CHAPTER 14

Parental effects in development and evolution

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14.1 Introduction

In contrast to the traditional conception that the genome provides a blueprint for development (e.g. Mayr 1982; Williams 1992), parents transfer a variety of non-genetic resources and templates that are as necessary for development as the transfer of genes (Fig. 14.1). Even in organisms with minimal parental care, maternally derived mRNA and proteins accumulated in the egg during oogenesis regulate early development, such as embryonic axis and pattern formation (Pelegri 2003; Li et al. 2010; Gilbert 2010). Maternal influences on offspring development continue during embryonic life, with the egg usually providing all or most of the macro- and micronutrients that are necessary for normal development. Furthermore, viviparity has evolved repeatedly in several of the major phyla (Hogarth 1976; Reynolds et al. 2002) and is associated with a further range of dependencies of offspring development on maternal transfer of resources and signalling molecules (e.g. Fowden and Forhead 2009). Finally, mothers and fathers have a significant impact on their offspring well beyond birth in many species through resource provisioning and behavioural interactions, which play important roles in the ontogeny of species-typical phenotypes and individual phenotypic variation (Gottlieb 1997; Avital and Jablonka 2000; Maestripieri 2009; Michel 2011).

Under the definition employed in this book, all these mechanisms that contribute to the 'continuity of the phenotype' across generations (West-Eberhard 2003; Fig. 14.1) are parental effects—causal effects of the parental phenotype on offspring phenotype (Wolf and Wade 2009; Chapter 1). The observation that maternal transfer of macro- and micronutrients, behavioural interactions, and reconstruction of the ecological conditions in which development takes place are necessary for expression of functional phenotypes show that many parental effects are developmentally entrenched, that is the transfer of developmental templates and resources from parents to offspring form an integral part of species-typical development (West-Eberhard 2003; Badyaev 2008; Badyaev and Uller 2009). Parental effects not only form an entrenched part of offspring development, however; they may also provide a source for expression of novel phenotypic variation (West-Eberhard 2003; Badyaev 2008), influence population dynamics (Inchausti and Ginzburg 2009), be a significant generator of natural and sexual selection (Donohue 2009), affect the rate and direction of phenotypic evolution (Kirkpatrick and Lande 1989; Bonduriansky and Day 2009; Chapter 15), contribute to the persistence of induced phenotypes across generations (Odling-Smee et al. 2003; Jablonka and Raz 2009; Chapter 17), and enable trans-generational transmission of acquired functions or information (Boyd and Richerson 1985; Jablonka and Lamb 1995, 2005; Uller 2008; for simplicity, I use the term 'trans-generational' to include both parental effects that involve only two generations (often referred to as 'intergenerational') and those where the effects of past generations of phenotypes accumulate, interact, or remain stable across more than two generations). These examples emphasize the need for evolutionary theory to take seriously the many ways by which parents reconstruct the developmental niche for their offspring and make parental effects central to the integration of developmental and evolution-

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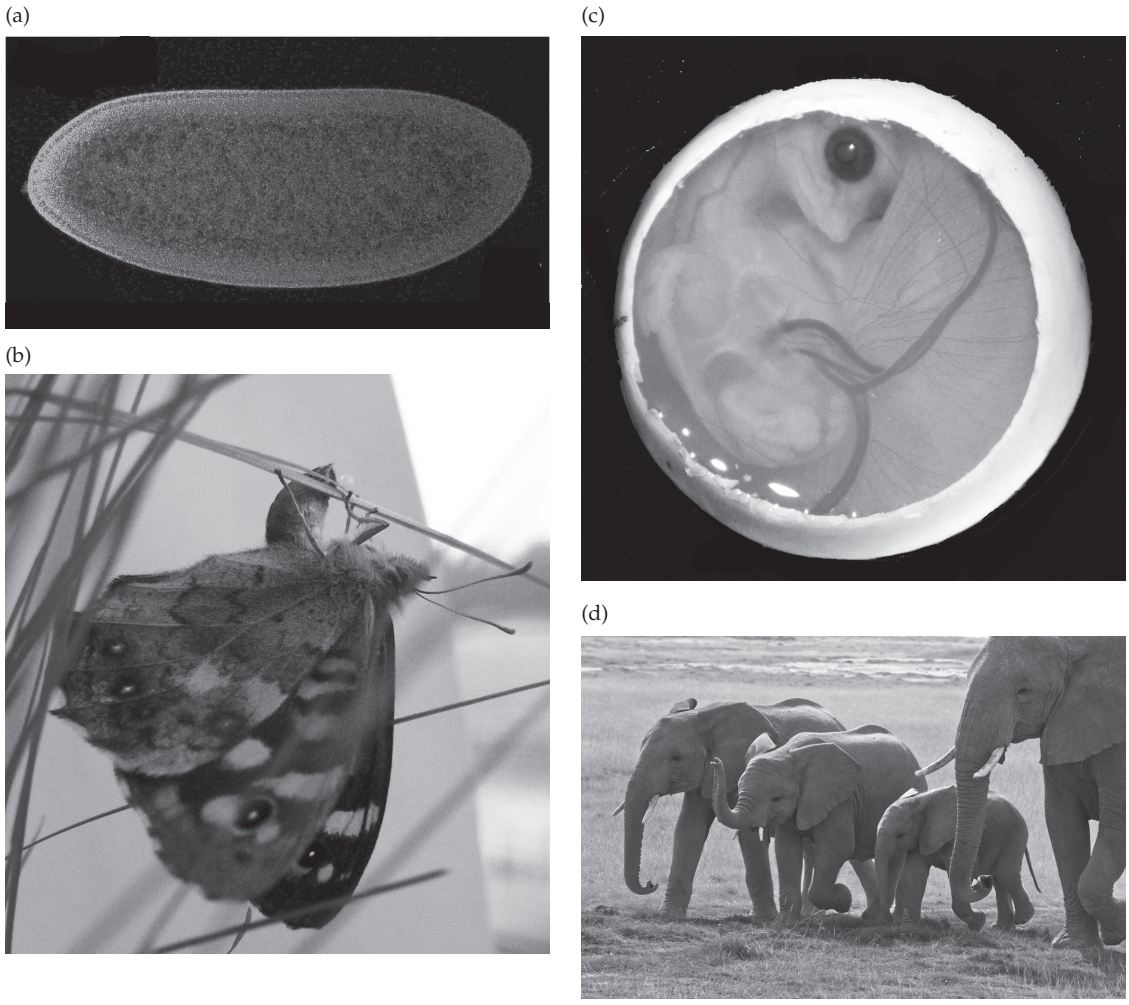

Figure 14.1 Parental effects—causal effects of the parental phenotype on offspring phenotype—are as fundamental to development as DNA. (a) All development starts with a responsive phenotype—an egg—produced by the parental phenotype. Early development occurs without expression of the offspring genome, as in maternal mRNA regulation of the anterior–posterior polarity in Drosophila embryos. (b) The parents also provide offspring with an ecological context for development, for example, by choosing where to lay the eggs. (c). Macro- and micronutrients in the egg of oviparous species, such as turtles, continue to nurse the embryo and contribute to its growth and differentiation. (d) Behavioural interactions between mothers and offspring after birth are necessary for the formation of species-typical behaviours expressed later in life in many social organisms. (Photo credits: (a): Stefan Baumgartner; (b): Casper Breuker; (c): Weiguo Du; (d): Joan Egert.)

ary biology (e.g. Jablonka and Lamb 2005; Badyaev 2008, 2009; Badyaev and Uller 2009; Bonduriansky and Day 2009; Odling-Smee 2010).

This chapter briefly covers three aspects of parental effects that are relevant to understanding their role in the evolution of parental care and offspring development (Fig. 14.2; see also Badyaev 2008, 2009 and Badyaev and Uller 2009). First, I will show how the mechanisms of parental effects can contribute to the generation of evolutionarily significant variation. I will argue that parental effects support the notion that phenotypic change may precede genetic change during adaptive evolution (Baldwin 1902; West-Eberhard 2003), and I suggest that parental effects are particularly well suited to addressing the role of developmental plasticity for

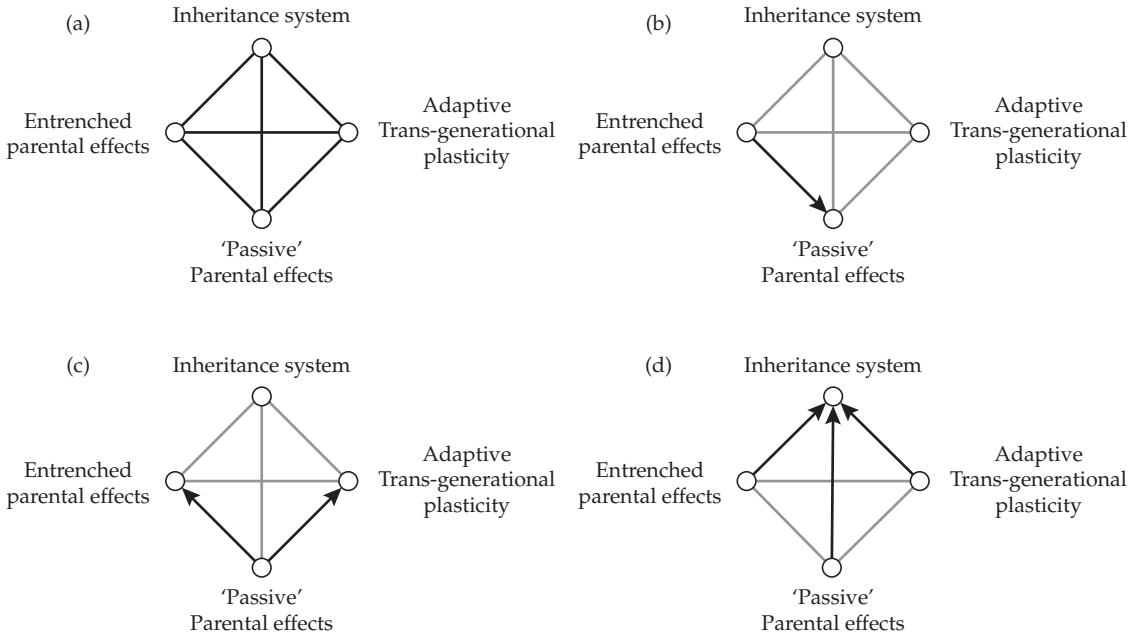

Figure 14.2 The mechanisms of parental effects can take on different 'roles' in development and evolution. (a) The open circles indicate four ways by which parental effects can contribute to the development and evolution of phenotypes. Each state may be connected to other states (black lines). Four of the possible transitions that are discussed in this chapter are shown in panels B–D. (b) The mechanisms of parental effects that contribute to species-typical development provide a source of induction of phenotypic variation via novel genetic or environmental input in the parental generations. The induced variants can be seen as 'passive' consequences of the evolved developmental reliance on parental phenotype. Nevertheless, such parental effects can influence the rate and direction of evolution by affecting the functionality of novel phenotypes and their recurrence (Sections 14.2 and 14.3). (c) Phenotypic variation expressed via parental effects is subject to natural selection, which, if variants are heritable, can result in the evolution of increased or reduced reliance on parental phenotype for normal development under species-typical developmental conditions via genetic assimilation (arrow pointing towards entrenched parental effects; Section 14.3.3), or, if the recurrence of more than one induced variant is sufficiently frequent, become stabilized by natural selection as conditionally expressed variants (arrow pointing towards adaptive trans-generational plasticity; Section 14.4). (d) Trans-generational plasticity can be seen as adaptive transmission of information across generations through a system of inheritance (e.g. chromatin-based systems, behaviours). However, if variants transmitted through non-genetic means become reliably reconstructed and sufficiently stable for natural selection to sort among their associated phenotypic effects, the mechanisms of parental effects that are involved in the replication of life cycles may evolve towards an inheritance system in a more strict sense that shares more features with the DNA-based system of inheritance (Box 14.1).

the origin of adaptive trait variation. Second, I will outline how the parental and offspring phenotypes that comprise parental effects evolve under natural selection and how this, under some circumstances, can lead to precise context-specific effects of the parental phenotype on offspring development in the form of adaptive trans-generational plasticity (Uller 2008). Third, I discuss the relationship between context-dependent parental effects and non-genetic 'systems of inheritance' (Jablonka and Lamb 2005), with the aim to provide an entry into the literature that explores the relationships between development, inheritance, and evolution from an information perspective. Finally, I provide a summary and some suggestions for how the evolutionary dynamics of parental effects can be further explored.

14.2 Parental effects and the origins of variation

Evolutionary change begins with developmental change, providing the phenotypic variation that is necessary for adaptive evolution. Developmental change, in turn, must begin with a phenotype that is responsive to novel genetic or environmen-

tal input. A description of the causes of evolution therefore requires an analysis of how existing mechanisms of development give rise to novel phenotypes (e.g. Mivart 1871; Gottlieb 1992; West-Eberhard 2003). The expression of phenotypic variation during development can be described as a two step process, by which novel genetic (via mutation, hybridization etc.) or environmental input is followed by accommodation of this input; that is, mutual adjustment of different parts that produces a functional phenotype (West-Eberhard 2003, 2005). Both genetic and environmental induction can contribute to evolutionarily relevant phenotypic variation since responses to novel environments often vary genetically between individuals, and hence can be heritable. Selection can therefore modify the regulation and form of genetically variable phenotypic accommodations over generations, a process referred to as genetic accommodation (West-Eberhard 2003; Moczek 2007). This perspective emphasizes that a complete understanding of adaptive evolution requires a description of both the developmental origin of adaptive phenotypes and the processes that result in an increase in the frequency of those phenotypes across generations. It also suggests a creative role of developmental plasticity in evolution since plasticity permits phenotypic accommodation and thus facilitates expression of novel, but functional, phenotypes in response to environmental change (West-Eberhard 2003).

Discussions of the role of developmental plasticity for expression of novel phenotypes tend to focus on the direct effect of genetic or environmental input on organisms within a single generation (e.g. Baldwin 1902; Wcislo 1989; Gottlieb 1992; West-Eberhard 2003, 2005; Moczek 2008; Pfennig et al. 2010; but see Badyaev 2009). However, parental effects allow maternal and paternal phenotypic accommodation to have carry-over effects on offspring development, thereby leading to expression of phenotypic variation in the following generation. Thus, genetic and environmental change affecting the parental generation may initiate evolutionary divergence in developmental trajectories (Badyaev 2008, 2009). For example, the reliance on maternally derived mRNA and proteins for the earliest stages in development implies that genetic or environmental modification of the regulation of oogenesis is involved in reorganization of developmental pathways (Sun et al. 2005; Minelli and Fusco 2010). Indeed, experimental studies of marine invertebrates suggest that both egg size and maternally derived factors (e.g. mRNA) that regulate embryonic development have contributed to evolutionary diversification of larval forms (Sinervo and McEdward 1988; Raff and Byrne 2006; Minelli and Fusco 2010).

The potential role of parental effects in the origin of novel variation is not restricted to regulation of early developmental patterning, but encompasses all parental influences on offspring phenotypes, including all forms of parental care (Chapter 1). In mammals, hormones of maternal origin play an important role in regulation of receptor densities, enzymes, growth factors, and other signalling molecules that are necessary for organ differentiation, including the brain (Fowden and Forehad 2009). Prenatal hormone exposure also plays a role in long-term regulation of gene expression via modification of patterns of DNA methylation (Weaver et al. 2004; Harris and Seckl 2011; Chapter 17). Variation in hormone exposure during development resulting from genetic variation between mothers or variation in maternal environment can cause short- and long-term physiological variation in the offspring, with concomitant effects on morphology, behaviour, and life-history. For example, prenatal exposure to high levels of glucocorticoids can reduce birth weight, cause hypertension, and increase the activity of the hypothalamus–pituitary–adrenal (HPA) axis, which is associated with changes in stress-related behaviours such as anxiety (reviewed in Meaney et al. 2007; Harris and Seckl 2011; Chapter 17). Experimental studies on fish, lizards, and birds suggest that many of the maternal effects observed in mammals are phylogenetically conserved and reflect shared developmental mechanisms among vertebrates (e.g. McCormick 1998; Uller and Olsson 2006; Love and Williams 2008).

Species-typical development also relies on maternal uptake and transmission of micronutrients directly from the environment. For example, development of the olfactory system in mammals requires olfactory stimuli obtained from the

amniotic fluid (Michel 2011). The composition of amniotic fluid is derived from the maternal plasma and is therefore directly influenced by maternal physiology and diet (Robinson and Méndez-Gallardo 2011). Consequently, maternal diet influences offspring response to potential food items by influencing the development of sensory neurons associated with particular olfactory receptors (Robinson and Méndez-Gallardo 2011; Todrank et al. 2011). Finally, behavioural interactions between parents and offspring are also a source of phenotypic novelties; offspring growing up in unusual family structures or under novel patterns of parental care may show changes in, for example, sociality, mate preferences, and parental behaviour (e.g. Bradshaw and Schore 2007; Hansen et al. 2010).

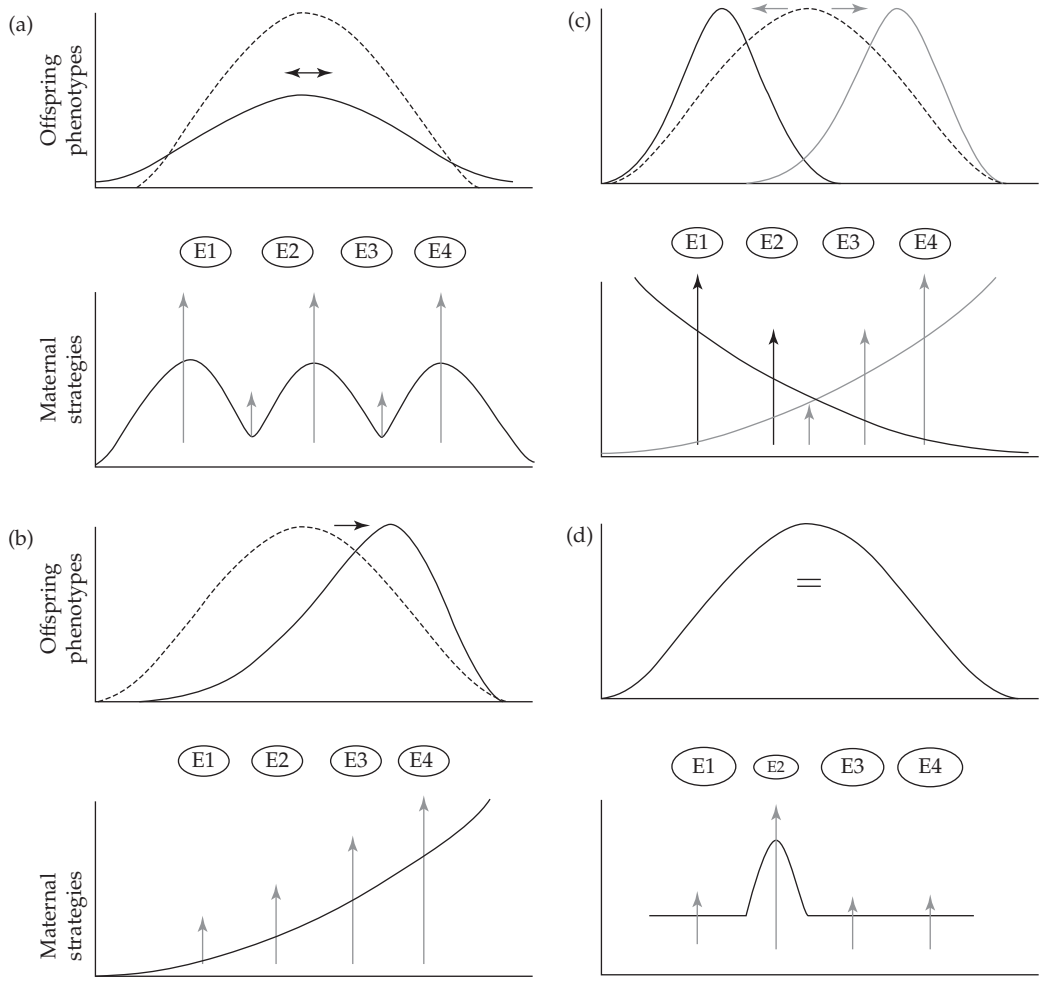

Figure 14.3 Parental effects in development. Each panel shows the distribution of offspring phenotypes (upper graphs) with (solid line) and without (dashed line) the distribution of parental effects (vertical arrows) on neonates (ovals labelled E1–E4). Horizontal grey arrows show the direction of change in the distribution of offspring phenotypes. (a) Variable parental transmission of developmental factors can increase variation in offspring phenotype. (b) Spatial or temporal variation in transmitted factors can cause directional change in offspring phenotype. (c) Morph-specific transmission can contribute to polymorphisms. (d) Context- or offspring-specific transmission can compensate for differences in developmental trajectories. (Redrawn from Fig. 2 in Badyaev 2008; reprinted with permission from the author and John Wiley & Sons.)

14.2.1 Patterns of phenotypic variation

These examples demonstrate how developmentally entrenched parental effects can contribute to modifications in the regulation and form of phenotypes. Badyaev (2008) has suggested that such parental induction can be captured in four ways (Fig. 14.3). First, parental effects may increase the variance in offspring phenotype (Fig. 14.3a). For example, maternal stress can result in increased variation in transfer of developmental resources among offspring with concomitant variation in offspring development (e.g. Badyaev 2005a). Increased variation among offspring may also result from maternal effects on the integration of developmental modules. Disruption of phenotypic integration results in greater variance in the connection of modules and thus greater among-individual variance in developmental outcomes (reviews in Hallgrímsson and Hall 2005).

Second, temporal or spatial variation in maternal transmission of resources can produce directional variation in offspring phenotypes (Fig. 14.3b). Such directional variation is common in birds, where egg size and yolk hormones often vary with laying order (Christians 2002; Groothuis et al. 2005). In several passerines, for example, environmental effects on ovarian activity and circulating levels of maternal hormones influence the accumulation of hormones by developing oocytes and results in within- and between-clutch variation in offspring phenotypes (e.g. Schwabl 1993; Badyaev et al. 2003). Highly divergent allocation of resources may also contribute to discontinuous phenotypic variation (Fig. 14.3c). A large number of polymorphisms depend on resource availability (e.g. Smith and Skúlason 1996), which provides substantial scope for maternal effects on morph expression in such systems. In spade-foot toads (*Spea multiplicata*), changes in maternal body condition under interspecific competition affect maternal egg investment and result in strong maternal effects on the development of resource-use polymorphism in the offspring (Pfennig and Martin 2009; Martin and Pfennig 2010). Furthermore, studies of mammals have shown that spatial or temporal variation in maternal resource allocation between sons and daughters contributes to morphological and behavioural divergence between the sexes (Moore 1995; Chapter 10). Thus, differential maternal allocation to offspring with different genotypes or developmental histories can exaggerate such differences and contribute to the expression of alternative phenotypes (Fig. 14.3c).

Finally, parental effects can constrain the production of novel phenotypes by limiting the effect of novel genetic and environmental input on offspring development (Fig. 14.3d). Female lizards are able to compensate for poor thermal conditions by adjusting their thermoregulatory behaviour or nest site choice. This can reduce the effect of ambient temperature on offspring development (Uller et al. 2011), maintain stasis in offspring reaction norms to temperature across climatic conditions (Doody et al. 2006), and may contribute to the build-up of cryptic genetic variation (which may prove important for a future response to selection; Schlichting 2008). Similarly, parental effects may compensate for genetic variation by genotype-specific allocation to offspring, which can limit the phenotypic effects of genetic variation and contribute to the resolution of constraints imposed by, for example, sexually antagonistic variation.

In summary, because development always relies on a parental phenotype, environmental or genetic changes to parent–offspring relationships can contribute—via phenotypic accommodation in both generations—to evolutionarily relevant variation. Parental effects can both facilitate expression of novel phenotypes and retard it, depending on to what extent parental accommodation of novel input results in differential transmission of resources in ways that influence pre-existing developmental mechanisms (Badyaev 2008).

14.3 Parental effects and adaptive evolution

The spread of a novel variant in a population partly depends on its fitness effects. Processes that positively affect the likelihood that novel genetic or environmental input will produce a functional phenotype should therefore promote adaptive evolution. However, even beneficial variants are likely to be lost by stochastic processes if they are rare. Thus, processes that increase recurrence of heritable

variation will also facilitate evolutionary change (West-Eberhard 2003). Parental effects can have a positive impact on both functionality and recurrence of novel phenotypes.

14.3.1 Parental effects can increase functionality of novel variation

Genetic or environmental induction of phenotypic variation via parental effects may initially be accommodated by a functional parental phenotype. Parental phenotypic accommodation of novel input should therefore reduce the risk of severe disruption of offspring development, and may even facilitate expression of functional phenotypes by capitalizing on pre-existing developmental mechanisms (Badyaev 2008). Although it may be tempting to interpret context-dependent parental effects as reflecting adaptations to past fluctuations in the environment (see Section 14.4), incorporation of recurrent environmental and parental components into development is a fundamental feature of evolution and is more likely when environments show low, rather than high, variation (Gottlieb 1992; West-Eberhard 2003; Lickliter and Harshaw 2011). This is because evolution of development capitalizes on resources that are reliably available in the environment. However, this reliance of development on maternally transmitted gene products and environmental components may promote biased responses to conditions not previously encountered and enable phenotypic responses to a broader range of conditions. For example, maternally transmitted immunoglobulins and other immune factors activate and regulate development of the offspring immune system with long-lasting consequences on, for example, B and T cell repertoires (Lemke et al. 2004). Maternal exposure to novel pathogens may therefore contribute to directional, and functional, change in offspring phenotype (e.g. resistance) in environments not previously encountered. Similarly, the integration of maternally derived substances in the neuro-anatomical development of olfactory organ in mammals (see Section 14.2 above) suggests that a change in diet can generate directional food preferences in the offspring, even if the mechanisms that are involved have not been selected as a system for transmission of information about food regimes between generations per se. Thus, despite the fact that directional, apparently adaptive, phenotypic change in novel environments is facilitated by a pre-existing developmental reliance on the maternal phenotype, this reliance need not have been selected for because it increases offspring fitness in *fluctuating* environments (see also Section 14.5 below). It could simply form a part of a developmentally entrenched mechanism that under most conditions contributes to development of a species-typical, invariant, phenotype.

14.3.2 Parental effects can increase recurrence of novel variation

Selection on rare variants is very inefficient as they are likely to be lost due to stochastic processes. Factors that promote recurrence of a novel phenotype should therefore increase the likelihood that the rare phenotype is being selected and, if it is heritable, facilitate adaptive evolution. This argument led West-Eberhard (2003) to conclude that environmentally induced phenotypes have greater evolutionary potential than those induced via mutation. Parental effects contribute to the recurrence of novel phenotypes in at least two ways. First, a rare genetic or environmental input can influence more than one individual if it is accommodated via a parent. Genetic variation, for example due to multiple paternity, allows recurrence in a diversity of genetic backgrounds and thus enhances the opportunity for genetic accommodation. Similarly, within-brood variation in a developmental context, for example due to laying order effects, may increase the probability of a favourable match between phenotype and selection (e.g. Badyaev 2005a), and thereby the likelihood that the rare variants persist and can spread.

Second, parental effects can contribute to persistence of induced phenotypes across several generations (Jablonka and Lamb 1995, 2005; West-Eberhard 2007; Fig. 14.4). For example, persistence of a novel food preference is facilitated by mechanisms that enable offspring to copy their parents' diet, which results in the incorporation of novel food types into development in each generation (Fig. 14.4a). Parental effects may

also allow environmentally induced phenotypic variation to be transmitted more or less stably down lineages via behavioural and epigenetic mechanisms even after the initial stimulus has ceased to exist (Fig. 14.4b). This is exemplified by research on the mechanisms of developmental plasticity and maternal care in rats (*Rattus norvegicus*) (Chapter 17). Cross-fostering of pups between mothers that direct high versus low levels of parental care towards their offspring (in the form of licking and grooming) showed that female offspring inherit the parental care behaviour of their foster mother, suggesting that maternal effects contribute to stability of between-lineage differences in maternal care.

The research on maternal care in rats emphasizes that the crucial element of trans-generational persistence of an environmentally induced phenotype is that the phenotype contributes to the reconstruction of the developmental niche, thus favouring its own expression (Fig. 14.4b). In the absence of germ-line transmission of induced variants (which does occur; see reviews by Youngson and Whitelaw 2008; Jablonka and Raz 2009; Fig. 14.4c), a limited period of parent–offspring interactions implies that parental effects must persist into adulthood to be maintained. For example, within- and trans-generational persistence of the effects of maternal care on pups have been linked to effects of maternal licking and grooming on the methylation status of the promoter regions of the estrogen receptor alpha and the glucocorticoid receptor genes, which remain stable throughout ontogeny and influence parental care in adulthood (Chapter 17). Stable inheritance of environmentally induced variants may often involve parental transmission of substances or behavioural interactions that affect epigenetic marks, which enables early environments to have long-lasting consequences via cellular epigenetic inheritance (Weaver et al. 2004; Gluckman et al. 2009; Chapter 17).

14.3.3 Parental effects and genetic accommodation

Environmental induction followed by reconstruction of the developmental niche via parental effects can initiate and maintain population differences without genetic divergence, as exemplified by dietary preferences in mammals (e.g. Avital and Jablonka 2000). However, if there is genetic variation in, for example, uptake, digestion, or circulation of novel odorants, or in offspring sensitivity to compounds circulating in the amniotic fluid (or in the milk), natural selection can fine-tune responses to local conditions and cause genetic divergence between populations. Mutation accumulation due to weak selection on rarely expressed alleles and costs associated with plastic responses can also contribute to further population divergence initiated by developmental plasticity (reviewed in Pfennig et al. 2010; Snell-Rood et al. 2010). This process in which changes in gene frequencies within populations is secondary to the origin of novel variation is referred to as genetic accommodation (West-Eberhard 2003). With respect to parental effects, genetic accommodation may occur in response to selection on the parental phenotype, offspring phenotype, or both, which should result in co-adaptation of parental and offspring phenotypes (Chapters 15 and 16; see also Section 14.4). Furthermore, initially deleterious effects can be eliminated via genetic accommodation (West-Eberhard 2003; Grether 2005). For example, the negative effects of maternal stress on offspring phenotype and fitness in mammals may gradually be reduced via selection on genetic variation in maternal stress response, transfer of corticosterone across the placenta, or offspring sensitivity to prenatal hormone exposure.

The evolution of integration of environmental input and offspring response capitalizes on pre-existing sensory systems in females (e.g. detection of photoperiod, or diet composition in the environment), physiological responses associated with reproduction (e.g. shared hormonal regulation of responses to environmental variation and breeding), and offspring sensitivity to maternal physiology (e.g. maternal hormones triggering expression of hormone receptors in the embryo) (Nijhout 2003; Badyaev 2009; Uller and Badyaev 2009). Changes in the environmental context of breeding, such as photoperiod, temperature, or food availability, will often be associated both with expression of novel variation in the offspring and selection on this variation, which enhances the scope for selection to effectively sort between phenotypes and hence

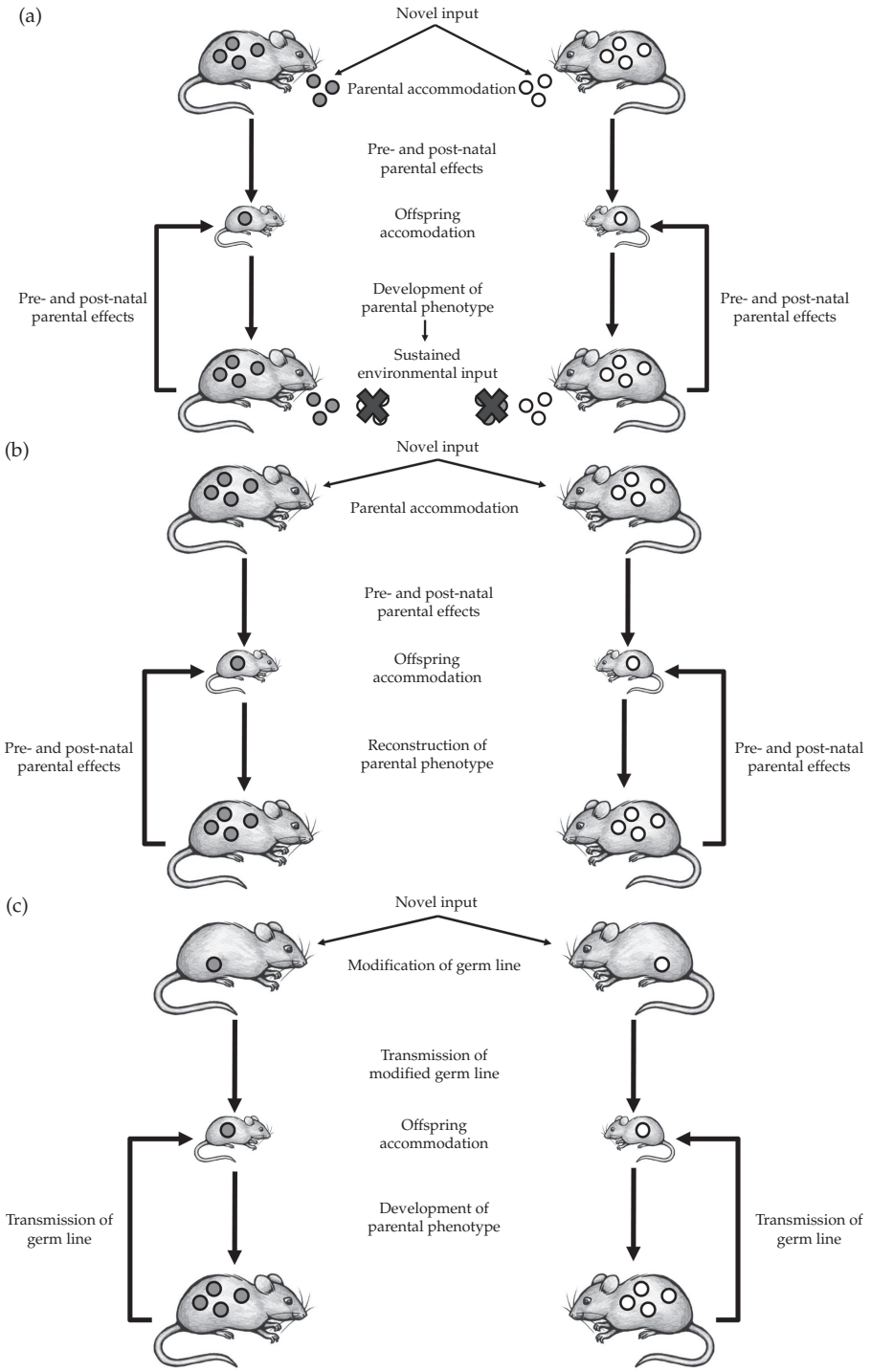

Figure 14.4 Parental effects can contribute to the recurrence of environmentally induced phenotypes. (a) An environmentally induced phenotypic change (a grey or white phenotype) can persist in a population because offspring from 'grey mothers' prefer 'grey environments' as adults and offspring from 'white mothers' prefer 'white environments' as adults. The induced phenotype only persists as long as all inducing environment persists. (b) An environmentally induced phenotypic change (a grey or white phenotype) can persist in a population even if the inducing factor is no longer is present in the population if the offspring develop a phenotype that reconstructs the parental effect that favoured its expression. (c) An environmentally induced phenotypic change can persist if it is transmitted via the germ line, either as a genetic variant or as an epigenetic variant that is not being erased during epigenetic reprogramming in the germ cells or during development.

increases the rate of evolution (West-Eberhard 2003; Badyaev 2005b; Badyaev 2009). The role of parental effects in evolution of local adaptation may be particularly important when the most reliable cue, like photoperiod, does not have a causal effect on offspring development unless it is mediated via parental responses.

In summary, parental effects facilitate development of functional phenotypes in response to novel input and increase the recurrence of those phenotypes by enabling cross-generational stability of environmentally induced variation. Both processes increase the potential for adaptive evolution and suggest substantial scope for environmentally induced variation to have evolutionary consequences via genetic accommodation. Furthermore, parental effects can enable persistence of induced phenotypes even if they are initially deleterious, which may result in genetic accommodation of the regulation of developmental processes to restore fitness.

14.4 Evolution of trans-generational plasticity

The evolution of developmentally entrenched parental effects can be seen as a process in which organisms accommodate and accumulate environmental input to pass on the most recurrent organism–environment configurations (Badyaev 2008, 2009; Badyaev and Uller 2009). Passive context-dependent parental effects may thus represent a transient period of phenotypic accommodation of environmental input that exposes phenotypic (and genetic) variation to selection. However, research in a wide range of disciplines—including behavioural and evolutionary ecology, evolutionary anthropology, and microbiology—emphasize that parental effects may also enable adaptive transfer of information about coming selective regimes across one or several generations (e.g. Feldman and Laland 1996; Mousseau and Fox 1998; Falkner and Falkner 2003; Gluckman et al. 2005; Jablonka and Lamb 2005; Uller 2008). Behavioural transmission of functional solutions to local environments from parents to offspring in humans is a familiar example, but similar processes also occur in organisms without sophisticated cognitive abilities. For example, the timing of germination in the herb *Campanulastrum americanum* depends on maternal light regime during seed production (Galloway 2005). Seeds from plants grown in light gaps tend to germinate in autumn and develop as annuals, whereas seeds from plants in shady conditions germinate in spring and develop as biennials. Because seeds tend to fall close to the maternal plant and light regimes are relatively stable across generations, but variable at the population level, adjustment of the timing of germination in relation to the maternal light environment is favoured over fixed or bet-hedging strategies (Galloway and Etterson 2007).

Maternal effects on seed germination can be seen as adaptive, trans-generational, plasticity (Mousseau and Fox 1998; Marshall and Uller 2007; Uller 2008). Furthermore, its similarity to behavioural transmission of adaptive strategies in animals shows that trans-generational plasticity can involve very different mechanisms and occur at different life-history stages (Fig. 14.1). This raises at least three questions. First, under what conditions can parental effects form an adaptive channel of transmission of information between generations? Second, are the mechanisms involved in adaptive trans-generational plasticity different from those of developmentally entrenched parental effects, and, finally, should they be seen as alternative systems of inheritance?

14.4.1 Adaptive evolution of trans-generational plasticity

In keeping with the standard framework of phenotypic plasticity (e.g. Schlichting and Pigliucci 1998), the evolution of trans-generational plasticity can be visualized as a set of two evolving reaction norms (Fig. 14.5). The parental phenotype may be responsive to some aspect of its environment by changing its morphology, physiology, or behaviour. Variation in the parental phenotype constitutes a fluctuating environment for the offspring, which is associated with a corresponding norm of reaction. Evolution of trans-generational plasticity is captured by the co-evolution of those two reaction norms so that a particular environmental context experienced by parents induces a particular phenotypic response in the offspring (Fig. 14.5).

Whether evolution of parental effects will be primarily driven by changes in the parental or offspring reaction norms is a question that has apparently received scant interest. However, the response is likely to depend on the relative amount of heritable variation, the genetic architecture of the phenotypes involved, and the relative strength of selection. If offspring plasticity is constrained, the offspring phenotype evolves only via evolution of the parental norm of reaction (i.e. via indirect genetic effects; Smiseth et al. 2008). Conversely, if parental plasticity is prevented from evolving, the evolution of parental effects reduces to evolution of within-generation phenotypic plasticity; the offspring reaction norm is expected to evolve to maximize fitness within the set of costs and constraints specified (reviewed in Berrigan and Scheiner 2004). Evolved parental strategies or 'passive' parental effects may impose selection on offspring development, thereby contributing to evolutionary diversification in developmental trajectories (Badyaev 2005a). For example, variation in egg size simultaneously affects offspring phenotype (e.g. size) and exercises selection on this phenotype, which provides substantial scope for evolution of alternative developmental strategies mediated via maternal effects (Badyaev 2005a; Pfennig and Martin 2009; Lancaster et al. 2010).

Making predictions regarding the conditions that promote evolution of trans-generational plasticity is currently hampered by the limited number of theoretical models, but should be subject to similar considerations as within-generation plasticity (Berrigan and Scheiner 2004) but framed within the context of the evolution of parental care (Smiseth et al. 2008). In particular the nature of fluctuating selection, the availability of cues, and benefits and costs of the transmission of information across generations for offspring and parents are expected to affect the evolution of trans-generational plasticity (Uller 2008). It can be useful to conceptualize the evolution of parental effects as evolution of developmental responses to different sources of input that carry information about coming selective regimes (Leimar et al. 2006; Fig. 14.5b, Box 14.1). Under this perspective, the parental phenotype is a source of information for the offspring when the parental phenotype correlates with current or future conditions experienced by the offspring (Shea et al. 2011; Box 14.1). Such correlations can arise in heterogeneous environments (e.g. Galloway 2005) or because of genetic or environmental variation in parental traits (e.g. Love & Williams 2008; Lancaster et al. 2010), both of which should favour evolution of trans-generational plasticity (Revardel et al. 2010; Shea et al. 2011). Assuming no parent–offspring conflict, we would expect selection to maximize information transfer between generations. Thus, not only should offspring evolve to adjust their phenotype according to the parental phenotype, but selection on parents may also favour transfer of developmental factors that are informative about the conditions that the offspring experience, or will experience. This should result in a tight integration between parental environment, parental phenotype, and offspring phenotype (Fig. 14.5c).

Empirical examples of potentially adaptive trans-generational plasticity mirror how parental phenotypes can modify offspring phenotypes more generally (see Fig. 14.3). Facultative diversifying maternal effects can be favoured in heterogeneous environments with different degrees of predictability (Crean and Marshall 2009). Directional changes in maternal allocation of androgens across the laying sequence in altricial birds might mitigate detrimental effects on hatching asynchrony (which is determined by parental onset of incubation; Groothuis et al. 2005). Discrete polyphenisms in insects also involve maternal effects. For example, the phase shift from solitary to gregarious morphs in locusts is initiated within generations but becomes progressively stronger across generations as a result of maternal transfer to the egg froth of a compound produced by the accessory glands (reviewed in Simpson and Sword 2009). Although the adaptive significance of trans-generational effect in locusts remains to be verified, it is possible that it might maximize the efficiency of phase shift in a gradually changing environment (Simpson and Sword 2009), conditions that potentially could favour parental effects that accumulate or persist across more than two generations (Jablonka et al. 1995).

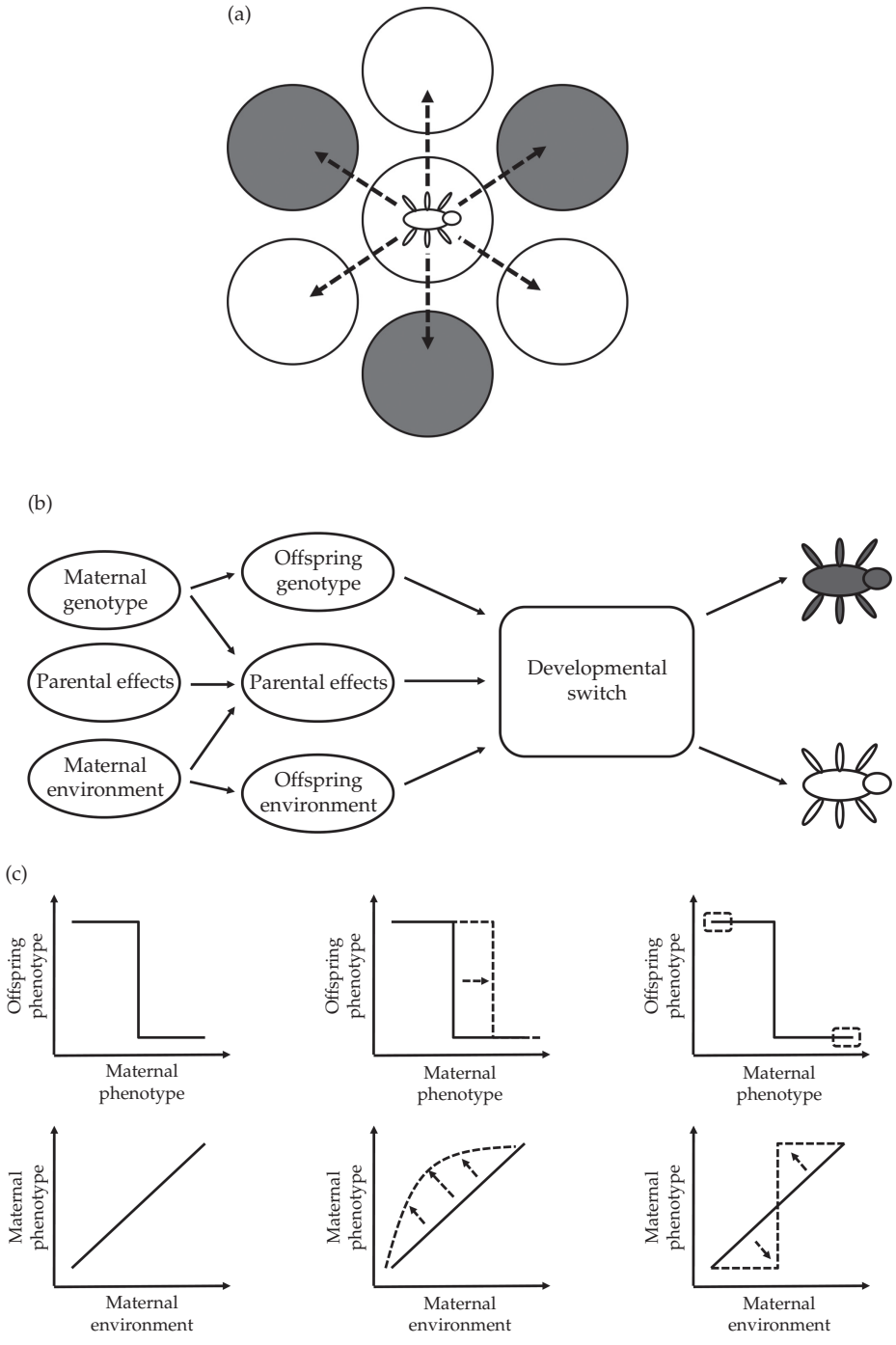

Figure 14.5 Evolution of trans-generational plasticity. (a) Evolution of adaptive plasticity requires temporal and/or spatial heterogeneity, for example, a meta-population structure with dispersal between patches with different environmental conditions. (b) Evolution of development can be conceptualized as evolution of sensitivity of a developmental switch to different sorts of input—genetic, parental, and environmental (modified from Leimar et al. 2006). Here the offspring phenotype is a polymorphism with only two possible states. Environmental conditions can vary at temporal or spatial scales so that the parental environment/phenotype carries information about the environment likely to be experienced by offspring. (c) If the maternal phenotype correlates with selection on the population of offspring phenotypes, this developmental switch can evolve to be responsive to variation in the parental phenotype (left panel). A shift in the parental phenotype (dashed lines) can change selection on the offspring reaction norm, which may evolve to maintain the fit between offspring phenotype and offspring environment (dashed lines, middle panel). Concordant selection across generations may allow evolution of parental reaction norms that maximize the information transfer between parents and offspring and thus minimize the risk for mismatch between offspring phenotype and their environment (right panel).

Box 14.1 Parental effects as systems of inheritance

From a perspective of modern biology, it may come as a surprise that the separation of development and heredity is a fairly recent innovation. Into the twentieth century, biologists saw inheritance (i.e. 'like begets like') as taking place throughout epigenesis via between-generation recurrence of the factors that build bodies (Amundson 2005). Only with the new field of genetics did heredity become the passing of traits between generations and, more specifically, transmission genetics (Amundson 2005). The discovery of a materialistic basis of the 'gene' thus turned DNA into the primary 'system of inheritance' upon which evolution relies. The unique position of DNA is challenged, however; parental effects show that phenotypic stability within lineages and differences between lineages can be maintained via parental transmission of non-genetic factors as well as DNA (Jablonka and Lamb 2005; Jablonka and Raz 2009). But are parental effects alternative systems of inheritance and, if so, how do those inheritance systems compare to genetic inheritance?

Biologists and philosophers alike often think about heredity as the passing of information between generations (e.g. Williams 1992; Maynard-Smith 2000; Jablonka 2002; Shea 2007; Bergstrom and Rosvall 2010). The concept of information in this context is not unproblematic (e.g. Oyama 2000), but one interpretation is that 'a source becomes an informational input when an interpreting receiver can react to the form of the source (and variations in this form) in a functional manner' (Jablonka and Lamb 2006; see also Jablonka 2002). This puts the study of inheritance systems into a broader context of signals and communication (Skyrms 2010; Shea in press), and focuses on the evolution of developmental responses to different sources of input, regardless of their origin (e.g. genetic versus environmental; Jablonka 2002; Shea et al. 2011). Using this approach, Jablonka and Lamb (2005) separated inheritance systems—ways to transmit information—as genetic, epigenetic, behavioural, and symbolic. By definition, parental effects do not fall under genetic inheritance (Chapter 1). However, they may fall under any of the other inheritance systems—epigenetic, behavioural, and symbolic (Helanterä and Uller 2010).

What makes the genetic inheritance system special and different from the mechanisms that contribute to parental effects (Shea 2007; Helanterä and Uller 2010)? One distinction between inheritance systems can be made based on the processes that generate correlational information between what is being transmitted and an adaptively relevant feature of the environment (Shea et al. 2011). The variants transmitted through inheritance systems involved in adaptive trans-generational plasticity carry information because the parental phenotype responds to some aspect of its environment that correlates with a feature that is of adaptive relevance to the offspring. This correlational information can be exploited by developmental processes because of the continuity between parental and offspring phenotypes (Fig. 14.5). In the genetic inheritance system, on the other hand, correlational information requires a process of selection that builds up gene frequency differences between environments (Leimar et al. 2006; Shea et al. 2011). Build-up of information through selection requires both stable transmission of developmental resources (such as genes) and sufficiently long time scales. Also, the DNA-based inheritance system seems to have the adaptive function to enable the transmission of heritable phenotypes down generations (Maynard Smith 2000; Shea 2007; Bergstrom and Rosvall 2010). These features may not be unique to DNA, however, and it is possible that epigenetic or behavioural mechanisms that initially contribute to parental effects (e.g. DNA methylation) can evolve to take on a similar role in heredity as the DNA-based system.

14.4.2 Trans-generational plasticity under parent–offspring conflict

As demonstrated by examples in this book, selection is not always concordant across generations. The implications of such parent–offspring conflict for the evolution of parental care in the form of resource provisioning have been explored in detail elsewhere (Chapter 7). Here I focus on how parent–offspring conflict will affect the evolution of trans-generational plasticity, that is the joint evolution of parental and offspring reaction norms in a heterogeneous environment, without assuming that the investment is costly to parents or beneficial to offspring (i.e. not only parental care).

Parent–offspring conflict is relevant for both continuous and discontinuous phenotypes that are of interest to a wide range of biologists. For example, in both plants and animals, competition between kin can generate parent–offspring conflict over natal dispersal. The parental inclusive fitness is often maximized at a higher dispersal rate than the offspring inclusive fitness because offspring that do not disperse compete with their parents and siblings for access to limited resources, and the cost of dispersal in terms of survival or reproductive success is usually paid for solely by the offspring (e.g. Frank 1986). A similar scenario may apply to diapause, which can be seen as dispersal in time (Tauber et al. 1986). Interestingly, related species often differ in the extent to which dispersal and diapause are genetically, maternally, or environmentally influenced (Tauber et al. 1986; Braendle et al. 2006), which provides opportunities for comparative tests of the adaptive significance of trans-generational plasticity and the role of parent–offspring conflict for the evolution of parental effects. For example, poor maternal nutrition and crowding increase the proportion of winged offspring in some aphid species, whereas in others genetic or direct environmental effects predominate (Braendle et al. 2006). Among vertebrates, research on the common lizard, *Lacerta vivipara*, has emphasized the role of kin competition for dispersal and provided experimental evidence that this conflict is modulated by the maternal environment (e.g. density; Meylan et al. 2007) and the maternal phenotype (e.g. maternal age; Ronce et al. 1998). Similar parent–offspring conflicts may arise over morphological and behavioural phenotypes associated with maternal hormone transfer, including offspring size, begging behaviour, and growth (Groothuis et al. 2005; Müller et al. 2007; Chapter 7).

Although context-dependent maternal effects on offspring behaviour can be interpreted as maternal 'manipulation' of offspring phenotype (e.g. Schwabl et al. 1997; Love and Williams 2008), parental manipulation will often be evolutionarily unstable (Müller et al. 2007; Uller and Pen 2011). The reason for this instability is that offspring can evolve to respond to the maternal phenotype in ways that maximize their own inclusive fitness, not that of the mother. In the absence of constraints on evolutionary counter-responses by the offspring, evolved patterns of trans-generational plasticity therefore often mirror those under offspring 'control' of trait expression (Uller and Pen 2011). This may apply even when offspring are unable to assess their own environment since the parental phenotype provides an additional source of information about local conditions (Müller et al. 2007; Uller and Pen 2011). Thus, even when the parent is the only individual who can directly detect the environment, context-dependent parental effects on traits that do not involve variation in costly resource allocation might most commonly represent the offspring optima. However, the simplifying assumptions of theoretical treatments (Revardel et al. 2010; Uller and Pen 2011)—for example two discrete environments, binary offspring response—question the generality of the conclusions. Indeed, 'deception' can be evolutionarily stable in signalling systems under certain conditions (Searcy and Nowicki 2005; Skyrms 2010) and may play a role also in signalling between parents and offspring (e.g. maternal manipulation of offspring phenotype; Chapter 7). Furthermore, costs associated with expression of particular phenotypes, such as parental investment (Chapter 3), complicate the interpretation of patterns of trans-generational plasticity, as the effect of the (parental) environment on offspring phenotype can be intermediate to that under complete maternal or offspring control (Uller and Pen 2011). Models trying to predict the shape of parental and offspring reaction norms will therefore have to carefully specify the temporal and spatial variation in environmental heterogeneity, limits on detection of environmental cues, costs involved with expression of particular phenotypes, the underlying genetic architecture, and the extent to which selection is concordant between parents and offspring.

14.4.3 Mechanisms of trans-generational plasticity

The often discrete and precise induction of offspring phenotype by parental effects may give the impression that the mechanisms of adaptive trans-generational plasticity, such as maternal effects on seed germination (Galloway and Etterson 2007), are different from developmentally

entrenched parental effects and those that promote diversifying or directional phenotypic variation in novel or stressful environments. However, adaptive trans-generational plasticity is more likely to represent a stable evolutionary state arising from emergent parental effects (e.g. initially induced by stress) that become stabilized by natural selection as maintenance of alternative phenotypes of both adults and developing offspring (Badyaev and Uller 2009; Fig. 14.2). For example, there is some evidence from birds and mammals that the same hormonal mechanisms that form an integral part of species-typical development also contribute to stress-induced, non-adaptive, variation and highly precise and adaptive environment-specific maternal effects on offspring phenotype (e.g. reviews by Fowden and Forhead 2009; Badyaev 2009; Uller and Badyaev 2009). Although mechanisms acting late in ontogeny may provide greater scope for information transfer between generations (Jablonka and Lamb 2005; Badyaev 2008), several authors emphasize the adaptive significance of environment-dependent reprogramming of epigenetic processes early in development (e.g. Mousseau and Fox 1998; Gluckman et al. 2005; Galloway and Etterson 2007). Thus, the mechanisms of adaptive trans-generational plasticity are not only similar to those involved in developmentally entrenched parental effects but may span the entire continuum from epigenetic modification of gene expression, maternal transfer of micro- and macro-nutrients to the egg yolk or the developing fetus, to post-natal behavioural interactions between parents and offspring.

14.5 Exploring the evolutionary dynamics of parental effects

A recent perspective (Badyaev 2009; Badyaev and Uller 2009) views parental effects as part of an evolutionary process in which the most recurrent parental resources are retained and eventually may become developmentally entrenched, visible only through genetic or environmental disruption of the species-typical developmental system. When such disruption is sufficiently recurrent for natural selection to stabilize the expression of induced alternative phenotypes, the result is adaptive trans-generational plasticity. Thus, developmentally entrenched and context-dependent parental effects are different outcomes of the same evolutionary process involving the same developmental mechanisms (Fig. 14.2). Badyaev (2009) has suggested that this process is an example of the Baldwin effect. The main tenet of the Baldwin effect is that phenotypic accommodation to environmental input can eventually become 'internalized' without any need for inheritance of acquired characters—all that is required is heritable variation in the initial response or that the initial response allows the population to persist until heritable variation accumulates (Baldwin 1902; see Weber and Depew 2002; West-Eberhard 2003 for discussion). This will result in a pattern of evolutionary diversification that reflects the ontogenetic flexibility of ancestral phenotypes. This chapter has emphasized three aspects of this process: 1) the role of parental effects for the origin of phenotypic variation via phenotypic accommodation of genetic or environmental input; 2) how particular aspects of parental effects (directionality and recurrence of novelties) can increase the likelihood that environmentally induced phenotypes can spread in the population, and 3) how selection on parents and offspring can sometimes maintain alternative phenotypes within a population in the form of adaptive trans-generational plasticity.

Parental effects in general, and parental care in particular, may thus contribute to evolutionary diversification or adaptation to novel environments in several ways. Evidence that maternal effects facilitate persistence in novel environments comes from studies of the seed beetle *Stator limbatus*, where offspring survival on a novel host species is facilitated by maternal plasticity in egg size (Fox and Savalli 2000). Diversifying maternal effects resulting from stress-induced changes in female reproductive physiology have contributed to the rapid and successful colonization of challenging climatic regions by house finches (Badyaev et al. 2003, 2008). Nevertheless, the available evidence that phenotypic accommodation via parental effects allows directional changes in response to novel environments that increase fitness and form the basis for local adaptation is often circumstantial, inferential, or based on laboratory conditions only.

Furthermore, the extent to which parental effects contribute to release of cryptic genetic variation that can enable evolution via genetic accommodation has not yet received much attention. Finally, although evidence for trans-generational persistence of novel phenotypic variation is rapidly accumulating (Jablonka and Raz 2009), whether or not this has played an important role in evolution by facilitating genetic accommodation (including the Baldwin effect), by enabling adaptive transfer of information across generations or as an inheritance system remains poorly understood.

Specific tests of whether parental effects have contributed to the origin and evolution of adaptation require investigation of the relationship between mechanism and regulation of developmentally entrenched and context-dependent parental effects in a historical context of environmental recurrence (Badyaev and Uller 2009). Support may be sought by assessing whether plastic responses in the ancestral state correspond to the developmental regulation of adaptive strategies in populations living under derived conditions. For example, evolution of relatively high developmental rate in colonial birds may have involved direct effects of high density and aggression on circulating levels of androgens in breeding females, which results in high androgen levels in eggs and an associated faster developmental rate (Gil et al. 2007). A similar scenario has been proposed for the evolution of alternative morphs in spadefoot toads (Pfennig and Martin 2009; Martin and Pfennig 2010). However, to show that developmental plasticity and parental effects played a role in the evolution of a particular phenotype, it is necessary to capture the ongoing process to document the transition from stress-induced variation to the evolution of local adaptation via phenotypic and genetic accommodation (Moczek 2007; Uller and Helanterä 2011). This requires an explicit focus on the developmental basis for evolutionary change on short time scales, which calls for innovative research programmes at the interface of developmental biology and evolutionary ecology. One such example is a long-term study of the house finch colonization of North America. Close integration of endocrinological regulation of female reproduction, oogenesis, and offspring growth facilitated evolution of local adaptation in sexual size dimorphism under novel climatic conditions via phenotypic accommodation of stress-induced variation, followed by cross-generational transfer of a subset of locally favoured phenotypes (summarized in Badyaev 2009).

As the house finch example demonstrates, human activities, such as species introductions, pollution, and habitat change can sometimes provide ideal settings for following populations as they encounter novel environments and gradually adapt (or go extinct). Such systems provide opportunities to link environmental induction of context-dependent parental effects and evolutionary change, and enables assessment of how important and general this process may be. For example, Marshall (2008) showed that in the bryozoan, *Bugula nerita*, a brief exposure to high levels of copper (a pollutant) resulted in offspring with reduced survival. This is expected since copper is toxic at high doses. However, offspring from copper-exposed mothers were relatively more tolerant to copper stress per se than offspring from non-exposed mothers. Although this may reflect an evolved adaptive trans-generational plastic response and a form of parental care (the history and heterogeneity of copper exposure in this species is unknown; Marshall 2008), it may also be a passive outcome of phenotypic accommodation and developmentally entrenched maternal effects. For example, an increase in circulation of metallothionein mRNA in response to copper exposure during oogenesis could have concomitant effects on egg composition, with carry-over effects on the development of heavy metal resistance in the offspring (e.g. Lin et al. 2000). Thus, the context-dependent parental effects observed in studies like this may be passive, capitalizing on pre-existing entrenched parental effects and representing a transient stage in the environmental induction of novel phenotypes, which may be followed by genetic accommodation in populations where the novel environmental factor is a recurrent feature (Fig. 14.2). A shift in focus from treating parental effects only as patterns of phenotypic variation, or as adaptive transfer of information across generations, towards viewing them as part of a process that connects environmental induction and adaptation will

allow us to gain novel insights into the mechanisms of evolutionary change.

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