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Review

Completing the cycle: maternal effects as the missing link in plant life histories

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Maternal effects on seed traits such as germination are important components of the life histories of plants because they represent the pathway from adult to offspring: the pathway that completes the life cycle. Maternal environmental effects on germination influence basic life-history expression, natural selection on germination, the expression of genetic variation for germination and even the genes involved in germination. Maternal effects on seed traits can even influence generation time and projected population growth rates. Whether these maternal environmental effects are imposed by the maternal genotype, the endosperm genotype or the embryonic genotype, however, is as yet unknown. Patterns of gene expression and protein synthesis in seeds indicate that the maternal genotype has the opportunity to influence its progeny's germination behaviour. Investigation of the phenotypic consequences of maternal environmental effects, regardless of its genetic determination, is relevant for understanding the variation in plant life cycles. Distinguishing the genotype(s) that control them is relevant for predicting the evolutionary trajectories and patterns of selection on progeny phenotypes and the genes underlying them.

Keywords: demography; flowering time; germination; life cycle; phenology; phytochrome

1. INTRODUCTION

A life cycle is a sequence of developmental events in which one life stage precedes another. In that sense, what happens later is contingent on what happened earlier: growth rate, for example, may depend on how big an egg or seed the hatchling or seedling came from. Notably, this temporal sequence of development occurs within a temporal sequence of seasons. Because seasons are not completely predictable, adaptive life histories frequently require a continuous assessment of seasonal changes and appropriate developmental responses to those seasonal changes. That is, adaptive life histories require matching the appropriate life stage with the appropriate season. In this sense, phenotypic plasticity—or developmental responses to external environments, and especially seasonal environments—becomes important for understanding adaptive life histories of organisms in nature—plants and animals alike.

Maternal effects are a particular kind of phenotypic plasticity that encompasses the developmental contingency of later life stages upon earlier stages. In the case of maternal effects, progeny phenotypes are altered as a function of the environment created by or experienced by the maternal parent. 'Maternal environmental effects' refer to the particular phenomenon in which the external ecological environment of the maternal parent influences the phenotype of its progeny.

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The influence of parental environments on offspring phenotypes is frequently a neglected pathway within organismal life histories. However, this pathway completes the life cycle, linking one generation to the next. Without considering that pathway, one cannot predict the critical first step in life histories: that of hatching out (or being born) in many animals and germination in plants. In plants, this first transition from seed to seedling is especially important, because the seasonal timing of germination determines the seasonal environment experienced by subsequent life stages of sessile plants throughout their lives (Donohue 2005). The same is true to varying degrees for many animals in regards to hatching phenology.

This paper discusses the influence of maternal effects on plant life cycles, focusing on maternal environmental effects on germination of the annual plant *Arabidopsis thaliana*. Maternal effects on germination influence the basic life cycle that is expressed, and empirical data show that they influence the overall phenology, projected population performance and even the genetic basis of life stage transitions.

2. MATERNAL EFFECTS AS THE MISSING LINK IN PLANT LIFE CYCLES

Annual and biennial plants exhibit variation in the basic life history they express. This variation can be attributed to the variation in two interacting phenological traits: germination timing and timing of reproduction (Chouard 1960; Venable 1984; Kalisz 1986; Masuda & Washitani 1990, 1992; Kalisz & Wardle 1994; Nordborg & Bergelson 1999). The *winter annual*

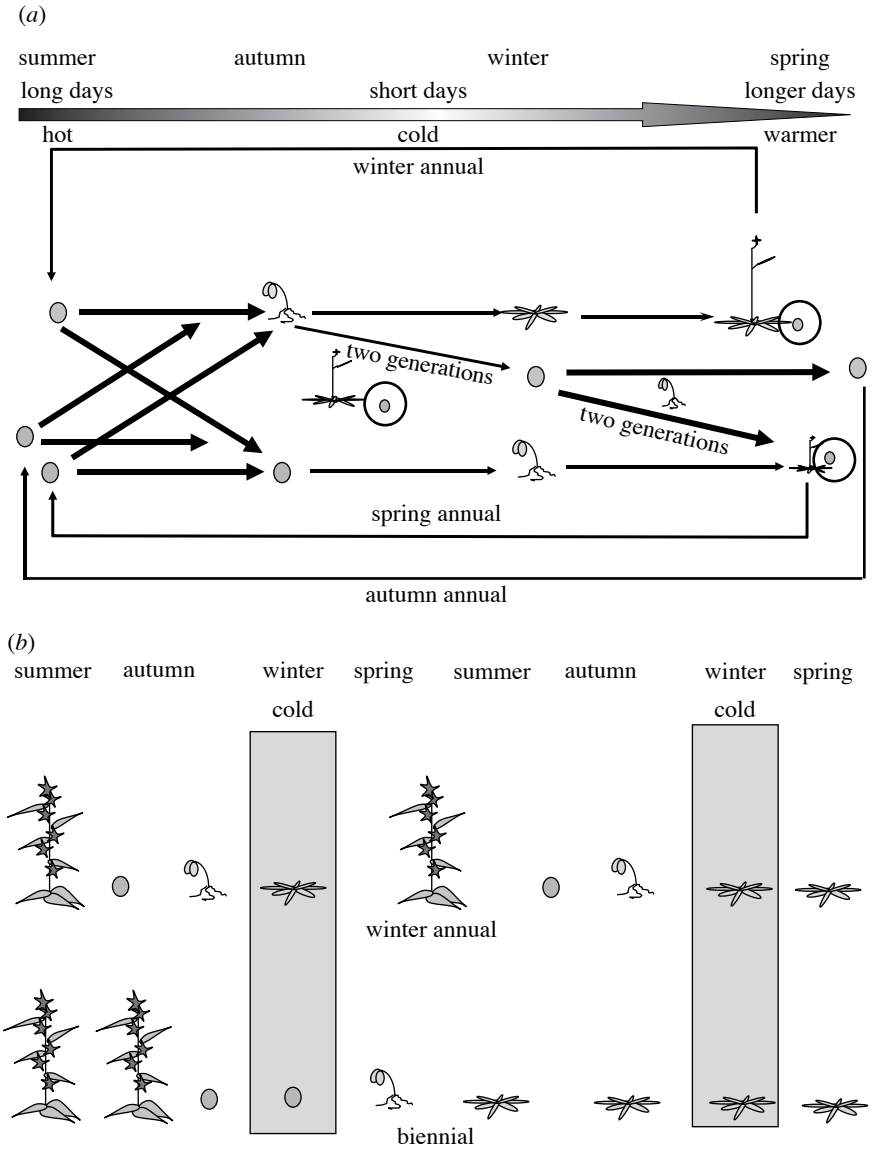

Figure 1. (*a*) Life cycles of annual plants. Bold lines indicate branch points determined by the germination behaviour of seeds (circled) that matured during different seasons. The pathway from flower to seed represents the maternal effects on germination, and this path also completes the life cycle. The life cycle that is expressed depends on which path the seeds follow, which can be a function of when the seeds were matured and dispersed. (*b*) Life cycles of biennial plants. Germination timing determines whether the seeds or rosettes experience cold (shaded). This, in turn, determines whether the plants flower the following season and express an annual life cycle as opposed to a biennial life cycle.

life history occurs when seeds germinate in autumn, seedlings or subadult plants overwinter and then flowering, seed set and dispersal occur during spring or early summer (figure 1*a*). The seeds of winter annuals that matured in spring and summer do not germinate until autumn, when the cycle begins again. By contrast, the *summer or spring annual* life history occurs when the seeds germinate in spring, seedlings mature into adult plants that same spring or summer and flowering, seed set and dispersal occur in spring or summer. The seeds of summer annuals overwinter and do not germinate until the following spring. Autumn flowering also occurs in many annuals. If autumn-matured seeds remain dormant and do not germinate until the next autumn, then an *autumn annual life history* would result. If those seeds are capable of germinating in springtime, however, then a spring annual generation could occur; these spring-flowering plants could set seeds in spring or summer, which could, in turn, germinate in autumn, resulting in a *rapid-cycling* or *'bivoltine' life history* with two (or more) generations per year instead of one. Thus, annual plant life histories can vary in the seasonal timing of the critical transitions of germination and reproduction, which can lead to variation in generation time.

Many common annual and biennial plants exhibit such phenological variation (e.g. *Lepidium*: Roberts & Boddrell 1983; *Capsella*: Neuffer & Hurka 1988, Neuffer 1990, Neuffer & Koch 1996; *Sisymbrium*: Bouwmeester & Karssen 1993; *Berteroa*: Brandes & Schrei 1997; *Arabidopsis*: see below). In several of these species, one can easily see new germinants in both autumn and spring, as well as flowers and maturing seeds in spring through to autumn. How this variation translates into variation of generation time and its accompanying life cycles is seldom known, however. Which life

cycle is actually expressed by any given seed depends critically on when that seed germinates, and especially whether it germinates in the autumn or the spring.

The season of seed germination influences adult phenology by determining the seasonal conditions experienced by seedlings and all subsequent vegetative stages. In particular, the season of seed germination determines the photoperiod and temperature experienced by seedlings and young rosettes—especially the probability of experiencing a prolonged winter. Both photoperiod and temperature (and especially prolonged cold) are well-known seasonal cues that regulate flowering. Many plants flower only after a prolonged cold or 'vernalization' period, and the vernalization pathway of flowering is one of the best-characterized genetic pathways in plants (Coupland 1995; Simpson & Dean 2002). Whether or not a plant receives vernalization before summer drought conditions and activates this flowering pathway depends on the season in which it germinated: autumn germinants would receive it, while spring germinants would not. Plants that do not need vernalization, however, may flower even if they germinate in autumn. Moreover, plants that do not receive vernalization may still be able to flower if they receive another seasonal cue, such as a long-day photoperiod (Lee & Amasino 1995). Spring annuals use this photoperiod pathway for flowering. Which pathway is most important for enabling flowering therefore depends on the season of seed germination. In short, both the life cycle that is expressed by annual plants and the genetic pathways that are required for flowering depend on the season of seed germination.

Likewise, the timing of germination may even determine whether a plant exhibits an annual or a biennial life cycle (figure 1*b*). In *Campanula americana*, seeds that are dispersed and germinate in late summer or early autumn receive cold vernalization and flower in spring, thereby exhibiting a winter annual life history (Galloway 2001*a*,*b*). By contrast, plants that flower and disperse seeds in late autumn do not germinate until spring. Those seedlings do not receive vernalization and consequently do not flower until the following spring, after they have experienced winter, and they consequently exhibit a biennial life history. In this case, the timing of germination determined whether an annual or biennial life history was expressed.

Equally important, but far less studied than the influence of germination phenology on flowering time via seasonal cues, is the influence of flowering phenology on germination. Since flowering time is a property of the maternal parent, the influence of flowering phenology on progeny germination can be considered a maternal effect. In the above example of *C. americana*, flowering time influenced whether the seeds germinated in autumn or spring, most likely through a combination of effects of pre- and post-dispersal seasonal conditions, and these maternal effects were even shown to be adaptive (Galloway & Etterson 2007).

Maternal effects on germination are ubiquitous, and substantial maternal control of dormancy and germination occurs in many species (reviewed in Roach & Wulff 1987; Fenner 1991; Lacey 1991; Gutterman 1992; Wulff *et al.* 1994; Schmitt 1995; Wulff 1995; Baskin & Baskin 1998; Donohue & Schmitt 1998). Maternal effects in plants, broadly defined, include (i) the maternal genetic effects caused by maternal inheritance of plastids, (ii) the effects of endosperm, which is triploid, with two-thirds of its genotype of maternal origin, (iii) the effects of the seed coat, which is maternal tissue, (iv) the effects of maternal provisioning during seed development, with nutrient resources, hormones, proteins and transcripts, all capable of being provisioned to seeds by the maternal parent, and (v) the maternal determination of the progeny environment via dispersal (similar to oviposition site preference in animals) or phenology. Germination is the result of complex interactions between the progeny and the maternal genotypes. The seed coat or testa, which comprises the maternal genotype, imposes mechanical constraints to germination in many species and can alter permeability and light exposure, which are well-known cues for germination (e.g. Dobrovolna & Cetl 1966; Goto 1982; Biere 1991; Platenkamp & Shaw 1993; Léon-Kloosterziel *et al.* 1994; Botto *et al.* 1995, reviewed in Baskin & Baskin 1998). Maternal provisioning of resources and hormones during seed development, moreover, can influence seed metabolism and gene expression during development and even after dispersal (e.g. Karssen *et al.* 1983, reviewed in Finch-Savage & Leubner-Metzger 2006; Holdsworth *et al.* 2008). Seed coat and provisioning effects commonly exhibit phenotypic plasticity in response to environmental conditions during seed development. As such, these maternal effects, along with maternal determination of progeny environments, are manifest as maternal environmental effects. It is through these maternal environmental effects that maternal flowering phenology can influence progeny germination phenology.

Perhaps the most direct effect of flowering phenology on germination is that flowering time determines the immediate post-dispersal seasonal conditions experienced directly by seeds. For example, seeds matured in summer will experience hot conditions immediately after dispersal, whereas those dispersed in autumn will experience cool or cold conditions immediately after dispersal. Post-dispersal seasonal conditions strongly influence germination behaviour (reviewed in Bewley 1997*b*; Baskin & Baskin 1998). A short cold spell breaks dormancy in many species, enabling them to germinate in autumn. Other species require prolonged cold to break dormancy, such that only seeds that have overwintered can germinate. Such seeds that require prolonged cold will not germinate unless they are shed before winter; seeds that are shed during spring will not complete a generation that same year. Likewise, warm temperatures can induce secondary dormancy or inhibit germination in a wide variety of species, whereas other species require warm conditions to break dormancy. Such seeds that require warm temperatures to break dormancy would not be able to germinate until after they have experienced summer, preventing autumn-shed seeds from germinating in their first autumn or even during the following spring. Therefore, the timing of seed dispersal can influence germination phenology, by determining the seasonal

conditions experienced by freshly shed seeds, and consequently determining whether they experience dormancy-breaking conditions.

Germination is also highly responsive to environmental conditions experienced before dispersal, during seed maturation on the maternal plant (reviewed in Gutterman 1992; Schmitt 1995; Donohue & Schmitt 1998). Environmental factors such as photoperiod, temperature, water availability, vegetative canopy development and even herbivore abundance all can vary seasonally, and all have the potential to act as cues of season during seed development. In particular, photoperiod is a very reliable seasonal cue, and many plant species exhibit plasticity of seed traits, including properties of seed coats, in response to the photoperiod during seed development (Karssen 1970; Gutterman *et al.* 1975; Gutterman 1978, 1994; Pourrat & Jacques 1978). Likewise, the temperature during seed development has major effects on dormancy and germination (Junttila 1971, 1973; Lacey & Pace 1983; Hume 1994; Lacey 1996; Donohue *et al.* 2005*a*,*b*) and varies seasonally in a somewhat predictable manner.

Reproductive phenology, therefore, imposes important maternal effects on seeds through influences of both pre-dispersal seed maturation conditions and post-dispersal seasonal conditions. Therefore, when a plant flowers very likely determines when its seeds germinate. Which of the many possible life cycles is expressed in the field depends critically on the season of seed germination. But germination phenology, in turn, depends on the season of seed maturation and dispersal via seasonal maternal effects. It is this pathway from adults to seeds that encompasses maternal effects on germination, which actually completes the life cycle. Without knowing how flowering and dispersal conditions influence germination, we cannot predict which life cycle will be followed. Therefore, maternal effects on germination are a significant missing link in our understanding of plant life cycles.

3. MATERNAL EFFECTS ON GERMINATION IN *Arabidopsis thaliana*

From this point forward, this paper will focus on the maternal effects on germination in the model genetic organism, *A. thaliana*. I chose this focus because it is the plant which I am currently working on, and it also enables a fuller discussion of the genetic basis of germination and maternal effects. Natural populations of *A. thaliana* vary geographically in their annual life histories and phenology, comprising winter annuals, spring annuals and mixed populations (Ratcliffe 1965; Effmertova 1967; Evans & Ratcliffe 1972; Thompson 1994; Nordborg & Bergelson 1999; Griffith *et al.* 2004), and as such provide a relevant ecological model for investigating how seasonal maternal effects influence annual plant life cycles. Being a colonizing species, germination phenology is likely to be an important determinant of whether a particular genotype with a particular germination phenology can successfully establish in an available location. Being widely geographically dispersed, *A. thaliana* also provides a research system for investigating the role of adaptive germination phenology in enabling plants to persist throughout wide geographical range, and how life-history variation enables adaptation to geographically variable seasonal environments.

(a) *Laboratory studies*

Natural populations of *A. thaliana* seeds cycle through dormancy from shedding through to germination (Baskin & Baskin 1972, 1983), with fresh spring-matured seeds unable to germinate under a wide range of conditions, including conditions resembling spring and summer seasonal conditions, while seeds that have been stored are capable of germinating under a wider range of conditions. The gradual loss of dormancy over the summer, and re-induction of secondary dormancy in late autumn, prevents seeds from germinating at times other than autumn in winter-annual populations. Thus, dormancy cycling and changes in the conditions under which germination can occur translate into restricted seasonal windows during which germination occurs in natural populations. Field and laboratory studies that assess the dynamics of dormancy loss and the conditions under which germination can proceed therefore provide valuable insights into the regulation of germination phenology in the field.

Most published studies of germination of *A. thaliana* show some evidence of maternal environmental effects, even if only in the form of differences in the germination of different seed batches (Koornneef & Karssen 1994). Such 'batch effects' alter the rate of loss of dormancy with dry after-ripening (Alonso-Blanco *et al.* 2003), germination percentages under constant imbibition conditions, or even responses to particular experimental factors such as exposure to plant hormones or other chemicals, cold, nitrate or light (e.g. Finch-Savage *et al.* 2007). While these batch effects combine potential uncontrolled differences in both plant growth conditions and experimental seed conditions during the germination assays, maternal plant growth conditions are notoriously influential and much more difficult to control than seed environments. The repeatability of results appears to depend on the density at which maternal plants were cultured, how frequently they were watered, the temperature and photoperiod at which they were grown, what pests they may have suffered, and many unidentified factors. Studies that have explicitly manipulated environmental factors during the cultivation of the maternal plants have found repeatable and large effects of specific environmental factors on seed germination and dormancy, and this has been found in countless other plant species as well (reviewed in Roach & Wulff 1987; Baskin & Baskin 1998; Donohue & Schmitt 1998).

One of the first such studies in *A. thaliana* showed that plants grown under a simulated canopy shade of low red : far red light produced seeds that required more light to germinate, suggesting that plants growing in a canopy in nature produce seeds that require light gaps before they will germinate (McCullough & Shropshire 1970; Hayes & Klein 1974). In a seasonal context, plants maturing late in the spring growing season, after the surrounding vegetation has formed a canopy, will produce seeds that will not germinate until the vegetation has died back again (likely in the autumn).

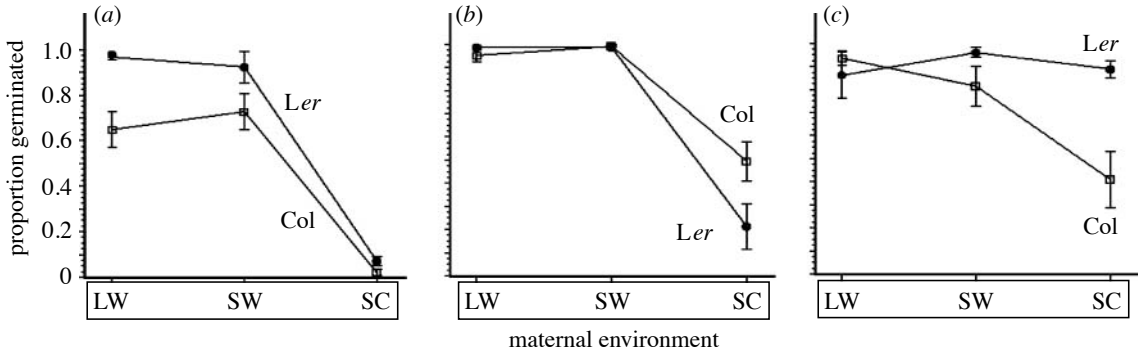

Figure 2. Germination proportions (y-axis) of seeds grown in three seed maturation conditions (x-axis). LW indicates long-day photoperiod (14 hours full spectrum light/10 hours dark) at a warm temperature of 22°C. SW indicates short-day photoperiod (10 hours/14 hours dark) at 22°C. SC indicates short-day photoperiod (10 hours) at a cool temperature of 10°C. Lines connect the mean germination proportion of each ecotype in the three seed maturation conditions. Standard error bars are shown. The seeds were put on water (imbibed) in the dark, and left to incubate for (*a*) 7 days at 22°C (neutral), (*b*) 7 days at 4°C (cold) or (*c*) 7 days at 31°C (warm) followed by 5 days at 4°C (cold). Col, Columbia ecotype; L*er*, Landsberg *erecta* genotype. Adapted from Donohue *et al.* (2007).

The adaptive significance of this maternal environmental effect has not as yet been tested in the field.

More recently, increased nitrate supplied to maternal plants was shown to reduce dormancy (Alboresi *et al.* 2005), not by preventing induction of dormancy but by preventing its maintenance. No effect of nitrogen application on germination was observed in another study, however, but removal of siliques and thereby nutrient sinks induced faster germination of the remaining seeds (Sills & Neinhuis 1995) again suggesting the possibility of nutrient effects on dormancy (although effects of wounding also could have contributed to these differences). Such maternal effects on germination may not influence the seasonality of germination, but they may enable opportunistic exploitation of impermanent resources by progeny.

Boyd *et al.* (2007) showed that the architectural position on the maternal plant at which seeds matured influenced the propensity of seeds to germinate without a prolonged cold treatment, as they would experience if they germinated in autumn, as opposed to with a cold period, as they would experience if they were to germinate in spring. This positional effect, moreover, differed greatly among natural ecotypes. Such maternal effects are likely to lead to sibships with heterogeneous germination behaviour, with some progeny germinating in autumn and others of the same genotype germinating in spring. As before, field studies testing this idea have yet to be executed.

Another recent study using two well-characterized lines of *A. thaliana* showed that cool seed maturation temperatures induced greater seed dormancy (figure 2; Donohue *et al.* 2007). In one line (Col), this dormancy was broken in approximately half the seeds by a short cold treatment, suggesting that many seeds of this ecotype that mature in cool days of autumn can germinate the following spring, facilitating a rapid-cycling life history. In the second genotype (L*er*), cold-induced dormancy was broken only after a cycle of warm followed by cold treatment, suggesting that seeds of this genotype that are matured in autumn will not germinate until the following autumn, after they have experienced warm summer temperatures. This would make them autumn or winter annuals, depending on when they flowered next. These are predictions that can be tested in the field.

Another study that used a set of recombinant inbred lines derived from crossing two natural ecotypes also showed that seed maturation conditions interact with post-dispersal conditions to influence germination (Munir *et al.* 2001). Seeds matured under short days (10 hours photoperiod) were more responsive than seeds matured under long days (14 hours photoperiod) to cold, germinating to higher percentages after a brief exposure to cold. Thus, seeds matured in autumn (short days) are expected to germinate to higher percentages later that autumn or during spring than seeds matured during late spring or summer (long days), all else being equal. A field study that used these same lines (plus several others) found some evidence that initially dormant seeds that had been matured under short days did germinate to slightly higher percentages in the autumn (Donohue *et al.* 2005*a*). This effect was quite subtle, however, suggesting that other post-dispersal seasonal conditions dilute this maternal effect.

In a comprehensive study of several natural ecotypes of *A. thaliana*, Schmuths *et al.* (2006) found that the temperature during seed maturation significantly influenced germination percentages, such that seeds matured at higher temperatures (22°C) germinated to higher percentages than those matured at cooler temperatures (14°C), and germination varied among ecotypes more when seeds were matured at the cooler temperature. The seeds that experienced warmer post-dispersal temperature also tended to germinate to higher percentages, and pre-and post-dispersal temperatures interacted differently for different ecotypes.

In summary, seed maturation conditions strongly influence dormancy loss and germination responses to particular post-dispersal conditions in *A. thaliana*. Factors that vary seasonally, such as photoperiod, temperature and even the seasonal development of vegetation canopy, strongly influence the germination responses in ways that can alter germination phenology under natural conditions. As such, these maternal effects on germination have the potential to be major

predictors of germination phenology and, in turn, major influences on the phenology of the entire plant life cycle. Put slightly differently, *in situ* variation in germination phenology may be, in part, due to the variation in reproductive phenology.

(b) *Field studies*

The laboratory studies of maternal effects on germination discussed above suggest that maternal effects have the potential to influence seasonal germination timing under natural conditions. However, few studies have tested these predictions in the field. One field study, however, did indicate that maternal effects induced under controlled conditions do influence germination phenology in the field. In this study (Donohue *et al.* 2005*a*), a set of recombinant inbred lines was exposed to different photoperiods during seed maturation, and the season of seed dispersal was also manipulated. Maternal photoperiod effects on germination timing in the field were subtle and affected only seeds with some degree of primary dormancy. When seeds were dispersed in June as opposed to November, seeds with primary dormancy that were matured under short days (10 hours) had significantly higher germination percentages than seeds matured under long days (14 hours), in accord with results conducted in the laboratory on a subset of these lines (see above and Munir *et al.* 2001). This effect was apparent only in one of the study sites, however.

In contrast to the minor effects of maternal photoperiod, the maternal effect of dispersal timing strongly altered germination phenology. Seeds dispersed in June had a pulse of germination in early summer, some germination throughout the summer, another pulse of germination in autumn and some germination throughout winter and spring. By contrast, nearly all the seeds dispersed in November germinated within a two-week period after dispersal, with a few seeds germinating throughout winter and spring. In short, germination was much faster and more synchronous when seeds were dispersed in autumn.

A striking result from this study was that stabilizing natural selection on germination behaviour was exceptionally strong when seeds were dispersed in June, with variance in germination explaining up to 72 per cent of the variance in fitness among genotypes (Donohue *et al.* 2005*b*). However, when seeds were dispersed in November, natural selection was weaker and directional. Remarkably, non-dormant seeds had the highest fitness when they were dispersed in autumn, but they had zero fitness when they were dispersed in June. Therefore, maternal effects operating through dispersal timing influenced not only germination phenotypes, but also natural selection on germination.

The genetic variance and heritability of germination also depended on the season of seed dispersal (Donohue *et al.* 2005*a*), with seeds dispersed in June having much higher heritability for germination, largely because autumn-dispersed seeds germinated synchronously. In this study, therefore, maternal effects influenced germination phenotypes, natural selection on germination and heritability of germination in the field.

Maternal effects on germination, and in particular the acceleration of germination of seeds dispersed in autumn, have the potential to enable a rapid-cycling life history. Without the maternal effect on germination, seeds would not have germinated until the following spring or even the following autumn. This is not a trivial accomplishment, since many seeds are shed with primary dormancy and require a prolonged period of 'after-ripening' to break that dormancy. Because seeds germinated very soon after they were dispersed in autumn, in contrast to the slow germination of June-dispersed seeds, seedlings were able to grow to an appreciable size, experience winter vernalization and flower and reproduce the next spring. That is, they were able to catch up in their phenology with seeds that were dispersed much earlier, even if they were not able to fully catch up in size. In this sense, maternal effects of dispersal season can influence later life stages and the overall life cycle.

4. DEMOGRAPHIC CONSEQUENCES OF MATERNAL EFFECTS ON GERMINATION

The study mentioned previously was conducted on recombinant inbred lines, which although derived from natural ecotypes are not natural genotypes. In order to test how seasonal maternal effects influence the life cycles expressed by natural ecotypes, and in particular whether this rapid-cycling life history actually occurs in natural ecotypes, a demographic study was conducted, the results of which are presented here.

In this study, seeds were collected from four natural populations of *A. thaliana* in MA, USA, during their two flowering periods: the first in June and the second in late October through to early December. The seeds from these two cohorts were planted in a common garden immediately after collection, and the cohorts were followed for two generations in order to obtain the demographic data. (See appendix A for details on experimental set-up.)

The study showed that these natural populations exhibited great variation in life history, exhibiting a winter annual, spring annual and rapid-cycling life history (figure 3). In particular, seeds that matured in autumn can germinate the following spring, and those germinants can survive to reproduce. While it is conceivable that autumn-flowering and spring-flowering sibships represent differentiated populations in some situations, in this study a single sibship that was collected from plants flowering in autumn actually produced progeny that reproduced in spring. This result indicates that autumn versus spring flowering is not completely genetically determined. Rather, plasticity of flowering time as a function of the timing of germination probably accounts for populations with mixed life histories.

The germination behaviour of spring-matured seeds differed greatly from that of autumn-matured seeds with respect to mortality and germination timing of surviving seeds (figure 3). Twenty per cent of spring-matured seeds germinated in the autumn, and 1.4 per cent remained dormant and germinated the following spring (while the remaining did not germinate over the 2-year period of the study and were probably dead). By contrast, germination percentages were lower overall

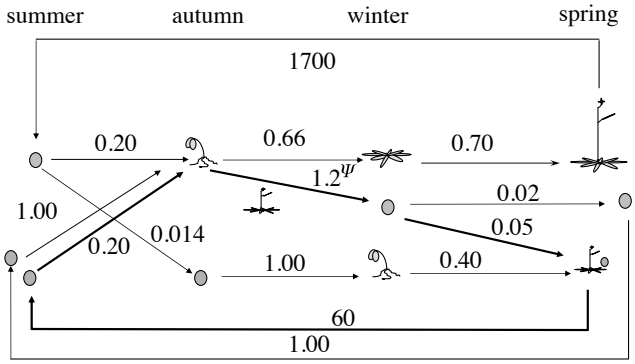

Figure 3. Transition probabilities between the stages of the life cycles shown in figure 1. Numbers represent the probability that one life stage will complete the transition to the next life stage. Probabilities less than 1 are due to mortality or choices at branch points in the life cycle. Values greater than 1 represent the transition from adult to seed, or the number of seeds produced by one adult. [Ψ]The transition probability combines the probability that the germinant bolted (0.03) and the number of seeds produced by those autumn-flowering individuals (40). Bold lines represent the rapid-cycling path.

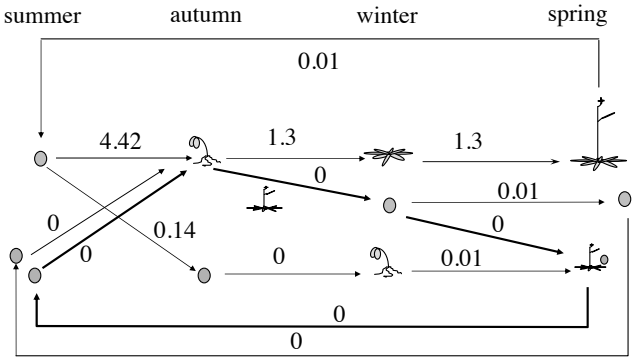

Figure 4. Sensitivity values for each transition. Sensitivities measure the unit change in the projected population growth rate (number of progeny *per capita* per generation) that would occur if the transition were changed by 1 unit.

for autumn-matured seeds, with 5 per cent of the seeds germinating in the spring and 2 per cent of them remaining dormant until the following autumn (the rest apparently being inviable). Estimated seed mortality, therefore, differed between seeds matured in spring (79%) versus autumn (93%). Moreover, germination phenology itself differed between cohorts; for those seeds that did germinate, 93.5 per cent of the spring-matured seeds germinated in autumn, whereas only 28.6 per cent of the autumn-matured seeds germinated in autumn. These results contrast to the previous experimental studies mentioned above (Munir *et al.* 2001; Donohue *et al.* 2005*a*), in that these autumn-dispersed seeds were not able to germinate during the same autumn in which they were dispersed, but rather delayed germination until spring. Very likely, the cooler temperatures experienced by autumn-matured seeds during seed maturation in the field contributed to their dormancy.

In agreement with the previous studies, germination timing had a strong effect on fitness, with winter annual autumn germinants producing an estimated 1700 seeds while spring germinants produce an estimated 60 seeds. In fact, a sensitivity analysis (Caswell 2001) showed that the transition from (spring matured) seed to germinant was the trait that, if altered, would have the largest effect on projected population growth rate (figure 4). The next most important transitions—specifically, the probability that autumn germinants flower in autumn, and the probability that over-wintering rosettes survive to reproduce—had sensitivities less than one-third of the transition from seed to seedling. Therefore, changes in germination behaviour are predicted to influence population performance more than changes in any other life stage.

While the rapid-cycling life history did occur in natural populations, that life-history pathway actually did not contribute much to the projected growth rate of these populations, as is clear from the extremely low sensitivities of the transitions in that pathway. This is largely because autumn-flowering plants produced so few viable seeds, and those seeds that did germinate (primarily in spring) flowered at a smaller size and consequently produced fewer seeds. However, the transition that avoids that pathway does have an appreciable sensitivity value (1.3); if fewer plants overwintered and reproduced the following spring, but instead reproduced in the previous autumn, then population growth rates would be depressed.

Given these observed transition probabilities, one can ask how population growth rates would be affected if maternal effects did *not* influence germination. If autumn-matured seeds had the same germination behaviour as spring-matured seeds (i.e. if they had a 20% probability of germinating in autumn, as opposed to 2%, and if they had 1.4% probability of germinating in spring, as opposed to 5%), projected population growth rate is barely altered (figure 5*a*). This is because only a small number of seeds were produced by autumn-flowering plants. If the converse were true, such that spring-matured seeds germinated in the same proportions as autumn-matured seeds, then projected population growth rates would be depressed by 30 per cent, indicating an appreciable influence of seasonal maternal effects on projected population growth rates under some conditions.

Moreover, projected population growth rates depend on the relative proportion of seeds that experience the different seed maturation conditions (figure 5*b*). If autumn-matured seeds were equally abundant as spring-matured seeds, the projected population growth rate would be lower, given the observed maternal effects; this is because a higher proportion of all seeds would germinate in spring (having been matured in autumn), and consequently be smaller and have lower fitness. If autumn-matured seeds had been equally abundant as winter annual spring-matured seeds, and if they exhibited the germination behaviour of spring-matured seeds, then the projected population growth rate would increase by 31 per cent compared with the case with the observed maternal effects on germination. By contrast, if spring-matured seeds behaved just as autumn-matured seeds, the projected population growth rate would be reduced by a further 11 per cent, and the population would actually decline, since λ would be less than 1. In this case, when the two seed types are

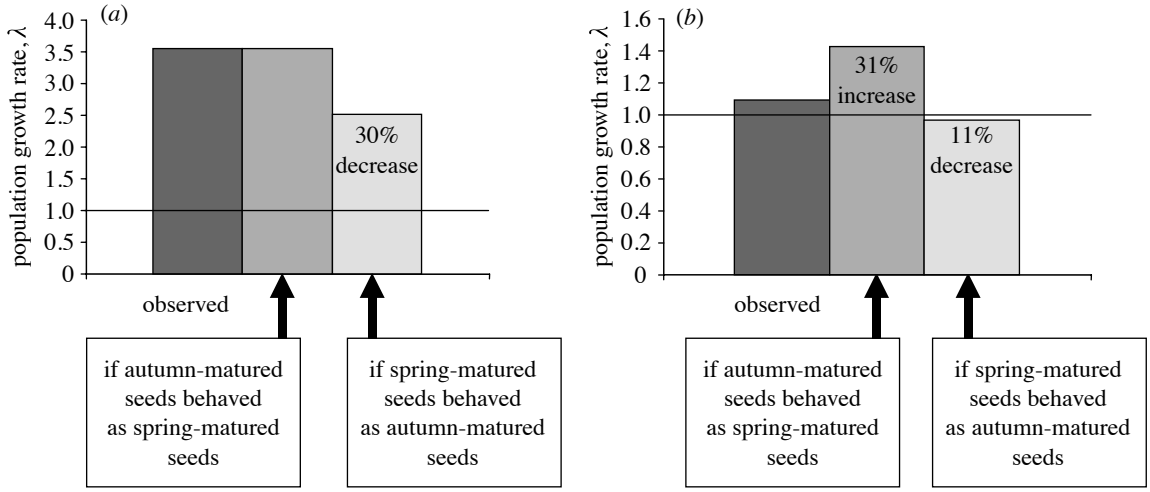

Figure 5. Projected population growth rates, λ, based on the matrix of transition probabilities presented in figure 3. (*a*) A comparison of the projected population growth rates when the actual observed transition probabilities were used to calculate λ (dark grey), when the germination behaviour of autumn-matured seeds was the same (no seasonal effects) as that of spring-matured seeds (intermediate grey), or when the germination of spring-matured seeds was the same as that of autumn-matured seeds (light grey). The observed seed production of the different cohorts was used to calculate λ. (*b*) Bars are the same as in (*a*), but the seed production of autumn-flowering plants was assumed to be the same as that of spring-flowering winter annuals, increasing the variance in the seed maturation conditions of the population. See text for more details.

equally abundant, the seasonal maternal effects on germination would actually determine whether the population increases or decreases in size.

These results show that seasonal maternal effects on germination are very likely manifest in natural populations, and they influence fitness, life cycles and the demographic performance of populations. These maternal effects would be more important, moreover, when the different maternal environments, in this case the frequency of the autumn- versus spring-flowering life histories, approach equal frequencies in the population. Thus, population demography is expected to be influenced by the opportunity for maternal environmental effects, via variation in reproductive phenology, and by the magnitude of these maternal effects on seed traits.

More generally, diverse demographic consequences of maternal effects can be expected in plant populations. In *C. americana*, for example, adaptive maternal effects caused higher projected population growth rates than experimentally disrupted maternal effects (Galloway & Etterson 2007), indicating that the matching of maternal cues and progeny environments is important for predicting demographic consequences of maternal effects. Moreover, maternal effects on between-year dormancy would influence bet-hedging dynamics, since greater dormancy can lead to lower temporal variance in fitness and consequently more stable population sizes in temporally variable environments (e.g. Venable 1985; Brown & Venable 1986; Venable *et al.* 1987; Evans *et al.* 2007). In perennial species, maternal effects on between-year dormancy have the potential to influence the age structure of populations, which in turn would influence projected population growth rates, probability of population extinction and genetic variation (Kalisz & McPeek 1992, 1993; Tonsor *et al.* 1993). Owing to the pronounced importance of dormancy to plant population dynamics, maternal effects on germination phenology have the potential to strongly determine the demographic dynamics of plant populations.

5. MATERNAL EFFECTS AND THE GENETIC BASIS OF GERMINATION

(a) *Maternal environmental effects on genes associated with germination*

Maternal effects have the potential to influence basic quantitative genetic parameters of seed germination, as mentioned previously (e.g. Donohue *et al.* 2005*a*). They have also been shown to influence specific genetic and biochemical processes associated with seed germination.

For example, a study that explicitly manipulated seed maturation conditions (Donohue *et al.* 2008) found that the particular phytochromes necessary for germination to proceed depend on the temperature of seed maturation. Phytochromes are red and far-red photoreceptors, and they regulate germination responses to light (Casal & Sanchez 1998). In the Brassicaceae, which includes *A. thaliana*, the phytochrome apoproteins are encoded by five genes—PHYA–PHYE—that have arisen through a series of gene duplications (Sharrock & Quail 1989; Clack *et al.* 1994; Mathews & Sharrock 1997). Donohue *et al.* (2008) have shown that when seeds are matured at warmer temperatures, knockout mutants of both PHYB and PHYD are able to germinate to comparable levels as wild-type if they are given a cycle of warm followed by cold dark imbibition treatment (figure 6). Thus, these two phytochromes are not required for germination under those conditions. By contrast, when seeds are matured at cool temperatures, the wild-type can germinate after warm–cold stratification, but these mutants are not able to germinate even though the seeds are viable. The results show that seeds can germinate without the function of these phytochromes if they are matured under one temperature, but not another. In particular, germination had not previously been known to be so dependent on functional PHYD; merely maturing seeds under a different (ecologically realistic) temperature exposed a new function for this gene.

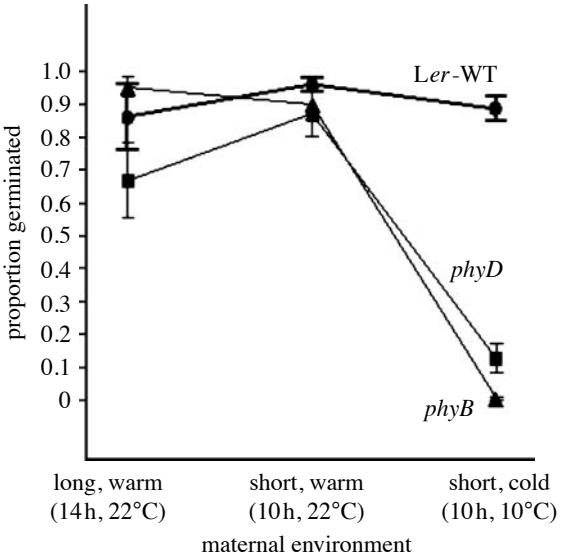

Figure 6. Germination proportions (y-axis) of the background genotype ('L*er*-WT') and two phytochrome mutants: *phyB*, which has a dysfunctional phytochrome-B and *phyD*, which has a dysfunctional phytochrome-D. Lines connect the mean germination proportion of each genotype in the three seed maturation conditions, as in figure 2. Standard error bars are shown. Adapted from Donohue *et al.* (2008).

The observation that maternal environments can alter which genes are most important for germination has some interesting implications for molecular population genetics more generally, since it suggests that maternal effects can influence which genes are exposed to natural selection. For example, the above study showed that the phytochromes necessary for germination depended on the temperature of seed maturation. An ongoing field study by D. Barua and colleagues suggests that the phytochromes that influence germination percentages in the field also depend on seed maturation temperature, and that as a consequence, the magnitude of natural selection on particular phytochrome genes and pathways depends on seed maturation temperature. In this manner, maternal effects on seed germination can contribute to variable natural selection on particular genes involved in germination. Specifically, seasonal maternal effects may vary geographically, when flowering seasons in different locations differ in photoperiod, climatic factors or biotic factors such as vegetation canopy or herbivores. If a species is distributed over a wide geographical range, differences in seed maturation conditions could impose requirements for different genes to elicit germination, and thereby impose geographical variation in natural selection on germination genes. In addition, flowering time can also vary as a function of genetic variation. Some genotypes are more likely to flower in autumn than others, for example. In this manner, maternal effects can contribute to epistasis for fitness, with selection on germination genes being contingent upon the flowering genotype. Both mechanisms of variable natural selection on germination genes—geographical and genotypic—are expected to contribute to the maintenance of genetic variation for germination. Maternal effects, therefore, have the potential to influence the geographical distribution of genetic variation and patterns of linkage disequilibrium in natural populations.

(b) *Genetic mechanisms of maternal effects*

The genetic mechanisms whereby maternal effects on dormancy and germination are imposed are poorly understood. Distinguishing between maternal versus embryonic control of germination is the first major challenge. The different genotypes of seeds—that of the seed coat (maternal genotype), the endosperm (triploid and two-thirds maternal) and the embryo (diploid and one-half maternal)—all contribute to dormancy and germination, and each may have distinct effects on seed processes during dormancy and germination. In fact, the global transcription profiles as well as the expression of individual germination genes have been shown to differ significantly between endosperm and embryos (Bewley 1997*a* and references therein; Dubreucq *et al.* 2003; Liu *et al.* 2005; Penfield *et al.* 2006).

Perhaps the most direct manner whereby the maternal genotype exerts control over dormancy and germination timing is through the testa, or seed coat, which is the maternal tissue. The testa imposes mechanical constraints on germination, and acts as an environmental filter. The testa regulates permeability to both water and oxygen and thereby regulates imbibition dynamics and oxidative processes, and it alters the intensity of particular light wavelengths that reach the embryo—all of which are known cues for germination (Finch-Savage & Leubner-Metzger 2006). Both the thickness and colour of the seed coats are known to be environmentally responsive in many species (reviewed in Roach & Wulff 1987; Baskin & Baskin 1998; Donohue & Schmitt 1998). In *A. thaliana*, testa mutants have altered mucilage and water uptake dynamics, are physically weaker and have reduced dormancy (Debeaujon & Koornneef 2000; Debeaujon *et al.* 2000, 2007). Therefore, environmental effects on mechanical and chemical properties of the testa are likely to be a major mechanism of maternal effects on germination, although the genetic basis of plasticity of these seed coat properties is not known.

In addition to the seed coat, the maternal plant influences dormancy and germination through provisioning seeds with proteins and transcripts during seed development (e.g. Dure & Waters 1965; Almoguera & Jordano 1992). Certainly, there exists plenty of opportunity for the maternal genome to affect seed dormancy and germination through stored compounds. Non-dormant seeds can complete germination without any post-dispersal transcription whatsoever (Rajjou *et al.* 2004). However, newly transcribed genes are necessary for timely germination, i.e. accelerated germination under favourable conditions, and for establishment. The same study showed that germination does require translation, indicating that de novo protein synthesis is necessary for germination. Surprisingly, the total proteomic profile of seeds that were prevented from transcribing RNA did not differ greatly from that of seeds without a transcription inhibitor (differing in the levels of accumulation of only

eight proteins), but the proteomic profile of newly synthesized proteins differed significantly, resembling instead the proteome of developing seeds. Therefore, both proteins and mRNA are stored in seeds during seed maturation, and these stored compounds are adequate to enable germination of non-dormant seeds. The demonstrated importance of post-transcriptional control of germination is perhaps one of the most significant developments in the studies of germination biology (Nakabayashi *et al.* 2005; Nishimura *et al.* 2005; Finch-Savage & Leubner-Metzger 2006; Holdsworth *et al.* 2007, 2008; Finkelstein *et al.* 2008). Its importance emphasizes the opportunity for maternal influences on dormancy and germination, since the stored mRNAs and proteins are synthesized during a time in which the connection between the maternal plant and the seeds is pronounced.

The role of these stored mRNAs and proteins is an area of exciting recent investigation. While stored compounds enabled the completion of germination of non-dormant seeds, their role in controlling the loss of dormancy and the conditions under which germination can proceed is largely unexplored territory. Stored RNAs have been shown to degrade during seed germination as a result of transcription during imbibition (Li *et al.* 2006). Nevertheless, seeds are metabolically active even before imbibition, since transcription (Bove *et al.* 2005; Leubner-Metzger 2005; Nakabayashi *et al.* 2005), protein synthesis (Chibani *et al.* 2006) and metabolism (Bailly 2004; Fait *et al.* 2006) occur even in dry seeds. Moreover, the metabolomes of dry seeds have been shown to differ among seed batches, suggesting that the metabolic processes of seeds are sensitive to seed maturation conditions (Fait *et al.* 2006).

The biochemical processes of dry seeds have a pronounced effect on dormancy and germination and can be relevant in the field when seeds experience dry conditions (as in summer in temperate climates) soon after dispersal. In general, seeds are induced into primary dormancy as a distinct process after seed development (Raz *et al.* 2001; Fait *et al.* 2006), and dormancy is gradually lost during dry storage, accompanying a process called after-ripening. How dormancy is lost during dry storage is one of the outstanding mysteries regarding dormancy. Recent studies showed that dry storage incurred the accumulation of new proteins (Chibani *et al.* 2006). The accumulation of new proteins and the persistence of existing stored proteins were inhibited by the application of abscisic acid (ABA), the phytohormone most important for dormancy induction and maintenance. Moreover, many genes that are expressed during dry storage are regulated by ABA (Nakabayashi *et al.* 2005). This evidence suggests that dormancy loss with dry storage is mediated by ABA, and that stored proteins and mRNA can participate in this process of dormancy loss, provided they are not inhibited by ABA. Interestingly, endosperm-associated genes tended to be downregulated during dry storage while embryo-associated genes tended to be upregulated (Carrera *et al.* 2008). Dry storage also alters gene expression profiles (Cadman *et al.* 2006), generally resulting in lower transcription of imbibed seeds. Dry storage specifically influenced the rates of degradation and new synthesis of ABA upon imbibition (Ali-Rachedi *et al.* 2004).

In one study, however, it was shown that even non-dormant, mutant seeds that are deficient in ABA synthesis undergo a process of dry after-ripening, during which their transcriptome changed in a manner similar to that of the wild-type transcriptomes (Carrera *et al.* 2008). The authors suggested that after-ripening and dormancy loss may be distinct processes, and that ABA may have little role in after-ripening *per se*. However, the results may also reflect the mutant status of the ABA-deficient seeds, such that the transcriptome changes would have been involved in dormancy loss had ABA been present. Regardless, ABA-mediated dormancy loss during dry storage may well involve interactions between the ABA and stored proteins and mRNAs.

An important dormancy gene associated with dormancy loss during dry storage, Delay of Germination 1 (DOG1), requires ABA to impose dormancy (Bentsink *et al.* 2006; Holdsworth *et al.* 2008). DOG1 is the only gene associated with natural variation in dormancy that has been cloned, and its biochemical function is as yet unknown. The variation in DOG1 expression is strongly associated with natural variation in the rate of loss of dormancy with dry storage (Alonso-Blanco *et al.* 2003). DOG1 expression levels increase at later stages of seed development, are high in fresh, dormant seeds, and decline in dry seeds after dry storage (Bentsink *et al.* 2006). DOG1 expression disappears completely in imbibed seeds whether they are fresh (and dormant) or after-ripened (and non-dormant). The timing of DOG1 expression—namely during seed development—suggests that the maternal genotype may conceivably mediate the induction of seed dormancy, although DOG1 itself appears to be expressed by the developing embryo.

While germination can proceed in non-dormant seeds without any transcription (Rajjou *et al.* 2004), germination of dormant seeds requires transcription. This result suggests that embryonic or endospermic, as opposed to maternal, gene expression is necessary for dormancy breakage (Finch-Savage *et al.* 2007). This is not to say that maternally provisioned compounds cannot regulate such transcription or alter the products of that transcription; the maternal genotype may still influence the dormancy-breaking processes even when it is no longer expressed.

With the exception of genes associated with testa quality, surprisingly few germination/dormancy genes have been actually demonstrated to be expressed by the maternal plant as opposed to the developing embryo. Two maternally expressed genes known to influence germination, DAG1 and DAG2, have been shown to be expressed only in the maternal vascular bundle of the funiculus connecting to the seed (Papi *et al.* 2000, 2002; Gualberti *et al.* 2002). The two genes work antagonistically with each other, with DAG1 repressing germination and DAG2 promoting it, probably in direct competition with DAG1. The germination factor that is repressed by DAG1 appears to be in the phytochrome pathway, since the germination response, when derepressed, is photoreversible. It has been proposed that DAG1 inhibits a germination factor that is transported to the embryo, where it is converted

to an active form by phytochrome under the appropriate light conditions. It would be interesting to test whether the balance of DAG1 repression and DAG2 promotion of germination changes as a function of environmental factors known to be associated with maternal environmental effects on germination—especially phytochrome-mediated maternal effects such as those mentioned above (e.g. Donohue *et al.* 2007, 2008).

A few other examples show that maternal gene expression influences dormancy and germination. Reciprocal crosses demonstrated that the dominance of two ABA response mutations (*abi1* and *abi2*), as assayed by seed sensitivity to ABA, depended on whether the maternal plant or the pollen donor carried the mutation; that is, it depended on whether the heterozygous embryo developed on a mutant or wild-type maternal plant (Finkelstein 1994).

Another study showed evidence that maternal and embryonic genes interact to influence vivipary (Raz *et al.* 2001), such that vivipary required a mutation in both a maternal gene (ABA1, an ABA biosynthetic gene) and an embryonic gene (FUS3, a gene associated with the control of accumulation of storage compounds and embryo growth arrest). Moreover, excised embryos of *fus3* mutants did not cease embryo growth even without maternal provisioning, whereas wild-type embryos ceased growth, suggesting that maternal provisioning in some way can derepress pathways that may be repressed by wild-type FUS3, and thereby promote embryo growth. Without excision of the embryo, *fus3* mutants also exhibited continued embryo growth and early germination, further suggesting that *fus3* activity may somehow be involved in germination. It is possible that germination effects of FUS3 may be regulated by maternal provisioning. This hypothesis is conjectural at this point, however.

The balance of maternal versus embryonic control of germination is still poorly characterized. Extensive studies of ABA, the primary hormone that induces and maintains dormancy, have shown that embryonically produced rather than maternally produced ABA is most effective at inducing dormancy (reviewed in Kucera *et al.* 2005). Moreover, embryonic rather than maternal control of seed development begins during seed maturation (at least in legumes; Weber *et al.* 2005). However, the demonstration of the extreme importance of stored proteins and mRNAs in regulating dormancy loss with dry storage, dormancy loss after imbibition and germination itself shows that the *opportunity* for maternal control of germination is vast. Nevertheless, developing and maturing embryos are also likely to contribute to these pre-dispersal reserves. We are far from characterizing the functions of these reserves, much less recognizing their origin—whether maternal or embryonic.

The so-called maternal environmental effects on germination occur when the conditions during seed maturation influence germination. Both maternal plants and embryos experience—and probably have the potential to respond to—environmental conditions during this time. Indeed, characterizing the contributions of each of these genotypes to maternal environmental effects needs to become a research goal if we are to understand the genetic basis of this major life-history transition.

6. CONCLUSIONS

Seed germination is a critical stage in the life cycle of plants, and the timing of seed germination has been shown to be under extremely strong and geographically variable natural selection. In some natural populations in the USA, the autumn transition from seed to seedling is the single life stage that, if it were to change, would have the largest effect on projected population growth rates. This is because the timing of seed germination determines to a great extent the basic life cycle that is expressed, including reproductive output and how many generations can be completed in a given year. However, it is the interplay between the flowering phenology and the germination phenology that ultimately determines plant life cycles and population demography. This effect of flowering phenology on germination—the pathway linking adult plants to particular seed phenotypes—is the pathway of maternal effects on germination. Maternal effects on germination are therefore a conspicuous missing link in our understanding of plant life histories.

Maternal environmental effects on dormancy and germination cuing influence germination phenology itself, and consequently life cycle expression and population demography. Maternal environmental effects also influence the strength and mode of natural selection on germination as well as the expression of genetic variation for germination. Maternal environmental effects even influence which genes are involved in the germination process. This has some important implications for molecular population genetics, since it suggests that maternal environmental effects can influence which genes are exposed to natural selection.

Maternal environmental effects on germination should not be seen as mere impediments to experimental investigations of germination and dormancy, but as a fundamental component of those processes. Understanding how maternal effects influence germination will add to our understanding of the genetic basis of this complex trait and provide insight into the patterns of variable natural selection on germination and germination genes as well as geographical and ecological variation in plant life cycles and population demography.

Maternal environmental effects, whether determined by the maternal or the embryonic genotype, will influence germination phenotypes and consequently influence plant life histories and patterns of selection on germination phenotypes and germination genes. Why should we care which genotype determines germination? The evolutionary dynamics of these maternal effects and of germination more generally depends on their genetic control. Indeed, the difference between the maternal and progeny control of this trait can determine whether responses to artificial and natural selection will be positive or negative (Kirkpatrick & Lande 1989), whether dynamics of parent–offspring conflict will be manifest in this trait (e.g. Goodrich 1998), and whether germination evolves primarily due to individual or kin selection. Therefore, fundamental evolutionary processes of

Table 1. Populations used for demographic observations.

population	collection site	description	no. collected in spring	no. collected in autumn
A	Cambridge, MA	lawn	24	2
B	Watertown, MA	naturalized park, along trail sides and minor disturbances in a hilly area overgrown with grasses, weedy herbs and small shrubs	24	2
C	Cambridge, MA	open pedestrian park in Cambridge, MA (Dennehy Park) in a gravelly exposed margin	24	37
D	Montague, MA	agricultural margin	24	28

natural selection and responses to selection depend on the genetic determination of offspring traits.

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APPENDIX A. EXPERIMENTAL SET-UP OF DEMOGRAPHIC STUDY OF SPRING- AND AUTUMN-FLOWERING COHORTS OF *Arabidopsis thaliana*

Seeds were collected from 24 individuals (seeds from a given individual are referred to as sibships henceforth) from each of four natural populations in Massachusetts, USA, during June. These populations were chosen because they had previously been observed to flower in both spring and autumn (table 1), and they thereby enable us to examine differences between these cohorts. Within a few days of collection, seeds were deposited into a prepared field at the Concord Field Station (Harvard University) in Bedford, MA. Twelve seeds from a given sibship (from a given population and seed collection time) were deposited into a given peat pot filled with Metromix 360 (Scotts Sierra, Marysville, OH, USA) using the planting methods of Donohue (2005).

The field site was divided into 12 blocks. In each block, four pots were left empty to assess contamination by dispersed seeds. For June-collected seeds, each block contained pots for two sibships per population. The seeds of each sibship were assigned to one of two germination cohort treatments: 'autumn germination' and 'spring germination', which corresponded to the season of germination of the focal individual in that pot (see below). This resulted in 192 pots of June-collected seed pots per population, or 2304 seeds (12 blocks $\times$ 2 individuals/pop/block $\times$ 4 populations $\times$ 2 germination cohorts $\times$ 12 seeds per individual). Seeds were collected from fewer autumn-flowering plants than spring-flowering plants because autumn flowering was not common during the year of the collection. In total, 67 plants were found to be maturing seeds in autumn (table 1). To balance the number of pots from both collection periods, seeds from the 67 autumn-flowering individuals were distributed over the same number of pots as those that contained seeds from the 96 spring-flowering plants, with each pot containing seeds from a single sibship, as before. This resulted in a total of 384 pots (4608 seeds).

After depositing seeds in the field, pots were monitored weekly for germination. The date of each germinant was recorded. In each pot, a random focal germinant was chosen to follow throughout its life, and all other germinants were plucked from the pot during each census. Only one germinant remained in the pot after each census so that germination would not be suppressed by a carpet of rosettes in the pot. At the end of the autumn, the autumn germination pots contained a single rosette, and all spring germinants were subsequently plucked. To prevent suppression of spring germinants by autumn germinants, we plucked all germinants from the spring germination pots through autumn, and then chose a random focal spring germinant the following spring. Thus, the spring germination pots enable the accurate assessment of the germination phenology of all seeds within the pot without suppression from previous germinants, and these pots were used to estimate the germination timing and the germination percentage for that replicate.

Each focal individual was followed throughout its life, and bolting date (the date on which reproduction was initiated), rosette diameter at bolting, number of rosette leaves at bolting, flowering date and death date were recorded. All individuals were harvested upon senescence, and the total number of siliques (fruits) produced by that individual was counted. The seeds were counted on a random sample of 20 siliques in the experiment, and the average (20 seeds per silique) was used to estimate the number of seeds produced by each plant. Pots that never produced a reproductive individual were followed for a second year. Pots with plants that reproduced were contaminated with their own seeds, so those pots could not be used further. While the uncontaminated, second season pots are a biased sample of sibships that failed to reproduce, they did provide some information on between-year dormancy.

As soon as most seeds of a focal individual matured, they were collected, aliquotted in the laboratory the next day, and then deposited back into the field the following day in an adjacent plot. The date of seed collection was recorded for each pot. Because less than one quarter of the pots in the experiment produced reproductive plants, the sample size of the second generation was much smaller than that of the first generation. The second-generation plot

contained five blocks, with representatives from each population in each block. As described above, the germination of seeds within each pot was recorded, and random focal individuals were followed throughout their life. The purpose of the second-generation observations was primarily to assess seed germination and consistency of life-history expression across generations. Results presented here are based on the more complete first-generation data.

For each seed-collection cohort in the first generation, we calculated the mean germination date, the total proportion of seeds that germinated and the proportion that germinated in the first autumn (for spring-collected seeds only), spring and second autumn. The plants were classified into the following categories: spring collected seeds that germinated in the first autumn; spring-collected seeds that germinated in the spring; spring-collected seeds that germinated in the second autumn; winter-collected seeds that germinated in the spring; and winter-collected seeds that germinated in the second autumn.

Transition probabilities between the life stages were calculated for demographic analysis using standard demographic calculations (Caswell 2001). Seed maturation cohorts were considered as distinct life stages since the transition probability to different germination cohorts was expected to differ between the two seed types. Likewise, autumn and spring germinants were considered distinct life stages because their flowering phenology was expected to differ. Finally, three flowering cohorts were recognized because their reproductive output was expected to differ: autumn-flowering individuals; spring-flowering individuals from autumn germinants (and thereby larger rosettes); and spring-flowering individuals from spring germinants (and thereby smaller rosettes). Projected population growth rates and element sensitivities are presented here.

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