Second, disease progression requires response to maize organ-specific properties so that *U. maydis* can tailor effector deployment to redirect physiology and development of a specific organ primordium. Sequential refinement of specificity may be of particular importance in this biotrophic interaction, which lasts 14 days from host penetration to fungal spore release. Within this conceptual framework, the next step is elucidation of distinct fungal and host factors interacting in a tissue-specific and temporal context. This new knowledge will clarify how organ-specific factors modulate biotrophy and, ultimately, tumor formation.

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Supporting Online Material

www.sciencemag.org/cgi/content/full/328/5974/89/DC1
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Cryptic Sex-Ratio Bias Provides Indirect Genetic Benefits Despite Sexual Conflict

Robert M. Cox* and Ryan Calsbeek

When selection favors sexual dimorphism, high-fitness parents often produce low-fitness progeny of the opposite sex. This sexual conflict is thought to overwhelm the genetic benefits of mate choice because preferred males incur a cost through the production of low-fitness daughters. We provide a counterpoint in a lizard (*Anolis sagrei*) that exhibits sexual conflict over body size. By using mate-choice experiments, we show that female brown anoles produce more sons than daughters via large sires but more daughters than sons via small sires. Measures of progeny fitness in the wild suggest that maximal fitness payoffs can be achieved by shifting offspring production from daughters to sons as sire size increases. These results illustrate how the resolution of sexual conflict can restore the genetic benefits of mate choice.

Because of their divergent reproductive roles, males and females often experience different selection pressures acting on the same phenotypic traits (*1*). However, sharing a common genome constrains the sexes from evolving independently in response to these antagonistic selection pressures (*2*–*4*). This can result in a genomic tug of war referred to as intralocus sexual conflict (*5*–*7*). When such conflict is widespread throughout the genome, high-fitness parents may actually produce low-fitness progeny of the opposite sex (*8*–*14*). This outcome can override the potential genetic benefits of mate choice because preferred males incur a net fitness cost through the production of low-fitness daughters (*8*–*10*). When sire genotypes have differential fitness effects on sons versus daughters, females are predicted to alter progeny sex ratio accordingly (*15*). We tested whether progeny sex-ratio bias can facilitate the sex-specific inheritance of good genes, thereby preserving the genetic benefits of mate choice in the face of sexual conflict.

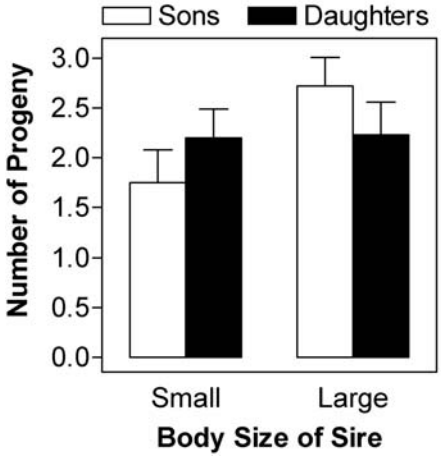

Fig. 1. Female anoles bias progeny sex ratio as a function of sire body size. Data are least-squares means ± 1 SEM from analyses weighted by the total number of progeny produced by each dam-sire pair. Size is dichotomized relative to the population mean.

The brown anole lizard (*Anolis sagrei*) exhibits signatures of intralocus sexual conflict over body size (fig. S1). On average, adult males are 30% longer and 150% heavier than adult females (*16*). Selection creates the potential for sexual conflict by favoring large size in males and intermediate size in females (*17*). However, anoles have also evolved several mechanisms that may resolve this conflict. First, body size and other morphological traits are heritable within each sex but exhibit negative genetic correlations between the sexes (*18*). Second, paternity analyses of wild populations reveal that females produce more sons via large sires but more daughters via small sires (*18*). This suggests a form of cryptic sex-ratio bias that may allow females to adaptively sort genes with sex-specific fitness effects into sons and daughters.

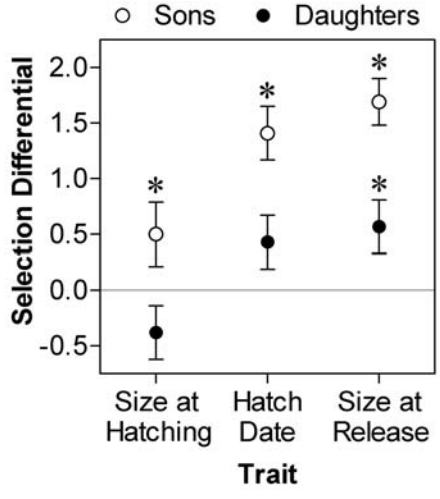

Fig. 2. Natural selection on three phenotypic traits differs between male and female progeny. Data are selection differentials ± 1 SEM derived from regressions of relative survival on trait values standardized to the population mean in unit variance. Asterisks indicate statistical significance ($P < 0.05$) on the basis of logistic regression.

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Because it is unknown whether this sex-ratio bias actually increases progeny fitness, we mated females to males of varying sizes, assigned paternity to their progeny, and then measured the fitness of these sons and daughters in the wild (*19*). Because the hypothesized fitness advantage of sex-ratio bias requires indirect genetic benefits, we hatched and raised all sires to adult size in a laboratory common garden to minimize nongenetic variation in body size. We provided each dam with two potential sires, paired such that some dyads consisted of two males of similar size whereas others consisted of two males of disparate sizes. We allowed dams to assess both males together for several days and then permitted each male to mate exclusively with the dam for 1 week to prevent direct male-male interactions from influencing mating success.

Overall, dams produced a balanced sex ratio (111 sons, 101 daughters; $\chi^2 = 0.47$; $P = 0.49$). However, as predicted by sexual conflict theory (*12*, *13*, *18*), dams produced relatively more sons with large sires and more daughters with small sires (sex × sire size: $F_{1,72} = 4.23$; $P = 0.043$) (Fig. 1). This bias became stronger when we increased the dichotomy between small and large sires by excluding males of intermediate size that fell within ±0.25 SD (±1 mm) of the population mean (sex × sire size: $F_{1,47} = 9.07$; $P = 0.004$). This confirms the pattern previously observed in wild populations (*18*) and suggests a refined form of cryptic fertilization bias. Moreover, this bias was driven primarily by the differential production of sons ($F_{1,72} = 4.95$; $P = 0.029$), rather than daughters ($F_{1,72} = 0.01$; $P = 0.945$), with sires of different sizes (Fig. 1). Therefore, we predicted that the fitness of sons in particular should be strongly tied to body size and positively correlated with the size of the sire.

To test these predictions, we released progeny from our breeding experiment into their natural habitat and followed their survival over 8 months to measure natural selection acting on juvenile phenotypes (Fig. 2). In both sexes, viability selection favored early hatch date ($\beta = 0.86 \pm 0.17$; $\chi^2 = 25.15$; $P < 0.001$) and large body size at release ($\beta = 1.06 \pm 0.16$; $\chi^2 = 35.81$; $P < 0.001$). However, this selection was significantly stronger for sons than it was for daughters (sex × hatch date: $\chi^2 = 7.55$; $P = 0.006$; sex × size at release: $\chi^2 = 8.82$; $P = 0.003$). Moreover, selection on size at hatching exhibited sexual antagonism, favoring large sons and small daughters (sex × size at hatching: $\chi^2 = 6.41$; $P = 0.011$) (Fig. 2). Collectively, these patterns of phenotypic selection indicate that large size and early hatch date are particularly important for the survival of sons, as predicted from patterns of cryptic sex-ratio bias.

To directly assess the adaptive value of cryptic sex-ratio bias, we examined the fitness of progeny as a function of sire body size. As predicted, survival of sons increased with sire size ($\chi^2 = 7.21$; $P = 0.007$). By contrast, survival of daughters was unrelated to sire size ($\chi^2 = 1.09$; $P = 0.30$). The resulting fitness functions (Fig. 3) reveal that maximal fitness payoffs are achieved by shifting progeny sex ratio from female-biased to male-biased as sire size increases. This suggests an adaptive function for sex-ratio bias, as previously hypothesized (*17*). However, sex-ratio bias at hatching could also arise as a nonadaptive by-product if small males sire sons with reduced embryonic viability. Given the fitness functions in Fig. 3, we favor an adaptive explanation, although it would be informative to generate analogous fitness functions for embryonic viability.

To date, adaptive interpretations of sex-ratio bias have been based on the assumption that fitness is negatively heritable from sire to daughter (*18*), as typically observed in the face of intralocus sexual conflict (*8*–*14*). By contrast, our data suggest that the fitness of daughters is independent of sire size. This implies that cryptic sex-ratio bias has not evolved to avoid the production of low-quality daughters but as a means of obtaining genetic benefits that are sex-specific to sons. This raises the question of why females mate with small males. In part, this reflects the nature of our experimental design, which constrained many females to mate only with small males (*19*). Those females that were given a choice between small and large males produced nearly twice as many total progeny with large sires (mean of 1.7 progenies) as they did with small sires (0.9 progeny; $F_{1,64} = 2.90$; $P = 0.09$). This suggests that females prefer large sires for all of their progeny, which is expected on the basis of progeny fitness functions (Fig. 3). Field studies of this and other territorial *Anolis* species indicate that females mate almost exclusively (95 to 98% of copulations) with males on whose territories they reside, displaying little precopulatory mate choice with respect to male phenotype (*20*, *21*). Our data suggest that, through post-copulatory control over progeny sex ratio, females can nonetheless minimize the production of low-quality sons when mating with small males.

Unexpectedly, although the survival of progeny was positively related to the size of the sire ($\chi^2 = 12.17$; $P < 0.001$), it was negatively related to the size of the dam ($\chi^2 = 8.09$; $P = 0.005$; Fig. 4). In particular, small dams that mated with large sires produced progeny with higher survival than any other combination of parental phenotypes ($\chi^2 = 14.49$; $P = 0.002$). This illustrates how intralocus sexual conflict can extend beyond sex differences in selection for adult viability and reproductive success and include the antagonistic effects of progeny survival on adult phenotypes. This form of intralocus sexual conflict may have been previously overlooked because of the lack of detailed pedigrees and intergenerational measures of fitness (*1*).

We note several important differences between brown anoles and other species (*8*–*10*) in which the costs of sexual conflict overwhelm the genetic benefits of mate choice. First, brown anoles exhibit strong intergenerational links between

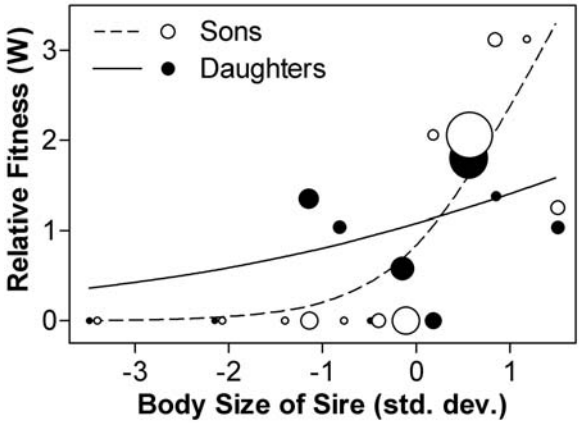

Fig. 3. Sex-specific fitness payoffs favor a transition from production of daughters to sons as sire body size increases. Body size is expressed in unit variance relative to the population mean. Data points report relative fitness for each increment of sire body size, calculated by dividing the mean probability of survival at each size by the overall probability of survival within each sex. The size of each point is proportional to the number of progeny (range from 1 to 15) produced by sires of that size. Fitness functions are best-fit cubic splines with standard errors omitted for clarity.

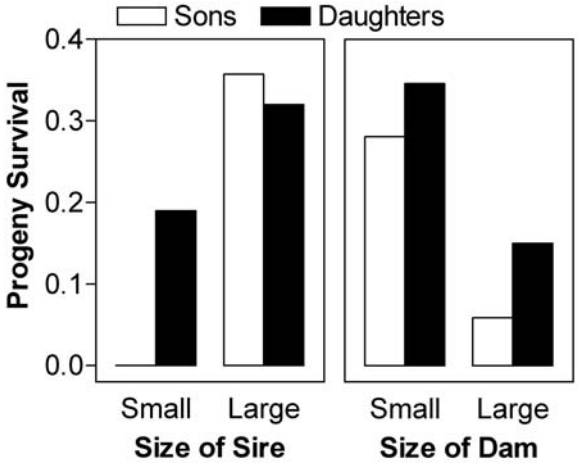

Fig. 4. Large sires had progeny with high survival (**left**), whereas large dams had progeny with low survival (**right**). Size is dichotomized relative to the separate population means for sires and dams.

the fitness of sires and sons. Natural selection favors large size in adult males (*17*), and dams produce more sons via large sires [Fig. 1 and (*8*)]. Body size is heritable from sire to son (*18*), and selection on juvenile viability favors large size and early hatch date in male progeny (Fig. 2). Consequently, large sires have high adult fitness and produce sons with high juvenile fitness (Fig. 3). Given the heritability of adult body size (*18*), large sires presumably also produce sons with high adult fitness. This differs from systems in which fitness is not heritable within males (*9*, *11*) because of the accumulation of sexually antagonistic genes on the X chromosome (*10*, *22*, *23*), which does not pass from sire to son. The mechanism of sex determination is unknown in brown anoles, but related *Anolis* species exhibit XY and XXY male heterogamety or genetic sex determination without heteromorphic sex chromosomes (*24*, *25*).

Second, in other species, males with high fitness often sire daughters with low fitness (*8*–*13*). This negative intersexual heritability of fitness may be common when sexually antagonistic X-linked genes are inherited from sire to daughter (*8*, *10*). However, the outcome of good-genes mate choice is complex and likely varies with patterns of sex linkage (*26*). Moreover, intersexual genetic correlations are often reduced or negative for sexually dimorphic traits (*27*–*29*), and the intersexual genetic correlation for body size is actually negative in brown anoles (*18*). This suggests that potential sexual conflict over body size has been largely resolved. Indeed, we found no evidence that large sires produce low-fitness daughters (Fig. 3). In this situation, any potential genetic benefits of mate choice should be preserved, contrary to the situation when high-fitness sires produce low-fitness daughters (*9*–*11*).

Our study suggests that indirect genetic benefits can be obtained even in the face of intralocus sexual conflict. However, this outcome is likely contingent on the evolution of mechanisms that resolve sexual conflict, thereby facilitating sex-specific inheritance and expression of good genes. In brown anoles, these mechanisms may include cryptic sex-ratio bias, which would allow females to preferentially produce high-fitness sons, and negative intersexual genetic correlations, which would mitigate the potential costs of producing low-fitness daughters. Because the underlying physiological mechanisms that produce cryptic sex-ratio bias are presently unknown, we cannot reject the alternative that this bias reflects differential embryonic mortality of sons and daughters with respect to sire size. Given the emerging perspective that intralocus sexual conflict can maintain genetic variation and constrain evolution via mate choice (*8*–*10*), further investigation of these mechanisms should clarify the implications of sexual conflict for a variety of fundamental evolutionary processes.

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Supporting Online Material

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Partitioning of Histone H3-H4 Tetramers During DNA Replication–Dependent Chromatin Assembly

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Semiconservative DNA replication ensures the faithful duplication of genetic information during cell divisions. However, how epigenetic information carried by histone modifications propagates through mitotic divisions remains elusive. To address this question, the DNA replication–dependent nucleosome partition pattern must be clarified. Here, we report significant amounts of H3.3-H4 tetramers split in vivo, whereas most H3.1-H4 tetramers remained intact. Inhibiting DNA replication–dependent deposition greatly reduced the level of splitting events, which suggests that (i) the replication-independent H3.3 deposition pathway proceeds largely by cooperatively incorporating two new H3.3-H4 dimers and (ii) the majority of splitting events occurred during replication-dependent deposition. Our results support the idea that "silent" histone modifications within large heterochromatic regions are maintained by copying modifications from neighboring preexisting histones without the need for H3-H4 splitting events.

Histone and DNA modifications provide key epigenetic information (*1*–*3*). A newly synthesized DNA strand acquires its DNA methylation pattern by copying the preexisting DNA methylation signature from the template strand (*1*, *4*, *5*). However, the mechanism by which patterns of histone modifications are passed on to daughter cells through mitotic divisions remains enigmatic. To understand this, the DNA replication–dependent nucleosome partition pattern must be unveiled first. Initial studies indicated that the nucleosomes do not dissociate (*6*, *7*), which was amended by the discoveries of "hybrid nucleosomes" that contain old H3-H4 tetramers and new H2A-H2B dimers or vice versa (*8*–*11*). Nevertheless, H3-H4 tetramers—the core particles of nucleosomes—do not dissociate during replication-dependent nucleosome assembly (*12*–*15*). Because all six major lysine methylation sites are present on either H3 (Lys4/9/27/36/79) or H4 (Lys20), newly deposited nucleosomes may become methyl-

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