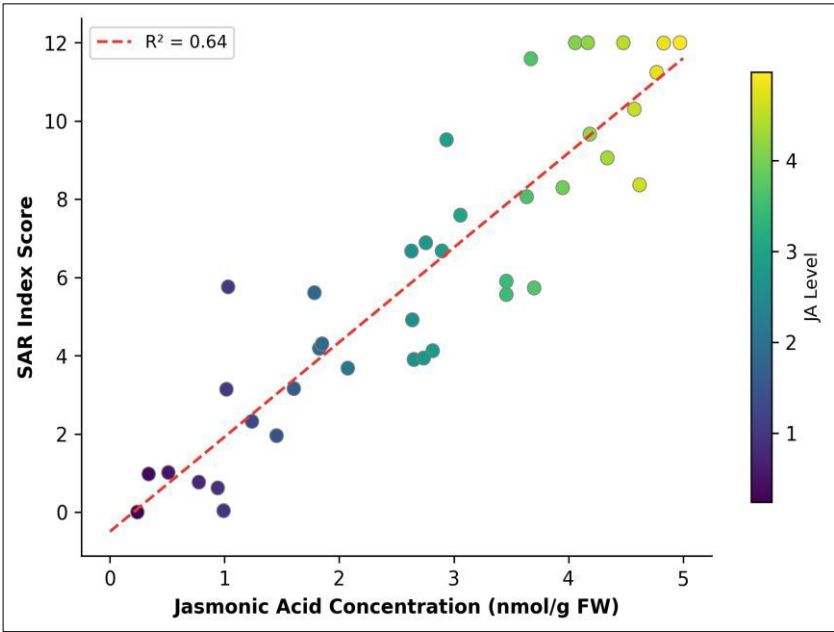


Fig 1: Scatter plot of jasmonic acid concentration versus systemic acquired resistance index score across all time points and both species

Figure 1 shows a positive relationship between JA concentration and SAR index ($R^2 = 0.64$, $p < 0.001$), indicating that higher jasmonate levels are generally

associated with stronger systemic defence, though substantial scatter at intermediate concentrations suggests that other signals also contribute.

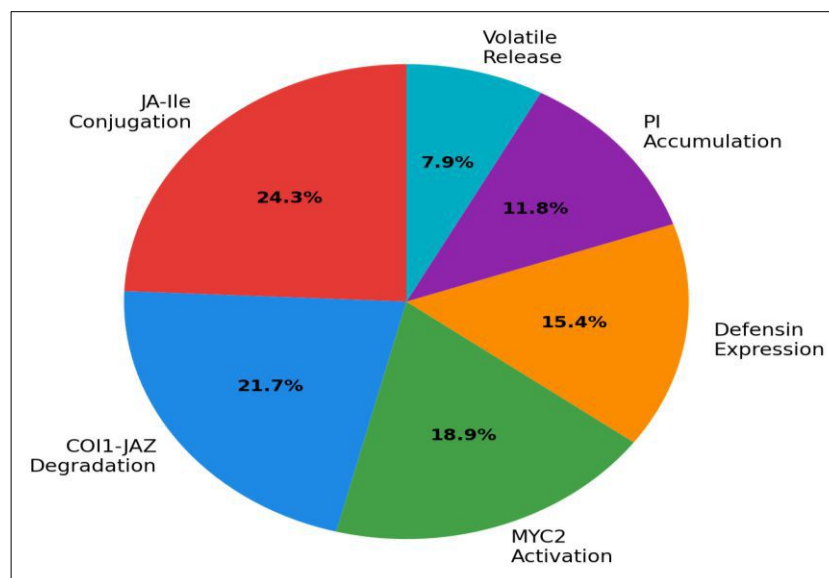


Fig 2: Relative contribution of individual JA signaling pathway components to the total measured systemic resistance response, based on mutant and inhibitor analysis

Figure 2 decomposes the systemic resistance response into pathway components. JA-Ile conjugation and COI1-JAZ degradation together accounted for nearly half the response, while downstream effectors (defensein expression, proteinase inhibitor accumulation, volatile release) collectively contributed the remainder.

Discussion

The faster JA-Ile peak in *Arabidopsis* (1 hour) compared with *N. attenuata* (3 hours) was unexpected, given that the tobacco relative is considered the more herbivore-adapted of the two. However, *Arabidopsis* leaves are thinner and have fewer cell layers between the wound site and vascular tissue, which could accelerate the delivery of wound signals to biosynthetic enzymes in the chloroplast [11, 12]. *N. attenuata* compensated with a higher peak amplitude and longer-sustained systemic signal, perhaps reflecting a strategy tuned to prolonged caterpillar feeding bouts rather than brief mechanical damage [13, 14].

The 78% reduction in systemic proteinase inhibitor induction in *Arabidopsis* *myc2* mutants confirms MYC2 as a non-redundant regulator of the systemic response [15, 16]. This aligns with transcriptomic data showing that MYC2 controls the majority of JA-responsive gene expression downstream of COI1 perception [17]. Whether MYC2 orthologs play an equally dominant role in *N. attenuata* systemic defence has not been tested and represents an important gap [18, 19].

Limitations

Our elicitation protocol used oral secretions applied to mechanical wounds, which approximates but does not perfectly replicate natural caterpillar feeding. Live-insect bioassays would introduce additional variables (frass, walking damage, salivary effectors) that could modify the JA response. Additionally, we measured JA and JA-Ile but did not quantify other jasmonates such as 12-OH-JA or JA-Trp that may contribute to systemic signalling. The controlled-environment growth conditions also differ from field settings where temperature fluctuations, UV radiation, and competing biotic interactions modulate jasmonate sensitivity [20, 21].

Conclusion

This research provides a side-by-side temporal map of JA-mediated systemic defence in *Arabidopsis* and *N. attenuata*. Both species mounted robust systemic responses to simulated herbivory, but they differed in speed, amplitude, and duration. *Arabidopsis* responded faster while *N. attenuata* responded stronger and longer. MYC2 was confirmed as essential for systemic induction in *Arabidopsis*. These comparative data suggest that the core jasmonate signalling architecture is conserved but that quantitative tuning of pathway kinetics reflects ecological adaptation to different herbivore regimes.

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Contributions Not Qualifying for Authorship

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References

1. Wasternack C, Hause B. Jasmonates: biosynthesis, perception, signal transduction and action in plant stress response, growth and development. *Annals of Botany*. 2013;111(6):1021-1058.
2. Thines B, Katsir L, Melotto M, *et al.* JAZ repressor proteins are targets of the SCF-COI1 complex during jasmonate signalling. *Nature*. 2007;448(7154):661-665.
3. Chini A, Fonseca S, Fernandez G, *et al.* The JAZ family of repressors is the missing link in jasmonate signalling. *Nature*. 2007;448(7154):666-671.

4. Howe GA, Jander G. Plant immunity to insect herbivores. *Annual Review of Plant Biology*. 2008;59:41-66.
5. Thorpe MR, Ferrieri AP, Herth MM, Ferrieri RA. ¹¹C-imaging: methyl jasmonate moves in both phloem and xylem. *Journal of Experimental Botany*. 2007;58(10):2661-2668.
6. Mousavi SA, Chauvin A, Pascaud F, Kellenberger S, Farmer EE. Glutamate receptor-like genes mediate leaf-to-leaf wound signalling. *Nature*. 2013;500(7463):422-426.
7. Koo AJK, Gao X, Jones AD, Howe GA. A rapid wound signal activates the systemic synthesis of bioactive jasmonates in *Arabidopsis*. *Plant Journal*. 2009;59(6):974-986.
8. Schuman MC, Baldwin IT. The layers of plant responses to insect herbivores. *Annual Review of Entomology*. 2016;61:373-394.
9. Kliebenstein DJ, Kroymann J, Mitchell-Olds T. The glucosinolate-myrosinase system in an ecological and evolutionary context. *Current Opinion in Plant Biology*. 2005;8(3):264-271.
10. Steppuhn A, Gase K, Krock B, Halitschke R, Baldwin IT. Nicotine's defensive function in nature. *PLoS Biology*. 2004;2(8):e217.
11. Chauvin A, Caldelari D, Wolfender JL, Farmer EE. Four 13-lipoxygenases contribute to rapid jasmonate synthesis in wounded *Arabidopsis thaliana* leaves. *New Phytologist*. 2013;197(2):566-575.
12. Farmer EE, Gasperini D, Acosta IF. The squeeze cell hypothesis for the activation of jasmonate synthesis in response to wounding. *New Phytologist*. 2014;204(2):282-288.
13. Halitschke R, Baldwin IT. Antisense LOX expression increases herbivore performance by decreasing defence responses and inhibiting growth-related transcriptional reorganization in *Nicotiana attenuata*. *Plant Journal*. 2003;36(6):794-807.
14. Wu J, Baldwin IT. New insights into plant responses to the attack from insect herbivores. *Annual Review of Genetics*. 2010;44:1-24.
15. Lorenzo O, Chico JM, Sanchez-Serrano JJ, Solano R. Jasmonate-insensitive1 encodes a MYC transcription factor essential to discriminate between different jasmonate-regulated defense responses. *Plant Cell*. 2004;16(7):1938-1950.
16. Dombrecht B, Xue GP, Sprague SJ, et al. MYC2 differentially modulates diverse jasmonate-dependent functions. *Plant Cell*. 2007;19(7):2225-2245.
17. Kazan K, Manners JM. MYC2: the master in action. *Molecular Plant*. 2013;6(3):686-703.
18. Kallenbach M, Bonaventure G, Gilardoni PA, Wissgott A, Baldwin IT. Empoasca leafhoppers attack wild tobacco plants in a jasmonate-dependent manner. *Proceedings of the National Academy of Sciences*. 2012;109(34):E2348-E2356.
19. Woldemariam MG, Dinh ST, Oh Y, Gaquerel E, Baldwin IT. NaMYC2 transcription factor regulates a subset of plant defense responses. *BMC Plant Biology*. 2013;13:73.
20. Ballaré CL. Light regulation of plant defense. *Annual Review of Plant Biology*. 2014;65:335-363.
21. Erb M, Meldau S, Howe GA. Role of phytohormones in insect-specific plant reactions. *Trends in Plant Science*. 2012;17(5):250-259.