Invited Review

Stress and sexual signaling: a systematic review and meta-analysis

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The vertebrate stress response has been shown to suppress investment in reproductive and immune function and may also lead to a reduced investment in the production of secondary sexual traits. However, it has been difficult to model roles of stress in sexual selection due to the inconsistent results seen in empirical studies testing for the effect of stress on the expression of secondary sexual traits. We conducted a phylogenetically controlled meta-analysis of published associations between physiological correlates of stress and sexual signaling in vertebrates in order to identify any consistent patterns. Our analysis included signaling in both males and females, 4 stress measures, and 4 categories of sexually selected traits (vocalizations, traits that varied in size, traits that varied in coloration, and opposite-sex preference). Across 38 studies of 26 species, there was no significant relationship between physiological correlates of stress and the expression of sexual signals. Mean effect size, however, varied significantly across the 4 types of sexually selected trait. We propose development of a model that incorporates the nuanced effects of species ecology, trait type, ecological context, and the complex nature of the physiological stress response, on the expression of sexually selected traits.

Key words: **glucocorticoid, meta-analysis, secondary sexual trait, sexual selection, sexual signaling, stress.**

INTRODUCTION

There is a growing movement toward incorporating the organizing role of stress (i.e., conditions where environmental demands exceed an organism's regulatory capacity; Koolhaas et al. 2011) on the allocation of somatic resources, into life-history models of behavior (Buchanan 2000; Korte et al. 2005; Husak and Moore 2008; Bonier et al. 2009; Moore and Hopkins 2009). The vertebrate stress response, for example, includes adaptive activation of the hypothalamic–pituitary–adrenal axis culminating in the release of glucocorticoids (GCs), which divert resources away from long-term functions and into short-term priorities (Cote et al. 2006). Although this promotes survival in the short term, chronically elevated GCs suppress reproduction (Sapolsky et al. 2000) and immune function (Martin 2009). GCs, then, may mediate the relationship between the environment and behavioral trade-offs.

The potential for stress to influence the expression of secondary sexual traits has long been recognized by ecologists (e.g., Buchanan 2000). Originally, GCs were predicted to influence sexual signals indirectly via effects on the immune system (Møller 1995; Buchanan 2000) either independently or in interaction with testosterone

(Buchanan 2000; Roberts et al. 2007; Husak and Moore 2008). Although testosterone has received most attention to date, it does not account for complexity in the cross-species data, and GCs have been proposed to interact with the sex hormone in effects on secondary sexual traits (Roberts et al. 2004). More recently, the physiological stress response itself has been proposed to be under sexual selection, such that secondary sexual traits provide cues to individual differences in, for example, stress reactivity or the efficiency of negative feedback (Pfaff et al. 2007; Roberts et al. 2007; Husak and Moore 2008; Bortolotti et al. 2009; Schmidt et al. 2012). Finally, GCs have been proposed to influence secondary sexual traits indirectly via effects on body condition (Husak and Moore 2008).

At first glance, empirical evidence for effects of stress on the expression of signals used to attract the opposite sex is inconsistent, with some studies reporting detrimental effects of physiological proxies of stress (e.g., Douglas et al. 2009), others an enhancing effect (e.g., Fitze et al. 2009) and some reporting no relationship (e.g., Setchell et al. 2010). Meta-analysis is well suited to determining common effects across a range of study systems, especially when empirical results are mixed and many studies may report nonsignificant results due to low statistical power (Arnqvist and Wooster 1995; Koricheva et al. 2013). Meta-analysis also allows us, sample size permitting, to investigate potential moderators of

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effect size, which may generate such inconsistent results (Jennions et al. 2012; Koricheva et al. 2013). We thus performed a phylogenetically controlled meta-analysis of published studies in which the effect of physiological proxies of stress on the expression of secondary sexual traits was reported. Our first aim was to crystallize any consistent relationships between proxies of stress and the expression of traits across species in order to determine which, if any, of the proposed roles of stress are best supported by the data. In addition, we analyzed 4 potential sources of variation in the observed effect sizes: 1) the sex of the signaler, 2) the measure of stress; 3) the type of signal; and 4) taxonomic group.

METHODS

Literature search

We conducted a systematic review of studies published up to November 2014 concerning the relationship between stress and secondary sexual traits. We followed the PRISMA protocol for conducting systematic reviews (Moher et al. 2009; Nakagawa and Poulin 2012). On 22 November 2014, we searched for the following keywords using the TOPIC field in Web of Science ("stress" OR "glucocorticoid" OR "corticoster*") AND ("sexual trait" OR "sexual selection" OR "sexual signal*" OR "mate choice" OR "attracti*"). We also contacted authors of relevant publications to identify any additional records. The number of records obtained from each of these approaches is given in the Supplementary Material. In Figure 1, we present a PRISMA flow diagram showing the number of records obtained from our searches and the number of records excluded following the application of our selection criteria outlined below.

Criteria for study inclusion

We only included those studies in which the following criteria were met: 1) subjects were adults; 2) subject sex was specified; 3) physiological indices of stress were measured; and 4) there was sufficient statistical information to calculate an effect size (either in the publication or provided by the author). We excluded 13 studies that did not meet these criteria, as well as a subset of results from 1 further study (see Figure 1 and Supplementary Table S1). This yielded a sample of 118 results from 38 studies of 26 species (for all effect sizes, see Supplementary Table S3). We included data concerning both males and females. We obtained effect sizes from 4 vertebrate classes: amphibians, reptiles, birds, and mammals (Burmeister et al. 2001; Saino et al. 2001; Parker et al. 2002; Saks et al. 2003; Leary et al. 2004; Garamszegi et al. 2006; Leary et al. 2006a, 2006b; Pfaff et al. 2007; Koren et al. 2008; Leary et al. 2008; Maney et al. 2008; Pérez-Rodríguez and Viñuela 2008; Bortolotti et al. 2009; Macdougall-Shackleton et al. 2009; Cote et al. 2010; del Cerro et al. 2010; Edler and Friedl 2010; Lobato et al. 2010; Mougeot et al. 2010; Setchell et al. 2010; Moore, Al Dujaili, et al. 2011; Moore, Cornwell, et al. 2011; Assis et al. 2012; Rantala et al. 2012; Barron et al. 2013; Henderson et al. 2013; Jenkins et al. 2013; Kennedy et al. 2013; Lendvai et al. 2013; Merrill et al. 2013; Rantala et al. 2013; San-Jose and Fitze 2013; San-Jose et al. 2013; Svobodová et al. 2013; Weiss et al. 2013; Merrill et al. 2014; Grunst ML and Grunst AS 2014).

Four categories of stress measurement were reported: baseline GCs, peak or total GCs produced in response to a stressor, experimental elevation of GCs, and long-term stress. Baseline GCs were typically measured within 3–5 min of capture (e.g., Douglas et al. 2009). Experimental elevation of GCs up to 4 times above baseline

was achieved via subcutaneous implants containing GCs (e.g., San-Jose and Fitze 2013). Long-term stress was assessed in 3 ways: GCs deposited in feathers, feces or hair; the ratio of heterophils to lymphocytes (a white blood cell count that correlates with baseline GCs; Vleck et al. 2000); and the expression of heat shock proteins (highly conserved proteins that are elevated under stress; Sørensen et al. 2003). Both heterophil-to-lymphocyte ratio (Davis et al. 2008) and heat shock proteins (Sørensen et al. 2003) are widely used as proxies of recent and long-term stress in the ecological literature.

The effect sizes we obtained considered a wide range of secondary sexual traits, which we sorted into 4 categories: coloration, vocalization, morphological traits, and opposite-sex preferences. The coloration category included examples in birds, mammals, and reptiles. The amount of coloration was measured in several different ways, including brightness, hue, saturation, proportion of structure (e.g., eye ring) that is pigmented, ultraviolet reflectance, and color reflectance. The vocalization category included singing in birds and calling in amphibians and a mammal species (rock hyrax *Procavia capensis*). The parameters measured varied according to the nature of vocalization in each species and included song rate, complexity, and repertoire size in birds; the latency to call, call duration, call rate, and vocal effort in amphibians, and whether calling/singing was observed or not (rock hyrax, amphibians). Effect sizes included in the morphological trait category all considered bird species and assessed the size of secondary sexual characters, such as comb or tail length. We also included in this category effect sizes considering the size of a colored structure (but not the coloration itself), such as epaulet size in the red-winged blackbird (*Agelaius phoeniceus*). Finally, while not a secondary sexual trait per se, opposite-sex preference was included as an indirect measure of the level of sexual signaling, with the assumption being that attractiveness to the opposite sex is a function of investment in secondary sexual traits. We rely on author judgments regarding whether each trait is a secondary sexual trait or not. For full coding of effect sizes for each moderator variable, see Supplementary Table S3.

Effect sizes

We used Pearson's product moment correlation coefficient (r) as the measure of effect size, as it was easily computable from statistical information included in most of the studies returned by the systematic review, and is an intuitive measure of effect size that is widely used in meta-analysis (Rosenthal 1991). Here, r represented the magnitude of an association between a physiological index of stress and the expression of a secondary sexual trait, or of a difference in expression of a secondary sexual trait between individuals exposed to exogenous GCs and controls. If studies did not report r, it was computed from the available statistical information or from additional information provided by the author using the Practical Meta-Analysis Effect Size Calculator (http://www.campbellcollaboration.org) following established methods (e.g., Rosenthal 1991). Supplementary Table S2 gives full details on the calculation of effect sizes when r was not reported. If multiple valid effect sizes were presented for a given study, we included them all and controlled for the possible nonindependence between effect sizes arising from this by including study ID as a random effect in all models (see below).

Before performing the analysis, all effect sizes were converted using Fisher's Z transform of the correlation coefficient (Zr), which has more desirable properties than r when approaching ±1 (Koricheva et al. 2013). All models were run using Zr. Mean effect size estimates derived from the models were then converted back to r for presentation. The associated variance for each effect size was calculated as $1/(n-3)$ (Borenstein et al. 2009).
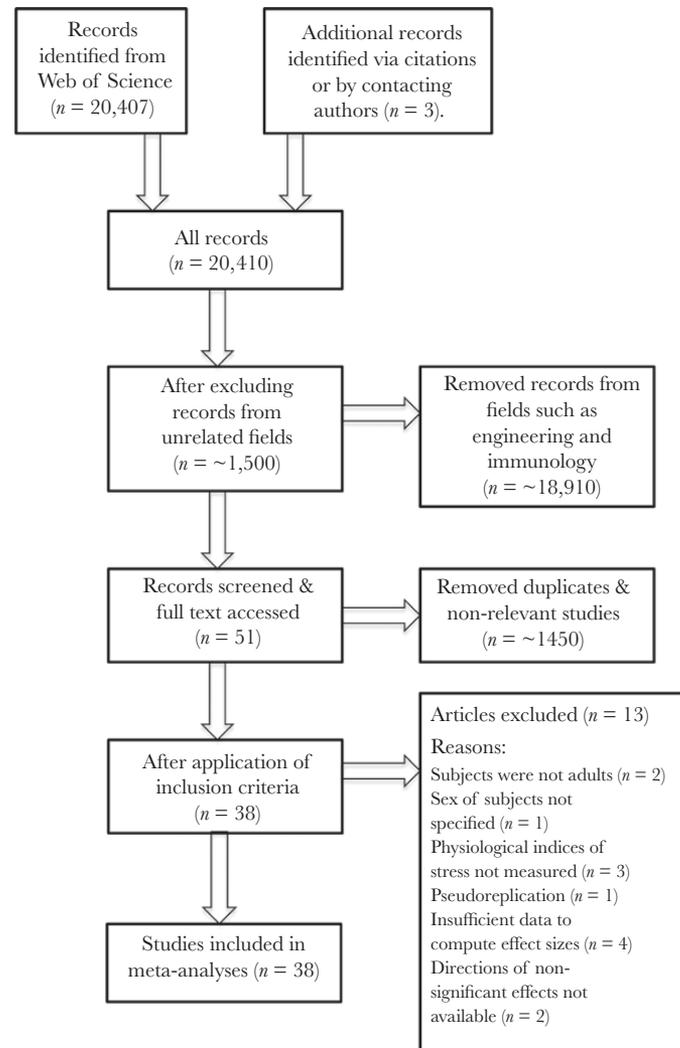
Figure 1
PRISMA flow chart showing results of literature search and study selection criteria and process. Supplementary Table S1 shows studies excluded from analyses.

Phylogeny

Recent developments in meta-analysis have allowed researchers to control for the potential nonindependence of effect sizes due to phylogenetic history, by incorporating phylogenetic relatedness as a random factor in meta-analysis models (Hadfield and Nakagawa 2010). This can be done even when accurate branch length data is lacking. As there is no single phylogeny available for all species included in the analysis, we constructed a supertree by combining multiple trees from several different sources. We used taxonomic groupings in cases where phylogenetic data were not available for species in our sample (Hadfield and Nakagawa 2010). We obtained phylogenetic trees from several sources. For the basal relationships among tetrapods, we used Xia et al. (2003). For the relationships among amphibians, we used Pyron and Wiens (2011). For the relationships among mammals, we used Murphy et al. (2001). For the relationships among birds, we used Hackett et al. (2008) and Ericson et al. (2006), with trees created using the online tool (birdtree.org) accompanying Jetz et al. (2012).

As branch length data were not available for this phylogeny, we first set all branch lengths to one. The tree was then transformed to make all tips contemporaneous using FigTree v1.4 using the cladogram option. Thus, total branch length was determined based on the total number of nodes in the tree. The final tree can be seen in Figure 2. Note that

branch lengths are likely underestimated for distantly related lineages and overestimated for lineages containing several species (e.g., *Ficedula*).

Meta-analysis

We implemented multilevel meta-analyses using a Bayesian linear mixed-effect model approach. Multilevel meta-analytic models are random-effects models (see Borenstein et al. 2009) incorporating additional random factors (following Nakagawa and Santos 2012). This allowed us to control for 3 potential sources of nonindependence in our dataset. In several cases, we obtained multiple effect sizes from a single study and from different studies testing a single species. We controlled for this by including study ID and species ID as random effects in all models. Nonindependence in effect sizes may also arise due to phylogenetic inertia, so that the relationship between stress and secondary sexual trait expression is more similar for closely related species (Hadfield and Nakagawa 2010; Koricheva et al. 2013). Phylogeny was thus included as a random effect by incorporating the phylogenetic tree shown above. All the models presented included study ID, species ID, and phylogeny as random factors.

Meta-analysis models were implemented using the MCMCglmm function from the package *MCMCglmm* (Hadfield 2010). Details on MCMCglmm model specification and testing are presented in the
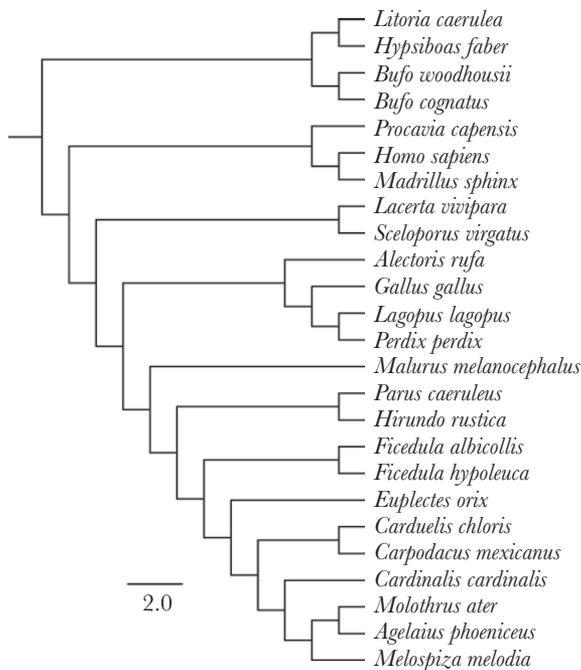
Figure 2
Phylogeny included in meta-analysis (see main text for details).

supplementary material. All results presented are based on models fitted using an inverse gamma prior for all random effects and residuals (following Lim et al. 2014). We first ran an intercept-only model to determine the mean effect size across all studies. We present our results as mean posterior estimates of r (back-converted from Zr after analysis), and consider a mean estimate to be significantly different from zero if the highest posterior density interval (lower highest posterior density interval [LHPD] to upper highest posterior density interval [UHPD], also known as the 95% credible interval) does not overlap zero.

We assessed the total level of heterogeneity among effect sizes using a modified version of the I^2 statistic (Higgins et al. 2003), following Nakagawa and Santos (2012). The original I^2 statistic describes the percentage of total variation in effect sizes that is due to heterogeneity rather than chance (Higgins et al. 2003). However, this statistic has to be modified when additional random effects are included in the model. This method can also be used to partition total heterogeneity into that associated with each of the random effects in the model (Nakagawa and Santos 2012). In other words, this allows us to assess the percentage variance in effect size explained by the different random effects (Lim et al. 2014). Substantial residual heterogeneity remaining after accounting for the random effects indicates that there may be further factors influencing effect size that are not included in the model. We follow Higgins et al. (2003) in considering I^2 values of 25%, 5%, and 75% as representing small, medium, and large amounts of heterogeneity, respectively.

The intercept-only model indicated significant heterogeneity in effect sizes even after variance associated with the 3 random factors was accounted for, and so we next investigated potential moderators of mean effect size using a model-selection approach (Nakagawa and Santos 2012). We performed a series of meta-regression models, each of which included study ID, species ID, and phylogeny as random effects, and one or more categorical fixed effects. Model fit was determined using the deviance information criterion (DIC), which is a Bayesian equivalent of traditional information theoretic

criteria, and a change in DIC of 2 or more was considered to significantly improve model fit (Spiegelhalter et al. 2002). Finally, we used a separate meta-regression model (minus the intercept) for each categorical fixed effect (taxonomic class, sex, stress measure, and trait type) to estimate the mean effect size for each factor level. Each model included study ID, species ID, and phylogeny as random effects.

We looked for signs of 2 types of publication bias in our dataset. First, we tested for a bias associated with the failure to publish nonsignificant or positive results (Koricheva et al. 2013) in 2 ways. We tested for a relationship between effect size and study precision (1/standard error) using linear regression (Egger et al. 1997). Due to the potential nonindependence of effect sizes in our dataset (due to being measured in the same study or species or due to shared ancestry), we used residual effect size, as residuals are theoretically independent of each other (Nakagawa and Santos 2012). We also performed a trim-and-fill analysis using the package *Metafor* (Viechtbauer 2010). This test explicitly searches for asymmetry in the funnel plot (showing the relationship between effect sizes and a measure of their variance), which is assumed to reflect publication bias (Duval and Tweedie 2000). The trim-and-fill function then imputes "missing" effect sizes until the funnel plot is symmetrical and then gives a new effect size estimate from a meta-analysis model including these new effect sizes (Duval and Tweedie 2000). Again, due to nonindependence of effect sizes, this analysis was performed on the residuals (Nakagawa and Santos 2012). The difference in mean effect size estimated from this analysis was then used to adjust the original mean effect size (and associated HPD interval) from the intercept-only model. Second, we assessed whether there was any temporal trend in mean effect size by testing for the rank correlation between effect size and year of publication (Koricheva et al. 2013). A significant temporal trend could reflect publication bias if, for example, studies showing nonsignificant effects are less likely to be published following the early buzz surrounding a new theory (Koricheva et al. 2013).

All analyses were performed using R v3.2.2 (R Development Core Team 2015). All code used in the analysis is included in the Supplementary Material.

RESULTS

Across all effect sizes there was no significant correlation between stress levels and the degree of secondary sexual signaling (intercept-only MCMCglmm; posterior mean = −0.08, LHPD = −0.22, UHPD = 0.03, k = 118, $N_{studies}$ = 38, $N_{species}$ = 25). There is, therefore, no general signaling of level of stress by secondary sexual traits across species, stress measures, and traits. Total heterogeneity was large however (I^2_{total} = 77.81%). The amount of variance explained by the 3 random factors was small (I^2_{study} = 26.76%, $I^2_{species}$ = 8.88%, $I^2_{phylogeny}$ = 5.59%), with substantial residual variance remaining after accounting for them ($I^2_{residual}$ = 36.59%).

As there was substantial heterogeneity in the dataset, we next used a model-selection approach to investigate potential categorical moderators of effect size. Adding taxonomic class, sex, or stress measure as a categorical fixed effect to the meta-analytic model did not improve the model fit (Figure 3; see also Supplementary Table S4). Accordingly, none of the categories associated with taxonomic class, sex, or stress measure exhibited a mean effect size that was significantly different from zero (Supplementary Table 1). However, model fit was significantly improved by the addition of secondary sexual trait type as a fixed effect (Figure 3; see also Supplementary

Table S4), suggesting this factor explains some of the observed heterogeneity in effect sizes.

A meta-regression indicated that there is a significant negative mean effect size when considering only those effect sizes associated with opposite-sex preferences (Table 1), such that stress rendered to be associated with mate preferences, with lower stress individuals favored. However, the upper highest posterior density estimate is very close to zero (−0.010). There was no significant effect of the remaining 3 secondary sexual trait types.

We found mixed evidence for publication bias in the dataset. Egger's regression suggested there was no significant funnel plot asymmetry ($F_{1,116} = 0.1$, $P = 0.75$; $\beta = 0.004$, intercept = −0.03).

However, a trim-and-fill analysis on the residual effect sizes suggested that 13 effect sizes were "missing" from the right hand side of the funnel plot. After imputing these missing effect sizes, the mean effect shifted by 0.048 (Supplementary Figure S1). Adjusting our original mean effect size estimate (from the intercept-only model) using this value still resulted in a nonsignificant result (mean = −0.032, LHPD = −0.169, UHPD = 0.075, $k = 131$). In terms of temporal patterns, we found no correlation between effect size and year (Spearman's rank correlation: $r_s = -0.03$, $P = 0.77$). However, all the studies included were published relatively recently (between 2001 and 2014), and a temporal trend is probably unlikely over such a short range.
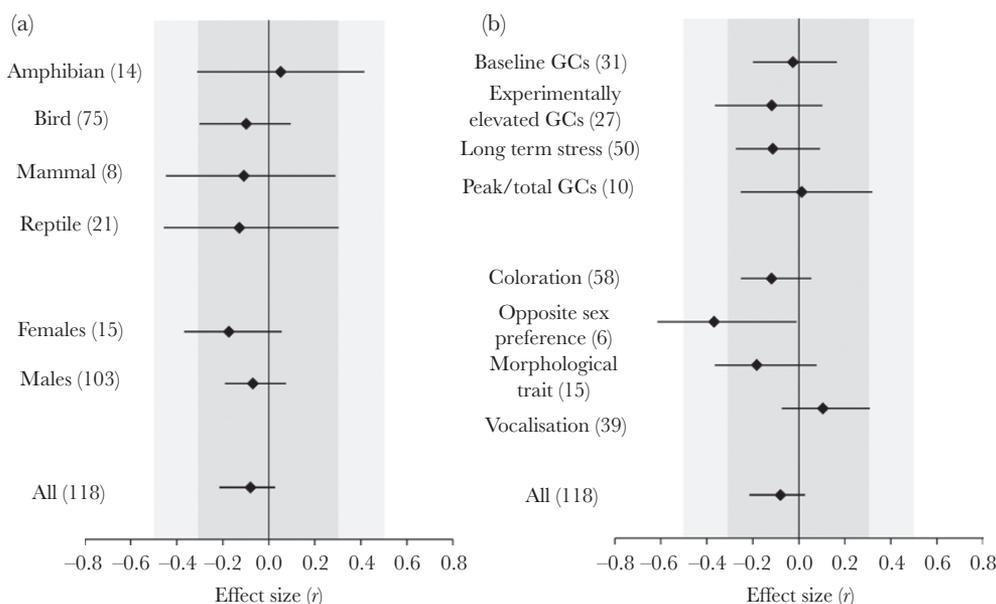

Figure 3

Forest plot showing the mean effect size estimate for each level of the 4 categorical moderator variables, considering (a) taxonomic class and sex and (b) stress measure and sexually selected trait. Diamonds show the mean posterior estimate from the model, and the error bars represent the 95% highest posterior density interval. Numbers in parentheses indicate the number of effect sizes for each subgroup. The dark gray and light gray areas represent "small" and "medium" effect sizes, respectively (Cohen 1992). Estimates were obtained by running a minus intercept multilevel MCMCglmm model for each factor separately. Models included 1 categorical fixed factor and 3 random factors (Study ID, species ID, and phylogeny). All models were run using Fisher's Z transform of the correlation coefficient (Zr) and then converted back to r for presentation.

Table 1

Mean effect size estimates for each level of the 4 categorical moderator variables included in the dataset

Factor	Category	Effect sizes	Studies	Species	Mean r	LHPD	UHPD
Taxonomic class	Amphibian	14	6	4	0.051	−0.312	0.415
	Bird	75	22	16	−0.098	−0.303	0.095
	Mammal	8	6	3	−0.109	−0.448	0.289
	Reptile	21	4	2	−0.128	−0.457	0.303
Sex	Female	15	6	5	−0.174	−0.368	0.056
	Male	103	35	24	−0.070	−0.192	0.075
Stress measure	BCORT	31	18	12	−0.026	−0.200	0.165
	ECORT	27	7	5	−0.118	−0.365	0.102
	LSTRESS	50	15	14	−0.114	−0.274	0.093
	PCORT	10	3	2	0.012	−0.253	0.320
Secondary sexual trait	Coloration	58	18	15	−0.119	−0.252	0.055
	Opposite-sex preference	6	4	1	**−0.368**	**−0.615**	**−0.010**
	Morphological traits	15	6	6	−0.183	−0.366	0.078
	Vocalization	39	12	8	0.105	−0.074	0.309

Estimates were obtained by running 4 minus intercept multilevel MCMCglmm models including one of the categorical fixed factors, and all 3 random factors (Study ID, species ID, and phylogeny). All models were run using Fisher's Z transform of the correlation coefficient (Zr) and then converted back to r for presentation. Values in bold are significant at $P = 0.028$. Mean r, mean posterior estimate.

DISCUSSION

Our meta-analysis did not detect a significant relationship between physiological indices of stress and the expression of sexually selected traits across 26 vertebrate species from 4 taxonomic classes. Furthermore, we failed to detect effects of stress in any of the 4 vertebrate orders, in either sex, or when controlling for the measure of stress employed. However, we did detect a significant effect of the type of sexually selected trait, such that stress had significant detrimental effect on opposite-sex preferences, but not on the expression of coloration or vocalization, or on trait size.

Opposite-sex preference is likely to reflect an aggregate response to the development of one or more sexually selected signals and the true "attractiveness" of an individual to the opposite sex (rather than relying on our judgments of the attractiveness of trait expression). Although our results suggest that members of the opposite sex attend to cues of stress, we do not know which traits are used in their assessment. It is possible, for example, that there are behavioral traits in addition to the morphological traits that we have included here which provide cues to physiological status (Roberts et al. 2007). In addition, it is possible that effects of stress on coloration or vocalization are more nuanced and complex than our analysis was able to detect.

For instance, effects of stress on coloration may be dependent on the nature of the coloration (e.g., melanic vs. carotenoid), the context (e.g., breeding season vs. moult), and species ecology (e.g., the mating system). Melanic coloration, for example, can provide insight into links between the stress response and sexual signaling as the melanocortins that control the expression of pheomelanic coloration (Ducrest et al. 2008) also influence sensitivity to stressors (Ducrest et al. 2008; Roulin and Ducrest 2011). There were, however, only a small number of studies in which melanic coloration was measured (8 effect sizes from 3 studies, see Supplementary Table S3) meaning that it was not possible to test effects of stress on these separately. A greater number of studies measured carotenoid coloration ($n = 14$), and in a high proportion of those of avian species (21 of 27 effect sizes from 9 studies, see Supplementary Table S3), stress had detrimental effects on carotenoid coloration. This coloration is dependent on antioxidants acquired in the diet (McGraw 2006), which can be diverted away from secondary sexual traits and into reduction of oxidative damage under stress (Fitze et al. 2009). Although this may explain the pattern of stress-induced color reduction in birds (but see Collins et al. 2008), there was some evidence that chronically elevated GCs enhanced ventral coloration in males of the common lizard (Cote et al. 2010). This discrepancy could stem from differences in species' ecology, meaning that it is adaptive for males of some species (e.g., the common lizard) to make a "terminal investment" in mating under stress, perhaps due to reduced chances of survival (Pryke et al. 2007; Fitze et al. 2009; Bonier et al. 2009; Huyghe et al. 2009). In biparental mating systems, such as those of many bird species, the optimal solution to the allocation of energy under stress may be away from mating effort and into, for example, parental investment. It may not be possible, then, to detect any effects of stress on coloration until a sufficient number of studies across species and coloration type are available.

We did not find an effect of stress on vocalizations. More than a third of these effect sizes were measured in anurans during the breeding season (14 of 37 effect sizes). A number of studies have shown GCs to be elevated across the breeding season, with those individuals who vocalize the most showing the highest levels

(Gladbach et al. 2010; Goymann and Wingfield 2004). At threshold GC levels, however, males change their strategy and stop calling, likely on reaching a negative energy balance (Emerson 2001). As this threshold depends on intrinsic (e.g., condition) and extrinsic (e.g., rainfall, chorus density) factors, despite the organizing role of GCs on calling strategy within individuals, consistent effects may not be easily detected across individuals and studies (Emerson 2001). The majority of the remainder of effect sizes categorized as "vocalizations" were for effects of stress on dimensions of birdsong. There is reason to predict that birdsong is linked to dimensions of the stress response as the brain centers responsible for song develop early in life, during which time conditions also determine adult stress resistance (Buchanan et al. 2004; Pfaff et al. 2007; Muller et al. 2010). Both may stem from a common phenotype (Spencer and MacDougall-Shackleton 2011), rather than song responding to fluctuations in adult stress. Failure to find effects on vocalization, then, may stem from the fact that relationships between stress and vocalization are nonlinear and context dependent and that effects on birdsong and amphibian vocalization may be functionally different.

Potential roles of stress on the expression of sexually selected traits have included indirect effects of GCs via the immune system (Møller 1995; Buchanan 2000), body condition (Husak and Moore 2008), or testosterone (Buchanan 2000; Roberts et al. 2007; Husak and Moore 2008), or via sexual selection on the physiological stress response itself. In the former, the effects of stress on sexual traits would likely be difficult to detect without measuring, and controlling for, its effects on testosterone, immune function, and body condition. In the latter, sexual signals would provide cues to individual differences in dimensions of the stress response such as stress reactivity or the efficiency of negative feedback (Pfaff et al. 2007; Roberts et al. 2007; Husak and Moore 2008; Bortolotti et al. 2009; Schmidt et al. 2012). While a number of studies included in our analyses reported various indices of immune function, testosterone, and body condition, there were insufficient numbers to test for their roles in our model. A promising avenue for future research is analysis of individual differences in the stress response, which may provide the necessary conditions for the evolution of condition dependent traits (e.g. Moore and Hopkins 2009). Individual differences in stress reactivity and the efficiency of negative feedback, for example, are heritable (Rowe and Houle 1996; Evans et al. 2005; Korte et al. 2005; Stöwe et al. 2010), and production and effects of GCs are related to measures of genetic quality (Olsson et al. 2005) and fitness (Bonier et al. 2009). An efficient stress response is likely to be comprised of low baseline GCs, moderate elevation, and rapid negative feedback once the stressor has passed (Olsson et al. 2005; de Kloet et al. 2008). Although peak GC response to a standardized stressor was not significantly related to expression of secondary sexual traits in our analyses (although the sample size was small; $n = 9$ effect sizes, see Supplementary Table S3), an inverse relationship between a sexual signal and sensitivity of negative feedback (Schmidt et al. 2012) and a finding (excluded from our analyses due to lack of statistical information) that female zebra finches preferred males from lines bred for low peak GC response (Roberts et al. 2007) further support this as an important future research direction.

It is, however, extremely difficult to measure these dimensions of the stress response, particularly in free-living individuals. Regulation of GCs in response to predictable seasonal challenges such as the moult (Husak and Moore 2008; Romero et al. 2005) or breeding (Kitaysky et al. 1999), for example, may have different

effects on the allocation of resources to sexual signaling than those due to unpredictable stressors (O'Reilly and Wingfield 2001). This demonstrates the need for multiple measures of the stress response, long-term stress, and stress history in future research. Although this is undoubtedly difficult, records of local weather conditions, season, resource availability, and population density, for example, could be controlled for in analyses. Repeated measures of stress provide a more ecologically valid assessment of the experience of stress (Bonier et al. 2009). More comprehensive measurements of the stress response, including duration, total GCs released in response to an ecologically valid, standardized stressor, and the efficiency of negative feedback (Romero 2004) provide a set of dimensions of the stress response with which to compare individuals. In addition, the concentration and distribution of GC receptors may be more meaningful measures of individual differences in stress reactivity in terms of effects on morphological and behavioral traits than GC production itself (Schmidt et al. 2012).

Our analysis controlled for phylogenetic relatedness among species. The amount of variance in effect size explained by phylogeny was very small. This could be for several reasons. First, it may be that the relationship between stress and secondary sexual trait expression is highly evolutionarily labile, so that phylogenetic effects are important only for very closely related species. This may be especially likely for those studies concerning male vocalization, as behavioral traits such as these may evolve particularly rapidly (Blomberg et al. 2003), and is frequently seen in meta-analyses concerning behavioral traits (e.g., Santos et al. 2011; Dougherty and Shuker 2015). Alternatively, this could be an artifact of the fact that the average phylogenetic distance between species in our tree is relatively large (Björklund 1997). With such a tree the power to detect a phylogenetic signal is reduced, especially if there is substantial variation across species in factors (such as physiology or behavior) that may affect the relationship we are investigating.

In conclusion, stress was not associated with the expression of sexually selected traits in our sample. The results therefore challenge any notion of a common stress-signaling function for sexual signals. It was, however, associated with the strength of preference for the opposite sex, suggesting that stress is relevant to mating decisions but that our analysis was not able to detect the specific traits through which it is signaled. This means that stress may be important for mate choice, but it does not provide a simple explanation for the role of any one sexual display or signal. This discrepancy therefore needs resolving. Our findings add to the body of work which seeks to identify how stress can moderate the expression of physical and behavioral traits more generally (e.g., Lupien et al. 2009; Buchanan et al. 2013). We argue that in order to advance our understanding of roles of stress in sexual selection, we need to develop a model which incorporates the nuanced effects of species ecology, trait type, ecological context, and the complex nature of the physiological stress response.

SUPPLEMENTARY MATERIAL

Supplementary material can be found at http://www.beheco. oxfordjournals.org/

FUNDING

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