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THE ECOLOGY OF STRESS

Life history and the ecology of stress: how do glucocorticoid hormones influence life-history variation in animals?

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Summary

1. Glucocorticoids hormones (GCs) are intuitively important for mediation of age-dependent vertebrate life-history transitions through their effects on ontogeny alongside underpinning variation in life-history traits and trade-offs in vertebrates. These concepts largely derive from the ability of GCs to alter energy allocation, physiology and behaviour that influences key life-history traits involving age-specific life-history transitions, reproduction and survival.

2. Studies across vertebrates have shown that the neuroendocrine stress axis plays a role in the developmental processes that lead up to age-specific early life-history transitions. While environmental sensitivity of the stress axis allows for it to modulate the timing of these transitions within species, little is known as to how variation in stress axis function has been adapted to produce interspecific variation in the timing of life-history transitions.

3. Our assessment of the literature confirms that of previous reviews that there is only equivocal evidence for correlative or direct functional relationships between GCs and variation in reproduction and survival. We conclude that the relationships between GCs and life-history traits are complex and general patterns cannot be easily discerned with current research approaches and experimental designs.

4. We identify several future research directions including: (i) integration of proximate and ultimate measures, including longitudinal studies that measure effects of GCs on more than one life-history trait or in multiple environmental contexts, to test explicit hypotheses about how GCs and life-history variation are related and (ii) the measurement of additional factors that modulate the effects of GCs on life-history traits (e.g. GC receptors and binding protein levels) to better infer neurendocrine stress axis actions.

5. Conceptual models of HPA/I axis actions, such as allostatic load and reactive scope, to some extent explicitly predict the role of GCs in a life-history context, but are descriptive in nature. We propose that GC effects on life-history transitions, survival probabilities and fecundity can be modelled in existing quantitative demographic frameworks to improve our understanding of how GC variation influences life-history evolution and GC-mediated effects on population dynamics

Key-words: stress, reproduction, survival, fitness, vertebrates

Introduction

Evolution has resulted in a phenomenal diversity in organisms' life histories, the products of interactions between an organism's intrinsic and extrinsic environment (Roff 1992; Stearns 1992). Intrinsic factors include resource allocation trade-offs among life-history traits (e.g. age-specific growth, reproduction and survival schedules) delimited by genetic variation enmeshed within an organism's phylogeny.

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Extrinsic factors are the environmental, ecological and ensuing demographic processes that create forces of selection acting on intrinsic processes (Stearns 2000; Zera & Harshman 2001). Cumulatively these factors define differences in transitions between life-history stages, the relative investment in competing life-history traits and ultimately variation in an organism's lifetime fitness. Complex interactions among phylogenetic constraints, macroevolutionary processes (e.g. environmental selection gradients) and contemporary ecological and demographic factors imply that optimizing life-history strategies of organisms involves multivariate selection. Therefore, a major challenge for understanding life-history evolution is to understand the proximate physiological mechanisms that link genotype and phenotype and hence integrate extrinsic factors into regulation of trade-offs among survival and reproduction. Physiological, and especially endocrine, mechanisms are key to understanding how life-history variation and trade-offs might arise and are maintained (Ketterson & Nolan 1992, 1999; Zera & Harshman 2001).

The glucocorticoid (GC) hormones corticosterone and cortisol produced by the hypothalamo-pituitary-adrenal axis (HP-interrenal axis in amphibians and fish, HPA/I axis) are broadly implicated in mediating the physiology and behaviour underlying transitions between life-history stages, as well as variation in the expression of and trade-offs among life-history traits. This is because GCs play a fundamental role in regulation of energy homoeostasis in relation to 'predictable' life-history events (e.g. growth, reproduction, migration; McEwen & Wingfield 2003). GCs are typically measured in two contexts. First, *baseline* activity of the HPA/I axis is related to an organism's energetic state because GCs play an integral role in regulating circulating glucose levels, and baseline GC levels are expected to correspond to variation in energetic demand in different life-history stages or seasonal activity patterns (reviewed in Sapolsky, Romero & Munck 2000; McEwen & Wingfield 2003). The action of the HPA/I axis is also important for the acquisition of food resources, as baseline GC concentrations are often be associated with circadian patterns of appetite and foraging behaviours (reviewed in Sapolsky, Romero & Munck 2000).

The second, and far more widely context in which GCs are measured in ecology is in *stress-induced* situations in which GCs coordinate of physiological and behavioural responses to unpredictable environmental changes or perceived challenges (i.e. emergency life-history stage *sensu* Wingfield *et al.* 1998). In this state, the HPA/I is one of the most important physiological systems that mediates processes needed to meet energetic demands and resource allocation trade-offs during environmental challenges (Wingfield & Sapolsky 2003). For example, in some animals, the trade-off between survival (self maintenance) and reproduction during acute or chronic stress are modulated by GCs inhibiting the hypothalamo-pituitary-gonadal axis (e.g. Wingfield & Romero 2000; Cox *et al.* 2010). GCs have also been implicated in mediating trade-offs in current versus future reproduction (e.g. Bókony *et al.* 2009), as well as energetic trade-offs between physiological systems (e.g. reproduction and immune function, French *et al.* 2007) that might influence the expression of life-history trade-offs between reproductive output, growth and survival.

These two functional domains of the HPA/I axis affect the expression of various life-history traits, and the differential activity of this axis has been invoked to explain intra- and interspecific variation in traits. McEwen & Wingfield (2003) and Romero, Dickens & Cyr (2009) have proposed the allostatic load and reactive scope models, respectively, that illustrate the interactions between HPA/I axis activity and environment across the life history of an organism, and there is increasing empirical work supporting the application of these models in diverse contexts (e.g. Romero 2002; Bókony *et al.* 2009; Hau *et al.* 2010). However, recent reviews consistently highlight the lack of uniform relationships between GCs and reproduction or survival among vertebrates (Breuner, Patterson & Hahn 2008; Bonier *et al.* 2009a), suggesting relationships between GCs and life-history traits are neither direct nor simple as is often hypothesized. Here, we focus on four topics that are critical to assessing the role of the HPA/I axis in the expression of life-history traits (fecundity and reproductive effort, survival) and trade-offs among these traits in vertebrates. We limited this review to these life-history traits, although we recognize that the relationships between GC levels and other physiological systems (e.g. immune function) and behaviour are also worthy of critical review; and while life span is an important life-history trait, we refer the reader to reviews on stress and ageing published elsewhere (e.g. Costantini *et al.* 2010; Haussmann & Marchetto 2010). Specifically, we aim to:

1. evaluate evidence that GCs mediate age-dependent transitions among life-history stages;
2. evaluate evidence that GCs mediate phenotypic variation in life-history traits within and between species, and whether GCs mediate trade-offs between life-history traits;
3. consider the utility and limitations of conceptual models of HPA/I axis actions in a life-history context, such as allostatic load (McEwen & Wingfield 2003, 2010) and reactive scope (Romero, Dickens & Cyr 2009) models; and
4. identify future research directions that contribute to elucidating the role of the HPA/I axis in mediating life-history transitions and life-history variation in vertebrates.

Do GCs mediate age-dependent life-history transitions?

The timing of important developmental phases, such as the age at birth, hatching, fledging or metamorphosis, and the age at sexual maturity are important life-history traits

(Stearns 1992). In addition to the key role of GCs as metabolic regulators described earlier, there is a large accumulation of empirical support for the idea that the neuroendocrine stress axis is an evolutionarily conserved regulator of transitions in early life-history stages across vertebrates (Denver 2009). Elevation in HPA/I axis activity has been described prior to metamorphosis in amphibians and fish, birth in mammals (reviewed in Crespi & Denver 2005a; Wada 2008), and hatching in most oviparous vertebrates (birds, reptiles and some amphibians) but not in fish (reviewed in Wada 2008; Ledon-Rettig, Pfennig & Crespi 2009). Increases in GCs have also been associated with the transition between freshwater to saltwater life-history stages in anadromous fish (reviewed in Wada 2008). Experiments in which GCs were elevated prior to metamorphosis or prior to hatching or birth (e.g. Weiss, Johnston & Moore 2007) or inhibited by treatments with GC synthesis blockers (e.g. metyrapone) or receptor antagonists (e.g. RU486, Glennemeir & Denver 2002) demonstrate that GCs play a causal role in precipitating these life-history transitions (also reviewed in Crespi & Denver 2005a; Wada 2008). Increased signalling of both corticotropin-releasing factor (CRF), the hypothalamic regulator of the HPA/I axis, and GCs are involved in inhibiting growth whilst stimulating developmental processes that prepare the individual for the transition from an aqueous developmental environment to a terrestrial environment, such as the preparation of lungs for air breathing and the restructuring of physiological systems involved in osmoregulation and nitrogenous waste removal (for a review see Denver 2009).

The up-regulation of HPA/I axis activity prior to and during early life-history transitions is genetically controlled, but is also regulated by environmental cues and thus, is involved in regulating intraspecific variation in the timing of early life-history transitions. In this context, the HPA/I axis is viewed as a modulator of developmental plasticity in vertebrates (Denver 2009). Depending on the stage of development, elevation of HPA/I axis activity in response to adverse environmental conditions (e.g. low oxygen, lowering water volumes, low food availability) can have opposing effects on developmental timing. For example, in most anuran tadpoles increased GC levels slow growth and development during early larval stages, while it accelerates developmental timing if experienced later in the tadpole period (reviewed in Crespi & Denver 2005a). Similarly, precocious elevation in HPA axis activity during late foetal/embryonic development also precipitates preterm birth in mammals and earlier hatching in birds and reptiles (e.g. Weiss, Johnston & Moore 2007), suggesting that HPA/I axis is an evolutionarily conserved system that fine-tunes the timing of these life-history transitions according to proximate environmental conditions.

Recent work has shown that the HPA axis activity in response to food availability in particular may be driving variance in the timing of fledging in birds, another life-history transition associated with morphological, physiological, and behavioural changes (e.g. feather growth, muscle growth, ability to fly and independently forage). For example, Laysan albatross chicks experience reduced food intake before fledging, which is associated with an increase in corticosterone and decrease in corticosteroid binding globulins (CBG; thus, higher bioavailability of GCs), a reduction in body mass at fledging, but an acceleration in feather growth needed for flight (Sprague & Breuner 2010). Similarly, fledging in white storks is preceded by a progressive fourfold increase in baseline plasma GC levels concomitant with food restriction and increase in locomotor activity (wing flapping) at the nest (Corbel & Groscolas 2008). Furthermore, in pied flycatchers while GC levels peaked prior to fledging, other factors such as brood size (i.e. sibling competition) and level of food restriction positively affected GC levels at fledging (Kern *et al*. 2001). Conversely, storm petral chicks that were supplementally fed, and thus had greater nutritional reserves, remained in the next longer and had lower free corticosterone than control chicks (Kozlowski *et al*. 2010). Fledging is not associated with increases in GCs in king penguin chicks, but this could be due to the greater utilization of lipid reserves during this time, which might prevent stimulation of the HPA axis (i.e. reduce nutritional stress at fledging) in these birds (Corbel, Geiger & Groscolas 2010). Taken together, while elevation in HPA activity has been associated with fledging in several bird species, this activity appears to be functionally associated with (i) the extent of developmental processes, and (ii) the reduction of food availability that occurs at this transition. This suggests some interplay between physiological energy balance indicators and the HPA axis, similar to what is involved in modulating the timing of birth in mammals (see Crespi & Denver 2005a).

While the increase in HPA/I axis activity is functionally related to the timing of early life-history transitions when studied within species, it is poorly understood how HPA/I axis activity varies across species with different life histories. For example, life-history theory would predict that species with relatively fast tadpole growth and development rates should have higher baseline GC levels to maintain high amounts of circulating energetic resources needed for rapid growth, and relatively earlier HPI axis maturation to allow for earlier onset of metamorphosis. Indeed, in spadefoot toads (Family Scaphiopodidae) Ledon-Rettig, Pfennig & Crespi (2009) found that *Scaphiopus couchii*, one of the fastest developing species, had higher baseline corticosterone levels compared with the more slowly developing *Spea bombifrons*. Buchholz & Hayes (2005) found higher thyroid hormone (TH) levels in faster developing spadefoot toads, and because CRF stimulates TH and GCs in tadpoles, corticosterone is likely to be higher in these animals too. These patterns suggest that genes and developmental processes that allow for elevated HPA/I activity during the tadpole phase may have been selected to allow for rapid development, however further studies are needed to more rigorously test this hypothesis.

It also remains unclear why the HPI axis is extremely sensitive to environmental conditions in some species and relatively insensitive in others (Buchholz *et al.* 2011). Although caution needs to be taken when examining interspecific neuroendocrine functions, as all levels of regulation must be considered (e.g. hormones, binding proteins, receptors, see Breuner & Orchinik 2002; Romero 2004; Romero, Dickens & Cyr 2009), a comparative approach, which is taken is many studies of life-history evolution (Roff 1992; Stearns 1992), would yield novel insights as to how HPA/I axis function during early development has evolved with life-history strategies (also see Future Directions).

AGE AT FIRST REPRODUCTION

Remarkably few studies have explicitly studied the effect of HPA/I axis activity on the timing of first reproduction/puberty in natural populations of animals, although many studies have investigated GCs in relation to seasonal reproduction in general. Elevated circulating GC levels appear to be correlated with later onset of puberty or first reproduction (Goos & Consten 2002; Shi *et al.* 2011), but Gesquiere *et al.* (2005) found that early maturing males had higher faecal GC levels than those maturing later. This finding is complicated by the fact that there was no within-individual change in GCs in males, so GC levels were not rising as puberty approached, and females actually experienced a decrease in GCs as they approached menarche (Gesquiere *et al.* 2005). This study suggests that relationships between GCs and timing of puberty are complex and differ depending on social context or sex. Such may be the case when relating GCs and sex change in fish. Perry & Grober (2003) proposed that elevated GCs inhibit sex change in fishes that exhibit socially mediated sex change, but this hypothesis was challenged by Frisch *et al.* (2007), who showed that cortisol implants did not prevent sex change (although a role for CRF could not be ruled out). Given that variation in age of first reproduction (and size when considering ectotherms) may have a greater contribution to an individual's fitness and population growth than variation in fecundity (Roff 1992), greater attention should be focused on how stress affects this life-history trait within and across species.

CONCLUSIONS

The timing of early life-history transitions in vertebrates shows a remarkably consistent association with increases in GCs. This is likely due, in part, to conserved effects of GCs on developmental processes and brain maturation that take place during these early transitions in vertebrates. Because food restriction and other environmental stressors often coincide with these transitions, stress-induced up-regulation of HPA/I axis activity is associated with generating variation in the timing of life-history transitions within species. If these environmental cues are predictable at these transitions, the responsiveness of the HPA/I axis to these signals could be considered an adaptation to stimulate the developmental events needed to make these transition at optimal times. However, more studies are needed to address whether HPA/I-mediated developmental plasticity is adaptive, relies on the measurement of survival at the time of transition, as well as survival and reproductive output throughout the life of the individual (e.g. Kitaysky *et al.* 2003). In addition, studies are needed to understand how alterations in the HPA/I axis are involved with interspecific differences in the timing of life-history transitions.

Do GCs mediate phenotypic variation and tradeoffs in life-history traits?

Many of the most familiar life-history traits are those that affect the reproductive success and survival of adult animals: age-specific reproductive output (clutch size and size of offspring, and parental care) reproductive lifespan and age-specific mortality rates are all familiar life-history traits. Moreover, tradeoffs between the number and size of offspring, or between reproduction and adult survival are well established in the literature. In vertebrates, most species are iteroparous (although there are some examples of semelparity, for example, Pacific salmon and Australian marsupial shrews), but life span and reproductive schedules vary tremendously: some are short-lived and therefore concentrate their reproductive effort into a few bouts (e.g. field mice), while others can live more than 100 years during which reproduction is spread throughout. In a life-history context, reproductive output in iteroparous species can be considered 'current' or 'future', and investment in reproduction can affect future reproduction and survival via longer-term 'costs of reproduction.'

While it is widely assumed that high GC levels negatively affect reproduction, in fact both baseline and stress-induced GC levels are typically highest during reproduction compared with other phases of the annual cycle in most vertebrates (although there was no clear pattern for stress-induced GCs in mammals; Romero 2002). Breuner, Patterson & Hahn (2008) and Bonier *et al.* (2009a) recently reviewed studies that have linked circulating GC concentrations with variation in fecundity, reproductive success or survival, either for baseline or stress-induced GC (see also Breuner 2011). These reviews show that variation in GC levels at any single point in time can be positively, negatively, or nonsignificantly related to surrogates of fitness. Although a range of methodologies have been used to assess functional consequences of variation in GC levels across studies (e.g. plasma GCs, faecal GCs, and exogenous GCs), these different measure should positively co-vary, and therefore, we would expect to see consistent relationships between GCs and reproduction with all three measures. However, there is also a range in the timing and life stage of GC measurements, the kinds of GC parameters measured, the other components of the HPA/I axis measured and the stress paradigms used, all of which can

further obscure detection of universal patterns that relate GCs to life-history traits and trade-offs (Fig. 1). Here, we provide a brief review what is known about the relationships between GCs and reproduction and survival across vertebrate groups, citing recent reviews on this topic but highlighting more recent studies and synthesizing some conclusions from these studies.

REPRODUCTION

Elevated baseline GC levels among individuals in the same life-history stage are often assumed to indicate individuals in poor condition with lower predicted reproductive output (i.e. Cort-Fitness Hypothesis, Bonier *et al*. 2009a), and while there are many examples where this is so, elevations in cortisol relative to other times in the year are often associated with increased energetic demands associated with reproductive function, particularly in oviparous vertebrates (Romero 2002). In fish, elevation in baseline circulating cortisol is associated with ovarian development and vitellogensis in females (Brodeur, Woodburn & Klecka 2005; Shankar & Kulkarni 2007), and stress-induced GC levels may not necessarily result in reduced reproductive output (e.g. Contreras-Sanchez *et al*. 1998; Pankhurst 1998) despite experimental evidence that cortisol inhibits vitellogenesis in females (e.g. Lethimonier *et al*. 2000). Similarly in amphibians, reptiles and birds and some mammals, baseline GC levels have been shown to increase during the transition from nonbreeding to a reproductive state in some species (e.g. Wilson & Wingfield 1994; Jessop 2001; reviewed in Romero 2002; Moore & Jessop 2003), but not always (e.g. Tyrrell & Cree 1998; Amey & Whittier 2000; Massot *et al*. 2011). However, a major unresolved question is whether elevation in baseline GCs is a requirement for reproduction, or simply associated with increased metabolic demands during reproduction (Moore & Jessop 2003). In two recent experimental studies of birds, Bonier, Moore & Robertson (2011) and Crossin *et al*. (2012), showed that higher plasma GC levels were associated with increased foraging or chick provisioning effort and resulted in higher fledging success or higher chick mass at fledging, respectively (also see the extensive studies by Kitaysky, Wingfield & Piatt 1999; Kitaysky *et al*. 2010; Satterthwaite, Kitaysky & Mangel 2012; and references therein). These positive relationships between GCs, parental care and reproductive success suggest that elevated GC levels do not always interfere with reproductive investment, and might instead facilitate it. Bonier, Moore & Robertson (2011) noted that they were able to detect the elevation in GCs in females with experimentally larger brood sizes because they measured GCs before and after rearing hatchlings, and those with larger broods had a greater difference in GC levels, suggesting that studies that control for within-individual variance in GC levels are more powerful than cross-sectional studies when relating GC levels to fitness (Williams 2008).

Studies measuring stress-induced GC levels have found variable associations with reproductive traits in fish, amphibians and reptiles. Stress-induced elevations in GCs have been shown to inhibit male brood guarding behaviour (Knapp, Wingfield & Bass 1999) and increased rate of nest failure (Dey *et al*. 2010) in fish species, but stress responses can be blunted during the parental care period in some fish species, thus minimizing changes in reproductive effort (O'Connor *et al*. 2011). In amphibians, elevated endogenous GC levels are associated with energetically expensive calling or reproductive behaviours in male frogs, and there are multiple studies that showed blunted stress responses during reproduction (reviewed in Moore & Jessop 2003). In male salamanders, which don't vocalize but exhibit courtship behaviours, stress-induced elevations in GC levels suppress testosterone levels and male sexual

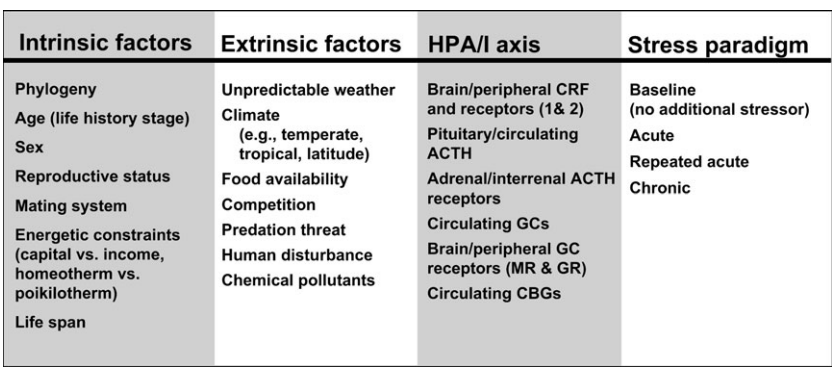

Fig. 1. Factors that affect variability in GC levels reported in the literature. A clear consensus of this review and others (Breuner, Patterson & Hahn 2008; Bonier *et al*. 2009a) is that GC levels inconsistently explain life-history variation across vertebrates, which is likely because of the multitude of intrinsic and extrinsic factors that affect the many elements of the HPA/I axis. Ideally, these factors need to be statistically or experimentally accounted for in order for robust patterns to emerge across taxa. Furthermore, the experimental designs and the context of GC measurements (e.g. stress paradigms used) also vary among studies, making it difficult to compare studies and interpret relationships between HPA/I axis activity, fitness, and the expression of life-history traits and trade-offs. CRF = corticotropin-releasing factor, ACTH = adrenocorticotropic-releasing hormone, GC = glucocorticoids, MR = mineralocorticoid receptor, GR = glucocorticoid receptor II, CBG = corticoid binding globulins.

behaviours in rough-skinned newts (Moore & Zoeller 1985; Coddington *et al.* 2007), but this may be a context-dependent effect as exogenous GC treatment does not inhibit clasping behaviours in males that had recently been engaged in clasping behaviours (Coddington & Moore 2003). Woodley & Lacy (2010) also found that exogenous GC treatment did not inhibit courtship behaviours in male or female *Desmognthus ocoee*, a plethodontid salamander, although it reduced general activity. Finally, in lizards reductions in territoriality and expression of male courtship and aggression can arise from elevated GC levels (Tokarz 1987; Denardo & Sinervo 1994; Moore & Mason 2001), yet GC-implanted male sand lizards (*Lacerta vivipara*) increased copulatory behaviour and reproductive effort (Gonzalez-Jimena & Fitze 2012). Indeed, more studies across different mating systems and phylogenetic groups are needed to better understand the context-dependent effects of stress-induced GCs on reproductive success.

Certainly, more studies are needed to understand the relationships between GCs and reproduction in female amphibians and reptiles. While females are rarely the subjects of such studies in amphibians, studies conducted with lizards suggest that high GC levels do not directly inhibit female reproduction (Sinervo & DeNardo 1996; Cadby, Jones & Wapstra 2010) but can modulate individual receptivity to mating (Vitousek *et al.* 2010) or morph-specific differences in reproductive performance, in particular offspring size vs. quantity trade-offs (Sinervo & Licht 1991a, b; Comendant *et al.* 2003; Lancaster *et al.* 2008). Elevated GCs cause morph-specific differences in the hierarchical organization of multidimensional trade-offs causing differential mortality, reproductive suppression and offspring quality (Lancaster *et al.* 2008), suggesting a fundamental role of GCs in the functional integration of several concurrent life-history trade-offs.

In contrast to the increase in GCs with reproduction in most oviparous vertebrates, wild mammalian species do not always experience elevations in baseline GCs during reproductive seasons (reviewed in Romero 2002; Breuner, Patterson & Hahn 2008; Bonier *et al.* 2009a) but relatively high GC levels seem to have negative effects on reproductive output. In snowshoe hares (*Lepus americanus*), which breed up to four times a year, maternal faecal cortisol metabolite (FCM) levels were negatively correlated with number and size of offspring (Sheriff, Krebs & Boonstra 2009). Moreover, experimentally stressed females, with higher FCM levels, were more likely to have a litter fail and their offspring were in poorer condition than control females. Snowshoe hares undergo decade-long population cycles in which predation pressure and measures of the severity of stress in hares becomes intense during the peak and decline phases of the cycle (Boonstra *et al.* 1998). Female FCM levels are positively correlated with predator density and offspring of these females are themselves more responsive to stressors than the offspring of mothers with lower FCMs (Sheriff, Krebs & Boonstra 2011). Thus, for the snowshoe hare, GCs have a clearly negative impact on many aspects of reproduction, which may relate to population cycles in this species. By contrast, in the communally breeding degu (*Octodon degus*), Ebensperger *et al.* (2011) found that female FCM levels *increase* with the *per capita* young produced in 2 of 3 years (there was no relationship in the middle year of the study). This latter study suggests that increased GCs were beneficial to fitness at least in some years, and highlights the need to measure these relationships in multiple reproductive bouts in variable environmental conditions.

This brief review supports the conclusions of Breuner, Patterson & Hahn (2008), Bonier *et al.* (2009a) and Breuner (2011): if there is a general relationship between GCs and reproduction, it is a pattern with many exceptions. Numerous studies in a wide range of vertebrate taxa suggest there can be a *positive* relationship between baseline GCs and reproduction, especially when energetically expensive behaviours are involved (e.g. mating behaviours or feeding young), or energetic resources need to be rerouted towards yolking eggs in females. But there are also examples across most taxa of *context-specific* negative effects of stressors on reproduction, and that the HPA/I axis can be insensitive to environmental stressors or experimentally induced elevations in GC levels during the reproductive season. Perhaps universal patterns have not yet revealed themselves as the phylogenetic diversity of free-living species representing the full-range of life histories may not be broad enough at this time. Furthermore, focusing just on circulating GCs without knowledge of the other components of the HPA/I axis (Fig. 1) may be giving us a limited snapshot of the ways in which environmental stress affects the many regulatory pathways involved in reproduction.

SURVIVAL

While some studies report a positive relationship between circulating GCs or HPA reactivity and survival in mammals (e.g. Cabezas *et al.* 2007) and reptiles (Romero & Wikelski 2001; Cote *et al.* 2006; Miles *et al.* 2007), several studies document no or negative relationships between GC levels and survival, or even conflicting relationships within the same species (e.g. Romero & Wikelski 2001; French *et al.* 2010). For example, Boonstra & Boag (1992) found no relationship between CGs and survival in the meadow vole (*Microtus pennsylvanicus*), but Charbonnel *et al.* (2008) found that faecal GC levels in the water vole (*Arvicola scherman*) were negatively correlated with body condition, immune function, and population size. Greater HPA reactivity also has been associated with lower annual survival in lizards (reviewed in Bonier *et al.* 2009a) and nestlings (Blas *et al.* 2007) and adult birds (Goutte *et al.* 2010; Kitaysky *et al.* 2010).

A frequent assumption of the Cort-Fitness hypothesis is that the relationship between GCs and fitness is linear, but a nonlinear relationship between baseline circulating GC levels and survival may be operating (Bonier *et al.* 2009a).

For example, Brown *et al.* (2005) showed that individual cliff swallows with very low and very high levels of corticosterone had lower survival than those with intermediate levels. Interestingly, when cliff swallows were sampled earlier in the season, annual survival declined with increasing corticosterone level (Brown *et al.* 2005), which illustrates that the relationships between baseline GCs and fitness likely vary depending on the specific life-history context in which they were measured. Growth in anuran tadpoles presents another example of nonlinear GC dynamics: tadpoles housed at too low or too high density grow slower than those at intermediate densities, and growth is associated with whole body GC levels (Glennemeir & Denver 2002). Uncovering these nonlinear relationships between GCs and fitness is labour intensive as it relies on either sampling during more than one time during the life history, sampling in multiple environmental contexts, or conducting experiments designed to expand the phenotypic variance needed to detect these patterns. The effort in acquiring this type of data may explain why there are so few examples of nonlinear GC-fitness relationships in the literature although it may be common.

As with the relationship between reproduction and GCs, there is no consistent relationship between stress responsiveness and survival among taxa, or even within taxa (e.g. Galapagos marine iguana, Romero & Wikelski 2001, 2002; French *et al.* 2010). One reason for the lack of patterns is that the specific contexts and kinds of stressors vary among studies, as does the time of year and life stage during which survival is measured (see Fig. 1). While these factors may be obscuring the relationships between HPA/I axis activity and fitness (Fig. 1), there are currently too few studies of taxa that span diverse life-history strategies to tease apart the influence of these potential factors. However, sufficient data within certain groups (birds and mammals) are accumulating to allow for meta-analyses to resolve general patterns relating GCs to life-history traits and fitness.

Most of the studies reviewed previously focus on correlations between GC levels and a single life-history trait (or surrogate trait), but life-history theory does not justify the assumption that such simple relationships should be widespread. There are well-described tradeoffs among certain life-history traits; notably between reproduction and survival (Stearns 1992). Thus, life-history theory warns us against assuming that fitness consequences of GCs can be predicted by measuring the effect of GCs on reproduction only, or on survival only. GCs can affect both, and researchers generating hypotheses and developing research programs need to account for this. The 'brood value' hypothesis, for example, predicts that elevation of GCs in response to a stressor should inhibit reproduction when the contribution of the current clutch/litter to lifetime reproductive fitness is low and the probability of survival to the next reproductive bout is high (Lendvai & Chastel 2008; Bókony *et al.* 2009). There could also opposing effects of GC levels on reproduction and survival (Bonier *et al.* 2009b), and therefore, the effects of GCs on both traits need to measured beyond a single time point to better estimate the relationship between GCs and lifetime reproductive success and survival (Breuner, Patterson & Hahn 2008; Breuner 2011).

COMPARATIVE ANALYSES OF GCS AND LIFE-HISTORY TRAITS

Another characteristic of most of the studies reviewed previously is that they examine the role of GCs on variation in life-history traits within a single species; however, some recent studies have taken a comparative approach to assess how GC modulation has evolved among species with different life-history strategies. Hau *et al.* (2010) combined original data and those collected from the literature to examine the hypothesis that variation in GCs is related to variation in life-history traits among different passerine bird species during the breeding season at a site in the tropics vs. a more temperate site at higher latitude. They found that baseline GC levels among species were higher with shorter breeding season length and lower body mass, and stress-induced GCs varied inversely with body mass and positively with annual adult survival rates. These correlations suggest that in passerine birds, baseline HPA activity has been selected to be higher when environmental constraints favour increased reproductive allocation/unit time at the cost of growth, and that stress responsiveness, as measured by maximum GC levels is positively associated with survival, as predicted by the Cort-Fitness hypothesis.

Bókony *et al.* (2009) used phylogenetic comparative analyses to test the 'brood value' hypothesis across 64 bird species. They found that species with a higher value of the current clutch relative to future breeding opportunities mounted weaker corticosterone responses during acute stress, and that females in species with more female-biased parental care had weaker corticosterone responses. Assuming that the weaker GC response is associated with reduced abandonment and higher survival of offspring, these results support the idea that the stress response is a trait that can evolve as part of a life-history strategy. These authors also found additional interesting trends, including a positive (but weak) correlation between GCs and brood value (i.e. increased reproductive allocation), and peak corticosterone was greater in species breeding at higher latitudes where breeding seasons are shorter.

In mammals, the most compelling evidence that GCs play an important role comes from studies of semelparous dasyurid marsupials in Australia. In at least 10 dasyurid species the entire male population dies at the end of their first breeding season (Lee & Cockburn 1985; Bradley 2003). In four of these species (*Antechinus stuartii*, *A. swainsonii*, *A. flavipes*, and *Phascogale calura*) there is a common physiological progression among males. As the breeding season approaches, free GC levels increase due to the cumulative effects of increased total GC production,

decreased CBG levels, and failure of negative feedback (Bradley, McDonald & Lee 1980; McDonald *et al*. 1981; Lee & Cockburn 1985; Bradley 1987, 1990). The elevated GC levels lead to gastric ulcers, suppression of immune and inflammatory responses, increased parasitism, and shifts in haematological parameters (Cheal, Lee & Barnett 1976; Barker *et al*. 1978; Bradley, McDonald & Lee 1980) that ultimately result in male die-off. In contrast, iteroparous species including fat-tailed dunnart, *Sminthopsis crassicaudata*, show no relationship between testosterone and CBG levels, and although cortisol concentrations fluctuate over the breeding season, they are lower than the maximum CBG binding capacity, resulting in low free GC levels (McDonald *et al*. 1981).

The distinct endocrine profile of the semelparous marsupials led Lee & Cockburn (1985) to propose that the gluconeogenic effect of cortisol allow these species to derive energy from the breakdown of muscle, thereby freeing them to spend time seeking mates instead of foraging during a time of scarce food. They also predicted that this strategy could apply to other small mammals and could explain the spring populations declines observed in a number of vole species (Lee & Cockburn 1985). This 'adaptive stress hypothesis' focused on the potential for the disregulation of the stress axis to support life-history strategies characterized by short lifespans and early reproduction. However, subsequent studies have failed to find a similar role for GCs in the semelparous didelphid marsupial, the Virginia opossum (*Didelphis virginiana*), or in other mammal species with short male reproductive lifespans like arctic ground squirrels (*Urocitellus parryii*; Delehanty & Boonstra 2011), although alterations in GCB binding could be working in these systems to regulate the bioactivity of GCs.

CONCLUSIONS

Whereas there was considerable uniformity in the role of GCs in early life-history transitions across vertebrate taxa, the preponderance of the literature suggests that there are no simple, general interspecific relationships between GCs and life-history traits related to reproduction and survival. Hau *et al*. (2010) did find relationships between breeding season length (a surrogate measure for number of annual breeding opportunities) and baseline and stress-induced GC levels, but they limited their analysis to passerines and they controlled for body mass and environment type. This suggests that patterns may emerge only when we conduct multivariate analyses that account for other intrinsic and extrinsic factors affecting life-history evolution. In other words, whereas most researchers have proposed simple and direct relationships between the HPA/I axis and life-history traits (e.g. the adaptive stress hypothesis of Boonstra & Boag 1992; the Cort-Fitness and Cort-Adaptation hypothesis of Bonier *et al*. 2009a), the evidence suggests that the relationship is more nuanced and complex. Although meta-analyses and interspecific comparative studies are complex, the use of multivariate analyses that control for phylogeny and methodological differences across studies offer a powerful, quantitative means of testing hypotheses and resolving patterns relating GC levels and HPA/I axis function to variation in life histories.

Conceptual models of HPA/I activity and life-history transition and variation

Given the emphasis on energetics in life-history theory (Levins 1968; Sibly & Calow 1986), the first conceptual model that integrated HPA/I axis activity, variation in life-history stages and fitness to understand how animals adjust their physiology to intrinsic and extrinsic changes was the 'allostatic load model' (McEwen & Wingfield 2003, 2010). This model assumes that fluctuations in HPA/I activity, as well as the physiological and behavioural phenotypes mediated by this activity, are adapted to the energetic demands associated with predictable changes in the environment and phylogenetically determined life-history transitions. This model introduced the concept of allostatic state from the biomedical literature, which is operationally defined as the summation of energetic demands and sustained activity of primary mediators (such as GCs) needed to maintain homoeostasis and obtain energetic resources in changing environments. Depending on the life-history stage, different kinds of energetic demands and constraints (e.g. food availability) can change in magnitude, creating temporal energetic limits within which the physiological responsiveness to predictable and unpredictable events can vary throughout the life of the animal (also see Landys, Ramenofsky & Wingfield 2006). When energetic demands exceed those stored internally or available in the environment, a state of allostatic overload will be eventually reached, which is considered a state of chronic stress that is not energetically sustainable and if prolonged will lead to mortality (McEwen & Wingfield 2003, 2010).

More recently, Romero, Dickens & Cyr (2009) proposed the 'reactive scope model', which changed the *Y*-axis from one that scales energy (in the allostatic load model) to one that scales a broader range of physiological mediators so it can be charted across life-history stages and environmental conditions over time. While GCs may be an indicator of HPA/I axis activity, other indicators of stress such as cytokines or catecholamine levels, heart rate, or locomotion could be modelled across the life history. The 'reactive scope' is the variation in the physiological mediator that lies within the normal homoeostatic function of the animal, including predictive perturbations and short-term responses to unpredictable environmental or social conditions. This model also predicts that for each physiological mediator there is a threshold above which animals enter homoeostatic overload leading to pathology (synonymous with allostatic overload), but the threshold for this state can vary depending environments experienced during early development and the amount of accumulated stress

experienced throughout life ('wear and tear', similar to allostatic load).

Life-history theory is incorporated within the allostatic load or reactive scope models of HPA/I activity by informing the predictions that can be made when applying these models to animals with different life styles, metabolic and physiological profiles, and phylogenetic constraints. For example, during a period of unpredictable food restriction, the HPA/I response that maximizes fitness may vary among organisms given their different energetic demands and physiological constraints (see Fig. 2a). Indeed, GCs and foraging behaviour have been shown to be positively related in homoeotherms (birds and mammals; Romero & Remage-Healey 2000; Sapolsky, Romero & Munck 2000), whereas GCs and locomotion are negatively related in a poikilotherm (frogs; Crespi & Denver 2005b). We expect that responses also will vary between capital breeders that use stored energetic reserves for reproduction, and income breeders that rely on current food intake to fuel reproduction, or between determinate vs. indeterminate growers that undoubtedly have different fitness trade-offs between reproduction and survival, or current and future reproduction.

The allostatic load and reactive scope models provide an excellent framework within which the relationships between GC levels and life-history traits of a species can be predicted while keeping in mind the many energetic, ecological, physiological, developmental, and phylogenetic differences that occur during the life span. These models also can be used to map contrasting predictions of the ways in which HPA/I axis activity varies among species of different life histories. In other words, these models are useful because they force us to recognize that the relationships between GCs and life-history traits are likely to be variable and state-dependent rather than simple. However, these models also have limitations. They are valuable schematic models, but neither the allostatic load nor reactive scope models are quantitative, computational models. Thus, they cannot be used to predict changes in GC levels over time or the complex relationships that GC levels have on fitness. In addition, while these models are accessible to the interests of physiological ecologists, these models are not structured to explicitly predict how GCs influence evolutionary or population dynamics. Using these models as a foundation, however, will allow for future empirical and theoretical work to make these advances.

Future research directions

It is clear that the HPA/I axis is an integral physiological system that underlies resource allocation trade-offs and the expression of vital life-history traits, although knowledge of the intrinsic energetic demands of the particular life-history stage and fitness consequences (i.e. the ecological and evolutionary context) are important in determining the specific nature of these trade-offs. At the organismal level, the function of the HPA/I axis has three tiers: (i) as a primary mediator of energy balance homoeostasis, (ii) as a master organizer of life-history transitions, and (iii) as an integral responder to stressors. None of these functions are mutually exclusive, although they seem to be studied in

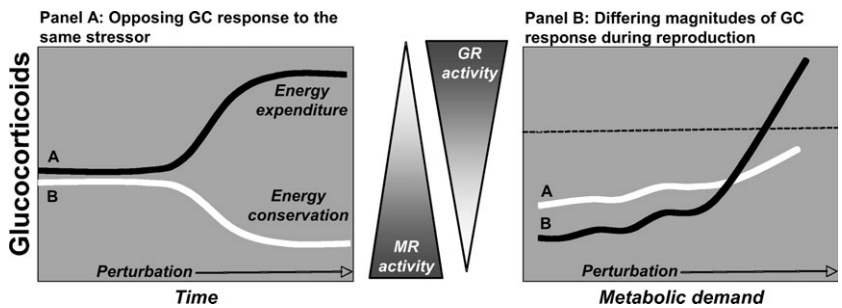

Fig. 2. Variation in glucocorticoid (GC) responses to environmental perturbations depending on mineralocorticoid receptor (MR) and GC receptor (GR) activity, life-history strategy, and fitness outcomes. Panel (a) depicts opposing GC responses of two genotypes, populations, or species to a perturbation, one in which an increase in energy expenditure increases fitness (survival/reproduction, mostly associated with endotherms) and the other in which a decrease in energy expenditure increases fitness (e.g. response of amphibians to unpredicted food restriction or detection of predation threat). These GC responses are associated with behavioural and metabolic effects caused in part by the alteration GR and MR signalling (see Marzolla *et al*. 2012 for relationship between MR and metabolism). Whether any of these 'stress' responses of the HPA/I axis are adaptive or pathological needs to be determined by experiments in which fitness consequences of phenotypic variance associated with these changes in HPA/I axis activity are actually measured. Panel (b) depicts variation in GC response in two hypothetical individuals (A and B) during reproduction according to metabolic demand and fitness outcomes: modest elevations in GCs modulate increases in reproductive effort and success in both A and B, as higher quality yolking of eggs, higher quality expression of courtship behaviours, or enhanced parental care may be facilitated; however, when an environmental stressor is experienced by individual A, GC levels cross a 'stress response' threshold (dashed line), above which GR activation is greater and behaviours change to reduce reproductive allocation. This individual trades off current reproductive success for future reproductive success or survival. By contrast, individual B has a blunted GC response to the perturbation and continues to allocate resources to the current reproductive bout. If either strategy enhances fitness, natural selection favours both strategies. A and B could also represent two individuals with different histories of stress, or the mean GC response of two populations of a species located in different environmental conditions, or of two species, whose response has been adapted according to their life history (e.g. short-lived species vs. a long-lived species, that is, following the 'prudent parenting hypothesis' Bókony *et al*. 2009).

isolation much of the time. We suggest that future studies of GCs need to be designed to explicitly incorporate life-history theory and test assumptions underlying the adaptive nature of stress responses, including measurement of survival and reproduction as well as intermediate behavioural or physiological fitness correlates. In this section, we address four main research directions and considerations that will most effectively advance our understanding of the role of the HPA/I axis in generating variation in life-history traits and trade-offs.

First, we need to better understand how the HPA/I axis mediates life-history traits across life stages and in different environments. So far this has been attempted with comparisons between distinct environmental types (e.g. tropical or temperate, see Wada *et al*. 2006; Hau *et al*. 2010), but additional approaches can be followed. For example, we need to define variation in environmental quality or conditions along gradients of what might be considered by the animal to be benign to severe, and measure HPA/I axis activity (baseline GCs, stress responsiveness), survival and reproduction at the population-level to best understand the ultimate effects of HPA/I axis function on fitness across environments (and potential selection gradients acting on HPA/I axis function). In pervasively harsh environments one might predict allostatic overload or extreme 'wear and tear' as predicted by the allostatic load or reactive scope model, but there may be selection for stress resistance in habitats that are consistently adverse (e.g. at the edge of a species range) pending a species life history. Because of the obvious interactions among different life-history and environmental factors, it may still be difficult to see clear cut patterns. However, one could certainly integrate these life-history and environmental components into improving the complexity of existing models and help improve a predictive framework. In addition, there is a need for field studies that show how endogenous GCs, as well as other components of the HPA/I axis, respond to natural perturbations. Large-scale ecological manipulations that alter environmental contexts could also offer powerful insights into how GCs, life history and environment interact to influence fitness in animals. These kinds of studies can be designed to address important landscape-level alterations in biotic and abiotic conditions of great interest to evolutionary ecologists, such as the impact of invasive species, habitat loss and climate change.

Second, we find that a major shortcoming in the area of stress physiology is the reliance on GC levels as an indicator of the biological effects experienced by the organism. The difficulties with this assumption lie in the multi-factorial regulation of the HPA/I axis (Fig. 1). First, many studies rely on total GC levels even though CBGs bind a large proportion of circulating GCs in most species. The biological significance of bound versus free hormone in the plasma continues to be debated (Malisch & Breuner 2010) but there are cases in which free and total GC levels reveal different trends (e.g. Boonstra & Singleton 1993; Breuner & Orchinik 2001; Love *et al*. 2004), making it critical to resolve the biological significance of CBG regulation of GC actions. In addition, CRF and ACTH, have behavioural and physiological effects that are independent of controlling GC secretion (e.g. ACTH can exert lipolytic effects and CRF affects food intake behaviour, learning and memory: Boston 1999; Croiset, Nijsen & Kamphuis 2000); therefore, they might be important factors that act to generate life-history traits and trade-offs. Study of these factors will also help us understand the mechanisms underlying variation in GC levels in different life-history contexts. For example, in studies of four birds species, all of which showed seasonal variation in GC levels, one species regulated GCs at the level of the pituitary, one at the adrenal and two at the hypothalamus (reviewed in Romero 2002). Furthermore, it is widely acknowledged that the downstream determinants of GC effects, including variation in receptor density, interconversion of active and inactive steroids by 11ß-hydroxysteroid dehydrogenases, and modulation of genomic effects of GC-receptor complexes by cofactor, could affect the interpretation of GC actions based only on measurement of circulating GC levels (Romero 2002; Love *et al*. 2004; Landys, Ramenofsky & Wingfield 2006; Malisch and Breuner 2010). However, few studies attempt to assess the importance of these downstream determinants of GC action, likely because it is difficult to measure these levels of HPA/I regulation in the natural populations. If any of these factors play a role in determining the biological effects of GCs, it would weaken relationships between total GC levels and life-history traits. Moreover, these complexities would challenge the usefulness of some standard techniques. The use of GC implants, for example, can (i) inhibit normal CRF signalling through endogenous negative feedback mechanisms, and (ii) prevent the assessment of stress-induced modulation of CBGs that would normally affect the bioactivity of GCs. We suggest that the development of a more diverse set of experimental tools is needed to probe the relationship between HPA/I function and life-history traits in field conditions. For example, finding ways to effectively treat animals in the field with agonists or antagonists would enhance our ability to evaluate the specific roles that CRF or GC signalling plays in stress responses or their relationships to fitness, life-history traits and trade-offs.

Third, we urge caution in interpreting the biological relevance of 'stress-induced' or 'maximum' GC levels, as measured using the standardized capture and restraint protocols, and variation in 'baseline' circulating GCs, especially in the context of physiological mechanisms underlying variation in life-history traits (also see Breuner, Patterson & Hahn 2008). The standardized stress protocol as well as the CRF or ACTH challenge protocol (e.g. Boonstra *et al*. 1998) are valuable tools to assay HPA/I *reactivity*, that is, the rate of increase and asymptote of GCs is response to a standard stressor, which could result from a range of intrinsic and extrinsic factors that chronically elevate baseline GC levels and/or decrease HPA/I axis responsiveness to acute, novel stressors. However, the

on-going focus on maximum, stress-induced GCs as the major correlate to the expression of life-history traits creates three conceptual problems. First, the main argument for a bi-modality in GC effects is the existence of two GC receptors (GRs): a high-affinity mineralocorticoid receptor (MR) and a low-affinity GR (Breuner & Orchinik 2002). The different affinities of these receptors has led to the suggestion that the high-affinity MR receptor is probably only responsive to changes in 'baseline' GC levels, whereas the low-affinity GR mediates the responses to 'stress-induced' GC levels (Breuner & Orchinik 2002). However, the biomedical literature indicates that both forms of receptor are involved in regulation of baseline HPA activity and energy balance (Herman *et al*. 2003; Joels *et al*. 2008; Marzolla *et al*. 2012; see Fig. 2). Thus, it is not clear that regulation of baseline levels can be quite so neatly separated from 'stress-induced' levels. Second, several studies in birds have shown that the maximum levels of 'baseline' GCs measured in relatively chronically stressful conditions (e.g. low food availability, high predation) are associated with decreased reproduction and survival, yet these GC levels do not approach 'stress-induced' levels as measured using the standardized stress protocols (Kitaysky, Wingfield & Piatt 1999; Clinchy *et al*. 2004; Kitaysky *et al*. 2010; Satterthwaite *et al*. 2010). In cases in which baseline and stress-induced GCs are similar (e.g. Delehanty & Boonstra 2012) the baseline measurement is likely to be more closely associated with the expression of life-history traits because it better reflects the integrated energetic expenditures and level of GC-mediated processes over a longer period of time (assuming MR, GR, and CBG levels are the same). Given this estimation, when GC levels are manipulated to investigate effects on life-history traits, we suggest that the circulating concentrations should not be calibrated/validated by reference to maximum stress-induced GC levels as measured by the standard stress protocol (e.g. Meylan, Dufty & Clobert 2003), but rather in relation to maximum, circulating GC levels measured in unmanipulated animals sampled in the field. In summary, we propose that it is more appropriate in a life-history context to consider continuous, phenotypic variation in *endogenous* plasma GC levels over a wide range of values (reflecting different environmental/social conditions), which reflect this hormone's role as a 'metabolic regulator' that adjusts to meet the varying physiological demands of predictable events such as reproduction, moult and migration. This approach may de-emphasize the tendency to classify GC measurements as 'baseline' or 'stress-induced' when the experience of these animals is sufficiently complex such that these categories are not biologically meaningful.

Finally, much needs to be been done to understand how an organisms' stress physiology is adapted to support (or constrain) the varied life-history strategies within and among species in any vertebrate group. While the allostatic load and reactive scope conceptual models (Romero, Dickens & Cyr 2009; McEwen & Wingfield 2010) provide a rubric for integrating variation in GC levels with intrinsic and extrinsic factors within a life history, the challenge ahead is to design studies to that test specific hypotheses that can be addressed with physiological data in an ecological and evolutionary context. Indeed, mathematical and computational approaches are a hallmark of life-history analyses, for example, optimality modelling with differential equations, dynamic programming, or matrix methods (Roff 1992; Stearns 1992). Therefore, we need to take concepts generated by either allostatic load or reactive scope models one step further and determine quantitative relationships between GCs and survival or reproduction through the use of demographic models (e.g. matrix models, see Fig. 3), for example, to estimate their effects on population and evolutionary dynamics. These models can also be used in a predictive way to assess which life-history traits we should be focusing on when relating the impact of GCs on life-history traits to project population-level effects (e.g. use of parameter elasticities within demographic models, Caswell 2001). Additional statistical methods can be used to quantitatively associate GCs and life-history traits, including survival analysis (Hosmer & Lemeshow 1999), which can be used to integrate GC levels with the timing of life-history transitions. If physiological measures are recorded repeatedly at different times or across different environmental conditions, path analysis (Scheiner, Mitchell & Callahan 2000) can be used to assess impacts of GCs across life-history stages and sum both the positive and negative associations with fitness components to ultimately measure selection differentials on traits

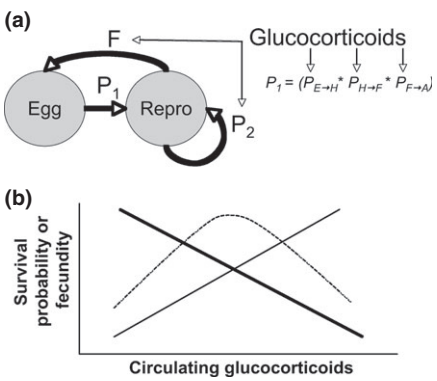

Fig. 3. (a) Given the importance of glucocorticoids (GCs) in the timing of life-history transitions, reproduction, and survival in vertebrates, stress physiologists can contribute to population and conservation biology by explicitly modelling the relationships between GC levels and the survival probabilities (P) between life-history states and fecundity (F) in age or state-based population models (e.g. matrix models, Caswell 2001). (a) Depicted is a hypothetical life stage model of a bird's life history that can be modelled in 1-year increments. Because of this constraint, data relating GC levels to the probability of transitioning from egg to hatching (E→H, hatching success), hatchling to fledging (H→F) and overwinter survival of young of year to the reproductive age class, that is, adults (F→A) need to be modelled as components of P1. P2 is the survival probability of adults (birds of reproductive age). (b) Depending on the species, age or state, the transition probability or fecundity can be a negative, positive, or nonlinear function of the GCs (or there can be no relationship all).

related to HPA/I axis function. Finally, the trajectory of GC levels over time, much like growth curves, can be analysed as a function-valued trait, the shape of which can be related to fitness (Kingsolver, Gomulkiewicz & Carter 2001). Collaborations among environmental endocrinologists and evolutionary and population biologists will facilitate these advances, promote rigorous hypothesis testing, and limit ad hoc analyses that allow for 'just-so' stories that link GC-mediation of life-history traits or trade-offs within evolutionary scenarios.

There is a vast literature accumulating that clearly shows that the HPA/I axis contributes to phenotypic variation in the timing of life stage transitions, survival and reproduction across vertebrates. However, there is still much to be done to garner a full understanding about the ways HPA/I axis activity relates to these fitness parameters across life histories, which is essential to our ability to predict how environmental change will affect the abundance and distributions of natural populations.

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