PHILOSOPHICAL TRANSACTIONS OF THE ROYAL SOCIETY B

Phil. Trans. R. Soc. B (2008) **363**, 1635–1645
doi:10.1098/rstb.2007.0011
Published online 28 November 2007

Review

Early growth conditions, phenotypic development and environmental change

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Phenotypic development is the result of a complex interplay involving the organism's own genetic make-up and the environment it experiences during development. The latter encompasses not just the current environment, but also indirect, and sometimes lagged, components that result from environmental effects on its parents that are transmitted to their developing offspring in various ways and at various stages. These environmental effects can simply constrain development, for example, where poor maternal condition gives rise to poorly provisioned, low-quality offspring. However, it is also possible that environmental circumstances during development shape the offspring phenotype in such a way as to better prepare it for the environmental conditions it is most likely to encounter during its life. Studying the extent to which direct and indirect developmental responses to environmental effects are adaptive requires clear elucidation of hypotheses and careful experimental manipulations. In this paper, I outline how the different paradigms applied in this field relate to each other, the main predictions that they produce and the kinds of experimental data needed to distinguish among competing hypotheses. I focus on birds in particular, but the theories discussed are not taxon specific. Environmental influences on phenotypic development are likely to be mediated, in part at least, by endocrine systems. I examine evidence from mechanistic and functional avian studies and highlight the general areas where we lack key information.

Keywords: fitness; phenotypic plasticity; compensatory growth; silver spoon; thrifty phenotype; development

1. INTRODUCTION

The environment is not merely 'permissive' of development, but to some extent also guides, or even induces, it (Gilbert 2001, 2005). This complex interplay, between the individual's genetic make-up and the conditions in which its growth and maturation take place, means that the same genotype can give rise to a variety of phenotypes (West-Eberhard 2003). These phenotypes can differ dramatically. The European map butterfly *Araschnia levana*, for example, develops such different colour forms under different environmental temperatures and photoperiods that Linnaeus originally classified them as different species; it is now known that this is due to environmentally induced changes in the pattern of secretion of the hormone ecdysone (Nijhout 2003; Gilbert 2005). Such distinct, discontinuous phenotypic variations are generally known as polyphenisms. More subtle, and probably more common, environmentally induced changes to physiology, behaviour or morphology also occur. This variation is more continuous and can be operational over varying time scales. Given the pace of global environmental change, understanding the nature and the consequences of these environmentally induced changes, and the extent to which they are adaptive, is of great importance.

Environmental effects on the phenotype can act directly on the developing organism. They can also come indirectly, when environmental effects on one individual, often the mother, influence phenotypic development in another, usually its offspring; these are generally referred to as maternal effects (Mousseau & Fox 1998; Wolf *et al.* 1998). Consequently, an organism's phenotype can be influenced not just by its own environment, but also by influences that result from current or past environmental effects on its parent(s) that are transmitted to their developing offspring in various ways and at various stages. In many insects, for example, breeding females change their production of diapausing or directly developing offspring in response to temperature, photoperiod and resource availability (Mousseau & Fox 1998). There is currently great interest in the extent to which environmentally induced phenotypic change is adaptive. This may be clear, as in the above insect example where changes in photoperiod signal the onset of winter, and diapausing offspring have a higher chance of survival (Mousseau & Fox 1998). However, environmentally induced changes to the phenotype need not be adaptive. Development of the optimum phenotype may be constrained by environmental circumstances, for example, where low resource availability during

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One contribution of 12 to a Theme Issue 'Integration of ecology and endocrinology in avian reproduction: a new synthesis'.

prenatal or post-natal development gives rise to a stunted, poorly performing individual. In other cases, for example, when the presence of high levels of maternally derived stress hormones in bird eggs alter the stress response of the resulting individuals, the adaptive significance is much less clear. We need to understand how environmentally induced changes occur and the capacity of organisms to produce phenotypes better adapted to cope with prevailing circumstances. In addition to its fundamental biological significance, this knowledge is essential for development of effective conservation policies and also to our understanding of the developmental origins of disease in humans and other animals (Bateson *et al.* 2004; Gluckman *et al.* 2005).

To obtain this knowledge, we need more integration across biological disciplines, such as the dialogue between endocrinologists and evolutionary biologists illustrated in this volume, since concerted mechanistic and functional approaches are required. The endocrine system mediates between the environment and gene expression, and thus plays a crucial role in epigenetic effects; changes can occur at many stages along the winding path from gene transcription, to functional proteins, to functioning individuals (Gilbert 2005). Hormones play a major role in mediating environmental effects on phenotypic development, and their pleiotropic effects will influence several traits simultaneously, causing positive covariation or mediating physiological trade-offs (see Lessells 2008; McGlothlin & Ketterson 2008). Interest in the links among conditions during early growth, phenotypic development and environmental change has come from scientists from many different disciplines, working within different theoretical frameworks (Bateson *et al.* 2004). This has resulted in apparently similar ideas and paradigms being superimposed on each other, when in fact they are not quite the same and give rise to different predictions. Key issues in this field for evolutionary ecologists are whether phenotypic changes induced by environmental effects during development are, or are not, adaptive, over what time scale any fitness benefits occur, and under what environmental circumstances. For endocrinologists, understanding how the environment influences development by triggering hormonal changes in parent and offspring is the main focus. For those in more applied disciplines, such as medical, veterinary or conservation research, understanding the mechanisms whereby health or performance is changed by the developmental environment, under what conditions negative effects arise, and what can be done to prevent them, is of paramount importance.

To answer both mechanistic and functional questions, we need clearly elucidated and testable hypotheses, and, where possible, experiments that allow us to tease apart confounding variables. In this paper, I outline how the different paradigms applied in this field relate to each other, the main predictions they produce and the kinds of experimental data that are needed to distinguish among competing hypotheses. I focus on birds in particular, but the theories discussed are not taxon specific. I then discuss evidence from avian studies and highlight the general areas where we lack key information.

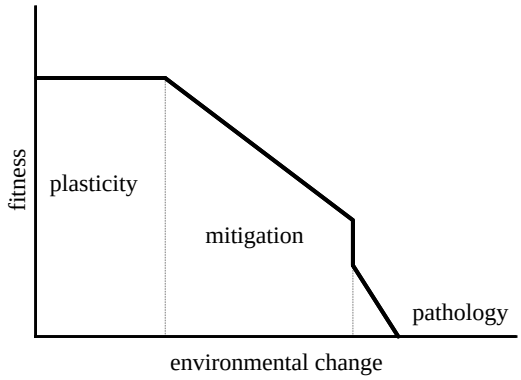

Figure 1. The effect of environmental change on fitness within the species reaction norm. As the environment during development changes (x-axis), phenotypic changes will be triggered. Within a certain range of environmental change, no negative fitness consequences result from the development of an alternative phenotype; outwith this range, fitness declines as the development of an optimal phenotype is constrained. The fitness decline is initially slow, since the phenotypic adjustments may mitigate the negative effects on fitness, and trade-offs between traits or across life-history stages may occur. Outwith this 'mitigation zone', pathologies develop and fitness drops dramatically.

2. PHENOTYPIC AND ENVIRONMENTAL CHANGES

Obviously, many features of the environment, such as resource availability, temperature, photoperiod or predator pressure, impinge on the reproducing parent and directly and indirectly on the developing offspring. These can affect the mother and its egg (foetus in viviparous organisms), the post-natal environment and the quality of post-natal care by the parents where this occurs (Zhang *et al.* 2006). Potentially, the offspring might receive signals during its embryonic development that are indicative of the environmental circumstances it is likely to experience after birth. In birds, such signals could come from egg composition, e.g. the nutrient, hormone or antibody content. The conditions during incubation, such as parental attendance, clutch size and incubation temperature, can also influence aspects of chick development (Reid *et al.* 2000; Gorman & Nager 2004). A familiar concept to evolutionary ecologists is that of the 'reaction norm', the range of phenotypes that can be produced by a single genotype in the environments in which it can survive (Stearns 1992, and see Lessells 2008). In terms of the resultant fitness of these phenotypes (taken here as the expected relative contribution to future generations) in different adult environments, we can usefully divide the reaction norm in three (figure 1). Within a certain level of environmental change, organisms may be able to adjust their phenotype such that fitness is maintained, that is they exhibit phenotypic plasticity in the sense of producing alternative phenotypes well matched to prevailing local conditions. This is a zone of tolerance, a level of environmental variability to which the individual organism can adjust without fitness penalties (figure 1). Such adjustment can take many forms. There might be different developmental routes to the same adult phenotype: alternatively, the adult phenotype itself may be permanently changed in physiology, morphology or behaviour. Greater

environmental change may take developing organisms into an area where fitness can no longer be maintained (figure 1). Within this zone, phenotypic changes that mitigate the detrimental effects on fitness can occur. It is here that we can apply the concept of trade-offs. These trade-offs may involve selective allocation of resources to some organs rather than others when conditions are poor. Or there may be trade-offs between beneficial effects in one life-history stage and detrimental effects in another; for example, changes that promote survival during the juvenile phase can carry survival penalties in later life. Nonetheless, this may still give the best fitness outcome given the circumstances, and hence be favoured by selection. A good example of such a trade-off is compensatory growth following a period of poor nutrition. While beneficial in enabling an individual that would otherwise be stunted and a poor competitor to attain a near normal adult size, such growth acceleration is associated with reduced adult lifespan (Metcalfe & Monaghan 2001, 2003). Even greater environmental perturbation may take the organisms outside the mitigation zone, into an area where pathology develops and fitness drops dramatically (figure 1). Obviously, the duration of the different phases and fitness declines depicted in figure 1 will vary among different organisms and with the nature of the environmental change.

3. THE INTERACTION BETWEEN DEVELOPMENTAL AND ADULT ENVIRONMENTS

Phenotypic plasticity is a property of a genotype, not something distinct from it. Genetic variation for plasticity has been demonstrated in a number of taxa, suggesting therefore that it is open to the forces of natural selection, and that adaptive phenotypic plasticity can evolve (West-Eberhard 2003); however, this has received relatively little attention until recently (Pigliucci 2005). The idea is essentially that the environmental 'shaping' of the phenotype 'matches' environment and phenotype in an adaptive way. Fitness is highest when the individual lives as an adult in the environment that matches the one in which it developed; where the developmental environment and the adult environment do not match, fitness is low (see figure 2*a* for a graphic representation of these relationships). For this 'tailoring' of the phenotype to work, we need a correlation between the developmental and the adult environments. Rapid environmental change could disrupt such a correlation.

Recently, this concept has been applied to the more complex, and probably more realistic and common, situation in which we have an environmental gradient, for example, related to food quality or availability or to stress (e.g. weather, predator or socially induced) or disease levels. What we then have are not alternative environments, but rather a quality continuum, stretching from low to high. The concepts of 'foetal programming' and the 'thrifty phenotype', which have their origins in human medicine, are based on this paradigm (Hales & Barker 2001; Bateson *et al.* 2004; Spencer *et al.* 2006). The thrifty phenotype hypothesis was formulated as an attempt to explain the association between low birth weight in humans and the increased risk of impaired glucose tolerance and metabolic syndrome in later life. The hypothesis originally was that developing in an environment in which resources are at the low end of the continuum induces changes to the endocrine system that serve to protect the supply of glucose to the developing brain, at the expense of other organs such as the pancreas. Permanent changes in the structure and the functioning of the body result, which become disadvantageous later in adult life. This explanation therefore involves trade-offs over two time scales that have not been explicitly separated. Firstly, during development itself, a trade-off in resource allocation to competing organs might give rise to a suboptimal phenotype from the outset—the best of a bad job, with investment being preserved in those organ systems that have the biggest positive impact on lifetime reproductive success. Secondly, there is also a potential trade-off operational over a longer time scale. Here, phenotypic changes that promote juvenile survival under poor conditions come at a price; costs are incurred in later life. Under the first scenario, one system, say pancreatic endocrine function, becomes a casualty of the juvenile environment, in the second it has been strategically adjusted to it.

The observation that the negative effects of poor developmental conditions are most marked when the adult environment is resource rich led to the thrifty phenotype theory being extended such that the developing phenotype is viewed as having been shaped (sometimes termed 'programmed') to perform best in a resource poor environment. In humans, the phenotypic changes induced by poor early nutrition are thought to involve, among other things, changes to insulin sensitivity and regulation of body fat levels. Hence, the suggestion is that it is only when a phenotype is tailored to resource-poor conditions, but then finds itself in a resource-rich environment, that early onset diabetes and obesity result; this also brings increased risk of cardiovascular and other degenerative diseases (Hales & Barker 2001; Fernandez-Twinn & Ozanne 2006; Spencer *et al.* 2006). Thus, we have a third explanation of the poor performance of the thrifty phenotype under good conditions. This is based not on trade-offs, but on an environmental mismatch; changes that are adaptive under poor conditions, such as storing food as fat and maintaining high blood glucose levels, now become maladaptive under good conditions (Bateson *et al.* 2004; Gluckman *et al.* 2005). Related to this is the concept of predictive adaptive responses (PARs; Gluckman *et al.* 2005). These are defined as changes that take place during development in response to environmental cues, but where the *advantage*, as opposed to the cost, is not evident until later in life. Thus, under this scenario, it is not the developing organism that benefits directly from these changes, but the adult form. This differs from the modified thrifty phenotype hypothesis, at least in the latter's original formulation, in that the phenotypic changes are not induced to promote survival of the developing organisms under poor conditions, and hence no trade-offs apply. Rather, the juvenile is preparing for a particular adult world. However, these phenotypic changes should not be costly in the juvenile

stages, otherwise they would be selected against since the forces of selection are likely to be stronger in the younger stages. Like the thrifty phenotype scenario, problems arise when the environment does not match the one that was anticipated during development. Both the modified thrifty phenotype hypothesis and the PARs hypothesis can then be viewed as environmental matching models, since in both there is an interacting effect of early and adult environment on fitness.

Figure 2*a* shows the expected fitness benefits that the environmental matching hypothesis predicts under different combinations of environment in early and in adult life. For the sake of simplicity, the developmental conditions are presented simply as good and poor as in the context of food supply, though of course like the adult environment, this would also be a continuum. (It need not be good and poor nutrition of course, but any environmental gradient, e.g. high and low temperature, predation pressure, etc.) There are four key predictions here.

(i) For those born in good conditions, fitness is highest under good adult conditions and progressively decreases as the adult environment deteriorates.
(ii) In good adult conditions, those individuals that developed under good conditions have higher fitness than those that developed under poor conditions.
(iii) For those born in poor conditions, fitness is highest when the environment matches that of development, that is poor, and progressively decreases as the adult environment departs from this, i.e. as the conditions in the adult environment improve.
(iv) In poor adult conditions, those individuals that developed under poor conditions have higher fitness than those that developed under good conditions. In effect, we have alternative phenotypes; to maintain fitness (i.e. to remain in the zone of tolerance shown in figure 1), the developmental and the adult environments must match.

An alternative model (figure 2*b*) is based on a concept with which functional and mechanistic researchers are familiar—the so-called silver spoon effect (Grafen 1988). This is a situation in which individuals born in good conditions have fitness or performance advantages in later life, and of which there are many examples (Lindstrom 1999). Those born in poor conditions are of lower quality. Here there is no phenotype–environment matching and those born under resource-poor conditions are at a permanent disadvantage. This silver spoon model is illustrated in figure 2*b* and the predictions are as follows.

(i) For those born in good conditions, fitness is highest under good adult conditions and progressively decreases as the adult environment deteriorates.
(ii) In good adult conditions, those individuals who developed under good conditions have higher fitness than those that developed under poor conditions.

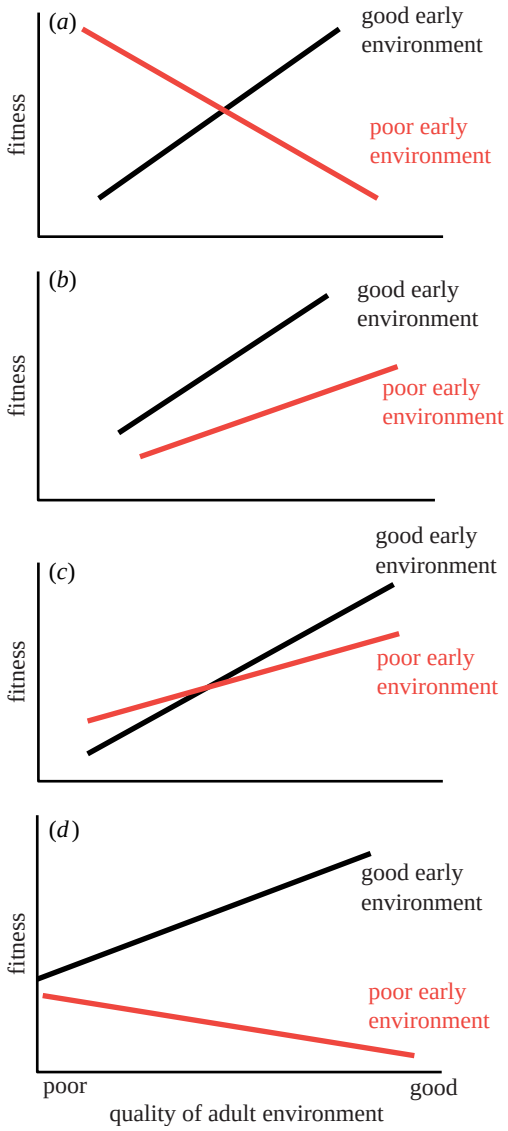

Figure 2. (*a*) Environmental matching. Here the adult environment changes in relation to levels of a particular parameter, e.g. resource low to resource high, stress low to stress high. Lines are shown for individuals developing at the low and high ends of the environmental spectrum. For those developing in both good and poor environments, fitness is the highest when the adult and developmental environments match. (*b*) Silver spoon effects. Here there is no environmental matching. Fitness always improves with improvement in the adult environment, and those born in poor conditions always have lower fitness than those born in good conditions. (*c*) Fitness always increases with resources in the adult environment, but when these are low, those born in such an environment have a fitness advantage over those not born in such conditions. (*d*) A combination of the silver spoon and environmental matching. For both those born in the good environment, fitness improves as the quality of the adult environment improves; for those born in the poor environment, fitness declines as the resources in the adult environment increase; however, whatever the adult environment, those born in the good environment always have higher fitness.

(iii) For those born in poor conditions, fitness is highest under good adult conditions and progressively decreases as the quality of the adult environment deteriorates.

(iv) Whatever the adult environment, those born in good conditions have a higher fitness than those born in poor conditions.

Thus, the predictions for those born in good conditions are the same under the environmental matching and the silver spoon models. For those born in poor conditions, however, the predictions are quite different in these two models. Under the silver spoon model, whatever the developmental environment, fitness improves as the adult environment improves; whatever the adult environment, those born in good conditions always outperform those born in poor conditions. Note that those born in poor conditions never attain the same fitness as those born in good conditions. Their relatively poor performance is a consequence of the trade-offs during development (delayed effects or trade-offs between traits competing for limited resources during development as mentioned previously). These individuals developed somewhere in the 'mitigation zone' shown in figure 1. The magnitude of the costs incurred will determine the distance apart of the two lines in figure 2*b*.

Obviously, other variations are also possible, depending on what happens to the fitness of those born in poor conditions. Figure 2*c*,*d* illustrate two of the most likely. In figure 2*c*, fitness again increases with the quality of the adult environment whatever the developmental environment; in poor adult conditions, however, those born in poor conditions do perform better than those born in good conditions. For example, if developing under poor conditions gives rise to a small body size, this may be less costly than a large body size when food is in short supply due to lower maintenance requirements, and hence is now advantageous. This model (figure 2*c*) seems more plausible than the environmental matching model as represented in figure 2*a*, in that all individuals benefit from a high-quality adult environment. It seems likely that selection would act strongly against the situation in figure 2*a*, where certain individuals are simply unable to benefit as adults from an improvement in conditions. The strength of this selection would of course depend on how variable the environment actually is, but for the capacity for phenotypic plasticity to evolve as a life-history strategy, significant environmental variability is necessary. This is because plasticity itself may well carry some costs, and hence would not be favoured by natural selection if the environment were stable (Pigliucci 2005).

Figure 2*d* is a combination of the silver spoon and the environmental matching models, and may in fact be a better representation of the thrifty phenotype concept. Here, those born in good conditions are always at a fitness advantage, but this lessens as the adult environment deteriorates; for those born in poor conditions, the phenotypic adjustments made to mitigate the effects of poor developmental conditions mean that these individuals perform less well than they could have done had the compromises not been necessary, and the disadvantage is exacerbated when the adult environment does not match that to which its phenotype is adjusted. So things get worse for these individuals when the adult environment is resource rich.

To distinguish between these models, we need experimental data from all four possible combinations of early and adult environments. In general, we lack good data on the performance of different phenotypes under poor conditions. We often have comparative data for the performance of those developing in relatively good and poor conditions experiencing good conditions in later life; we rarely have the comparative data for performance under poor adult conditions.

4. DO WE HAVE EVIDENCE OF INTERACTING EFFECTS OF EARLY AND ADULT ENVIRONMENTS ON FITNESS IN BIRDS?

(a) *Long-term effects of early developmental conditions*

That conditions during early development can have long-term consequences in birds is well established (Lindstrom 1999; Metcalfe & Monaghan 2001). Such effects have been studied at the individual and cohort level, and in both the field and the laboratory. In the field, a number of fitness-related measures in adults have been found to correlate with conditions during development. In the red-billed chough *Pyrrhocorax pyrrhocorax*, for example, adults originating from cohorts reared in poor environmental conditions on average have shorter breeding lives. Similarly, individual oystercatchers, *Haematopus ostralegus*, reared in high-quality habitats have higher adult survival and are much more likely to recruit to high-quality habitat as breeders in comparison with those reared in low-quality habitats (van de Pol *et al.* 2006). One sex may be more vulnerable to long-term effects of poor early conditions than the other. For example, female, but not male, tawny owls, *Strix aluco*, that developed in good conditions have better breeding performance later in life (Appleby *et al.* 1997). In choughs, the breeding performance of young adult males varies positively with the conditions under which they were reared, but this effect does not occur in females (Reid *et al.* 2003).

While working in the field does of course give more realistic measures of the effects of differing environmental conditions early and late in life on fitness or (more usually) fitness correlates, identifying causal factors is much more difficult since genetic and environmental factors are usually confounded, and many environmental factors will be operating simultaneously and sequentially. Laboratory studies provide the potential to manipulate different components of the early and the adult environments, with comparisons being possible among siblings given different treatments, or genotypes randomized across treatment groups to try to control genetic background at least to some extent. Such experiments have been conducted using a variety of different environmental treatments applied at different stages in development, with follow-ups to adulthood and sometimes to subsequent generations. While fitness consequences are more difficult to measure in the laboratory, realistic measures of aspects of performance that are likely to relate to fitness are often possible.

For example, resource availability during development has been manipulated indirectly, through brood size manipulation. Chicks of the altricial song bird, the zebra finch *Taeniopygia guttata*, reared in experimentally enlarged broods have higher standard metabolic rates as adults (Verhulst *et al.* 2006) and take longer to begin breeding (Alonso-Alvarez *et al.* 2006). Adult size and immune function can also be affected (Gil *et al.* 2004). Furthermore, females that were reared in enlarged broods, then subsequently kept on ad libitum food rations, still produce offspring of a smaller size, showing that these effects can span the generations (Naguib & Gil 2005). While it could be hypothesized that such a size effect would be advantageous if the poor conditions persisted into the next generation, with small size then being advantageous, this has not been tested.

While brood manipulations are a relevant way of reducing food availability to developing offspring, it is not clear in such experiments exactly what features of the early environment are being altered for such chicks. The competitive environment in which they are being reared is different, some offspring might get more food than others, the state of the parents will change, the nature of the parental care that the offspring receive and their post-fledging social environment will all be different as a consequence of the brood size change.

An alternative approach is to manipulate the quality of food available during development. Zebra finch males reared on a poor quality (low protein) diet have substantially shorter adult lives (Birkhead *et al.* 1999) and both sexes have also been found to have reduced antioxidant defences as adults (Blount *et al.* 2003) and take longer to produce their first clutch when breeding for the first time (Blount *et al.* 2006). Early nutrition has also been shown to have important implications for the structure and complexity of songs that males sing as adults in several species, with those experiencing poor nutrition performing less well; it has been suggested that, because the neurological development of the song system is very sensitive to early nutrition, male song gives females a reliable cue as to the developmental history, and hence quality, of prospective mates (Nowicki *et al.* 2002; Buchanan *et al.* 2003; Buchanan *et al.* 2004; Searcy *et al.* 2004). Other areas of neural development can also be affected by poor early nutrition. For example, western scrub-jays, *Aphelocoma californica*, reared on reduced ration as nestlings performed less well on spatial, but not associative, learning tasks and had lower mean hippocampal volumes as adults (Pravosudov & Kitaysky 2006).

A complication with this approach, however, is that early nutritional deficits are often accompanied by subsequent compensatory growth, involving growth acceleration and/or prolongation of the growth period such that normal adult size it attained. Such growth compensation carries costs; such costs can detrimentally affect adult performance or their manifestation may be delayed but traded-off against long-term survival (Metcalfe & Monaghan 2003). With respect to elucidating underlying mechanisms, it is therefore difficult to identify whether or not the observed long-term effects are a consequence of the nutritional deficit itself or the subsequent growth compensation.

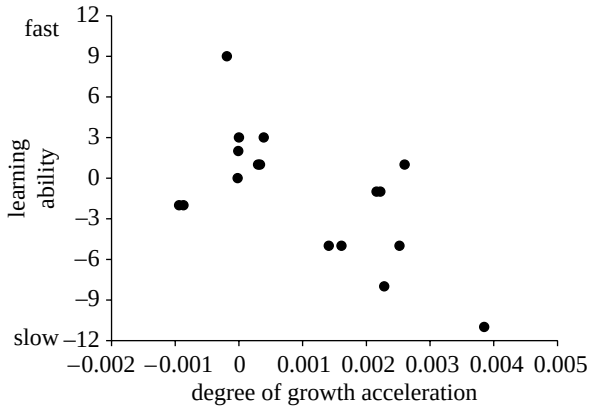

Figure 3. The relationship between the degree of growth acceleration following a period of poor nutrition early in life and cognitive ability as an adult. Those birds that showed the most growth acceleration relative to a control sibling had the lowest relative cognitive ability. Adapted from Fisher *et al.* (2006).

However, it has recently been found in zebra finches, subjected to a period of poor nutrition during early post-hatching growth, that cognitive performance in adulthood was linked not to the early nutritional deficit, but to the extent to which growth had been accelerated relative to a control sibling (Fisher *et al.* 2006; figure 3). Similar detrimental effects of compensatory growth on cognitive abilities (Singhal & Lucas 2004) and on the onset of degenerative disease (Eriksson *et al.* 2001) have been found in humans. Such data suggest that, whatever the mechanisms, the phenotype that develops under resource poor conditions is permanently impaired in some features. It seems unlikely that such individuals would perform well as adults in situations where food is hard to find, but this has not been tested. Much more experimental work is needed in this area.

There are many other factors that also need to be considered. The timing of the nutritional deficit might be very important in influencing the outcome of experiments, since there might be sensitive periods during development when particular systems are especially vulnerable, as seems to be the case for the song centres in the avian brain. Different kinds of birds may prioritize different body components during shortage. Most nutritional manipulations, including those mentioned above, are undertaken from the beginning or part way through the nestling phase. However, embryonic development is likely to be a particularly important phase and egg quality is known to have substantial effects on parents and offspring (Monaghan & Nager 1997; Nager *et al.* 1999). It is generally held that the earlier an environmental perturbation takes place, the more severe will be its long-term effects (Lindstrom 1999). Furthermore, the great variation in the pattern of development in birds along the altricial–precocial spectrum means that hatching takes place at different stages of maturation, adding a further layer of complexity to the effects of the early developmental environment that has received little attention. A number of field and laboratory studies in birds suggest that one sex is more vulnerable to poor early environmental conditions than the other

(Nager *et al.* 1999; Martins 2004; Spencer & Verhulst 2007). We need also to consider therefore whether we get differing fitness outcomes for males and females.

These long-term effects of conditions during early development are likely to occur over vaying time scales; some, such as changes in body adiposity, may begin to manifest themselves during the latter part of development. We therefore need more studies that try to identify effects over differing time frames. Such changes are presumably mediated by changes in the endocrine system, probably at many levels. This is obviously an extremely complex area, since many hormonal systems are likely to be involved. Furthermore, some effects may come about through maternal hormones transmitted via the egg, while others come about through effects on the endocrine system of the developing chick itself; these direct and indirect effects have not been well separated so far. Direct hormonal manipulations, albeit relatively crude, have a role in helping to identify mechanisms. By carefully controlling the timing of administration, it may be possible to experimentally separate effects likely to be of maternal origin. But which hormones should be manipulated? In avian studies, most effort so far, at least outside of the poultry field, has gone into examining the consequences for chicks of maternally derived androgens in eggs. These are influenced by maternal state, and have substantial effects on chick growth and development (see Groothuis & Schwabl 2008; McGlothlin & Ketterson 2008). However, the extent to which maternal androgens provide environmental signals for the developing offspring, 'preparing' it for the environment it is most likely to encounter after fledging, has received little attention. One system that is likely to play a key role in transmitting environmental signals is the hypothalamic–pituitary–adrenal axis (HPA axis). The effects of early environmental circumstances, particularly maternal grooming and nursing behaviour (Champagne *et al.* 2007), on the development of this axis in rodents are profound and relatively well studied (Fish *et al.* 2004). It has been suggested in mammals that adult metabolic disease is a result of the so-called foetal programming of the HPA axis that occurs during conditions of poor foetal growth (Fernandez-Twinn & Ozanne 2006). Such changes may well involve both prenatal hormones of maternal origin and post-natal environmental effects on the developing organism's own hormone system; thus both direct and indirect environmental effects may be involved. In birds, work with this hormone system in the context of its mediating environmental effects on the development of traits other than song learning (see above) is currently relatively limited, but increasing (Kitaysky *et al.* 2003; Hayward & Wingfield 2004; Spencer & Verhulst 2007). Furthermore, different baseline levels of corticosterone in the plasma of breeding male, but not female, white-crowned sparrows have been found in urban and rural areas, so again sex specific effects on the development of the HPA axis might be very important (Bonier *et al.* 2007). Administration of corticosterone to breeding female quail, a highly precocial species, resulted in their laying eggs with elevated corticosterone in their yolks. The resulting chicks grew more slowly, and as adults had a higher activity of the HPA axis in response to capture and restraint (Hayward & Wingfield 2004). In this case, the change is driven at least initially by maternally derived hormones present in the egg. Post-natal corticosterone administration during chick growth has also been found to produce long-lasting effects in altricial species. In zebra finches, administration of corticosterone to nestlings during the chick phase again slowed growth, and on reaching adulthood the males were found to be less competitive (Spencer & Verhulst 2007). However, that the administration of corticosterone generally slows growth (Hayward & Wingfield 2004; Spencer & Verhulst 2007) means that any long-term effects could be due to the effects of growth compensation, rather than the effects of the corticosterone. Furthermore, the effects of corticosterone are pervasive; it is a proteolytic hormone and can influence protein metabolism, making it difficult to pin down specific effects.

There are of course many other mechanisms that need to be investigated if we are to predict the responses of birds to changes in conditions during early development. For example, adipose tissue, the storage of which is strongly influenced by early growth circumstances, does more than simply store energy; it is an endocrine tissue, involved in energy balance and glucose tolerance. Thyroid-related hormones are also likely to be very important, but these have been very little studied in birds (McNabb & Wilson 1997; Wilson & McNabb 1997). In other taxa, such as flies, rodents and nematodes, there is a strong research focus on the insulin-like growth factor-1, growth hormone and thyroid hormone, all of which are interdependent and play an important role in regulating growth and metabolism, in enabling organisms to survive periods of food shortage and in determining lifespan (Gems & Partridge 2001; Tatar *et al.* 2003; Clemmons 2004). Studies in this area in birds are again very limited, and almost entirely confined to poultry (Leili & Scanes 1998; McMurtry 1998; Kita *et al.* 2002; Decuypere *et al.* 2005). A further potentially profitable area that has been attracting increasing attention is that of the uncoupling proteins, which are involved in many aspects of metabolism including glucose tolerance, immune efficiency and the level of production of free radicals by mitochondria; these proteins are sensitive to conditions during early development, but we know very little about their operation in birds (Criscuolo *et al.* 2005; Mostyn *et al.* 2005). We need to know more about what changes occur in these systems under different developmental conditions, whether observed effects on development are direct or indirect, and the consequences over varying time scales. Obviously, we need to go beyond simple dosage with hormones, but the tools available with which to do this are not well developed outside of the 'model organisms'. That different types of birds are likely to give differing results is in itself of great interest, and interspecific comparisons, central to evolutionary biology, are killed off by a model organism approach. We need more studies of longer-term effects under different environmental circumstances in developmentally and ecologically different species. But we also need to know more about many mechanistic processes, such as the links and feedback loops between hormones and responses

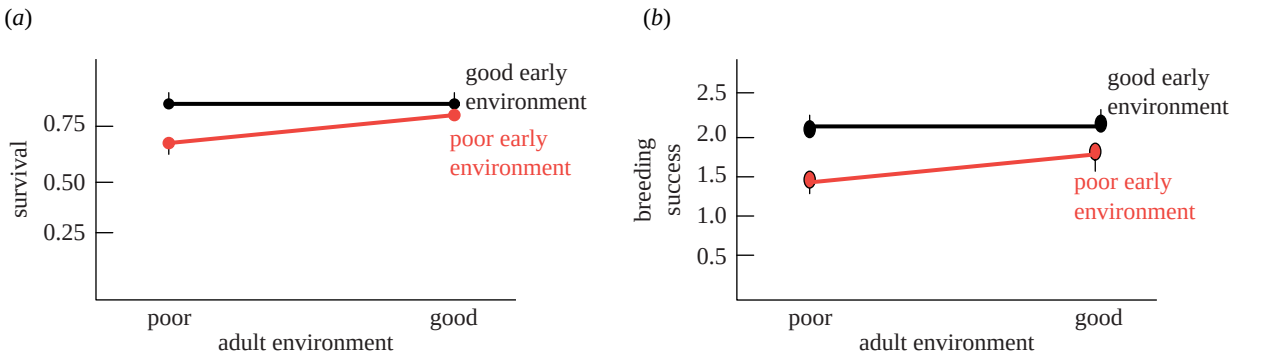

Figure 4. (*a*) Adult survival and (*b*) breeding success of red-billed choughs born in poor and good environments in relation to the quality of the environment in which they bred as adults. Those born in good conditions performed best in general. Data from Reid *et al.* (2006).

to help us separate cause and effect. Changes in hormone-binding proteins and receptor levels during development also need to be studied since, among other things, we need to know how closely offspring 'listen' to signals provided via maternal hormones (see also Groothuis & Schwabl 2008).

(b) *Interaction between developmental and adult environments*

We know very little about the adaptive significance of the phenotypic changes that take place in birds, or in other taxa, during a particular set of environmental circumstances (Pigliucci 2001). The key question here is whether the fitness of a phenotype produced as a consequence of poor nutrition is simply impaired—that is, in the 'mitigation' phase of the reaction norm as shown in figure 1, and thereby consistently lower than the non-impaired individuals as in figure 2*b*. Or do the developmental circumstances experienced trigger the development of an alternative phenotype such that fitness is maintained, provided the adult environment matches that of the developmental stage as in figure 2*a*? The necessary data need to involve the full set of environmental crossovers between developmental and adult environments. In the field, we have very little information for birds since it is difficult to manipulate settlement patterns. In the red-billed chough, a long-term study of an island population in western Scotland has demonstrated that distinct poor and good areas (based on the probability of producing recruits to the breeding population) can be identified. Tracking of the performance of birds born in these areas that survived to breeding age, in relation to whether they recruited to the poor or good areas, gives a result that is more in line with the silver spoon model than the environmental matching model (figure 4, compared with figure 2*a*,*b*). However being a field study, many aspects of settlement are not controlled; it is possible, for example, that genetic effects are also involved here, though this seems to be unlikely since the population is small, and birds do move around the island making it unlikely that specific lineages have developed in the two areas (Reid *et al.* 2003, 2006).

Experimental data are needed, where genetic status is controlled, and where subjects do not self-select into particular adult environments. We need to be clear as to what theoretical framework should be applied in such studies, and what signals the offspring might obtain. Being in a large brood, for example, could be taken to signal that the environmental conditions were good, at least during laying and incubation, and thus it would not make adaptive sense to 'programme' the phenotype based on nestling food availability. Such experiments therefore belong more within the developmental stress/silver spoon/mitigation framework. To test the phenotypic plasticity/phenotypic programming hypotheses, we also need to ensure that the full environmental crossovers occur. Interestingly, a well-controlled experimental study on female cichlid fish, *Simochromis pleurospilus*, in which the necessary environmental crossovers were carried out, also supported the silver spoon model; the total biomass of young produced (considered the best fitness-related trait measured) was consistently poorer for those females reared under poor conditions, whatever the adult environment (Taborsky 2006). There is obviously scope for much more experimental work here, in relation to manipulations of key parameters such as nutrition and stress hormones. Environmental factors other than food are also likely to be very interesting. For example, ectoparasite levels in the environment have been linked to egg hormones and dispersal behaviour (Tschirren *et al.* 2007).

5. CONCLUSIONS

Clearly, the effect of conditions during early growth and maturation on phenotypic development is a huge and burgeoning field, which can be studied from many different angles. There is much scope for novel experimental work in which the effects are assessed over different stages of the life cycle, and performance measured in differing adult environments. For birds in particular, it is important to take account of the timing of the manipulation relative to the developmental mode of the species involved, and the time scales over which effects are measured. Most work to date has involved altricial species such as zebra finches. Manipulations of the embryonic environment are also likely to be important, especially in precocial species. In addition, we need to consider the magnitude of the experimental manipulations relative to the responses shown in figure 1, since creating environmental circumstances

that induce pathologies is generally not the experimental aim. However, we need to understand how organisms can respond to environmental change; we need to know the limits to their flexibility and mitigation responses. We need to try to separate different environmental variables that may be confounded, and take into account the effect of the manipulation on subsequent growth. We also need to look further than just hormone levels; we need to know about how the environment alters gene expression. This probably means that more interactions are needed between researchers in poultry science and those in other areas of avian biology, since there is currently rather little exchange of information.

However, we must not lose sight of ecological relevance and diversity in responses. To an evolutionary biologist, it does not make adaptive sense for a mother to shape its offspring to transitory environmental conditions. Under some circumstances therefore, where the maternal or early growth environment is not predictive of the adult environment, we would expect development to be buffered against environmental effects; where the environment is very variable, we might expect organisms to retain flexibility into adulthood. Can we identify such species? Are they more or less likely to be able to cope with environmental change? What physiological constraints are there on flexibility? Does it pay to produce offspring of variable phenotypes and is there evidence that birds do this within or between clutches? Does a variable and unpredictable food supply have different effects on development than do stable high or low food supplies? Can we separate direct and indirect environmental effects? Only by combining studies of function and mechanisms in a wide range of organisms, will we answer these kinds of questions. Challenging, yes, but exciting.

I thank Neil Metcalfe, Karen Spencer, Francois Criscuolo, Lawrence Weaver, Kate Arnold, John Wingfield and three referees for comments on an earlier draft of this manuscript.

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