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Essay

The stress of elaborate male traits: integrating glucocorticoids with androgen-based models of sexual selection

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Stress hormones are emerging as major factors regulating the expression of elaborate male traits. Surprisingly, however, the effects of glucocorticoids on such traits have not been formally integrated with androgen-based models of sexual selection. Here we point out that consideration of glucocorticoid-mediated effects on the phenotype provides new insight into long-standing hypotheses and controversies associated with such models. In particular, androgen-based 'handicap' models of sexual selection characteristically hinge on graded effects of androgens on male traits, but few studies have found support for such a relationship, suggesting that androgens may not be a primary target of selection. We propose, however, that in many instances, androgens may not appear to have a graded effect on the phenotype because elevated glucocorticoids mask the effects of androgens. Glucocorticoids may be inextricably linked to elaborate traits because the energetic demands associated with such traits promote glucocorticoid production. We thus propose that glucocorticoid-mediated effects on male traits warrant re-evaluation of androgen-based models of sexual selection. In particular, we argue that androgen-based handicap models cannot be dismissed based on the lack of evidence for a graded relationship between androgen level and the extent or magnitude of the trait. Our review of the literature indicates that most studies have examined either the effects of androgens or the effects of glucocorticoids, but not both. A more integrated approach involving the effects of both steroids is necessary to fully understand the role of androgens and the endocrine targets of selection associated with the expression of elaborate male traits.

Hormones often give rise to variable phenotypes that are screened by selection (Adkins-Regan, 2005; Ketterson, Nolan, Cawthorn, Parker, & Ziegenfus, 1996; Ketterson et al., 2001; Moore, Hews, & Knapp, 1998; West-Eberhard, 2003). Androgens, for example, often have a dramatic effect on male secondary sex characteristics and reproductive behaviour and have thus become integral components of current models linking proximate physiological mechanisms and the evolution of elaborate male courtship displays (Adkins-Regan, 2005; Emerson, 2001; Folstad & Karter, 1992; Hau, 2007; John-Alder, Cox, Haenel, & Smith, 2009; Ketterson, Atwell, & McGlothlin, 2009; McGlothlin et al., 2008; Moore & Hopkins, 2009). Despite the historical emphasis on androgens in behavioural endocrinology (Berthold, 1849; Nelson, 2011) and continued interest in androgen-dependent sexually selected male traits, we still have a relatively poor understanding of the nature of the relationship between androgen level and the extent or magnitude of trait expression (Adkins-Regan, 2005; Ball & Balthazart, 2008; Fusani, 2008; Hews & Moore, 1997; Rubenstein & Hauber, 2008). An understanding of this relationship, however, is a central consideration for many androgen-based models of sexual selection. A graded or dose-dependent relationship, for example, emerges as a critical factor in maintaining the honesty of androgen-mediated traits, yet in most cases androgens appear to affect trait expression in a threshold or nonlinear manner (Adkins-Regan, 2005; Hews & Moore, 1997). The preponderance of threshold androgenic effects has stimulated considerable debate and even the dismissal of androgen-based models of sexual selection. Below, we describe an alternative explanation for this hormone–trait relationship that incorporates recent findings associated with glucocorticoid-mediated effects on the phenotype. The traits of interest here are male reproductive behaviours and/or traits associated with courtship. Specifically, we describe how glucocorticoids could mask graded androgenic effects on the phenotype and give rise to a nonlinear association between androgen level and the extent or magnitude of sexually selected male traits. This perspective provides new avenues for continued research aimed at understanding the evolution of the endocrine system as a determinant of male phenotype.

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ANDROGEN LEVEL AS A MEDIATOR OF HONEST SIGNALS

The immunocompetence handicap hypothesis (ICHH) (Folstad & Karter, 1992) has been particularly pivotal in bringing androgens to the forefront of models of sexual selection. The ICHH describes a trade-off associated with androgen level wherein elevations in testosterone level promote the development and expression of elaborate male traits but simultaneously compromise the immune system. Androgen-mediated courtship displays are thus expected to provide females with 'honest signals' when choosing mates because only males that are resistant to pathogens (i.e. males that possess 'good genes') would be able to afford the immunosuppressive costs associated with elevated androgen level (see Greives, McGlothlin, Jawor, Demas, & Ketterson, 2006; Mills et al., 2009; Roberts, Buchanan, & Evans, 2004). Androgen-mediated trade-offs, however, are not necessarily limited to potential immunosuppressive effects; the costs could be associated with other androgen-mediated effects as well (e.g. decreased parental care and/or survivorship; Ketterson et al., 2001; Ketterson et al., 2009; Marler & Moore, 1998; McGlothlin et al., 2010; Reed et al., 2006).

A central criticism associated with the ICHH involves the presumed graded dose-dependent relationship between androgen level and the magnitude and/or intensity of elaborate male traits (Adkins-Regan, 2005; Evans, Goldsmith, & Norris, 2000; Hews & Moore, 1997; Hillgarth & Wingfield, 1997; Poulin & Vickery, 1994; Roberts et al., 2004). Specifically, individuals must show a graded dose-dependent relationship between androgen level and the magnitude or intensity of the trait (Fig. 1a) for androgens to maintain the honesty of the signal (Adkins-Regan, 2005; Hews & Moore, 1997). If the extent or magnitude of the trait does not reliably track intraindividual variation in androgen level, as with a threshold scenario (Fig. 1b), then males could down-regulate circulating androgens to the threshold level to circumvent the potential negative fitness consequences associated with higher androgen levels (e.g. immunosuppression) without negatively affecting the quality of the trait (Fig. 1b). In such circumstances, androgen level does not reliably maintain the honesty of the signal (see also Roberts et al., 2004).

Studies examining the relationship between androgen level and the expression of secondary sexual traits are often inadequate to address whether androgens have graded or threshold effects on traits (Adkins-Regan, 2005; Ball & Balthazart, 2008; Hews & Moore, 1997; Roberts et al., 2004). Relatively few studies, for instance, demonstrate that incremental changes in androgen level, above the level required for development or expression of the trait, result in concordant incremental changes in the magnitude or extent of the trait (reviewed by Adkins-Regan, 2005; Hews & Moore, 1997). There is, however, no reason that hormones cannot affect target tissues in a graded fashion (Ball & Balthazart, 2008) and, indeed, there are many cases in which hormones do appear to alter behaviour or trait expression in such a manner (Adkins-Regan, 2005; Balthazart & Ball, 2006; Emlen, Warren, Johns, Dworkin, & Lavine, 2012; Enstrom, Ketterson, & Nolan, 1997; Hill, Enstrom, Ketterson, Nolan, & Ziegenfus, 1999; Ketterson, Nolan, Wolf, & Ziegenfus, 1992; Ketterson et al., 2001; Ketterson & Nolan, 1994; Lewis & Rose 2003; Moore & Miller, 1984; Roberts et al., 2004; Zuk, Johnsen, & Maclarty, 1995). Hence, the apparent prevalence of threshold androgenic effects in the literature is puzzling. Below, we describe how androgen-dependent effects on male phenotype are likely to increase circulating glucocorticoid levels. We then provide an overview of the evidence suggesting that glucocorticoids obscure graded relationships between androgens and sexually selected male traits.

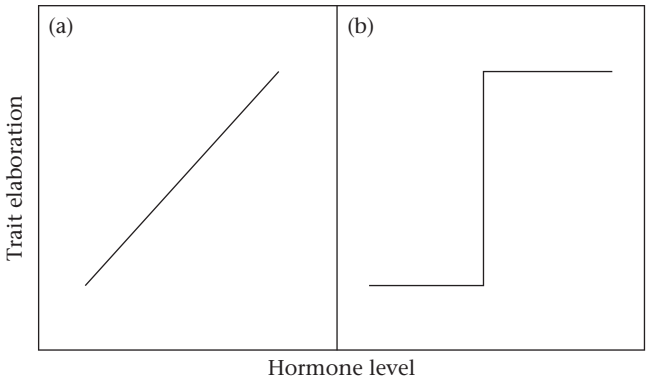

Figure 1. (a) Graded or dose-dependent effects versus (b) threshold effects of hormones on trait elaboration (modified from Hews & Moore, 1997).

GLUCOCORTICOID EFFECTS ON SEXUALLY SELECTED MALE TRAITS

Evidence that Glucocorticoids Promote the Expression of Sexually Selected Male Traits

Sexually selected male traits are often energetically taxing (Andersson, 1994) and glucocorticoids play a prominent role in energy balance and liberating stored energy (Laugero, 2001; McEwen & Wingfield, 2003; Sapolsky, 1992; Sapolsky, Romero, & Munck, 2000; Wingfield & Sapolsky, 2003). Thus, a number of researchers have linked the well-known metabolic effects of glucocorticoids to sexually selected male traits (Bonier, Martin, Moore, & Wingfield, 2009; Bonier, Moore, Martin, & Robertson, 2009; Buchanan, 2000; Eikenaar, Husak, Escallón, & Moore, 2012; Emerson, 2001; Hau, Ricklefs, Wikelski, Lee, & Brawn, 2010; Husak & Moore, 2008; Leary, 2009; Leary, Garcia, Knapp, & Hawkins, 2008; Moore & Jessop, 2003; Romero, 2002; Rubenstein & Hauber, 2008; Wada et al., 2008). The general concept is that moderate elevations in circulating glucocorticoids may be required to meet the metabolic demands associated with an increase in the magnitude or intensity of such traits (e.g. concepts of the 'energy mobilization hypothesis'; Romero, 2002).

Seasonal patterns of glucocorticoid production (e.g. high glucocorticoid levels during the reproductive period) are consistent with this hypothesis (Romero, 2002). Moreover, interspecies differences in baseline glucocorticoid levels have recently been linked to the intensity, and thus metabolic demands, of reproduction. Species where individuals invest more in reproduction, although not necessarily related to the extent or magnitude of elaborate male traits, tend to have higher baseline glucocorticoid levels (Hau et al., 2010).

Considerable correlative evidence suggests that moderate elevations in circulating levels of glucocorticoids are required to channel energy to support costly courtship displays (Buchanan, 2000; Emerson, 2001; Hau et al., 2010; McEwen & Wingfield, 2003; Moore & Jessop, 2003; Romero, 2002). Moreover, the administration of small amounts of glucocorticoids can increase the investment in reproductive behaviours (e.g. parental care in birds), indicating that increased glucocorticoid levels are not merely a consequence of increased reproductive investment but can also promote reproductive behaviour (Ouyang, Muturi, Quetting, & Hau, 2013).

Evidence that Glucocorticoids Negatively Affect the Expression of Sexually Selected Male Traits

While moderate elevations in circulating glucocorticoids potentially promote the elaboration of courtship displays by

mobilizing available energy reserves, high glucocorticoid levels typically negatively affect reproduction (Adkins-Regan, 2005; Greenberg & Wingfield, 1987; Sapolsky, 1992; Sapolsky et al., 2000; Schoech, Rensel, Bridge, Boughton, & Wilcoxen, 2009). Such effects may occur independently of changes in circulating androgen level or via suppression of the hypothalamic–pituitary–gonadal (HPG) axis.

Work performed in male roughskin newts (*Taricha granulosa*) provides one of the most thoroughly studied examples of how changes in circulating glucocorticoids may alter the expression of androgen-dependent male behaviours independently of changes in androgen level. Administration of corticosterone (CORT) in this species inhibits amplexus behaviour (clasping of females by males during mating) in a graded fashion; higher doses elicit a greater decrease in clasping quality (Lewis & Rose, 2003; Moore & Miller, 1984). Moreover, direct application of CORT to medullary motor neurons controlling this behaviour suppresses the amplexus response in seconds, indicating that CORT-induced changes in behaviour occur through nongenomic mechanisms and independently of changes in circulating androgens (Evans, Searcy, & Moore, 2000; Moore & Evans, 1999; Moore & Miller, 1984; Moore, Boyd, & Kelley, 2005). Intensive research aimed at understanding the mechanistic basis for this effect has led to the discovery of membrane-bound neural glucocorticoid receptors (Orchinik, Murray, & Moore, 1991) and evidence that activation of these receptors suppresses the release of the neuropeptide arginine vasotocin (AVT) from medullary neurons controlling amplexus behaviour (Moore & Evans, 1999; Evans, Searcy, et al., 2000). Interestingly, the synthesis of AVT is androgen dependent (Boyd, 1994), illustrating how glucocorticoid effects may override androgenic effects on neural target tissues regulating reproductive behaviour.

Recent evidence suggests that high glucocorticoid levels also negatively affect the extent or magnitude of courtship signals that are important in mate selection by females. For example, in male barn owls, *Tyto alba*, CORT administration decreases melanin-based coloration; females prefer males with melanin-based colour patterns characteristic of males that were not exposed to high CORT levels during the period when melanin-based coloration develops (Roulin et al., 2008). Similarly, male barn swallows, *Hirundo rustica*, with the longest tails (tail length is under directional selection via female mate choice) generally have lower levels of CORT than males with the shortest tails (Saino, Incagli, Mertinelli, & Møller, 2002). However, this correlation must be interpreted with caution because CORT levels at the time of feather development are unknown. In red grouse, *Lagopus lagopus*, CORT level was negatively related to the magnitude of the effect of testosterone treatment on comb size (Bortolotti, Mougeot, Martinez-Padilla, Webster, & Piertney, 2009). In this case, the negative effects of high CORT appear to have been related to a reduction in immunocompetence (e.g. increased parasite infestation), suggesting that the effects of CORT on elaborate traits can be indirect. Importantly, this study showed a reduced effect of androgen treatment on trait elaboration as a result of elevated CORT level, providing another example where CORT overrides and masks androgenic effects on male traits (see also references above to CORT effects in *T. granulosa*).

Considerable evidence exists for a negative effect of elevated glucocorticoids on acoustic courtship signals. In the Great Plains toad, *Bufo cognatus*, for example, CORT injections (resulting in high physiological levels of circulating CORT) caused a reduction in call duration independently of changes in circulating androgens (Leary, Garcia, & Knapp, 2006a, 2006b). Females preferred males producing longer calls (characteristic of males with low CORT levels) in two-speaker playback trials, indicating that males with low levels of circulating CORT are more attractive to females (Leary et al., 2006b).

Work in birds has also emphasized the potential negative effects of high CORT level on sexually selected male vocalizations. Female zebra finches, *Taeniopygia guttata*, preferred males artificially selected for low over high peak levels of circulating CORT (Roberts, Buchanan, Bennett, & Evans, 2007; Roberts, Buchanan, Hasselquist, Bennett, & Evans, 2007). Of the measured morphological traits, none differed between the two artificially selected lines (Roberts, Buchanan, Bennett, et al., 2007; Roberts, Buchanan, Hasselquist, et al., 2007), but male zebra finches with low total and free baseline CORT levels produced songs that were longer in duration and produced them more frequently than males with high total and free baseline CORT levels (Wada et al., 2008). Oral administration of CORT also increases the pitch of evoked calls in this species (Perez et al., 2012), suggesting that CORT-mediated effects on vocalization may be an important factor affecting female mate choice for males with low CORT levels. Similarly, male song sparrows, *Melospiza melodia*, with the lowest CORT responses to restraint stress tended to produce the most complex song (Schmidt, Furlonger, Lapierre, MacDougall-Shackleton, & MacDougall-Shackleton, 2012).

These studies in birds, however, have not shown that females express a preference for the calls characteristic of males with low versus high CORT levels (i.e. this is generally inferred from other research), nor have such studies addressed whether the negative effects of CORT treatment on song are associated with a reciprocal relationship between CORT and androgen levels. Glucocorticoids, for example, can suppress the HPG axis, inhibit the activity of enzymes involved in the synthesis of androgens and/or stimulate the gonadotropin inhibitory hormone production (Calisi, Rizzo, & Bentley, 2008; Chand & Lovejoy, 2011; Greenberg & Wingfield, 1987; Kirby, Geraghty, Ubuka, Bentley, & Kaufer, 2009; Michael & Cooke, 1994; Monder, Sakai, Miroff, Blanchard, & Blanchard, 1994; Moore, Thompson, & Marler, 1991; Sapolsky, 1992; Wingfield & Sapolsky, 2003). Further work is needed to determine whether glucocorticoids or androgens give rise to variation in vocalization in birds. Establishing a reciprocal relationship between the two steroids does not, however, sufficiently address this issue because such a relationship does not eliminate potential direct (i.e. non-androgenic) effects of glucocorticoids on the expression of sexually selected male traits (e.g. DeNardo & Licht, 1993).

Current evidence thus suggests that moderate elevations in circulating glucocorticoids promote the expression of sexually selected male traits via the mobilization of energy reserves, but high levels of glucocorticoids typically inhibit reproductive activity (Breuner, Patterson, & Hahn, 2008; Calisi et al., 2008; Emerson, 2001; Leary, Jessop, Garcia, & Knapp, 2004; Leary et al., 2006a; Leary et al., 2006b; Leary et al., 2008; Moore & Jessop, 2003). Such varied effects could result from disparate actions of multiple glucocorticoid receptor types that differ in ligand affinity (e.g. type I and type II corticosteroid receptors; Sapolsky et al., 2000). Below, we build upon the existence of such varied effects to illustrate the complex relations that are likely to exist among androgen level, glucocorticoid level and sexually selected male traits. We propose that an emphasis on androgens, to the exclusion of glucocorticoids, may provide an incomplete, or even inaccurate, representation of how, and the extent to which, androgen level influences the expression of male traits.

INTEGRATING GLUCOCORTICOIDS WITH ANDROGEN-BASED MODELS OF SEXUAL SELECTION

The Conceptual Model

We begin with a hypothetical scenario, depicted in Fig. 2, to illustrate how glucocorticoids could override and thus mask a graded relationship between androgen level and the magnitude or

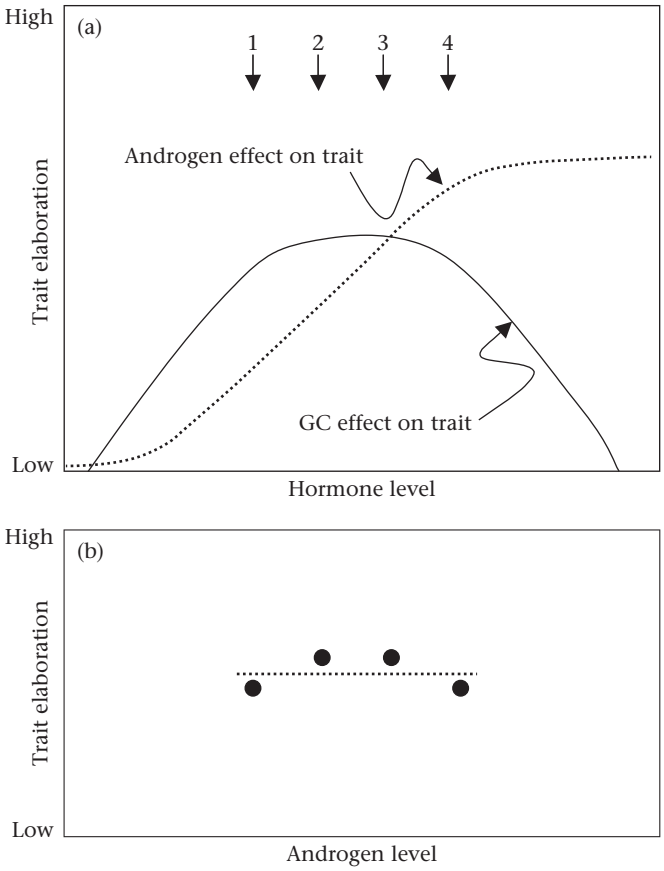

Figure 2. Hypothetical scenario of how glucocorticoids could mask a graded relationship between androgen level and the magnitude or intensity of elaborate male traits. (a) Androgens have a graded effect on trait elaboration that promotes glucocorticoid (GC) production. Glucocorticoids, however, override androgenic effects on trait elaboration. Hence, examination of the relationship between androgen level and trait elaboration (b) over points 1–4, indicated in (a), would suggest no significant relationship between these two variables.

intensity of sexually selected male traits. In this scenario, androgens have a graded effect on the trait across a particular range of androgen levels, below which the trait is not observed and above which there is no further increase in the magnitude or extent of the trait (see Ball & Balthazart, 2008). Glucocorticoid level is positively correlated with androgen level because androgenic effects on the magnitude or extent of the trait promote glucocorticoid production. The effects of high glucocorticoids, however, override androgenic effects on trait expression. Hence, androgens may not appear to have a graded effect on the trait across a specified range of androgen levels (levels 1–4 in Fig. 2a, b).

Our proposed scenario (depicted in Fig. 2) rests on the assumption that androgen and glucocorticoid production are positively correlated as a result of the metabolic demands associated with androgenic effects on sexually selected elaborate male traits (see also Eikenaar et al., 2012 and citations therein; Emerson, 2001). A positive relationship between androgens and glucocorticoids is central to the 'stress-linked' version of the ICHH as well. This modification to the ICHH proposes that glucocorticoids, not androgens, suppress the immune system to maintain the honesty of the signal (see Bortolotti et al., 2009; Evans, Goldsmith, et al., 2000; Moore et al., 2011; Roberts, Buchanan, Hasselquist, & Evans, 2007). Although a positive relationship between androgens and glucocorticoids often does not appear to exist and is potentially problematic for both the stress-linked version of the ICHH (Moore et al., 2011) and our proposed model, the reported discrepancies in this relationship may lie in (1) the statistical approach used to examine the relationship and (2) variation in the sensitivity of the HPG axis to elevated glucocorticoids (e.g. see Jennings, Moore, Knapp, Matthews, & Orchinik, 2000; Wingfield, O'Reilly, & Astheimer, 1995; Wingfield et al., 1998). For example, simple linear regression analysis using hormone data that span the range of circulating levels may be misleading if there is a threshold level of glucocorticoids that suppresses androgen production (e.g. the relationship with androgen level may be nonlinear over the entire range of glucocorticoid levels). Such an approach could easily result in a positive or negative relationship between the two steroids, depending upon the range of glucocorticoid levels present and the relative proportions of individuals with low versus high circulating glucocorticoid levels. This analytical approach may also be misleading for understanding the relationship between hormone level and the extent or magnitude of the trait. If, for example, low to moderate levels of glucocorticoids promote trait expression but high glucocorticoids negatively affect the extent or magnitude of the trait, then glucocorticoid level may not appear to be related to trait expression across the entire range of circulating glucocorticoid levels. Thus the same potential problem of interpretation of a glucocorticoid–trait relationship exists that we described above for androgen–trait relationships. The relationship between hormone level and trait is further complicated by variation in the sensitivity of target tissues, including components of the HPG axis, as a result of variation in receptor affinities or density and/or variation in hormone-binding globulins. For example, in the tree lizard *Urosaurus ornatus*, males from the two alternative reproductive tactics differ in the effect of CORT on testosterone levels. In response to elevations in CORT, the more aggressive, territorial male morph shows smaller declines in circulating testosterone levels than does the less aggressive, nonterritorial morph (Knapp & Moore, 1997), likely due at least in part to the greater plasma corticosteroid-binding capacity of territorial males (Jennings et al., 2000).

Stabilizing selection on androgen level and sexually selected male traits as a result of glucocorticoid actions

Under the scenario depicted in Fig. 2, glucocorticoids are expected to effectively stabilize selection on androgen level. Consider, for example, directional selection via female mate choice on the intensity and/or magnitude of elaborate courtship signals that indirectly selects for increased androgen level in males (Fig. 3a). If female preferences drive concordant elevations in glucocorticoid level (e.g. to support energetically costly traits), then directional selection on androgen level may be effectively truncated by the negative consequences (i.e. decreased signal quality) associated with high glucocorticoid level (Fig. 3b).

Stabilizing selection on androgen level is also expected to occur when elevated glucocorticoid level suppresses reproductive behaviour via negative effects on circulating androgen level. To illustrate such a scenario, we highlight concepts of the 'energetics–hormone–vocalization' model (EHV model; Emerson, 2001) that describes the predicted relationship between androgens, glucocorticoids and vocalizations in anuran amphibians. The EHV model predicts a graded effect of androgen level on vocal effort (e.g. the total energy invested in vocalization, which is often related to vocal quality), which, in turn, modulates circulating glucocorticoid level (Fig. 4). Androgen-mediated effects on the energy invested in vocalization are expected to drive elevations in circulating glucocorticoids to levels that negatively affect androgen level and, thus, vocal effort. Hence, the negative effects of high glucocorticoids on circulating androgens are expected to stabilize selection on circulating androgens and elaborate traits.

Consistent with the above scenarios, androgen and glucocorticoid levels are often highly correlated (either positively or

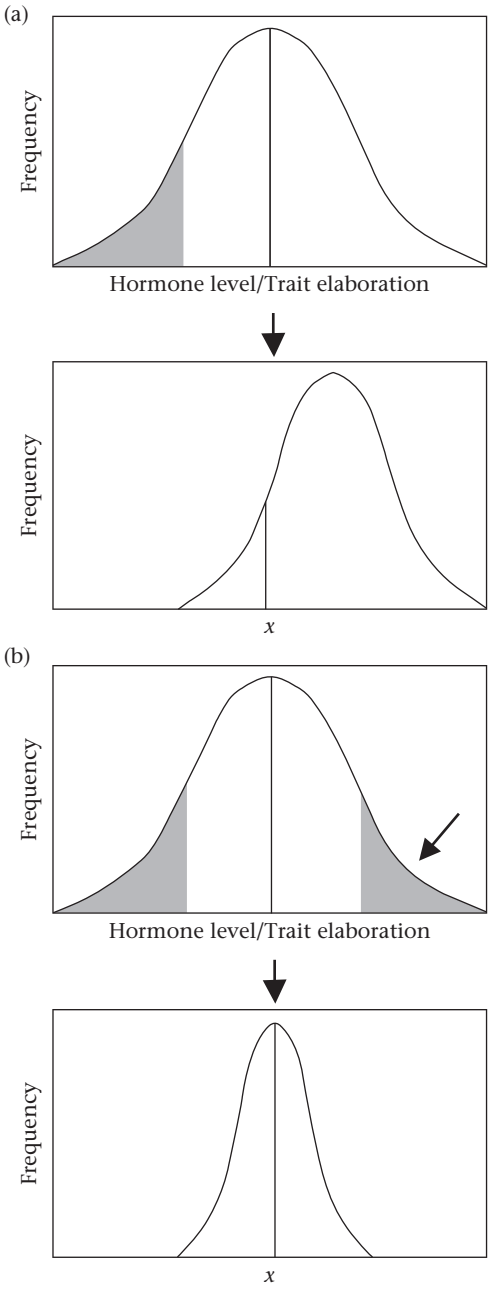

Figure 3. Model for selection under two scenarios of endocrine effects on elaborate male traits. (a) Female mate choice is expected to result in directional selection on elaborate male traits and androgen level (shaded portion = low androgen level disadvantage; x = mean hormone level/trait elaboration). (b) Negative effects of high glucocorticoid level on signal quality, however, could stabilize selection on androgen level (shaded portion indicated with arrow = disadvantage resulting from high glucocorticoid level).

negatively) (Adkins-Regan, 2005; Bonier, Martin, et al., 2009; Bonier, Moore, et al., 2009; Eikenaar et al., 2012; Roberts et al., 2004; Romero, 2002) and androgen administration often results in an increase in glucocorticoid levels (Adkins-Regan, 2005; Benner & Woodley, 2007; Casto, Nolan, & Ketterson, 2001; Duffy, Bentley, Drazen, & Ball, 2000; Evans, Goldsmith, et al., 2000; Ketterson et al., 1991; Ketterson et al., 2001; Owen-Ashley, Hasselquist, & Wingfield, 2004; Poiani, Goldsmith, & Evans, 2000; Van Hout, Eens, Darras, & Pinxten, 2010).

These examples thus illustrate how immunosuppressive costs associated with the ICHH and the 'stress-linked' version of the ICHH

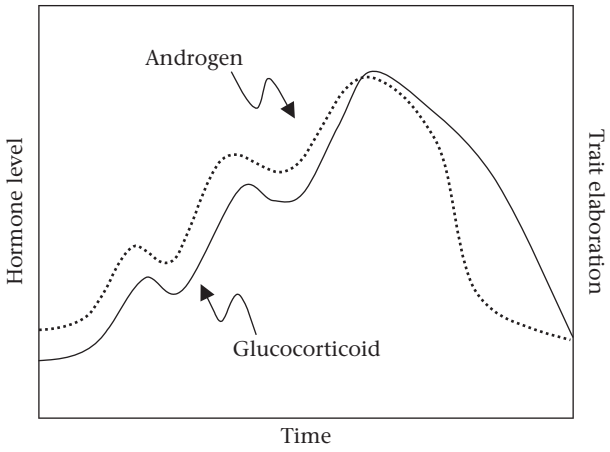

Figure 4. The energetics–hormone vocalization model (Emerson, 2001). Androgen level reflects the extent of trait elaboration, and an increase in the extent or magnitude of the trait drives a concordant increase in glucocorticoid level. At some point, however, glucocorticoid level negatively affects androgen level and, thus, the expression of the trait. Glucocorticoid level subsequently declines because of reduced energetic demands of a less elaborate trait. Although this model was originally proposed in the context of anuran advertisement calls, it may also apply to morphological traits that are energetically expensive to produce (figure modified from Emerson, 2001).

may not be required to stabilize selection on androgen level; glucocorticoids could also be important mediators of honest male signalling. The interrelationships among energetic state, glucocorticoid level and trait elaboration, for example, suggest that more elaborate male traits could be a reliable indicator of a male's ability to cope with metabolic stress (e.g. see reviews on developmental stress effects by Schoech et al., 2009; Schoech, Rensel, & Heiss, 2011; Schoech, Rensel, & Wilcoxen, 2012; Spencer & MacDougall-Shackleton, 2011).

CONCLUSIONS AND FUTURE DIRECTIONS

Adrenal glucocorticoids play major roles in metabolism, immunity, reproduction and behaviour, which has led numerous investigators to emphasize their potential importance in measures of performance and fitness (Adkins-Regan, 2005; Bonier, Moore, et al., 2009; Breuner et al., 2008; Buchanan, 2000; Emerson, 2001; Evans, Goldsmith, et al., 2000; Hews & Moore, 1997; Husak & Moore, 2008; Leary, 2009; Leary et al., 2006b; Love, Breuner, Vézina, & Williams, 2004; Møller, 1995; Moore & Hopkins, 2009; Poiani et al., 2000; Roberts et al., 2004). Surprisingly, however, glucocorticoids have received far less attention than androgens in endocrine-based models of sexual selection (Adkins-Regan, 2005; Bortolotti et al., 2009; Buchanan, 2000; Møller, 1995; Moore et al., 2011). As outlined here, however, glucocorticoids can directly or indirectly (e.g. via reciprocal interactions with androgens) modulate the expression of sexually selected elaborate male traits and may thus be critical to understanding androgen-based models of sexual selection. Glucocorticoids appear to be intimately linked to the expression of many elaborate male traits because the energetic costs associated with such traits may promote production of these hormones. Yet glucocorticoids may also negatively affect trait elaboration. The latter effect may help explain the reported discrepancies associated with graded versus threshold androgenic effects on elaborate male traits and the related controversy associated with the role of androgens in maintaining the honesty of male signals.

Although several lines of evidence invoke glucocorticoid-mediated mechanisms to explain variation in elaborate male traits, most studies to date have not addressed whether such effects

are a result of glucocorticoid-mediated effects on androgen level. An inverse relationship between androgen level and glucocorticoid level introduces the problem of assessing whether variation in trait elaboration is related to glucocorticoid level or to androgen level. Less elaborate traits, for example, may be the result of low androgens or high CORT or, alternatively, more elaborate traits could be the result of high androgens or the 'release' from inhibitory effects of high CORT on trait elaboration (Fig. 5); both situations would result in the same plot of trait elaboration and hormone level we depict here. Determining which situation holds in a particular case is essential for identifying the source of variation in trait expression and, thus, the endocrine target(s) of selection. However, glucocorticoids emerge as important factors regulating the expression of elaborate male traits, whether they act directly on the target tissues or via negative effects on androgen level. Further studies that incorporate measurement of androgen and glucocorticoid concentrations should assist in identifying the potential endocrine targets of selection. Such studies should be carefully designed to examine the nature of the relationship between androgen level, glucocorticoid level and the magnitude or extent of trait elaboration. Repeated measures approaches using hormonal manipulation with varying doses of one hormone while controlling for circulating levels of the other, while potentially challenging, would be extremely beneficial for understanding how these hormones are involved in regulating trait expression.

An additional fruitful avenue for future research is related to selection for hormone level or hormone sensitivity. Androgen level and glucocorticoid level are both known to have high heritability values in a variety of vertebrate species (Bartels, de Geus, Kirschbaum, Sluyter, & Boomsma, 2003; Bartels, Van den Berg, Sluyter, Boomsma, & de Geus, 2003; Bogaert et al., 2008; Evans, Roberts, Buchanan, & Goldsmith, 2006; Kampenaers, Peters, & Foerster, 2008; King, Cline, & Hubbard, 2004; Ring et al., 2005; Roberts, Buchanan, Bennett, et al., 2007; Roberts, Buchanan, Hasselquist, et al., 2007; Satterlee & Johnson, 1988; Tschirren, Sendecka, Groothuis, Gustafsson, & Doligez, 2009), supporting the premise that indirect selection for steroid hormone level via female mate choice can have a significant evolutionary impact on the endocrine system. However, to our knowledge, no studies have examined whether selection for androgen levels results in concordant selection on glucocorticoid levels. Such studies are needed to understand what females potentially gain by selecting males with high androgen levels. In other words, do offspring with high androgen levels also tend to have high glucocorticoid levels? We were able to find only one CORT selection study where testosterone was also monitored. In that study of zebra finches, the relationship between testosterone and CORT levels varied depending on the generation of offspring used for the analysis (Roberts, Buchanan, Hasselquist, et al., 2007).

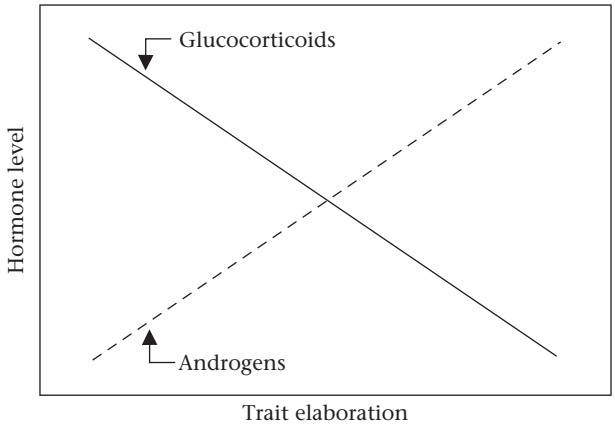

Figure 5. Challenge to interpreting hormone effects on elaborate male traits when androgens and glucocorticoids are reciprocally related. It is often assumed that the negative effects of high glucocorticoids on androgen level negatively influence trait expression. However, glucocorticoids can influence traits independently of changes in circulating androgen levels. Hence, in cases where a reciprocal relationship between the two steroids exists, it is not clear whether more elaborate traits are the result of high androgens or low glucocorticoids (e.g. the 'release' of glucocorticoid-mediated effects on elaborate male traits). Note that the figure focuses on a reciprocal relationship between the two steroids and does not incorporate potential positive effects of low to moderate levels of glucocorticoids on elaboration of the trait.

Throughout this essay, we have emphasized circulating hormone levels as they relate to trait elaboration. We did this because these are the most common data currently available related to this topic. In doing so, we recognize that we have neglected other critical components of the endocrine system that are undoubtedly important in regulating trait expression (see Ketterson et al., 2009), or other means of assessing hormone levels such as response to injections of trophic hormones. Most notably, variation in binding globulins and receptor densities/affinities can profoundly alter individual sensitivity to circulating hormone levels (see Breuner & Orchinik, 2002; Lattin & Romero, 2013; Rosvall et al., 2012; but see Schoech, Romero, Moore, & Bonier, 2013 for an alternative interpretation of the importance of corticosteroid-binding globulins). Similarly, organizational effects of androgens or glucocorticoids could influence the sensitivity of adult males to activational effects of either of these hormones. Studies related to trait elaboration that incorporate all of these components of the endocrine system are, to our knowledge, currently lacking and are likely to require collaborative efforts among researchers with expertise in different methodological techniques. Nevertheless, such efforts are needed and are likely to contribute significantly to our understanding of the endocrine system, how it regulates the expression of elaborate male traits, and how such regulation has evolved and is maintained.

The emphasis placed here on glucocorticoid level as it relates to sexually selected elaborate male traits is not intended to merely redirect concepts of androgen-based models in favour of another hormone. Rather, we have attempted to identify a component of the endocrine system that, when integrated with current androgen-based models, provides important insights into the hormonal mechanisms regulating elaborate male traits and the selective pressures involved in shaping the relationship between such traits and the endocrine system. We argue that androgen-based handicap models of sexual selection should not be dismissed based on the lack of evidence for graded androgenic effects on elaborate male traits if glucocorticoids had not been measured simultaneously. We hope that this essay stimulates further study on the interrelationships among glucocorticoid level, androgen level and trait elaboration.

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