J Ornithol
DOI 10.1007/s10336-015-1236-z

REVIEW

# Developmental stress: evidence for positive phenotypic and fitness effects in birds

**Ondi L. Crino[1] · Creagh W. Breuner[2]**

Received: 10 January 2015 / Revised: 23 March 2015 / Accepted: 6 May 2015


**Abstract** The developmental environment has strong and pervasive effects on animal phenotype. Exposure to stress during development (in the form of elevated glucocorticoid hormones or food restriction) is one environmental cue that can have strong formative effects on morphology, physiology, and behavior. Although many of the effects of developmental stress appear negative, there is increasing evidence for an adaptive role of developmental stress in shaping animal phenotype. Here, we take a three-pronged approach to review studies that have uncovered positive effects of developmental stress on phenotype in birds. We focus on studies that: (1) examine phenotypic effects likely to increase fitness in offspring, (2) directly identify increased fitness in offspring, or (3) provide evidence of fitness benefits to the mother, at a cost to the offspring. Throughout, we focus on studies that evaluate the environment when assessing the 'costs/benefits' of phenotype alterations and examine the effects of developmental stress across life-history stages. Finally, we consider the two common methods used to simulate developmental stress: food restriction and direct hormone manipulation. Although these methods are often considered to elicit equivalent responses, there has been very little discussion of this in the literature. To this end, we review the main methods used to implement developmental stress in experimental studies and discuss how they may simulate different environmental conditions. In light of our conclusions, we propose possible avenues for future research, stressing the need for a greater focus on direct fitness metrics, longitudinal studies, and experiments in free-living animals.

**Keywords** Birds · Corticosterone · Developmental stress · Fitness · Nestlings · Performance · Phenotypic plasticity

## Introduction

The environment animals experience during development has strong and pervasive effects on phenotype and fitness (Mousseau and Fox 1998; Sheldon 2002; Stamps 2003). Across taxonomic groups, the developmental environment has been shown to affect morphology, physiology, behavior, neural function, survival, and reproduction (e.g., Franzke and Reinhold 2013; Lucassen et al. 2013; Miller et al. 2012; Noble et al. 2014). In some systems, developmentally influenced phenotypic alterations have been shown to affect future generations through epigenetic mechanisms (e.g., Francis et al. 1999; Liu et al. 1997; Weaver et al. 2004). In this way, environmental cues experienced by an individual during development can modulate phenotype across generations, making the developmental environment a potential driver of rapid evolutionary change (Badyaev 2014; Prudic 2011; Sheldon 2002).

The effects of the developmental environment on animal phenotype have been studied extensively in the context of developmental stress (reviewed in Henriksen et al. 2011; Matthews 2002; Monaghan 2008; Nesan and Vijayan 2013; Schoech et al. 2011; Spencer and MacDougall-Shackleton

Communicated by E. Matthysen.

✉ Ondi L. Crino
ondi.crino@deakin.edu.au

[1] Centre for Integrative Ecology, Deakin University, Victoria, Australia

[2] Organismal Biology and Ecology, University of Montana, Missoula, MT, USA

Published online: 21 May 2015

2011). Although developmental stress is a broad term, it generally refers to any sub-optimal condition experienced during either the prenatal or postnatal developmental period, including: food restriction, elevated glucocorticoid stress hormones, extreme ambient temperature, varied incubation temperature, immune challenges, psychological disturbances such as altered paternal care and social contact, and anthropogenic disturbances (e.g., Banerjee et al. 2012; Buchanan et al. 2003; Crino et al. 2011; Fairhurst et al. 2012; Grindstaff et al. 2012; Honarmand et al. 2010; Lynn and Kern 2014; Pravosudov and Kitaysky 2006; Spencer et al. 2009; Walker et al. 2005a, 2005b; Weaver et al. 2004; Wilsterman 2015). The effects of developmental stress have been widely studied across taxonomic groups. In recent years, birds are emerging as a model system for investigations into how the early developmental environment shapes phenotype, because the distinct egg stage in their development allows for relative ease in separating prenatal versus postnatal effects. In birds, the majority of developmental stress studies have focused on food restriction and elevated corticosterone (CORT; the dominant avian glucocorticoid) as stressors (reviewed in MacDougall-Shackleton and Spencer 2012; Schoech et al. 2011). Although these two factors likely simulate different environmental conditions (see below), they are collectively referred to in the literature and hereafter as developmental stressors.

Historically, the effects of developmental stress on phenotype and fitness have predominantly been viewed as deleterious. For example, in birds, developmental stress decreases immune function, suppresses growth, decreases neural function, and can suppress the expression of sexually selected traits in adulthood (Buchanan et al. 2003; Carmona-Isunza et al. 2013; e.g., Chin et al. 2013; Crino et al. 2014a; but see Gil et al. 2006). Indeed, it is difficult to see how these effects are anything but negative. However, in recent years, studies have gone beyond examining the discrete phenotypic responses to developmental stress, to investigate how perturbations during development affect animals across life-history stages and in an ecological and evolutionary context (e.g., Crino et al. 2014b; Love and Williams 2008a, 2008b; Monaghan 2008; Monaghan et al. 2012; Sheriff and Love 2013). These studies have revealed that, rather than acting as a purely negative force shaping phenotype and fitness, developmental stress can be a cue that sets animals on a developmental pathway that matches phenotype to the postnatal environment in order to maximize fitness (i.e. developmental programming; Gluckman and Hanson 2004; McMillen and Robinson 2005; Nesan and Vijayan 2013).

Here, we take a three-pronged approach to review studies that have uncovered positive effects of developmental stress on phenotype in birds. First, we focus on studies examining phenotypic effects that are thought to increase fitness in offspring. Second, we consider studies that directly measure fitness (specifically, reproductive success) of those offspring as adults. And third, we consider the possibility that developmental stress may benefit the mother, at a cost to the offspring. Finally, although developmental stress is a term used broadly here and elsewhere, the type of stressors animals are exposed to during development (e.g., food restriction vs. CORT exposure) may indicate different things about the postnatal environment. We finish with a discussion of the two major methods used to induce developmental stress: food restriction and direct CORT administration. Whether or not developmental stress induces a positive or negative phenotypic effect is likely influenced by the context within which the trait is evaluated. Therefore, studies that evaluate the effects of developmental stress within an ecological context have much to add to our understanding of how developmentally induced traits affect fitness.

## First: a discussion of terms

The term ‘stress’ is often misleading, as it can refer to the application of challenge (the stressor), as well as the state of the organism experiencing challenge (Romero 2004). Additionally, the term stress is often used synonymously with glucocorticoids, as though glucocorticoids were the only physiological response to a stressor. In this paper, we refer to the stress-inducing treatments given during development as ‘stressors’, and to the condition immediately resulting as ‘developmental stress’. In this way, many different treatments (stressors) can each induce developmental stress, and the applications of glucocorticoids during development are a direct manipulation of developmental stress (the organismal state). When discussing hormonal responses, we either refer directly to glucocorticoids or to the glucocorticoid stress response.

## Developmental stress—positive phenotype and fitness effects

Developmental stress has been associated with seemingly deleterious consequences, such as reduced growth and development, impaired immunocompetence, altered hypothalamic-pituitary-adrenal activity, and impaired neurological function (Liu et al. 1997; Loiseau et al. 2008; Muller et al. 2009; Weaver et al. 2004). Although the phenotypic consequences of developmental stress appear overwhelmingly negative, there is increasing evidence for an adaptive function of developmental stress (reviewed in Breuner 2008). Specifically, it has been proposed that

developmental stress may induce phenotypic changes that prepare developing animals to live in harsh environments or match offspring needs to maternal capabilities (Gluckman and Hanson 2004; Love and Williams 2008a; McMillen and Robinson 2005; Sheriff and Love 2013). In these scenarios, developmental stress may modulate phenotypic changes that allow animals to maximize fitness in the environment that they are about to enter by doing a 'best of the bad job' (Monaghan 2008). Studies that examine the effects of developmental stress on fitness or fitness-related traits of both parents and offspring provide some of this first evidence that developmental stress promotes adaptive phenotypic responses.

## Phenotype studies

Developmental stress may cue offspring to program their phenotype for maximum fitness in harsh environments (Gluckman and Hanson 2004). For example, in birds, investing in systems that modulate flight performance could increase survival in predator dense environments where developing individuals may be exposed to higher levels of glucocorticoids. Experimental evidence for this has been described in barn swallows (*Hirundo rustica*), where egg-laying females exposed to a predator cue increased CORT deposition in eggs, resulting in smaller nestlings (Saino et al. 2005). Although smaller body size may appear detrimental, it could be advantageous to flight performance by decreasing wing loading (body mass/wing area), which enables birds to achieve faster speeds at take-off (e.g., Metcalfe and Ure 1995). Chin et al. (2009) found evidence of this in European starlings (*Sturnas vulgaris*), where elevated CORT in ovo caused juveniles to develop heavier pectoral muscles, have lower wing loading, and achieve higher energy gain on take-off compared to control birds (Fig. 1; Chin et al. 2009). Similarly, a study in great tits (*Parus major*) found that nestlings reared by mothers exposed to acoustic predator cues during egg laying were smaller and lighter and prioritized wing growth over other morphological structures compared to control nestlings (Coslovsky and Richner 2011). Cumulatively, these studies provide a compelling example of how developmental stress can have profound effects on performance traits important for fitness.

Other studies examining the effects of developmental stress on learning have provided further support for an adaptive role of the early environment in shaping fitness-related phenotypic traits. Historically, developmental stress has been strongly associated with reduced song learning in passerines (i.e. the developmental stress hypothesis; Nowicki et al. 1998; Spencer et al. 2005). Developmental stress decreases development of the brain regions that control song learning and production (Nowicki et al. 2002). Consequently, males exposed to stressors during development sing less attractive songs as adults and are less preferred by females. Although studies testing the developmental stress hypothesis provide compelling support that developmental stress has negative consequences on adult phenotype, recent studies examining the effects of developmental stress on non-song learning have revealed positive effects. For example, Crino et al. (2014b) found that adult zebra finches fed CORT as nestlings learned a novel foraging task faster compared to siblings exposed to a control treatment (Fig. 2a). Likewise, studies in Japanese quail (*Coturnix japonica*) and domesticated chickens (*Gallus gallus domesticus*) have also described positive effects of developmental stress on spatial (Fig. 2b) and associative learning (respectively; Calandreau et al. 2011; Goerlich et al. 2012). The effects of developmental stress on learning are clearly mixed with both positive and negative effects. Developmental stress may create resource trade-offs that cause individuals to invest in some neural structures at the expense of others during development (e.g., Sewall et al. 2013). In this scenario, developmental stress may decrease some types of learning (e.g., song learning), but increase other types (e.g., motoric learning, but see Bonaparte et al. 2011; Kitaysky et al. 2003). In stressful environments, trade-offs in types of learning may benefit survival at early life-history stages at the expense of reproductive success in later life-history stages. Despite a loss of reproductive success, greater survival could result in higher net fitness in the appropriate postnatal environment.

Similarly, the effects of developmental stress on glucocorticoid stress responsiveness have previously been considered negative, but may program animals to thrive in suboptimal environments. Numerous studies have found that developmental stress organizes the neuroendocrine pathway responsible for CORT output (the hypothalamic–pituitary–adrenal or HPA axis) such that animals exposed to stressors during development respond more strongly to stressors as adults (but see Crino et al. 2014a). For example, nestling zebra finches fed CORT during the nestling period respond to stressors with greater CORT output in adulthood (Kriengwatana et al. 2014; Spencer et al. 2009). Another study in house sparrows (*Melospiza melodia*), found that adults treated with CORT during the nestling period responded with greater CORT output compared to control birds (Schmidt et al. 2014). Higher CORT reactivity may benefit adults in sub-optimal environments: greater CORT reactivity predicts greater sensitivity to environmental perturbations, and may induce behavioral switches to promote survival sooner than in non-developmentally stressed conspecifics. Greater CORT reactivity has been associated with greater survival across species (e.g., Cabezas et al. 2007; Patterson et al. 2014), although counter examples exist (e.g., Blas et al. 2007). Again, we

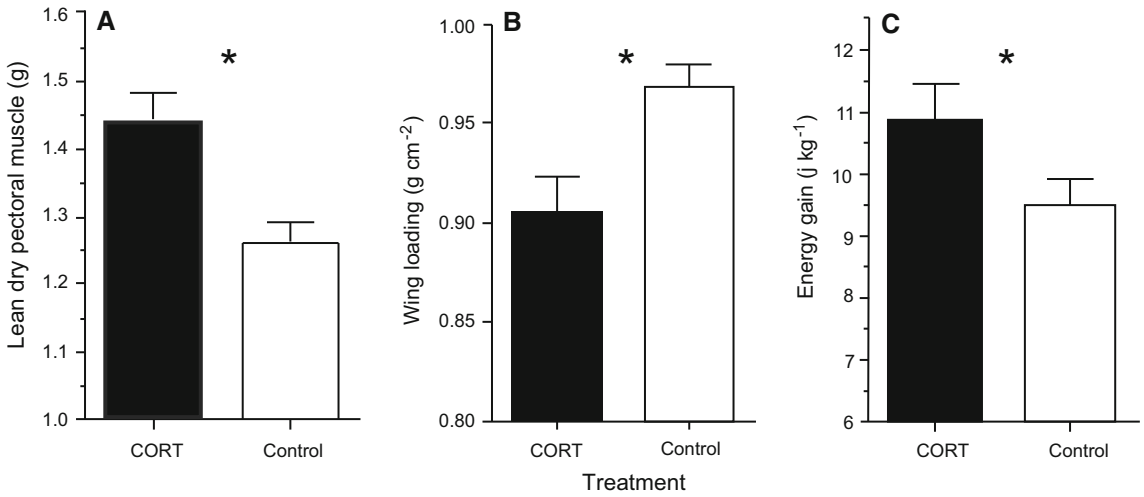


**Fig. 1** As adults, European starlings exposed to experimentally elevated CORT in ovo had **a** heavier pectoral muscles and **b** lower wing loadings. These phenotypic changes lead to **c** energy gains during take-off compared to control birds, possibly enhancing predator escape performance post-fledging. Figure redrawn from Chin et al. (2009)

believe it is important to test the effects of developmental stress in a context-appropriate adult environment. These altered phenotypes may not be beneficial under optimal conditions, but may increase survival under sub-optimal conditions (e.g., Sheriff and Love 2013).

## Fitness studies

Although studies examining the effects of developmental stress on proxies of fitness are informative, in order to truly understand how developmental stress affects fitness, studies need to directly capture measures of reproductive success and survival. Very few studies have extended phenotypic effects to fitness metrics. Recently, Crino et al. (2014c) measured the effects of developmental stress on reproductive success in male zebra finches. They experimentally manipulated developmental stress by feeding nestlings CORT. When these birds reached sexual maturity, control and CORT-treated males were put in common garden breeding experiments to determine how developmental stress affected reproductive success. Crino et al. (2014c) predicted that males exposed to developmental CORT would have less attractive sexually selected traits (colored check patches), and therefore, sire fewer offspring through extra pair copulations (EPCs). However, to compensate for a lower quantity of genetic offspring, Crino et al. predicted that developmentally stressed males could invest more heavily in parental care and raise offspring of greater condition. Indeed, nestlings raised by developmentally stressed males received more parental care and were in better condition compared to nestlings reared by control males (Fig. 3a). Surprisingly, developmentally stressed males also sired a greater number of offspring compared to control males (Fig. 3b). One explanation the authors proposed to explain this unexpected finding is that developmental stress may decrease longevity (Monaghan et al. 2012; Tissier 2014), causing developmentally stressed males to front load their overall reproductive investment early in their life (Crino et al. 2014c). This explanation is supported by a study by Monaghan et al. (2012) that found that developmentally stressed males had shorter lifespans compared to control males. Exciting new studies examining the effects of stress on the ability of animals to cope with oxidative stress may provide a mechanism to explain greater rates of senescence in animals exposed to developmental stress (Haussmann et al. 2012; Tissier 2014).

## Maternal benefits

The majority of developmental stress studies to date have focused on changes to offspring phenotype, considering the fitness costs to the offspring. However, changes in offspring phenotype may benefit the mother when environmental conditions are difficult. Hayward and Wingfield (2004) proposed that maternal stress may slow growth in offspring to enable the mother to raise all hatched young at lower cost to her when food availability is low. This is difficult to test under laboratory conditions, which usually are accompanied by ad libitum food availability. Love and Williams (2008a) evaluated this possibility with an elegant set of experiments in European starlings (*Sturnus vulgaris*). The authors manipulated yolk CORT and then evaluated maternal survival when mothers were challenged during the nestling phase (wings clipped to make foraging more difficult). Clipped mothers raising control young had

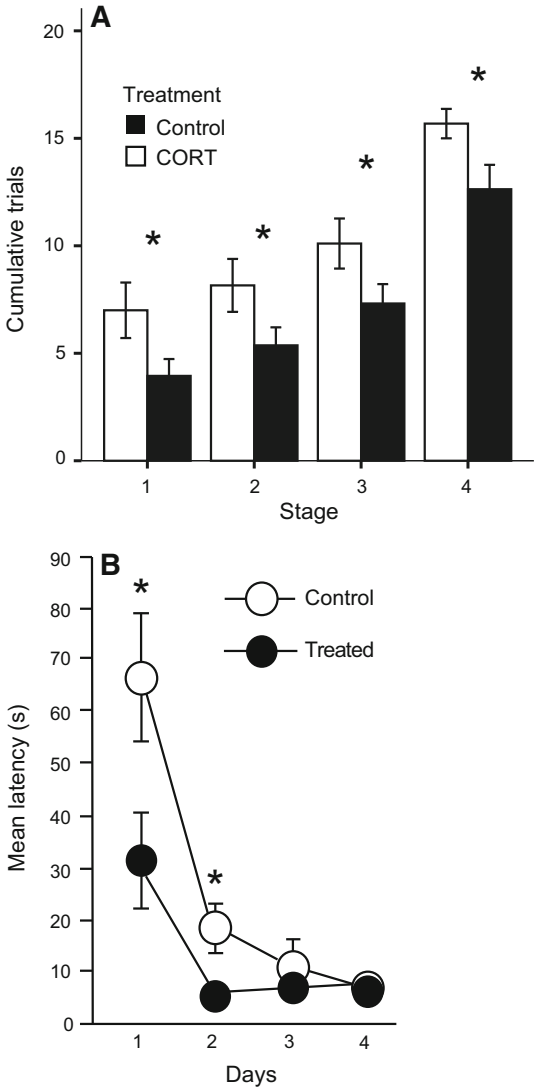


**Fig. 2** There are now several examples demonstrating an increase in learning ability with developmental stress: **a** developmentally stressed adult zebra finches learned a novel foraging task more rapidly than their control siblings (Crino et al. 2014b); **b** Japanese quail exposed to restraint stress during development had a shorter latency to locate a food reward (Calandreau et al. 2011). These examples run counter to previous work and demonstrate that developmental stress can have positive effects on learning. Figures redrawn from Crino at al. (2014b) and Calandreau et al. (2011)

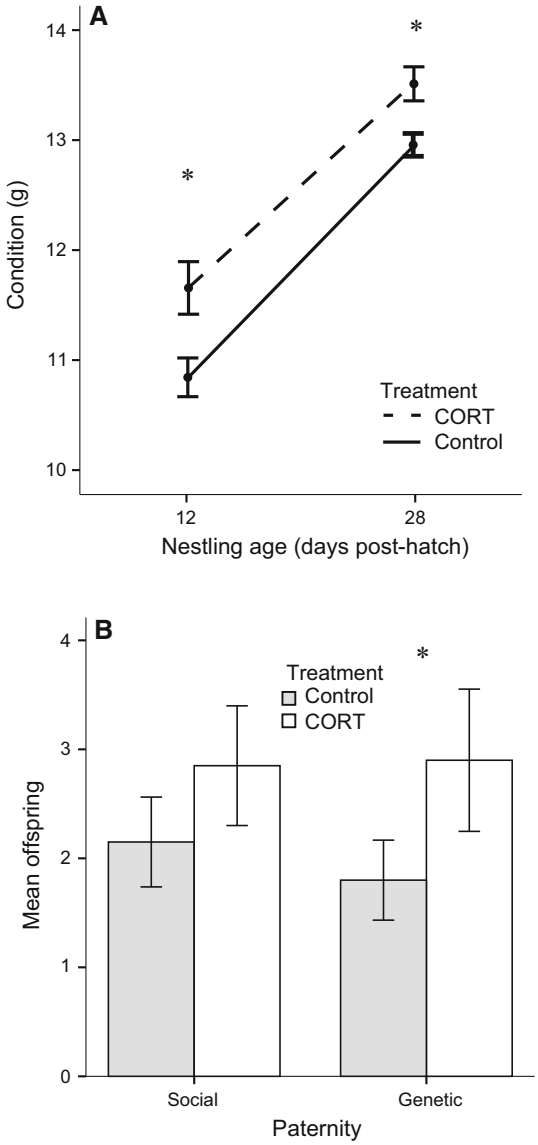


**Fig. 3** Male zebra finches exposed to stress during development **a** reared offspring in better condition, and **b** sired more offspring compared to control males. Figure redrawn from Crino et al. (2014c)

significantly lower annual survival rates compared to control (non-clipped) mothers. However, if a clipped mother was raising a CORT-injected brood (with smaller hatch size and slower growth rates), her annual survival matched that of control females, and was three-fold higher than clipped mothers raising control broods. Hence, phenotypic changes in offspring may exist to benefit the mother. Sheriff and Love (2013) extend these ideas in a review across vertebrate classes, suggesting productive avenues of research to advance the field.

## Experimental manipulation of stress—elevated CORT versus food restriction

Most studies that examine the effects of developmental stress on birds either restrict food or elevate prenatal or postnatal CORT exposure. The 'developmental stress hypothesis' was borne out of the food restriction literature (Nowicki et al. 1998), but has since been extended to include direct treatment with glucocorticoids (Spencer and MacDougall-Shackleton 2011) and other stressors, such as manipulations of social contact, immune and parasite challenges, and varied incubation temperatures (e.g., Banerjee et al. 2012; Grindstaff et al. 2012; Lynn and Kern

2014). These varied treatments are often referred to synonymously as 'developmental stressors', potentially because they result in many similar phenotypic effects; however, they likely simulate different environmental scenarios. Here, we describe the two primary methods used to induce developmental stress, and discuss the ways in which they may result in different outcomes.

### Direct treatment with Glucocorticoids

In vertebrates, the endocrine response to stress involves activation of the HPA axis and results in the release of glucocorticoid hormones (e.g., CORT in birds). Glucocorticoids modulate physiological responses and behaviors that allow animals to counter or cope with stressors (Wingfield et al. 1998). Like adults, embryos and nestlings secrete CORT in response to stressors, but the age when the HPA axis becomes fully activated is highly dependent on developmental mode (precocial vs. altricial). Experimental techniques used to manipulate CORT are divided primarily by prenatal vs. postnatal application. Prenatal treatment is generally done through maternal implant (e.g., Hayward and Wingfield 2004) or direct application to the egg (e.g., Love and Williams 2008a). Postnatal treatment is accomplished primarily through oral administration (e.g., Crino et al. 2014a; Spencer and Verhulst 2007), but dermal patches (e.g., Wada and Breuner 2008) and hormone implants (e.g., Roulin et al. 2008) have been used as well.

Prenatal CORT treatment simulates an environment that is stressful for the mother during egg production. Females exposed to stressors during egg development deposit greater amounts of CORT in egg yolk (Hayward and Wingfield 2004; Saino et al. 2005). In this way, experimentally increasing prenatal CORT levels simulates stressors that mothers may be experiencing prior to egg-laying rather than stressors that nestlings might experience post-hatching. Prenatal CORT treatment causes a wide variety of changes, including altered sex ratios, smaller body size that can persist through fledging, slower plumage development, and altered HPA-axis function as adults (Hayward and Wingfield 2004; Love and Williams 2008a; Saino et al. 2005).

Alternatively, postnatal CORT treatments (from dermal patches or oral administration) simulate a hormonal stress response that nestlings might experience in response to an environmental disturbance such as inclement weather, predation pressure, or anthropogenic disturbance. When nestlings are still confined to the nest, and therefore, unable to avoid noxious stimuli, elevated levels of CORT may promote changes in energy allocation such that nestlings prioritize development of some systems at the expense of others. For example, nestlings in predator-dense environments may prioritize the growth of wings over other body systems in order to decrease time to fledging and predation risk. Experimental evidence of this in great tits was described above, where nestlings reared by mothers exposed to acoustic predator cues during egg laying were smaller and lighter and prioritized wing growth over other morphological structures compared to control nestlings (Coslovsky and Richner 2011). Directly manipulating CORT in nestlings is an efficient method to investigate how nestling phenotype and fitness responds to any perturbation that increases endogenous CORT. Although there are clear links to why nestlings may experience elevated CORT in free-living conditions (e.g., inclement weather, anthropogenic disturbance, food restriction), the connection between these perturbations and the ultimate organismal consequences are often indirect and will benefit from comprehensive studies in free-living animals.

In the mammalian literature, prenatal and postnatal glucocorticoid manipulation can result in very different results (e.g., Vallee et al. 1997). There is growing evidence for this in the avian literature as well. Several recent studies have combined prenatal and postnatal manipulations to investigate how exposure to developmental stressors at these two developmental stages produces different outcomes and how they may interact. For example, in Japanese quail (*Cortunix japonica*), prenatal and postnatal stressors have different effects on adult HPA function. Adult quail exposed to prenatal stressors showed a greater attenuation of the CORT response over time compared to control birds, while birds exposed to postnatal stressors did not differ in CORT responses compared to control birds (Zimmer et al. 2013). Changes in HPA function in response to developmental stressors may stem from changes in mineralocorticoid (MR) and glucocorticoid (GR) receptors that affect negative feedback of the HPA axis. A recent study by Zimmer and Spencer (2014) found that developmental stressors had sustained effects on MR and GR receptor mRNA expression in the HPA axis in Japanese quail. These changes support a more efficient negative feedback of the HPA stress response and corroborate previous findings showing an attenuated physiological stress response following prenatal stress (Zimmer et al. 2013). In European starlings, prenatal stress decreases stress reactivity, while postnatal stress increases it (Love and Williams 2008b). Therefore, methods of manipulating CORT during the prenatal period may represent a different ecological context and result in different phenotypic effects than postnatal manipulation. An interesting issue in birds is that prenatal and postnatal stressors target different developmental stages, depending on whether the species has precocial or altricial development. This issue adds a level of complexity above what is found in the mammalian literature, and deserves more attention.

### Food restriction

Compared to elevating CORT either prenatally or postnatally, food restriction is a more ecologically relevant, but more complex stressor used to manipulate developmental stress. Food can be experimentally limited by artificially increasing clutch size, handicapping parents (e.g., feather clipping), or providing substandard food in laboratory studies. Food restriction is often associated with elevated levels of CORT in nestlings (e.g., Kitaysky et al. 2001; Walker et al. 2005a), and could provide a signal to nestlings to prioritize systems to enable faster fledging and decrease time to nutritional independence. In addition to eliciting elevated levels of CORT, however, food restriction also results in deficiencies of essential energy resources for optimal development. With limited food, developing animals face trade-offs in energy allocation such that they must devote resources to some systems at the expense of others. A classic example of this is the effects of food restriction on the development of the song system in passerines (i.e. the 'nutritional stress' hypothesis; Nowicki et al. 1998, 2002). Nestling passerines learn their species specific song during early development. Food restriction during the nestling period results in fewer resources devoted to the brain nuclei associated with song learning and production (e.g., Buchanan et al. 2004; Nowicki et al. 2002). Thus, only individuals that can cope well with stressors as nestlings are able to invest in neural structures associated with song learning (Nowicki et al. 2002); individuals that cope poorly with food restriction as nestlings sing less attractive songs and are less preferred by females (Spencer et al. 2005). Nestling food restriction has also been shown to reduce hippocampal volume and subsequent spatial memory tasks in adults (Pravosudov et al. 2005). Brain tissues have high metabolic demands, and nestlings faced with calorie and protein deficiencies may sacrifice developing these tissues in order to develop systems and structures more pertinent to immediate survival.

Comparing results from the two techniques (direct CORT treatment vs. food restriction) is risky. The first is a controlled treatment that alters one component of a complex pathway. The second is more ecologically relevant, but changes energy availability as well as, likely, a suite of hormone systems (such as those involved in stress, feeding, and energetics). Both techniques have strengths and weaknesses, and depending on the given question, one type of manipulation may be more appropriate than the other. For example, food restriction experiments (especially those where parents are handicapped or clutch size is experimentally increased) are more logistically feasible in free-living birds compared to CORT manipulations, which often employ methods that require nestlings be treated after they fledge from the nest (e.g., Crino et al. 2014a; Spencer et al. 2009). Food restriction, where parents are handicapped or provided with substandard food, also affects all nestlings in a nest and does not allow for intra-nest comparisons. In contrast, when CORT manipulations are used, nestlings are individually dosed, allowing for direct comparison to siblings. Beyond logistics, food restriction experiments allow researchers to comprehensively examine how an ecological relevant stressor affects both parental and nestling responses. In comparison, CORT manipulations provide a method for partly separating parental and nestling responses, allowing researchers to examine more specific outcomes. Both methods have their place, and although researchers are often limited in the approach they can take, studies that evaluate the effects of both food restriction and CORT manipulations in one experiment (e.g., Schmidt et al. 2014; Spencer et al. 2003) have much to add to our understanding of how these two approaches produces different phenotypic effects.

## Conclusions and future research directions

The adaptive potential of developmental stress may largely be influenced by the context within which phenotypic alterations are evaluated. The outcome could be dependent on environmental quality, or the life-history stage at which it is assessed. Phenotypic effects may have negative effects on fitness at early life-history stages, but positive effects at later life-history stages. In this way, the overall effect could be a neutral or net positive gain in fitness, but this would only be revealed by longitudinal studies. Similarly, developmentally induced phenotypic effects could have positive fitness effects in stressful postnatal environments and negative fitness effects in comparatively non-stressful environments. Admittedly, studies that examine the longitudinal fitness effects of developmental stress are logistically challenging, and few researchers have attempted such experiments (but see Monaghan et al. 2012). However, examining lifetime reproductive success is essential to addressing hypotheses about how developmental stressors affect fitness. Studies in captive birds more easily allow researchers to conduct such long-term studies and control the developmental environment. However, domesticated and captive birds can differ in remarkable ways compared to their wild congeners with respect to morphology, longevity, behavior, and reproduction (Forstmeier et al. 2007; Schutz 2001; Tschirren 2009). Future studies that evaluate the effects of developmental stress on fitness across life-history stages, especially in free-living birds, will add much to our understanding of the potential of developmental stress to drive evolutionary change.

**Acknowledgments** We would like to thank Kendra Sewall, Haruka Wada and Brit Heidinger for organizing the symposium on developmental stress. The Wildlife Biology Program at the University of Montana provided travel support to CWB to attend the IOC.

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