


**Stress and sexual traits: why are there no clear relationships? A comment on Moore et al.**

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The last 20 years have seen considerable interest in the physiological links between sexual signaling and the maintenance of essential homeostasis (Folstad and Karter 1992; von Schantz et al. 1999; Buchanan 2000; Roberts et al. 2004; Garratt and Brooks 2012). Broadly speaking, however, Moore et al. (2016) find little evidence for any such links. We suggest that this is perhaps not at all surprising, for reasons outlined below.

Conceptually, it seems highly unlikely that a simple relationship exists between physiological stress levels and sexual trait expression. Studies quantifying physiological stress assess the ability of an animal to maintain homeostasis, while exposed to environmental challenges. Throughout, Moore et al. (2016) refer to the effects of stress, but they are actually assessing the response to external stressors (Romero 2004). This nuance is crucial, because an animal with high levels of, for example, glucocorticoids is not necessarily “more stressed,” but rather is meeting the demands of a transient challenge. Consequently, it may not be required to compromise trait expression. This rationale may underlie the inconsistencies in the literature relating stress indices to trait expression, highlighted by Moore et al. (2016).

The relevant mechanisms for any direct stress-trait trade-off are therefore entirely unclear. In the original immunocompetence handicap hypothesis, Folstad and Karter (1992) suggested that glucocorticoid hormones, which can be immunosuppressive when chronically elevated (Sapolsky et al. 2000), might play a role in mediating the “honesty” of signal production. But the relationship is not simple. Androgens, which are more directly related to sexual trait expression, compete with glucocorticoids for binding proteins that regulate the availability of steroid hormones to target tissues (Breuner and Orchinik 2002). The complex interplay between androgens, glucocorticoids, immune function, and secondary sexual trait expression makes a simple association between physiological stress and sexual traits even more unlikely. Without well-designed manipulative experiments, it seems unlikely that we can resolve the causal relationships.

Timing is also crucial. In order for the physiological stress response to be linked directly to the expression of secondary sexual traits, it should be quantified during the time when the signal is being produced, but in many studies, this is not the case. Alternatively, if this is not the case then the stress response should be highly repeatable over time, within individuals. Although a number of studies report significant repeatability of the glucocorticoid stress response (Wada et al. 2008; Cockrem et al. 2009; Careau et al. 2014), the moderate repeatability values reported suggest that the plasticity of the glucocorticoid stress response is hard to characterize at the individual level.

One final point, surely raised with all meta-analyses, is the issue of comparability of studies. Stress indices indicate changes in physiological condition over different time frames, creating undesirable noise in any analysis. Although there has been tremendous growth in interest, we would suggest that the sample of relevant, comparable studies is still quite small. We therefore laud Moore et al.’s (2016) attempt to quantify the broad patterns in the literature to date and anticipate exciting avenues for future work in this area.

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