Causality and sex roles: prejudice against patterns? A reply to Ah-King

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Ah-King [1] presents three arguments as to why being male or female (by definition, producing small or large gametes) does not underlie other sex differences in reproductive behaviour ('sex roles'). These are: that correlation does not equal causation; there is too much variation among and within species to talk about 'sex roles'; and that 'stochastic demographic' (SD) models by Gowaty and Hubbell [2,3] have more explanatory power than Schärer et al. [4] implied in a recent Opinion piece.

We suspect that explaining sex roles generates fiercer debate than, say, asking whether radiation can cause cancer, not only because experimentation is harder, but because of concerns that researchers are prejudiced by social values that prevent (or delay) discoveries. For example, in the 1970s it took many researchers by surprise that female birds are often unfaithful to their mates. However, it also took surprisingly long for researchers like Marie Curie and her contemporaries to realise that radioactivity could be dangerous [5]. Failure to see what is obvious in hindsight is not always attributable to societal prejudices.

In our view, however, there appear to be prejudices at play when claiming [1–3] that mainstream models of sex roles that assign explanatory power to anisogamy are impossible to reconcile with cases of an allegedly 'unexpected' nature. This requires a wilful misunderstanding of recent theoretical models. Our work [6], for example, confirmed [7,8] that anisogamy's effect does not follow from original verbal arguments [9]. We showed that understanding anisogamy's role requires more sophisticated models where sperm competition, by influencing the certainty of parentage, leads to greater female care. We also explicitly noted that which sex provides more care is sensitive to mortality (and other) differences between the sexes [6]. We find it disheartening that Ah-King selectively chose one quote from near the beginning of a sequence of logical arguments, which distorts our findings as if we had disproven the role of anisogamy. This reflects either a failure to understand the models or, more worryingly, a decision to place rhetoric before facts. It is blantly false to claim that existing 'mainstream' models are incapable of explaining 'exceptions to the rule', e.g., what happens when parenting itself becomes sexually selected [10,11]. A good theory will explain what has happened when an unusual case is encountered, but it should also explain why such cases are uncommon.

One can legitimately refer to several factors all causing the same outcome. The scientific method is such that experimental manipulation of a factor that significantly alters a measured outcome allows us to refer to this factor as having a causal effect. Of course, the depth of the explanation can vary (i.e., how much of the causal ‘pathway’ is revealed). For example, mutations can be said to cause cancer, but so can radiation (by increasing mutation rates). If one wants to focus on ‘exceptions to the rule’, one can produce models [2,3] that are framed to avoid any explicit link between anisogamy ('radiation') and factors such as encounter probability with mates or survival probability ('mutations'). The task of a scientist is, however, to explain broad patterns, and not merely celebrate a model's ability to fit single cases. Proponents of SD models prefer to quote single cases of 'unexpected' sex roles as evidence against anisogamy models instead of presenting their own falsifiable prediction: no broad relationship between sex and behaviour. This may be understandable, because if a model predicts the absence of an intriguing pattern, it might divert one's attention away from testing for it in data.

Yet it remains a testable prediction, and it reveals the central weakness of SD models. Although they identify important factors that affect choosiness (and, incidentally, competition for mates), such as mating latency and encounter probability with potential mates, these parameters are deliberately not linked to sex. If the models remain truly non-sex-specific, this implies that sex differences – which these models do not preclude [1] – will arise at random due to extrinsic ecological processes impacting one sex more strongly than the other. So we should find that in 50% of species (or populations) that show detectable sex differences, males are choosier/less competitive/provide more post-mating parental care than females. Yet this is not the case. We now undeniably know enough about the genetic breeding system, degree of competitiveness, parental care asymmetries and (perhaps to a lesser degree) choosiness of each sex to be confident in stating that despite exciting and well-studied [4] diversity, there are systematic differences in male and female behaviour captured by the umbrella term 'sex roles' that needs to be explained. Neither do we see differentated roles in species with isogamy despite different mating types. Following the logic of SD arguments, by chance ecological/social variables should just as readily become associated with + strains or – strains and lead to divergence between them.

Unfortunately, observational correlations (radiation–cancer incidence), unlike correlations derived from experiments, do not establish causal links, but they should alert us to their possibility. In some fields (e.g., astronomy, geology and parts of evolutionary biology) it is not possible to conduct experiments, which makes it hard to determine

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casualties. Fortunately, meiosis in species with genetic sex determination provides a ‘natural experiment’ that is close to a designed experiment with subjects randomly assigned to treatments. As though a coin were flipped, an individual develops as a male or a female depending on whether an egg fuses with an X- or Y-bearing sperm. Can we now predict its sexual behaviour based on knowing how the coin landed? If a biologist can, on average, win money by participating in this bet, then there is something about anisogamy (used to define the two sexes in all taxa) that is important, and researchers should seek to understand why this is the case. SD models [2,3] essentially claim that one cannot win money betting on the meiosis experiment.

Theories are designed to explain correlations. In astronomy, if models of star formation that are based on established theoretical physics succeed in predicting the observed distributions of spectral properties of stars, science can be said to have proceeded beyond merely reporting correlations and distributions. Given the imperfect, but statistically significant, correlation between reproductive roles and whether individuals produce sperm or eggs, and the plausibility (established through mathematical models, and biophysical principles) of causal routes linking gamete size to various life history trade-offs (including the likely time spent in various breeding activities [4,12]), we can only reiterate that anisogamy causes sex roles.

Finally, there is nothing circular about defining ‘reversed sex roles’ based on rarity. Context-dependence of the term ‘reversed’ might, in hindsight, have made it preferable simply to categorize cases as ‘females more competitive than males’ or vice versa. Despite its historical baggage, the ‘reversed’ label is not circular when applied as intended, across rather than within taxa. Reversal in this context describes rarity rather than pathology. Sex role terminology boils down to a simple factual statement: it is more common to find that the producers of smaller rather than larger gametes compete more intensely for mates. This is a falsifiable claim that, to the best of our knowledge, is true.

Note added in proof
The original letter by Ah-King contained material that the author chose to remove when correcting the proofs. Our reply was written assuming this content will be present at publication. Our claims regarding selective quoting ought to be interpreted with this in mind.

References