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Meta-analysis

Differences in pathogen resistance between diploid and polyploid plants: a systematic review and meta-analysis

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Polyploidy, the state of having more than two full sets of chromosomes, has been hypothesized to provide several evolutionary advantages to flowering plants including increased ability to resist pathogens and parasites. However, studies comparing pathogen resistance in conspecific and congeneric diploids and polyploids have produced mixed results. While the supposed relationship between polyploidy and pathogen resistance has been commented on in several narrative reviews, it has never been subjected to a systematic meta-analysis. We examined the effect of polyploidy on pathogen resistance by synthesizing 214 effect sizes from 128 studies. We find that, overall, there is no consistent effect of polyploidy on pathogen resistance. Subgroup analyses suggest that polyploids perform significantly better than diploids only in resisting hemibiotrophic pathogens, and autopolyploids tend show greater resistance than allopolyploids. This is surprising given the fact that polyploids possess extra allele copies of R-gene alleles that provide resistance to biotrophic pathogens, and this pattern may indicate that signaling cascades needed to elicit hypersensitive responses are disrupted by polyploidy. Disruption is supported by the observation that, across all pathogens, autopolyploids show significantly greater resistance compared to diploids, whereas allopolyploids do not. This is corroborated by the observation that synthetic autopolyploids perform significantly better than their allopolyploid and established counterparts. Regarding pathogen type, diploids show greater resistance than polyploids to pathogens that are fungi or nematodes. Analyses of publication bias indicate little to no bias, and analyses of heterogeneity indicate that phylogeny explains almost none of the observed heterogeneity. These results underscore the importance of not only systematic review but also the strong degree to which the effects of polyploidy depend on ecological context.

Keywords: coevolution, plant–fungal interactions, plant–pathogen interactions, polyploidy, whole genome duplication

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Introduction

Polyploidy, the state of having more than two full sets of chromosomes, has been studied in plants for over one hundred years by scientists in several biological fields (Ramsey and Ramsey 2014). This immediate doubling, tripling, or further multiplication of genetic variation on which evolution can act can lead to immediate speciation and considerable phenotypic changes, sometimes being called a 'macromutation' (Goldschmidt 1940, Doyle and Coate 2020). Numerous reviews discuss the supposed benefits and disadvantages that polyploidy provides plants, arguing, for example, that it could increase growth rates (Udall and Wendel 2006), can enhance tolerance of various environmental stresses (Stebbins 1950), is associated with phenotypic 'key innovations' that increase diversification rates (Soltis and Soltis 2016), and is associated with increased resistance to pathogens (Levin 1983).

In the case of pathogen resistance specifically, polyploidization can increase the production of existing secondary defense compounds (Levin 1976, Dhawan and Lavania 1996) or lead to the creation of novel metabolites (Schranz et al. 2012, Su et al. 2021). For these reasons, among others, polyploidy is commonly employed to improve cultivated plants (Touchell et al. 2020), including in some of the most common groups of crops in the world, such as wheat, bananas, brassicas, potatoes and coffee (Kyriakidou et al. 2018). Polyploid cultivars in major crops often show the greatest tolerance to biotic stresses like infections, leading some to argue that artificially inducing polyploidy could be effective in mitigating the increased susceptibility of crops to pathogens that is expected under future climate change (Ruiz et al. 2020).

However, despite the purported positive relationship between polyploidy and pathogen resistance, empirical support for this association is lacking. The physiological effects of polyploidization are, in general, poorly understood (Soltis et al. 2010), and few non-agricultural studies have examined the effect of polyploidy on tolerance of pathogens and parasites (Segraves and Anneberg 2016). While ecological modeling studies have shown support for a positive effect (Oswald and Nuismer 2007), narrative reviews of the literature suggest mixed results, indicating that the relationship is complex and dependent on ecological context (King et al. 2012, Segraves 2017). Additionally, in a recent systematic review and meta-analysis of the effect that plant polyploidy has on secondary metabolite composition, Gaynor et al. (2020) found no support for a consistent relationship. Despite the wide use of induced polyploidy as a method of crop improvement, there appears to be no existing meta-analysis that explicitly quantifies pathogen resistance in diploid and polyploid plants.

One difficulty with such an undertaking is that most appropriate studies that could be included in a meta-analysis examine human-bred plant cultivars in agricultural settings (Segraves and Anneberg 2016). If one finds a positive influence of polyploidy on pathogen resistance, it may be because the polyploids under study were not only induced but also subsequently selectively bred for favorable traits. Conversely, if one finds a negative relationship, this could be the result of comparing newly induced polyploids to diploid crops that have been selectively bred for specific resistances. Additionally, some cultivars used in such studies have been genetically modified for pathogen resistance and other traits, making it difficult to determine the specific contribution of ploidy to observed differences. To control for such factors, meta-analysis would need to not only include publications that studied both cultivated and wild species, but it would also need to filter out studies where genetic editing was used.

Here we report the results of the first such meta-analysis conducted to date. In our analysis, we included studies of cultivated species (those with a history of human cultivation and thus subject to either methodical or unconscious artificial selection; Darwin 1868) only if they met several criteria, including that the aim of the paper must not involve active breeding of more resistant plants. Overall, we were able to calculate 214 effect sizes from 128 different studies. We find that current evidence supports no consistent effect of polyploidy on pathogen resistance in flowering plants, and any observed improvement in resistance that coincides with polyploidy is likely contingent on random chance and biological context.

Material and methods

Literature search and selection

The following literature search is briefly summarized in a PRISMA flow diagram (Fig. 1, O'Dea et al. 2021). Searches were performed in March 2022 with Google Scholar, employing individual searches for studies comparing diploids and triploids, tetraploids, pentaploids, hexaploids, octaploids and decaploids (septaploids, dodecaploids and others were excluded due to their rarity in the literature). The query terms used for each search, the number of papers that each returned, and the number of papers that remained after screening are shown in the Supporting information. The queries were highly specific due to the necessities of removing studies that examined plants bred or genetically modified to be pathogen resistant, removing ploidy comparison studies not focused on pathogen resistance, and capturing the many different types of plant pathogens under study.

In total, our searches returned 1602 studies. During the abstract screening process, papers were removed from consideration if they involved breeding for superior traits, focused on genetic or biochemical underpinnings rather than pathogen resistance, had appeared in a previous search, included confounding variables, focused on non-pathogen organisms like aphids, were not written fully or partially in English, or generally studied irrelevant subject matter. Additionally, 53 papers were removed from screening because, while Google Scholar showed full abstracts for these studies, Internet searches and/or interlibrary loan requests turned up no results for existing full texts. In many cases, these may have been conference proceedings or other publications of sets of

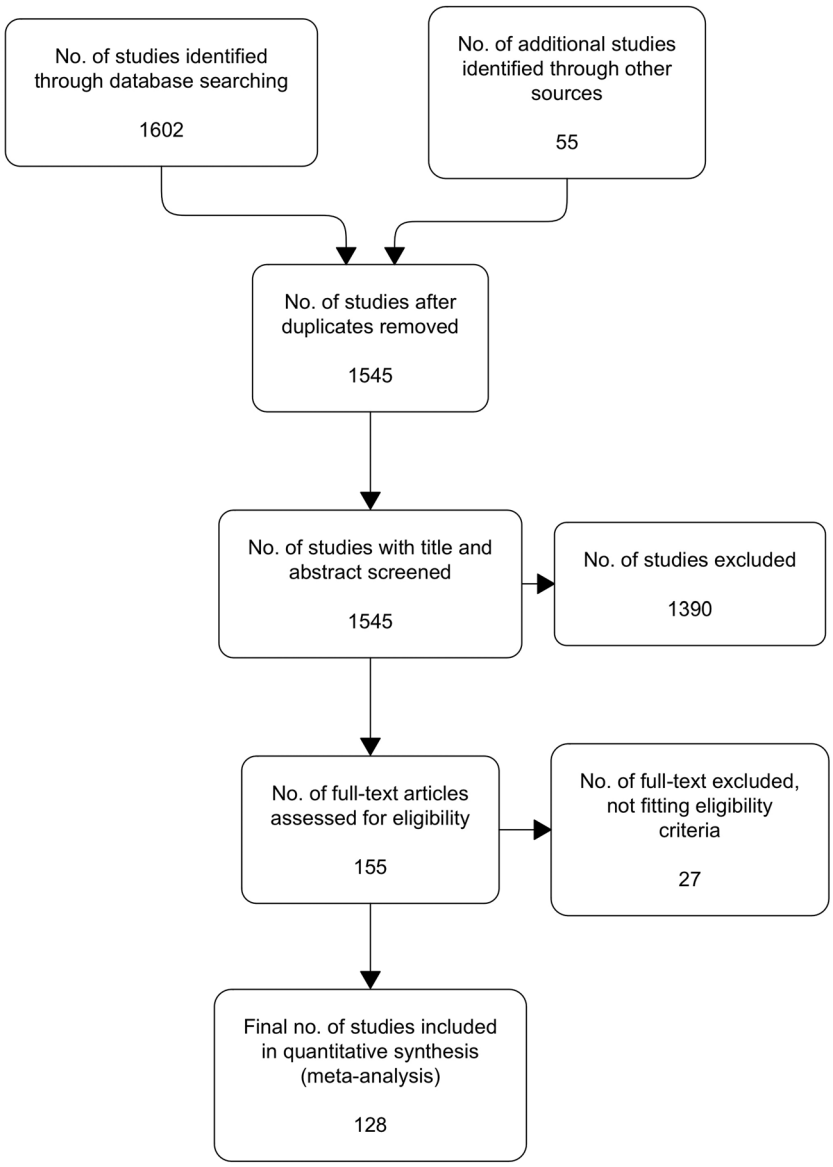

Figure 1. PRISMA flow diagram depicting our systematic literature search and application of inclusion-exclusion criteria.

abstracts without the publication of full texts. In the end, our search produced 100 articles that were determined to be eligible for meta-analysis, of which 73 were appropriate to be analyzed. In addition to articles identified through our systematic literature search, we also included 55 papers found through other means which met our inclusion/exclusion criteria, mainly previous ad hoc non-systematic searches on Google Scholar with variable Boolean language ('Group' column in the Supporting information). The data is available and is briefly summarized in the Supporting information.

Data extraction

For both sets of articles, we extracted data from each paper. In addition to the means, standard deviations, and sample sizes (number of genotypes/varieties in each category) for both the diploid and polyploid groups for each effect size entry, the following items were recorded: paper authors, plant family, polyploid plant family, ploidy level of the polyploid, whether or not the diploids under study were hybrids (or a mix of hybrids and non-hybrids), whether the polyploids under study were autopolyploids (non-hybrids) or allopolyploids (hybrids) or a mix of both, whether the species under study were wild or cultivated (or a mix of both), whether the polyploids under study were synthetic (i.e. anthropogenically induced) or established (i.e. have undergone significant genome reorganization since polyploidization), the type of pathogen with which plants were infected (e.g. fungus, virus, etc.), and the effect direction (i.e. whether a higher value indicates greater or lesser pathogen resistance).

Scoring the moderators was straightforward except for polyploid type (allopolyploid or autopolyploid), cultivation status

(cultivated or wild), and whether polyploids were synthetic versus established. Polyploid type is difficult to categorize in binary form due to the many different, and often controversial, definitions of polyploidy that exist (Parisod et al. 2010). Additionally, cultivation histories of plants can be complicated or unclear, such as in einkorn wheat (Zaharieva and Monneveux 2014), and because of this it can also be difficult to determine whether a polyploid cultivar is man-made or naturally established. Therefore, in cases where studies did not explicitly label plants with regard to these variables, we defined inclusion criteria for these three moderators.

For polyploid type, we followed the simple definitions of Ramsey and Ramsey (2014), who designate as autopolyploids any polyploids arising from parents that are members of the same single species and define as allopolyploids any polyploids that derive from interspecific hybridization. Effect sizes with the label 'both' come from papers where the polyploid group contained both allopolyploids and autopolyploids that were not separable. Studies for which it was still unclear what type of polyploid was under study were labeled as 'unknown.' For cultivation status, species were defined as 'wild' very narrowly, following the definition in Gaynor et al. (2020) as having no history of anthropogenic manipulation whatsoever (whether through cultivation, induced polyploidy, or manual hybridization). Otherwise, the species were labeled as 'cultivated,' or 'both' if both wild and anthropogenically manipulated species were not separable and contained in a single effect size. Studies for which we could not gather information about the presence or absence of anthropogenic manipulation were marked as 'unknown.' To remove the potential bias of studies where researchers bred plants for improved polyploid crops, publications were excluded from consideration if one group (diploids or polyploids) was anthropogenically improved while the other was not. Improvement does not encompass papers that merely crossed species rather than used species which had undergone artificial selection for trait improvement or were otherwise explicitly identified as 'improved.' To score polyploids as synthetic or established, we labeled as 'synthetic' any paper which explicitly stated that the polyploid was developed anthropogenically during the study or soon before. Wild polyploids were automatically labeled as 'established,' and all others were labeled as 'unsure'. Because rates of genome reorganization vary widely (Li et al. 2021), and because it is unclear for many older polyploid cultivars whether polyploidization occurred naturally or anthropogenically, we erred on the side of caution in labeling most effect sizes as 'unsure'.

We used WebPlotDigitizer ver. 4.5 (Rohatgi 2021) to extract values for some articles that only included bar graphs instead of tables. Papers for which both mean and standard deviation could not be calculated were removed. In total, 214 effect sizes from 128 articles were recorded and able to be meta-analyzed.

Meta-analyses

We used the R statistical software (www.r-project.org) ver. 4.0.3 and 4.2.1 to perform all the following analyses. Effect sizes were calculated using standardized mean difference (SMD, i.e. Hedges's g; Hedges 1981) with the *escalc* function from the R package 'metafor' (www.r-project.org, Viechtbauer 2010). We used this metric because all studies measured pathogen resistance, but different studies used a variety of metrics to compare diploids and polyploids, from the proportion of surviving plants after infection to the area under disease progress curve (AUDPC; Van der Plank 1963). Seven effect sizes were missing standard deviations for the diploid group, and five had no standard deviations for the polyploid group. So, following the method of Bracken (1992), these values were imputed prior to effect size calculation by multiplying the mean of the entry by the quotient of the sum of all standard deviations from entries with complete information in the dataset, divided by the sum of all means.

After effect size directions were standardized based on the 'Effect Direction' column in the dataset, we used multi-level meta-analytic models to systematically assess the data. This was done using the *rma* and *rma.mv* functions from 'metafor'. The initial *rma.mv* model included four random effects: average infection time in days before disease incidence was calculated, the between-study effect (variation among effect sizes from different studies), the within-study effect (variation among effect sizes from the same study), and a *phylo* variable calculated using a family-level phylogeny of angiosperms (Qian and Zhang 2014). We were unable to examine the effect of phylogeny in the pathogen column due to the diversity of included organisms as well as the lack of robust phylogenies for pathogens like fungi, bacteria and viruses (Gani et al. 2019). It also included six categorical moderator variables: different diploids (to account for effect sizes from the same publication comparing different sets of polyploids to the same set of diploids), ploidy level (triploid, tetraploid, etc.), polyploid type (autopolyploid, allopolyploid, or both included in the study), cultivation status (cultivated, wild, or both), whether polyploids were synthetic or established, and pathogen type (fungus, oomycete, bacterium, virus, or nematode). Since phylogeny explained almost none of the observed heterogeneity, we analyzed a second model in which between- and within-study effects were the only included random effects. We examined the amount of heterogeneity explained by each random effect using the I^2 statistic (Higgins and Thompson 2002) calculated using the *i2_ml* function from the package 'orchaRd' (Nakagawa et al. 2020), and we determined whether there were significant differences in mean effect size between subgroups (i.e. moderators) by looking at the p-value of the 'test of moderators' (Q_M statistic; Deeks et al. 2001) provided in the *rma.mv* output.

We examined the influence of publication bias on our results by creating a funnel plot (Egger et al. 1997) of all SMD values against their respective standard errors. Using that same plot, we tested for asymmetry using the trim-and-fill method (Duval and Tweedie 2000). Since funnel plot asymmetry can be caused by things other than publication bias (Nakagawa et al. 2022), we also used the following regression-based tests of publication bias: Egger's test of the relationship between residual effect size and study precision

(Egger et al. 1997) and a test for time lag bias (Jennions and Møller 2002). Finally, even though fail-safe numbers do not adequately control for heterogeneity and non-independence (Nakagawa et al. 2022), we calculated Rosenthal's (1979), Orwin's (1983), and Rosenberg's (2005) fail-safe n statistics.

Results

Multi-level modeling

Overall, we found no difference between diploids and polyploids in their abilities to resist pathogens ($Q_M = 25.645$, $p = 0.267$). Effect sizes in the full dataset ranged from a standardized mean difference of −6.73 to 4.75, with a mean effect size of −0.028. A caterpillars plot of these results can be seen in the Supporting information.

The first iteration of the mixed-effects model indicated that the random effect of phylogeny explained essentially none of the observed heterogeneity ($I^2 = 5.8 \times 10^{-8}$), so the mixed-effects model was run again with only between-study and within-study heterogeneity included as random effects. The results of this model suggest that none of the included moderators have significant influences on the degree to which polyploidy affects pathogen resistance, with no significant p-values inferred for any moderators. Additionally, the confidence intervals for all moderators overlap 0. Of the total observed heterogeneity ($I^2 = 93.4\%$), between-study heterogeneity was larger than within-study heterogeneity ($I^2 = 74.4$ and 19%, respectively). The insignificant value of the test of moderators (Q_M) also indicates little variation between subgroups.

Subgroup analysis

Despite the lack of significant moderators in our full multi-level model, single-moderator models shed light on interesting patterns of resistance in subgroups. Diploids seem to slightly outperform polyploids overall when all families are examined, but the performance is about equal when the two largest ones, Musaceae (n = 55) and Poaceae (n = 46), are removed ($Q_M = 17.749$, $p = 0.34$). While no family showed statistically significant resistance in either direction, Asparagaceae (n = 1) shows the strongest signal for diploid resistance over polyploid resistance (estimate = 2.163, $p = 0.233$), while Apocynaceae (n = 1) shows the strongest pattern in the opposite direction, though it is not significant (estimate = −1.941, $p = 0.27$; Fig. 2).

Diploids do not exhibit greater pathogen resistance when compared against allopolyploids than against autopolyploids (Supporting information). A simple *rma* model with 'both' and 'unknown' values removed shows that autopolyploids exhibit slightly greater resistance than diploids (estimate = −0.313, $p = 0.073$) while allopolyploid resistance is not significantly different from that of diploids (estimate = 0.126, $p = 0.467$). This pattern holds when pathogens are broken down by lifestyle, though diploids do show slightly greater resistance to biotrophic pathogens compared to autopolyploids (Fig. 3). When polyploids are divided into synthetic versus established, synthetic autopolyploids tend to outperform diploids (estimate = −0.637, $p = 0.068$; Fig. 4), and synthetic polyploids perform better relative to diploids than their counterparts in the 'established' and 'unknown' categories (estimate = −0.409, $p = 0.05$).

Diploids show significantly greater resistance to fungal (estimate = 0.911, $p = 0.047$) and nematode (estimate = 1.185, $p = 0.018$) pathogens (Fig. 5), while polyploids outperformed diploids in resisting hemibiotrophic pathogens (estimate = −0.895, $p = 0.043$). In individual subgroup analyses, no significant differences in resistance were observed on the basis of cultivation status.

Publication bias

Across all included effect sizes, there is little evidence that publication bias significantly affects our meta-analysis. Visual inspection of our funnel plot (Supporting information) shows a symmetrical distribution of SMD and standard error values. This was corroborated by trim-and-fill analysis, which produced no imputed studies and showed no evidence of significant bias ($p = 0.92$). However, Egger's regression test does suggest significant funnel plot asymmetry ($p = 0.02$), and individual trim-and-fill analyses of effect sizes found through systematic search and those found from other sources each showed evidence of significant bias ($p < 0.0001$ for both). While fail-safe n values varied widely (3408 for Rosenthal's, 0 for Orwin's, and 33 141 for Rosenberg's), they generally suggest little bias. We also found no influence of publication year on our results ($p = 0.38$).

The standardized mean differences of the in-search effect sizes are significantly different from those found outside the systematic search ($Q_M = 8.22$, $p = 0.042$, Supporting information), with studies found outside our search showing greater pathogen resistance in diploids relative to polyploids. Trim-and-fill analysis of each group showed bias in opposite directions, but when these are combined, the total data shows little bias.

Discussion

Based on our analyses there is no evidence that polyploidy is consistently associated with overall increased (or decreased) resistance to pathogens and parasites in flowering plants. While the association has been suggested in previous narrative reviews (Levin 1983, Van de Peer et al. 2017), many have been cautious about proposing a general effect (King et al. 2012, Segraves and Anneberg 2016). Given the lack of any significant moderators in our general multi-level model, as well as the lack of any effect of phylogeny, our results support this caution. The effect of polyploidy on pathogen resistance likely depends greatly on factors like ecological context, time since polyploid formation and the degree of subsequent genome rearrangement, and the luck of the genomic draw.

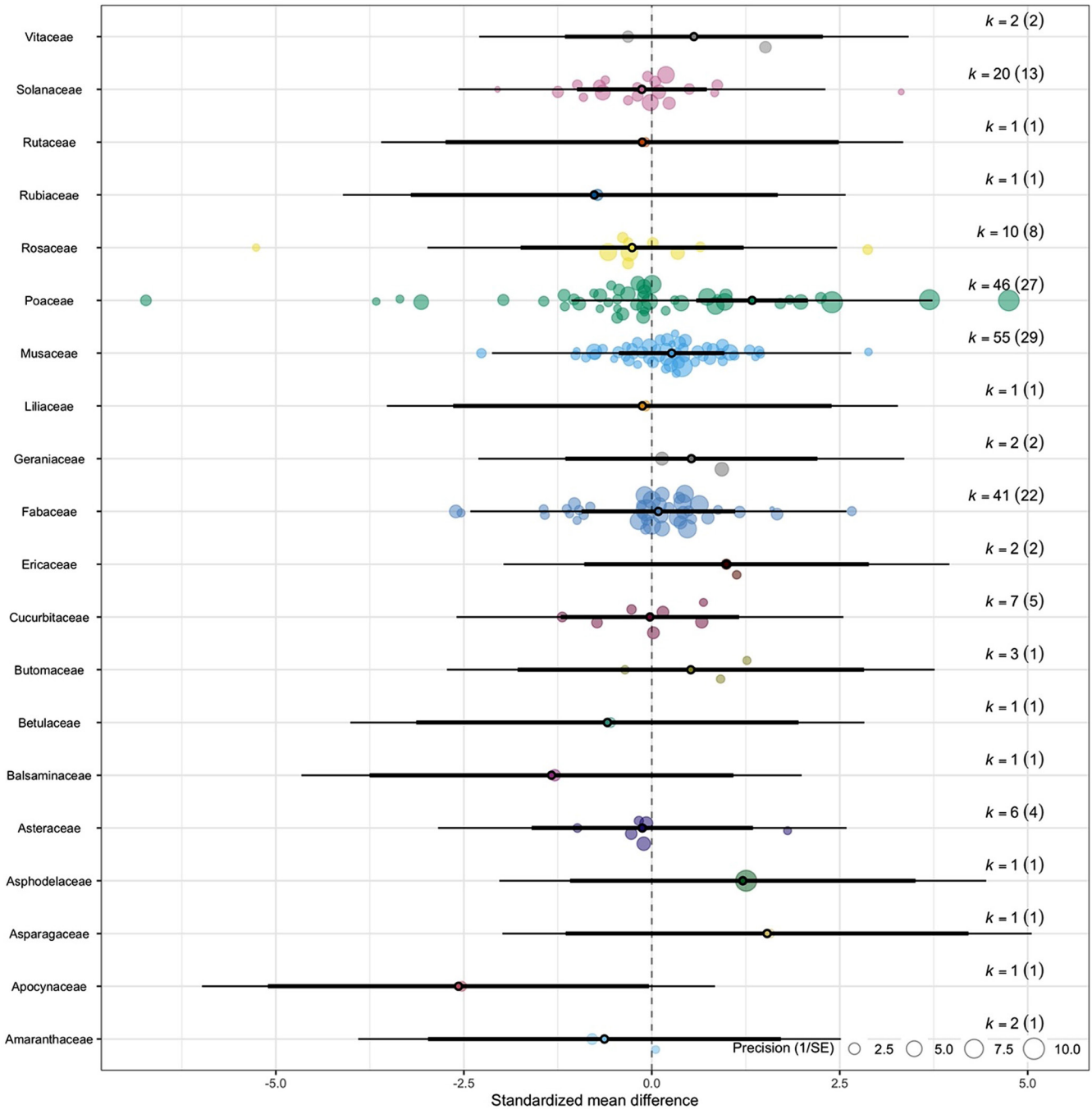

Figure 2. An orchard plot showing the distribution of effect sizes across plant families. *k* is the number of effect sizes, and numbers in parentheses are the number of studies. 95% confidence intervals are displayed as bold lines around the overall estimates (bold circles) while 95% prediction intervals are shown with thinner lines. Positive standardized mean difference values indicate greater pathogen resistance in diploids than in polyploids. Poaceae shows the strongest advantage of diploids over polyploids in pathogen resistance, while Apocynaceae shows the strongest difference in resistance in favor of polyploids.

We expected that polyploids would exhibit superior resistance than diploids because R-gene alleles, which mediate resistance to biotrophic and hemibiotrophic pathogens, might be present in double their quantity in polyploids relative to diploids. Instead, we found no significant differences in resistance to biotrophs between diploids and polyploids. While this may be due to chance alone, especially since polyploids show significantly greater resistance to hemibiotrophs, these results may instead indicate that polyploidy causes breakdown in R-gene signaling pathways, or that doubled R-genes are lost during diploidization (Innes et al. 2008, Soltis et al. 2010). This seems to be especially the case in

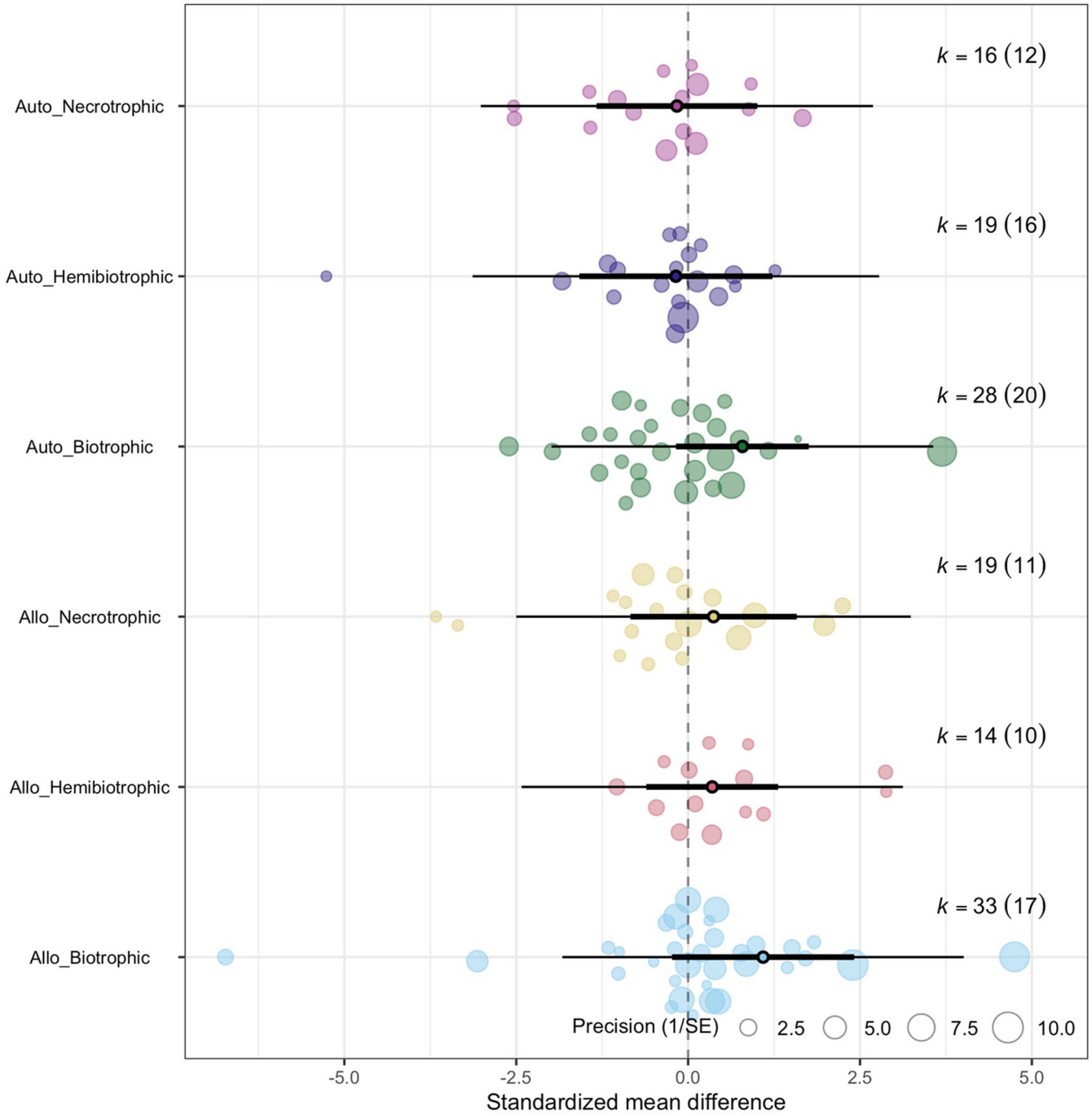

Figure 3. An orchard plot showing the distribution of effect sizes across combinations of polyploid types ('Auto' for autopolyploids and 'Allo' for allopolyploids) and pathogen lifestyles. *k* is the number of effect sizes, and numbers in parentheses are the number of studies. 95% confidence intervals are displayed as bold lines around the overall estimates (bold circles) while prediction intervals are shown with thinner lines. Positive standardized mean difference values indicate greater pathogen resistance in diploids than in polyploids. Effect sizes for groups with polyploid types or pathogen lifestyles labeled 'Both' or 'Unknown' are not shown.

allopolyploids, in which one would expect to see higher allelic diversity of R-genes, yet which consistently show decreased resistance relative to diploids. While the degree to which allopolyploidy disrupts proper genomic functioning is still uncertain (Parisod et al. 2010), it is plausible that signaling pathways are generally disrupted after allopolyploidization, and heterosis effects often seen in allopolyploids may require processes like diploidization to reorganize genomes before beneficial traits can appear (Dodsworth et al. 2016).

The apparent superiority of synthetic polyploids, particularly synthetic autopolyploids, over established ones in resisting pathogens relative to their diploid counterparts may

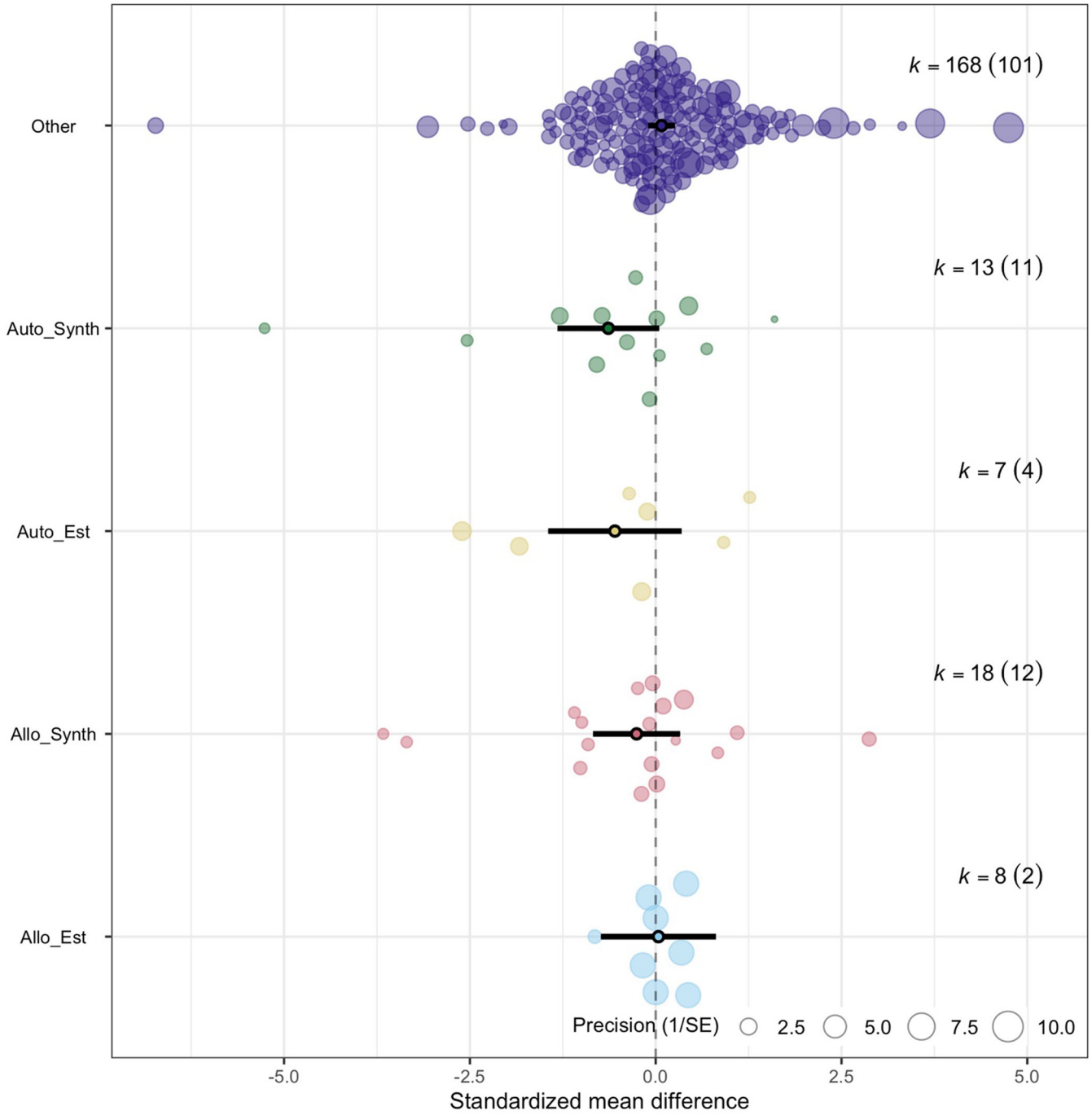

Figure 4. An orchard plot showing the distribution of effect sizes across combinations of whether polyploids were labeled as autopolyploid ('Auto') or allopolyploid ('Allo') as well as synthetic ('Synth') or established ('Est'). All other effect sizes, where these designations were unable to be made with certainty, fall into the 'Other' category. 95% confidence intervals are displayed as bold lines around the overall estimates (bold circles) while prediction intervals are shown with thinner lines. Positive standardized mean difference values indicate greater pathogen resistance in diploids than in polyploids. Synthetic autopolyploids show the greatest resistance relative to diploids.

also be evidence that genome reorganization leads to a loss of R-gene alleles, though this finding is open to interpretation. For example, Clo and Kolář (2022) found that younger, synthetic polyploids exhibit lower amounts of inbreeding depression relative to ones that are older and/or established. Our finding suggests that, in crop improvement efforts, any initial advantages of polyploidy may be short-lived (without, perhaps, subsequent breeding efforts). However, we are cautious about these findings due to the difficulty in demarcating 'synthetic' from 'established' polyploids (Tayalé and Parisod 2013), especially in crop plants with unclear histories of anthropogenic intervention.

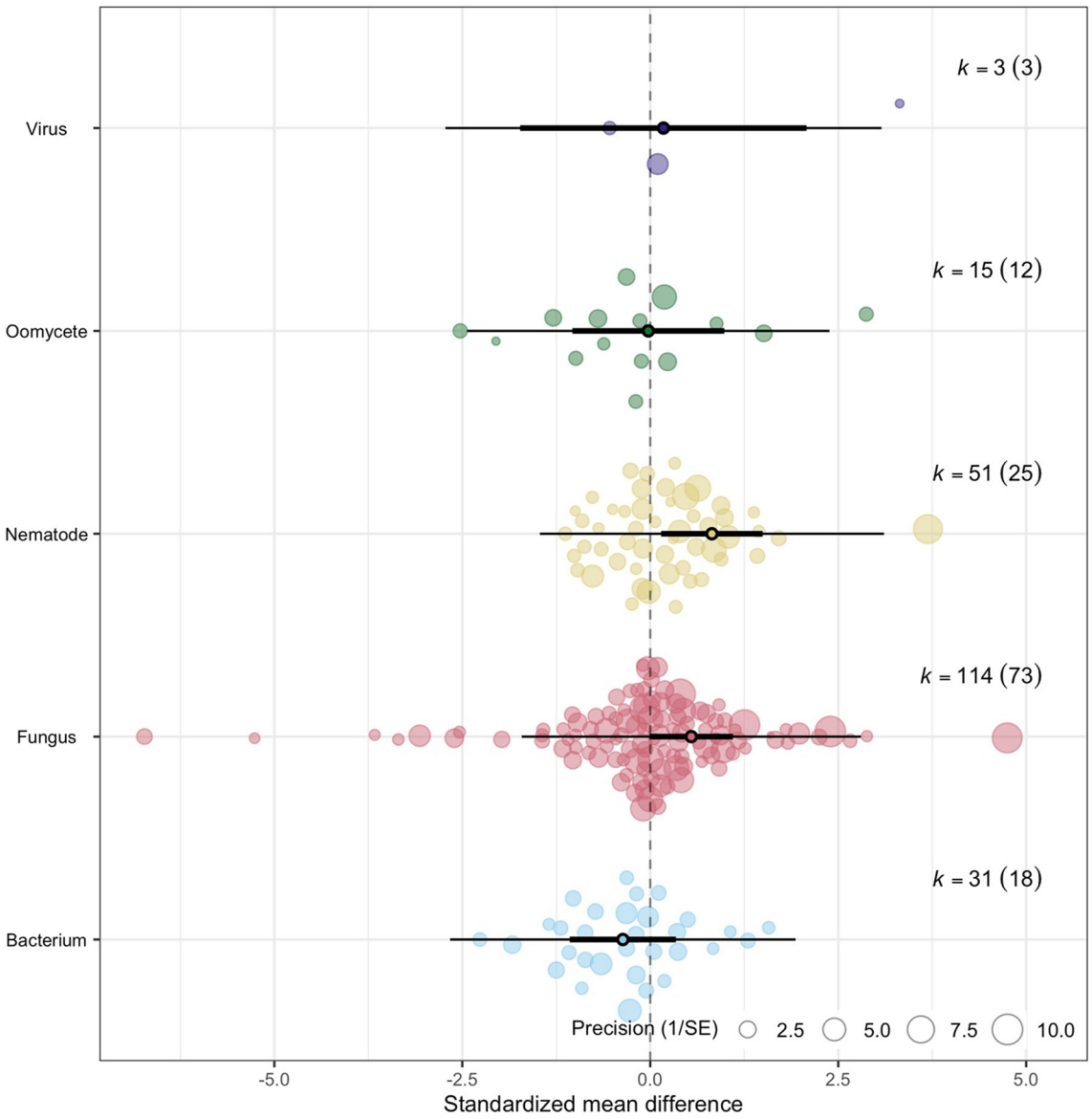

Figure 5. An orchard plot showing the distribution of effect sizes across pathogen types. *k* is the number of effect sizes, and numbers in parentheses are the number of studies. 95% confidence intervals are displayed as bold lines around the overall estimates (bold circles) while prediction intervals are shown with thinner lines. Positive standardized mean difference values indicate greater pathogen resistance in diploids than in polyploids. Diploids outperform polyploids in resistance to pathogens that are fungi or nematodes.

The large amount of heterogeneity indicated by the I^2 statistics (93.4% for the robust model with all effect sizes included) suggests that other factors besides those examined in our study may shed further light on the differences in pathogen resistance between polyploids and diploids. Within-study effects (19%) are present, but not terribly large, which bodes well for the ability of future researchers to identify other explanatory moderators, especially given the complexity of the effects of polyploidy and the difficulty of generalizing them across clades and ecological conditions (Stebbins 1950, Soltis and Burleigh 2009). This randomness as well as dependence on ecological context are likely large parts of what is being captured by the within-study effect. Polyploid success is highly contingent, depending on being at the 'right place at the right time' (Oswald and Nuismer 2011). Regarding diversification over long periods, Sessa (2019) calls polyploidy a 'Las Vegas strategy,' where genome multiplications usually end in 'losses' (i.e. extinction), but on rare occasions cause plants to 'win big' (i.e. succeed and diversify). When it comes to pathogen resistance, this randomness seems very explanatory, but in most cases, polyploidy appears to lead to only small losses and wins relative to diploids, being more of a 'Reno strategy.'

As anthropogenic climate change continues to raise global temperatures and atmospheric carbon dioxide concentrations, it is possible that plants could become more susceptible to pathogen attacks, raising the specter of massive crop

losses (Lake and Wade 2009, Velásquez et al. 2018). While it has been proposed that experimentation with polyploidy may improve crops in the face of nutritional and growth losses expected under future climate change (Cheng et al. 2022), our results indicate that polyploidy is not a reliable path forward for increasing pathogen resistance in crops.

Conclusions

We found that there are no consistent overall differences between diploids and polyploids in their abilities to resist pathogens. None of the moderators included in our multilevel model showed significant effects. The overall similarity in resistance to biotrophic and hemibiotrophic pathogens between diploids and polyploids suggests that increased numbers of R-gene alleles do not lead to decreased infections in polyploids, and the lack of difference between diploids and polyploids in cultivated plants calls into question the utility of using polyploid crops for decreasing susceptibility to disease. Given the need for crop breeding strategies that can address the likely increase in disease susceptibility that will accompany future climate change, our results are disconcerting, but they may guide agriculturists toward other strategies for increasing crop resistance to infections.

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Author contributions

Eric R. Hagen: Conceptualization (lead); Data curation (equal); Formal analysis (equal); Methodology (equal); Writing – original draft (lead); Writing – review and editing (equal). **Chase M. Mason**: Data curation (equal); Formal analysis (equal); Methodology (equal); Writing – original draft (equal); Writing – review and editing (equal).

Data availability statement

Data are available from the Dryad Digital Repository: https://doi.org/10.5061/dryad.4qrfj6qg7 (Hagen and Mason 2023).

Supporting information

The Supporting information associated with this article is available with the online version.

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