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RESEARCH ARTICLE

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Interactions between plant defence signalling pathways: Evidence from bioassays with insect herbivores and plant pathogens

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Abstract

1. Sequential damage by attackers is hypothesized to result in reciprocal antagonism (crosstalk) between jasmonic acid (JA) and salicylic acid (SA) defence signalling pathways in plants. However, evidence for this crosstalk is not universal and several studies have found positive interactions (i.e. synergism) or no interaction whatsoever between JA and SA pathways.
2. Here we conducted a meta-analysis of studies on plant-mediated effects of initial attackers on performance of subsequent attackers to test the hypothesis of crosstalk between plant hormonal signalling pathways.
3. We found a significant negative mean effect size of JA-inducing initial attackers on both JA- and SA-inducing subsequent attackers, but a non-significant effect of SA-inducing initial attackers on both JA- and SA-inducing subsequent attackers. Effects on subsequent herbivores were contingent on the biology of the initial attacker, with negative effects of JA-inducing initial herbivores but no effect of SA-inducing initial herbivores, whereas pathogens on average did not influence subsequent herbivores. Furthermore, negative effects of JA-inducing initial herbivores on JA- and SA-inducing subsequent herbivores held when both attacks occurred on the same plant part, but when attacks were on different plant parts the effect of JA-inducing herbivores on SA-inducing herbivores was non-significant.
4. *Synthesis*. These results indicate that reciprocal antagonism between jasmonic acid and salicylic acid pathways is not universal, and suggest assymetries and specificity in the strength of plant-mediated interactions.

KEYWORDS
biotrophic pathogens, initial attacker, insect herbivores, meta-analysis, necrotrophic pathogens, plant-mediated interactions, sequential attack, subsequent attacker

1 | INTRODUCTION

Plants are attacked by multiple above- and below-ground enemies such as insect and vertebrate herbivores, as well as pathogens such as fungi, viruses and bacteria. Multiple attacks by different enemies on plants, either simultaneously or sequentially, frequently lead to plant-mediated indirect interactions among enemies where effects of early-arriving attackers determine the performance of subsequent attackers (Erb, Robert, Hibbard, & Turlings, 2011; Moreira et al., 2015; Poelman, Broaekgaarden, Van Loon, & Dicke, 2008) as well

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as attacker community assembly (Biere & Goverse, 2016; Poelman & Dicke, 2014). These plant-mediated effects are determined by plant induced responses to damage by insect herbivores or pathogens that involve changes in a wide array of physical (e.g. thorns, spines, trichomes) and chemical (secondary metabolites) defences, and the plant signalling pathways involved in upregulating these defences are thought to be highly conserved across many plant taxa (Mithöfer & Boland, 2012; Núñez-Farfán, Fornoni, & Valverde, 2007) and therefore presumably lead to predictable outcomes of plant-mediated interactions among attackers.

The two most important hormonal signalling pathways associated with induced plant defences are the jasmonic acid (JA) and salicylic acid (SA) pathways (Erb, Meldau, & Howe, 2012; Howe & Jander, 2008). The JA signalling pathway is thought to be primarily involved in defence against chewing and mining herbivores, necrotrophic pathogens, bacteria and nematodes, whereas the SA signalling pathway is mainly associated with defences against sucking herbivores, biotrophic pathogens and viruses (Pieterse, Schaller, Mauch-Mani, & Conrath, 2006; Stout, Thaler, & Thomma, 2006; Thaler, Humphrey, & Whiteman, 2012). The fact that plants are frequently attacked sequentially by enemies arriving at different time points and these enemies induce potentially different pathways sets the stage for potential interactions between plant defence signalling pathways. Indeed, a number of studies have empirically demonstrated that sequential damage by attackers inducing different plant hormonal pathways may result in antagonism (i.e. crosstalk) between signalling pathways (Erb et al., 2012; Pieterse et al., 2006), with this ultimately determining the magnitude and sign of effects of initial attackers on subsequent ones (Ali & Agrawal, 2014; Pieterse et al., 2006; Stout et al., 2006; Thaler et al., 2012). Accordingly, the crosstalk hypothesis predicts reciprocal antagonism or interference between plant signalling pathways when initial and subsequent attackers induce defences associated with different pathways (Erb et al., 2012; Pieterse et al., 2006), with this leading to predictable outcomes from plant-mediated effects between attackers. Specifically, initial attackers inducing defences through the JA pathway should have negative effects on subsequent attackers inducing the JA pathway but positive effects on subsequent attackers inducing the SA pathway, whereas initial attackers inducing defences through the SA pathway should have negative effects on subsequent attackers inducing the SA pathway but positive effects on subsequent attackers inducing the JA pathway (Figure 1; Pieterse et al., 2006; Thaler et al., 2012).

A number of studies have found patterns that fit the proposed antagonism between JA and SA pathways, either by directly measuring plant hormones or upregulation of JA or SA pathway-related defences or chemical precursors, or by means of herbivore or pathogen performance bioassays (see revision by Thaler et al., 2012). However, evidence for this crosstalk is not universal and several studies have found positive interactions (i.e. synergistic, e.g. Stout, Fidantsef, Duffey, & Bostock, 1999; Zehnder, Murphy, Sikora, & Kloepper,

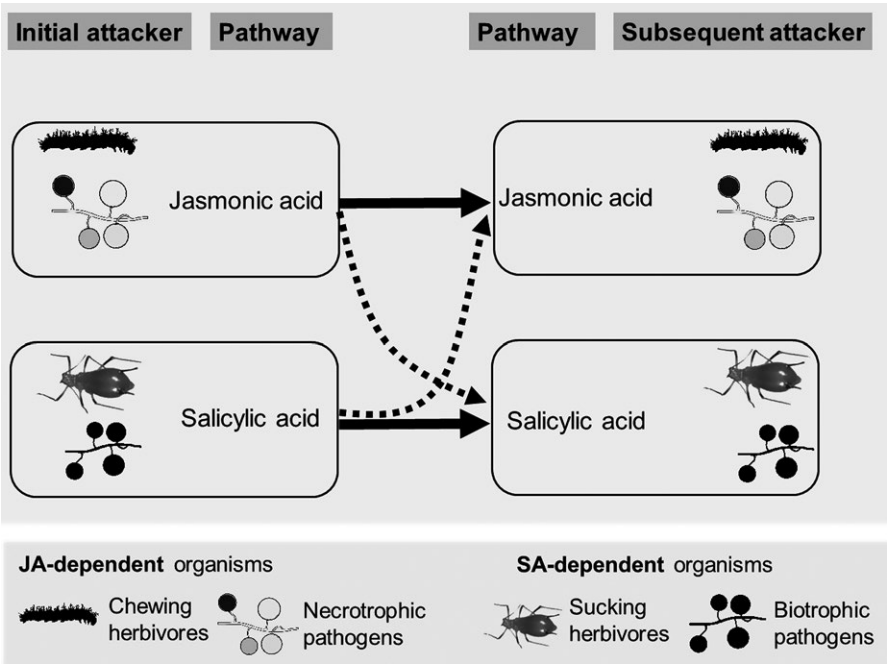

FIGURE 1 Predictions for the effects of the plant signalling pathway induced by an initial attacker (jasmonic acid [JA] or salicylic acid [SA] pathways) on the performance of subsequent attackers inducing JA or SA pathways. The crosstalk hypothesis predicts that initial attackers inducing defences through the JA pathway will have negative effects on performance of subsequent attackers inducing the JA pathway but positive effects on subsequent attackers inducing the SA pathway. Similarly, initial attackers inducing defences through the SA pathway will have negative effects on performance of subsequent attackers inducing the SA pathway but positive effects on subsequent attackers inducing the JA pathway. Predicted positive effects are shown by dashed lines, whereas predicted negative effects are shown by solid lines. The JA signalling pathway is primarily involved in the induction of defences against insect leaf chewers and necrotrophic pathogens, whereas the SA signalling pathway is primarily involved in the induction of defences against piercing sucking herbivores and biotrophic pathogens

2001) or no interaction between JA and SA signalling pathways (e.g., Ajlan & Potter, 1992; Inbar, Doostdar, Sonoda, Leibee, & Mayer, 1998; van Dam, Witjes, & Svatos, 2004). These inconsistencies can be attributed to several non-mutually exclusive factors. First, most studies reporting on interactions between JA- and SA-related defences have used model herbaceous plants such as *Arabidopsis* or crops such as tobacco or tomato (see Table 1 in Thaler et al., 2012), which may not be representative of responses across a more diverse pool of species including non-domesticated species, and other plant life-forms or taxonomic groups. Second, because plants respond with highly specific responses to different biotic stimuli, the nature and strength of their induced responses to attack may be strongly contingent on general aspects of the biology of the enemies under study, such as whether they are insect herbivores or pathogens, or consumer traits such as diet breadth (generalist vs. specialist) within a given functional group (Agrawal, 2000; Ali & Agrawal, 2012; Bingham & Agrawal, 2010; Moreira et al., 2015; Van Zandt & Agrawal, 2004). However, due to logistical limitations, most studies have not conducted replicated experiments to control for type of attacker or traits of specific attacker groups or guilds. Finally, there are a number of specificities related to interactions between plants and multiple attackers which may influence the outcome of these dynamics and these frequently vary among studies. One of these is whether initial and subsequent attackers feed on the same or different plant part or organ (e.g. roots, leaves, stems), as plant induced responses may be wired differently in each case and plant parts vary in their degree of inducibility (Agrawal, 2011; Karban, 2011) which could lead to variable outcomes.

Our goal was to test whether evidence from research conducted to date on plant-mediated interactions between initial and subsequent attackers supports the expectation of crosstalk between plant signalling pathways associated with defences against phytophagous insects and plant pathogens. We did this by performing a meta-analysis of work conducted from 1950 to 2017 on plant-mediated effects of initial attackers on performance of subsequent attackers as a proxy for assessing the presence of interactions between plant defensive pathways. We included 108 published studies comparing the performance of an insect herbivore or plant pathogen on plants that were previously damaged by an initial attacker vs. plants not previously damaged. First, we investigated whether effects of plant induction on performance of the subsequent attacker depended on the plant pathway induced by the initial attacker and the pathway induced by the subsequent attacker, that is, whether attackers induced the same or different pathway (pathway induced by initial attacker → pathway induced by subsequent attacker: JA→JA, JA→SA, SA→SA, and SA→JA). Second, we evaluated whether the biology of the initial attacker (i.e. herbivore or pathogen) influenced performance of subsequent attackers as well as the hypothesized interaction between pathways. Finally, we tested if effects on subsequent attackers and the hypothesized crosstalk between plant defensive pathways were contingent on whether initial and subsequent attackers fed on the same or a different plant part. In addressing the above, we deliver a novel assessment and reveal general patterns of interactions between plant defence-related hormonal pathways and its effects on attackers. Results will inform research on the evolution and constraints of plant induced resistance, as well as improve our understanding and ability to predict the outcome of plant-mediated interactions involving herbivores and pathogens.

2 | MATERIALS AND METHODS

2.1 | Data collection

From 18th April to 25th June 2017, we carried out an extensive literature search in the ISI Web of Knowledge database using a combination of the following keywords: "(Plant or tree) and (herbivore or herbivores or herbivorous or fungus or fungi or oomycota or virus or nematode or bacteria or bacterium) and (induction or sequential)." We retained only articles, book chapters, reviews, theses, dissertations and abstracts published in English. To further limit the search to relevant papers, we filtered outputs to retain only those matching with the following research areas: plant sciences, environmental sciences, ecology, pathology, agriculture, zoology, forestry, chemistry, physiology, behavioural sciences, microbiology, entomology, biochemistry, molecular biology, parasitology and mycology. The search was limited to the period 1950–2017. Our initial search yielded 7,638 papers (the number of papers retained at each stage is reported in the PRISMA flow chart, Figure S1 in the Appendix S1). To complete our dataset, we also surveyed the cited references in relevant review or meta-analysis articles about interactions between plant defence pathways (Biere & Goverse, 2016; Erb et al., 2012; Erb, Ton, Degenhardt, & Turlings, 2008; Fernández-Conradi, Jactel, Robin, Tack, & Castagneyrol, 2018; Johnson et al., 2012; Pieterse, Van der Does, Zamioudis, Leon-Reyes, & Van Wees, 2012; Tack & Dicke, 2013; Thaler et al., 2012; Wondafrash, van Dam, & Tytgat, 2013), as well as in those finally considered in this meta-analysis (next).

To be considered in the analysis, studies had to meet the following criteria: (1) report on the performance (damage, growth, size, developmental time or reproduction) of a subsequent attacker on control (uninduced) plants vs. plants induced by an initial attacker (when there was more than one initial attacker these had to be of the same type, i.e. herbivore or pathogen, and induce the same signalling pathway), (2) report on initial and subsequent attackers feeding on living plants, (3) report taxonomic information about plant and the initial and subsequent attackers, at least at the genus level and (4) provide a measure of the treatment level means and variability (i.e. variance, standard error or standard deviation) as well as the sample size in either the text, figures, tables or appendices. We excluded case studies where plants were exposed to human-driven abiotic treatments such as fertilization, drought, light exposure, CO_2 enrichment or flooding. In addition, we also excluded data on traits associated with preference of subsequent attackers (e.g. insect orientation in wind tunnel) because the subsequent attacker has to necessarily feed on tissues of a living plant in order for there to be crosstalk between initial and subsequent induction events. We excluded 97% of the papers from our initial search (7,601 papers, see Figure S1 in

the Appendix S1) because they did not include sequential attacks by real herbivores or pathogens based on criterion "1" above. We then excluded an additional 89 papers which did not meet one or more of the remaining criteria ("2–4" above).

It is important to note that most of the studies conducted thus far and included in our analyses measured attacker performance but not expression of plant hormone levels associated to each pathway. Despite this limitation, the association between specific groups of pathogens and phytophagous insects and each of these pathways is well-established in the literature for several groups of attackers (e.g. leaf chewing and sucking insects, necrotrophic and biotrophic pathogens; Stout et al., 2006; Thaler et al., 2012). Thus, while there is clearly variation in responses among plant species and higher order taxonomic groups, as well as in the induction effects by different herbivore species within specific guilds, in the absence of plant biochemistry data (e.g. hormonal levels) we used the best available evidence linking a given guild of consumers to either the JA or SA pathway (Thaler et al., 2012).

After applying the search keywords and excluding papers based on the above criteria, we obtained a dataset consisting of 774 case studies from 108 studies from the primary literature published between 1986 and 2017 in 34 scientific journals (see the list of references in the Appendix S2). These studies included 72 plant species (56 herbaceous and 16 woody), and reported initial damage by 63 insect herbivore and 30 pathogen species and subsequent feeding or infection responses by 112 insect herbivore and 20 plant pathogen species respectively. We did not find any study on mammalian herbivory which met our search criteria. Viruses, bacteria, fungi and nematodes were grouped as pathogens for statistical analyses.

For each study case, we extracted the following variables: plant identity and growth form (herbaceous or woody); attacker type (insect herbivore, biotrophic or necrotrophic fungi, virus, bacteria, nematode), feeding guild in the case of insect herbivores (chewer, miner or piercing sucking) and diet breadth (specialist or generalist, as indicated by authors of primary papers) of the initial and subsequent attackers; experimental conditions (field or controlled, i.e. greenhouse or laboratory); spatial scale of the interaction (initial and subsequent attackers targeted the same or different plant parts [e.g. leaves, stem, roots]); type of response of the subsequent attacker (abundance, growth, weight, size, developmental time, longevity, survival, consumption, damage, number of eggs, eggs mass, number of larvae, fecundity); and finally, the signal transduction pathway of the initial and subsequent attackers.

We assigned each study case a single identifier (Case ID) and assigned these study cases (one or more) to each corresponding paper (Study ID). A Study ID corresponded to a single published paper retained in our analysis. Within each Study ID, we considered as a Case ID any performance-related response variable measured for each pair of induced and control plants. In most studies, more than one response variable was measured for the same system. Although variables from the same study were not strictly independent (e.g. developmental time, mortality, growth of the same pool of individuals), we used all variables to include the greatest amount of information and to avoid possible biases due to a priori exclusion of some variables. The non-independence among case studies belonging to a single study was accounted for in the analyses (see Section next). In addition, some studies compared different plant treatments to the same control (e.g. the responses of a given herbivore to initial induction by different species were compared to the same uninduced control plant). In these cases, we retained all comparisons and controlled statistically for non-independence of multiple comparisons to the same control in the analyses. When needed, we extracted data from figures following digitalization using ImageJ 1.51j8 software.

2.2 | Statistical analyses

For each study case, we calculated an effect size using Hedges' *d* metric and its confidence interval (CI; Hedges, 1981) in the "metafor" package 1.9-8 version in R 3.2.3 (R Core Team, 2016; Viechtbauer, 2010). Hedges' *d* was the standardized mean difference between induced and control plants, such that negative values indicate that subsequent attackers performed worse on previously induced plants compared to control plants, whereas positive values indicate higher performance on previously induced than control plants (see Appendix S3 for details on effect size calculation). For mortality and developmental time, positive values indicated lower performance of subsequent attackers on previously induced plants than on control plants. In both of these cases, we multiplied the effect size by −1 so that the interpretation of these variables was consistent with all other performance traits in that lower values were indicative of reduced performance of subsequent consumers.

Initially, we calculated a grand mean effect size across all studies to assess whether there was an overall effect of plant induction by initial attackers on the performance of subsequent attackers. This grand mean effect size was considered significant if its confidence interval did not overlap with the zero (Koricheva, Gurevitch, & Mengersen, 2013). We also estimated the level of consistency among studies by calculating between-studies heterogeneity (τ^2 and associated *Q* statistics). Because τ^2 is dependent on sample size, we calculated I^2 value which is a standardized estimate of total heterogeneity ranging from 0 to 1 (Koricheva et al., 2013; Nakagawa, Noble, Senior, & Lagisz, 2017). Second, we investigated whether performance of subsequent attackers was affected by the signalling pathways induced by the initial and subsequent attacker. For this, we estimated mean effect sizes for each combination of signalling pathways induced by the initial and subsequent attackers using the full dataset (pathway of initial attacker → pathway of subsequent attacker: JA→JA, JA→SA, SA→JA, and SA→SA). We inspected each effect size and determined whether it was significant if its confidence interval did not overlap with zero. A negative mean effect size means that subsequent attackers associated to a given pathway performed worse on plants previously induced by an initial attacker associated with a given pathway than on controls, and a positive effect size means subsequent attackers performed better on previously induced plants. The expectation under the crosstalk hypothesis is that initial attackers inducing defences through the JA pathway should

have negative effects on subsequent attackers inducing the JA pathway (i.e. mean effect size significantly smaller than zero) but positive effects on subsequent attackers inducing the SA pathway (i.e. mean effect size significantly greater than zero). Similarly, initial attackers inducing defences through the SA pathway should have negative effects on subsequent attackers inducing the SA pathway but positive effects on subsequent attackers inducing the JA pathway.

After conducting analyses and calculating effect sizes using all studies, we used subsets of the database to test specific hypothesis by controlling for potential confounding factors. Specifically, we tested whether the effects of type of pathway induced by initial and subsequent attackers on the performance of the latter were contingent on (1) the biology of the initial attacker (i.e. whether the initial attacker was an insect herbivore or a pathogen, "attacker type" hereafter) and (2) on whether initial and subsequent attackers fed on the same or different plant part. For attacker type, we ran a mixed model with the type of initial attacker (insect herbivore vs. pathogen) as a moderator and then estimated the mean effect sizes for each combination of signalling pathways induced by the initial and subsequent attackers as described above (JA→JA, JA→SA, SA→JA, and SA→SA) for insect herbivore and pathogen initial attackers. Due to the lack of replication for studies measuring performance of subsequent pathogens for some of the pathway combinations, we restricted our analyses to insect herbivores as subsequent attackers. Here, we followed the same approach described previously to assess overall effects of pathway combination types, which consisted in inspecting each pathway combination effect size under each initial attacker type and determining whether these were significant if their confidence interval did not overlap with zero. Likewise, we followed the same approach for effects of plant part targeted by attackers using a subset of the full dataset restricted to studies where both attackers were insect herbivores (replication for studies with pathogens was too low or in some cases lacking). In this case, we ran a mixed model with the plant part targeted by initial and subsequent attackers (same vs. different) as a moderator, and then assessed separately for each plant part whether mean effect sizes of each combination of pathways were significant if their confidence intervals did not overlap with zero.

We also ran preliminary analyses to evaluate the effects of the diet breadth of the initial and subsequent attackers (specialists vs. generalists), experimental conditions (field or laboratory/greenhouse studies) and plant growth form (herbaceous or woody plants) on effect sizes of performance of subsequent attackers. Results indicated that performance of subsequent attackers did not differ (1) when initial attackers were specialists vs. generalists (Q_M = 5.70, p = .127) or when subsequent attackers were specialists vs. generalists (Q_M = 1.24, p = .266), (2) between controlled and field conditions (Q_M = 2.27, p = .132), or (3) between herbaceous and woody plants (Q_M = 0.85, p = .357). Based on these results, we did not include these predictors in our main statistical analyses.

In all cases, including tests of the effects of plant part and attacker type, we performed multi-level error meta-analyses (Nakagawa et al., 2017) with the *rma.mv* function of the R package metafor v. 2.0-0 (R Core Team, 2016; Viechtbauer, 2010) and included Study ID and Case ID nested within Study ID as random factors in order to account for correlations among effect sizes calculated from the same primary study. Multiple comparisons of induced plants with the same control plant were controlled for by computing the variance–covariance matrix among all effect sizes.

To ensure that our results were robust and unbiased by non-independence among effect sizes from the same primary studies, we conducted two sensitivity analyses. First, we randomly selected a single Case ID per study ID and system ID (i.e. the combination of plant and initial and subsequent attacker corresponding to each effect size) and quantified mean effect sizes for each combination of signalling pathways induced by the initial and subsequent attacker using mixed-effect meta-analysis (Fernández-Conradi et al., 2018). We repeated this procedure 100 times and checked whether mean effect sizes for each combination of pathways induced by initial and subsequent attackers obtained from this random subset of case studies were significantly different from zero, as well as compared whether the 95% distribution of model parameter estimates was consistent with those observed from the complete dataset. Second, we repeated the main analyses (i.e. multi-level error meta-analysis using the complete dataset) but sequentially removing one primary study at a time. This analysis aimed at testing whether the main result could have resulted from the inclusion of any particularly influential study, in particular studies providing a large number of case studies. For each of the 108 runs, corresponding to removing each of the 108 primary studies included in the main analysis, we checked that model parameter estimates for each pathway were comparable, regardless of whether each study was included or not in the analyses. These two analyses indicated that our findings were robust and unbiased by non-independence among effect sizes (see Appendix S4).

Finally, we used several approaches to verify that our results were not affected by publication bias (Koricheva & Gurevitch, 2014; Koricheva et al., 2013): (1) inspection of funnel plots, (2) cumulative meta-analysis, (3) calculation of fail-safe number and (4) exploration of the relationship between effect sizes and journal impact factor (Murtaugh, 2002). These analyses are fully described in the Appendix S4. They indicated that our findings were robust to selective reporting and dissemination bias.

3 | RESULTS

3.1 | Overall effect of initial attackers on subsequent attackers

The grand mean effect size (±95% CI) for performance of subsequent attackers was significantly negative (−1.34 ± [−1.93, −0.76], k = 774) indicating that, on average, subsequent attackers performed worse on previously induced plants than on control plants. There was a large amount of total heterogeneity (τ^2 = 16.18, Q_T = 25213.59, p < .001), most of which (98%) was attributable to among-study heterogeneity (i.e. I^2 = 0.98).

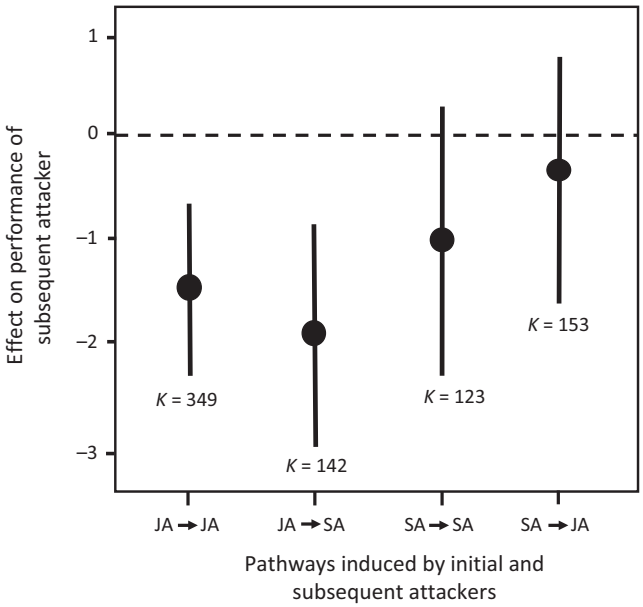

FIGURE 2 Mean effect size of the combination of signalling pathways induced by the initial and subsequent attackers (JA→JA, JA→SA, SA→SA, and SA→JA) on the performance of subsequent attackers. Dots and error bars represent model parameter estimates and corresponding 95% bias-corrected confidence intervals (CI). *k* = number of case studies. The vertical dashed line centred on zero represents the null hypothesis (i.e. no difference in performance of subsequent attacker between control and induced plants). The effect size of combined pathways induced by initial and subsequent attacker is significant if the 95% CI does not include zero

3.2 | Interaction between plant signalling pathways

A comparison of mean effect sizes and their 95% CI against zero for each combination of defensive pathways induced by initial and subsequent attackers did not support the crosstalk hypothesis, that is that initial attackers decrease the performance of subsequent attackers inducing the same pathway but increase the performance of subsequent attackers inducing a different pathway than them. Rather, initial attackers inducing the JA pathway significantly reduced the performance of both JA- and SA-inducing subsequent attackers (mean effect sizes of JA→JA and JA→SA pathways were negative and significantly different from zero; Figure 2), whereas initial attackers inducing the SA pathway did not significantly affect the performance of either JA- or SA-inducing subsequent attackers (mean effect sizes of SA→JA or SA→SA pathways were not significantly different from zero; Figure 2).

3.3 | Effects of initial attacker type on plant signalling pathway interactions

We found a significant effect of initial attacker type on the performance of subsequent insect herbivores (Q_M = 3.87, p = .049). A comparison of mean effect sizes and their 95% CI vs. zero for each pathway combination revealed distinct patterns depending on whether the initial attacker was an insect herbivore or a pathogen. When the initial attacker was an insect, we found that JA-inducing initial herbivores drove a reduction in performance of both JA- and SA-inducing subsequent herbivores (mean effect sizes of JA→JA and JA→SA pathways were negative and significantly different from zero; Figure 3a), whereas induction by SA-inducing initial insect herbivores did not significantly affect the performance of either JA- or SA-inducing subsequent herbivores (mean effect sizes of SA→JA or SA→SA pathways were not significantly different from zero, Figure 3a). These results closely resembled patterns for effect sizes of each pathway combination not accounting for attacker type (Figure 2), which is to be expected since most of the case studies used to compute such effect sizes involved insect herbivores as subsequent attackers. On the other hand, when the initial attacker was a pathogen we found that both JA- and SA-inducing initial pathogens did not significantly affect the performance of either JA- or SA-inducing subsequent insect herbivores (mean effect sizes of JA→JA, JA→SA, SA→JA and SA→SA were not significantly different from zero; Figure 3b).

3.4 | Effects of plant part attacked on plant pathway interactions

Based on a subset of the dataset limited to insect herbivores as initial and subsequent attackers, we found that plant part significantly affected the performance of subsequent insect herbivores (Q_M = 8.30, p = .040). A comparison of mean effect sizes and their 95% CI vs. zero for each pathway combination revealed distinct patterns depending on whether insect herbivores fed on the same or a different plant part. When initial and subsequent insects fed on the same part, we found the previously described pattern when not accounting for attacker type where JA-inducing initial herbivores reduced the performance of both JA- and SA-inducing subsequent herbivores (mean effect sizes of JA→JA and JA→SA pathways were negative and significantly different from zero; Figure 4a), but SA-inducing initial insect herbivores did not significantly affect the performance of either JA- or SA-inducing subsequent herbivores (mean effect size of SA→JA or SA→SA pathways were not significantly different from zero, Figure 4a). However, when insect herbivores fed on different plant parts JA-inducing initial herbivores influenced JA-inducing but not SA-inducing subsequent herbivores (mean effect size of JA→JA pathway were negative and significantly different from zero and mean effect size of JA→SA pathways were not significantly different from zero; Figure 4b). Again, SA-inducing initial insect herbivores did not significantly affect the performance of either JA- or SA-inducing subsequent insect herbivores feeding on different plant parts (mean effect sizes of SA→JA and SA→SA were not significantly different from zero; Figure 4b).

4 | DISCUSSION

The overall pattern obtained from studies involving bioassays with insect herbivores and pathogens on plants did not support the

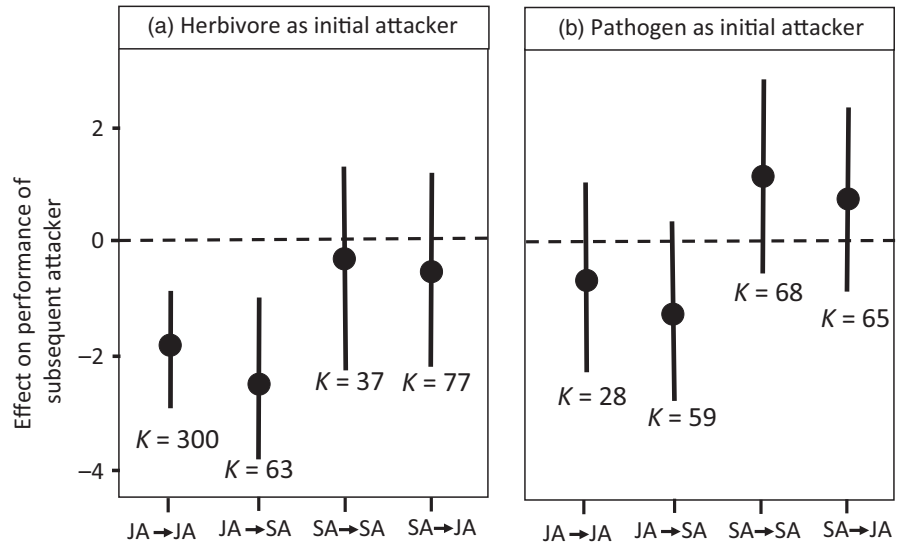

FIGURE 3 Mean effect size of the combination of signalling pathways induced by the initial and subsequent attackers (JA→JA, JA→SA, SA→SA, and SA→JA) on the performance of subsequent attackers. (a) Insect herbivores and (b) pathogens as initial attackers. Due to the lack of replication for studies measuring responses by subsequent pathogens, we restricted our analyses to insect herbivores as subsequent attackers. Dots and error bars represent model parameter estimates and corresponding 95% bias-corrected confidence intervals. *k* = number of case studies. The vertical dashed line centred on zero represents the null hypothesis (i.e. no difference in performance of subsequent attacker between control and induced plants). The effect size of combined pathways induced by initial and subsequent attacker is significant if the 95% CI does not include zero

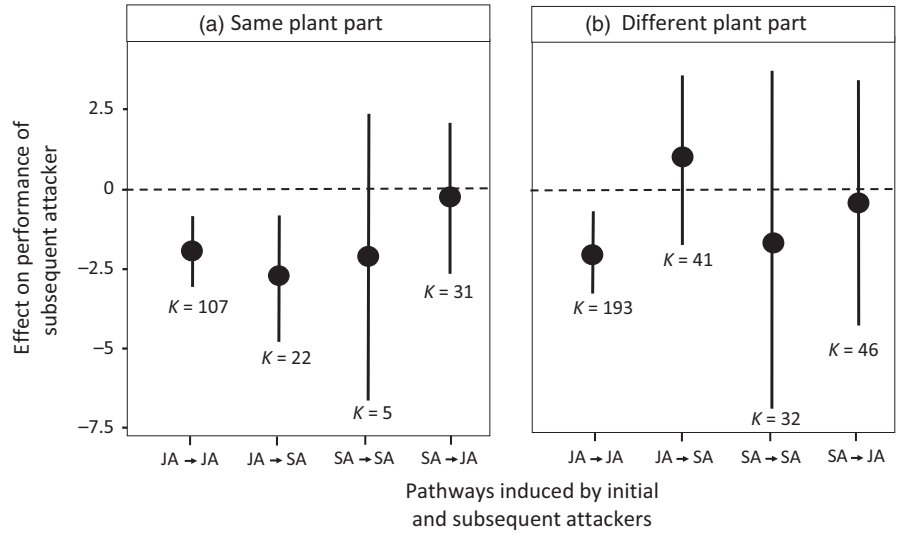

FIGURE 4 Mean effect size of the combination of signalling pathways induced by the initial and subsequent insect herbivores (JA→JA, JA→SA, SA→SA, and SA→JA) on the performance of subsequent insect herbivores. Initial and subsequent insect herbivores feeding on (a) the same plant part or (b) different plant parts. Dots and error bars represent model parameter estimates and corresponding 95% bias-corrected confidence intervals. *k* = number of case studies. The vertical dashed line centred on zero represents the null hypothesis (i.e. no difference in performance of subsequent attacker between control and induced plants). The effect size of combined pathways induced by initial and subsequent attacker is significant if the 95% CI does not include zero

predicted antagonism between plant defence signalling pathways. Instead, we found a significant negative effect of initial attackers inducing the JA pathway on both JA- and SA-inducing subsequent attackers, whereas initial attackers inducing the SA pathway had, on average, no significant effect on either JA- or SA-associated subsequent attackers. Interestingly, a closer inspection at this pattern comparing cases where the initial attacker was an insect herbivore or pathogen indicated that the biology of the initial attacker is important for predicting these plant-mediated effects. When the initial attacker was an insect herbivore, the above pattern held in that

initial JA-inducing insect herbivores had a significant negative effect on subsequent herbivores associated with both pathways, whereas SA-inducing herbivores, on average, did not influence any type of subsequent herbivore. However, when the initial attacker was a pathogen there was no significant effect of either JA- or SA-inducing pathogens on subsequent herbivores associated to either pathway. Further, when looking at interactions involving only herbivores we found that JA-inducing initial herbivores influenced both JA- and SA-inducing subsequent herbivores when interactions involved the same plant part (e.g. leaves, roots, stems), but when attackers fed on different plant parts the effect of JA-inducing herbivores on SA-inducing herbivores was no longer significant. Together, these findings suggest a degree of specificity in attacker plant-mediated interactions where effects on subsequent insect herbivores are present when the initial attacker is also an herbivore but not when the initial inducer is a pathogen, and interactions involving JA-associated initial herbivores are stronger or more consistent when both attackers feed on the same plant part.

A number of studies based on attacker bioassays have reported the expected pattern of crosstalk between plant signalling pathways, where initial attackers increase performance of subsequent attackers associated to a different defensive pathway than the former, presumably due to reciprocal antagonism between plant signalling pathways (reviewed by Pieterse et al., 2006; Stout et al., 2006; Thaler et al., 2012). Counter to this prediction, we found no evidence of increased performance of subsequent attackers associated to different plant signalling pathways than initial attackers, and this held true for both pathways. Rather, we found a strong pattern where JA-associated initial attackers had consistent negative effects on all attacker types (regardless of inducing pathway), whereas SA-associated initial attackers elicited weaker and more variable plant-mediated effects on subsequent attackers. The fact that JA-inducing initial attackers influenced both JA- and SA-inducing subsequent attackers suggests consistently stronger (negative) effects of JA-inducing attackers on all types of subsequent attackers on the one hand, but also a potential synergism between JA- and SA- related defence responses when initial attackers are JA-associated (De Vos et al., 2006; Hatcher, Paul, Ayres, & Whittaker, 1994). These patterns, however, held primarily for cases where both attackers were insect herbivores and not when initial attackers were pathogens, as we shall discuss ahead. In addition, it is important to note that there tended to be overall more variability in responses to SA-inducing attackers than JA-inducing attackers, particularly for effects of SA-inducing initial attackers on SA-inducing subsequent attackers where the mean effect size was reasonably strong but variability was high (Figure 2). Increased variability in the effects by SA inducers could reflect concomitant variability in the degree of plant induction by these attackers, perhaps because this group includes species that produce a greater diversity of plant induced responses, from manipulation of plant metabolism to supress plant induction to induction of both signalling pathways (see Section ahead). If SA-associated attackers as a whole do in fact produce more variable plant responses, then this would potentially weaken selection on the plant for consistent responses and thus limit signalling pathway interactions and increase variability in the direction and strength of plant-mediated effects on subsequent attackers. A good way forward to address this would be to perform phylogenetically controlled experiments with congeneric plant and herbivore species that compared the magnitude of plant induced responses (hormone concentrations and defence levels) in response to multiple species of JA and SA-associated attackers and measured effects on subsequent attackers.

One often cited cause behind patterns that do not fit the prediction of reciprocal antagonism between plant signalling pathways is that insect herbivores and pathogens have evolved strategies to manipulate plant signalling pathways and attenuate defence induction (Consales et al., 2012; Diezel, von Dahl, Gaquerel, & Baldwin, 2009; Pieterse & Dicke, 2007). Attenuation of induced responses by initial attackers may weaken plant-mediated effects on subsequent feeders as well as prevent crosstalk between plant signalling pathways. This phenomenon may be particularly important in the case of piercing–sucking SA-inducing insect herbivores. For example, SA-inducing sap-feeders such as aphids typically cause less damage than (JA-inducing) chewing herbivores and also inject specific compounds ("effectors") that manipulate or attenuate the plant's hormonal defensive response (Züst & Agrawal, 2016). Similarly, SA-inducing biotrophic pathogens have been shown to actively suppress plant defences (Abramovitch & Martin, 2004; Vargas et al., 2012). On the other hand, however, findings from other studies suggest that attacker manipulation of plant defences may in fact promote interactions between plant hormonal pathways. For example, studies with aphids suggest that these insects actively induce (by manipulation) the SA-pathway to suppress the JA-pathway, and this in turn benefits JA-inducing herbivores (Züst & Agrawal, 2016). In addition, attacker manipulation of plant hormonal signalling has been predicted to be more prone when interactions involve dietary specialist consumers. In this case, we found no difference in the observed patterns after controlling for attacker diet breadth, suggesting that plant manipulation did not play a preponderant role based on analysed studies. Other explanations for lack of interactions between plant pathways or variability in sign or magnitude of effects on attackers could be differences in the specific responses measured (e.g. herbivore feeding vs. performance; Stenberg & Muola, 2017) or in the environmental conditions (e.g. plants grown under limiting conditions might be more likely to exhibit pathway interactions or defence trade-offs; Sampedro, Moreira, & Zas, 2011). Unfortunately, we lacked replication to test for these potentially confounding factors.

Subsequent analyses by type of initial attacker showed the previously described pattern where JA-inducing initial herbivores had a significant negative effect on the performance of both JA- and SA-inducing subsequent herbivores but SA-inducing initial herbivores did not influence either group of subsequent herbivore. In contrast, pathogen initial attackers did not have a significant effect on any type of herbivore regardless of pathway type combination (SA-inducing pathogens tended to have a positive effect on SA-inducing subsequent herbivores, but this mean effect was not significant). A plausible explanation for the lack of plant-mediated effects involving

pathogens could be the predominance of manipulation of the host plant's induced defence responses reported for some groups such as necrotrophic pathogens (which have been shown to strongly induce JA). For example, previous work has shown that necrotrophic pathogens disrupt plant defence signalling (Prins et al., 2000; Sharon, Elad, Barakat, & Tudzynski, 2004) or detoxify host metabolites that interfere with virulence (Morrissey & Osbourn, 1999), and phenomena such as these may prevent effects of initial infection on subsequent insects feeding on plants. In addition, previous work has reported that leaf-chewing insects and necrotrophic pathogens are mainly associated with the production of JA, but this may not always be the case. For example, a recent study reported that damage by leaf-chewing insects usually only induces the JA-pathway, whereas necrotrophic pathogens inducing the JA pathway may also moderately induce the SA-pathway which could in turn inhibit the expression of genes associated with JA-related defences (Biere & Goverse, 2016). This concomitant induction of both pathways by necrotrophic pathogens may lead to interference or variable levels of induction of JA- or SA-related defences, which could explain the observed lack of a significant effect size of pathogens on subsequent insect herbivores. Last but not least, we must note that the lack of effect of pathogens on subsequent attackers could also be explained by the lower number of studies used, particularly in the case of JA-inducing initial pathogens. Only by increasing the number of studies with pathogens will it be possible to refute or corroborate these patterns. In particular, ethylene has been shown to play an important role in plant responses to pathogen attack (Broekaert, Delauré, De Bolle, & Cammue, 2006). For example, work has shown that ethylene modulates the sensitivity to JA-related responses and its downstream responses (von Dahl & Baldwin, 2007; see also Caarls et al., 2017). Further studies are much needed to better understand the interactions between ethylene, JA and SA.

When looking exclusively at plant-mediated interactions between insect herbivores, results showed that negative effects of JA-inducing initial herbivores on both JA- and SA-inducing subsequent herbivores occurred only when both attackers fed on the same plant part. Effects of JA-inducing initial herbivores on SA-inducing subsequent herbivores were no longer significant when interactions involved different plant parts. Similarly a recent meta-analysis found that initial pathogen infections drove stronger reductions on the performance of subsequent chewing insect herbivores when both attackers feed on the same plant part (vs. different parts; Fernández-Conradi et al., 2018). The proposed explanation for this pattern is that interactions between initial and subsequent attackers feeding on different plant parts are more likely to be exclusively plant-mediated since both groups of insects are spatially separated, whereas interactions involving the same plant part may lead to both direct (e.g. via competition or interference) and indirect (i.e. plant-mediated) interactions. In this sense, direct and indirect effects may have additive effects and this could explain the observed stronger (or more consistent) negative effects of JA-inducing herbivores when initial and subsequent attack were on the same plant part (Fernández-Conradi et al., 2018). A related aspect which was not addressed by our analyses, which may influence the strength of plant-mediated effects and interact with effects of plant part is distance between feeding sites of initial and subsequent attackers (Agrawal, 2011). However, the effect of distance may not always correlate with the occurrence of interactions within vs. between plant parts (i.e. distant attacks on the same plant part or attacks on different parts that are close to each other). Unfortunately, most studies do not report information on whether damage by initial vs. subsequent attackers was conducted on the same leaf or branch, and insect feeding is usually not constrained to a given leaf or part. As a result, it was not possible to control for distance to damage site. Further work is necessary to assess the independent and combined effects of plant part attacked and distance between feeding sites to better characterize the specificity of within-plant induction patterns in response to multiple attacks as well as its underlying mechanisms.

4.1 | Gaps in research and future work

There are several factors as well as biases and limitations in research that likely influence the occurrence or magnitude of interactions between plant JA and SA pathways and these might explain some of the patterns reported here. First, many of the attackers might have mechanisms to cope with (or, as mentioned earlier, even manipulate) plant hormone-regulated immune responses which globally might have averaged out (or attenuated) many of the stronger effects observed in some individual studies. Second, JA–SA antagonism has been reported for *c*. 20 plant species, most of which (>60%) are model herbaceous plants (e.g. *Arabidopsis*) and crops (e.g. tomato, tobacco) (Thaler et al., 2012), which may not be representative of induced responses across a more diverse pool of plant life-forms or taxonomic groups. Third, evidence for reciprocal antagonism reported in studies measuring gene expression, hormone levels or even defensive compounds does not always translate into changes in actual resistance (see Thaler et al., 2012 and references therein). This may occur, for example, because readouts of pathway end product are not effective against all species of herbivores or pathogens (Thaler, Karban, Ullman, Boege, & Bostock, 2002). Such uncoupling of defence or hormonal expression and attacker performance likely explain some of the discrepancy in studies reporting effects on plant defensive traits vs. studies reviewed here measuring attacker performance.

Our analyses of the literature also highlights a severe bias in research towards specific groups of attackers used as either initial or subsequent attackers. Although there is a vast amount of literature in plant responses to pathogen infection (reviewed by Glazebrook, 2005), clearly, much fewer studies addressing plant-mediated interactions have involved pathogens compared to insect herbivores (reviewed by Biere & Goverse, 2016), therefore limiting our understanding of plant-mediated pathogen–herbivore and pathogen–pathogen interactions. Another bias relates to plant growth forms as most of the analysed studies (78%) used herbaceous plants which, in comparison with woody plants, may largely differ in the nature and magnitude of induced defensive responses (e.g. qualitative changes for herbaceous vs. quantitative changes

for woody. Without more research exploring the outcomes of interactions involving different functional groups or species of plants and pathogens, our knowledge will remain incomplete and drawing robust generalizations about these phenomena will not be possible.

Finally, another key challenge will be to develop experiments with the appropriate replication for testing the effects of plant and attacker traits (e.g. contrasting plant growth forms and different attacker functional groups or guilds). Results from such studies will improve our understanding and ability to predict plant induced responses to attackers and plant-mediated indirect interactions. In addition, as plant induced responses by different groups of attackers are better described (e.g. phloem-feeding insects, necrotrophic pathogens) we will achieve a better characterization of induced responses by different groups of attackers. This will allow to establish more direct and reliable links between plant induced responses and bioassays measuring attacker performance.

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AUTHORS' CONTRIBUTIONS

X.M. formulated the idea of the manuscript; X.M., B.C., L.A.-R. designed the searching protocol; X.M. searched the literature and collected data; X.M. contributed materials/analysis tools; B.C. analysed the data; X.M. wrote the manuscript; L.A.-R. and B.C. contributed critically to the writing.

DATA ACCESSIBILITY

Data available from the Dryad Digital Repository: https://doi.org/10.5061/dryad.0nd4r91(Moreira,Abdala-Roberts,&Castagneyrol, 2018).

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