Maternal Effects as Generators of Evolutionary Change

A Reassessment

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Despite being a center of debate in biology for centuries, the connection between the generation of novel adaptive variation and its inheritance remains a contentious issue. In evolutionary and behavioral ecology, assigning natural and sexual selection a creative and anticipatory role unmasks the need to explicitly consider the link between a trait's functional importance and its inheritance and results in confusion about selection as an adaptive modifier of development versus selection as a passive filter of already produced forms. In developmental genetics, an emphasis on regulatory versus coding aspects of molecular evolutionary change overlooks the fundamental question of the origination of an inherited developmental toolkit and assignment of its regulatory functions. Because maternal effects, by definition, combine developmental induction of functionally important changes and their inheritance, they bridge the origin and evolution of organismal adaptability, at least on short time scales. The explosion of empirical studies of maternal effects raises a question—are maternal effects ubiquitous but short-term adjusters and fine-tuners of an evolved form with only secondary importance for evolutionary change? Or are they a particularly clear example of a stage in a continuum of inheritance systems that accumulates, internalizes, and passes on the most consistent and adaptive organism–environment interactions? Here I place recent empirical studies of avian maternal effects into the evolutionary framework of variation, selection, and inheritance to examine whether maternal effects provide a window into evolutionary processes.

Key words: **inheritance; evolution; adaptation; selection; development; maternal effects**

Maintenance of Adaptation versus Evolutionary Change

Even after being a center of scientific debate for several centuries, the relationship among origination, modification, and inheritance of organismal systems remains a contentious issue. Two questions seem particularly difficult to resolve: How is novel variation generated, and what accounts for its discreteness? And what is the connection between generation of novel variation and its inheritance? It is often stated that the success of Charles Darwin's theory was facilitated by setting aside the second question, a theory of inheritance (Darwin 1872), which made the link between natural selection and evolutionary change particularly direct. Similarly, a conceptual breakthrough in the theoretical development of the neo-Darwinian Modern Synthesis in the 1930s came from a strategic abandonment of the first question—the origin of developmental and physiological variation (Mayr & Provine 1980; Mayr 1982)—and explicit refocusing on the link between inheritance of randomly generated genetic changes and natural selection—the

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processes considered to be strictly independent of one another (Dobzhansky *et al.* 1977). Yet, although the central requirement of strict independence among developmental variation, function, and inheritance formed the foundation of population genetics theory of the maintenance and modification of existing variation (e.g., maintenance of adaptation), it has had considerably less influence on the progress of scientific fields concerned with the evolution of innovations, adaptability, and diversification. Not surprisingly, the application of the conceptual framework of the Modern Synthesis to fields in which integration of development, function, and inheritance is crucial—such as developmental biology, behavioral ecology, comparative anatomy, and animal physiology—significantly extended the original role of natural selection from a passive postproduction filter to "a dynamic force that actively molds and shapes organic forms" (Eldredge 1985), despite warnings that natural selection is not a force that "acts or favors" (Endler 1986), and is far better suited to explain the maintenance of adaptation than evolutionary change (Williams 1966; Gould 2001). Yet the ease with which we assign a creative and anticipatory role to natural selection such that it "favors a particular form" or "acts on its development" unmasks the need to explicitly consider, once again, the link between a trait's functional importance and its inheritance (Lewontin 1970; Van Valen 1982).

The central problem in considering such a link is envisioning the evolution of an organism–environment system that enables both allowance and accommodation of continuing environmental input through developmental or genetic induction, and at the same time, integration and homeostatic stability of already evolved adaptive structures. The connection between generation and maintenance of environmentally induced variation is a contentious issue, with both pan-selectionism and pan-environmentalism essentially predicting the same ultimate outcome—the eventual absence of developmental variation due either to complete surrendering to natural selection as a shaping force, where all extant species are adapted to their environments, or to organisms' evolving sufficient internal controls of their development to gain complete independence from the constraining effects of natural selection and environmental influence (Whyte 1965; Lewontin 1983; Matsuda 1987; Oyama 2000; Hall *et al.* 2004). Because maternal effects—epigenetic influences of parental phenotypes on offspring—combine developmental induction of functionally important changes in traits and their inheritance (Mousseau & Fox 1998b), they bridge the origin and evolution of organismal adaptability and thus provide an opportunity to examine the evolution of both adaptability and the connection between generation and maintenance of variation.

The enormous literature on maternal effects reflects the need for an accepted way to link the functional importance of traits and their inheritance by either integrating epigenetic effects into the statistical framework of population genetics (Kirkpatrick & Lande 1989; Atchley & Zhu 1997; Wade 1998; Räsänen & Kruuk 2007) or through empirical studies showing that maternal effects' producing rapid appearance of adaptive organismal forms are too common and important to be ignored or assigned secondary importance (Mousseau & Fox 1998a; Gilbert 2005). Here I emphasize three points. First, I suggest that because maternal effects on offspring development essentially represent environmental induction delivered (and prescreened) by a fully functioning phenotype (the mother), they might be particularly suitable for subsequent accommodation into preexisting developmental and genetic organismal systems. Second, I suggest that maternal effects are uniquely placed to produce discrete and functional novel phenotypes by acting at epigenetic thresholds of previously accumulated complex adaptations and by influencing hierarchical developmental processes. Third, I suggest that the epigenetic interactions associated with maternal effects can capture and retain a novel

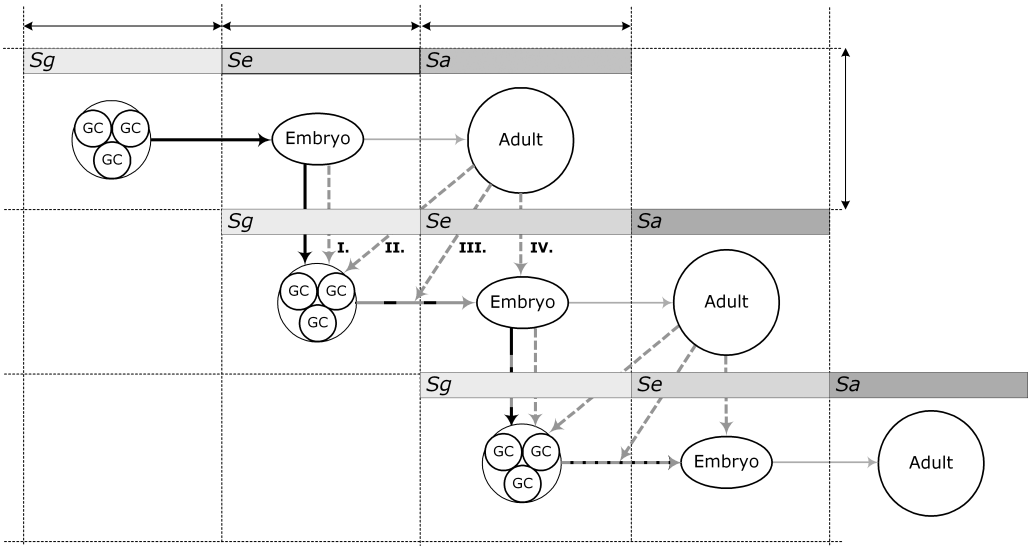

Figure 1. Continuity of phenotype and information between developmental stages (columns; GC [germ cells], embryo stage, and adult stage) and generations (rows) in birds. Each developmental stage is a subject to selection pressure (S_g, natural selection during GC stage; S_e, selection during embryo stage; S_a, selection during adult stage). Because the stages (horizontal double-headed arrows) and especially generations (vertical double arrow) can partially overlap, each developmental stage is a subject to a combination of selection pressures specific to its level and time. Black single-headed solid arrows show induction of germ cells from embryonic tissues; gray dashed arrows show epigenetic effects typically classified as "maternal effects." Solid gray lines indicate individual development. Figure illustrates the concept of phenotype-specific effects (integration of dashed gray and solid black arrows) on various developmental stages and corresponding accumulation, retention, and transmission of functionally important phenotypic modifications through maternal effects. Path I, effect of somatic tissues of embryo on induction and migration of germ cells; path II, maternal effects on germ cell apoptosis, induction of maturation, and meiosis; path III, maternally derived cytoplasmic gradients and protein production machinery; path IV, maternal allocation of growth-influencing substances and immunofactors, offspring provisioning, sexual imprinting, learning, and inheritance of maternal ecological and social environments.

adaptation for long evolutionary periods even before genetic determination of the new adaptation can evolve and with considerably lesser lethality than direct genetic effects. I will conclude by suggesting that maternal effects are not a special case but instead a transitory state in the spectrum of inheritance systems that consolidates and retains environment–organism developmental configurations in relation to their repeatability and predictability.

Maternal Effects in Development and Evolution

In birds, as in most vertebrates, germ cell induction and germline and somatic line differentiation occur after some somatic tissues are already formed, leading to close interaction between germ cells and somatic tissues (Buss 1988) and providing many opportunities for epigenetic influences on germ cell lineages (Box 1, Fig. 1), contrary to a widespread assumption of their isolation. Importantly, although each developmental stage—germ cells, embryo, and adult—is under its own natural selection, significant and variable overlap among the stages of different generations, in addition to direct and indirect epigenetic effects between the stages (Fig. 1), results in continuity of both phenotypes (e.g., somatic induction of germ cells) and information transferred along the phenotypic lineage (e.g., maternal modification of embryo development).

Although early epigenetic effects provide the most direct effects on germ cells (paths I and II in Fig. 1) and thus the strongest link to heritability, these effects are mostly nonspecific in relation to a particular adaptation and commonly represent either the outcome of maternal selection of germ cells or "developmentally entrenched" maternal effects that enable proper development of an embryo (see following discussion and Box 1). At the same time, whereas late epigenetic effects (paths III and IV in Fig. 1) do not directly affect germ cell lineages, they nevertheless represent the shortest link to inheritance of adaptive functions by connecting two functioning phenotypes at different temporal stages (Fig. 1, Box 1). The commonly documented greater expression of maternal effects at earlier developmental stages might be caused by decreased similarity of environments across developmental stages between maternal and offspring generations or an ontogenetic increase in offspring counteradaptations to maternal strategies. Further, because of the timing of their action, early maternal effects can facilitate emergence of novel developmental pathways by which they act, whereas later maternal effects mostly capitalize on existing developmental pathways.

Developmentally Contingent Maternal Effects and Generation of Variation

Maternal effects are expressed as offspring developmental variation before selection that the offspring will experience. Thus, depending on the interplay of maternal adaptations and strategies, offspring developmental responses to these strategies, and the intergenerational similarity of selection pressures, maternal effects can be categorized by four general patterns of developmental variation that they generate (Fig. 2). Each of these patterns can represent either a by-product of a maternal adaptation passively transferred to the offspring generation or an active maternal strategy to adjust the offspring's phenotype.

First, maternal effects can directionally adjust offspring development and thus accomplish overproduction of some phenotypes and underproduction of others (Fig. 2A). For maternal allocation of growth-affecting substances into an egg, such effects are proximately enabled by temporal changes of these substances in breeding females (Fig. 2). For example, the frequently documented temporal gradient in allocation of steroids and antioxidants into eggs can produce a corresponding directional shift in offspring phenotypes, such as changes in growth, morphology, and behavior (Schwabl 1996; Eising *et al.* 2001; Lipar 2001; Badyaev *et al.* 2002; Tschirren *et al.* 2007).

Second, avian maternal effects can produce divergent offspring phenotypes (Fig. 2B) either by influencing developmental thresholds of offspring development or by providing morph- or sex-specific resources (Burke 1989; Adkins-Regan *et al.* 1995; Velando 2002; Badyaev *et al.* 2006a). For example, sex-specific provisioning of developing embryos can result in pronounced sexual dimorphism of resultant phenotypes (Badyaev 2002a; Carere & Balthazart 2007). Mothers can allocate resources into eggs by temporally or spatially separating offspring by sex or by their growth requirements (Uller 2006) so that simultaneously growing neonates are exposed to different concentrations of maternal hormones (Young & Badyaev 2004; Badyaev *et al.* 2005, 2008).

Third, maternal effects can modify variance in offspring phenotypes (Fig. 2C), either by providing variable resources or by directly effecting integration of offspring development (e.g., Badyaev 2005a; Love *et al.* 2005). For example, females experiencing stressful environmental changes during reproduction often produce offspring with a greater range of morphological variation in skeletal structures than that of females breeding under normal environments (references in Badyaev 2005c). Finally, maternal effects can adjust offspring growth by compensating for other developmental inputs (Fig. 2D), such as poor environmental and

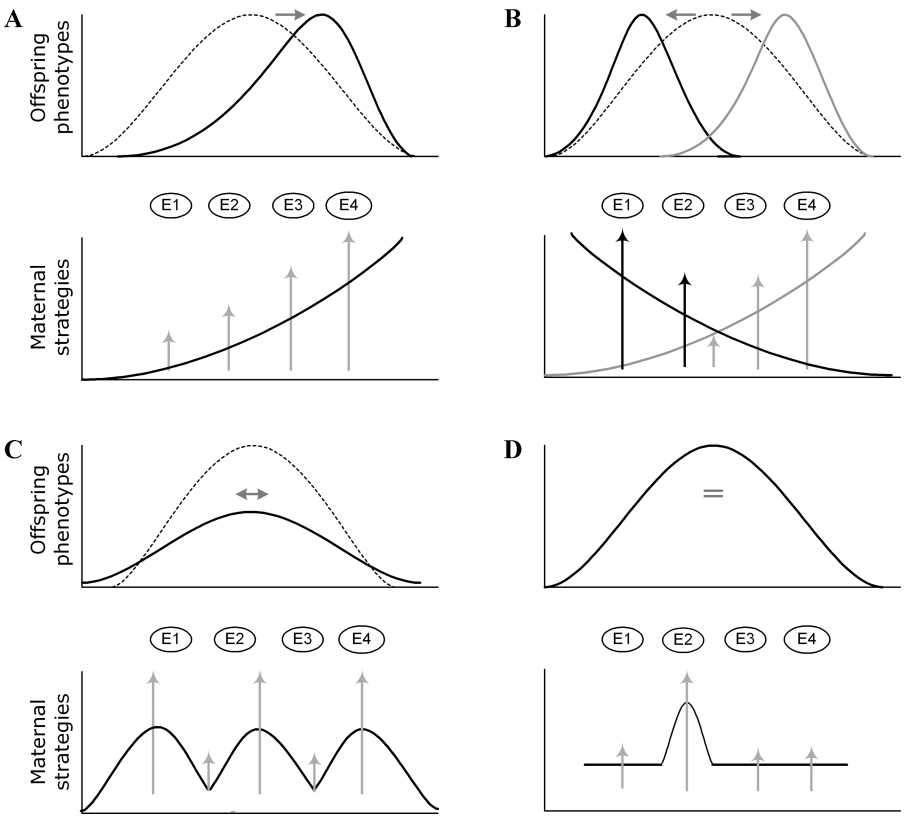

Figure 2. Maternal effects on offspring developmental variation. Each panel shows offspring distribution (upper graph) with (solid line) and without (dashed line) maternal effects (vertical arrows) on neonates (four ovals labeled E1–E4) and the corresponding distribution of maternal strategies (lower graph). Horizontal gray arrows show direction of distribution change. **(A)** Directional adjustment of offspring variation by temporal or spatial gradients in maternal strategies. **(B)** Maternal induction of offspring polymorphism by morph-specific allocation. **(C)** Maternal effect on offspring variability by variable allocation. **(D)** Compensatory maternal effects by neonate- or context-specific allocation.

social conditions, mate quality, parasites, and inbreeding effects (e.g., Gasparini *et al.* 2001; Tschirren *et al.* 2004; Michl *et al.* 2005; Badyaev *et al.* 2006b; Groothuis *et al.* 2006; Russell *et al.* 2007; Oh & Badyaev 2008).

Developmentally Entrenched Maternal Effects

Early maternal effects are often overlooked in evolutionary ecology and behavioral biology because, unlike context-dependent maternal effects that effectively link developmental induction, observed adaptive function, and inheritance, developmentally entrenched maternal effects often enable species-specific, "normal," and buffered early development of embryo (Gilbert 2006). For example, maternally derived mRNAs and cytoplasm gradients provide a developmental template for avian blastula differentiation and induce protein synthesis during early morphogenesis (e.g., Dworkin and Dworkin-Rastl 2005). At the same time, early maternal effects can result in substantial reorganization of offspring phenotypes and formation of novel developmental pathways—to a greater degree than later-acting maternal effects. Further, maternal transfer of steroids, immunofactors, and antioxidants is crucial for formation of receptor fields and sensitivities

(e.g., Gatford *et al.* 1998; Hassan *et al.* 2001), carotenoid biochemical pathways, and immunological pathways that at later stages affect an embryo's sensitivity to its own or maternal production of these substances (e.g., Surai & Speake 1998; Karadas *et al.* 2005; Badyaev 2006).

Maternal Effects and Inheritance of Functionally Important Traits

By linking developmental induction, adaptation, and inheritance, maternal effects provide important insight into the origin of adaptations; the maintenance of adaptability and evolvability; and most importantly, the link between micro- and macroevolutionary changes. A recent explosion of empirical evidence of maternal effects on each of these fundamental evolutionary processes and the diversity of epigenetic phenomena classified under maternal effects raises two questions: Are maternal effects a special case—ubiquitous but short-term adjusters that have only secondary importance for evolutionary change? Or are they a particularly clear example of a stage in the continuum of inheritance systems (Jablonka & Lamb 1995; Newman & Müller 2000; Oyama *et al.* 2001) that links function and inheritance by continuously accumulating, internalizing, and passing on the most consistent and adaptive organism–environment interactions? And does recent attention to the evolutionary effect of maternal effects represent a long-awaited shift toward appreciating the plurality of inheritance systems and their developmental linkage to evolutionary change (e.g., Oyama *et al.* 2001)—a view that might once again make evolutionary theory needed for progress in functional morphology, developmental biology, and behavioral ecology?

Importantly, maternal effects clarify the paradox of observed organismal adaptability that often occurs within a generation in contrast to the perceived rarity of appearance of novel adaptations that are heritable. The speed with which organisms mount novel biochemical, physiological, and behavioral responses to changing environmental conditions, and observations of direct transgenerational transfers of such responses (Mousseau & Dingle 1991; Mousseau & Fox 1998a; Gluckman *et al.* 2007), suggests that the widely held assumption of evolution as a slow process is due to the efficacy of stabilizing natural selection at maintaining stasis of existing adaptations (Simpson 1984) and building in genetic redundancies for the most recurrent organism–environment configurations (Wagner 2005) and is not due to the speed with which novelties can appear (Schmalhausen 1969). By modifying both offspring developmental variation and selection pressures on this variation, maternal effects can be more effective in generating and retaining adaptive novelties than genetic inheritance systems (Schmalhausen 1938; empirical examples, Badyaev & Oh 2008), can stabilize and internalize the response to an external stimulus even after the stimulus ceases (argument from development, Baldwin 1896, 1902; West-Eberhard 2003; argument from quantitative genetics, Kirkpatrick & Lande 1989; argument from population biology, Ginzburg 1998), and can produce similar changes in multiple offspring phenotypes (West-Eberhard 1989; Jablonka & Lamb 1995). Thus, epigenetic interactions arising during modifications of established developmental systems by maternal effects can be an important and ubiquitous source of novelties.

By acting at developmental thresholds of complex and hierarchical developmental processes and bypassing conserved developmental stages and controls, maternal effects can facilitate production of diverse but functional phenotypes—long a puzzle in evolutionary biology (Goldschmidt 1940; West-Eberhard 2003; Reid 2007). Further, because maternal transference involves multiple phenotypes, both the transmission and receipt of the information requires evolved organismal systems suited for such transmission, receipt, and reaction (Rollo 1994; Jablonka 2002). The extent of coevolution of these phenotypes (Wolf &

Brodie 1998; Wolf & Wade 2001) and, thus, the evolutionary efficacy and eventual genetic determination of maternal effects depend on the recurrence and similarity of mother–offspring adaptations.

The ontogenetic predictability of components of organism–environment interactions might account for the evolutionary transfer from epigenetic to genetic inheritance systems, with epigenetic (e.g., maternal) inheritance of the least reliable and most contingent components and genetic inheritance of the most predictable (and often earliest ontogenetic) processes (Oyama 1988; Newman & Müller 2000; Badyaev 2007). In turn, successive accumulation of the most recurrent organism–environment configurations over many generations, accommodated by homeostatic systems and internalized by genetic determination, can account for contingency in organism–environment interactions that can be expressed when the present and past environmental conditions match (Chetverikov 1926; Schmalhausen 1938; Wimsatt 1986; Wagner 2003; Badyaev 2005b; Gilbert 2005; Young & Badyaev 2007). Depending on their placement in the continuum of such successive accumulations, maternal effects can act as either fine-scale and short-term adjusters of organismal forms or as entrenched and genetically determined developmental factors. Thus, studies of maternal effects can provide important insight into the evolution of organismal systems that enable both continuing environmental input and the maintenance of evolved adaptive structures and therefore bring us closer to a more realistic understanding of the processes accounting for life's complexity and diversity.

Box 1. Place and time of maternal effects.

In birds, germ cells are induced from epiblast cells during the first 20 h of development. After induction, the germ cells migrate individually from the induction site to the edge of the area pellucida, where they aggregate and divide mitotically (e.g., Fujimoto *et al.* 1976). By 24 h of development, when blood vessels reach the area pellucida, the germ cells enter these vessels and are carried by blood circulation across embryonic tissues, for several hours, toward the site of the future gonads. The germ cells then exit the blood vessels by squeezing between the cells of vessel walls and surrounding tissues and crawl toward the future gonad sites (e.g., Ginsburg & Eyalgiladi 1986; Kuwana & Rogulska 1999). The somatic tissues of the embryo, which are under strong maternal influences at this stage, are thought to exercise substantial control over induction, traveling, and movement of germ cells (e.g., Nakamura *et al.* 1988, 2007; Fig. 1, path I).

After the establishment of functioning gonads, somatic tissues of the embryonic and adult phenotype maintain a significant effect on an isolated population of germ cells (Fig. 1, path II) by inducing apoptosis in some germ cells and promoting others to further development and maturation (Gilbert *et al.* 1983; Bahr & Johnson 1984; Yoshimura *et al.* 1993; Johnson 2000, 2003). The mechanisms by which only some precursor cells are selected to respond to adult hormonal signaling are not known, but it is thought that different groups of germ cells can be induced for further development in different breeding contexts (e.g., Johnson 1996; Zakaria 1999). Further, maternal strategies can have direct effects on meiosis and mitosis of germ cells (reviewed in Rutkowska & Badyaev 2007), which along with mate choice constitutes an additional opportunity for maternal selection and modification of germ cells (Fig. 1, path II). Finally, the early development of an avian zygote and early morphogenesis are directed by cytoplasmic gradients and protein production machinery that are provided largely by maternal allocation (Olszanska & Malewska 1999; Olszanska *et al.* 2002; Fig. 1, path III).

Perhaps the most studied expression of avian maternal effects is maternal allocation of growth-affecting substances and immunofactors to developing embryos (Groothuis *et al.* 2005, 2008; Sockman *et al.* 2006; Fig. 1, path IV). These substances can influence offspring development either directly, by transferring developmental resources used in offspring development (e.g., lipids, immunofactors, carotenoids), or indirectly, by transferring substances that facilitate formation of an offspring's own production of developmental resources (Badyaev 2002b). Finally, maternal provisioning of offspring, sexual imprinting, learning, and inheritance of maternal ecological and social environment constitutes additional components of the effects of maternal phenotype on offspring (Jablonka & Lamb 1995; Mousseau & Fox 1998b; Qvarnström & Price 2001).

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Conflicts of Interest

The author declares no conflicts of interest.

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