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RESEARCH ARTICLE

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Effect of maternal infection on progeny growth and resistance mediated by maternal genotype and nutrient availability

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Abstract

1. Maternal effects of pathogen infection on progeny development and disease resistance may be adaptive and have important consequences for population dynamics. However, these effects are often context-dependent and examples of adaptive transgenerational responses from perennials are scarce, although they may be a particularly important mechanism generating variation in the offspring of long-lived species.
2. Here, we studied the effect of maternal infection of *Plantago lanceolata* by *Podosphaera plantaginis*, a fungal parasite, on the growth, flower production and resistance of the progeny of six maternal genotypes in nutrient-rich and nutrient-poor environments. For this purpose, we combined a common garden study with automated phenotyping measurements of early life stages, and an inoculation experiment.
3. Our results show that the effects of infection on the mother plants transcend to impact their progeny. Although maternal infection decreased total leaf and flower production of the progeny by the end of the growing season, it accelerated early growth and enhanced resistance to the pathogen *P. plantaginis*.
4. We also discovered that the effects of maternal infection affected progeny development and resistance through a three way-interaction between maternal genotype, maternal infection status and nutrient availability.
5. *Synthesis*. Our results emphasize the importance of maternal effects mediated through genotypic and environmental factors in long-living perennials and suggest that maternal infection can create a layer of phenotypic diversity in resistance. These results may have important implications for both epidemiological and evolutionary dynamics of host–parasite interactions in the wild.

KEYWORDS

automated phenotyping, host–parasite interactions, maternal effects, perennial plant, *Plantago lanceolata*, transgenerational defence priming

1 | INTRODUCTION

Plant pathogens are ubiquitous in both natural and managed ecosystems, and they have complex and diverse effects on their host's survival and reproduction. These effects include host mortality, reduced growth and lower seed production, which is caused either directly by the pathogen or indirectly due to hampered plant growth (Agrios, 2005; Alexander, 2010; Gilbert, 2002; Jarosz & Davelos, 1995). Natural plant populations support extensive phenotypic variation in disease resistance (Antonovics et al., 2011; Laine et al., 2011; Lebeda et al., 2014).

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Traditionally, phenotypic variation in resistance is assumed to have a genetic basis (Flor, 1956), where variation is maintained through a co-evolutionary arms race with pathogens (Bergelson, Dwyer, et al., 2001; Bergelson, Kreitman, et al., 2001). However, we are becoming increasingly aware that the local context—consisting of both abiotic and biotic variables—may alter the resistance phenotype directly and through genotype-by-environment interactions (Blanford et al., 2003; Raghavendra & Newcombe, 2013). To date, remarkably little is known about how the conditions experienced by the parental generation contribute to phenotypic resistance diversity through non-genetic inheritance (Bonduriansky & Day, 2009). In particular, transgenerational priming (Bruce et al., 2007; Luna et al., 2012; Martinez-Medina et al., 2016; Mauch-Mani et al., 2017)—whereby maternal infection renders the offspring more resistant against pathogen attack (Holeski et al., 2012)—could create a layer of phenotypic diversity in resistance to provide further protection from infection.

It is well established that pathogen infection can prime defence plasticity in plant hosts, resulting in enhanced activation of induced defence mechanisms (Bruce et al., 2007; Mauch-Mani et al., 2017). Although often the primed response may be limited to a short time window (Frost et al., 2008; Luna et al., 2012), there is also evidence of a response that is maintained throughout the plant's life cycle (Martinez-Medina et al., 2016; Mauch-Mani et al., 2017). The effects of priming can be passed on to subsequent generations through the embryo via non-genetic mechanisms including DNA methylation, chromatin remodelling and small RNA signalling (Mauch-Mani et al., 2017; Singh & Roberts, 2015), and therefore transgenerational priming represents a type of plant immunological memory (Martinez-Medina et al., 2016). Increased resistance against pathogens in the progeny of infected mothers was first shown in tobacco plants inoculated with tobacco mosaic virus (Roberts, 1983), and later studies mostly performed with short-living annuals and model species have described similar responses (reviewed by Holeski et al., 2012). Results from these studies have confirmed that transgenerational priming may enhance resistance in the progeny of plants exposed to the same or different pathogen species (Kathiria et al., 2010; Slaughter et al., 2012) and hence, maternal effects are often viewed as a form of adaptive, transgenerational plasticity (Agrawal et al., 1999; Frost et al., 2008; Marshall & Uller, 2007; Mauch-Mani et al., 2017; Uller et al., 2013; Yin et al., 2019).

Under pathogen attack, both host growth and reproduction are typically reduced, host resources are allocated to defence and depleted by the parasite, and in some cases hosts are exposed to pathogen-produced toxins (Tsuge et al., 2013; Van Kal, 2006; Yoder, 1980). Hence, transgenerational effects of maternal infection—or other stressors—are not always expected to be adaptive (Burton & Metcalfe, 2014; Marshall & Uller, 2007). In agricultural crops, it is well established that maternal infection may reduce the quantity of the produced seeds (Agrios, 2005; Argyris et al., 2003; Strange & Scott, 2005). In addition to producing fewer seeds, infected plants have been reported to produce smaller seeds, and seeds that have reduced protein content when compared to seeds produced by healthy plants (Edwards et al., 2001; Murray et al., 1995). Parental pathogen infection may also limit the growth in asexually spreading clonal species (Piqueras, 2001). While such reductions in seed quality due to parental infection are expected to affect seed viability, seedling emergence, growth and reproduction, to date this has not been verified, particularly in natural populations (Burdon & Laine, 2019).

How consistently maternal infection generates variation in offspring fitness and resistance depends on how the effects vary across genotypes and environments. Experiments that focused on maternal effects induced by insect herbivores have shown that maternal effects can be highly genotype specific (Agrawal, 2002; Colicchio, 2017; Holeski et al., 2013), and may vary across genotypes in different abiotic conditions (Münzbergová & Hadincová, 2017; Rendina González et al., 2018; Vivas et al., 2013; Vu et al., 2015). Hence, how strong and durable the pathogen-induced priming effects are, may depend on the genotype-specific response to infection, and the quality and predictability of the environment that both mothers and their offspring encounter (Agrawal et al., 1999; Herman & Sultan, 2011; Kuijper & Hoyle, 2015; Luna et al., 2012; Mauch-Mani et al., 2017).

In this study, we were particularly interested in whether the effects of maternal infection depend on the level of nutrients available to the offspring. Nutrient availability is considered a key determinant of plant growth but may also affect plant disease tolerance and resistance (Agrios, 2005; Amtmann et al., 2008; Dordas, 2008; Mittler & Blumwald, 2010; Suzuki et al., 2014). Generally, nutrient stress is shown to weaken plant defences (Amtmann et al., 2008; Dordas, 2008; Suzuki et al., 2014). However, there is also evidence that increasing nutrients can increase the severity of infection, especially by obligate pathogens (Dordas, 2008), and that the effects of nutrients on disease resistance vary among plant species (Veresoglou et al., 2013) and genotypes (Laine, 2007). Agriculture has replaced natural habitats across the world, and those remaining are increasingly occurring adjacent to agricultural practices (Foley et al., 2005). With both nutrient spillover from agricultural habitats (Matson et al., 1997), and nitrogen deposition Hyvönen et al., 2007), natural populations are increasingly exposed to new levels of nutrients, and there is a pressing need to quantify the consequences of this.

Long-lived perennial plants may face short periods of stress with respect to their life span, and thus transgenerationally induced phenotypic plasticity may be a particularly advantageous strategy to cope with changing conditions (Auge et al., 2017; Donelson et al., 2018). Here, we investigate whether maternal infection by powdery mildew *Podosphaera plantaginis* affects progeny growth, flower production and resistance phenotype in the perennial ribwort plantain *Plantago lanceolata* in a series of experiments described in Figure S1. We also tested whether the effect varies depending on the maternal genotype, and between two contrasting nutrient environments of the offspring. In our study area in the Åland archipelago *P. lanceolata* grows on open dry meadows that are characterized by low in nutrient concentrations. However, soils in populations adjacent to agricultural fields have significantly higher nutrient levels (H. Susi & A.-L. Laine, pers. comm.), with potentially far reaching implications for the

ecology and evolution of these populations. We used seeds from six maternal *P. lanceolata* genotypes that had been clonally propagated into multiple individuals and grown in common garden conditions for 3 years with and without infection. First, we monitored early-life development of the offspring, in two contrasting nutrient environments in the greenhouse conditions using automated phenotyping technology. Subsequent life-history traits were then monitored under the common garden settings. Finally, the progeny resistance against powdery mildew strains was assessed in a laboratory inoculation trial. The epidemic of *P. plantaginis* peaks late in the growing season, and hence we measured the resistance of mature progeny.

2 | MATERIALS AND METHODS

2.1 | *Plantago lanceolata* and *Podosphaera plantaginis* interaction

Plantago lanceolata L., the ribwort plantain, is a perennial monoecious herb (Sagar & Harper, 1964). It is an obligate outcrosser that can reproduce sexually via wind pollination, or asexually by producing vegetative side rosettes (Laine, 2004). Seeds drop close to the mother plant forming a long-term seed bank (Bos, 1992). Obligate biotroph *P. plantaginis* (Castagne; U. Braun and S. Takamatsu), belonging to the order *Erysiphales* in Ascomycota, is a powdery mildew parasite which is specific to *P. lanceolata*. Like all powdery mildews, *P. plantaginis* completes its life cycle as localized lesions on host tissue where it obtains nutrients via haustorial feeding roots. Infection causes significant stress for host plant, decreasing host growth and increasing host mortality (Laine, 2004; Susi & Laine, 2015). Almost 4,000 natural *P. lanceolata* populations located in the Åland islands, SW of Finland, have been surveyed annually since 2001 to study the metapopulation dynamics of *P. plantaginis* in this highly fragmented pathosystem (Jousimo et al., 2014). Between epidemic seasons, the parasite survives on decaying host leaves by producing overwintering structures, chasmotechia (Tack & Laine, 2014).

2.2 | Plant material

To produce genetically identical mother plants that differed in their infection status, we set up a common garden study in Lammi Biological station (Hämeenlinna, Finland), where cloned *P. lanceolata* plants were grown over years 2011–2013 with and without the pathogen *P. plantaginis* (Susi & Laine, 2015). Originally, seeds for the six mother plants were collected in 2010 from six distinct natural populations located in the Åland islands. The grown six mother genotypes were then propagated into altogether 51 individuals (2–14 clones representing each genotype; Figure S2a) and planted into common garden populations (Susi & Laine, 2015). Soil used in the experimental populations was a mixture of commercial soil substrate and sand (1:1), and the abiotic environmental conditions were same for all the plants. In this multi-year experiment, half of the mothers were inoculated with two *P. plantaginis* strains (strain IDs L3 and L10, harvested from two allopatric populations in Åland islands) for three consecutive years. The infection level of each host plant was measured four times during every growing season through 2011–2013 (for more details, see Susi & Laine, 2015). We also measured the growth and reproduction of the host plants as number of leaves and flowers, respectively, first in July and secondly at the end of the growing season in September. The results of the analysis of this multi-year study are reported in Susi and Laine (2015), here we focus on reporting the findings regarding the six maternal genotypes that were included in this study.

After 3 years of repeated infection, we collected seeds from the six maternal genotypes, two clones of each (in total from 12 plants), where one was always heavily (more than 30% of leaves) infected (infected mothers) and another clone represented the same genotype, but was never infected (healthy mothers). From each of the 12 maternal plants, 18 seeds (half- or full-sibs, resulting from natural wind-pollination) were sown into 0.75 L pots. Half of the seeds ($n = 9$) were sown into fertilized, nutrient-rich soil and the other half ($n = 9$) to nutrient-poor soil (1:1 mixture of commercial soil substrate without added nutrients and sand). The plants in the nutrient-rich treatment received slow-release fertilizer twice, first portion (25 g of NPK 4-1-2 per plant) prior to the planting, and the second portion (20 g/plant) when we started the second common garden experiment (details below), in total 1.8 g of N, 0. 45 g of P and 0.9 g of K. No fertilizer was added to the plants in the nutrient-poor treatment. Finally, the experiment consisted in total of 216 plants in four experimental groups: offspring in nutrient-rich or nutrient-poor soils originating from the healthy and infected mothers, 54 plants in each group. Seedlings were grown in the greenhouse conditions at 20 ± 2°C (day) and 16 ± 2°C (night) with 16:8 L:D photoperiod. When the seedlings were 18 days old, we moved them into National Plant Phenotyping Infrastructure (NaPPI, https://www.helsinki.fi/en/infrastructures/national-plant-phenotyping) platform.

2.3 | Early life measurements

We used the plant phenotyping facility located at the University of Helsinki, Viikki campus, for the phenotypic characterization of progeny's growth patterns. The plants were randomly arrayed into rows that were rotated every night to avoid microclimate differences influencing growing conditions. Plants were imaged every 72 hr by overhead CCD camera for Red Green Blue (RGB) images positioned in a PlantScreen™ analysis chamber (PSI, Czech R.). In addition, images from three sides (angles 0°, 120° and 240°) were taken simultaneously with overhead imaging. The images were obtained for all 216 plants in total 13 times during the 8-week period and pre-processed online as described in Awlia et al. (2016) to allow collecting RGB data. Obtained binary images from top view were used for calculating growth parameter of leaf area (PlantScreen™ analyzer, PSI, Czech R.), which has been found to yield precise data for several plant species (Humplík et al., 2015).

Because all three side angles (0°, 120° and 240°) resulted in similar estimations, the parameter 'height' only from angle 0° was included in the further analyses. The bolting day (a day when first flower stem was visible) and the number of developing flowers was manually observed and counted for each plant individual from the top RGB images.

2.4 | Common garden populations to measure offspring development over the growing season

To determine progeny growth and flowering traits under semi-natural conditions, we established a common garden population in the Viikki campus experimental field (Helsinki, Finland) after the phenotyping characterization. When the plants were 8-week old, they were acclimated for 1-week outdoors and then were planted into six common garden plots. In each plot, there were 36 plants of which half (18) of the plants originated from infected mothers, and half (18) from healthy mothers. Each maternal genotype was represented by three offspring from healthy plants, and three offspring from infected plants in each plot. The positions of the plants within plots were randomly assigned. To exclude the variation in the soil, the top layer (30 cm) was replaced with a mixture of commercial substrate without nutrients and sand (1:1) and the experimental area was covered with plastic to avoid weeds and asexual reproduction of the experimental plants. The plants in the nutrient-rich treatment (i.e. in the three experimental plots) received a second portion of fertilizer (20 g/plant of NPK 4-1-2) prior to planting. The experimental plots (three fertilized and three non-fertilized) were randomly arrayed in the experimental field. We counted the number of leaves, the length and width of the longest leaf, the number of flowers, the height of the longest stalk and the height of the inflorescence in the longest stalk, when the progeny plants were 16, 20 and 24 weeks old. After the last manual measurements, flower stalks were removed and the above-ground biomass was collected, dried and weighted.

2.5 | Inoculation experiment

To characterize the resistance of the progeny of infected and healthy mothers against powdery mildew *P. plantaginis*, we performed an inoculation experiment when the progeny plants were 24-weeks old. Leaves collected from 216 offspring plants were placed in random order on moist filter paper in Petri dishes and inoculated with conidial spores from purified powdery mildew lesions, by evenly brushing the spores on a leaf. We tested the resistance of the progeny plants against four *P. plantaginis* strains (L3, L10, 1630_2 and 877), originally collected from natural populations in the Åland Islands. Lesion development was monitored under a dissecting microscope every second day, starting at day 3 and continued until day 13. For the statistical analysis, resistance was scored on day 13 as 0 = no resistance (infection) or 1 = resistance (no infection), when there was no growth, or if the test plant showed rapid cell death (hypersensitive response) around the inoculum area.

2.6 | Statistical analysis

We ran all the analyses, including linear fixed effect models, in R software (R Development Core Team, 2016), using the LME4 package (Bates et al., 2015). Each analysis was started with a full model with all the interactions included. Minimum adequate models were assessed through stepwise simplification and selection based on likelihood ratio or X^2 values to compare significant interactions (Crawley, 2012). The effect of significant independent variables was derived from analysis of the minimum adequate model with the CAR package (Fox & Weisberg, 2011). Overdispersion was tested when necessary and accounted for by fitting an observation-level random effect. To detect the significant differences in resistance among genotypes in different nutrient treatments, a post hoc analysis was performed using the MULTCOMP package (Hothorn et al., 2008).

2.7 | Growth and reproduction of the mother plants

To test whether the maternal genotypes differed in their infection levels, we first ran a generalized linear model where we defined the proportion of diseased leaves (n = 25 plants) at the highest peak of epidemics (July 2013) as the response variable, and genotype as categorical explanatory variables. To test whether the genotypes, and infected and healthy plants differed in their growth, we defined the number of leaves (in September 2013 count) as the response variable, and genotype and pathogen treatment (inoculation or no inoculation) as the categorical explanatory variables in the analysis. To understand the effect of infection and genotype on plant reproduction, we used the summed number of the flowers counted in July and September as the response variable, and genotype and pathogen treatment as the class explanatory variables. In models with two explanatory variables, the interaction of these variables was included in the model. In all models, we assumed a Poisson distribution of errors.

2.8 | Early growth and flower production of the progeny

To analyse the differences in early growth between nutrient treatment and maternal infection groups, we fitted mixed polynomial linear model (Mirman, 2014; Pavicic et al., 2017) for measurements obtained from the RGB images. The class explanatory variables treatment, maternal genotype, maternal infection and their interactions were fixed effects. The polynomial order of days after sowing was added as fixed effect to take into account the curved growth patterns over time. Plant ID was nested under a day and included as random effect to account for the repeated measurements. This structure was fitted to response variables area in mm^2 and height

computed from the top RGB images. To test whether experimental groups varied in their flower production, we analysed the number of flowers in every imaging day using a generalized linear mixed-effect model with log link and Poisson distribution of errors, with the same explanatory variables as above. The proportion of plants that were flowering (0/1) measured in every imaging day was analysed as a likelihood using a generalized linear model with a logit link function. The polynomial order of days and class explanatory variables treatment, maternal infection, maternal genotype and their interactions were defined as fixed effects.

2.9 | Growth and flower production of the progeny in the common garden study

To determine the differences between progeny groups among manually measured traits in the common garden experiment, we analysed the data from the last measuring point (24 weeks). The number of leaves and flowers was analysed as generalized linear mixed-effect models with a Poisson distribution of errors. Continuous response variables (the length and width of the longest leaf, the height of the tallest stalk, the height of inflorescence in the tallest stalk and the total biomass from dried plants) were analysed as linear mixed-effect models. The fixed class explanatory variables were nutrient treatment, maternal infection, maternal genotype and their interactions. The common garden plot was defined as a random effect in the models.

2.10 | Progeny resistance to powdery mildew measured through inoculations

To test whether maternal infection, genotype or offspring nutrient treatment affected the resistance responses against powdery mildew strains, we analysed the inoculation experiment data derived from laboratory observations. The inoculation response (0/1) on day 13 post-inoculation was modelled as a likelihood using a generalized mixed-effect model with a logit link function. Maternal infection, genotype, offspring nutrient treatment and their interactions were fixed effects, and plant ID was nested under a mildew strain (the four mildew genotypes) as a random intercept.

3 | RESULTS

3.1 | Growth and reproduction of the mother plants

All the mother plants grown in common garden conditions with pathogen inoculation became heavily infected (33%–100% of the leaves infected). There were no significant differences among the genotypes in the proportion of infected leaves ($p = 0.9126$; Tables S1 and S2; Figure S2a). We found that the mother plant genotypes varied in the number of leaves they produced ($p < 0.0001$; Tables S1 and S2; Figure S2b), and that the effect of pathogen inoculation on growth was genotype specific ($p < 0.0001$; Tables S1 and S2; Figure S2b). However, there was no direct significant effect of pathogen treatment on growth ($p = 0.749$; Tables S1 and S2; Figure S2b). When we analysed the reproduction of the mother plants, we found that healthy plants generally produced more flowers than infected plants (96.9 ± 5.6 vs. 77.1 ± 5.2; $p < 0.0001$; Tables S1 and S2; Figure S2c). We also found a significant interaction between genotype and pathogen treatment ($p < 0.0001$; Table S1; Figure S1c). Genotype alone had a significant effect on flower production ($p < 0.0001$; Table S1; Figure S1c).

3.2 | Early growth and flower production of the progeny

To understand the differences in the progeny growth rates during the first 8 weeks, we compared the accumulation of the total leaf area (mm^2) and height using the RGB imaging approach. The measured leaf area and plant height increased with time ($p < 0.0001$, Table 1; Figure 1; Table S3), and plants in the nutrient-rich conditions grew

TABLE 1 Factors contributing to early growth of *Plantago lanceolata* plants in the greenhouse study analysed with polynomial linear mixed-effect models. Statistics for minimum adequate models with smallest AIC values are reported. Significant values are highlighted in bold

	Area (mm^2)		Height		Number of flowers		Flowering individuals	
Source	**LRT**	***p***	**LRT**	***p***	**LRT**	***p***	**LRT**	***p***
Days from sowing, polynomial order, $df = 3$	1,239.67	**<0.0001**	961.68	**<0.0001**	858.77	**<0.0001**	21.64	**<0.0001**
Nutrient treatment, $df = 1$	96.91	**<0.0001**	19.28	**<0.0001**	377.05	**<0.0001**	3.16	0.07
Maternal infection, $df = 1$	16.38	**<0.0001**	0.42	0.51	2.81	0.09	3.42	0.06
Genotype, $df = 5$	9.24	0.099	9.63	0.086	6.07	0.2	15.62	**0.008**
Nutrient treatment × maternal infection, $df = 1$	10.21	**0.001**	6.09	**0.01**	12.99	**0.0003**	1.54	0.21
Random	**ID**	***Days***	**ID**	***Days***	**ID**	***Days***	**ID**	***Days***
Variance	159,622,863	251,066	23,784.8	32.85	22.76	0.03	0.01	18.38
St. dev.	12,634.20	501.10	154.22	5.73	4.77	0.16	0.11	4.29

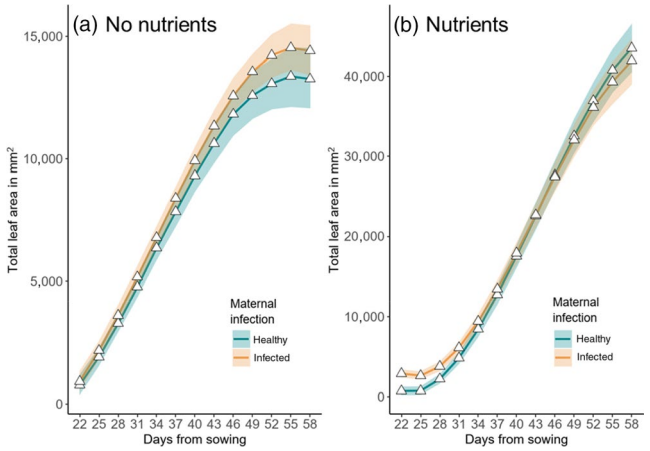

FIGURE 1 Development of detected top leaf area (mm^2) from RGB images. Plant early development between first 22–58 days as measured in pixels and transferred into mm^2, detected by RGB imaging. Growth of progeny of healthy and infected mothers was followed in two treatments, where (a) no nutrients were added and (b) nutrients were added. Curves and confidence intervals from fitted models, $n = 54$ plants per treatment and maternal infection group. The triangles represent the average for each group at a given time point

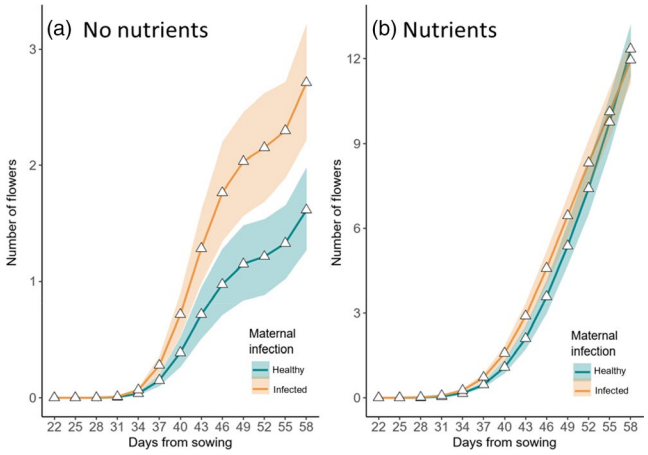

FIGURE 2 Flower development in the progeny of healthy and infected mothers. Number of flowers detected from top RGB images through early life in two treatments, where (a) no nutrients were added and (b) nutrients were added. Curves from fitted models with 95% confidence intervals, $n = 54$ plants per treatment and maternal infection group. The triangles represent the average for each group at a given time point

faster and produced more leaves than the plants in the nutrient-poor conditions ($p < 0.0001$, Table 1; Figure 1; Table S3). The progeny of infected mothers produced more leaf area ($p < 0.0001$, Table 1; Figure 1; Table S3), especially in the nutrient-poor treatment (interaction between nutrition treatment and maternal infection, $p < 0.0001$, Table 1; Figure 1a; Table S3). Maternal genotype had no effect on the detected leaf area ($p = 0.09$, Table 1) or height ($p = 0.51$, Table 1). Plant height was affected by both the nutrient treatment ($p < 0.0001$, Table 1), and a significant interaction between nutrient treatment and maternal infection ($p = 0.01$, Table 1; Table S3).

To understand how maternal infection and nutrient treatment affect offspring flower production over the first 8 weeks, we quantified the number of flowers from top RGB images. As expected, the proportion of plants that were flowering, and the number of flowers increased with time ($p < 0.0001$ and $p < 0.0001$, respectively, Table 1; Figure 2; Table S2). In the last imaging round on day 58, 67% of progeny of healthy mothers and 81% of progeny of infected mothers were flowering in the nutrient-poor treatment, and 96% of healthy and 98% of progeny of infected mothers were flowering in the nutrient-rich treatment (nutrient treatment, $p = 0.07$, Table 1; Table S3). Maternal infection had no significant effect on the proportion of flowering plants ($p = 0.06$, Table 1), but the genotypes differed in their flowering efforts ($p = 0.008$; Table 1). In general, plants produced significantly more flowers in the nutrient-rich treatment than in the nutrient-poor treatment ($p < 0.0001$, Table 1; Figure 2; Table S3). Neither maternal infection nor genotype had a significant effect on the number of flowers produced (infection $p = 0.09$, genotype $p = 0.2$, Table 1; Table S3). However, we found a significant interaction between nutrient treatment and maternal infection for the number of flowers produced. The progeny of infected mother produced more flowers in the nutrient-poor treatment, while in the high nutrient treatment flower production was similar between the progeny of both infected and healthy mothers (interaction between nutrient treatment and maternal infection, $p = 0.0003$, Table 1; Figure 2; Table S2).

3.3 | Growth and flower production of the progeny in the common garden study

To understand how maternal infection and nutrient treatment affect vegetative growth and the number of flowers produced in 8-week-old to 24-week-old progeny, we measured phenotypic traits manually in common garden populations. The phenotypic measurements conducted at the last time point revealed that the accelerated growth and reproduction rate detected in the progeny of infected mothers during early life were not detected anymore when the plants were 24 weeks old (Figure S3). By the last manual measurement time point in early September, offspring of infected mothers had produced less flowers and leaves than the offspring of healthy mothers (maternal infection $p < 0.0001$ and $p < 0.0001$, respectively; Table 2; Figure 3; Table S4). Plants produced more flowers and leaves in the nutrient-rich treatment (Table 2; Figure 3), and the genotypes also differed in their flower and leaf production ($p < 0.0001$ and $p < 0.0001$, respectively; Table 2; Figure 3; Table S4). We found a significant three-way interaction between maternal infection, nutrient treatment and maternal genotype on both the number of flowers and leaves produced ($p < 0.0001$ and $p < 0.0001$, respectively, Table 2; Figure 3; Table S4). These significant three-way interactions indicate that the effects of maternal infection on leaf size and flower production vary depending on the maternal genotype and the nutrient conditions that the progeny encounter.

TABLE 2 Factors contributing to the growth and resistance of progeny plants in the common garden study and in the laboratory experiment, analysed with linear mixed-effect models. Statistics for minimum adequate models with smallest AIC values are reported. Significant values are highlighted in bold

	No. of leaves		No. of flowers		Longest leaf		Leaf width		Longest stalk		Inflorescence		Biomass		Resistance	
Source	**LRT**	***p***	**LRT**	***p***	**LRT**	***p***	**LRT**	***p***	**LRT**	***p***	**LRT**	***p***	**LRT**	***p***	**LRT**	***p***
Nutrient treatment, $df = 1$	12.93	**0.0003**	6.22	**0.01**	—	*ns*	—	*ns*	12.90	**0.0003**	7.87	**0.005**	5.39	**0.02**	0.12	0.72
Maternal infection, $df = 2$	19.12	**<0.0001**	42.24	**<0.0001**	—	*ns*	2.78	0.095	0.13	0.714	—	*ns*	—	*ns*	3.77	**0.049**
Genotype, $df = 5$	129.35	**<0.0001**	127.77	**<0.0001**	11.35	**0.044**	5.25	0.386	5.88	0.318	11.48	0.04	—	*ns*	25.03	**<0.001**
Maternal infection × Nutrient treatment, $df = 1$	16.99	**<0.0001**	2.86	0.09	—	*ns*	—	*ns*	—	*ns*	—	*ns*	—	*ns*	—	*ns*
Maternal infection × Genotype, $df = 5$	96.19	**<0.0001**	85.68	**<0.0001**	—	*ns*	14.45	**0.013**	24.75	**0.0001**	11.9	0.03	—	*ns*	—	*ns*
Genotype × Nutrient treatment, $df = 5$	97.24	**<0.0001**	23.90	**0.0002**	—	*ns*	—	*ns*	—	*ns*	—	*ns*	—	*ns*	—	*ns*
Maternal infection × Genotype × Nutrient treatment, $df = 5$	77.96	**<0.0001**	68.36	**<0.0001**	—	*ns*	—	*ns*	—	*ns*	—	*ns*	—	*ns*	11.31	**0.04**
Random (plot)														**(Genotype)**		**(Mildew)**
Variance	0.00		0.02		6.18		0.05		3.24		0.02		18.8	1.34		0.30
St. dev.	0.07		0.14		2.49		0.23		1.8		0.16		4.34	1.15		0.54

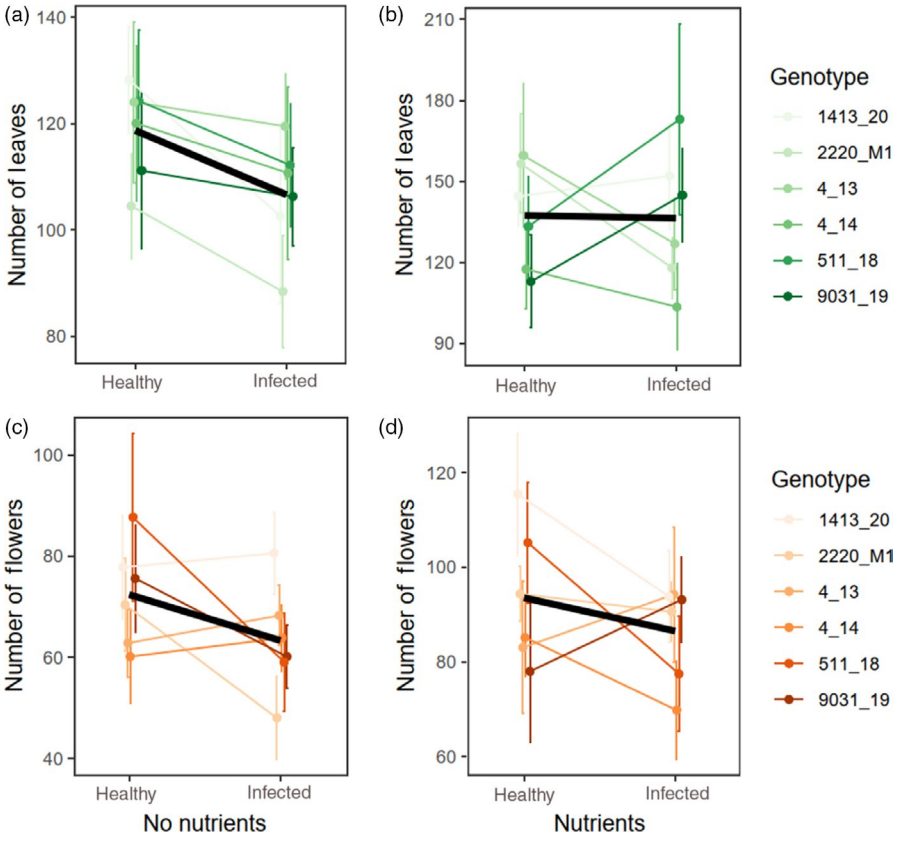

FIGURE 3 Total number of leaves and flowers produced at the end of growing season. Reaction norms for number of leaves in progeny of healthy and infected mothers by genotype in two treatments, where (a) no nutrients were added and (b) nutrients were added. Number of flowers in (c) no nutrients (d) nutrients treatment by genotype. The mean values for reaction norms in each nutrient treatment–maternal infection groups are shown by black lines. Standard errors for each genotype are shown

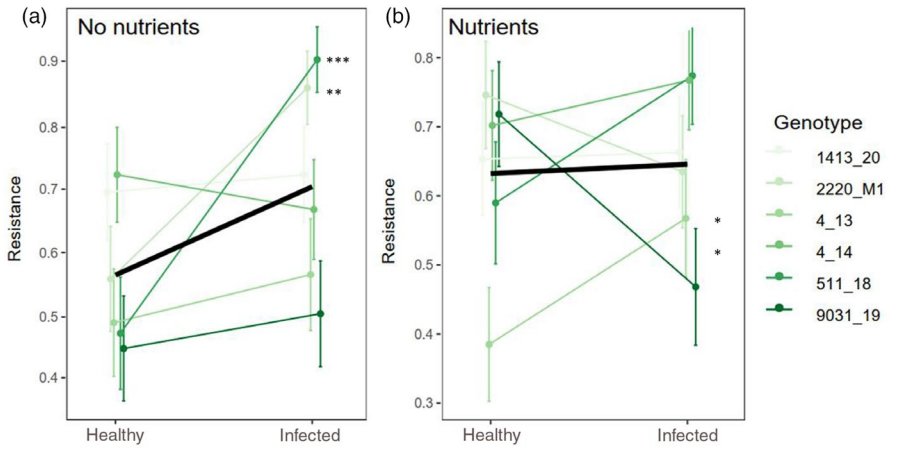

FIGURE 4 Mean resistance of progeny of six *Plantago lanceolata* maternal genotypes in nutrient-poor and nutrient-rich treatments. Reaction norms for resistance in the progeny of healthy and infected mothers in two treatments, where (a) no nutrients were added and (b) nutrients were added. Resistance was tested against four *Podosphaera plantaginis* strains. The mean values for reaction norms in both nutrient treatment–maternal infection groups are shown by black lines. Standard errors for each genotype are shown. Significant interactions among genotypes are indicated by asterisks

For the other measured life-history traits, nutrient treatment was the most significant factor affecting growth ('Nutrient treatment' row in Table 2; Figure S3). Total growth was highest in the high nutrient treatment for the following measurements: number of leaves (on average 111.2 in nutrient-poor and 136.4 in nutrient-rich treatment), number of flowers (68.4 and 89.3), longest stalk (42.6 and 49.1 cm), largest inflorescence size (4.3 and 4.9 cm) and dried biomass (19.6 and 28.6 g), respectively (Figure S3). The length of the longest leaf (on average 24.4 cm) was defined by the maternal genotype, and the leaf width (2.3 cm) and the longest stalk (47.3) by the interaction between maternal genotype and infection (Table 2; Figure S3).

3.4 | Progeny resistance to powdery mildew measured through inoculations

To study how maternal infection and nutrient treatment affect progeny disease resistance, we characterized the progeny phenotypic resistance against powdery mildew *P. plantaginis* strains in a laboratory inoculation experiment when the progeny were 24 weeks old. On day 13 post-inoculation, 36.3% of the leaves showed infection. The resistance phenotype of the progeny was significantly affected by maternal genotype ($p < 0.001$, Table 2; Figure 4; Table S4). There was no significant difference between the detected resistance responses of progeny grown in nutrient-rich or nutrient-poor

conditions (Table 2, $p = 0.72$, Figure 4; Table S4). In general, there was a tendency for progeny of infected mothers to be more resistant than the progeny of healthy mothers ($p = 0.049$, Table 2; Figure 4; Table S4). However, measured levels of resistance varied significantly according to genotype and nutrient as shown by a significant three-way interaction between maternal nutrient treatment, maternal infection and genotype ($p = 0.04$, Table 2; Figure 4). This significant three-way interaction indicates that the effects of maternal infection on progeny resistance vary depending on the mother plant genotype and nutrient conditions that the progeny encounter.

4 | DISCUSSION

The extensive variation in disease resistance that natural populations support (Laine et al., 2011) has been traditionally considered to reflect genetic diversity. However, recent studies have highlighted that the effects of pathogen infection may transcend generations also via non-genetic mechanisms (Holeski et al., 2012; Martinez-Medina et al., 2016; Mauch-Mani et al., 2017; Singh & Roberts, 2015). Pathogen infection in a perennial, long-living plant may affect offspring fitness and resistance phenotype, yet its extent remains largely unknown. Here, we determined how maternal infection affects progeny fitness and resistance in contrasting nutrient environments. Our results reveal that the negative effect powdery mildew infection has on the mother plants also transcends to impact their progeny. By the end of the growing season, the progeny of infected mothers had grown less, and produced fewer flowers than the progeny of healthy mothers. The offspring of infected mothers also had a tendency to have higher resistance against infection by *P. plantaginis*. Moreover, we discovered that maternal infection affected progeny development and resistance both directly, as well as through a three way-interaction between maternal genotype, maternal infection status and nutrient availability.

In line with previous studies (Laine, 2004; Penczykowski et al., 2015), we found a negative effect of infection on the maternal plant generation in the common garden study where plants were grown with and without infection. The negative effect of *P. plantaginis* on the growth of *P. lanceolata* was genotype specific while flower production was negatively affected by infection both directly and via genotype-specific effects. Indeed, there is a strong evidence showing that pathogen infection may reduce host fitness and the quantity and quality of produced seeds (Agrios, 2005; Argyris et al., 2003). Asexual reproduction may also be altered by pathogen infection, as shown in *Trientalis europea* where plants infected by the systemic smut *Urocystis trientalis* produced smaller ramets than healthy plants (Piqueras, 2001). Hence, it is not surprising that the effects of infection may transcend to affect a wide range of life-history traits also in the progeny. We found that both infected mothers and their non-infected offspring produced fewer flowers than the healthy mothers and their offspring. Similarly, progeny of wheat plants infected by powdery mildew *Blumeria graminis* produced fewer and lighter seeds in two generations after an epidemic (Jarosz et al., 1989), and ryegrass *Lolium multiflorum* progeny produced smaller and less vigorous seeds when their parents were infected by a rust pathogen *Puccinia coronata* (Mattner & Parbery, 2007).

We found that the offspring of mothers that had experienced powdery mildew infection had adapted a life-history strategy that favours early reproduction. Initially, in the greenhouse study, the size of progeny plants—measured as leaf area—grew faster if the mother had been infected. Similarly, in their study, Latzel et al. (2009) showed that *Plantago* plants produced more and longer leaves, and had higher photosynthetic capacity if mothers were mechanically disturbed and grown in stressful nutrient-poor environments, even in their second year (Latzel et al., 2010). In our study, the progeny of infected mothers not only grew faster but also bolted earlier in both nutrient treatments. Increased abiotic or biotic stress is known to accelerate flowering in many plant species (Banday & Nandi, 2015; Brachi et al., 2012; Cho et al., 2017). As the differences in progeny growth and flowering patterns due the maternal infection were evidenced mostly in nutrient-poor conditions similar to those experienced by the mother plants, our results are in line with previous studies suggesting that maternal effects are responsible for enhanced growth of progeny in maternal conditions (Donohue & Scmitt, 1998; Galloway, 2005), and the maternal stress results in enhanced growth in progeny (Latzel et al., 2009; Sultan, 1996; Yin et al., 2019). However, we found the opposite effect of maternal infection later in the growing season with progeny of infected mothers being smaller and producing fewer flowers than those of healthy mothers. The epidemic of *P. plantaginis* in Åland islands does not peak until August (Ovaskainen & Laine, 2006) while flowering starts in early June. Hence, this strategy would allow individuals to maximize their fitness when risk of infection is expected to be high.

We found that the progeny of infected *Plantago* mother clones had a tendency to be more resistant to powdery mildew infection. Both the strength and the direction of this trend depended on maternal genotype and nutrient availability. Increasing evidence has shown that pathogens can prime defence plasticity in plants, and that the enhanced resistance may be transgenerational (Holeski et al., 2012; Mauch-Mani et al., 2017). The mechanisms underpinning pathogen-induced epigenetic changes have been detected in several annual species such as *Arabidopsis* (reviewed by Martinez-Medina et al., 2016; Mauch-Mani et al., 2017). Since the discovery that defence priming can be transmitted to future generations, similar effects have been described in cultivated crops (Walters et al., 2013) and in legumes (Martinez-Aguilar et al., 2016; terHorst & Lau, 2012). We tested priming effects after three consecutive years of maternal infection, which may have strengthened the detected resistance responses in the progeny. Because our study spans only one growing season of progeny growth and reproduction, we cannot determine the durability of increased resistance over multiple seasons or generations. However, a study conducted in natural populations of the *Plantago–Podosphaera* system has revealed that resistance is higher in areas where disease encounter rates have been high compared to areas where it has been low (Laine, 2006). This phenomenon could have arisen both via direct genetic changes in these populations or

via transgenerational priming. An exciting future avenue of research would be to determine the extent to which transgenerational priming contributes to the high levels of phenotypic resistance diversity observed in natural populations (Laine et al., 2011).

Importantly, we found evidence for a strong direct genotype effect on disease resistance, and both direct and genotype-by-maternal infection interactions on progeny growth, and reproduction. The strong genotypic differences in disease resistance detected here are in line with earlier studies on *P. lanceolata* (Laine, 2004, 2007). Overall, genotype-specific responses in the progeny to maternal infection may be an important mechanism generating phenotypic variation in these key life-history traits within and among the local populations of *P. lanceolata*. Similarly, maternal effects under herbivory stress in long-living *Populus* sp. varied over genotypes, and offspring differed significantly in their constitutive allocation to growth and resistance traits (Holeski et al., 2013). In the clonal species, for example, in *Trifolium repens*, the direction and strength of maternal effects on offspring biomass were altered positively or negatively depending on the type of abiotic maternal stress, and these effects were highly genotype specific (Rendina González et al., 2018).

A recent meta-analysis (Yin et al., 2019) found that while transgenerational effects often enhance offspring performance in response to both stressful and benign conditions, perennial plants show hardly any transgenerational responses at all, suggesting other strategies for adaptation. However, it should be noted that there are relatively few case studies on perennial plants (21 studies were identified in meta-analysis by Yin et al., 2019), and these have generally not accounted for the high genotypic and environmental variation typical of natural plant populations. The three-way interactions we find between maternal infection, genotype and nutrient availability highlight that maternal effects on progeny fitness and disease resistance are not consistent across genotypes and environments. Our results suggest that the advantage of transgenerational defence priming occurs when environmental conditions become more challenging (Kuijper & Hoyle, 2015; Reiss & Drinkwater, 2018). Overall, fine-scale variation in genetic diversity and environmental conditions typical of natural plant populations are likely to amplify the extent to which maternal effects may generate variation in offspring quality both within and between populations. Our results are in line with findings by Gáspár et al. (2019), who discovered that epigenetic differences across *P. lanceolata* populations were consistently related to genetic and environmental variation. Although the mechanistic underpinning maternal effects of infection in *P. lanceolata* is not known, the strong phenotypic responses we find are compelling evidence of the role that maternal effects in heterogeneous environments can have in generating phenotypic diversity in perennial plants.

Jointly, our results support the idea that phenotypic variation within a generation stems not only from the genetic inheritance of parental alleles but can also be adjusted by maternal effects in response to abiotic and biotic stress experienced by the parental generation (Auge et al., 2017). Overall, our results indicate that maternal infection may influence progeny growth, flower production and phenotypic resistance diversity in long-lived plant species. Both empirical and theoretical work have shown that variation in resistance to natural enemies across spatially structured populations can fundamentally alter epidemiological and evolutionary patterns of infectious disease (Laine et al., 2011; Salvaudon et al., 2008; Tack et al., 2012), yet to our knowledge this is the first study to demonstrate how the effects of maternal infection on progeny resistance vary depending on the mother plant genotype and nutrient conditions that the progeny encounter. This work provides novel insights into how present conditions may shape future generations of perennial plants to help them cope with the same stressors as their parental generation in an adaptive manner.

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AUTHORS' CONTRIBUTIONS

A.-L.L., L.H. and H.S. conceived the ideas and designed the experiment; L.H. and H.S. conducted the experimental work and L.H. analysed the data; L.H. and A.-L.L. led the writing of the manuscript; all the authors contributed to the drafts and gave final approval for publication.

DATA AVAILABILITY STATEMENT

Data available from the Dryad Digital Repository https://doi.org/10.5061/dryad.0zpc866wm (Höckerstedt et al., 2020).

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REFERENCES

Agrawal, A. (2002). Herbivory and maternal effects: Mechanisms and consequences of transgenerational induced plant resistance. *Ecology*, *83*(12), 3408–3415. https://doi.org/10.1890/0012-9658(2002)083[3408:HAMEMA]2.0.CO;2

Agrawal, A., Laforsch, C., & Tollrian, R. (1999). Transgenerational induction of defences in animals and plants. *Nature*, *401*, 61–63. https://doi.org/10.1038/43425

Agrios, G. N. (2005). *Plant pathology* (5th ed.). Elsevier Academic Press.

Alexander, H. M. (2010). Disease in natural plant populations, communities, and ecosystems: Insights into ecological and evolutionary processes. *Plant Disease: An International Journal of Applied Plant Pathology*, *94*(5), 492–503. https://doi.org/10.1094/PDIS-94-5-0492

Amtmann, A., Troufflard, S., & Armengaud, P. (2008). The effect of potassium nutrition on pest and disease resistance in plants. *Physiologia Plantarum*, *133*(4), 682–691. https://doi.org/10.1111/j.1399-3054.2008.01075.x

Antonovics, J., Thrall, P. H., Burdon, J. J., & Laine, A.-L. (2011). Partial resistance in the *Linum-Melampsora* host–pathogen system: Does partial resistance make the red queen run slower? *Evolution; International Journal of Organic Evolution*, *65*(2), 512–522. https://doi.org/10.1111/j.1558-5646.2010.01146.x

Argyris, J., Van Sanford, D., & TeKrony, D. (2003). *Fusarium graminearum* infection during wheat seed development and its effect on seed quality. *Crop Science*, *43*(5), 1782–1788. https://doi.org/10.2135/cropsci2003.1782

Auge, G. A., Leverett, L. D., Edwards, B. R., & Donohue, K. (2017). Adjusting phenotypes via within- and across-generational plasticity. *New Phytologist*, *216*(2), 343–349. https://doi.org/10.1111/nph.14495

Awlia, M., Nigro, A., Fajkus, J., Schmoeckel, S. M., Negrão, S., Santelia, D., Trtílek, M., Tester, M., Julkowska, M. M., & Panzarová, K. (2016). High-throughput non-destructive phenotyping of traits that contribute to salinity tolerance in *Arabidopsis thaliana*. *Frontiers in Plant Science*, *7*, https://doi.org/10.3389/fpls.2016.01414

Banday, Z. Z., & Nandi, A. K. (2015). Interconnection between flowering time control and activation of systemic acquired resistance. *Frontiers in Plant Science*, *6*. https://doi.org/10.3389/fpls.2015.00174

Bates, D., Maechler, M., Bolker, B., & Walker, S. (2015). Fitting linear mixed-effects models using lme4. *Journal of Statistical Software*, *67*(1). https://doi.org/10.18637/jss.v067.i01

Bergelson, J., Dwyer, G., & Emerson, J. J. (2001). Models and data on plant-enemy coevolution. *Annual Review of Genetics*, *35*, 469–499. https://doi.org/10.1146/annurev.genet.35.102401.090954

Bergelson, J., Kreitman, M., Stahl, E. A., & Tian, D. (2001). Evolutionary dynamics of plant R-genes. *Science*, *292*, 2281–2285. https://doi.org/10.1126/science.1061337

Blanford, S., Thomas, M. B., Pugh, C., & Pell, J. K. (2003). Temperature checks the Red Queen? Resistance and virulence in a fluctuating environment. *Ecology Letters*, *6*, 2–5. https://doi.org/10.1046/j.1461-0248.2003.00387.x

Bonduriansky, R., & Day, T. (2009). Nongenetic inheritance and its evolutionary implications. *Annual Review of Ecology, Evolution, and Systematics*, *40*, 103–125. https://doi.org/10.1146/annurev.ecolsys.39.110707.173441

Bos, M. (1992). Gene flow characters and population structure in *Plantago lanceolata*. In P. J. C. Kuiper & M. Bos (Eds.), *Plantago: A multidisciplinary study* (pp. 221–231). Springer-Verlag.

Brachi, B., Aimé, C., Glorieux, C., Cuguen, J., & Roux, F. (2012). Adaptive value of phenological traits in stressful environments: Predictions based on seed production and laboratory natural selection. *PLoS ONE*, *7*(3). https://doi.org/10.1371/journal.pone.0032069

Bruce, T. J. A., Matthes, M. C., Napier, J. A., & Pickett, J. A. (2007). Stressful 'memories' of plants: Evidence and possible mechanisms. *Plant Science*, *173*(6), 603–608. https://doi.org/10.1016/j.plantsci.2007.09.002

Burdon, J., & Laine, A.-L. (2019). *Evolutionary dynamics of plant-pathogen interactions* (pp. I-Ii). Cambridge University Press.

Burton, T., & Metcalfe, N. B. (2014). Can environmental conditions experienced in early life influence future generations? *Proceedings of the Royal Society B: Biological Sciences*, *2014*(281), 1785. https://doi.org/10.1098/rspb.2014.0311

Cho, L.-H., Yoon, J., & An, G. (2017). The control of flowering time by environmental factors. *The Plant Journal*, *90*(4), 708–719. https://doi.org/10.1111/tpj.13461

Colicchio, J. (2017). Transgenerational effects alter plant defense and resistance in nature. *Journal of Evolutionary Biology*, *30*(4), 664–680. https://doi.org/10.1111/jeb.13042

Crawley, M. J. (2012). Statistical modelling. *The R Book*, 388–448. https://doi.org/10.1002/9781118448908.ch9

Donelson, J. M., Salinas, S., Munday, P. L., & Shama, L. N. S. (2018). Transgenerational plasticity and climate change experiments: Where do we go from here? *Global Change Biology*, *24*(1), 13–34. https://doi.org/10.1111/gcb.13903

Donohue, K., & Scmitt, J. (1998). Maternal environmental effects in plants: Adaptive plasticity? In T. A. Mousseau & C. W. Fox (Eds.), *Maternal effects as adaptions* (pp. 137–158). Oxford University Press.

Dordas, C. (2008). Role of nutrients in controlling plant diseases in sustainable agriculture. *A Review. Agronomy for Sustainable Development*, *28*(1), 33–46. https://doi.org/10.1051/agro:2007051

Edwards, M. C., Fetch, T. G., Schwarz, P. B., & Steffenson, B. J. (2001). Effect of Barley yellow dwarf virus infection on yield and malting quality of barley. *Plant Disease*, *85*(2), 202–207. https://doi.org/10.1094/PDIS.2001.85.2.202

Flor, H. H. (1956). The complementary genic systems in flax and flax rust. *Advances in Genetics*, *8*, 29–54. https://doi.org/10.1016/S0065-2660(08)60498-8

Foley, J. A., De Fries, R., Asner, G. P., Barford, C., Bonan, G., Carpenter, S. R., Chapin, F. S., Coe, M. T., Daily, G. C., Gibbs, H. K., Helkowski, J. H., Holloway, T., Howard, E. A., Kucharik, C. J., Monfreda, C., Patz, J. A., Colin Prentice, I., Ramankutty, N., & Snyder, P. K. (2005). Global consequences of land use. *Science*, *309*(5734), 570–574. https://doi.org/10.1126/science.1111772

Fox, J., & Weisberg, S. (2011). *An R companion to applied regression* (2nd ed.). SAGE Publications.

Frost, C. J., Mescher, M. C., Carlson, J. E., & Moraes, C. M. D. (2008). Plant defense priming against herbivores: Getting ready for a different battle. *Plant Physiology*, *146*(3), 818–824. https://doi.org/10.1104/pp.107.113027

Galloway, L. F. (2005). Maternal effects provide phenotypic adaptation to local environmental conditions. *The New Phytologist*, *166*(1), 93–99. https://doi.org/10.1111/j.1469-8137.2004.01314.x

Gáspár, B., Bossdorf, O., & Durka, W. (2019). Structure, stability and ecological significance of natural epigenetic variation: A large-scale survey in *Plantago lanceolata*. *New Phytologist*, *221*(3), 1585–1596. https://doi.org/10.1111/nph.15487

Gilbert, G. S. (2002). Evolutionary ecology of plant diseases in natural ecosystems. *Annual Review of Phytopathology*, *40*, 13–43. https://doi.org/10.1146/annurev.phyto.40.021202.110417

Herman, J. J., & Sultan, S. E. (2011). Adaptive transgenerational plasticity in plants: Case studies, mechanisms, and implications for natural populations. *Frontiers in Plant Science*, *2*, 102. https://doi.org/10.3389/fpls.2011.00102

Höckerstedt, L., Susi, H., & Laine, A. L. (2020). Data from: Effect of maternal infection on progeny growth and resistance mediated by maternal genotype and nutrient availability. *Dryad Digital Repository*, https://doi.org/10.5061/dryad.0zpc866wm

Holeski, L. M., Jander, G., & Agrawal, A. A. (2012). Transgenerational defense induction and epigenetic inheritance in plants. *Trends in Ecology & Evolution*, *27*(11), 618–626. https://doi.org/10.1016/j.tree.2012.07.011

Holeski, L. M., Zinkgraf, M. S., Couture, J. J., Whitham, T. G., & Lindroth, R. L. (2013). Transgenerational effects of herbivory in a group of long-lived tree species: Maternal damage reduces offspring allocation to resistance traits, but not growth. *Journal of Ecology*, *101*(4), 1062–1073. https://doi.org/10.1111/1365-2745.12110

Hothorn, T., Bretz, F., & Westfall, P. (2008). Simultaneous inference in general parametric models. *Biometrical Journal*, *50*(3), 346–363. https://doi.org/10.1002/bimj.200810425

Humplík, J. F., Lazár, D., Husičková, A., & Spíchal, L. (2015). Automated phenotyping of plant shoots using imaging methods for analysis of plant stress responses – A review. *Plant Methods*, *11*(29). https://doi.org/10.1186/s13007-015-0072-8

Hyvönen, R., Ågren, G. I., Linder, S., Persson, T., Cotrufo, M. F., Ekbland, A., Freeman, M., Grelle, A., Jansses, I. A., Jarvis, P. G., Kellomäki, S., Lindroth, A., Loustau, D., Lundmark, T., Norby, R. J., Oren, R.,

Pilegaard, K., Ryan, M. G., Sigurdsson, B. D., … Wallin, G. (2007). The likely impact of elevated [CO_2], nitrogen deposition, increased temperature and management on carbon sequestration in temperate and boreal forest ecosystems: A literature review. *New Phytologist*, *173*, 463–480.

Jarosz, A. M., Burdon, J. J., & Muller, W. J. (1989). Long-term effects of disease epidemics. *Journal of Applied Ecology*, *26*(2), 725–733. https://doi.org/10.2307/2404096

Jarosz, A. M., & Davelos, A. L. (1995). Effects of disease in wild plant populations and the evolution of pathogen aggressiveness. *New Phytologist*, *129*(3), 371–387. https://doi.org/10.1111/j.1469-8137.1995.tb04308.x

Jousimo, J., Tack, A. J. M., Ovaskainen, O., Mononen, T., Susi, H., Tollenaere, C., & Laine, A.-L. (2014). Ecological and evolutionary effects of fragmentation on infectious disease dynamics. *Science*, *344*(6189), 1289–1293. https://doi.org/10.1126/science.1253621

Kathiria, P., Sidler, C., Golubov, A., Kalischuk, M., Kawchuk, L. M., & Kovalchuk, I. (2010). Tobacco mosaic virus infection results in an increase in recombination frequency and resistance to viral, bacterial, and fungal pathogens in the progeny of infected tobacco plants. *Plant Physiology*, *153*(4), 1859–1870. https://doi.org/10.1104/pp.110.157263

Kuijper, B., & Hoyle, R. B. (2015). When to rely on maternal effects and when on phenotypic plasticity? *Evolution; International Journal of Organic Evolution*, *69*(4), 950–968. https://doi.org/10.1111/evo.12635

Laine, A.-L. (2004). Resistance variation within and among host populations in a plant–pathogen metapopulation: Implications for regional pathogen dynamics. *Journal of Ecology*, *92*(6), 990–1000. https://doi.org/10.1111/j.0022-0477.2004.00925.x

Laine, A.-L. (2006). Evolution of host resistance: Looking for coevolutionary hotspots at small spatial scales. *Proceedings of the Royal Society B: Biological Sciences*, *273*(1584), 267–273. https://doi.org/10.1098/rspb.2005.3303

Laine, A.-I. (2007). Pathogen fitness components and genotypes differ in their sensitivity to nutrient and temperature variation in a wild plant–pathogen association. *Journal of Evolutionary Biology*, *20*(6), 2371–2378. https://doi.org/10.1111/j.1420-9101.2007.01406.x

Laine, A.-L., Burdon, J. J., Dodds, P. N., & Thrall, P. H. (2011). Spatial variation in disease resistance: From molecules to metapopulations. *Journal of Ecology*, *99*(1), 96–112. https://doi.org/10.1111/j.1365-2745.2010.01738.x

Latzel, V., Hájek, T., Klimešová, J., & Gómez, S. (2009). Nutrients and disturbance history in two Plantago species: Maternal effects as a clue for observed dichotomy between resprouting and seeding strategies. *Oikos*, *118*(11), 1669–1678. https://doi.org/10.1111/j.1600-0706.2009.17767.x

Latzel, V., Klimešová, J., Hájek, T., Gómez, S., & Šmilauer, P. (2010). Maternal effects alter progeny's response to disturbance and nutrients in two Plantago species. *Oikos*, *119*(11), 1700–1710. https://doi.org/10.1111/j.1600-0706.2010.18737.x

Lebeda, A., Křístková, E., Kitner, M., Mieslerová, B., Jemelková, M., & Pink, D. A. C. (2014). Wild *Lactuca* species, their genetic diversity, resistance to diseases and pests, and exploitation in lettuce breeding. *European Journal of Plant Pathology*, *138*(3), 597–640. https://doi.org/10.1007/s10658-013-0254-z

Luna, E., Bruce, T. J. A., Roberts, M. R., Flors, V., & Ton, J. (2012). Next-generation systemic acquired resistance. *Plant Physiology*, *158*(2), 844–853. https://doi.org/10.1104/pp.111.187468

Marshall, J. D., & Uller, T. (2007). When is a maternal effect adaptive? *Oikos*, *116*(12), 1957–1963. https://doi.org/10.1111/j.2007.0030-1299.16203.x

Martinez-Aguilar, K., Ramirez-Carrasco, G., Hernandez-Chavez, J. L., Barraza, A., & Alvarez-Venegas, R. (2016). Use of BABA and INA as activators of a primed state in the common bean (Phaseolus vulgaris L.). *Frontiers in Plant Science*, *7*, 653. https://doi.org/10.3389/fpls.2016.00653

Martinez-Medina, A., Flors, V., Heil, M., Mauch-Mani, B., Pieterse, C. M. J., Pozo, M. J., Ton, J., van Dam, N. M., & Conrath, U. (2016). Recognizing plant defense priming. *Trends in Plant Science*, *21*(10), 818–822. https://doi.org/10.1016/j.tplants.2016.07.009

Matson, P. A., Parton, W. J., Power, A. G., & Swift, M. J. (1997). Agricultural intensification and ecosystem properties. *Science*, *277*(5325), 504–509. https://doi.org/10.1126/science.277.5325.504

Mattner, S. W., & Parbery, D. G. (2007). Crown rust affects plant performance and interference ability of Italian ryegrass in the post-epidemic generation. *Grass and Forage Science*, *62*(4), 437–444. https://doi.org/10.1111/j.1365-2494.2007.00598.x

Mauch-Mani, B., Baccelli, I., Luna, E., & Flors, V. (2017). Defense priming: An adaptive part of induced resistance. *Annual Review of Plant Biology*, *68*(1), 485–512. https://doi.org/10.1146/annurev-arplant-042916-041132

Mirman, D. (2014). *Growth curve analysis and visualization using R* (1st ed.). CRC Press.

Mittler, R., & Blumwald, E. (2010). Genetic engineering for modern agriculture: Challenges and perspectives. *Annual Review of Plant Biology*, *61*(1), 443–462. https://doi.org/10.1146/annurev-arplant-042809-112116

Münzbergová, Z., & Hadincová, V. (2017). Transgenerational plasticity as an important mechanism affecting response of clonal species to changing climate. *Ecology and Evolution*, *7*(14), 5236–5247. https://doi.org/10.1002/ece3.3105

Murray, G. M., Ellison, P. J., & Watson, A. (1995). Effects of stripe rust on the wheat plant. *Australasian Plant Pathology*, *24*(4), 261–270. https://doi.org/10.1071/APP9950261

Ovaskainen, O., & Laine, A.-L. (2006). Inferring evolutionary signals from ecological data in a plant-pathogen metapopulation. *Ecology*, *87*(4), 880–891. https://doi.org/10.1890/0012-9658(2006)87[880:iesfed]2.0.co;2

Pavicic, M., Mouhu, K., Wang, F., Bilicka, M., Chovanček, E., & Himanen, K. (2017). Genomic and phenomic screens for flower related RING type ubiquitin E3 ligases in *Arabidopsis*. *Frontiers in Plant Science*, *8*. https://doi.org/10.3389/fpls.2017.00416

Penczykowski, R. M., Walker, E., Soubeyrand, S., & Laine, A.-L. (2015). Linking winter conditions to regional disease dynamics in a wild plant-pathogen metapopulation. *The New Phytologist*, *205*(3), 1142–1152. https://doi.org/10.1111/nph.13145

Piqueras, J. (2001). Infection of *Trientalis europaea* by the systemic smut fungus *Urocystis trientalis*: Disease incidence, transmission and effects on performance of host ramets. *Journal of Ecology*, *87*(6), 995–1004. https://doi.org/10.1046/j.1365-2745.1999.00409.x

R Development Core Team. (2016). *R: A language and environment for statistical computing*. R Foundation for Statistical Computing.

Raghavendra, A. K., & Newcombe, G. (2013). The contribution of foliar endophytes to quantitative resistance to Melampsora rust. *New Phytologist*, *197*(3), 909–918. https://doi.org/10.1111/nph.12066

Reiss, E. R., & Drinkwater, L. E. (2018). Cultivar mixtures: A meta-analysis of the effect of intraspecific diversity on crop yield. *Ecological Applications*, *28*(1), 62–77. https://doi.org/10.1002/eap.1629

Rendina González, A. P., Preite, V., Verhoeven, K. J. F., & Latzel, V. (2018). Transgenerational effects and epigenetic memory in the clonal plant *Trifolium repens*. *Frontiers in Plant Science*, *9*. https://doi.org/10.3389/fpls.2018.01677

Roberts, D. A. (1983). Acquired resistance to tobacco mosaic virus transmitted to the progeny of hypersensitive tobacco. *Virology*, *124*(1), 161–163. https://doi.org/10.1016/0042-6822(83)90299-4

Sagar, G. R., & Harper, J. L. (1964). Biological Flora of the British Isles. *Plantago major* L., *P. media* L. and *P. lanceolata* L. *Journal of Ecology*, *52*, 189–221. Retrieved from https://www.jstor.org/stable/pdf/2257792.pdf

Salvaudon, L., Giraud, T., & Shykoff, J. A. (2008). Genetic diversity in natural populations: A fundamental component of plant–microbe interactions. *Current Opinion in Plant Biology*, *11*(2), 135–143. https://doi.org/10.1016/j.pbi.2008.02.002

Singh, P., & Roberts, M. R. (2015). Keeping it in the family: Transgenerational memories of plant defence. *CAB Reviews: Perspectives in Agriculture, Veterinary Science, Nutrition and Natural Resources*, *10*(26). https://doi.org/10.1079/PAVSNNR201510026

Slaughter, A., Daniel, X., Flors, V., Luna, E., Hohn, B., & Mauch-Mani, B. (2012). Descendants of primed *Arabidopsis* plants exhibit resistance to biotic stress. *Plant Physiology*, *158*(2), 835–843. https://doi.org/10.1104/pp.111.191593

Strange, R. N., & Scott, P. R. (2005). Plant disease: A threat to global food security. *Annual Review of Phytopathology*, *43*, 83–116. https://doi.org/10.1146/annurev.phyto.43.113004.133839

Sultan, S. E. (1996). Phenotypic plasticity for offspring traits in *Polygonum persicaria*. *Ecology*, *77*(6), 1791–1807. https://doi.org/10.2307/2265784

Susi, H., & Laine, A.-L. (2015). The effectiveness and costs of pathogen resistance strategies in a perennial plant. *Journal of Ecology*, *103*(2), 303–315. https://doi.org/10.1111/1365-2745.12373

Suzuki, N., Rivero, R. M., Shulaev, V., Blumwald, E., & Mittler, R. (2014). Abiotic and biotic stress combinations. *The New Phytologist*, *203*(1), 32–43. https://doi.org/10.1111/nph.12797

Tack, A. J. M., & Laine, A.-L. (2014). Spatial eco-evolutionary feedback in plant-pathogen interactions. *European Journal of Plant Pathology*, *138*(3), 667–677. https://doi.org/10.1007/s10658-013-0353-x

Tack, A. J. M., Thrall, P. H., Barrett, L. G., Burdon, J. J., & Laine, A.-L. (2012). Variation in infectivity and aggressiveness in space and time in wild host–pathogen systems: Causes and consequences. *Journal of Evolutionary Biology*, *25*(10), 1918–1936. https://doi.org/10.1111/j.1420-9101.2012.02588.x

terHorst, C., & Lau, J. (2012). Direct and indirect transgenerational effects alter plant-herbivore interactions. *Evolutionary Ecology*, *26*, 1469–1480. https://doi.org/10.1007/s10682-012-9560-8

Tsuge, T., Harimoto, Y., Akimitsu, K., Ohtani, K., Kodama, M., Akagi, Y., & Otani, H. (2013). Host-selective toxins produced by the plant pathogenic fungus *Alternaria alternata*. *FEMS Microbiology Reviews*, *37*(1), 44–66. https://doi.org/10.1111/j.1574-6976.2012.00350.x

Uller, T., Nakagawa, S., & English, S. (2013). Weak evidence for anticipatory parental effects in plants and animals. *Journal of Evolutionary Biology*, *26*(10), 2161–2170. https://doi.org/10.1111/jeb.12212

Van Kal, J. (2006). Licensed to kill: The lifestyle of a necrotrophic plant pathogen. *Trends in Plant Science*, *11*(5), 247–253. https://doi.org/10.1016/j.tplants.2006.03.005

Veresoglou, S., Barto, E., Menexes, G., & Rillig, M. (2013). Fertilization affects severity of disease caused by fungal plant pathogens. *Plant Pathology*, *62*(5), 961–969. https://doi.org/10.1111/ppa.12014

Vivas, M., Zas, R., Sampedro, L., & Solla, A. (2013). Environmental maternal effects mediate the resistance of maritime pine to biotic stress. *PLoS ONE*, *8*(7), e70148. https://doi.org/10.1371/journal.pone.0070148

Vu, W. T., Chang, P. L., Moriuchi, K. S., & Friesen, M. L. (2015). Genetic variation of transgenerational plasticity of offspring germination in response to salinity stress and the seed transcriptome of *Medicago truncatula*. *BMC Evolutionary Biology*, *15*(59). https://doi.org/10.1186/s12862-015-0322-4

Walters, D. R., Ratsep, J., & Havis, N. D. (2013). Controlling crop diseases using induced resistance: Challenges for the future. *Journal of Experimental Botany*, *64*, 1263–1280. https://doi.org/10.1093/jxb/ert026

Yin, J., Zhou, M., Lin, Z., Li, Q. Q., & Zhang, Y.-Y. (2019). Transgenerational effects benefit offspring across diverse environments: A meta-analysis in plants and animals. *Ecology Letters*, *22*(11), 1976–1986. https://doi.org/10.1111/ele.13373

Yoder, O. C. (1980). Toxins in Pathogenesis. *Annual Review of Phytopathology*, *18*(1), 103–129. https://doi.org/10.1146/annurev.py.18.090180.000535

SUPPORTING INFORMATION

Additional supporting information may be found online in the Supporting Information section.

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