The adaptive significance of maternal effects

Timothy A. Mousseau and Charles W. Fox

Recently, the adaptive significance of maternal effects has been increasingly recognized. No longer are maternal effects relegated as simple 'troublesome sources of environmental resemblance' that confound our ability to estimate accurately the genetic basis of traits of interest. Rather, it has become evident that many maternal effects have been shaped by the action of natural selection to act as a mechanism for adaptive phenotypic response to environmental heterogeneity. Consequently, maternal experience is translated into variation in offspring fitness.

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The causes and consequences of phenotypic variation among individuals are of fundamental interest to students of evolutionary ecology because it is this variation that provides the raw material for natural selection. We are accustomed to envisioning an individual's phenotype as the result of its own genotype plus the environmental effects experienced during development. However, in recent years, with the increasing use of quantitative genetic designs for the study of life history, behavior and development, it is becoming evident that individual phenotype is frequently, and sometimes dramatically, influenced by the environmental experience of other individuals in the population. Not surprisingly, most often (but not exclusively), these interindividual interactions occur between mothers and their offspring.

Mothers determine propagule size, where, when and how propagules are dispersed, protection of young from inclement conditions or predators, parental care and provisioning to developing young, as well as the attributes of the offspring's father if mate choice is operating. In addition, a mother's experience of the environment can lead to variation in her growth (i.e. body size), condition and physiological state that can be transmitted to offspring via cytoplasmic factors (e.g. yolk amount, hormones and mRNAs) in the egg that may directly (via maternal programming) or indirectly (via offspring sensitivity to maternally transmitted factors) influence offspring development. The extent to which maternal environment and behavior influence offspring phenotype and fitness will determine the likelihood that such maternal effects themselves will be shaped by the action of natural selection.

In recent symposia[1–3], it has been repeatedly suggested that maternal effects often provide a mechanism for adaptive transgenerational phenotypic plasticity, in which the environment experienced by the mother is translated into phenotypic variation in the offspring, and that this relationship can be envisioned (and modeled) as a reaction norm (Box 1). Here, we explore four broad classes of environmentally induced maternal effects that have received considerable attention in recent years: (1) maternal effects on offspring development, (2) the influence of maternal oviposition behavior on offspring fitness, (3) maternal effects on propagule resources, and (4) the influence of female mate choice on offspring. This review complements a recent *TREE* article by Wolf *et al.*[4] that deals with the genetic complications that emerge from maternal effects.

Maternal effects on offspring development

There are numerous reported examples of maternal environmental influences on offspring development. In many insects, the photoperiod, temperature, or host availability experienced by an ovipositing female will determine the probability of diapause in her offspring[5] (Box 2). In general, females that experience short photoperiods, cool temperatures or few potential hosts (i.e. cues that predict deteriorating environmental conditions) tend to produce a high proportion of diapausing offspring. Although the details of the response curve (i.e. the reaction norm) have rarely been investigated, environmentally induced maternal effects have been demonstrated for more than 70 insects. Similarly, environmentally induced maternal effects on seed dormancy and/or germination have been reported in many plants[6,7]. In many cases, maternal photoperiod will influence the probability of seed dormancy, although other cues, including crowding, light, interspecific competition, maternal size and the position of the seed on the maternal plant, have been shown to exert influence on dormancy or germination. In most cases, the adaptive significance of maternal effects on diapause and dormancy is obvious: survival during the winter (or summer in hot desert environments) is dependent upon the physiological state of the propagule. Dormancy and diapause are protective mechanisms that enhance survival during inclement seasons.

For insects using seasonal or ephemeral resources, environmental cues, such as crowding, temperature and photoperiod, are often predictable indicators of the future deterioration of their habitat and impending food shortage. In some insects, females (and sometimes males) respond to their environmental conditions by stimulating the production of winged and/or flight-phenotype progeny, or by influencing how sensitive their progeny are to subsequent environmental conditions[8]. These flight polymorphisms are best studied in aphids and grasshoppers but have also been studied in other insects. The production of dispersing progeny often increases with maternal age, possibly because environmental quality tends to decrease throughout the season, which corresponds to increasing maternal age.

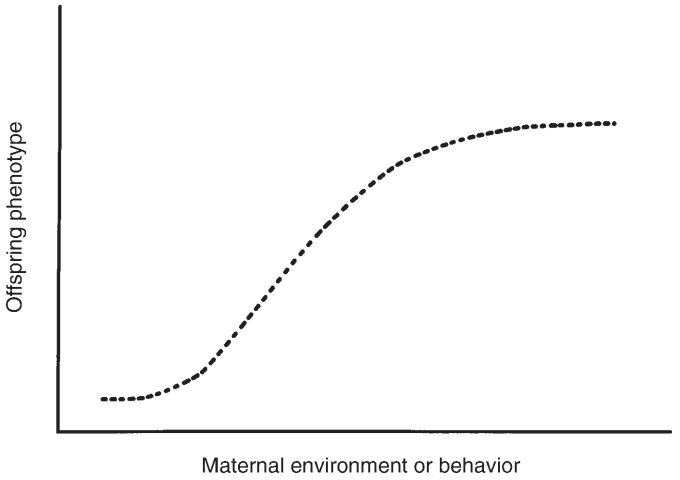
Effects of maternal oviposition decisions on offspring

Even in species with no direct parental care, when, where and how mothers place their offspring is often the single greatest determinant of offspring success[9]. For example, a female herbivore that places her eggs on an appropriate host

  0169-5347/98/$19.00 PII: S0169-5347(98)01472-4

Box 1. Maternal effects as transgenerational phenotypic plasticity

Many maternal effects can be visualized as a reaction norm describing the influence of maternal environment or behavior on an individual offspring's phenotype (as in the figure). Maternal effects occur when a mother's phenotype influences her offspring's phenotype independently of the female's genetic contributions to her offspring. Many maternal effects can be modeled as environmentally modulated transgenerational phenotypic plasticity, in which environmental variation (e.g. temperature, photoperiod and nutrients) experienced by mothers is translated into phenotypic variation in offspring. Similarly, maternal behavior (e.g. host choice, oviposition behavior and parental care) will often influence offspring phenotype and fitness.



In many cases, maternal effects can be visualized as reaction norms that extend across generations. For example, variation in maternal photoperiod often influences the expression of diapause in insects[5] and dormancy in plants[6], and the temperature of a nest chosen by many female reptiles can influence the sex of offspring[18,19]. Similarly, female choice of male secondary sexual characters can influence aspects of offspring fitness[12,22,40,43], and female host choice by herbivores and parasites can dramatically influence offspring growth and survival[17,24,36]. In addition, many aspects of female environmental experience and behavior can influence offspring fitness via effects on propagule (i.e. eggs or seeds) size[27,28,32,33,35,36] or through competitive interactions among offspring (i.e. clutch size effects)[23–25].

plant at an appropriate time is likely to have higher fitness than a female that oviposits randomly (Box 3). Similarly, dispersal of seeds is often controlled by the female plant.

Host choice

For organisms developing on discrete resource patches (such as parasitoids, seed beetles and leaf-miners), host size can dramatically affect the survival and growth of progeny. Thus, as expected, females of most parasitoids preferentially lay eggs on larger hosts[10].

The plant species or plant part that a female develops on can have dramatic consequences for the phenotype of her offspring, mediated via maternal or paternal effects. For example, the plant species that a mother develops on can affect the composition of her eggs, which subsequently affects the growth and development of her progeny[11]. Thus, female oviposition decisions can affect not only the growth and development of her progeny, but also that of her grandprogeny. Host-plant effects on progeny are not restricted to maternal effects – paternally-derived substances are frequently incorporated into eggs during oogenesis (compounds sequestered by fathers can be transferred to the eggs of their progeny) where they might affect progeny fitness[12].

Variation in maternal and paternal diet-mediated effects on progeny growth provide the potential for adaptive transgenerational plasticity – if the host plant on which a female (or a male) rears its offspring is predictive of local or future host-plant availability (which it generally is), then it is advantageous for them to produce offspring that are 'acclimatized' to the host on which they have been reared. However, whether females produce offspring that are acclimatized via nongenetic effects has rarely been tested, and little evidence exists for the acclimatization of host-plant suitability[13,14]. More work needs to be done in this area because maternal conditioning of host suitability, if demonstrated for any organism, could have profound implications for our understanding of host-use evolution of herbivores, host-race formation and sympatric speciation. For example, host experience often influences oviposition preference of females[15] and if host experience also influences larval performance on these hosts, then correlations mediated via linkage disequilibrium between oviposition preference and larval performance could be maintained in a randomly mating population through the effect of maternal host experience[16]. Such epigenetic interactions could result in a runaway process that facilitates rapid local adaptation and, eventually, speciation in systems where maternal-oviposition choices determine the environments for offspring development[16,17].

Progeny sex ratio

In addition to affecting progeny survival and growth, where and when a female lays her eggs can affect the progeny sex [i.e. environmental sex determination (ESD)][18,19]. Progeny sex can be influenced by biotic factors, such as the density of conspecifics, the quality of food resources or even by the quality of mate (biotic ESD). Abiotic factors, such as temperature, pH or photoperiod, can also affect progeny sex (abiotic ESD). In many species, mothers can manipulate or respond to their environment in a manner that suggests adaptive adjustment of progeny sex. Perhaps the best studied examples are in the Hymenoptera (wasps, bees and ants), in which females can manipulate sex ratio by simply choosing whether to fertilize an egg (haplodiploidy; fertilized eggs generally produce females, with diploid males uncommon in most species)[20]. For example, in most parasitic wasps, females manipulate progeny sex ratio in response to: (1) host size – producing female progeny on larger hosts because host size affects the lifetime reproductive success of female progeny more than that of male progeny; and (2) local female density (i.e. the number of other mothers present) – producing a greater proportion of sons when female densities are higher, which is likely because either high female:male ratios imply that there is increased competition for males in the population (and thus individual males have higher fitness than females), or increased densities of egg-laying females result in increased larval competition and, therefore, smaller progeny[21]. Temperature dependent ESD is also common in reptiles (e.g. turtles and alligators), providing the opportunity for females to adaptively manipulate the sex ratio of their progeny (although the adaptive significance of sex ratio adjustment is still speculative)[18,19]. Recently, it has been found that female collared flycatchers (*Ficedula albicollis*) can adjust offspring sex ratio in response to mate quality, favoring sons when mated to males with large forehead patches and daughters when the forehead patch is small. This has been interpreted as female variation in sex allocation in response to sex specific fitness differences (large-patched males are more attractive to females and sire higher quality offspring)[22].

Clutch size and superparasitism

Many animals lay their eggs in clutches. How many eggs a female produces in a clutch, or how readily she superparasitizes a resource patch (i.e. lays eggs on the same patch

as another female), will affect the degree of resource competition her progeny experience and thus their growth and survival, particularly if eggs are laid on discrete resource patches[23–25]. Females of many animals can adjust the number of eggs that they lay depending on where they are laying them, which is consistent with predictions of optimality models[23]. For example, females generally lay smaller clutches on smaller resource patches[14], and they also generally avoid ovipositing on hosts that already bear conspecific eggs[24]. Superparasitism is often necessary when hosts are limiting, females are old or host handling time is high, but it can come at a substantial cost to progeny in terms of reduced growth and survival. However, in some cases, superparasitism can improve progeny growth or survivorship (an 'Allee' effect) if it improves the quality or accessibility of a resource (possibly by improving the ability of individuals to overcome a host's defenses) or if it reduces the *per capita* risk of predation or parasitism[24,26].

Maternal effects on propagule resources

Propagule size

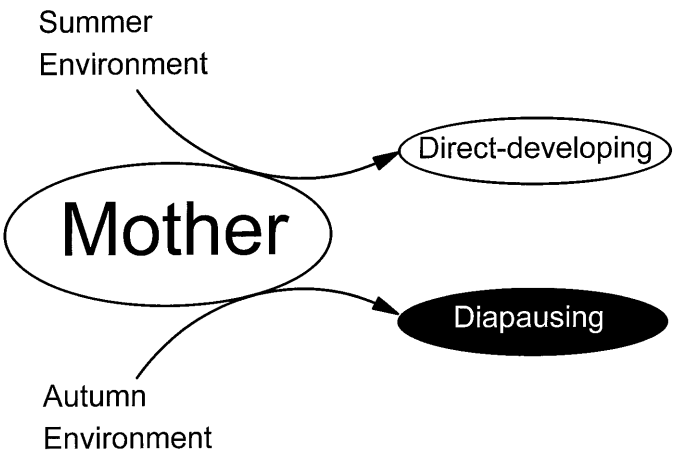
Propagule size is a particularly important life history trait mediating maternal influences on progeny phenotypes because it is simultaneously both a maternal and offspring character – eggs are produced by mothers, but also determine initial offspring resources and size[9,27], and variation in initial size can be propagated through the individual's life[28]. Thus, the amount and quality of resources allocated to propagules by mothers often profoundly influence the growth and survival of their progeny[14,25–35]. In natural populations, the consequences of variation in propagule size depend on the environmental conditions experienced by progeny[35], with fitness differences between large and small propagules generally greatest in adverse environments[36]. Thus, selection favors the production of different sized propagules in different environments.

In many animals, egg size and number vary within and among females depending on the environmental conditions experienced by the female[37], generally reflecting a constraint associated with maternal nutrient status or age. However, in some animals, the egg-size plasticity associated with maternal age or nutrient status appears to prepare their progeny for the environmental conditions they will experience[38]. In some insects, females can respond rapidly to their immediate oviposition environment by modifying the size and/or composition of the eggs they produce independently of their physiological state. For example, in the seed beetle, *Stator limbatus*, females respond to the host plant they lay eggs on by adjusting the size of their eggs, laying substantially larger eggs on poorer quality host plants[14,36]. However, increasing egg size comes at a cost; mothers laying large eggs must lay fewer eggs because of the tradeoff between size and number of progeny[36,39].

Maternal effects and sexual selection

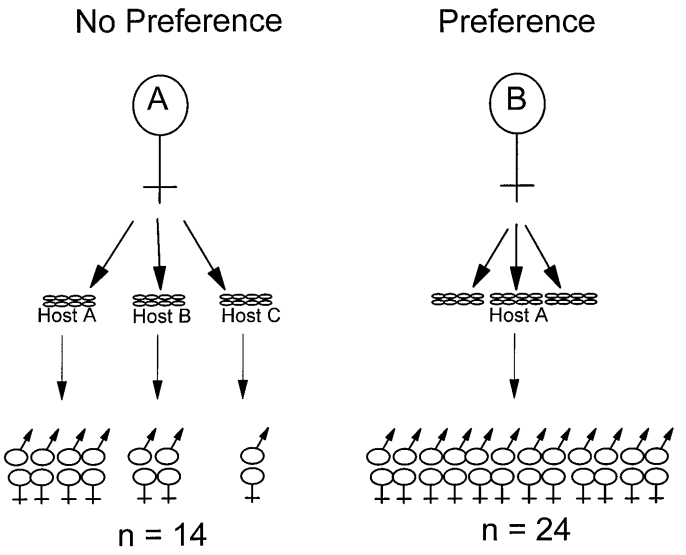
In many organisms, mothers actively select particular males to sire their offspring, and the adaptive significance of mate choice has been the subject of considerable interest to evolutionary biologists. Recently, several studies have found relationships between indicator traits used by females to select males and measures of fitness in the offspring[22,40] (Box 3). Such a relationship between female mate choice and offspring phenotype falls within the realm of maternal effects because variation in offspring phenotype is mediated via maternal behavior, rather than any 'genes' she may pass along. The causes of phenotypic variation in offspring might result from variation among males in territory

Box 2. Environmentally modulated transgenerational plasticity in offspring development



The figure illustrates the influence of maternal environment on offspring development. In many insects and plants, the expression of diapause or dormancy is regulated by maternal environmental experience[5,6]. In most cases, the photoperiod experienced by a mother will influence whether an offspring enters a diapausing or dormant state, although temperature and host availability have also been found to affect diapause. Often, the environmental cue to which females are receptive is a good predictor of future environmental conditions (i.e. short photoperiods and cooling temperatures are good indicators of an impending winter). In many species, a female will change from producing diapausing (or dormant) offspring to directly developing offspring (and vice versa) in response to a change in environmental conditions[5]. Similar responses are observed for maternal effects on flight and wing polymorphisms[8,14].

Box 3. Maternal host/mate preferences often influence offspring fitness



The figure illustrates the potential effects of maternal host or mate preference on maternal and offspring fitness. Variation in maternal preferences for hosts that vary in their suitability for growth and survival frequently result in maternal effects. Maternal host preferences are expected to evolve when offspring fitness varies with host because a female who places her eggs randomly among hosts (female A) will have lower fitness than a female with the ability to discern among potential hosts (female B). Relationships between maternal-host preference and offspring performance are frequently observed in host–parasite and plant–insect interactions[17]. Similarly, there is mounting evidence that females often show preferences for mates who positively affect offspring fitness in some way[22,40,43]. Females choosing to mate with high quality males often obtain resources (e.g. nuptial gifts and parental care) that are translated into increased offspring quality (e.g. increased size and/or increased survival), making female mate choice an adaptive maternal effect.

quality, male parenting abilities or extragametic contributions transmitted from the father and expressed by offspring (all of which might also lead to paternal effects on offspring). As long as female choice affects offspring phenotype, this choice can be considered a maternal effect. One could argue that female preferences for 'good genes' might also be considered a maternal effect because it is maternal behavior that leads to variation among offspring. However, female choice for good genes extends beyond popular definitions of maternal effects, which currently include only nongenetic sources of phenotypic variation.

The tie between sexual selection and maternal effects is an exciting one for evolutionary biologists because it lays the foundation for future studies of 'interacting phenotypes'[41]. Although the significance of 'extended phenotypes' has long been appreciated[42], recent theoretical explorations have provided novel insights to the evolutionary dynamics that can result from interactions among individuals[16,41,43,44]. Of particular interest are the findings that maternally affected traits are likely to harbor significantly more additive genetic variation than 'regular' traits, and that special linkage disequilibrium can exist between maternal and offspring traits[16,17]. Wade[16] and others have suggested that these genetic peculiarities of maternally affected traits could promote a runaway process and rapid speciation, especially in host–parasite systems where female host preferences and offspring performance jointly determine fitness. In addition, the relative rate of evolution for traits involving 'interacting phenotypes' can be either significantly slower or faster than that predicted by classical genetic models[41,43–45]. Maternal effects could even be a significant factor underlying periodic cycles in population density in many mammals and insects[46]. The potential for unexpected evolutionary dynamics, combined with their taxonomic and phenomenological ubiquity, will make the study of maternal effects a worthy investment.

Acknowledgements

The authors thank Allen Moore, Trevor Price and Heather Preston for valuable discussions concerning the adaptive nature of maternal effects. We also thank the US National Science Foundation and Oxford University Press for promoting this project.

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The role of soil community in plant population dynamics: is allelopathy a key component?

In his recent *TREE* news & comment, Watkinson[1] drew attention to the role of soil microorganisms in plant population dynamics. In particular, he reported on the dynamical framework for the inter-relations between the composition of plant and soil communities, proposed last year by Bever *et al.*[2] It is worth adding allelopathic interactions to this picture.

Allelopathy has been defined by Rice[3] as 'any direct or indirect harmful or beneficial effect by one plant (including microorganisms) on another through production of chemical compounds that escape into the environment'. If we apply this definition to Bever *et al.*'s framework for the feedback interaction between the soil community and two plant species, at least two further aspects come into play.

The first concerns the potential mechanisms for positive and negative feedbacks. We can hypothesize direct interactions, such as mycorrhizal systems, for positive feedback (the fungal symbiont allows plant species to explore more soil resources, thus augmenting the autotrophic community) and pathogens for negative feedback. But we can also hypothesize indirect interaction: the plant produces allelochemicals that are metabolized by soil microorganisms[4], leading to the release of compounds into the soil that might affect (positively or negatively) the plant species[5].

The second concerns the feedback model proposed by Bever *et al.*[2] The authors did not depict any direct or indirect interaction between the two plant species in their model. Nevertheless, one could add connections between them because of the potential occurrence of direct allelopathic interactions among plant species.

I agree with Watkinson's conclusion that 'the soil community is something that plant population biologists can no longer ignore' (if indeed they do), but they should not ignore allelopathy either.

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Reply from A.R. Watkinson

Pellisier is quite right to draw our attention to allelopathy as a potential component in the interaction between plants and microorganisms in the soil. But readers familiar with John Harper's strong views on the subject[1,2] will not be surprised to know that I, having been a student of his, am also rather sceptical about it. Unfortunately, Rice's definition quoted above is not at all helpful in defining allelopathy so broadly as any harmful or beneficial effect, direct or indirect, produced by a chemical that just happens to have escaped into the environment. That means that carbohydrate exudate from the root or the chemical compounds from a damaged piece of root are potential allelopathic agents. Most people would not accept that as allelopathy and indeed it is not what is studied.

Others restrict the definition of allelopathy to a form of interference competition by means of chemical compounds produced by one species that reduce the performance of other species[3]. Whether this interaction is direct or indirect is – I believe – critical, especially when one considers how allelochemicals may have evolved; I suspect that the direct interaction is relatively rare. Unfortunately it is impossible to say how rare or common a phenomenon it is, as many of the criticisms made by Harper[2] and others[4] of the methodologies involved in demonstrating allelopathy, and in particular the use of leachates, still apply. I would not dispute that chemical compounds (carbohydrates, proteins, phenols) from the roots of plants may have an impact on the microbial community and thus potentially on other plants as outlined in my original article. But are simple carbohydrates allelochemicals?

A computer literature survey (BIDS) of references to allelopathy in the past 10 years revealed that the subject barely merits a mention in the mainstream ecological literature: e.g. *American Naturalist* (0), *Journal of Ecology* (1), *Oikos* (1), *Ecology* (6), *Oecologia* (10). While agronomists, weed scientists and foresters clearly have more time for the concept, most of the 455 references to allelopathy are in the specialist *Journal of Chemical Ecology* (109) and *Phytochemistry* (23). There remain few attempts to relate the results of laboratory experiments to field situations.

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Reply from J. Bever, K.M. Westover and J. Antonovics

Watkinson[1] and Pellissier provide valuable perspectives on our model of the impact of the soil community on plant population dynamics[2]. The routes for such feedback can indeed be quite complex. In our work within a grassland in North Carolina, USA, we found that the accumulation of host-specific pathogens from the genus *Pythium* plays an important role in generating the negative feedbacks on plant growth that are common within the system[3,4]. However, we have also found