Ecology, 83(12), 2002, pp. 3408–3415

HERBIVORY AND MATERNAL EFFECTS: MECHANISMS AND CONSEQUENCES OF TRANSGENERATIONAL INDUCED PLANT RESISTANCE

ANURAG A. AGRAWAL[1]

Department of Botany, 25 Willcocks Street, University of Toronto, Toronto, Ontario M5S 3B2 Canada

Abstract. Many plants induce defenses against herbivores following initial attack. Maternal effects associated with herbivory could mediate induced defenses across generations of plants if damaged plants produce more resistant progeny than undamaged plants. I report that wild radish plants (*Raphanus raphanistrum*) damaged by herbivores (*Pieris rapae*) or treated with a chemical elicitor of induced resistance (jasmonic acid) during the vegetative growth stage, induced resistance of the plants' progeny compared to controls. Conversely, clipping of leaves with scissors, which results in loss of photosynthetic area but not induced resistance, tended to increase susceptibility of progeny plants to *P. rapae* compared to controls. Progeny plants exhibited further induced resistance when damaged as seedlings, although maternally induced plants were less inducible than maternal-control plants. Herbivory in the maternal generation also affected the growth of progeny plants in two ways: (1) seed mass, which was influenced by maternal herbivory, strongly positively correlated with early plant growth; and (2) when seed mass was accounted for, maternal herbivory still had an effect on plant growth, but these effects varied by plant family. Taken together, these results demonstrate that the defensive phenotype of plants is determined, in part, by the maternal environment, and that this allocation can affect the growth trajectories of progeny.

Key words: herbivory and maternal effects; jasmonic acid; Pieris rapae; *plant–insect interactions; transgenerational adaptive plasticity; wild radish* (Raphanus raphanistrum).

INTRODUCTION

Phenotypic plasticity is the ability of single organisms to express different phenotypes in contrasting environments (Agrawal 2001*a*). Maternal effects are a form of phenotypic plasticity that occurs across generations. For example, the environment in the maternal generation can cause the activation of genes that are expressed in the progeny's phenotype (Mousseau and Fox 1998). Plant defenses that are induced within a generation following herbivory have been the subject of much study over the past two decades, and recent work indicates that such plant responses may be adaptive (Agrawal 1998, 1999, 2000*a*, Baldwin 1998, Agrawal et al. 1999*b*). In contrast, plant defenses induced across generations have been little studied (Roberts 1983, Lammerink et al. 1984, Tuzun and Kuc 1987, Shattuck 1993, Agrawal et al. 1999*a*), perhaps in part due to the Lamarckian feel of the hypothesis that progeny of plants may acquire a defensive phenotype from their parents without genetic change. However, biologists have recently recognized that maternal effects are not only common, but may be adaptive if parents respond to information in the environment to improve the fitness of progeny by altering the progeny's phenotype (Mousseau and Fox 1998).

Manuscript received 13 November 2001; revised 1 May 2002; accepted 2 May 2002.

[1] E-mail: agrawal@botany.utoronto.ca

Wild radish (*Raphanus raphanistrum* L. Brassicaceae) is an annual plant that has been used as a model for examining phenotypic plasticity in defense against herbivores (Strauss et al. 1996, Agrawal 1998, 1999, 2000*b*, Agrawal et al. 1999*a*, *b*, Lehtilä and Strauss 1999). Induction of putatively defensive glucosinolates (typically indolyl glucosinolates) following herbivory or jasmonic acid application, has been reported for wild radish and several related *Brassica* species (Bodnaryk 1992, 1994, Agrawal et al. 1999*b*, Agrawal 2000*a*). Glucosinolates and their breakdown products have been studied extensively and have been shown to have negative effects on vertebrates, invertebrates, and microorganisms (Louda and Mole 1992, Giamoustaris and Mithen 1995), including some specialists such as *Pieris rapae* (Stowe 1998). In field experiments, plants exposed to early season herbivory had 60% higher relative fitness than undamaged controls (Agrawal 1998). In the absence of herbivory, the herbivore-induced responses were costly in terms of reduced pollination (Strauss et al. 1996) and reduced production of male reproductive characters in damaged plants compared to controls (Agrawal et al. 1999*b*, Lehtilä and Strauss 1999). Induced responses in wild radish exhibit specificity to the type of damage (mechanical vs. herbivore), and show differential induction following attack by four lepidopteran herbivores (Agrawal 1998, 1999, 2000*b*). Finally, it was demonstrated that folivory on maternal plants caused plant progeny to be more re-

sistant (i.e., 20% reduced caterpillar growth) than progeny from undamaged plants (Agrawal et al. 1999*a*). This transgenerational effect, termed a maternally induced defense, was a novel addition to our understanding of the defensive response repertoire of plants.

In this study I examined the consequences of treating maternal plants with herbivory, a natural elicitor of induced defenses, jasmonic acid, and clipping with scissors to test the hypothesis that maternally induced defenses are activated by the jasmonate signal transduction pathway, and not by the simple stress of leaf tissue loss. Jasmonate is a ubiquitous plant hormone that has been directly implicated as a key signaling molecule in induced resistance of plants to insect herbivores (Thaler et al. 1996, Karban and Baldwin 1997, Staswick and Lehman 1999). Maternally induced plants may produce maximally defended progeny, or progeny may be able to induce even higher levels of resistance. I previously hypothesized that greater induction in damaged progeny from damaged mothers compared to damaged progeny from undamaged mothers may serve as a mechanism to overcome developmental constraints early in an organism's life, especially since this stage may be particularly vulnerable to predation or herbivory (Agrawal et al. 1999*a*). Thus, in this study I also address the consequences of maternal environment treatments for subsequent inducibility in response to herbivory in the progeny generation and for progeny chemistry and growth.

Materials and Methods

Grandmaternal and maternal generation

I started these experiments with *R. raphanistrum* seeds from a second (grandmaternal) generation of untreated, greenhouse-grown plants in order to equalize maternal environment effects between families. From 17 grandmaternal families, I collected ~10 seeds each (full- or half-sibs, maternal generation). These maternal generation plants were grown in a greenhouse in 0.5-L pots in Sunshine Soil Mix no. 1 (Sun Grow Horticulture, Bellevue, Washington). Natural light was augmented with sodium vapor lights on a 16:8 light : dark cycle. Pots were randomly placed in trays and were watered from below using automated emitters. At germination, each plant received one dose of 0.3 g of 17:9:13 N:P:K Osmocote slow release micro-fertilizer (Scotts-Sierra, Marysville, Ohio). To minimize the effects of temperature and light heterogeneity on plants within the greenhouse, each tray was moved (both within and among rows) every other day for the duration of the experiment. At the four leaf stage of the maternal generation, each plant was randomly assigned to one of four treatments (3–5 plants per treatment per family): (1) unmanipulated control, (2) 50% of each leaf consumed by a caged *Pieris rapae* larva, a natural herbivore of *R. raphanistrum*, (3) sprayed to run-off with 0.5 mmol/L jasmonic acid (~1 mL solution, or 0.1 mg of jasmonic acid, was delivered to each plant), and (4) 50% of each leaf clipped and removed using scissors. We used this jasmonic acid concentration because it mimicked induction caused by real herbivory in this and other systems (Thaler et al. 1996, Agrawal et al. 1999*b*). The clipping treatment followed the phenology and damage pattern of the *P. rapae* treatment. Thus, the experimental design was a 2×2 factorial with the main fixed effects being with or without induced responses and with or without leaf tissue removal. Clipped plants were equivalent to controls and jasmonate treated plants were equivalent to caterpillar damaged plants as measured by glucosinolate responses (Agrawal et al. 1999*b*). Each of the 280 treated plants was considered a replicate in this study.

The caterpillar herbivory and clipping treatments were maintained throughout the growth of the plants (4–6 wk of additional foliar growth), while the jasmonic acid treatment was applied once, ~2 wk before flowering commenced. To allow seed set, plants were hand pollinated every other day using a makeup brush wherein a brush loaded with pollen collected from many haphazardly chosen plants across all treatments was dabbed across every open flower. Seeds were collected and counted upon plant senescence (Agrawal et al. 1999*b*).

Does jasmonate activate maternally induced resistance?

Experiment 1.—I tested the hypothesis that activation of the jasmonic acid pathway was sufficient to cause maternally induced resistance. This experiment was conducted ~5 yr after the maternal generation treatments were imposed and seeds were collected. The seeds were stored in paper bags in a cool dry dark cabinet. I employed all of the treatments described above and conducted two trials of the experiment: the first trial employed 3–5 maternal families from each of 10 of the grandmaternal families (259 plants) and the second trial employed 3–5 maternal families from all 17 grandmaternal families (218 plants). Sample sizes were nearly equal for the treatment by family combinations. Seeds were extracted from pods using pliers and weighed; I then planted two seeds from each maternal plant. Seed mass was used as a covariate in all analyses because allocation to seed mass is typically considered one of the potentially important maternal effects in plants including wild radish (Stanton 1984*a*, Mazer 1987, Roach and Wulff 1987). Seeds were planted in 210 mL pots in Sunshine Soil Mix no. 1 with ~0.25 g of 13:13:13 N:P:K Nutricote slow release fertilizer (McCalif Grower Supplies, Ceres, California) in a greenhouse with augmented sodium vapor lights on a 16:8 light : dark cycle.

Five days after planting, most seedlings had emerged (~90% germination) and had one true leaf beginning to expand. At this stage, seedlings were completely randomized and a freshly hatched neonate *P. rapae*

larva was introduced to each seedling. Because *P. rapae* is a sluggish caterpillar that usually does not leave an acceptable host plant (*personal observations*), no cages were required to confine caterpillars to plants. After 4 d (trial 1) and 4.5 d (trial 2) I collected, froze, and weighed the larvae. Effects of grandmaternal family (random effect), maternal family (random effect), leaf removal (fixed effect), induced response (fixed effect), seed mass (covariate), and trial (random effect) on caterpillar growth were analyzed using ANOVA. Caterpillar masses were natural log-transformed to reduce heteroscedasticity. Maternal family was nested in the three-way interaction between leaf removal, induced responses, and grandmaternal family because maternal plants came from one combination of these three factors. All interactions were included in the statistical model with the following exception: interactions between trial and other terms were not included because trial was an arbitrary blocking factor included simply to correct for variance associated with the different trials.

All analyses were conducted using PROC MIXED in SAS (SAS Institute 1999). Degrees of freedom for F tests of fixed effects were estimated using the Satterthwaite approximation (SAS Institute 1999). As suggested by the SAS Institute (1999), the likelihood ratio χ^2 test was employed for tests of the random effects. The likelihood ratio χ^2 tests the hypothesis that the variation due to the random effect is >0, and is a one-sided single degree of freedom test.

To test for changes in the chemistry of undamaged seedlings among the four treatments on the maternal generation, I examined their glucosinolate profiles. I collected undamaged seedlings (cotyledons and first true leaves) from each of the four treatment groups of the 17 grandmaternal families. However, because each sample of the chemical analysis required at least 100 mg of dried plant tissue, I pooled seedlings from several families for each replicate ($n = 6$–8 per treatment, $\sim$14 seedlings pooled for each sample). Nevertheless, each of the 50 chemical analyses is an independent estimate of maternal treatment because replicate samples did not contain seedlings from the same treated maternal plant. The analytical procedure was modified from published protocols (Brown and Morra 1995, Agrawal et al. 1999*b*). I classified glucosinolates into indolyl, aliphatic, and aromatic glucosinolates using predetermined high-pressure liquid chromatography retention times and used two-way MANOVA with leaf removal (fixed effect) and induced response (fixed effect) as main effects in the analysis of concentrations of the three classes of glucosinolates.

Are maternally and progeny induced resistance additive?

Experiment 2.—I tested whether maternally induced resistance affected the ability of progeny to induce resistance. I employed 3–5 maternal families from each of the 17 grandmaternal families for this experiment (427 plants) in an identical planting design to experiment 1. Five days after planting, one plant from each grandmaternal family by leaf removal by induced response treatment combination was inoculated with a neonate *P. rapae* larva. These plants were the same as those used in experiment 1, trial 2. In addition, an undamaged control plant from each treatment combination was randomized within the array of plants. After 5 d of feeding, caterpillars were removed and a single new neonate *P. rapae* larva was introduced to all plants. At this stage, half of the plants had received herbivory while the other half had not. Many of the herbivore damaged plants had most of their first true leaf consumed, while undamaged plants had a full leaf and second expanding true leaf.

The caterpillars were removed after 4 d of feeding, frozen, and then weighed as a bioassay for induced resistance. Caterpillar mass was natural log-transformed to reduce heteroscedasticity. Effects of grandmaternal family (random effect), maternal leaf removal (fixed effect), maternal induced response (fixed effect), seedling herbivory (fixed effect), and seed mass (covariate) on caterpillar growth were analyzed using ANOVA. All interaction terms were included.

Experiment 3.—As in experiment 2, this experiment tested for the effects of maternal environment on induced resistance in progeny. I used 80 maternal families from 10 of the grandmaternal families, chosen to span the range of effects of herbivory on seed mass. Previous experiments showed that there was strong variation among grandmaternal families for effects of herbivory on individual seed mass, with some families increasing and other families decreasing seed mass compared to controls (Agrawal 2001*b*). A total of 157 plants from two treatments, with and without maternal herbivory, were grown in a completely randomized design as in experiment 1. At the cotyledon/first true leaf stage I introduced a single neonate *P. rapae* larva to half of the plants in each treatment combination. After 4 d of feeding, the caterpillar was removed. The plants were then allowed to grow for 5 d until they had produced 2–3 true leaves. At this stage, a fresh neonate *P. rapae* larva was introduced to each plant. After 4 d of feeding and growth the caterpillar was removed, frozen, and weighed. In addition, I examined the first true leaf for area, trichome number, and trichome density (Scion Image, Scion Corporation, Frederick, Maryland). Here I report only the effects on trichome density because this is likely a relevant measure of resistance (Agren and Schemske 1993). In previous experiments using wild radish I have shown that the effects on total trichome number are consistent with the effects on trichome density (Agrawal 1999).

In this experiment I examined two progeny seedlings from each of the maternally treated plants. Thus, maternal plant was nested in the two-way interaction between grandmaternal family and maternal herbivory

TABLE 1. The effects of maternal treatments on the growth of caterpillars on progeny plants analyzed using SAS PROC MIXED.

Source	df	*F*	χ^2†	*P*
Leaf removal [L]	1, 185	3.49		0.0635
Induced response [I]	1, 188	7.27		0.0077
L × I	1, 192	0.68		0.4106
Seed mass	1, 385	15.24		0.0001
Grandmaternal family [G]	1		0.2	0.3274
G × L	1		54.6	<0.0001
G × I	1		1.2	0.1367
G × L × I	1		0	0.5000
Maternal family(G × L × I)	1		0	0.5000
Trial	1		0	0.5000

Note: Letters in brackets represent shorthand definitions of the factor and parentheses represent nesting.

† Likelihood-ratio χ^2 test for random effects.

treatment. Effects of grandmaternal family (fixed effect), maternal family (random effect), maternal herbivory (fixed effect), progeny herbivory (fixed effect), and seed mass (covariate) on caterpillar mass and trichome density were analyzed using ANOVA. All interaction terms were included.

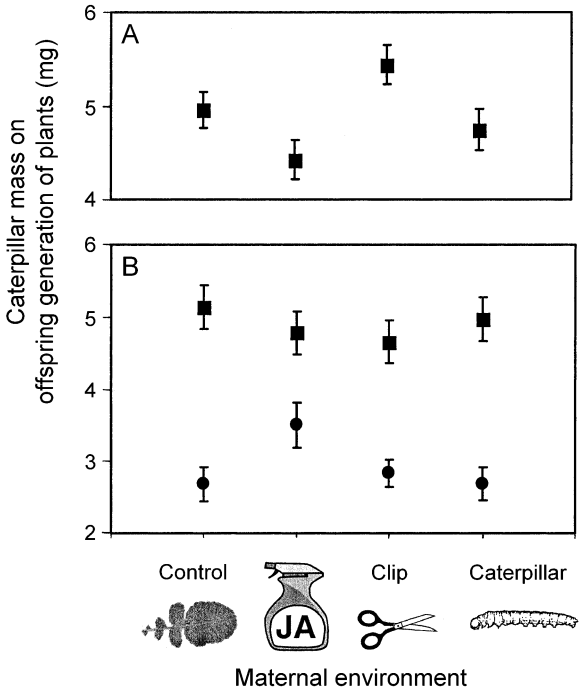

FIG. 1. Growth of *P. rapae* caterpillars (mean ± 1 SE): the maternal parent of these plants was untreated (control), treated with jasmonic acid to stimulate induced responses in the absence of leaf tissue removal (JA), clipped with scissors to remove 50% of each leaf without the associated induced responses (clip), or eaten by caterpillars to remove 50% of each leaf, combining leaf tissue removal and induced responses. (A) The pooled means of two trials where caterpillars were grown on seedling plants. (B) Squares represent caterpillars growing on undamaged plants at rosette stage (two true leaves), and circles represent caterpillars growing on plants at the same stage that were damaged as seedlings.

Does maternal induction affect progeny growth?

Experiment 4.—This experiment tested for the effects of maternal herbivory on progeny growth. As in experiment 3, I used 80 maternal families from 10 of the grandmaternal families, chosen to span the range of effects of herbivory on seed mass. A total of 98 plants from two treatments, with and without maternal herbivory, were grown up in a completely randomized design. When the plants reached the 3–4 true leaf stage (15 d after planting the seeds) I harvested the aboveground biomass of the plants. Plants were freeze-dried and then weighed. Early seedling growth was measured because maternal effects are generally stronger in the early life stages.

As in experiment 3, I examined two progeny seedlings for many of the maternally treated plants. Thus maternal plant was nested in the two-way interaction between grandmaternal family and maternal herbivory treatment. Effects of grandmaternal family (fixed effect), maternal family (random effect), maternal herbivory (fixed effect), and seed mass (covariate) on plant mass were analyzed using ANOVA. All interaction terms were included.

RESULTS AND DISCUSSION

Does jasmonate activate maternally induced resistance?

Induced resistance and leaf tissue removal each had an effect on the resistance of progeny plants (Table 1, Fig. 1A). Treatment of maternal plants with jasmonate decreased caterpillar growth by 11% and leaf tissue removal by scissors increased caterpillar growth by 10% (marginally significant), compared to controls. Seed mass was positively correlated with caterpillar growth, although this was a weak association, explaining 6% of the variance in caterpillar mass. These results were consistent over two experimental trials, each with >200 experimental plants (Table 1).

My results have two major implications. First, activation of the jasmonate pathway via application of jasmonic acid is sufficient to cause maternally induced

resistance. In this experiment, plants were only treated once with jasmonic acid, several days before plants bolted and commenced flowering. Thus, the maternally inducing signal need not be present during floral or fruit development. In transgenerational induced responses of water fleas, *D. cucullata*, chemical cues of the predators are also sufficient to cause maternally induced progeny, even in unformed eggs (Agrawal et al. 1999*a*). Second, leaf tissue removal in the absence of insect-specific inducing signals (i.e., caterpillar saliva, jasmonates) resulted in increased susceptibility. Why are the progeny of clipped plants more susceptible to caterpillars than controls? One hypothesis is that the 50% leaf loss of clipped plants weakens plants, alters allocation patterns, and the lack of an inducing signal prevents allocation to defense in progeny. Although light and nutrient stressed plants are quite capable of inducing responses to herbivores (Zangerl and Berenbaum 1994/1995, Stout et al. 1998), in the absence of a herbivory signal these plants may be more susceptible to herbivores. Seedlings from the four maternal treatments did not vary in their concentrations of the three glucosinolate groups (induced response, Wilks' lamba = 0.994, $F_{3,22} = 0.046$, $P = 0.987$; leaf removal, Wilks' lamba = 0.828, $F_{3,22} = 1.523$, $P = 0.236$; interaction, Wilks' lamba = 0.879, $F_{3,22} = 0.010$, $P = 0.407$; data not shown). My previous work indicated that seeds from maternally damaged plants had shifted glucosinolate concentrations (reduced indolyl and increased hydroxylated glucosinolates) compared to controls, but they did not differ in nitrogen or carbon composition (Agrawal et al. 1999*a*). Thus, the mechanism of maternally induced resistance in the progeny of wild radish remains elusive.

Are maternally and progeny induced resistance additive?

In this experiment I assayed caterpillar growth on progeny from the four maternal treatments. The first result from this experiment was that damaged seedlings induced resistance across maternal treatments to reduce caterpillar growth by 40% ($F_{1,17} = 80.37$, $P < 0.0001$, compare squares to circles in Fig. 1B). Second, although control plants no longer showed strong maternally induced resistance at this stage of development, I observed a three-way interaction between maternal induction, maternal leaf tissue removal, and progeny induction ($F_{1,385} = 5.30$, $P < 0.0218$, Fig. 1B). Although this interaction is difficult to interpret, it appears to result from maternally jasmonate-treated plants showing weaker induced resistance than other treatments in the progeny generations (Fig. 1B). This effect is likely a result of maternally jasmonate-treated plants receiving less herbivory during the induction period as a seedling, thus reducing the within-generation induced resistance. None of the other 16 main effects or interactions approached significance in this analysis (table not shown). Thus, the key result from this experiment

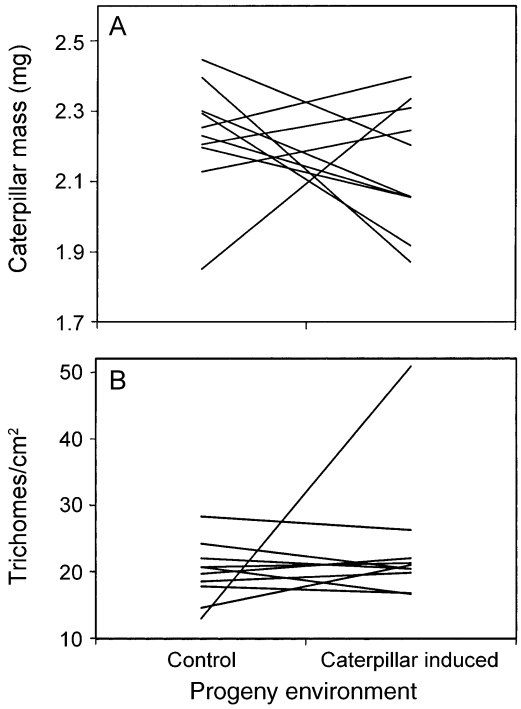

FIG. 2. (A) Effects of seedling herbivory on resistance of plants and (B) trichome density on first true leaves of 10 families of wild radish. In these norm-of-reaction plots each line represents a grandmaternal family with the mean of 3–5 maternal plants in each environment.

is that maternally induced resistance does not strongly influence inducibility of the progeny. Although maternal induction protects plants very early in the development of progeny (cotyledons and first true leaves), after this period within-generation induced resistance is of greater importance.

In the second experiment to test for effects of maternal herbivory on induction in the progeny (experiment 3) I again found no evidence that maternal herbivory influenced the ability of progeny to induce resistance (data not shown, all P values $\gg 0.1$). These plants were at the 3–4 true leaf stage, and it is likely that much of the maternal effects had dissipated by this time. As in experiment two, there were no family effects or interactions between maternal environment and family. The only effect in this experiment approaching significance was a grandmaternal family by progeny herbivory (induction) interaction for effects on caterpillar growth (Fig. 2A, $F_{9,177} = 1.85$, $P = 0.0666$ for the full model, $P = 0.0540$ for the model with the nonsignificant covariate removed). I also measured the density of trichomes on leaves of another set of plants and again found that the only significant effect was the grandmaternal family by progeny herbivory interaction (Fig. 2B, $F_{9,63.8} = 2.34$, $P = 0.0240$). The results from both progeny induction experiments provide suggestive evidence for genetic variation for induced resistance within a generation of wild radish (although primarily

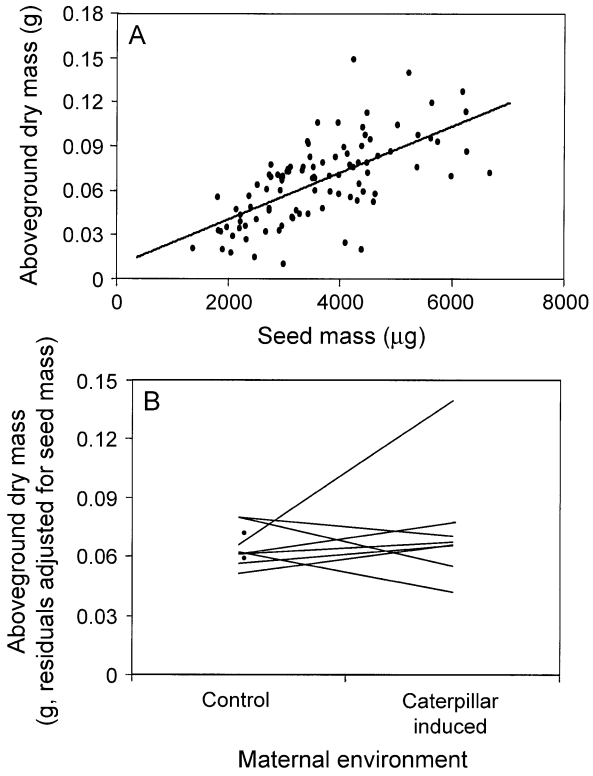

FIG. 3. (A) The relationship between seed mass and early plant growth. (B) Effects of herbivory on maternal parents on growth of progeny when the effect of seed mass is removed. In this norm-of-reaction plot each line represents a grandmaternal family with the mean of several maternal plants in each environment. Although 10 grandmaternal families were represented in this experiment, two of these families (single dots) did not have representatives in both environments.

caused by one family) and emphasize that maternally induced resistance is a short lived response in progeny plants. Consistent with theory, my data show that maternally induced resistance relaxes in the face of low herbivore pressure. In other words, damaged plants produce progeny that are maternally induced and this induction is likely to protect seedling plants. However, if the maternal environment is not reflected in the early progeny environment, the plant relaxes the maternally induced resistance and relies more heavily on within-generation induced resistance if it is required.

Does maternal induction affect progeny growth?

Individual seed mass is strongly influenced by maternal herbivory depending on the plant family: in a companion study (Agrawal 2001*b*) I reported that in some of the same 17 grandmaternal families used in this experiment, maternal herbivory reduced individual seed mass by as much as 50%. Other families showed no effect of maternal herbivory on individual seed mass, and some families showed an equal positive effect, with maternal herbivory increasing seed mass by up to 50% (Agrawal 2001*b*). Accordingly, maternal herbivory can influence seed mass, which was shown to influence emergence in field plots (Agrawal 2001*b*) and is likely to influence progeny growth. Indeed, I found a positive correlation ($r^2 = 0.43$, Fig. 3A, Table 2) between seed mass and progeny growth. However, progeny growth was also apparently influenced by grandmaternal family, and its interaction with maternal herbivory, irrespective of seed mass (Fig. 3B, Table 2). Thus, when the effect of seed mass is removed from the data, there is a residual effect of maternal herbivory on progeny growth, and the magnitude and direction of this effect is dependent on grandmaternal family. Although it is unclear why maternal herbivory influences progeny growth, especially given the benign conditions of the greenhouse, this result strongly indicates that the maternal environment may have profound consequences for development, growth, and defense in progeny.

Ecology and evolution of maternally induced resistance

Other studies have suggested the possibility of maternally induced resistance in plants attacked by aphids and pathogens (Roberts 1983, Lammerink et al. 1984, Shattuck 1993), and vertebrate and invertebrate animals threatened by predators (Gilbert 1966, Brambell 1970, Agrawal et al. 1999*a*, Shine and Downes 1999, Buechler et al. 2002). Like all forms of phenotypic plasticity, maternally induced plant resistance will only be effective if the response to herbivore environment in one generation protects the seedling in the herbivore environment of the next generation (Karban et al. 1999). A plant that employs maternally induced resistance but whose progeny are not subject to attack may suffer energetic and ecological costs without benefits (Agrawal 2001*b*). Few data exist on the year-to-year variation in *P. rapae* numbers. However, if the herbivore environment is essentially constant over time, I expect that fixed or constitutive resistance will evolve. The persistence of maternally induced resistance suggests variability in herbivory through time.

Consistent with mathematical theory (Jablonka et al. 1995), in a scenario where any two consecutive gen-

TABLE 2. Mixed-model analysis for effects of herbivory on maternal plants on growth (dry aboveground biomass at the 3–4 leaf stage) of progeny plants using SAS PROC MIXED.

Source	df	*F*	χ^2†	*P*
Caterpillar herbivory [C]	1, 79	2.30		0.1330
Grandmaternal family [G]	1, 79	1.99		0.0515
C × G	1, 79	2.86		0.0103
Seed mass	1, 79	66.96		<0.0001
Maternal family(C × G)	1		0.0	0.5000

Note: Letters in brackets represent shorthand definitions of the factor, and parentheses represent nesting.

† Likelihood-ratio χ^2 test for random effects.

erations have similar levels of attack, although across many generations there are variable levels of herbivory, natural selection may favor maternally induced resistance. The fact that maternally induced resistance rapidly declines in wild radish seedlings that are not attacked by herbivores further supports the view that maternal determination of the progeny's phenotype is a reversible ''guess'' at the progeny's environment. Resistance at the seedling stage may be critical for fitness (Stanton 1984*b*), and thus maternal influences at this stage should be advantageous. However, the relaxation of maternally induced resistance should reduce potential costs associated with induction and defers the defensive needs of the plant to within-generation strategies.

The generality of transgenerational induced resistance may also depend, in part, on the mode of action of maternally induced resistance and species-specific seed sizes. Virus infected *Brassica* plants also produced seeds with shifted glucosinolate profiles, in addition to seeds with altered concentrations of total and soluble nitrogen (Shattuck 1993). Given that seed chemistry determines the glucosinolate profile of seedlings, and in some cases, the resistance of seedlings to herbivores (Glen et al. 1990), maternally induced resistance may simply lie in maternal allocations to the seed. If maternally induced resistance is based on allocation of resources, defense chemicals, or hormones in the seed tissue, then I predict that large-seeded species will be more likely to show maternally induced resistance than relatively small-seeded species. However, if maternally induced resistance is activated by the expression of genes in progeny tissue, I predict that seed size will be independent of maternally induced resistance.

Speculation on maternally induced resistance and agricultural crops

Are seedlings of agricultural crops more susceptible to herbivores because of benign conditions during seed development in the maternal plant? Conventional wisdom has been that favorable growing conditions will yield the largest, most vigorous progeny. However, if maternally induced responses are a general phenomena, and plants grown for seed are typically protected from pests using pesticides, then the seeds being produced for agriculture may be particularly susceptible. For example, broccoli and other *Brassica* crops that are closely related to wild radish have seedlings that are severely attacked by caterpillars, other herbivores, and pathogens (UC-IPM 1992). These plants may be better able to cope with seedling herbivores if they are maternally induced. In particular, low to intermediate levels of herbivory on maternal plants, below thresholds of yield loss, may result in the most vigorous seeds and seedlings because they are resistant to pests. Although detailed studies are required, manipulation of the maternal environment may be a valuable tool in producing more resistant crop seedlings.

Acknowledgments

I thank Sharon Strauss, Mike Stout, and Joel Kniskern for stimulating the maternal portion of this work and Marc Johnson for helping with the experiments conducted on the progeny. Matt Morra and Vladamir Borek (University of Idaho) conducted the chemical analyses of foliar glucosinolates. The manuscript was improved by the comments of Matt Aryes, Marc Johnson, Nile Kurashige, Ken Raffa, Jennifer Thaler, Danush Viswanathan, Pete Van Zandt, and an anonymous reviewer. My research (http://www.botany.utoronto.ca/researchlabs/agrawallab/index.stm) is supported by the Botany Department at the University of Toronto, an operating grant from the Natural Sciences and Engineering Research Council of Canada, Connaught grants (University of Toronto), the Canadian Foundation for Innovation, and a Premier's Research Excellence Award (Ontario).

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