

Review

Parental effects in ecology and evolution: mechanisms, processes and implications

Alexander V. Badyaev^{1,*} and Tobias Uller^{1,2}

¹*Department of Ecology and Evolutionary Biology, University of Arizona, Tucson, AZ 85721, USA*

²*Edward Grey Institute, Department of Zoology, University of Oxford, Oxford OX1 3PS, UK*

As is the case with any metaphor, parental effects mean different things to different biologists—from developmental induction of novel phenotypic variation to an evolved adaptation, and from epigenetic transference of essential developmental resources to a stage of inheritance and ecological succession. Such a diversity of perspectives illustrates the composite nature of parental effects that, depending on the stage of their expression and whether they are considered a pattern or a process, combine the elements of developmental induction, homeostasis, natural selection, epigenetic inheritance and historical persistence. Here, we suggest that by emphasizing the complexity of causes and influences in developmental systems and by making explicit the links between development, natural selection and inheritance, the study of parental effects enables deeper understanding of developmental dynamics of life cycles and provides a unique opportunity to explicitly integrate development and evolution. We highlight these perspectives by placing parental effects in a wider evolutionary framework and suggest that far from being only an evolved static outcome of natural selection, a distinct channel of transmission between parents and offspring, or a statistical abstraction, parental effects on development *enable* evolution by natural selection by reliably transferring developmental resources needed to reconstruct, maintain and modify genetically inherited components of the phenotype. The view of parental effects as an essential and dynamic part of an evolutionary continuum unifies mechanisms behind the origination, modification and historical persistence of organismal form and function, and thus brings us closer to a more realistic understanding of life's complexity and diversity.

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1. PARENTAL EFFECTS ON DEVELOPMENT ENABLE GENETIC EVOLUTION

As any metaphor, parental effects—that occur when the phenotype of an individual is affected by the phenotype or environment of its parents (Roach & Wulff 1987; Bernardo 1996; Mousseau & Fox 1998a)—mean different things to different scientists and in different areas of biology (Mousseau *et al.* 2009). This diversity of perspectives is amply demonstrated by the contributions to this theme issue. To some, parental effects represent discrete static components of variance in phenotypes (e.g. Wolf & Wade 2009)—a view originating from the statistical framework of quantitative genetics. Reflecting its focus on optimality in organismal form and function, behavioural ecologists commonly treat parental effects as an adaptation (e.g. Crean & Marshall 2009; Harris & Uller 2009), while an extension of this view in evolutionary ecology considers parental effects to be an evolved compromise between responses of parental and offspring generations to natural selection (e.g. Duckworth 2009; Russell & Lummaa 2009). In ecology and population biology, the

focus on ecological succession, niche construction and population cycling emphasizes the inheritance and time-lag components of parental effects (e.g. Donohue 2009; Inchausti & Ginzburg 2009; Plaistow & Benton 2009), whereas animal physiology and developmental biology often focus on parental effects' role in transgenerational transfer of essential developmental templates and resources (e.g. Brown & Shine 2009) as well as maintenance of organismal homeostasis. Studies explicitly dealing with establishing links between the origin of adaptation and evolutionary change consider parental effects a stage in an evolutionary cycle connecting initial phenotypic retention of adaptive changes and their eventual genetic determination (Badyaev 2009).

Such a diversity of perspectives is expected from the biological reality of the composite nature of parental effects, but it also highlights a lack of agreement about their place in the evolutionary cycle. For example, parental effects are most commonly treated as an evolved adaptive outcome of natural selection—a static direct bridge that provides a discrete channel of transmission of functions and environments between parental and offspring phenotypes and whose configuration and placement are subject to natural selection. An effect of such a discrete and static channel of

* Author for correspondence (abadyaev@email.arizona.edu).

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transmission is then assessed in statistical partitioning of phenotypic variance. This perspective is problematic for several reasons. First, the view of parental effects as an evolved and static pattern obscures their transient nature as a *developmental process* and is not consistent with new findings on the interchangeability and interrelatedness of epigenetic and genetic mechanisms of which they consist (e.g. Allis *et al.* 2007). Parental effects are crucial aspects of development not because they contain evolved adaptive instructions about phenotypes, but because they have an impact (i.e. an effect) on developing phenotypes. The extent of this impact is a function of the reliability and availability of the repertoire of developmental resources transferred or reconstructed by parental effect processes. In other words, parental effects on development *enable* evolution by natural selection by reliably transferring the developmental resources needed to reconstruct and modify genetically inherited components of ontogenies in the offspring environment. Second, the impact of parentally transferred developmental resources itself has an ontogeny, such that 'sensitivity' of developing offspring to parentally transferred developmental resources needs to be constructed and can evolve, resulting in the variable expression of parental effects both in individual development and over evolutionary time.

Whereas most of the attention has been paid to variation in the outcomes of parental effects on offspring phenotype and the similarity between offspring and parents, here we focus on the mechanisms behind these effects. We specifically emphasize three points. First, we highlight the distinction between parental effects that, by providing developmental templates or essential resources, are involved in the *reconstruction* of developmental niches and pathways (i.e. 'developmentally entrenched' parental effects) versus parental effects that capitalize on the existing developmental pathways to *modify* the developmental niche (i.e. 'context-specific' parental effects). We are specifically interested in establishing an evolutionary link between these two classes of parental effects. Second, we discuss two contrasting aspects of parental effects in evolution by natural selection: on the one hand, parental effects can enable rapid evolution of adaptations by sustaining and regulating complex ontogenies across generations, yet, on the other hand, their modifications of offspring ontogenies can impose strong constraints on diversification and adaptability when the environment of offspring and parental generations differ. Finally, we address the relationship between parental effects and evolution of heredity with specific focus on the mechanisms and conditions under which organismal adaptations generate hereditary variation.

2. PARENTAL EFFECTS AND CONSTRUCTION OF THE DEVELOPMENTAL NICHE

(a) *Mechanisms of parental effects*

Parental effects construct the developmental niche of offspring by transferring or creating developmental resources that can (i) enable reliable implementation of genetically encoded contingencies, (ii) maintain trans-generational transfer of developmental variation and

thus increase phenotypic similarity between parent and offspring generations, (iii) induce novel variation in offspring in response to the conditions present in parental generations, and (iv) modify development to expose previously accumulated variability and hitherto unexpressed developmental pathways. These general outcomes can be accomplished by any of the three general categories of transmission between parental and offspring generations: (i) germ cells to germ cells (path I, [figure 1](#), e.g. epigenetic marking of germ cells' chromatin, sex-specific genomic imprinting; [Narasimha *et al.* 1997](#); [Kimmins & Sassone-Corsi 2005](#); [van de Lavoie *et al.* 2006](#)), (ii) somatic tissues to germ cells (path II, [figure 1](#), e.g. somatic effects on gametogenesis, germ cell apoptosis and transfer of organelles, migration, mitosis and meiosis; [Nakamura *et al.* 1988](#); [Barber *et al.* 1991](#); [Karagenc *et al.* 1996](#); [Johnson 2003](#); [Bekaert *et al.* 2004](#); [Hashimoto *et al.* 2004](#); [Renault *et al.* 2004](#); [Platonov & Isaev 2006](#); [Rutkowska & Badyaev 2008](#)), and (iii) somatic tissues to somatic tissues (paths III and IV, e.g. effects of hormones, nutrients, transfer of symbionts and immunodefence factors, transfer of environment created or modified by parental activity; [Odling-Smee *et al.* 2003](#); [Turner 2004](#); [Weaver *et al.* 2004](#); [Moran 2007](#); [Groothuis & Schwabl 2008](#)). Here, we address three outstanding issues in the evolution of parental effects: (i) causes of differential prevalence of particular paths of parental effects across taxa and environments ([figure 1](#)), (ii) the historical relationship between parental effects that evolve by natural selection and those that emerge as a result of development, and (iii) evolutionary transitions between the paths of parental effects.

Prevalence of a particular channel in the construction of the offspring developmental niche depends on (i) the duration and discreteness of each stage in [figure 1](#), (ii) the extent of temporal offset between generations, (iii) similarity in developmental resources between the sexes, and (iv) similarity of environment between the stages and generations (see §3; [Oyama 1988](#); [Jablonka *et al.* 1995](#); [Badyaev 2005a](#); [Uller 2008](#)). For example, in species such as plants and fungi that have late or continuous induction of germ cells from somatic tissues, the induction of changes in somatic tissues that become germ cells could be present across a lifetime, while in vertebrates it is often limited to a short period of early embryonic induction of germ cells from somatic tissues ([Buss 1988](#); [Extavour & Akam 2003](#)). Greater developmental offset between generations ([figure 1](#)) should reduce the possibility of identical somatic modifications in both generations and thus lead to lesser fidelity in the reconstruction of the same developmental niche across generations. In addition, sex specificity in developmental resources can greatly limit parental effects on the offspring generation (reviewed in [Badyaev 2002](#); [Carere & Balthazart 2007](#)). Both developmental offset between the generations and sex specificity in parental effects contribute to modification and diversification of offspring phenotypes and both can generate strong natural selection to minimize the limiting effects of sex specificity or developmental offset between the generations ([Rossiter 1998](#); [Uller 2003, 2006](#)).

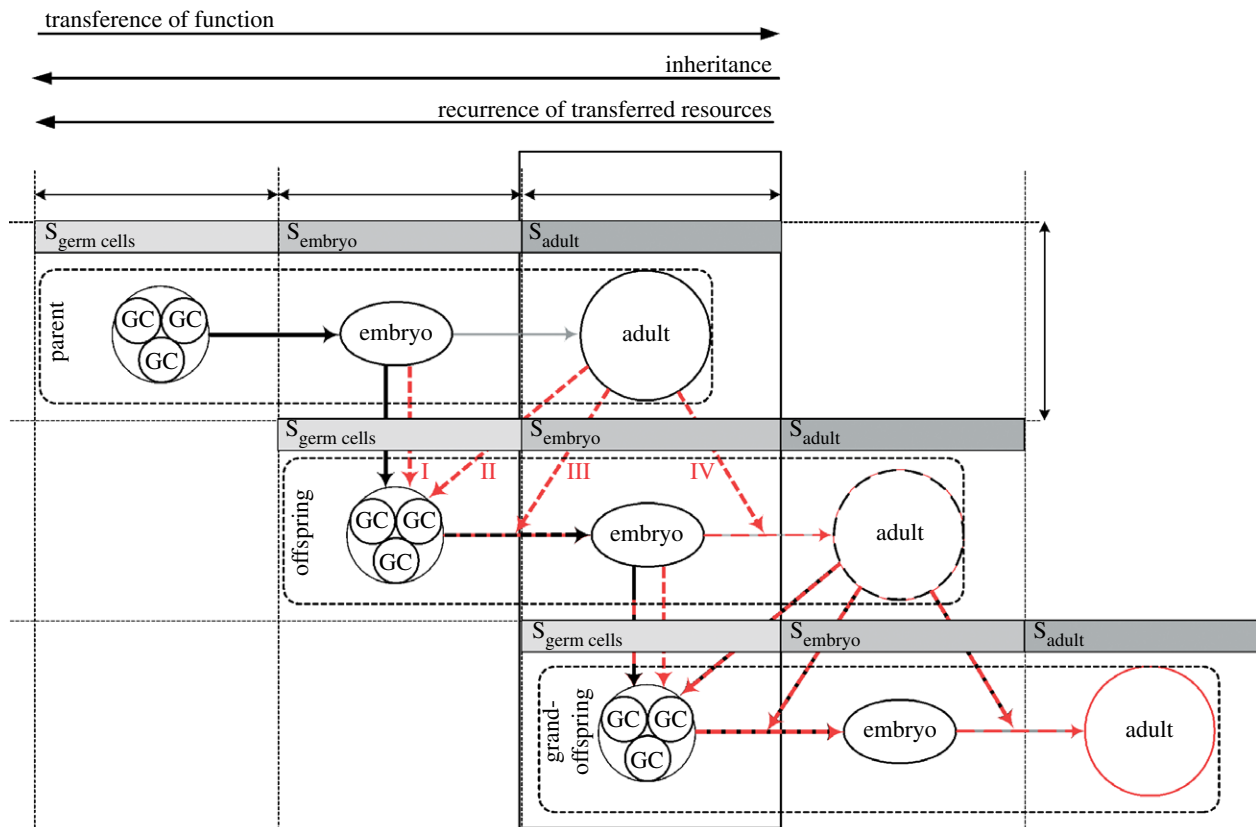


Figure 1. Conceptual illustration of continuity of phenotype and information between developmental stages (columns; germ cells (GC), embryo stage and adult stage) and generations (rows). Each developmental stage is a subject to selection pressure ($S_{\text{germ cells}}$, natural selection during GC stage; S_{embryo} , selection during embryo stage; S_{adult} , selection during adult stage). Developmental offset between generations results in similar selection pressures (black rectangle shows similar selection on function (adult stage), development (embryo stage) and germ cell induction (germ cell stage) in three overlapping generations). Black single-headed solid arrows show induction of germ cells from embryonic tissues, and red dashed arrows show epigenetic effects typically classified as parental effects. Solid grey lines indicate individual development. The figure also illustrates the concept of phenotype-specific effects (integration of dashed red and solid black arrows) on various developmental stages and the corresponding accumulation, retention and transmission of functionally important phenotypic modifications through parental effects. Overlap between developmental stages (e.g. germ cells and embryo) will result in the overlap of paths of parental effects. Transference of adaptive function mostly involves late ontogeny somatic tissue transmissions, while the importance of genetic inheritance decreases with ontogenetic decrease in the predictability of parentally transferred resources. Path I is parental effects on germ cells (including germ cell-to-germ cell and somatic tissue-to-germ cell effects), and path II shows somatic tissue-to-germ cell effects. Path III—somatic tissue-to-somatic tissue effects on offspring development, including templating and priming parental effects, and path IV includes somatic tissue-to-somatic tissue parental effects on offspring growth and development, including provisioning, sexual imprinting, learning and inheritance of parental ecological and social environments (see text for examples of parental effects for each path). Adapted from Badyaev (2008).

Paths of parental effects also differ in the extent to which they are suited to reconstruct an *evolved* specific adaptation versus induce *novel* phenotypic and genetic variation, i.e. in their contribution to the link between 'continuity of phenotype' versus 'continuity of information' (figure 1) in the evolution of parental effects (Jablonka 2002; Badyaev 2008). Later acting, somatic tissue-to-somatic tissue parental effects (paths III and IV, figure 1) might have greater involvement in transgenerational transference of novel adaptive variation (i.e. transference of 'function') because they represent developmental induction pre-screened and shaped by functioning (e.g. maternal) somatic tissues. In addition, because of the necessary temporal offset between the generations, parental effects can influence progressively earlier developmental processes and channel offspring's developmental processes to effectively reconstruct functional phenotypes. Both of these characteristics make somatic tissue-to-somatic

tissue parental effects well suited for short-term modifications of offspring phenotype (Jablonka & Lamb 1995; Schlichting & Pigliucci 1998; Pigliucci 2001; West-Eberhard 2003; Räsänen & Kruuk 2007). Owing to the complexity and abundance of genetic variance in the pre-existing developmental systems underlying such parental effects, they are good candidates for phenotypic accommodation of novel developmental variation and, over evolutionary time, for eventual genetic assimilation (Baldwin 1902; Schmalhausen 1938). However, the coevolution of offspring and parental adaptations in somatic tissue-to-somatic tissue transmissions is constrained by limited inheritance of late ontogeny modifications, especially in fluctuating environments (see §3, figure 1). By contrast, earlier acting germ cell-to-germ cell or somatic tissue-to-germ cells parental effects are less suited for transference and reconstruction of novel adaptive variation, but by providing templates and

through priming effects on offspring development, they can strongly facilitate continuity of a species-specific developmental niche of offspring.

Parental effects can be classified by their outcomes and developmental targets into three general categories: (i) developmentally entrenched parental effects that represent highly coevolved parent–offspring adaptations that consistently produce species-specific developmental dynamics in most species-specific environments, (ii) context-specific parental effects that modify offspring ontogeny and expose or induce developmental variation specific to a particular environmental context, and (iii) ‘passive’ parental effects that under a stressful environment (capitalizing on their role as developmental regulators of offspring growth) expose previously unexpressed developmental variation in offspring ontogeny. A central question is whether these paths are time-specific adaptations that fine-tune an evolved form to a particular environmental context or whether they are stages in an evolutionary continuum of inheritance systems that retain, accumulate and pass on the most recurrent organism–environment associations, so that developmentally entrenched parental effects are an evolved form of context-specific (and thus less recurrent) or passive parental effects.

(b) Parental effects as reconstruction of the developmental niche: entrenched parental effects

Species-specific phenotypes are as much a product of species-specific environments of development as they are of species-specific genotypes, and parental effects play a crucial role in constructing such environments. Some of these parental effects, such as maternally derived RNAs, organelles, cytoplasmic gradients and symbionts, can be transferred directly to influence developmental variation in offspring, and some parental effects in this category provide a developmental template and resources for early embryonic morphogenesis. However, direct transfer of resources is not confined to early ontogeny—transfer of species-specific environment modified by parental activity (such as plant succession, direct inheritance of environment, social rank or parental resources) could occur throughout organismal lifetime (Fairbanks 1996; Avital & Jablonka 2000; Jablonka 2001; McComb *et al.* 2001; Donnell *et al.* 2004; Dłoniak *et al.* 2006). Other parental effects, such as early transfer of steroids and antioxidants, are crucial for the formation of receptor fields and sensitivities, as well as priming of the offspring’s own receptors for subsequent reaction to self-synthesized products (Gatford *et al.* 1996; Lucas *et al.* 1996; Lung *et al.* 1996; Surai & Speake 1998; Karadas *et al.* 2005). Additional developmentally entrenched parental effects operate through highly predictable, developmental stage-specific signalling between parental and offspring phenotypes (e.g. time-specific effects of maternal hormones on DNA methylation or offspring somatic growth; Adkins-Regan *et al.* 1995; Meaney 2001; Rapp & Wendel 2005).

Precise stage specificity of these developmentally entrenched parental effects illustrates their highly evolved recurrent nature. Priming effects on offspring form and function are a particularly interesting example

because they involve modulation of parental–offspring adaptation in relation to the costs of de novo versus facilitated development in different environments (Badyaev 2005a, see §4). More generally, such developmentally entrenched and highly reliable parental effects play an important role in the construction of developmental pathways that later enable context-specific parental effects on offspring phenotypes.

(c) Parental effects as modification of the developmental niche: context-specific parental effects

Variation in the reliability and availability of parentally transferred resources can result in the lack of transgenerational persistence of some phenotypes and is a powerful force for the induction, transference and inheritance of novel developmental variation. Such introduction of novel variation can operate through all three main channels (germ cells to germ cells, somatic tissue to germ cells and somatic tissue to somatic tissue), although introduction of adaptive (i.e. context dependent) developmental variation usually involves functional soma (e.g. maternal phenotype) and thus are often limited to somatic tissue-to-germ cells and somatic tissue-to-somatic tissue channels (e.g. Fleming *et al.* 2002; Jablonka 2002; Badyaev 2008). By contrast, diversifying and inducing parental effects seem to be distributed across the routes of transmission and reconstruction; when reliability or availability of parentally transferred resources change (including as a result of changes in other components of phenotype or environment), components of parental effects themselves become a powerful source of natural and sexual selection (see §3, Qvarnström & Price 2001; Zeh & Zeh 2005). Here, we partition context-dependent parental effects by their outcomes into two categories: diversifying and directional effects on offspring phenotypes.

First, variation in repertoire and availability of developmental resources transferred by parental effects can increase variance in offspring phenotypes. Proximally, such outcomes can be a result of (i) developmental offset between generations or of sex or morph specificity of parental effects (and thus mismatch between parental strategy and effects; Lachmann & Jablonka 1996; Piersma & Drent 2003; Carere & Balthazart 2007), (ii) parental modification of developmental niche associated with a change in environments between generations (Atchley *et al.* 1991; Clark & Galef 1995; Gil *et al.* 1999; Rhees *et al.* 1999; Agrawal 2002; Whittingham & Schwabl 2002; Donohue *et al.* 2005; Plaistow *et al.* 2006; Uller 2006), (iii) direct and indirect parental effects on developmental integration of offspring ontogeny and resulting exposure and repatterning of novel phenotypic and genetic variation (Schlichting 2003; Badyaev 2005a), including through the effect of stress hormones on the induction of behavioural and morphological variation in offspring (Sibly & Calow 1989; Denver 1999; Boyce & Ellis 2005), and (iv) direct parental effects on the elevation in mutation and recombination rates and direction of their phenotypic outcomes (Jablonka & Lamb 1995; Imasheva 1999).

Second, when the environment of breeding is similar over long time scales, recurrent parental effects (i.e. recurrent maternal transference of context-specific

developmental resources) can lead to directional exaggeration of some components of offspring phenotype. For example, maternal transference of stress hormones is commonly associated with diversifying effects on offspring development (McCormick 1998; Uller & Olsson 2006). However, when a stressor is recurrent, maternal transference of stress hormones can induce precise and directional effects on offspring development (Boorse & Denver 2004; Love & Williams 2008). Similarly, directional variation in offspring phenotype can be produced by a consistent gradient in repertoire or availability of maternally transferred resources, such as variation in offspring size owing to systematic variation in hormone or nutrients allocation during gametogenesis or consistent seasonal variation in nutrients passed to offspring (Miao *et al.* 1991; Cordero *et al.* 2001; Williams *et al.* 2004). In addition, when variation in maternally transferred resources is sex or morph specific (either in availability or in repertoire), it can facilitate production of discrete distributions of offspring phenotypes. For example, parental effect on offspring growth is a powerful factor in the evolution of sexual size dimorphism (e.g. Ono & Boness 1996; Guégan *et al.* 2000; reviewed in Badyaev 2002). More generally, developmental offset between the generations enables parental effects to act at developmentally conserved regulatory stages that often harbour a diversity of previously accumulated complex adaptations (Goldschmidt 1940; West-Eberhard 2003), thereby facilitating rapid production of discrete phenotypes.

3. SELECTION ON PARENTAL EFFECTS

Parental transfer of developmental resources to offspring capitalizes on both the evolved sensitivity of offspring growth to these resources and the ability of offspring phenotype to use these resources in ontogeny (Rossiter 1996; Mousseau & Fox 1998a; Uller 2008). In turn, exposure of developmental variation by parental effects to natural and sexual selection results in the accumulation of the most recurrent (and fit) organism–environment configurations. Crucial for the evolution of parental effects is the scale of environmental fluctuations in relation to the generation time that determines concordance of natural selection on parental and offspring strategies (Mousseau & Dingle 1991; Jablonka *et al.* 1995; Piersma & Drent 2003; Badyaev 2005b), such that selection on context-dependent parental effects can lead to their stabilization and the reliable formation of sets of traits in parents and offspring under specific conditions, some of which may be adaptive for parents, offspring or both.

The evolution of adaptive parental effects should be particularly rapid when environmental fluctuations do not last more than a few generations and when there is a strong correlation between maternal and offspring environments, which facilitates stabilization and canalization of context-dependent parental effects (Jablonka *et al.* 1995; Lachmann & Jablonka 1996; Galloway 2005; Marshall & Uller 2007; Uller 2008). Furthermore, stage- and generation-specific selection together with limited opportunity for the incorporation of ‘external’ environmental cues in some developmental stages (figure 1) may lead to capitalization on

the pre-existing, developmentally entrenched parental effects to enable context-dependent offspring development (e.g. Young & Badyaev 2007). For example, parental effects often involve organization of major life-history traits early in life when offspring sensory organs are poorly developed, such as initiation of dormancy, diapause or dispersal (Mousseau & Fox 1998a; Massot & Clobert 2000; Donohue 2009; Duckworth 2009).

Selection on parental effects combines life stages of two generations, which generates scope for antagonistic selection across generations (i.e. parent–offspring conflict; Trivers 1974; Godfray 1995; Parker *et al.* 2002). Consequently, selection on parents might favour strategies that reduce offspring fitness and vice versa (Scholl *et al.* 1994; Haig 1996; Zeh & Zeh 2000; Crespi & Semeniuk 2004). The evolutionary dynamics of parental effects under parent–offspring conflict should depend on the relative costs and benefits of a particular set of strategies to the interacting individuals, the strength and consistency of selection on those strategies, and mechanistic aspects of interactions that influence whether or not one of the generations can impose their strategy on the other (Price 1998; Müller *et al.* 2007; Uller 2008). This coevolution of parental and offspring traits results in the formation of phenotypic and genetic covariance between parents and offspring (Wolf & Brodie 1998; Smiseth *et al.* 2008), with the most consistent and recurrent configurations ultimately producing developmentally entrenched parental effects that are part of species-specific normal development (Badyaev 2007, 2008).

4. A NOVEL PERSPECTIVE ON PARENTAL EFFECTS IN EVOLUTION

Through their transfer of developmental resources needed to reconstruct or modify offspring ontogeny, parental effects combine elements of developmental variation, natural selection and inheritance, and these processes can generate contrasting patterns depending on the context-specific relationship between them. As this review shows, parental effects can generate novel developmental variation or prevent its expression, generate natural selection or retard it through matching developmental and functional periods of two generations, enhance heredity by increasing similarity between ancestors and descendants and enabling inheritance of adaptive modifications, or reduce it through diversifying effects on development. Thus, the study of parental effects calls our attention to the complexity of causes and influences in development and evolution that go well beyond genetic inheritance and natural selection and emphasizes the importance of a deeper understanding of developmental dynamics of life cycles.

We think that a novel perspective on the evolution of parental effects is emerging. Parental effects can be viewed as a stage in an evolutionary continuum—a composite entity that capitalizes on the functional and developmental offset between the generations, and continuously retains and reconstructs the most reliable organism–environment configurations (Schmalhausen 1938; Newman & Müller 2000; Badyaev 2007; Callebaut *et al.* 2007). Under this perspective, initially

emergent or stress-induced parental effects continuously give rise to short-term context-dependent parental effects, which, depending on the reliability of transferred developmental resources and recurrence of environmental context, can eventually become developmentally entrenched, species-specific parental effects and, over evolutionary time, a part of genetically determined components of development. The degree of environmental fluctuation is a key factor in this process, such that the effects of stable, essentially constant over a wide range of species-specific conditions, internal environments become a characteristic of normal species-specific development and are no longer detectable as 'maternal effects' in statistical models.

Under this perspective, ongoing evolutionary transitions between the stages of parental effects can be interrupted such that a particular context-dependent parental effect can be stabilized and canalized by natural selection, limiting its variation and resulting in its long-term persistence on a particular set of parental and offspring traits in a particular set of circumstances. Similarly, a build-up of adaptive parental effects on offspring phenotype (such as epigenetic marking of genes in offspring germ line or an expansion of hormone receptor fields in offspring somatic tissues) can be reset and erased by a stressor, resulting in adaptively neutral parental effects that expose novel developmental variance.

Parental effects combine a bewildering diversity of phenomena—from epigenetic modification of activity and structure of genes in germ cells, to hormonal induction of novel morphological and behavioural variation, to transference of immunological factors produced as a result of exposure to pathogens, to behavioural inheritance of novel traditions, modified song dialects, food and mate preferences, and to ecological inheritance of modified environments (Jablonka & Lamb 1995; Mousseau & Fox 1998b; West-Eberhard 2003; contributions in this issue). Some of these effects act early in ontogeny, while others require sufficient development of offspring to have an effect (e.g. cultural inheritance of song dialect). Both of these classes of parental effects can be developmentally entrenched or 'context dependent', and the perspective outlined here, with its emphasis on the reconstruction of all aspects of offspring phenotype and plurality of inheritance systems, provides a mechanism for their evolution. Thus, this perspective brings forward the original notion in evolutionary theory that organismal functioning itself generates aspects of hereditary variation (Darwin 1859) and builds a continuum of this variation from short-term within-generation physiological and behavioural responses to environmental change to genetic inheritance of the most persistent adaptations (Oyama 2000; Müller & Newman 2003).

The main advantage of such a perspective is that it makes explicit the link between within-generation modification of phenotype and among-generation change, origination of novel variation and its maintenance, and, more generally, connects adaptation and evolutionary change (e.g. Badyaev 2008). Generational offset in development and function in relation to the external environment links generation

of developmental variation, its selection and inheritance (e.g. overlap between stage-specific selection pressures of different generations in figure 1). This highlights parental effects as an important source of evolutionary diversifications and innovations of modern organisms: parental effects can generate and retain adaptive novelties and produce coordinated changes in multiple offspring phenotypes simultaneously. Owing to these features, Badyaev (2009) suggested that parental effects are an illustration of the Baldwin effect—the process by which non-heritable developmental accommodation of novel inputs, which increase an organism's fit in its current environment, can become internalized and affect the course of evolution (Baldwin 1902; Schmalhausen 1969)—a perspective that, if correct, puts parental effect processes at the forefront of diversification and innovation in modern organism.

What this perspective makes clear is that the composite nature of parental effects phenomena makes it difficult to extrapolate processes and causes from their effect on offspring development or to assign a greater causal role to only some components of parental effects when all are required for normal development. What are needed to test this perspective empirically are the studies that place parental effects processes in an explicitly historical context by (i) examining evolutionary transference in their mechanisms, paths and effects in relation to recurrence of environments and (ii) examining the relationship between genetic and epigenetic variation in the development of organismal form and function in relation to such recurrence. By providing essential developmental resources for the reconstruction of past development, parental effects link the continuity of phenotype and continuity of information in evolutionary cycles. In doing so, they determine the direction of evolution—'the direction of the ontogenetic accommodations of the earlier generations' (Baldwin 1902)—and thus seamlessly integrate development and evolution—a major and long-awaited step in evolutionary theory.

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REFERENCES

- Adkins-Regan, E., Ottinger, M. A. & Park, J. 1995 Maternal transfer of estradiol to egg yolk alters sexual differentiation of avian offspring. *J. Exp. Zool.* **271**, 466–470. (doi:10.1002/jez.1402710608)
- Agrawal, A. A. 2002 Herbivory and maternal effects: mechanisms and consequences of transgenerational induced plant resistance. *Ecology* **83**, 3408–3415. (doi:10.2307/3072089)
- Allis, C. D., Jenuwein, T., Reinberg, D. & Caparros, M. L. (eds) 2007 *Epigenetics*, Cold Spring Harbor, NY: Cold Spring Harbor Laboratory Press.
- Atchley, W. R., Logsdon, T., Cowley, D. E. & Eisen, E. J. 1991 Uterine effects, epigenetics, and postnatal skeletal development in the mouse. *Evolution* **45**, 891–909. (doi:10.2307/2409697)
- Avital, E. & Jablonka, E. 2000 *Animal traditions: behavioural inheritance in evolution*. Cambridge, UK: Cambridge University Press.

- Badyaev, A. V. 2002 Male and female growth in sexually dimorphic species: harmony, conflict, or both? *Comments Theor. Biol.* **7**, 10–33.
- Badyaev, A. V. 2005a Stress-induced variation in evolution: from behavioral plasticity to genetic assimilation. *Proc. R. Soc. B* **272**, 877–886. (doi:10.1098/rspb.2004.3045)
- Badyaev, A. V. 2005b Maternal inheritance and rapid evolution of sexual size dimorphism: passive effects or active strategies? *Am. Nat.* **166**, S17–S30. (doi:10.1086/444601)
- Badyaev, A. V. 2007 Evolvability and robustness in color displays: bridging the gap between theory and data. *Evol. Biol.* **34**, 61–71. (doi:10.1007/s11692-007-9004-5)
- Badyaev, A. V. 2008 Maternal effects as generators of evolutionary change: a reassessment. In *The year in evolutionary biology 2008* (eds C. D. Schlichting & T. A. Mousseau), pp. 151–161. New York, NY: Wiley-Blackwell.
- Badyaev, A. V. 2009 Evolutionary significance of phenotypic accommodation in novel environments: an empirical test of the Baldwin effect. *Phil. Trans. R. Soc. B* **364**, 1125–1141. (doi:10.1098/rstb.2008.0285)
- Baldwin, J. M. 1902 *Development and evolution*. New York, NY: Macmillan.
- Barber, D. L., Sanders, E. J., Aebersold, R. & Schneider, W. J. 1991 The receptor for yolk lipoprotein deposition in the chicken oocyte. *J. Biol. Chem.* **266**, 18 761–18 770.
- Bekaert, S., Derradji, H. & Baatout, S. 2004 Telomere biology in mammalian germ cells and during development. *Dev. Biol.* **274**, 15–30. (doi:10.1016/j.ydbio.2004.06.023)
- Bernardo, J. 1996 Maternal effects in animal ecology. *Am. Zool.* **36**, 83–105.
- Boorse, G. C. & Denver, R. J. 2004 Endocrine mechanisms underlying plasticity in metamorphic timing in spadefoot toads. *Integr. Comp. Biol.* **43**, 646–657. (doi:10.1093/icb/43.5.646)
- Boyce, W. T. & Ellis, B. J. 2005 Biological sensitivity to context: I. An evolutionary–developmental theory of the origins and functions of stress reactivity. *Dev. Psychopathol.* **17**, 271–301. (doi:10.1017/S0954579405050145)
- Brown, G. P. & Shine, R. 2009 Beyond size–number trade-offs: clutch size as a maternal effect. *Phil. Trans. R. Soc. B* **364**, 1097–1106. (doi:10.1098/rstb.2008.0247)
- Buss, L. W. 1988 Diversification and germ-line determination. *Palaeobiology* **14**, 313–321.
- Callebaut, W., Müller, G. B. & Newman, S. A. 2007 The organismic systems approach. Streamlining the naturalistic agenda. In *Integrating evolution and development. From theory to practice* (eds R. Sansom & R. N. Brandon), pp. 25–92. Cambridge, MA: MIT Press.
- Carere, C. & Balthazart, J. 2007 Sexual versus individual differentiation: the controversial role of avian maternal hormones. *Trends Endocrinol. Metab.* **18**, 73–80. (doi:10.1016/j.tem.2007.01.003)
- Clark, M. M. & Galef Jr, B. G. 1995 Prenatal influences on reproductive life history strategies. *Trends Ecol. Evol.* **10**, 151–153. (doi:10.1016/S0169-5347(00)89025-4)
- Cordero, P. J., Vinuela, J., Aparicio, J. M. & Veiga, J. P. 2001 Seasonal variation in sex ratio and sexual egg dimorphism favouring daughters in first clutches of the spotless starling. *J. Evol. Biol.* **14**, 829–834. (doi:10.1046/j.1420-9101.2001.00320.x)
- Crean, A. J. & Marshall, D. J. 2009 Coping with environmental uncertainty: dynamic bet hedging as a maternal effect. *Phil. Trans. R. Soc. B* **364**, 1087–1096. (doi:10.1098/rstb.2008.0237)
- Crespi, B. & Semeniuk, C. 2004 Parent–offspring conflict in the evolution of vertebrate reproductive mode. *Am. Nat.* **163**, 635–653. (doi:10.1086/382734)
- Darwin, C. 1859 *The origin of species by means of natural selection, or the preservation of favoured races in the struggle for life*. London, UK: John Murray.
- Denver, R. J. 1999 Evolution of the corticotropin-releasing hormone signaling system and its role in stress-induced phenotypic plasticity. *Ann. NY Acad. Sci.* **897**, 46–53. (doi:10.1111/j.1749-6632.1999.tb07877.x)
- Dloniak, S. M., French, J. A. & Holekamp, K. E. 2006 Rank-related maternal effects of androgens on behaviour in wild spotted hyenas. *Nature* **440**, 1190–1193. (doi:10.1038/nature04540)
- Donnell, D. M., Corley, L. S., Chen, G. & Strand, M. R. 2004 Caste determination in a polyembryonic wasp involves inheritance of germ cells. *Proc. Natl Acad. Sci. USA* **101**, 10 095–10 100. (doi:10.1073/pnas.0403625101)
- Donohue, K. 2009 Completing the cycle: maternal effects as the missing link in plant life histories. *Phil. Trans. R. Soc. B* **364**, 1059–1074. (doi:10.1098/rstb.2008.0291)
- Donohue, K., Dorn, L., Griffith, C., Kim, E., Aguilera, A., Polisetty, C. R. & Schmitt, J. 2005 Niche construction through germination cueing: life-history responses to timing of germination in *Arabidopsis thaliana*. *Evolution* **59**, 771–785. (doi:10.1554/04-655)
- Duckworth, R. A. 2009 Maternal effects and range expansion: a key factor in a dynamic process? *Phil. Trans. R. Soc. B* **364**, 1075–1086. (doi:10.1098/rstb.2008.0294)
- Extavour, C. G. & Akam, M. 2003 Mechanisms of germ cell specification across the metazoans: epigenesis and preformation. *Development* **130**, 5869–5884. (doi:10.1242/dev.00804)
- Fairbanks, L. A. 1996 Individual differences in maternal style: causes and consequences for mothers and offspring. *Adv. Study Behav.* **25**, 579–611. (doi:10.1016/S0065-3454(08)60343-5)
- Fleming, A. S., Kraemer, G. W., Gonzalez, A., Lovic, V., Rees, S. & Melo, A. 2002 Mothering begets mothering: the transmission of behavior and its neurobiology across generations. *Pharmacol. Biochem. Behav.* **73**, 61–75. (doi:10.1016/S0091-3057(02)00793-1)
- Galloway, L. F. 2005 Maternal effects provide phenotypic adaptation to local environmental conditions. *New Phytol.* **166**, 93–99. (doi:10.1111/j.1469-8137.2004.01314.x)
- Gatford, K. L. *et al.* 1996 Sexual dimorphism of circulating somatotropin, insulin-like growth factor I and II, insulin-like growth factor binding proteins, and insulin: relationships to growth rate and carcass characteristics in growing lambs. *J. Anim. Sci.* **74**, 1314–1325.
- Gil, D., Graves, J., Hazon, N. & Wells, A. 1999 Male attractiveness and differential testosterone investment in zebra finch eggs. *Science* **286**, 126–128. (doi:10.1126/science.286.5437.126)
- Godfray, H. C. J. 1995 Evolutionary theory of parent–offspring conflict. *Nature* **376**, 133–138. (doi:10.1038/376133a0)
- Goldschmidt, R. B. 1940 *The material basis of evolution*. New Haven, CT: Yale University Press.
- Groothuis, T. G. G. & Schwabl, H. 2008 Hormone-mediated maternal effects in birds: mechanisms matter but what do we know of them? *Phil. Trans. R. Soc. B* **363**, 1647–1661. (doi:10.1098/rstb.2007.0007)
- Guégan, J.-F., Teriokhin, A. T. & Thomas, F. 2000 Human fertility variation, size-related obstetrical performance and the evolution of sexual statute dimorphism. *Proc. R. Soc. B* **267**, 2529–2535. (doi:10.1098/rspb.2000.1316)
- Haig, D. 1996 Placental hormones, genomic imprinting, and maternal–fetal communication. *J. Evol. Biol.* **9**, 357–380. (doi:10.1046/j.1420-9101.1996.9030357.x)

- Harris, W. E. & Uller, T. 2009 Reproductive investment when mate quality varies: differential allocation versus reproductive compensation. *Phil. Trans. R. Soc. B* **364**, 1039–1048. (doi:10.1098/rstb.2008.0299)
- Hashimoto, Y., Maegawa, S., Nagai, T., Yamaha, E., Suzuki, H., Yasuda, K. & Inoue, K. 2004 Localized maternal factors are required for zebrafish germ cell formation. *Dev. Biol.* **268**, 152–161. (doi:10.1016/j.ydbio.2003.12.013)
- Imasheva, A. G. 1999 Environmental stress and genetic variation in animal populations. *Russ. J. Genet.* **35**, 343–351.
- Inchausti, P. & Ginzburg, L. R. 2009 Maternal effects mechanism of population cycling: a formidable competitor to the traditional predator–prey view. *Phil. Trans. R. Soc. B* **364**, 1117–1124. (doi:10.1098/rstb.2008.0292)
- Jablonka, E. 2001 The systems of inheritance. In *Cycles of contingency: developmental systems and evolution* (eds S. Oyama, P. E. Griffiths & R. D. Gray), pp. 99–116. Cambridge, MA: MIT Press.
- Jablonka, E. 2002 Information: its interpretation, its inheritance, and its sharing. *Philos. Sci.* **69**, 578–605. (doi:10.1086/344621)
- Jablonka, E. & Lamb, M. J. 1995 *Epigenetic inheritance and evolution: the Lamarckian dimension*. Oxford, UK: Oxford University Press.
- Jablonka, E., Oborny, B., Molnar, I., Kisdi, E., Hofbauer, J. & Czarán, T. 1995 The adaptive advantage of phenotypic memory in changing environments. *Phil. Trans. R. Soc. B* **350**, 133–141. (doi:10.1098/rstb.1995.0147)
- Johnson, A. L. 2003 Intracellular mechanisms regulating cell survival in ovarian follicles. *Anim. Reprod. Sci.* **78**, 185–201. (doi:10.1016/S0378-4320(03)00090-3)
- Karadas, F., Pappas, A. C., Surai, P. F. & Speake, B. K. 2005 Embryonic development with carotenoid-enriched eggs influences the post-hatch carotenoid status of the chicken. *Comp. Biochem. Physiol. B* **141**, 244–251. (doi:10.1016/j.cbpc.2005.04.001)
- Karagenc, L., Cinnamon, Y., Ginsburg, M. & Petite, J. N. 1996 Origin of primordial germ cells in the prestreak chick embryo. *Dev. Genet.* **19**, 290–301. (doi:10.1002/(SICI)1520-6408(1996)19:4<290::AID-DVG2>3.0.CO;2-4)
- Kimmins, S. & Sassone-Corsi, P. 2005 Chromatin remodeling and epigenetic features of germ cells. *Science* **434**, 583–589.
- Lachmann, M. & Jablonka, E. 1996 The inheritance of phenotypes: an adaptation to fluctuating environments. *J. Theor. Biol.* **181**, 1–9. (doi:10.1006/jtbi.1996.0109)
- Love, O. P. & Williams, T. D. 2008 The adaptive value of stress-induced phenotypes in the wild: effects of maternally-derived corticosterone on sex allocation, cost of reproduction and maternal fitness. *Am. Nat.* **172**, E135–E149. (doi:10.1086/590959)
- Lucas, A., Baker, B. A., Desai, M. & Hales, C. N. 1996 Nutrition in pregnant or lactating rats programs lipid metabolism in the offspring. *Br. J. Nutr.* **76**, 605–612. (doi:10.1079/BJN19960066)
- Lung, N. P., Thompson, J. P., Kollias, G. V., Olsen, J. H., Zdziarski, J. A. & Klein, P. A. 1996 Maternal transfer of IgG antibodies and development of IgG antibody responses by blue and gold macaw chicks (*Ara ararauna*). *Am. J. Vet. Res.* **57**, 1157–1161.
- Marshall, D. & Uller, T. 2007 When is a maternal effect ‘adaptive’? *Oikos* **116**, 1957–1963. (doi:10.1111/j.2007.0030-1299.16203.x)
- Massot, M. & Clobert, J. 2000 Processes at the origin of similarities in dispersal behavior among siblings. *J. Evol. Biol.* **13**, 707–719. (doi:10.1046/j.1420-9101.2000.00202.x)
- McComb, K., Moss, C., Durant, S. M., Baker, L. & Sayialel, S. 2001 Matriarchs as repositories of social knowledge in African elephants. *Science* **292**, 491–494. (doi:10.1126/science.1057895)
- McCormick, M. I. 1998 Behaviorally induced maternal stress in a fish influences progeny quality by a hormonal mechanism. *Ecology* **79**, 1873–1883. (doi:10.2307/176695)
- Meaney, M. J. 2001 Maternal care, gene expression, and the transmission of individual differences in stress reactivity across generations. *Annu. Rev. Neurosci.* **24**, 1161–1192. (doi:10.1146/annurev.neuro.24.1.1161)
- Miao, S. L., Bazzaz, F. A. & Primack, R. B. 1991 Effects of maternal nutrient pulse on reproduction in two colonizing *Plantago* spp. *Ecology* **72**, 586–596. (doi:10.2307/2937198)
- Moran, N. A. 2007 Symbiosis as an adaptive process and source of phenotypic complexity. *Proc. Natl Acad. Sci. USA* **104**, 8627–8633. (doi:10.1073/pnas.0611659104)
- Mousseau, T. A., Uller, T., Wapstra, E. & Badyaev, A. V. 2009 Evolution of maternal effects: past and present. *Phil. Trans. R. Soc. B* **364**, 1035–1038. (doi:10.1098/rstb.2008.0303)
- Mousseau, T. A. & Dingle, H. 1991 Maternal effects in insects: examples, constraints, and geographical variation. In *The unity of evolutionary biology* (ed. E. C. Dudley), pp. 745–761. Portland, OR: Dioscorides Press.
- Mousseau, T. A. & Fox, C. W. 1998a The adaptive significance of maternal effects. *Trends Ecol. Evol.* **13**, 403–407. (doi:10.1016/S0169-5347(98)01472-4)
- Mousseau, T. A. & Fox, C. W. (eds) 1998b *Maternal effects as adaptations*. Oxford, UK: Oxford University Press.
- Müller, G. B. & Newman, S. 2003 *Origination of organismal form: beyond the gene in developmental and evolutionary biology*. Cambridge, MA: MIT Press.
- Müller, W., Lessells, C. M., Korsten, P. & von Engelhardt, N. 2007 Manipulative signals in family conflict? On the function of maternal yolk hormones in birds. *Am. Nat.* **169**, E84–E96. (doi:10.1086/511962)
- Nakamura, M., Kuwana, T., Miyayama, Y. & Fujimoto, T. 1988 Extragonadal distribution of primordial germ-cells in the early chick-embryo. *Anat. Rec.* **222**, 90–94. (doi:10.1002/ar.1092220113)
- Narasimha, M., Barton, S. C. & Surani, M. A. 1997 The role of the paternal genome in the development of the mouse germ line. *Curr. Biol.* **7**, 881–884. (doi:10.1016/S0960-9822(06)00377-0)
- Newman, S. A. & Müller, G. B. 2000 Epigenetic mechanisms of character origination. *J. Exp. Zool.* **288**, 304–314. (doi:10.1002/1097-010X(20001215)288:4<304::AID-JEZ3>3.0.CO;2-G)
- Odling-Smee, F. J., Laland, K. N. & Feldman, M. W. 2003 *Niche construction: the neglected process in evolution*. Monographs in Population Biology. Princeton, NJ: Princeton University Press.
- Ono, K. A. & Boness, D. J. 1996 Sexual dimorphism in sea lion pups: differential maternal investment, or sex-specific differences in energy allocation? *Behav. Ecol. Sociobiol.* **38**, 31–41. (doi:10.1007/s002650050214)
- Oyama, S. 1988 Stasis, development and heredity. In *Evolutionary processes and metaphors* (eds M.-W. Ho & S. W. Fox), pp. 255–274. New York, NY: Wiley.
- Oyama, S. 2000 *The ontogeny of information: developmental systems and evolution*. Science and cultural theory. Durham: Duke University Press.
- Parker, G. A., Royle, N. J. & Hartley, I. R. 2002 Intrafamilial conflict and parental investment: a synthesis. *Phil. Trans. R. Soc. B* **357**, 295–307. (doi:10.1098/rstb.2001.0950)

- Piersma, T. & Drent, J. 2003 Phenotypic flexibility and the evolution of organismal design. *Trends Ecol. Evol.* **18**, 228–233. (doi:10.1016/S0169-5347(03)00036-3)
- Pigliucci, M. 2001 *Phenotypic plasticity: beyond nature and nurture*. Baltimore, MD: John Hopkins University Press.
- Plaistow, S. J. & Benton, T. G. 2009 The influence of context-dependent maternal effects on population dynamics: an experimental test. *Phil. Trans. R. Soc. B* **364**, 1049–1058. (doi:10.1098/rstb.2008.0251)
- Plaistow, S. J., Lapsley, C. T. & Benton, T. G. 2006 Context-dependent intergenerational effects: the interaction between past and present environments and its effects on population dynamics. *Am. Nat.* **167**, 206–217. (doi:10.1086/499380)
- Platonov, E. S. & Isaev, D. A. 2006 Genomic imprinting in epigenetics of mammals. *Russ. J. Genet.* **42**, 1030–1042. (doi:10.1134/S1022795406090092)
- Price, T. 1998 Maternal and paternal effects in birds: effects on offspring fitness. In *Maternal effects as adaptations* (eds T. A. Mousseau & C. W. Fox), pp. 202–226. Oxford, UK; New York, NY: Oxford University Press.
- Qvarnström, A. & Price, T. D. 2001 Maternal effects, paternal effects and sexual selection. *Trends Ecol. Evol.* **16**, 95–100. (doi:10.1016/S0169-5347(00)02063-2)
- Rapp, R. A. & Wendel, J. F. 2005 Epigenetics and plant evolution. *New Phytol.* **168**, 81–91. (doi:10.1111/j.1469-8137.2005.01491.x)
- Räsänen, K. & Kruuk, L. E. B. 2007 Maternal effects and evolution at ecological time-scales. *Funct. Ecol.* **21**, 408–421. (doi:10.1111/j.1365-2435.2007.01246.x)
- Renault, A. D., Sigal, Y. J., Morris, A. J. & Lehmann, R. 2004 Soma-germ line competition for lipid phosphate uptake regulates germ cell migration and survival. *Science* **305**, 1963–1966. (doi:10.1126/science.1102421)
- Rhees, B. K., Ernst, C. A., Miao, C. H. & Atchley, W. R. 1999 Uterine and postnatal maternal effects in mice selected for differential rate of early development. *Genetics* **153**, 905–917.
- Roach, D. A. & Wulff, R. D. 1987 Maternal effects in plants. *Annu. Rev. Ecol. Syst.* **18**, 209–235. (doi:10.1146/annurev.es.18.110187.001233)
- Rossiter, M. 1996 Incidence and consequences of inherited environmental effects. *Annu. Rev. Ecol. Syst.* **27**, 451–476. (doi:10.1146/annurev.ecolsys.27.1.451)
- Rossiter, M. C. 1998 The role of environmental variation in parental effects expression. In *Maternal effects as adaptations* (eds T. A. Mousseau & C. W. Fox), pp. 112–134. Oxford, UK; New York, NY: Oxford University Press.
- Russell, A. F. & Lummaa, V. 2009 Maternal effects in cooperative breeders: from hymenopterans to humans. *Phil. Trans. R. Soc. B* **364**, 1143–1167. (doi:10.1098/rstb.2008.0298)
- Rutkowska, J. & Badyaev, A. V. 2008 Meiotic drive and sex determination: molecular mechanisms of sex ratio adjustment in birds. *Phil. Trans. R. Soc. B* **363**, 1675–1686. (doi:10.1098/rstb.2007.0006)
- Schlichting, C. D. 2003 Origins of differentiation via phenotypic plasticity. *Evol. Dev.* **5**, 98–105. (doi:10.1046/j.1525-142X.2003.03015.x)
- Schlichting, C. D. & Pigliucci, M. 1998 *Phenotypic evolution: a reaction norm perspective*. Sunderland, MA: Sinauer Associates.
- Schmalhausen, I. I. 1938 *Organism as a whole in individual development and history*. Leningrad, Russia: Academy of Sciences, USSR.
- Schmalhausen, I. I. 1969 *Problems of Darwinism*. Leningrad, Russia: Nauka.
- Scholl, T. O., Hediger, J. I., Schall, J. I., Khoo, C.-S. & Fischer, R. L. 1994 Maternal growth during pregnancy and the competition for nutrients. *Am. J. Clin. Nutr.* **60**, 183–188.
- Sibly, R. M. & Calow, P. 1989 A life-cycle theory of responses to stress. *Biol. J. Linn. Soc.* **37**, 101–116. (doi:10.1111/j.1095-8312.1989.tb01908.x)
- Smiseth, P. T., Wright, J. & Kölliker, M. 2008 Parent–offspring conflict and co-adaptation: behavioural ecology meets quantitative genetics. *Proc. R. Soc. B* **275**, 1823–1830. (doi:10.1098/rspb.2008.0199)
- Surai, P. F. & Speake, B. K. 1998 Distribution of carotenoids from the yolk to the tissues of the chick embryo. *J. Nutr. Biochem.* **9**, 645–651. (doi:10.1016/S0955-2863(98)00068-0)
- Trivers, R. L. 1974 Parent–offspring conflict. *Am. Zool.* **14**, 249–261.
- Turner, J. S. 2004 Extended phenotypes and extended organisms. *Biol. Philos.* **19**, 327–352. (doi:10.1023/B:BIPH.0000036115.65522.a1)
- Uller, T. 2003 Viviparity as a constraint on sex-ratio evolution. *Evolution* **57**, 927–931. (doi:10.1554/0014-3820(2003)057[0927:VAACOS]2.0.CO;2)
- Uller, T. 2006 Sex-specific sibling interactions and offspring fitness in vertebrates: patterns and implications for maternal sex ratios. *Biol. Rev.* **81**, 207–217. (doi:10.1017/S1464793105006962)
- Uller, T. 2008 Developmental plasticity and the evolution of parental effects. *Trends Ecol. Evol.* **23**, 432–438. (doi:10.1016/j.tree.2008.04.005)
- Uller, T. & Olsson, M. 2006 Direct exposure to corticosterone during embryonic development influences behaviour in an ovoviparous lizard. *Ethology* **112**, 390–397. (doi:10.1111/j.1439-0310.2006.01164.x)
- van de Lavoie, M.-C. *et al.* 2006 Germline transmission of genetically modified primordial germ cells. *Nature* **441**, 766–769. (doi:10.1038/nature04831)
- Weaver, I. C. G., Cervoni, N., Champagne, F. A., D'Alessio, A. C., Sharma, S., Seckl, J. R., Dymov, S., Szyf, M. & Meaney, M. J. 2004 Epigenetic programming by maternal behavior. *Nat. Neurosci.* **7**, 847–854. (doi:10.1038/nn1276)
- West-Eberhard, M. J. 2003 *Developmental plasticity and evolution*. Oxford, UK: Oxford University Press.
- Whittingham, L. A. & Schwabl, H. 2002 Maternal testosterone in tree swallow eggs varies with female aggression. *Anim. Behav.* **63**, 63–67. (doi:10.1006/anbe.2001.1889)
- Williams, T. D., Kitaysky, A. S. & Vezina, F. 2004 Individual variation in plasma estradiol-17 β and androgen levels during egg formation in the European starling *Sturnus vulgaris*: implications for regulation of yolk steroids. *Gen. Comp. Endocrinol.* **136**, 346–352. (doi:10.1016/j.ygcen.2004.01.010)
- Wolf, J. B. & Brodie, E. D. 1998 The coadaptation of parental and offspring characters. *Evolution* **52**, 299–308. (doi:10.2307/2411068)
- Wolf, J. B. & Wade, M. J. 2009 What are maternal effects (and what are they not)? *Phil. Trans. R. Soc. B* **364**, 1107–1115. (doi:10.1098/rstb.2008.0238)
- Young, R. L. & Badyaev, A. V. 2007 Evolution of ontogeny: linking epigenetic remodeling and genetic adaptation in skeletal structures. *Integr. Comp. Biol.* **47**, 234–244. (doi:10.1093/icb/icm025)
- Zeh, D. W. & Zeh, J. A. 2000 Reproductive mode and speciation: the viviparity-driven conflict hypothesis. *Bioessays* **22**, 938–946. (doi:10.1002/1521-1878(200010)22:10<938::AID-BIES9>3.0.CO;2-9)
- Zeh, J. A. & Zeh, D. W. 2005 Maternal inheritance, sexual conflict and the maladapted male. *Trends Genet.* **21**, 281–286. (doi:10.1016/j.tig.2005.03.006)