primarily through the display of signs of youth (where thinness is a marker for youth). Youth is a major determinant of reproductive potential in females, whereas men compete among themselves through the display of physical dominance and prowess, which is enhanced through the size and muscularity of the upper body. The increase in the rates of eating disorders in the past several decades in Western and Westernised countries is explained by the 'mismatch' between the human psychological systems for mate attraction and retention and for competition with same-sex rivals that evolved in small-scale societies on the one hand, and the novel realities of the modern urban Western environment on the other.

The SCH has been supported by findings of studies of disordered eating in non-clinical populations (female undergraduates) both in the USA[3] and the UK.[4] Further support for predictions made by the SCH came from another US study of disordered eating in a non-clinical population of male and female homosexual and heterosexual participants.[5]

The consistency of the clinical features of eating disorders across different high-income countries, as well as their persistence (or even rising levels), argues strongly against the arbitrary cultural standard view dominant in eating disorder circles and in favour of a model in which these disorders are the outcome of a gene–environment interaction.

1 Murray SB, Griffiths S, Mond JM. Evolving eating disorder psychopathology: conceptualising muscularity-oriented disordered eating. *Br J Psychiatry* 2016; **208**: 414–5.

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5 Li NP, Smith AR, Griskevicius V, Cason MJ, Bryan A. Intrasexual competition and eating restriction in heterosexual and homosexual individuals. *Evol Hum Behav* 2010; **31**: 365–72.

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Authors' reply: We thank Dr Abed for his comment on our recent editorial but dispute the implication that delineating the symptom profiles of male versus female eating disorder pathology neglects the aetiological precursors of eating disorders. On the contrary, careful delineation of the spectrum of disordered eating that occurs at the population level is essential in developing comprehensive aetiological frameworks. For many years, the male experience of disordered eating has been under-represented in empirical and clinical research. The purpose of our editorial[1] was to highlight both the male experience of eating disorder pathology, and the historical failure to recognise and index this pathology.

Although our piece was not intended to address considerations of aetiology, it is important to note that there is strikingly little evidence to support the sexual competition hypothesis as an aetiological framework for muscularity-oriented disordered eating, as proposed by Abed. Indeed, much of the work cited by Abed was conducted in female samples, with a consequent focus on the drive for thinness, and the study mentioned by Abed,[2] in which the sexual competition hypothesis was assessed in non-clinical male and female populations, is merely another example of male eating disorder pathology being assessed through the lens of female-oriented measurement tools. As such, the statement that the sexual competition hypothesis provides a cogent framework in accounting for the aetiology of muscularity-oriented disordered eating goes beyond what the current evidence base permits. The propensity to extrapolate findings from thinness-oriented studies of eating disorder pathology to males is the very problem which our editorial aimed to highlight.

In our view, the assertion that evolution-instilled competition for mates provides a framework for understanding the aetiology of eating disorder pathology is problematic for at least two reasons. First, eating disorders are associated with a substantially elevated risk of mortality,[3] a distinct evolutionary disadvantage. Second, clinical eating disorder pathology is frequently characterised by a reduction in libido and active avoidance of sexual relationships,[4] observations that are at odds with the purported goal of dietary restraint. Additionally, this framework offers little insight into how to distinguish those who develop clinical eating disorders from the far greater numbers of individuals who engage in purposeful dietary restraint with the goal of modifying their shape or weight, many of whom presumably desire a mate. No doubt a more comprehensive aetiological framework for eating disorder pathology needs to take better account of the reciprocal influences of a broad range of factors, but to say that 'these disorders are the outcome of a gene–environment interaction' is a diffuse and perhaps oversimplified statement which is likely more harmful than helpful.

1 Murray SB, Griffiths S, Mond JM. Evolving eating disorder psychopathology: conceptualising muscularity-oriented disordered eating. *Br J Psychiatry* 2016; **208**: 414–5.

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