REVIEW AND SYNTHESIS

Determining the adaptive potential of maternal stress

M. J. Sheriff[1]* and **O. P. Love[2]**

[1]*Institute of Arctic Biology, University of Alaska Fairbanks, Fairbanks, AK, USA*

[2]*Department of Biological Sciences, University of Windsor, Windsor, ON, Canada*

**Correspondence:
E-mail: mjsheriff@alaska.edu*

Abstract

Ecological and medical researchers are investing great effort to determine the role of Maternally-Derived Stress (MDS) as an inducer of phenotypic plasticity in offspring. Many researchers have interpreted phenotypic responses as unavoidable negative outcomes (e.g., small birth weight, high anxiety); however, a biased underestimate of the adaptive potential of MDS-induced effects is possible if they are not viewed within an ecologically relevant or a life-history optimization framework. We review the ecological and environmental drivers of MDS, how MDS signals are transferred to offspring, and what responses MDS induces. Results from four free-living vertebrate systems reveals that although MDS induces seemingly negative investment trade-offs in offspring, these phenotypic adjustments can be adaptive if they better match the offspring to future environments; however, responses can prove maladaptive if they unreliably predict (i.e., are mismatched to) future environments. Furthermore, MDS-induced adjustments that may prove maladaptive for individual offspring can still prove adaptive to mothers by reducing current reproductive investment, and benefitting lifetime reproductive success. We suggest that to properly determine the adaptive potential of MDS, researchers must take a broader integrated life-history perspective, appreciate both the immediate and longer term environmental context, and examine lifetime offspring and maternal fitness.

INTRODUCTION

The adaptive value of a maternal effect as a driver of variation in offspring and maternal fitness is frequently an assumption in many evolutionary life-history studies (Fox & Mousseau 1998; Räsänen & Kruuk 2007). Often this assumption lacks rigorous testing, and significance is frequently assigned following only a short-term examination of the proximate effects on offspring phenotype (Marshall & Uller 2007), without a full understanding of life-history or even environmental context (Love *et al.* 2009, 2012). For example, although physiological traits have become the focus of many maternal effects studies (e.g., Groothuis *et al.* 2005; Williams 2008), we generally still lack strong models combining experimental manipulations and observational studies tracking free-living individuals over longer periods of time (Williams 2012). Determining the adaptive value of any maternal effect requires knowledge of: i) the environmental/ecological context inducing the effect, ii) the mechanism(s) by which environmental/ecological variation is translated into phenotypic changes in offspring, and iii) how these environmentally-induced effects influence offspring and maternal fitness within an appropriate life-history framework (Marshall & Uller 2007; Love & Williams 2008a).

Evolutionary biologists and medical practitioners are investing great effort to determine the role of Maternally-Derived Stress (MDS) as a significant inducer of trans-generational phenotypic plasticity in offspring (Gluckman *et al.* 2005; Meaney *et al.* 2007; Love & Williams 2008a; Sheriff *et al.* 2010, 2011; Love *et al.* 2012). Given their significant role in the management of homeostatic energy balance in vertebrates (Sapolsky *et al.* 2000; Landys *et al.* 2006), stress hormones (glucocorticoids – GCs) have been

proposed as a primary mechanistic translator providing offspring with information on MDS, and hence the quality of their immediate post-natal and even future environments (Love *et al.* 2005). Embryos in a wide range of taxa show developmental sensitivity following exposure to maternally-derived GCs (i.e., altered size, growth, physiology, behavior; see Love & Williams 2008a), and as such these hormones could play key roles by inducing adaptive phenotypic responses in offspring in relation to variation in maternal and environmental quality (Meylan & Clobert 2005; Love *et al.* 2009). However, short-term examinations of these phenotypic effects are commonly labeled as unavoidable negative side effects of exposure to MDS by biomedical researchers and many ecologists (Seckl & Meaney 2004; Meaney *et al.* 2007). Ecologists can significantly improve this perspective by experimentally examining the fitness consequences of MDS-induced phenotypic effects across multiple components of the life-histories of free-living species (see Harshman & Zera 2007 for general framework; Sinervo & DeNardo 1996; Love & Williams 2008a; Sheriff *et al.* 2010, 2011; Meylan *et al.* 2012; Fig. 1).

We expand the "Maternal Match Hypothesis" (Love & Williams 2008a; reviewed in Breuner 2008, 2010) towards a much broader "integrated life history perspective" (*sensu* Sinervo & DeNardo 1996; Lancaster *et al.* 2007) which simultaneously examines two life-history investment components linked by MDS: offspring phenotypic quality investment trade-offs and the maternal costs of reproduction (Fig. 1). A holistic examination of both components is critically important for properly determining the realized adaptive potential of MDS. For example, assigning the (commonly reported) MDS-induced reduction in offspring quality or quantity as maladaptive would be highly premature if this reduction in the
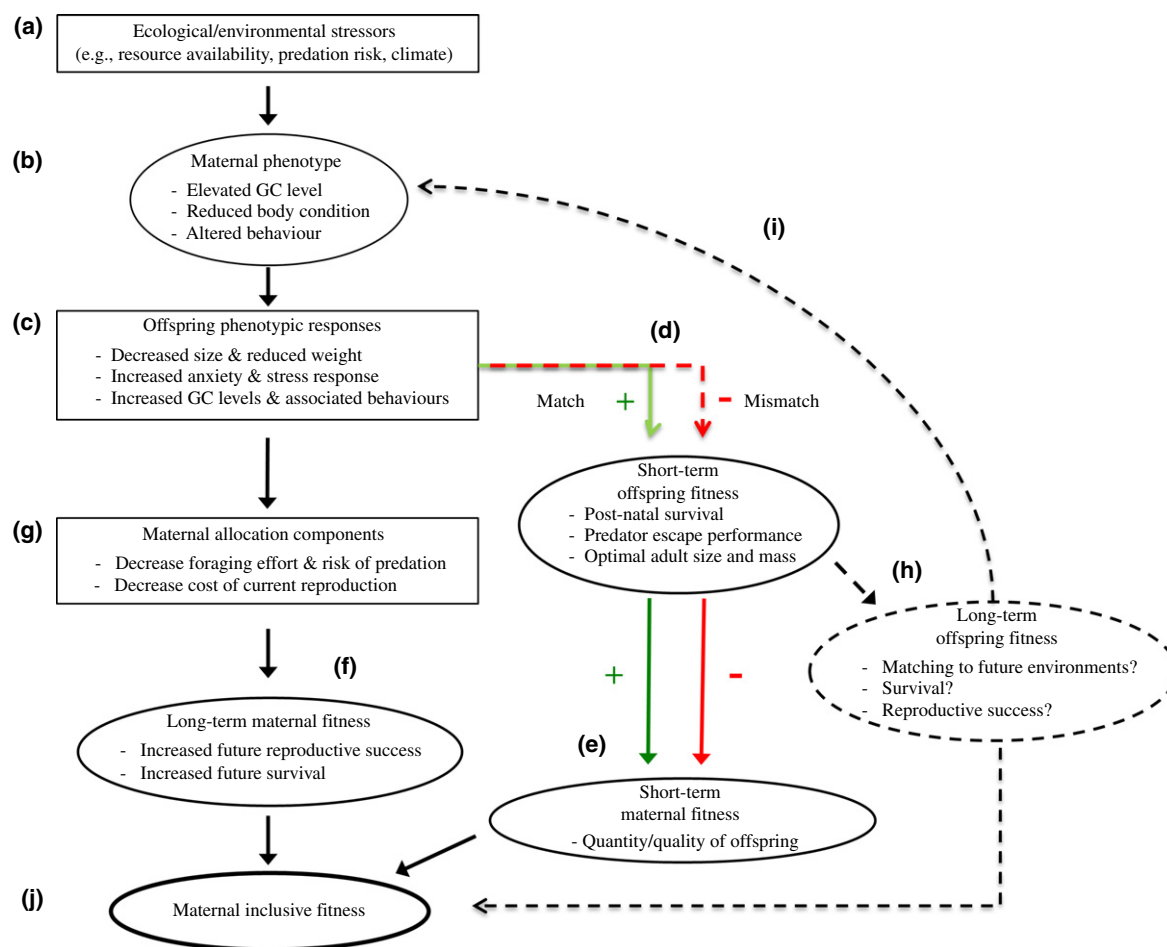
Fig 1 The potential impact of an ecological stressor on maternal inclusive fitness acting through maternally-derived stress (MDS)-mediated effects on both offspring and maternal life-history components. (a) Ecological/Environmental stressors alter (b) maternal phenotypes resulting in (c) MDS-induced phenotypic responses in offspring. Dependent upon the relative maternal-offspring environmental match these responses may positively or negatively impact (d) short-term offspring fitness. Short-term offspring fitness will directly impact (e) short-term maternal fitness and indirectly contribute to (f) long-term maternal fitness via changes to (g) maternal allocation components. Although not yet rigorously tested, short-term offspring fitness is expected to contribute to (h) long-term offspring fitness and impact (i) offspring phenotype as reproductive adults. (j) Together the sum of these components will contribute to maternal inclusive fitness.

current cost of reproduction improved future reproductive success or survival for mothers (Sinervo & DeNardo 1996). In this review we aim to uncover the means by which ecological researchers can help to determine the adaptive potential of MDS. Although important recent reviews have examined exogenous drivers on stress-induced phenotypes (e.g., Meylan *et al.* 2012), our goal was to examine specifically whether MDS acts to translate both endogenous and exogenous environmental variability into adaptive outcomes for mothers and offspring. We begin by briefly discussing the various ways in which ecological and environmental stressors cause increases in maternal GCs, and the means by which vertebrate offspring can be exposed to MDS during development. To demonstrate the phenotypic capacity and constraints that offspring face, we then examine the diversity of known offspring phenotypic responses to MDS and the importance of environmental context in mediating these responses. We then review four diverse and rigorously-tested ecological systems as models for examining the hypothesized evolutionary role of MDS in vertebrates within a life-history framework. Finally, to encourage robust experimental testing of an MDS-induced phenotypic matching paradigm across

ecological systems, we discuss the environmental and life-history prerequisites for MDS to produce adaptive outcomes for mothers and offspring.

GLUCOCORTICOIDS AS POTENTIAL ECOLOGICAL INTEGRATORS

Glucocorticoids are often referred to as 'stress' hormones in ecological studies since they have primarily been examined for their role in physiological and behavioral 'stress' responses to acute, unpredictable environmental stressors (Sapolsky *et al.* 2000; Romero 2002, 2004; Wingfield 2005). However, the primary metabolic role of baseline GCs on an hourly to daily basis, as their name implies, is to increase circulating glucose levels, primarily through increasing gluconeogenesis (Sapolsky *et al.* 2000). Vertebrates exhibit a daily rhythm in baseline GCs with highest levels in early morning, prior to feeding, as a means to induce glucose mobilization and behaviors that increase resource acquisition (Sapolsky *et al.* 2000). Elevated baseline GCs i) promote energy acquisition during demanding life-history stages such as breeding and migration (reviewed in Romero 2002; Holberton 1999; Pravosudov 2003; Breuner & Hahn 2003; Love *et al.* 2004, 2012), ii) are

often responsive to ecological (predator, resource, conspecifics, etc.) and environmental (weather, season, habitat, etc.) variation (Marra & Holberton 1998; Romero & Wikelski 2001; Kitaysky *et al.* 2007; Sheriff *et al.* 2011, 2012; Wasser *et al.* 2011; although see Romero *et al.* 2000) and iii) are often linked to reduced energetic state or body condition (Kitaysky *et al.* 1999a, b; Romero & Wikelski 2001; Love *et al.* 2005). Given their central role in energetic management and resource acquisition, baseline GCs have been proposed as significant mediators of state-mediated life-history trade-offs and decisions (Love *et al.* 2005) and are therefore predicted to act as key physiological mechanisms mediating life-history evolution (Zera & Harshman 2001; Ricklefs & Wikelski 2002; Love & Williams 2008a).

Ecological and environmental inducers of MDS

Although many ecological and environmental stressors can increase GCs (Breuner 2010), we focus on variables that ecologists habitually study, and those that have the greatest impact during key life-history stages including emerging human-induced stressors. Nonetheless, relatively few studies have measured variation in GCs in free-living female vertebrates at the pre-natal stage when offspring can be directly exposed to MDS (i.e., gestation, egg production).

Variability in predators and resources are well known physiological stressors in ecological systems (Clinchy *et al.* 2004; Hawlena & Schmitz 2010; Clinchy *et al.* 2012). An increase in the number of predators, the risk of predation, or even the perceived risk of predation can increase maternal GCs (mammals: Boonstra *et al.* 1998; Monclús *et al.* 2011; Sheriff *et al.* 2009, 2010, 2011; birds: Saino *et al.* 2005; Love *et al.* 2008; Travers *et al.* 2010; Zanette *et al.* 2011; fish: McCormick 1998; Giesing *et al.* 2011). Increases in maternal or female GCs can also occur directly via reductions in the quantity, quality and predictability of resources (Kitaysky *et al.* 1999b, 2007; Shultz & Kitaysky 2008; Jeanniard du Dot *et al.* 2009; Welcker *et al.* 2009), or indirectly via a reduction in maternal condition (de Fraipont *et al.* 2000; Meylan *et al.* 2002; Love *et al.* 2005, 2009; Monclús *et al.* 2011). Interactions between resources and predation risk can also act synergistically to increase GCs (Clinchy *et al.* 2004; Sheriff *et al.* 2010).

Social stressors also modulate circulating GCs in vertebrates (Sapolsky *et al.* 2000; Creel 2001; Creel *et al.* 2012). Subordinate reproductive females can show either elevated or lowered GCs to dominant females depending on a species' social system (social: Sapolsky 1992; Sapolsky *et al.* 2000; cooperatively breeding: Young *et al.* 2006; both: Creel 2001). Aggressive interactions and perceived increases in competition are known to increase maternal GCs in fish (McCormick 1998, 1999, 2006), reptiles (Comendant *et al.* 2003) and birds (Love *et al.* 2008).

Declines in habitat integrity are often related to elevated maternal GCs; however, complex interactions between resources and other extrinsic variables can complicate this relationship (Madliger & Love 2011). Elevated GCs are related to spatial changes in resource abundance/quality (Marra & Holberton 1998; Kitaysky *et al.* 1999a, b, 2007; Shultz & Kitaysky 2008) and increased human recreational and industrial activity (Creel *et al.* 2002; Thiel *et al.* 2008; Wasser *et al.* 2011). Theoretical work predicts direct and indirect links between climatic stressors and increases in GCs (Boonstra 2004; Wingfield 2008) and has suggested that trans-generational phenotypic plasticity, mediated through maternal effects, may allow animals to adapt to changing environments (Meylan *et al.* 2012). In support, Sheriff *et al.* (2012) reported that the timing of snowmelt and increased variation in spring climate appear to alter seasonal patterns of GC secretion in a free-living mammal.

Offspring exposure to MDS

Mothers can expose offspring to the environmental and ecological stressors they face through exposure to maternal GCs via the placenta in mammals and the yolk in non-mammalian vertebrates (Seckl & Meaney 2004; Almasi *et al.* 2012). The mammalian fetus appears to have some control over maternal GC exposure by placental expression of 11β-hydroxysteroid dehydrogenase type 2 (11β-HSD2; Burton & Waddell 1999), where 11β-HSD2 interconverts GCs (cortisol and corticosterone) to the inert forms cortisone and 11-dehydrocorticosterone, respectively (Funder 1996). However, when GCs increase beyond normal baseline limits, greater exposure to maternal GCs results from a decrease (or lack of increase) in placental expression of 11β-HSD2 (Lesage *et al.* 2001; Lucassen *et al.* 2009). Since maternal GC levels are up to 10-fold higher than those of the fetus (Owen *et al.* 2005), subtle changes in 11β-HSD2 activity may have profound effects on fetal GC exposure.

In oviparous species, maternal GCs may be easily transferred from mother to yolk given the lipophilic nature of GCs (McCormick 1998; Hayward & Wingfield 2004; Love *et al.* 2005, 2008; Almasi *et al.* 2012; although see Henriksen *et al.* 2011). Experimental increases in maternal GCs during egg production can increase GC concentration in the yolks and albumin of eggs (Hayward & Wingfield 2004; Love *et al.* 2005; Saino *et al.* 2005; Almasi *et al.* 2012). Further, unlike the potentially constant exposure and maternal-fetal feedback found in mammals, egg-bound embryos are only exposed to maternal GCs deposited into the egg during follicular recruitment. Unfortunately, little is known about the mechanisms of GC transfer between the mother and the egg (Groothuis *et al.* 2005) and even less is known about the potential mechanisms of GC transfer between the mother and offspring in viviparous species, although this likely occurs in a manner more similar to mammals than true oviparous species (Dauphin-Villemant & Xavier 1986; Meylan & Clobert 2005).

In addition to the direct exposure of developing offspring, MDS has the potential to alter maternal care of offspring which can have a profound impact on offspring, leading to changes in the stress (hypothalamic-pituitary-adrenal - HPA) axis and causing a potentially permanent increase in offspring GC levels (Weaver *et al.* 2004; Meaney *et al.* 2007; Love *et al.* 2012). Moreover, it is also possible that elevating maternal GCs alters the quantity/quality of resources invested in offspring before or after hatch/birth (Sinervo & DeNardo 1996). Although neither of these indirect influences of MDS on offspring phenotype are routinely investigated in free-living systems (although see Sinervo & DeNardo 1996), they nonetheless represent significant potential sources of MDS-related variation in offspring performance that could be measured relatively easily.

PHENOTYPIC RESPONSES OF OFFSPRING TO MDS

Phenotypic offspring responses to MDS exposure can be physiological, behavioral and morphological, and are commonly not mutually exclusive. Changes in offspring physiology and behavior involve direct effects on neural development, particularly in stress (HPA) axis and amygdala (Hayward & Wingfield 2004; Seckl & Meaney 2004; Meaney *et al.* 2007; Love & Williams 2008b; Sheriff *et al.*

2010) and can involve decreases glucocorticoid and mineralocorticoid receptor levels in the brain (critical components of the GC-HPA axis negative feedback loop) resulting in offspring with elevated GC levels, and altered HPA axis and amygdala brain activity (Matthews 2002; Weaver *et al.* 2004; Bogoch *et al.* 2007). In mammals, outcomes include increased anxiety, depression and fearfulness leading to decrease locomotor and exploratory activity in novel environments and associated impairment of spatial learning. Furthermore, MDS exposure increases cardiovascular tone and glucose production, and reduces insulin sensitivity (Lesage *et al.* 2001; Zagron & Weinstock 2006; Abe *et al.* 2007; Bogoch *et al.* 2007).

Exposure to MDS has also been linked to altered morphological structure in offspring including low birth weight and small structural size (Saino *et al.* 2005; Meaney *et al.* 2007; Love & Williams 2008a; Sheriff *et al.* 2009), as well as the capacity for morphological structures to function optimally (i.e., altered flight muscle morphology and physiology leading to reduced escape performance; Chin *et al.* 2009). Although the mechanisms for morphological changes are not well understood, GC regulation of key developmental pathways such as insulin-like growth factor I, the secretion of pituitary growth hormone and the production of thyroid-stimulating hormone, as well as the activation of glucocorticoid response elements and DNA methylation at various tissues are all thought to be involved (Byrne 2001; Porter & Dean 2001; Fowden *et al.* 1996; Meaney *et al.* 2007).

Importance of environmental context

Within the biomedical community, altered phenotypic traits are often viewed as negative functional outcomes of exposure to MDS representing fitness costs (Seckl & Meaney 2004; Meaney *et al.* 2007). However, from an evolutionary perspective, different phenotypes can achieve different fitness values, and their success largely depends upon the environment in which they occur (Gross 1996). For example, changes in phenotypic traits such as blood pressure, body size and/or anxiety are not fitness values unto themselves, but instead alter fitness depending upon their interaction with the environment. Thus, to appreciate how MDS-induced phenotypic responses impact fitness, the environmental context in which they are generated and occur in must be considered. For example, in high-predation environments, MDS inductions of greater anxiety/fearfulness and reduced locomotor and exploratory activities could translate into highly adaptive anti-predator behaviors which promote offspring survival. Giesing *et al.* (2011) found that stickleback females under greater predation risk produced eggs with greater GC levels and higher oxygen consumption, and as juveniles, the offspring exhibited tighter shoaling behaviour even before being exposed to a threat compared to control offspring. Thus, through MDS exposure, mothers may translate the environment they encounter into potentially adaptive phenotypic responses in their offspring. Alternatively, an MDS-induced phenotype may be maladaptive if the offspring encounter a different-than-predicted environment, providing a further test of the potential value of the phenotype. For example, within the stickleback system above, offspring born to females under greater predation risk had reduced anti-predator behaviour and lower survival when they were placed alone with a live predator (McGhee *et al.* 2012). These divergent fitness outcomes from the same MDS-induced phenotypic trait exemplify the need to examine phenotypic responses within the environmental context that they are induced and experienced. The stickleback results are a reminder that MDS-induced phenotypes have the potential to produce both adaptive and maladaptive fitness outcomes dependent upon the predictability of future environments (i.e., phenotype-environment (mis)match).

EVALUATING THE ADAPTIVE POTENTIAL OF MDS-INDUCED PHENOTYPES

Here we review results from four free-living study systems that have used biologically- and species-relevant experimental manipulations to test the adaptive potential of MDS. Importantly, many have examined both offspring phenotypic quality investment trade-offs and their impact on maternal costs of reproduction, and how these integrative life-history components influence fitness.

Snowshoe hares and the 10-year predator-prey population cycle

The predator-prey system of the snowshoe hare was the first mammalian system studied to examine how MDS effects in free-living prey function under fluctuating predation risk (Sheriff *et al.* 2009, 2010, 2011; Fig. 2). Snowshoe hare populations undergo a regular cyclic fluctuation, with 8–10 years between peak densities (Krebs *et al.* 1995; Fig. 2A). As hare populations increase so do those of their predators, but with a lag of 1–2 years (Fig. 2A). During the hare population decline, predators are the direct cause of nearly all hare deaths. Following the decline phase, hare populations remain inexplicably low for 2–5 years despite predator numbers being low and vegetation ample (Krebs *et al.* 1995). Hare reproduction also cycles: maximum reproductive output occurs during the early increase phase (when predator numbers are lowest), progressively declines to a nadir during the decline (when predator numbers are highest; Krebs *et al.* 1995), and does not increase again until the late low-phase. During the decline phase, the greater risk of predation increases maternal GCs (Sheriff *et al.* 2011; Fig. 2B, b), and predator-induced MDS results in a decline in litter size, and offspring birth weight and size (Sheriff *et al.* 2009; Fig. 2C, D, d). Furthermore, prenatally-stressed offspring have higher GCs and a greater stress response compared to offspring that are not prenatally stressed, and these effects persist into adulthood (Sheriff *et al.* 2010; Fig. 2E, e). Although seemingly negative, these phenotypic responses in offspring appear to be highly advantageous during the decline phase when both the maternal and offspring environment are matched under high predation. The lower reproductive output would increase maternal (and thus offspring) survival and fitness by decreasing foraging and provisioning time, thus reducing maternal exposure to predators. The increase in offspring stress (and anti-predator behaviours associated with prenatal MDS exposure) would increase offspring survival (Fig. 2G). Conversely, giving birth to fewer, smaller offspring with a greater stress response at the end of the decline phase and beginning of the low phase when there is a mismatch between maternal and offspring environment (mothers experience high predation during the decline but their offspring experience very low predation during the low phase) would be highly disadvantageous (Fig. 2H). This combination of MDS-induced effects and the degree of maternal-offspring environmental mismatch during the transition phase is likely a major factor affecting the lack of reproductive, and hence population, recovery when predators are virtually absent and food is abundant. Considering MDS-induced phenotypic effects within recognized environmental contexts emphasizes that adaptive advantages when mothers and offspring are environmentally-matched can nonetheless become maladaptive if a mismatch occurs.
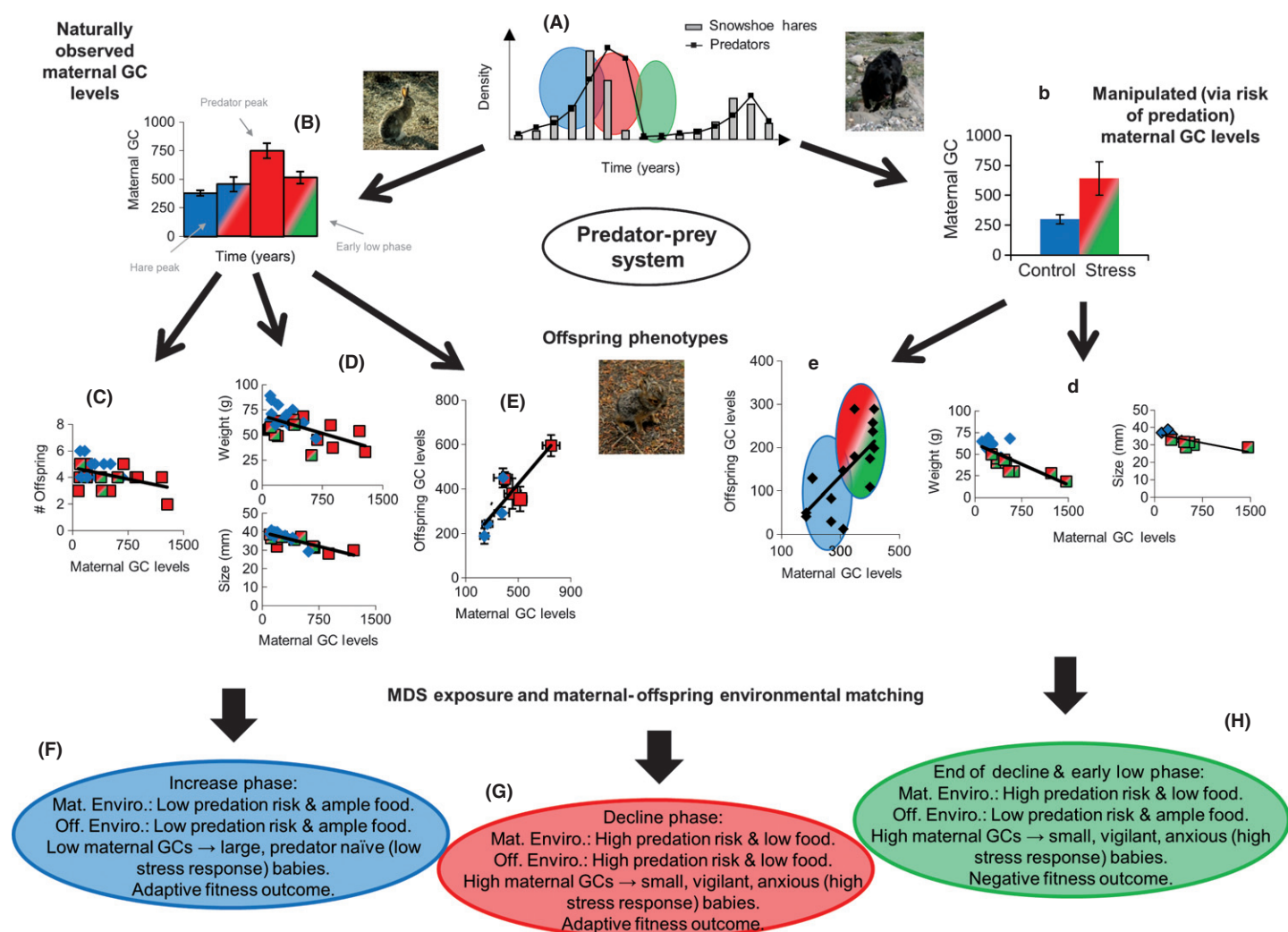
Fig 2 Changes in maternal GC levels and MDS exposure in the predator-prey system of the snowshoe hare. As the hare population declines, the risk of predation increases (a) directly affecting maternal GC levels (B, b). Mothers with greater GC levels (naturally (b) or via an experimental manipulation (b)) give birth to fewer (c), smaller (D, d) babies with compromised stress-axis (E, e). When the maternal-offspring environment match (F, G), MDS cues may be adaptive, producing offspring with the appropriate phenotype. However, during transitions between phases, such as from the decline to the low phase (H), MDS-exposure results in maladaptive phenotypes. This system illustrates the necessity of considering the resultant phenotype of MDS exposure in the appropriate environmental context.

Reef fish, social competition and growth-mortality trade-offs

Tropical reef fish exist in areas with high species diversity and population density, especially during the breeding season, and therefore provide an excellent model system to examine how intra- and inter-specific socially-mediated MDS exposure influences offspring phenotype and fitness (McCormick 1998 and references there-in). In the tropical damselfish (*Ponacentrus amboinensis*), males guard a demersal nest of eggs contributed to by many nearby females, including a dominant female that contributes the majority of eggs. The density of females interacting with breeding mothers positively influences maternal cortisol levels and directly reduces larval size through MDS exposure (McCormick 1998, 2006), without affecting yolk size (Gagliano & McCormick 2009). During the pelagic larval stage (lasting between 15–23 days), where intense size-selected mortality occurs, survival is greatest for small juveniles with large yolk sacs (Gagliano et al. 2007). In areas of high density, dispersal away from the natal location is likely beneficial, but would require a large larval:yolk reserve size given the potentially longer time spent in the pelagic phase. Thus, contrary to the idea that 'bigger-is-always-better', a density-mediated MDS-induced reduction in hatching larval size is adaptive because it is coupled with greater relative energetic yolk reserves. Conversely, large size and faster growth rates during the initial reef settlement stage increases survival (Holmes & McCormick 2006; Gagliano et al. 2007), likely due to decreased vulnerability to gape-limited predation. Low density areas with reduced social interactions are associated with unstressed mothers that produce larger offspring (McCormick 1999, 2006) where reduced dispersal distances are likely beneficial. Results further emphasize that to determine the adaptive potential of MDS-induced changes in offspring size and quality, fitness outcomes must be measured across life-history stages and interpreted with respect to the prenatal environment in which they were induced.

Lizards, population density and juvenile philopatry

Studies in lizards linking MDS and proximate effects in offspring have made some of the most important contributions to disentangling the possible evolutionary role of MDS (Dufty et al. 2002). In 1996, Sinervo & DeNardo reported that exogenous GC administration to female oviparous side-blotched lizards (Uta stansburiana) increased energy invested into clutches resulting in larger female hatchlings. Furthermore, this MDS-induced alteration of current reproductive investment appeared to have downstream effects on maternal survival, with the direction of the effect being dependent upon environmental conditions (e.g., population density) within a given year (Sinervo & DeNardo 1996). Since population density increases female plasma GCs in this species, Comendant et al. (2003) proposed that the MDS-induced increase in female hatchling size may be an adaptive mechanism to increase offspring survival in a highly competitive environment. Supporting this supposition, Clobert and colleagues have shown that although MDS exposure reduces male offspring size, body condition and growth in the viviparous common lizard (Lacerta vivipara) it increases survival (Meylan & Clobert 2005; Coté et al. 2006). Further, they found higher juvenile philopatry in offspring born to older and poor body condition mothers exhibiting higher GCs (de Fraipont et al. 2000; Meylan et al. 2002). They suggested that a chronic elevation of maternal GCs during pregnancy signals a mother's poor survival prospects following birth, reducing expected parent-offspring competition indicating that an MDS-induced increase in offspring philopatry would be adaptive (Meylan & Clobert 2005). As such, MDS exposure has the potential to alter meta-population and population dynamics by influencing recruitment or subsequent life-history characteristics (Meylan et al. 2012).

European starlings and the maternal match hypothesis

Experimental work in European starlings (Sturnus vulgaris) was the first to simultaneously manipulate MDS-induced offspring phenotype and the future environment those offspring expected to face to test whether MDS-induced phenotypes benefited offspring (Love et al. 2005; Love & Williams 2008a,b; Love et al. 2012). Researchers found a negative relationship between maternal quality and maternal baseline GCs (Love et al. 2005; Love & Williams 2008b; Fig. 3A), and that maternal GCs were proportionally transferred to yolks (Love et al. 2005; Fig. 3A). An experimental elevation of yolk GCs (Fig. 3B) induced a sex-specific trade-off between the quantity and quality of offspring, where the usually larger and faster-growing male offspring in this sexually-size dimorphic species were smaller at hatching, grew more slowly and had higher early post-natal mortality, all apparently maladaptive side-effects of MDS exposure (Love et al. 2005; Love & Williams 2008a; Fig. 3C). However, when MDS-exposed males were paired with low quality mothers, those that survived outperformed control counterparts for a number of fitness-related phenotypic metrics (Love & Williams 2008a; Chin et al. 2009). Importantly, at parental independence MDS-exposed males showed no costs to the body mass or size traits important for future intra-sexual competition despite having hatched at a smaller mass (Love & Williams 2008a). Although mothers paired to these MDS-exposed males raised fewer offspring in the current attempt, the optimized maternal-offspring match benefitted mothers via trade-offs between current reproductive investment and (i) future condition, (ii) future reproductive investment, and (iii)

survival influenced by the cost of reproduction; i.e. MDS-matched mothers (i) were in better body condition during second broods, (ii) produced more and better quality offspring in future attempts, and (iii) had higher inter-annual survival measured across multiple years (Love & Williams 2008a; Fig. 3D). Taken together, improving the match between offspring phenotype and maternal quality via MDS exposure ultimately increased maternal inclusive fitness (Fig. 3D), even when some of the short-term offspring responses such as reductions in size and growth rate appeared maladaptive (Love & Williams 2008a).

THE ROLE OF LIFE-HISTORY AND ENVIRONMENTAL VARIATION

Life-history traits and environmental circumstances that increase the temporal or spatial similarity between prenatal and future environments are expected to improve the adaptive potential of MDS. Environmental circumstances can represent both extrinsic (e.g., habitat quality, climate, predation risk) and intrinsic factors inherent to individual mothers (e.g., maternal condition/quality). Adaptive responses to MDS are possible when intra-annual environmental predictability is high (Love et al. 2009) where offspring can benefit from relying on prenatal signals to predict their developmental future. Conversely, in consistent environments (high inter-annual predictability), there is no benefit for variability in phenotypes to begin with. The relative strength of the MDS cue also informs offspring about its reliability as a signal: considerable or chronic exposure to MDS may be indicative of a tangible, persistent reduction in environmental conditions that offspring should respond to, whereas minor or ephemeral environmental stressors could be ignored with little consequence. Notably, it may not necessarily be the absolute change in MDS that is important for offspring to respond, but rather the relative change in the MDS signal from background (baseline) levels.

Variation in key life-history traits (developmental mode, mating system, reproductive investment; Stearns 1992) should determine the adaptive potential of MDS at the species and individual level (Love et al. 2009). Variation in developmental mode alters the temporal match between prenatal and future environments: r-selected animals (faster maturing, short-lived) should be more heavily influenced by the quality of the prenatal environment than k-selected (slower maturing, longer-lived) animals which may encounter a variety of environments during their lifetime (see examples in Love et al. 2009). Likewise, species with high natal philopatry (i.e., spatial match between parental-adult environments) should rely on MDS cues more heavily than species with high rates of juvenile dispersal. Similarly, income-breeding species where pre- and post-natal resources match strongly should rely on MDS cues more heavily than capital-breeding species. Variation in mating systems likely fine-tunes the adaptive value of MDS, rather than driving its absolute occurrence, depending on the relative differences in the proximate and ultimate value of raising sons vs. daughters and which sex provides parental care. For example, selection on the phenotypic responses to MDS is expected to be much stronger in sons than daughters in polygynous species with strong reproductive skew and sexual size dimorphism, where there are significantly greater costs to producing (and being) a male offspring (Love et al. 2005; Love & Williams 2008a). Conversely, in polyandrous species where maternal quality may have little to do with the degree of post-natal care, signals of MDS may not translate into reliable predictions of male paternal care. Finally, variation in reproductive investment
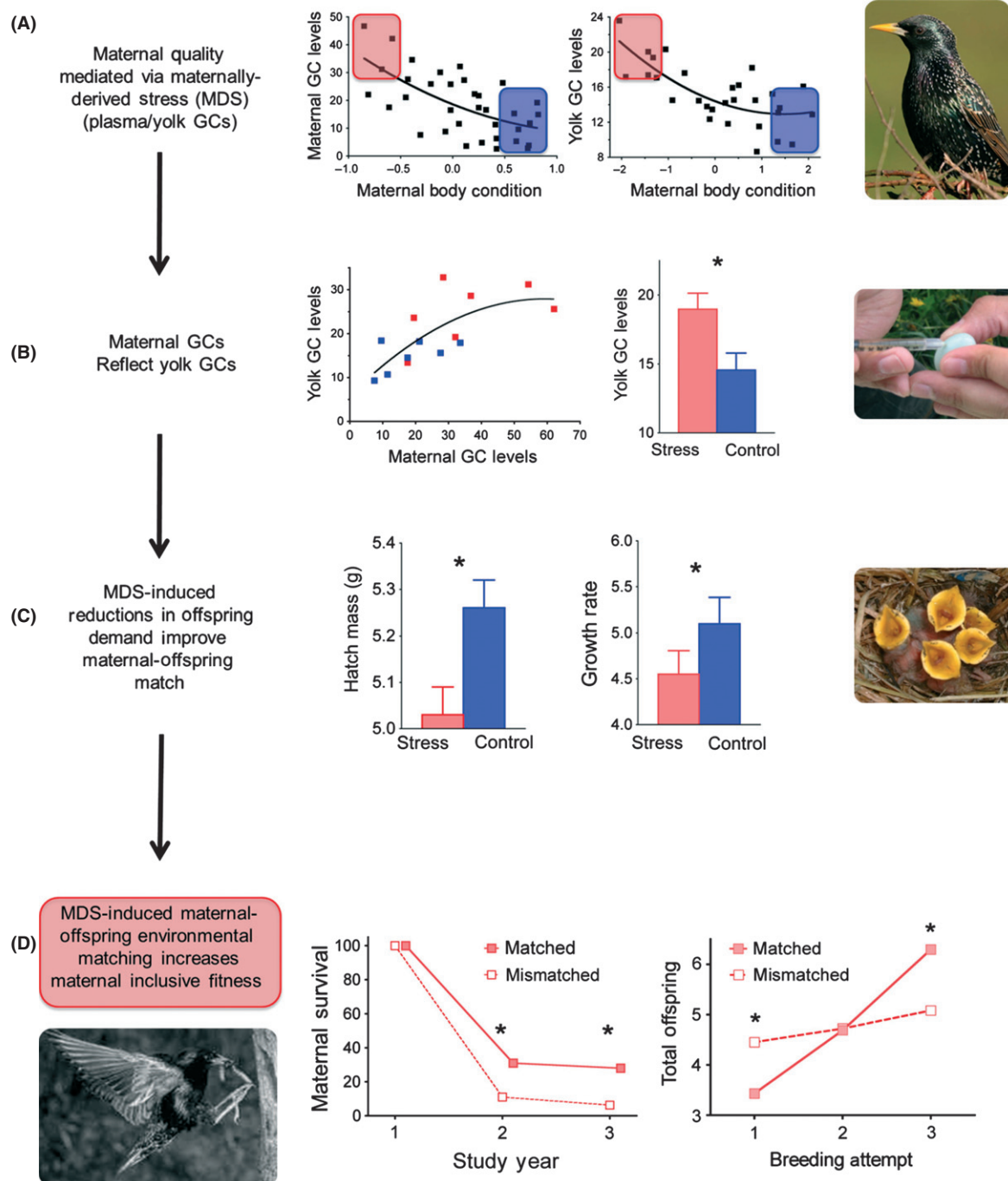
Fig 3 Maternally-Derived Stress (MDS - as represented by maternal and yolk Glucocorticoids - GCs) mediates the link between maternal quality (e.g., body condition) and offspring phenotypic effects to maximize maternal inclusive fitness in European starlings. Maternal body condition-Glucocorticoid (GC) relationships (A) were used to experimentally increase offspring's exposure to MDS via elevation of yolk GC levels (B). This reduced developmental demand by male offspring via lower hatching body masses and slower growth rates (C). Pairing MDS-exposed offspring to mothers with experimentally-reduced rearing capacity revealed that MDS improves the match between offspring demand and maternal quality increasing maternal inclusive fitness, compared to mismatched mothers (D). Based on and redrawn from Love et al. (2005); Love and Williams (2008a).

(e.g., quantity or quality of young) will profoundly adjust the adaptive value of MDS as a cue of maternal capacity since it will alter inter-offspring competition (Love et al. 2009), a potential reason for the presence of large intra-clutch variation in MDS signals for species with strong post-natal competition (Love et al. 2009).

UNRAVELING THE REALIZED ADAPTIVE POTENTIAL OF MDS

Exposure to MDS and its effects on offspring phenotype appears to be a widespread phenomenon across vertebrate taxa. MDS-induced phenotypic effects can be adaptive for offspring if they reli-

ably predict future environments (i.e., when the maternal environment matches that of the offspring; Love *et al.* 2005; Love & Williams 2008a); however, when MDS is a poor predictor of the future environment for offspring (i.e., when the maternal and offspring environment become mismatched), MDS exposure can produce maladaptive phenotypic outcomes in offspring (Sheriff *et al.* 2009, 2010). Importantly, even when MDS-induced effects appear maladaptive for offspring (i.e., slower growth rates, increased mortality) they can nonetheless be adaptive for mothers since they may optimize the cost of current reproductive investment thereby maximizing lifetime fitness (Love & Williams 2008a). Taking a broad "integrated life history perspective" (*sensu* Sinervo & DeNardo 1996; Lancaster *et al.* 2007) is therefore critically important for determining the realized adaptive potential of MDS (Fig. 1). Within this framework, we encourage ecologists to: (i) recognize the prenatal/maternal environmental context that induces MDS, (ii) use biologically- and species-relevant experimental manipulations of MDS in free-living systems to test the relative match between phenotypes and environmental variation, (iii) appreciate that variation in life-histories will influence the shape of MDS-fitness relationships, and (iv) recognize that the potential adaptive value of MDS exposure may occur at the level of both the offspring and mother. From a human health and disease perspective, the robust evolutionary ecological perspective we have outlined here has the potential to significantly improve our understanding of the Developmental Origins of Health and Disease (DOHaD), where a potentially preventable mismatch between the prenatal and future environment results in anticipatory responses in offspring that become maladaptive and lead to elevated risks of disease in adulthood (Low *et al.* 2012).

ACKNOWLEDGEMENTS

AUTHORSHIP

Both MJS and OPL contributed equally to the ideas and writing of this paper.

REFERENCES

Abe, H., Hidaka, N., Kawagoe, C., Odagiri, K., Watanabe, Y., Ikeda, T., Ishizuka, Y., Hashiguchi, H., Takeda, R., Nishimori, T. & Ishida, Y. (2007). Prenatal psychological stress causes higher emotionality, depression-like behavior, and elevated activity in the hypothalamo-pituitary-adrenal axis. *Neurosci. Res.*, 59, 145–151.

Almasi, B., Rettenbacher, S., Müeller, C., Brill, S., Wagner, H. & Jenni, L. (2012). Maternal corticosterone is transferred into the egg yolk. *Gen. Comp. Endocrinol.*, 178, 139–144.

Bogoch, Y., Biala, Y.N., Linial, M. & Weinstock, M. (2007). Anxiety induced by prenatal stress is associated with suppression of hippocampal genes involved in synaptic function. *J. Neurochem.*, 101, 1018–1030.

Boonstra, R. (2004). Coping with changing northern environments: the role of the stress axis in birds and mammals. *Integr. Comp. Biol.*, 44, 95–108.

Boonstra, R., Hik, D., Singleton, G.R. & Tinnikov, A. (1998). The impact of predator-induced stress on the snowshoe hare cycle. *Ecol. Monogr.*, 68, 371–394.

Breuner, C.W. (2008). Maternal stress, glucocorticoids, and the maternal/fetal match hypothesis. *Horm. Behav.*, 54, 485–487.

Breuner, C.W. & Hahn, T.P. (2003). Integrating stress physiology, environmental change, and behavior in free-living sparrows. *Horm. Behav.*, 43, 115–123.

Breuner, C.W. (2010). *Stress and Reproduction in Birds*. In: *Hormones and Reproduction in Vertebrates* (eds Norris, D.O.). Vol 4, chapter 5, Elsevier, Oxford.

Burton, P.J. & Waddell, B.J. (1999). Dual function of 11B-hydroxysteroid dehydrogenase in placental: modulating placental glucocorticoid passage and local steroid action. *Biol. Reprod.*, 60, 234–240.

Byrne, C.D. (2001). Programming other hormones that affect insulin. *Brit. Med. Bull.*, 60, 234–240.

Chin, E.H., Love, O.P., Verspoor, J.J., Williams, T.D., Rowley, K. & Burness, G. (2009). Juveniles exposed to embryonic corticosterone have enhanced flight performance. *Proc. R. Soc. Lond. B.*, 276, 499–505.

Clinchy, M., Zanette, L., Boonstra, R., Wingfield, J.C. & Smith, J.N.M. (2004). Balancing food and predator pressure induces chronic stress in songbirds. *Proc. R. Soc. Lond. B.*, 271, 2473–2479.

Clinchy, M., Sheriff, M.J. & Zanette, L. (2012). Predator induced stress and the ecology of fear. *Funct. Ecol.*, DOI: 10.1111/1365-2435.12007.

Comendant, T., Sinervo, B., Svensson, E.I. & Wingfield, J. (2003). Social competition, corticosterone and survival in female lizard morphs. *J. Evol. Biol.*, 16, 948–955.

Coté, J., Clobert, J., Meylan, S. & Fitze, P.S. (2006). Experimental enhancement of corticosterone levels positively affects subsequent male survival. *Horm. Behav.*, 49, 320–327.

Creel, S. (2001). Social dominance and stress hormones. *Trends Ecol. Evol.*, 16, 491–497.

Creel, S., Fox, J.E., Hardy, A., Sands, J., Garrot, B. & Peterson, R.O. (2002). Snowmobile activity and glucocorticoid stress responses in wolves and elk. *Conserv. Biol.*, 16, 809–814.

Creel, S., Dantzer, B., Goymann, W. & Rubenstein, D.R. (2012). Ecological processes and the ecology of stress: the impact of the social environment. *Funct. Ecol.*, (in press).

Dauphin-Villemant, C. & Xavier, F. (1986). Adrenal activity in the female *Lacerta vivipara* Jacquin: Possible involvement in the success of gestation. In: *Endocrine Regulation as Adaptive Mechanisms to the Environment* (eds Assenmacher, I. & Boissin, J.) CNRS, Paris, pp. 241–250.

de Fraipont, M., Clobert, J., John-Alder, H. & Meylan, S. (2000). Increased prenatal maternal corticosterone promotes philopatry of offspring in common lizard *Lacerta vivipara*. *J. Anim. Ecol.*, 69, 404–413.

Dufty, A.M., Clobert, J. & Møller, A.P. (2002). Hormones, developmental plasticity and adaptation. *Trends Ecol. Evol.*, 17, 190–196.

Fox, C.W. & Mousseau, T.A. (1998). Maternal effects as adaptations for transgenerational phenotypic plasticity in insects. In: *Maternal effects as adaptations* (eds Mousseau, A. & Fox, C.W.). Oxford University Press, New York, pp. 159–177.

Fowden, A.L., Giussani, D.A. & Forhead, A.J. (1996). Intrauterine programming of physiology systems: causes and consequences. *Physiology.*, 21, 29–37.

Funder, J.W. (1996). 11b-Hydroxysteroid dehydrogenase: new answers, new questions. *Euro. J. Endocrinol.*, 134, 267–268.

Gagliano, M. & McCormick, M.I. (2009). Hormonally mediated maternal effects shape offspring survival potential in stressful environments. *Oecologia.*, 160, 657–665.

Gagliano, M., McCormick, M.I. & Meekan, M.G. (2007). Survival against the odds: ontogenetic changes in selective pressure mediate growth-mortality trade-offs in a marine fish. *Proc. R. Soc. B.*, 274, 1575–1582.

Giesing, E.R., Suski, C.D., Warner, R.E. & Bell, A.M. (2011). Female sticklebacks transfer information via eggs: effects of maternal experience with predators on offspring. *Proc. R. Soc. Lond. B.*, 278, 1753–1759.

Gluckman, P.D., Hanson, M.A. & Spencer, H.G. (2005). Predictive adaptive responses and human evolution. *Trends Ecol. Evol.*, 20, 527–533.

Groothuis, T.G.G., Müller, W., von Engelhardt, N., Carere, C. & Eising, C. (2005). Maternal hormones as a tool to adjust offspring phenotype in avian species. *Neurosci. Biobehav. Rev.*, 29, 329–352.

Gross, M.R. (1996). Alternative reproductive strategies and tactics: Diversity within sexes. *Trends Ecol. Evol.*, 11, 92–98.

Hawlena, D. & Schmitz, O.J. (2010). Physiological stress as a fundamental mechanism linking predation to ecosystem functioning. *Am. Nat.*, 175, 537–556.

Harshman, L.G. & Zera, A.J. (2007). The cost of reproduction: the devil in the details. *Tr. Ecol. Evol.*, 22, 80–86.

Hayward, L.S. & Wingfield, J.C. (2004). Maternal corticosterone is transferred to avian yolk and may alter offspring growth and adult phenotype. *Gen. Comp. Endocrinol.*, 135, 365–371.

Henriksen, R., Rettenbacker, S. & Groothuis, T.G.G. (2011). Prenatal stress in birds: Pathways, effects, function and perspectives. *Neurosci. Biobehav. Rev.*, 35, 1484–1501.

Holberton, R.L. (1999). Changes in patterns of corticosterone secretion concurrent with migratory fattening in a neotropical migratory bird. *Gen. Comp. Endocrinol.*, 116, 49–58.

Holmes, T.H. & McCormick, M.I. (2006). Location influences size-selective predation on newly settled reef fish. *Mar. Ecol. Prog. Ser.*, 317, 203–209.

Jeanniard du Dot, T., Rosen, D.A.S., Richmond, J.P., Kitaysky, A.S., Zinn, S.A. & Trites, A.W. (2009). Changes in glucocorticoids, IGF-I and thyroid hormones as indicators of nutritional stress and subsequent refeeding in Steller sea lions (*Eumetopias jubatus*). *Comp. Biochem. Physiol. A.*, 152, 524–534.

Kitaysky, A.S., Piatt, J.F., Wingfield, J.C. & Romano, M. (1999a). The adrenocortical stress-response of black-legged kittiwake chicks in relation to dietary restrictions. *J. Comp. Physiol. B.*, 169, 303–310.

Kitaysky, A.S., Wingfield, J.C. & Piatt, J.F. (1999b). Dynamics of food availability, body condition and physiological stress response in breeding black-legged kittiwakes. *Funct. Ecol.*, 13, 577–584.

Kitaysky, A.S., Piatt, J.F. & Wingfield, J.C. (2007). Stress hormones link food availability and population processes in seabirds. *Mar. Ecol. Prog. Ser.*, 352, 245–258.

Krebs, C.J., Boutin, S., Boonstra, R., Sinclair, A.R.E., Smith, J.N.M., Dale, M.R. T., Martin, K. & Turkington, R. (1995). Impact of food and predation on the snowshoe hare cycle. *Science.*, 269, 1112–1115.

Lancaster, L.T., McAdam, A.G., Wingfield, J.C. & Sinervo, B. (2007). Adaptive social and maternal induction of antipredator dorsal patterns in a lizard with alternative social strategies. *Ecol. Lett.*, 10, 798–808.

Landys, M.M., Ramenofsky, M. & Wingfield, J.C. (2006). Actions of glucocorticoids at a seasonal baseline as compared to stress-related levels in the regulation of periodic life processes. *Gen. Comp. Endocrinol.*, 148, 132–149.

Lesage, J., Blondeau, B., Grino, M., Bréant, B. & Dupouy, J.P. (2001). Maternal undernutrition during late gestation induces fetal overexposure to glucocorticoids and intrauterine growth retardation, and disturbs the hypothalamo-pituitary adrenal axis in the newborn rat. *Endocrinology.*, 142, 1692–1702.

Love, O.P., Breuner, C.W., Vézina, F. & Williams, T.D. (2004). Mediation of a corticosterone-induced reproductive conflict. *Horm. Behav.*, 46, 59–65.

Love, O.P., Chin, E.H., Wynne-Edwards, K.E. & Williams, T.D. (2005). Stress hormones: a link between maternal condition and sex-biased reproductive investment. *Am. Nat.*, 166, 751–766.

Love, O.P., Wynne-Edwards, K.E., Bond, L. & Williams, T.D. (2008). Determinants of within- and among-clutch variation of yolk corticosterone in the European starling. *Horm. Behav.*, 53, 104–111.

Love, O.P. & Williams, T.D. (2008a). The adaptive value of stress-induced phenotypes: effects of maternally derived corticosterone on sex-biased investment, cost of reproduction, and maternal fitness. *Am. Nat.*, 172, E135–E149.

Love, O.P. & Williams, T.D. (2008b). Plasticity in the adrenocortical response of a free-living vertebrate: the role of pre- and post-natal developmental stress. *Horm. Behav.*, 54, 496–505.

Love, O.P., Gilchrist, H.G., Bêty, J., Wynne-Edwards, K.E., Berzins, L. & Williams, T.D. (2009). Using life-histories to predict and interpret variability in yolk hormones. *Gen. Comp. Endocrinol.*, 163, 169–174.

Love, O.P., McGowan, P. & Sheriff, M.J. (2012). Maternal adversity and ecological stressors in natural populations: the role of stress axis programming in individuals, with implications for populations and communities. *Funct. Ecol.*, DOI: 10.1111/j.1365-2435.2012.02040.x

Low, F.M., Gluckman, P.D. & Hanson, M.A. (2012). Developmental plasticity, epigenetics and human health. *Evol. Biol.*,(in press).

Lucassen, P.J., Bosch, O.J., Jousma, E., Kramer, S.A., Andrew, R., Seckl, J.R. & Neumann, I.D. (2009). Prenatal stress reduces postnatal neurogenesis in rats selectively bred for high, but not low, anxiety: possible key role of placental 11β-hydroxysteroid dehydrogenase type 2. *Eur. J. Neurosci.*, 29, 97–103.

Madliger, C. & Love, O.P. (2011). Links between baseline stress physiology, habitat quality, and fitness in an aerial insectivore. Frontiers in Endocrinology Conference Abstract: NASCE 2011: The inaugural meeting of the North American Society for Comparative Endocrinology.

Marra, P.P. & Holberton, R.L. (1998). Corticosterone levels as indicators of habitat quality: effects of habitat segregation in a migratory bird during the non-breeding season. *Oecologia.*, 116, 284–292.

Marshall, D.J. & Uller, T. (2007). When is a maternal effect adaptive?. *Oikos.*, 166, 1957–1963.

Matthews, S.G. (2002). Early programming of the hypothalamo-pituitary-adrenal axis. *Trends Endocrinol. Metab.*, 13, 373–380.

McCormick, M.I. (1998). Behaviourally induced maternal stress in a fish influences progeny quality by a hormonal mechanism. *Ecology.*, 79, 1873–1883.

McCormick, M.I. (1999). Experimental test of the effect of maternal hormones on larval quality of a coral reef fish. *Oecologia.*, 118, 412–422.

McCormick, M.I. (2006). Mothers matter: crowding leads to stressed mothers and smaller offspring in marine fish. *Ecology.*, 87, 1104–1109.

McGhee, K.E., Pintor, L.M., Suhr, E.L. & Bell, A.M. (2012). Maternal exposure to predation risk decreases offspring antipredator behaviour and survival in threespined stickleback. *Funct. Ecol.*, 26, 932–940.

Meaney, M.J., Szyf, M. & Seckl, J.R. (2007). Epigenetic mechanisms of perinatal programming of hypothalamic-pituitary-adrenal function and health. *Trends Mol. Med.*, 13, 269–277.

Meylan, S. & Clobert, J. (2005). Is corticosterone-mediated phenotype development adaptive? Maternal corticosterone treatment enhances survival in male lizards. *Horm. Behav.*, 48, 44–52.

Meylan, S., Belliure, J., Clobert, J. & de Fraipont, M. (2002). Stress and body condition as prenatal and postnatal determinants of dispersal in the common lizard (*Lacerta vivipara*). *Horm. Behav.*, 42, 319–326.

Meylan, S., Miles, D.B. & Clobert, J. (2012). Hormonally mediated maternal effects, individual strategy and global change. *Phil. Trans. R. Soc. B.*, 367, 1647–1664.

Monclús, R., Tiulim, J. & Blumstein, D.T. (2011). Older mothers follow conservative strategies under predator pressure: the adaptive role of maternal glucocorticoids in yellow-bellied marmots. *Horm. Behav.*, 60, 660–665.

Owen, D., Andrews, M.H. & Matthews, S.G. (2005). Maternal adversity, glucocorticoids and programming of neuroendocrine function and behaviour. *Neurosci. Biobehav. Rev.*, 29, 209–226.

Porter, T.E. & Dean, K.J. (2001). Regulation of chicken embryonic growth hormone secretion by corticosterone and triiodothyronine: evidence for a negative synergistic response. *Endocrine*, 14, 363–368.

Pravosudov, V.V. (2003). Long-term moderate elevation of corticosterone facilitates avian food-caching behaviour and enhances spatial memory. *Proc. R. Soc. Lond. B.*, 270, 2599–2604.

Räsänen, K. & Kruuk, E.B. (2007). Maternal effects and evolution at ecological time-scales. *Funct. Ecol.*, 21, 408–421.

Ricklefs, R.E. & Wikelski, M. (2002). The physiology-life-history nexus. *Trends Ecol. Evol.*, 17, 462–468.

Romero, L.M. (2002). Seasonal changes in plasma glucocorticoid concentrations in free-living vertebrates. *Gen. Comp. Endocrinol.*, 128, 1–24.

Romero, L.M. (2004). Physiological stress in ecology: lessons from biomedical research. *Trends Ecol. Evol.*, 19, 249–255.

Romero, L.M. & Wikelski, M. (2001). Corticosterone levels predict survival probabilities of Galápagos marine iguanas during El Niño events. *Proc. Natl. Acad. Sci.*, 19, 7366–7370.

Romero, L.M., Reed, J.M. & Wingfield, J.C. (2000). Effects of weather on corticosterone responses in wild free-living passerine birds. *Gen. Comp. Endocrinol.*, 118, 113–122.

Saino, N., Romano, M., Ferrari, R.P., Martinelli, R. & Møller, A.P. (2005). Stressed mothers lay eggs with high corticosterone levels which produce low-quality offspring. *J. Exp. Zool.*, 303A, 998–1006.

Sapolsky, R.M. (1992). Cortisol concentrations and the social significance of rank instability among wild baboons. *Psychoneuroendocrinology.*, 17, 701–709.

Sapolsky, R.M., Romero, L.M. & Munck, A.U. (2000). How do glucocorticoids influence stress responses? Integrating permissive, suppressive, stimulatory, and preparative actions. *Endocr. Rev.*, 21, 55–89.

Seckl, J.R. & Meaney, M.J. (2004). Glucocorticoid programming. *Ann. N.Y. Acad. Sci.*, 1032, 63–84.

Sheriff, M.J., Krebs, C.J. & Boonstra, R. (2010). The ghosts of predators past: population cycles and the role of maternal programming under fluctuating predation risk. *Ecology.*, 91, 2983–2994.

Sheriff, M.J., Krebs, C.J. & Boonstra, R. (2009). The sensitive hare: sublethal effects of predator stress on reproduction in snowshoe hares. *J. Anim. Ecol.*, 78, 1249–1258.

Sheriff, M.J., Krebs, C.J. & Boonstra, R. (2011). From process to pattern: how fluctuating predation risk impacts the stress axis of snowshoe hares during the 10-year cycle. *Oecologia.*, 166, 593–605.

Sheriff, M.J., Wheeler, H., Donker, S.A., Krebs, C.J., Palme, R., Hik, D. & Boonstra, R. (2012). Mountain-top and valley-bottom experiences: the stress axis as an integrator of environmental variability in arctic ground squirrel populations. *J. Zool.*, 287, 65–75.

Shultz, M.T. & Kitaysky, A.S. (2008). Spatial and temporal dynamics of corticosterone and corticosterone binding globulin are driven by environmental heterogeneity. *Gen. Comp. Endocrinol.*, 155, 717–728.

Sinervo, B. & Denardo, D.F. (1996). Costs of reproduction in the wild: Path analysis of natural selection and experimental tests of causation. *Evolution.*, 50, 1299–1313.

Stearns, S.C. (1992). *The evolution of life histories*. Oxford University Press, Oxford, 249 pp.

Thiel, D., Jenni-Eiermann, S., Braunisch, V., Palme, R. & Jenni, L. (2008). Ski tourism affects habitat use and evokes a physiological stress response in capercaillie *Tetrao urogallus*: a new methodological approach. *J. Appl. Ecol.*, 45, 845–853.

Travers, M., Clinchy, M., Zanette, L., Boonstra, R. & Williams, T. (2010). Indirect predator effects on clutch size and the cost of egg production. *Ecol. Lett.*, 13, 980–988.

Wasser, S.K., Keim, J.L., Taper, M.L. & Lele, S.R. (2011). The influences of wolf predation, habitat loss, and human activity on caribou and moose in the Alberta oil sands. *Front. Ecol. Environ.*, 9, 546–551.

Weaver, I.C.G., Cervoni, N., Champagne, F.A., D'Alessio, A.C., Sharma, S., Seckl, J.R., Dymov, S., Szyf, M. & Meaney, M.J. (2004). Epigentic programming by maternal behavior. *Nature Neurosci.*, 7, 847–854.

Welberg, L.A.M. & Seckl, J.R. (2001). Prenatal stress, glucocorticoids and the programming of the brain. *J. Neurosci.*, 13, 113–128.

Welcker, J., Harding, A.M.A., Kitaysky, A.S., Speakman, J.R. & Gabrielsen, G.W. (2009). Daily energy expenditure increases in response to low nutritional stress in an Arctic-breeding seabird with no effect on mortality. *Funct. Ecol.*, 23, 1081–1090.

Williams, T.D. (2008). Individual variation in endocrine systems: moving beyond the 'tyranny of the Golden Mean'. *Phil. Trans. R. Soc. B.*, 363, 1687–1698.

Williams, T.D. (2012). *Physiological adaptations for breeding in birds*. Princeton University Press, Princeton & Oxford, 379pp.

Wingfield, J.C. (2005). The concept of allostasis: coping with a capricious environment. *J. Mammal.*, 86, 248–254.

Wingfield, J.C. (2008). Comparative endocrinology, environment and global change. *Gen. Comp. Endocrinol.*, 157, 207–216.

Young, A.J., Carlson, A.A., Monfort, S.L., Russell, A.F., Bennett, N.C. & Clutton-Brock, T. (2006). Stress and the suppression of subordinate reproduction in cooperatively breeding meerkats. *Proc. Natl. Acad. Sci., U.S.A.*, 103, 12005–12010.

Zanette, L.Y., White, A.F., Allen, M.C. & Clinchy, M. (2011). Perceived predation risk reduces the number of offspring songbirds produce per year. *Science.*, 334, 1398–1401.

Zagron, G. & Weinstock, M. (2006). Maternal adrenal hormone secretion mediates behavioural alterations induced by prenatal stress in male and female rats. *Behav. Brain Res.*, 175, 323–328.

Zera, A.J. & Harshman, L.G. (2001). The physiology of life-history trade-offs in animals. *Annu. Rev. Ecol. Syst.*, 32, 95–126.