Parasites as a Viability Cost of Sexual Selection in Natural Populations of Mammals

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Sexual selection in mammals has resulted in the evolution of sexual size dimorphism (SSD), with males usually being the larger sex. Comparative analyses indicate that the evolution of SSD is associated with the evolution of male-biased mortality, suggesting a possible causal link between the two. Here, we use a comparative approach to investigate the possible role of parasites in generating this relation. We show that there is a robust association between male-biased parasitism and the degree of sexual selection, as measured by mating system (monogamous or polygynous) and by the degree of SSD. There is also a positive correlation, across taxa, between male-biased mortality and male-biased parasitism. These results are consistent with the hypothesis that parasites contribute to the observed association between SSD and male-biased mortality.

In mammals, male reproductive success is most strongly correlated with competitive ability, and this has resulted in the evolution of large body size and weaponry (*1*). As a consequence, polygynous mating systems in mammals are characterized by SSD, with males generally being larger than females (*1*, *2*). Previous comparative studies have indicated that there may be a viability cost associated with SSD, because there is a strong positive relation across taxa between SSD and male-biased mortality (*3*). Because parasites are an important source of mortality in wild mammal populations (*4*) and because there is some evidence that males tend to be more heavily parasitized than females (*5*–*7*), we conducted a series of comparative analyses aimed at testing the hypothesis that parasites play a role in mediating the observed relation between SSD and male-biased mortality. More generally, we sought to determine the role of sexual selection in generating sex-biased parasitism (SBP) in mammals (*7*).

A data set on sex-related incidence of parasitism was compiled using information extracted from the literature (*8*). For each host-parasite interaction, the extent of SBP was defined as the rate difference in parasite prevalence between male and female hosts (i.e., male prevalence minus female prevalence) (*9*). We then used a meta-analytic model to assess the average sex difference in the prevalence of infection (*10*). Overall, there was a small but statistically significant male bias in the parasitism of mammals, with the mean cumulative effect size being +0.022 (95% confidence interval, 0.011 to 0.031) (*8*). This result is consistent with two previous meta-analyses based on much smaller sample sizes (*5*, *6*). Although there was considerable variation in the extent of SBP within host orders [the heterogeneity statistic (*8*), $Q_T = 542$, d.f. = 355, $P < 0.0001$], in 8 out of the 10 orders examined males were on average more likely to be parasitized than females, and in four of these orders the difference from zero was statistically significant (Fig. 1A). Only one host order exhibited significant female-biased parasitism (Perissodactyla); but because this was represented by a single host species in our analysis (*Diceros bicornis*), this trend may not be typical of the order as a whole. There was also significant heterogeneity in SBP within parasite taxa ($Q_T = 567$, d.f. = 353, $P < 0.0001$). However, the mean prevalence of infection was male biased for all three parasite types examined (arthropods, helminths, and unicellular parasites), and the extent of the male bias was significantly different from zero for two of the three taxa (Fig. 1B).

Sexual selection and male-biased parasitism. Although these data suggest an overall male bias in the prevalence of infection, they do not shed any light on the factors generating variation in SBP within or between host orders. To address this issue and to examine the influence of sexual selection on the evolution of SBP, we reanalyzed these data incorporating species-specific information and using a comparative method based on independent contrasts (*8*, *11*). This allows us to control for the fact that different species may exhibit similar traits (e.g., the extent of SBP) because of their common ancestry rather than because of any evolutionary convergence (*8*). If sexual selection is important in generating SBP, we should observe an association between mating system and the extent of any sex bias (*12*). As predicted, we found that evolutionarily independent increases in the incidence of polygyny were associated with significant increases in the extent of male-biased parasitism [$z = -1.98$, $n = 11$ independent contrasts, one-tailed $P = 0.024$) (*8*)] (Fig. 2).

Mating system is a fairly crude description of the intensity of sexual selection acting on a species. However, it is widely accepted that sexual selection is generally the main driving force behind SSD in mammals (*1, 13*); hence, this should act as a more sensitive measure of the strength of sexual selection. Using each host species as an independent data point, we found a strong and statistically significant positive relation between SBP and SSD [$F_{1,92} = 10.92$, $P < 0.001$ (*8*)]. Generally, mammal species in which the male is the larger sex exhibited male-biased parasitism, and those species in which the female is the larger sex displayed female-biased parasitism (Fig. 3A). Using the independent contrasts method, we found evolutionarily independent increases in SBP to be associated with increases in SSD [$F_{1,70} = 15.87$, $P < 0.001$ (*8*)] (Fig. 3B). This relation was a consequence of a significant positive relation across taxa between SSD and parasite prevalence in males ($F_{1,70} = 7.73$, $P = 0.007$) and a nonsignificant relationship between SSD and parasite prevalence in females ($F_{1,70} = 0.34$, $P = 0.56$). Using current comparative methods, it is impossible to simultaneously control for both host and parasite phylogeny. However, it is possible to

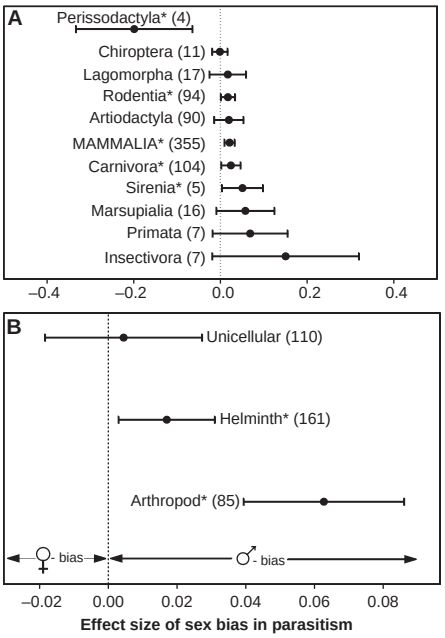

Fig. 1. Effect sizes and 95% confidence interval plots of sex bias in parasitism in relation to (**A**) mammalian host orders and (**B**) parasite taxa. An asterisk denotes statistical significance at the 5% level; number of studies is indicated in parentheses.

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determine whether the relation between SBP and SSD is contingent on the type of parasite considered. We found that increases in SBP across taxa were associated with increases in SSD for all three of the parasite taxa considered: arthropods ($F_{1,22} = 12.85$, $P < 0.001$), helminths ($F_{1,31} = 12.57$, $P < 0.001$), and unicellular parasites ($F_{1,42} = 4.27$, $P = 0.022$). Hence, our comparative analyses provide robust evidence that SBP in mammals is associated with the intensity of sexual selection, as measured by both the extent of SSD and by mating system.

SBP and mortality. The results of this analysis are consistent with the hypothesis that increased parasitism is a cost of sexual selection in mammals (*12*, *14*, *15*). A previous comparative study on mammals showed that the degree of male bias in adult mortality was significantly higher in nonmonogamous than monogamous taxa and was positively correlated with the degree of SSD across taxa (*3*). Thus, the ultimate cost of sexual selection and SSD for males appears to be enhanced adult male mortality. But is this cost mediated by parasites? To investigate this further, we analyzed the relation between SBP and sex-biased mortality (SBM) (*8*). Using the raw species data, we found that there was a significant positive relation between SBP and SBM ($F_{1,27} = 6.70$, $P = 0.008$) (Fig. 4A). More important, using the comparative method we found that evolutionarily independent increases in SBP were associated with increases in SBM ($F_{1,25} = 8.04$, $P = 0.004$) (Fig. 4B). Both of these relations remained significant after SSD had been controlled for (raw data, $F_{1,22} = 6.23$ and $P = 0.010$; independent contrasts, $F_{1,22} = 3.98$ and $P = 0.029$). Thus, the available evidence is consistent with the notion that sexual selection leads to enhanced risk of parasitism and elevated mortality in males due, in part at least, to the negative impact of parasites [e.g. parasite-induced host mortality or parasite-enhanced predation risk (*4*)].

Though our analyses implicate sexual selection as the ultimate driving force behind SBP and suggest that factors associated with relative body mass, growth rate, or size-dependent resource allocation may be important, they cannot identify the proximate mechanisms underlying these trends. However, they may provide us with some insight into which processes may be important. There are two major classes of hypotheses for the proximate control of SBP. One class invokes androgenic hormones, such as testosterone, and posits that though these are required for the full expression of sexually selected traits, they have immunodepressive side effects (*16*, *17*), and males consequently cannot achieve large body size, for example, without suffering a relative depression of immune function and increased susceptibility to parasites. Under this scenario, males are envisaged to suffer a sex-specific handicap associated with the production of immunodepressive androgens. Another class of hypotheses suggests that it is body size per se that is important in generating SBP, either because the larger sex is exposed more to infection (e.g., because it offers a larger target for parasites and their vectors) (*18*, *19*) or because some limiting resource (e.g., energy) constrains both somatic growth and immune function such that, all else being equal, species in which the males invest in enhanced growth do so at the expense of their immune systems, making them more susceptible to parasites (*20*, *21*). Under this scenario, males are not "special"; rather, there is a parasitism cost associated with being relatively large or growing to a relatively large size. If this cost explains SBP, then we might expect the prevalence of parasitism to be greater in larger species than smaller species. As predicted, this trend was observed in both the raw data ($F_{1,88} = 10.83$, $P < 0.001$) and in the contrast scores ($F_{1,66} = 3.70$, $P = 0.029$). We would also predict that females should suffer higher parasitism rates than males in those species in which females are the larger sex (i.e., in those species exhibiting "reversed," as opposed to "conventional," SSD). Such a relation is observed in the raw species data set (Fig. 3A). Moreover, levels of SBP were more female biased in the 10 taxa displaying reversed SSD than in the nearest taxon exhibiting conventional SSD (paired t test: $t = -4.03$, d.f. = 10, $P = 0.002$) (Fig. 5). Thus, the qualitative relation between SBP and SSD was independent of whether the male or the female was the larger sex in size-dimorphic species. We can also determine whether the quantitative relation between size dimorphism and SBP is dependent on whether the male or the female is the larger sex. Using both the raw species data ($t = 0.69$, d.f. = 87, $P = 0.49$) and the contrast scores ($t = -0.68$, d.f. = 69, $P = 0.50$), we found that for a given degree of (absolute) size dimorphism, the degree of sex bias in parasitism rate (as measured by the slope of the SBP – |SSD| regression line) was similar in taxa exhibiting reversed and conventional SSD (*8*). Thus, it appears that differences in body mass, growth rate, or resource allocation may be sufficient to explain the degree of sex bias in parasitism in size-dimorphic species; there is no evidence from these analyses that males suffer an additional sex-specific handicap [though this does not, in itself, exclude the possibility of testosterone-mediated sex differences in immune function (*22*)].

Correlation and causation. Correlations cannot reveal the direction of causation, and it is possible that enhanced sexual selection is a consequence rather than a cause of SBP. Hamilton and Zuk (*23*) proposed that in

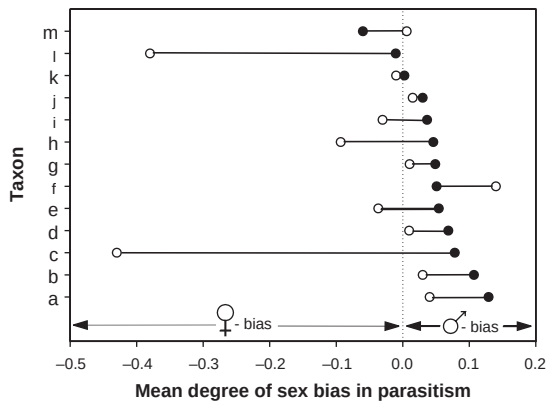

Fig. 2. Differential parasitism in polygynous and monogamous mammals. Contrasts in SBP of polygynous taxa (closed circles) and the most closely related, monogamous sister taxa for which data were available (open circles). Estimation of mean degree of SBP is described in (*32*), and the dendrogram of the sister taxa used in the analysis is shown in fig. S1.

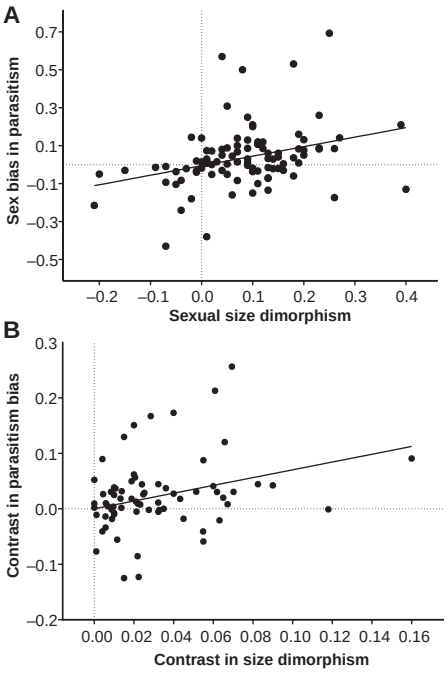

Fig. 3. Sex bias in parasitism rate (male prevalence–female prevalence) in relation to SSD (logarithm of male mass/female mass). (**A**) Plot of raw data. (**B**) Plot of independent contrast scores. The lines show the least-squares regression lines; in (B), the intercept is forced through the origin (*33*).

those species that are consistently exposed to high levels of parasitism, the intensity of sexual selection would be greater, as males advertise to females their levels of disease resistance. However, the Hamilton-Zuk hypothesis makes no specific predictions regarding sex differences in parasitism rates across species (though it could be argued that, if anything, it predicts female-biased parasitism, because selection for parasite resistance is envisaged to be stronger on males than females). More importantly, if the extent of any sex bias in parasitism influences the strength of sexual selection, then we would expect to observe some evidence for sex role reversal in those species exhibiting reversed SSD and SBP. Yet, in those species that exhibit reversed SSD (mostly Chiroptera, Rodentia, and Lagomorpha in our data set), large female body size is related to maternal care or intrasexual competition for nest sites not competition for mates (*24*–*26*). Indeed, in an extensive review of reversed SSD in mammals it was concluded that "the phenomenon may have evolved in a variety of ways, but it is rarely, if ever, the result of sexual selection acting upon the female sex" (*24*).

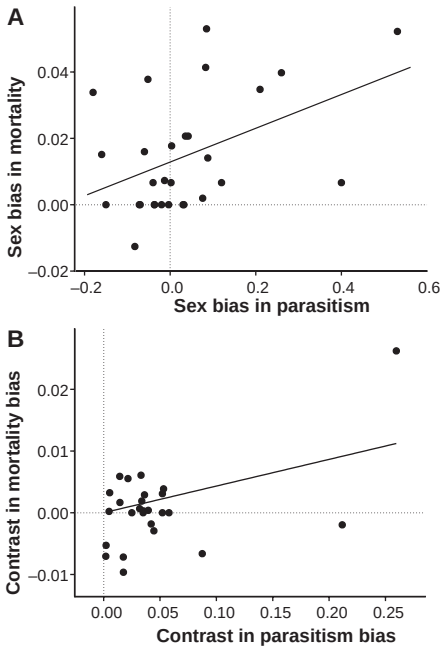

Fig. 4. Sex bias in parasitism rate in relation to sex bias in mortality rate (logarithm of female life expectancy/male life expectancy). (**A**) Plot of the raw data. (**B**) Plot of independent contrast scores. The lines show the least-squares regression lines; in (B), the intercept forced through the origin (*33*).

Lastly, we have argued that parasites may mediate, or at least contribute to, the relation between SSD and sex-biased mortality (*3*). By investing in growth and reproduction, males of sexually dimorphic species may expose themselves to a greater risk of parasitism and may pay the ultimate cost of reduced survival. An alternative interpretation of these results is that the degree of SSD reflects the strength of intrasexual competition and aggression and that the higher mortality generally observed in males is a consequence of their greater risk of injury and susceptibility to mortality factors, including parasitism. Under this scenario, differential parasitism of males contributes to male-biased mortality but is secondary to the lethal effects of male-male combat. Although it is impossible, at this stage, to disentangle the relative importance of parasites and other mortality agents in generating sex-biased mortality, the fact that SBP explained variation in SBM even after SSD had been controlled for suggests that differential parasitism influences mortality patterns independently of any effects due to intrasexual combat. Moreover, if this hypothesis explains our results, then we would predict that in species exhibiting reversed SSD, competition between females would be more injurious than it is in males, making them more susceptible to parasites and pathogens. Yet, it appears that even in species exhibiting reversed SSD, males generally exhibit higher levels of intrasexual aggression than females (*24*).

Testing hypotheses. Empirical support for the idea that sex differences in susceptibility to parasitism might yield sex differences in mortality is provided by recent field studies on Soay sheep (*Ovis aries*) on St. Kilda, Scotland (*27*). Soays exhibit a relatively high degree of SSD (*28*), and the average mortality rate of males is approximately double that of females (*29*). Males have significantly higher rates of infection with gastrointestinal nematodes (*30*), and crucially experimental removal of these gut parasites from yearling males and females completely reverses the male-biased mortality observed in nontreated animals (*31*), strongly implicating male-biased parasitism as a source of male-biased mortality in this population (*27*). This system provides an ideal opportunity to tease apart the proximate mechanisms underlying the link between SSD, SBP, and SBM by conducting experimental manipulations of parasite and hormone levels and by applying appropriate epidemiological models. In tandem with similar analyses across a range of species exhibiting both conventional and reversed SSD, we should be in a stronger position to critically appraise competing hypotheses for the mechanistic basis for SBP in mammals.

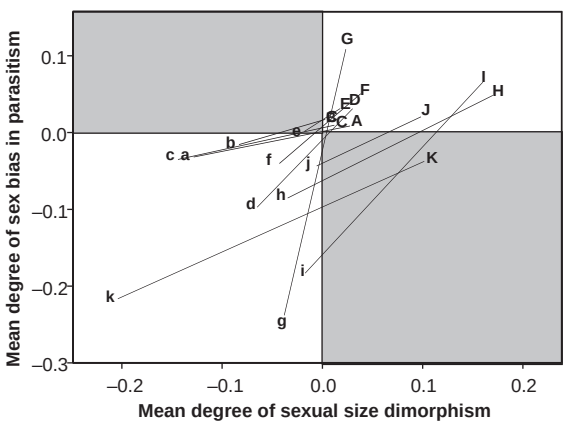

Fig. 5. Contrasts in SBP of taxa exhibiting reversed size dimorphism against the most closely related taxon displaying conventional or no SSD. Lowercase letters represent taxa in which females are larger than the males (reversed SSD); uppercase letters represent the most closely related taxon in which males are larger than the females (conventional SSD) or exhibit no SSD. The estimation of mean degree of SBP is described in (*32*). Unshaded areas indicate regions where the biases in size dimorphism and parasitism are in the same direction. The dendrogram of the sister taxa used in this analysis is shown in fig. S2.

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nogamy (Fig. 2) and conventional or reversed SSD (Fig. 5) must necessarily be made at bifurcating nodes. Where multiple nodes (consisting of sister taxa exhibiting, for example, all conventional SSD) made up one side of the node, the mean value of their individual SSD scores was used in the contrast against the taxa exhibiting reversed SSD, at that particular node.

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34. We are grateful to I. Owens, A. Read, M. Zuk, and three anonymous reviewers for commenting on and improving earlier drafts of this manuscript, and to R. Johnstone and I. Owens for providing valuable guidance on the CAIC analyses. We also acknowledge the help of S. Cotter, R. Hails, C. Lapsley, and D. Promislow. The authors declare that there was no sex bias in the contributions made to this paper. Supported by the Natural Environment Research Council, UK.

Supporting Online Material
www.sciencemag.org/cgi/content/full/297/5589/2015/DC1
Materials and Methods
SOM Text
Figs. S1 and S2
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21 May 2002; accepted 25 July 2002

Nucleotide Control of Interdomain Interactions in the Conformational Reaction Cycle of SecA

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The SecA adenosine triphosphatase (ATPase) mediates extrusion of the amino termini of secreted proteins from the eubacterial cytosol based on cycles of reversible binding to the SecYEG translocon. We have determined the crystal structure of SecA with and without magnesium–adenosine diphosphate bound to the high-affinity ATPase site at 3.0 and 2.7 angstrom resolution, respectively. Candidate sites for preprotein binding are located on a surface containing the SecA epitopes exposed to the periplasm upon binding to SecYEG and are thus positioned to deliver preprotein to SecYEG. Comparisons with structurally related ATPases, including superfamily I and II ATP-dependent helicases, suggest that the interaction geometry of the tandem motor domains in SecA is modulated by nucleotide binding, which is shown by fluorescence anisotropy experiments to reverse an endothermic domain-dissociation reaction hypothesized to gate binding to SecYEG.

SecA uses ATP-driven cycles of insertion and retraction from the membrane-bound SecYEG translocon to mediate processive extrusion of preproteins through the cytoplasmic membrane of eubacteria (*1–4*). This cyclical interaction explains the presence of SecA in vivo in both soluble and membrane-bound forms (*5*). In vitro translocation assays show that the ATPase activity of SecA (*6*, *7*) is required for the transmembrane transport of the NH_2-terminus of the preprotein, which includes the signal sequence (*8*, *9*) that targets proteins for export through the translocon (*3*, *10*). The ATPase activity of SecA is also required to drive the transport of downstream COOH-terminal segments of the preprotein in the absence of a transmembrane protonmotive force (*10–12*). Understanding the structure of SecA and its ATP-driven conformational reaction cycle is essential to understanding the mechanism by which the energy of ATP binding and hydrolysis is exploited to produce vectorial preprotein transport.

Crystal structure determinations and refinements. The soluble form of SecA from *Bacillus subtilis* was crystallized (*13*) and solved (*14*) in space group $P3_112$. Multiple isomorphous replacement phases were obtained to 4.4 Å by using heavy-atom derivatives of wild-type protein and an N96C mutant. The phases were extended to high resolution by using density modification procedures (*15*) combined with partial-model building. Assignment of the sequence was facilitated by the use of an anomalous difference Fourier map from a selenomethionine derivative that showed the locations of 33 of the 36 methionines, and a real-space cross-validation procedure was used when adding the side chains to prevent phase bias from impeding the procedure. Because the refined phases are ultimately derived from the coordinate model in all macromolecular crystal structure determinations, the quality of the map (fig. S1) (*14*) and of the model (Table 1 and Fig. 1) produced in this way is equivalent to that of a structure solved in a more traditional way and refined to an equivalent *R* factor.

SecA crystallized isomorphously either in the presence or absence of adenine nucleotides or analogs, consistent with solution studies showing that nucleotide binding does not produce large conformational changes in the free enzyme (*16*). Isomorphous difference Fourier maps from all nucleotide-containing crystals showed electron density for a single ligand bound at the high-affinity ATPase site identified in mutagenesis studies (*17*). Slightly weaker diffraction (by ~0.5 Å) was obtained when nucleotides were cocrystallized rather than soaked in after growth, so that we chose to refine a data set obtained from a native crystal soaked in 5 mM Mg-ATP for 2.5 hours. However, there was no evidence of the γ-phosphate of ATP during the subsequent refinement, indicating that Mg-ADP was bound. Additional soaking experiments suggested that a kinetic barrier prevents Mg-ATP binding in the crystal lattice (*18*). Given the high affinity of SecA for Mg-ADP [dissociation constant (K_d) ≈ 100 nM] (*19*), we assume that the protein selectively bound the low concentration of Mg-ADP in the stock.

There was no credible evidence for a second bound nucleotide molecule in the refined Mg-ADP–bound structure, indicating that the low-affinity ATP-binding site (*17*, *20*) was not stably occupied by well-ordered nucleotide in our crystals. Binding could have been inhibited by the >2 M $NH_2(SO_4)_2$ present in the mother liquor or prevented by crystal packing interactions, which could either obstruct the site or restrain the protein in an incompatible conformation. (General considerations concerning the potential location of this site are presented in the SOM text.)

The *apo* structure was refined (*21*) at 2.7 Å resolution to working and free *R* factors of 22.1 and 30.4%, respectively, whereas the Mg-ADP–bound structure was refined at 3.0 Å resolution to working and free *R* factors of 21.8 and 29.4%, respectively (Table 1 and Fig. 1; figs. S2 and S3). The same set of free reflections was used for both refinements. Only ~83% of the residues in the refined SecA structures have most-favored Ramachandran angles. However, there are very few Ramachandran outliers, and the residues with noncore angles occur primarily in weakly ordered regions with very high backbone *B* factors. The well-ordered regions have good backbone geometry, and the low root-

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Science **297** (5589), . DOI: 10.1126/science.1074196