Response to Comment on "Parasites as a Viability Cost of Sexual Selection in Natural Populations of Mammals"

We are grateful to Brei and Fish (*1*) for highlighting a number of issues relating to our respective articles (*2*, *3*). First, Brei and Fish argue that the Moore and Wilson study (*2*) ignored the importance of sex differences in home range size as a proximate cause of sex differences in exposure to parasites and as a potential mechanism for generating sex-biased parasitism (SBP) in wild mammals. Parasite load is a complex function of both the rate of exposure to parasites and physiological responses activated against parasites once exposed ("immunity"), and we certainly did not exclude the possibility that sex differences in behavior (and hence exposure) could generate the patterns we observed in wild mammal populations. Indeed, there is good evidence that sex differences in behavior can contribute to SBP for some host-parasite interactions in both humans (*4*) and nonhuman mammals (*5*).

We tested the Brei and Fish hypothesis that SBP was generated by sex differences in home range size by extracting home range size data from the literature (*6*, *7*). We found that, as observed in previous studies (*7*, *8*), across the mammal species in our data set, there was a positive relation between mean body mass and mean home range size (linear regression using the logged data: $F_{1,53} = 109.5$, $P < 0.0001$, $r^2 = 0.67$). However, there was no relation between mean home range size and mean parasite prevalence ($F_{1,54} = 0.42$, $P = 0.52$). More important, we found no significant relation between sex-biased home range size (calculated as the logarithmically transformed ratio of male range size to female range size) and SBP ($F_{1,26} = 0.180$, $P = 0.894$), even after controlling for sexual size dimorphism ($F_{1,25} = 0.070$, $P = 0.794$; Fig. 1A).

When we controlled for host phylogeny, using the independent-contrasts method, the relation between SBP and sex-biased home range size remained nonsignificant ($F_{1,25} = 0.010$, $P = 0.920$; Fig. 1B). It is unlikely that these results were a consequence of the analyses lacking sufficient power, because the relationship between SBP and sexual size dimorphism (SSD) remained highly significant, even though the number of species used was much smaller than that used in our original analysis (raw data: $F_{1,26} = 11.25$, $P = 0.0024$; independent contrasts: $F_{1,25} = 20.54$, $P = 0.0001$). Thus, the available evidence does not support the notion that SBP in mammals is generated by sex differences in home range size.

Second, Brei and Fish question whether the results obtained from nonhuman mammals can be extended to include humans, as suggested in the Perspective by Owens (*3*). In particular, they argue that the late onset of the sex difference in death rate in the United States from parasitic and infectious diseases (that is, after 25 years of age) is more consistent with sex differences in behavior than with sex differences in energetic or hormonal investment in growth. We disagree, and argue that the timing of the onset of the sex difference in mortality tells us little about when the underlying sex difference in susceptibility to infection first appears. Indeed, much like the costs of reproduction (*9*), we should not be surprised if the costs of immune deficiency become apparent only later in life, when the fitness consequences are much lower.

Brei and Fish also question the validity of using the World Health Organization (WHO) 1997 USA dataset to examine parasite-induced sex-biased mortality (SBM) in humans, in part because of the high incidence of deaths attributable to HIV infection. That data set was used

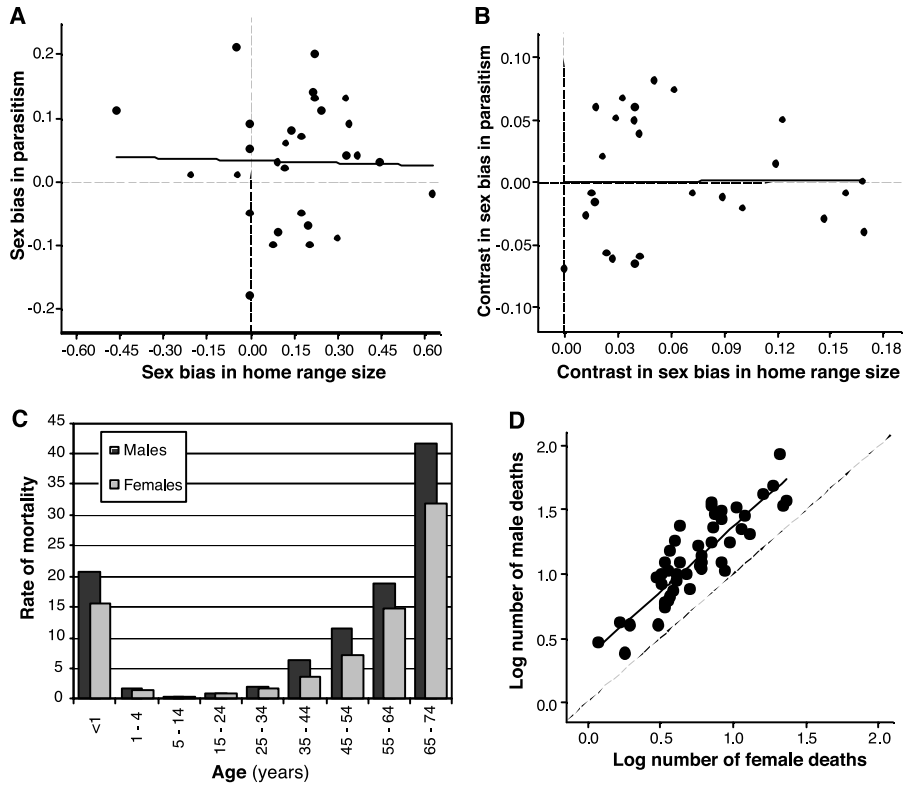

Fig. 1. SBP and mortality in wild mammals and humans. (**A**) Relation between sex bias in parasitism and sex bias in home range size in wild mammals. Each point represents a single species, and the line shows the least-squares regression (see main text for details). (**B**) Relation between the independent contrast scores for sex bias in parasitism and sex bias in home range size in wild mammals. Each point represents an independent contrast score generated using the program CAIC, and the line represents the least-squares regression, with the intercept forced through the origin. (**C**) Age-dependent variation in the rate of mortality (deaths per 100,000 population) attributed to non-HIV parasitic and infectious diseases in the United States in 1997 (*10*). (**D**) Relation between number of deaths caused by parasitic and infectious diseases in males and females. Data were $\log_{10}$-transformed. Each point represents data from a single country during the year 1996 (when available). The solid line represents the least-squares regression ($y = 0.3617 \pm 1.0080x$; $F_{1,49} = 156.6$, $P < 0.0001$, $r^2 = 0.76$). The dashed line shows the expected relationship (1:1) if males and females were equally likely to die from parasitic and infectious diseases.

purely for illustrative purposes, as a conservative test of the general principle of a greater impact of parasites on males than on females. We disagree with the view suggested by the Brei and Fish comment (*1*) that HIV is fundamentally different from other parasitic and infectious disease agents and should have been omitted from the analysis. However, when deaths due to HIV infection are excluded, mortality in the United States due to non-HIV parasitic and infectious diseases remains male-biased (Fig. 1C).

To determine the generality of this result, we conducted a preliminary analysis of the full WHO data set, which includes information on the causes of deaths in more than 50 countries. We found that, overall, males were more than twice as likely to die from parasitic and infectious diseases than females (ratio of the number of male deaths to the number of female deaths: $b = 2.51 \pm 0.14$; 95% confidence interval = 2.22 to 2.79; one-sample t test comparing b to unity: $t = 10.70$, d.f. = 50, $P < 0.001$; Fig. 1D). This was true even after HIV-related deaths were excluded from the analysis ($b = 1.85 \pm 0.17$; 95% CI = 1.51 to 2.19; $t = 5.13$, d.f. = 30, $P < 0.001$) (*10*). Thus, there is good evidence that parasite-induced SBM is not confined to wild mammal populations but is evident also in contemporary human populations. Indeed, data from studies on humans may provide a rich resource for understanding the mechanisms underlying SBP parasitism and mortality in other mammals.

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References and Notes

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10. Data were extracted from the WHO Web site (www3.who.int/whosis/) and included information on the causes of death in 51 countries for the year 1996 (when data were not available for 1996, the nearest year for which data were available was used). To exclude deaths attributed to HIV infections, we used data only from those countries for which HIV-related deaths were distinguished from deaths due to other parasitic and infectious diseases.

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