Notes

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Genetic variation in resistance to leaf fungus indirectly affects spider density

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Abstract. Many host-plants exhibit genetic variation in resistance to pathogens; however, little is known about the extent to which genetic variation in pathogen resistance influences other members of the host-plant community, especially arthropods at higher trophic levels. We addressed this knowledge gap by using a common garden experiment to examine whether genotypes of *Populus trichocarpa* varied in resistance to a leaf-blistering pathogen, *Taphrina* sp., and in the density of web-building spiders, the dominant group of predatory arthropods. In addition, we examined whether variation in spider density was explained by variation in the density and size of leaf blisters caused by *Taphrina*. We found that *P. trichocarpa* genotypes exhibited strong differences in their resistance to *Taphrina* and that *P. trichocarpa* genotypes that were more susceptible to *Taphrina* supported more web-building spiders, the dominant group of predatory arthropods. We suspect that this result is caused by blisters increasing the availability of suitable habitat for predators, and not due to variation in herbivores because including herbivore density as a covariate did not affect our models. Our study highlights a novel pathway by which genetic variation in pathogen resistance may affect higher trophic levels in arthropod communities.

Key words: *arthropods; plant pathogens;* Populus trichocarpa; Taphrina; *tripartite interactions.*

Introduction

Genetic variation within host-plant species is a key factor governing its associated arthropods (Fritz and Price 1988, Maddox and Root 1990, Johnson 2008). Studies have now shown that the effects of host-plant genetic variation can extend beyond herbivores (Fritz and Price 1988, Maddox and Root 1990, Barbour et al. 2015) to determine predator abundances (Bailey et al. 2006, Johnson 2008) and the structure of trophic interactions (Barbour et al. 2016). The few studies that have examined potential mechanisms underlying the communities of higher trophic levels on host-plants suggest that genetic variation in plant traits indirectly affects predators primarily through variation in herbivore abundances and traits (Bailey et al. 2006, Johnson 2008, Mooney and Agrawal 2008, Barbour et al. 2016). However, plants often host a diversity of organisms beyond arthropods, whose abundances and species composition are also determined by genetic differences in plant traits (Whitham et al. 2012). Microorganisms including fungi and bacteria that live inside plant tissue are affected by plant genetic variation but can also influence population dynamics of arthropods (Stout et al. 2006). These three-way interactions between plants, microorganisms and arthropods are known as tripartite interactions. While we know that host plants often mediate interactions between fungi and arthropods (Hatcher 1995, Stout et al. 2006), few studies have examined whether host-plant genetics mediate the strength of these tripartite interactions (but see, Saikkonen et al. 2001, Ahlholm et al. 2002, Busby et al. 2015).

Fungal pathogens are often dominant members of host plant communities and there is substantial evidence that pathogen-induced changes in plant traits can alter plant-arthropod interactions (Hatcher 1995, Stout et al. 2006, Tack and Dicke 2013, Busby et al. 2015). In particular, pathogen-induced changes in chemical-signaling

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pathways have been shown to alter the preference and performance of sap-sucking and leaf-chewing insects on host plants (Thaler et al. 2012). Similarly, plant pathogens can induce changes in plant volatile chemistry that directly attract or deter insect parasitoids (Biere et al. 2002, Dicke et al. 2003, Tack et al. 2012*a*). Besides leaf chemistry, plant pathogens can induce changes in plant morphology (Lake and Wade 2009, Giron et al. 2013), although these morphological effects have historically been focused on changes induced by herbivores (Ohgushi 2005). Studies have shown that herbivore-induced changes in leaf and stem morphology attract arthropods and often have a large effect on predatory arthropods (Langellotto and Denno 2004, Crawford et al. 2007, Wetzel et al. 2016). However, little is known about the effects of pathogen-induced changes in plant morphology on higher trophic levels.

In this study, we investigated whether genetic variation in pathogen resistance alters the availability of arthropod habitat, thus directly influencing the abundance of arthropods at higher trophic levels. Preliminary observations of the dominant riparian tree species, black cottonwood (*Populus balsamifera* L. ssp. *trichocarpa* (Torr. and A. Gray ex Hook.); hereafter *Populus trichocarpa*), suggested that individual trees varied substantially in their susceptibility to *Taphrina* sp., an ascomycete (fungus) that induces cup-like leaves (leaf blisters) in the spring (Newcombe 2005). In addition, we noticed that the undersides of leaf blisters were often being used as shelters by small web-building spiders (Fig. 1). Based on these observations, we designed our study to answer three questions: (1) Do spiders prefer to colonize *Taphrina*-blistered vs. unblistered leaves of *P. trichocarpa*? (2) Does *P. trichocarpa* exhibit genetic variation in its resistance to *Taphrina* and in the density of spiders? (3) Is variation in spider density explained by variation in resistance to *Taphrina*? Taken together, our study tests a novel pathway whereby genetic variation in pathogen resistance directly influences arthropods at higher trophic levels.

FIG. 1. An example of a spider in a *Taphrina* blistered leaf of a *Populus* tree. [Color figure can be viewed at wileyonlinelibrary.com]

METHODS

Common garden

We established a common garden experiment consisting of replicate clones of five *P. trichocarpa* genotypes in March 2012. The five genotypes were randomly selected from a larger common garden experiment (McKown et al. 2014) with the prerequisites that they were equally related and represented trait variation in height, phenology (e.g., bud set, leaf flush), growth rate, leaf C:N, and leaf tannins within southern BC localities, which is where the experiment was established (Latitude range: 49–52° N) (Crutsinger et al. 2014). The average genetic distance of the clones, which was calculated using 1-IBS (identity by state), was 0.326 with a range between 0.317–0.334 (Crutsinger et al. 2014). Due to the wide sampling of genotypes relative to the spatial scale of the experimental setup, we acknowledge that the effects of genetic variation in our study may be greater than what would be observed in nature (Tack et al. 2012*b*). Further methodological details about where the genotypes came from are available in McKown et al. (2013, 2014). For each genotype, we propagated 38–42 clones (G1: 38, G2: 41, G3: 41, G4: 41, G5: 42) in large 95 L nursery containers. The containers were placed on top of weed barrier cloth and spaced 2 m apart in a 30 × 40 m area on the campus of the University of British Columbia. We watered trees as needed throughout the growing season. For detailed tree propagation methods, see Crutsinger et al. (2014).

Do spiders prefer to colonize Taphrina*-blistered vs. unblistered leaves of* Populus trichocarpa*?*

To determine whether spiders preferentially use blistered leaves, we haphazardly surveyed up to 15-blistered and unblistered leaves per tree for evidence of use by spiders (presence of spiders and/or spider-webs) and analyzed the data with a Wilcoxon signed-rank test.

Does Populus trichocarpa *exhibit genetic variation in its resistance to* Taphrina *and in the density of spiders?*

Genotypic variation in resistance to *Taphrina* is a prerequisite for genotype to mediate the effect of *Taphrina* on spiders. We measured resistance to *Taphrina* fungus infection in two ways. First, we counted the total number of leaves infected by *Taphrina* fungus per tree. To account for variation in tree height, which is correlated with whole-tree biomass in *P. trichocarpa* ($r = 0.7$, McKown et al. 2014), we converted all estimates of abundance into density by dividing the number of individuals in a trophic group by tree height (m). Second, we randomly chose five blistered leaves from each tree and measured the width, length and depth (all in mm) of each leaf blister. We converted width, length, and depth measurements to volume (mm^3) using the formula for half a sphere. We then averaged blister volume estimates from the five leaves to obtain a single estimate of blister size per replicate tree. We used generalized linear models (GLMs) to test if blister density and blister size varied among *P. trichocarpa* genotypes. We specified a negative binomial error distribution in our GLMs (link function = log) to account for the over-dispersion of our data. We then used a post-hoc Tukey honestly significant difference (HSD) test to evaluate pairwise comparisons between the genotypes.

To evaluate the effect of *P. trichocarpa* genotypes on herbivore and predator densities, we vacuum-sampled the entire crown of each *P. trichocarpa* tree using a modified leaf blower/vacuum (Craftsman 25 cc 2-cycle) with a fine insect net attached. We immediately brought samples to the lab where we counted each individual and identified them as herbivores or predators based on taxonomy and feeding morphology (Grissell and Schauff 1990, Borror and White 1998). We further identified predators as ''web-building spiders'' and ''other predators,'' since preliminary surveys suggested that web-building spiders were associated with leaf blisters. To quantify herbivory, we visually estimated damage for four leaves starting with the first fully expanded leaf on each shoot. We assigned each leaf to 1 of 11 damage categories based on percent leaf area removed (0%, 1–5%, 6–10%, 11–20%, 21–30%…91–100%). The same observer scored all damage to maintain consistency across samples. We averaged damage scores for each shoot and then for all six shoots to obtain a single estimate of percent leaf area removed per replicate tree. We then ran a GLM to determine the effect of genotype on the density of web-building spiders. Again, we specified a negative binomial error distribution to account for the over-dispersion of our data. Using our spider survey data, we also analyzed a GLM to examine whether the proportion of spiders exploiting leaf blisters varied among *P. trichocarpa* genotypes after accounting for the proportion of unblistered leaves with spiders. We then used a post-hoc Tukey HSD test to test how genotypes differed from one another. We specified a quasi-binomial error distribution to account for the over-dispersion of our data. We also weighted the analysis by the number of blistered leaves sampled to account for the fact that estimates of spider occupation should increase in accuracy with more sampling.

Is variation in spider density explained by variation in resistance to Taphrina*?*

We used multiple regression models to evaluate whether factors related to variation in *Taphrina* resistance influenced spiders. Specifically, we used separate GLMs to test whether blister density, blister size, and their interactive effect influenced the probability of finding a spider in a leaf blister (error distribution = quasibinomial, link function = logit) as well as spider densities (error distribution = negative binomial, link function = log). We scaled predictor variables (mean = 0 and SD = 1) to eliminate collinearity with the interaction term in the model (Schielzeth 2010). In other words, scaling predictor variables enabled us to reliably interpret the importance of both main and interaction effects in the same model (Schielzeth 2010). Since prey availability could also influence spider occupancy and densities, we included herbivore density and percent leaf herbivory as covariates in our null model to account for these potential confounding effects. We then used a likelihood-ratio test to determine whether variation in *Taphrina* resistance still had a significant effect after accounting for the variation explained by herbivores. Finally, to determine whether we captured the effects of *P. trichocarpa* genotype, we used a likelihood-ratio test to test whether plant genotype still had a significant effect after accounting for the variation explained by both herbivores and *Taphrina* resistance.

Results

Do spiders prefer to colonize Taphrina*-blistered vs. unblistered leaves of* Populus trichocarpa*?*

During our spider survey we observed that blistered leaves were 35 times more likely to have a spider and/or spider web than unblistered leaves ($W = 6895$, $P < 0.001$), suggesting that spiders were attracted to certain characteristics of blistered leaves.

Does Populus trichocarpa *exhibit genetic variation in its resistance to* Taphrina *and in the density of spiders?*

Genotypes of *P. trichocarpa* exhibited substantial variation in resistance to *Taphrina* leaf blisters. We found that the absolute density of blisters varied 2.4-fold ($F_{4,198} = 25.57$, $P < 0.001$) among *P. trichocarpa* genotypes

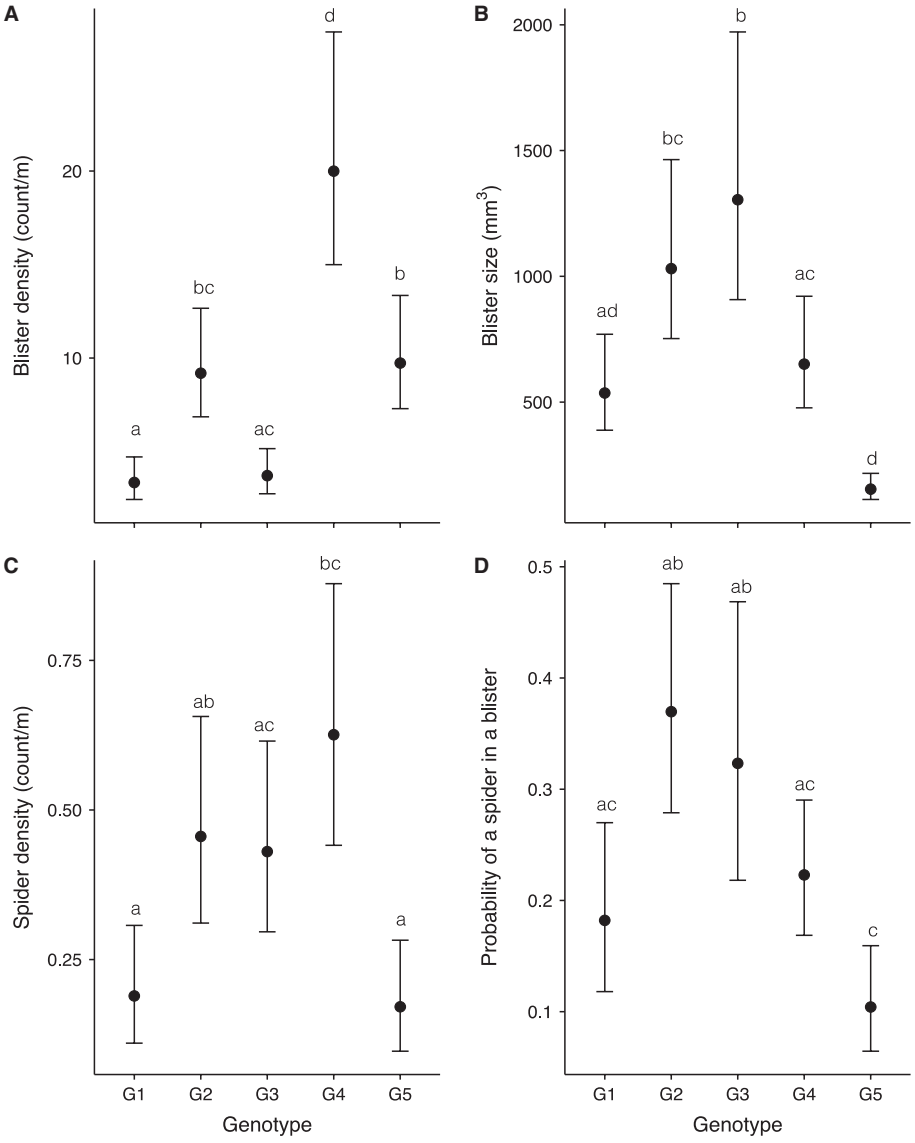

FIG. 2. Plots of the effects of *Populus trichocarpa* genotype on the leaf pathogen *Taphrina* and its associated arthropod community: (A) blister density (number of leaf blisters/tree height [m]), (B) blister size (mm^3), (C) spider density (number of individuals/tree height [m]), (D) probability of a spider in a leaf blister. Points and error bars correspond to means and 95% confidence intervals, respectively. Letters refer to Tukey honestly significant differences between plant genotypes.

(Fig. 2A). Moreover, we found 2.1-fold variation in the size of *Taphrina* leaf blisters ($F_{4,176} = 17.49$, $P < 0.001$) among *P. trichocarpa* genotypes (Fig. 2B). Importantly, genetic variation in resistance to *Taphrina* was not solely due to the effects of a single *P. trichocarpa* genotype, as indicated by post-hoc Tukey tests (letters denote significant differences, Fig. 2A, B).

Genotypes of *P. trichocarpa* also exhibited clear differences in the density of web-building spiders, the dominant group of predatory arthropods. The probability of observing a spider associated with a leaf blister ($F_{4,182} = 3.63$, $P = 0.007$) varied 3.7-fold among *P. trichocarpa* genotypes (Fig. 2D), suggesting that variation in leaf blister characteristics among genotypes may be influencing spider colonization of leaf blisters. In addition, we found a 2.5-fold difference in spider density ($F_{4,198} = 7.30$, $P < 0.001$) among *P. trichocarpa* genotypes (Fig. 2C). As with *Taphrina* resistance, the observed differences in spider density among *P. trichocarpa* genotypes were not solely due to the effects of a single genotype, as indicated by post-hoc Tukey tests (letters denote significant differences, Fig. 2C, D).

Is variation in spider density explained by variation in resistance to Taphrina*?*

We found that variation in *Taphrina* resistance (blister density and size) had a large effect on both the occupancy of spiders in leaf blisters ($F_{3,172} = 12.61$, $P < 0.001$) as well as spider densities ($\chi_{3,174} = 18.36$, $P < 0.001$). The probability of finding a spider in a leaf blister was higher on trees with large blisters (coef = 0.43, $F_{1,172} = 33.99$, $P < 0.001$) and high blister densities (coef = 0.16, $F_{1,172} = 4.44$, $P = 0.036$), but the effect of blister size was 2.7-fold greater than blister density. For spider density, we found a strong positive association with high blister densities (coef = 0.28, $\chi_{1,174} = 10.12$, $P = 0.001$). While we did not detect an association between spider density and blister size (coef = 0.14, $\chi_{1,174} = 0.80$, $P = 0.370$), we found that the positive association between spider density and blister density was even more pronounced on trees with large leaf blisters (coef = 0.38, $\chi_{1,174} = 9.23$, $P = 0.002$) (Fig. 3A). While our small number of *P. trichocarpa* genotypes prevents an analysis of genetic correlations, we qualitatively observed that genotypes with large leaf blisters at high densities supported higher spider densities (Fig. 3B).

Although spider occupancy in leaf blisters and spider densities exhibited strong associations with *Taphrina* resistance, we found that *P. trichocarpa* genotype still had a significant effect on both spider occupancy ($F_{4,168} = 4.01$, $P = 0.004$) and densities ($\chi_{4,170} = 21.26$, $P < 0.001$), suggesting that genetic variation in unmeasured characteristics of *P. trichocarpa* were also important.

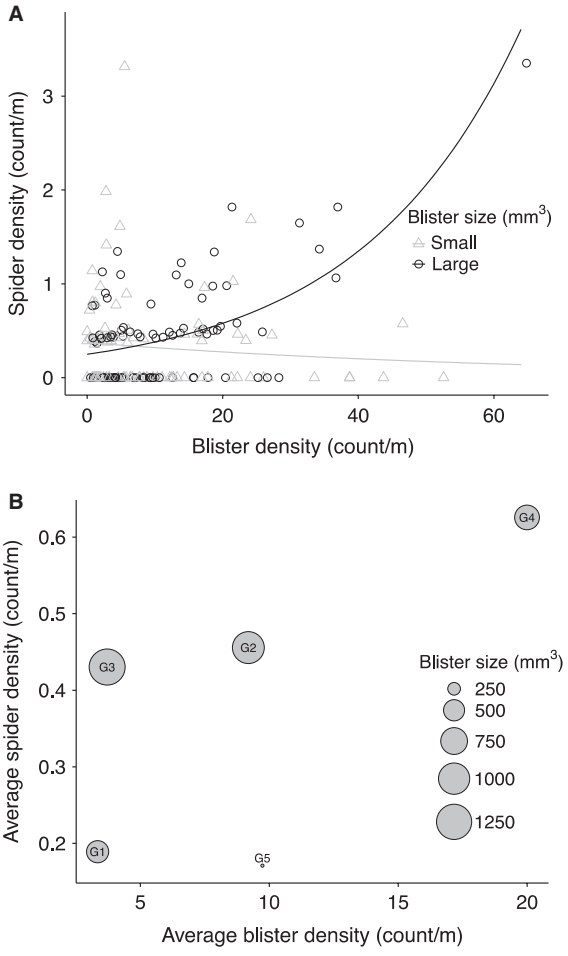

FIG. 3. Variation in resistance to *Taphrina* mediates the density of spiders on *Populus trichocarpa*. (A) Response of spider density (number of individuals/tree height [m]) to variation in leaf blister density (number of leaf blisters/tree height [m]) and size (mm^3). Black circles and grey triangles correspond to spider densities observed on large (>39.3 mm^3) vs. small (<39.5 mm^3) leaf blisters, respectively. We used 39.3 mm^3 as the cutoff for large and small leaf blisters because this value was the median size of leaf blisters in our dataset. Black and grey lines correspond to slopes from a generalized linear model. We divided blisters into two different categories in order to illustrate the interaction between blister density and spider density but we did not analyze the data this way. (B) Scatter plot of the average genotypic means of blister density (number of leaf blisters/tree height [m]) and spider density (number of individuals/tree height [m]).

DISCUSSION

Our study sheds light on decades of work showing that host-plant genetic variation affects arthropods (Maddox and Root 1987, 1990, Fritz and Price 1988, Whitham et al. 2012) by offering a novel mechanism—pathogen susceptibility increases predator density through the creation of favorable habitat. We found that plant genotype influenced multiple fungal and arthropod responses including: blister density, blister size, spider density and the probability of finding a spider in a blister (Fig. 2). Variation in *Taphrina* density and size influenced the probability of finding a spider in a blister. Therefore, trees with larger leaf blisters and higher blister densities had a greater probability of spiders colonizing blisters (Fig. 3).

While we know that genetic variation in host plants can indirectly affect predators, most of these effects are thought to be driven by bottom-up effects via herbivores (Bailey et al. 2006, Johnson 2008, Barbour et al. 2016). Instead, our results suggest that the increase in spiders on more *Taphrina*-susceptible trees is caused by increased habitat availability. Plant genetic variation not only influenced blister density and size but also the probability of finding a spider associated with a leaf blister (Fig. 2D). We suspect that spiders prefer living in blistered leaves because the depressed blisters offer more protection from abiotic factors and other predators (Langellotto and Denno 2004). Additionally, larger blisters are likely better habitat for laying eggs and building webs than smaller ones. Our finding is consistent with prior work documenting positive correlations between habitat availability and arthropod predators, an effect that appears to be particularly strong for spiders (Langellotto and Denno 2004, Wetzel et al. 2016). The fact that our spiders were

still influenced by *Taphrina*, independent of herbivore density—which was included as a covariate in the statistical models—suggests that habitat availability was more important than bottom-up effects caused by differences in herbivore density. However, because we did not experimentally manipulate blisters and monitor spider colonization, we cannot say definitively that blisters are the cause of increased spider density. Although, the effect size of our observational data on spider colonization of blisters is so large (35 times more likely to find a spider/web in a blister) that inferring an association between blisters and spiders seems to be a reasonable conclusion. Alternatively, bottom-up effects caused by variation in herbivore density may be important in this system, but we lack the temporal resolution to detect this effect. If host plants are consistently infected with *Taphrina* over multiple years (H. L. Slinn, *personal observation*), and the positive effect of *Taphrina* on spiders is really mediated through increase in herbivore density increasing predator density, we may not be able to detect this if predators have already decreased the population size of herbivores at the time of sampling. Temporal sampling or *Taphrina*-free trees would help us distinguish between these competing hypotheses.

Several studies have now demonstrated that pathogens can alter plant traits (e.g., plant chemistry and morphology) which influence plant—arthropod interactions, (Hatcher 1995, Stout et al. 2006, Tack and Dicke 2013, Busby et al. 2015). While prior work has focused on tripartite interactions between pathogens, plants, and insect herbivores, our work showed that pathogen-induced changes in plant traits could directly affect predatory arthropods. Taken together, this suggests that there may be a dynamic interplay between pathogens, plants, and multi-trophic interactions that has yet to be explored. Future studies should explore how pathogens alter plant traits, such as leaf chemistry and whether these chemical changes alter food quality for herbivores (Thaler et al. 2012), that indirectly affect with upper trophic levels. While our study and many others have looked at how plant pathogens shape other members of the community through tripartite interactions, there has been little work on how higher trophic levels influence plant pathogens after the pathogens have been established. It would be interesting to perform an additional study to look at the importance of herbivores on plant pathogen prevalence and how this in turn alters arthropod community composition since herbivores are a common mechanism for pathogen transmission in plants (dutch elm disease—Anagnostakis 1987, chestnut blight—Hunt and Meagher 1989, white pine blister rust—Hansen and Somme 1994). Our findings and future studies on this topic could have important agricultural implications for plant disease management, arthropod interactions in crops and biodiversity conservation. As climate change increases the frequency and intensity of disease outbreaks, it is important to understand how plant pathogens influence arthropods at all trophic levels for these reasons (Fisher et al. 2012).

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Author's contributions

HLS, MAB and GMC designed the experiment and HLS performed the fieldwork. HLS and MAB analyzed the data. HLS wrote the first draft, but all authors contributed to additional drafts of the manuscript.

Conflict of interest

The authors declare that there is no conflict of interest.

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