Correlated evolution of female and male testosterone—internal constraints or external determinants? A response to comments on Goymann and Wingfield

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We very much appreciate the thoughtful comments of our colleagues Buchanan and Fanson (2014), Garamszegi (2014), Groothuis (2014), and Ketterson (2014) on our study on male-to-female testosterone in birds (Goymann and Wingfield 2014). Buchanan and Fanson (2014) and Groothuis (2014) highlight the importance of recognizing that different selection pressures could act on testosterone levels in females and males. Further, Groothuis (2014) elaborates on an important conceptual issue: selection acts on phenotypes and not on hormone levels. If hormone levels are just one out of many potential mechanisms that are shaping phenotypes, it may be hard (or even impossible) to disentangle the effect of one of these mechanisms.

Ketterson (2014) disagrees with our perspective on hormone manipulation because “the difference between pharmacological and physiological can be informative rather than problematic.” This may be true in some cases, but we think that we as experimenters need to be cautious in selecting the dose and agent. All hormone implants mask the daily pattern of hormone release. If a trait depends on the daily pattern, then hormone implants cannot effectively manipulate the trait in question. But even if the daily pattern is not crucial for the expression of a trait, implants often fail to elevate hormone levels in a naturalistic way. Most implants may release hormones within or close to the physiological range 1 or 2 weeks after the implantation. But for a few days after the implantation, hormone titer can be much higher and far from the physiological range (see Fusani 2008). Because we do not really know the effects that such short-term peak elevations may have in the longer term, we remain skeptical that such high levels may be informative in ecological or evolutionary contexts (see also Groothuis, 2014, who extends this critique to purely mechanistic studies).

Garamszegi (2014) offers a very stimulating extension of our analysis, which also exemplifies the importance of the assumptions that we as researchers have when we conduct such analyses. In our comparative study, we used untransformed hormone data for the male-to-female regression (Goymann and Wingfield 2014, Fig. 1; normality was not an issue). But we agree with the scaling argument that a logarithmic comparison of absolute male and female levels could be more appropriate and that the regression coefficient is the more relevant factor to consider when looking at the strength of a relationship. Thus, the relationship between male and female testosterone may indeed be stronger than we thought. The follow-up analysis of Garamszegi (2014)—in which male testosterone was used as a covariate—is valid, assuming that testosterone levels in females and males are connected because of correlated evolution due to (internal) constraints. Garamszegi (2014) found that female testosterone concentrations are higher in colonial than in noncolonial species when controlling for male testosterone. Although this kind of analysis absolutely makes sense from an evolutionary and statistical point of view, we wonder how this scenario could work taking a physiological perspective: Absolute levels of testosterone did not significantly differ between colonial and noncolonial females. Thus, in order to generate a physiological signal with regard to coloniality, the female physiology needs to “know” something about the respective male physiology. A possible explanation could be that other parameters in the hormonal transduction cascade are modified differently in females of colonial than in those of noncolonial species, so that colonial females can respond to similar levels of testosterone with a higher sensitivity.

But correlated evolution of testosterone in which one sex constrains the possibility to express testosterone in the other sex is only one possible explanation for a correlation of female and male testosterone. Alternatively, a third (and potentially external) factor could influence both female and male testosterone levels in a similar manner. In this scenario, females and males would not constrain each other’s circulating testosterone concentrations, but their testosterone levels would both respond to a third factor. If the latter is the case, it would not be appropriate to use male testosterone as a covariate when investigating female testosterone concentrations, because female testosterone is independent of male testosterone, even though they are correlated.

What third factor that is shared by females and males could lead to the impression that female and male circulating testosterone levels are constraining each other? In males, the length of the breeding season is a major determinant of maximum testosterone concentrations (Goymann et al. 2004; Garamszegi et al. 2008). If this would be similar in females, then the correlation of male and female testosterone may not be caused by correlated evolution but by an external parameter related to the breeding phenology of both sexes in each species investigated. Indeed, the length of the breeding season (data estimated as egg-laying period, mainly extracted from Garamszegi et al. 2008) is significantly related to maximum testosterone in female birds (MCMCglmm model: \(\beta = -0.051\), CI_{lower/upper} = −0.099/−0.003; \(\log_{e}(\text{egg-laying period} < 0.039; \text{DIC} = 53.1\)). Similar to males, females of species with shorter breeding seasons express higher levels of maximum testosterone than females of species with longer breeding seasons. Thus, the length of the breeding season could represent an external determinant of maximum testosterone concentrations in females and males, leading to correlation between females and males without the necessity to assume causation of correlated evolution due to one sex constraining the testosterone levels of the other sex.
If we now run a similar MCMCglmm model following Garamszegi (2014) but replacing male testosterone with egg-laying period as a covariate of coloniality, we again find the significant effect of the egg-laying period, but not of coloniality (egg-laying period: $\beta = -0.052, CI_{lower/upper} = -0.097/-0.005; P_{egg-laying\ period} = 0.028$; coloniality: colonial testosterone mean = 0.896 ng/mL, CI_{lower/upper} = 0.422/1.378 ng/mL, noncolonial testosterone mean = 0.619 ng/mL, CI_{lower/upper} = 0.291/0.988 ng/mL; $P_{coloniality} = 0.121$; DIC = 52.2). Thus, the breeding season effect remains, but coloniality does not explain a significant proportion of the variance. In our main article, we wondered about the particularly high levels of testosterone in female albatrosses, penguins, and Lapland longspurs. It now turns out that a very short breeding period during which females lay their eggs is common to these species and that this short breeding period is associated with high levels of testosterone.

In conclusion, assuming a weak correlation between male and female testosterone levels (Goymann and Wingfield 2014), we did not consider alternative explanations for correlated evolution of testosterone due to one sex constraining the expression of testosterone in the other sex. In his thoughtful reanalysis of female testosterone data, Garamszegi (2014) made us think about the correlation of testosterone in males and females in greater depth. For the reasons given in our main article (supported by Groothuis 2014), we still consider it unlikely that one sex constrains the expression of circulating testosterone in the other sex. In our response, we offer an alternative explanation as to why female and male testosterone concentrations may be correlated. It is up to future research to determine whether the existing correlation is due to constraints or due to a common response to environmental factors that shape testosterone profiles in females and males in a similar manner. The length of the breeding season could indeed represent such a common factor, in this case leading to similar selection pressures in male and female birds.

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