LETTERS

Sexually antagonistic genetic variation for fitness in red deer

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Evolutionary theory predicts the depletion of genetic variation in natural populations as a result of the effects of selection, but genetic variation is nevertheless abundant for many traits that are under directional or stabilizing selection[1]. Evolutionary geneticists commonly try to explain this paradox with mechanisms that lead to a balance between mutation and selection[2]. However, theoretical predictions of equilibrium genetic variance under mutation–selection balance are usually lower than the observed values, and the reason for this is unknown[3]. The potential role of sexually antagonistic selection in maintaining genetic variation has received little attention in this debate, surprisingly given its potential ubiquity in dioecious organisms. At fitness-related loci, a given genotype may be selected in opposite directions in the two sexes. Such sexually antagonistic selection will reduce the otherwise-expected positive genetic correlation between male and female fitness[4]. Both theory[5–7] and experimental data[8–12] suggest that males and females of the same species may have divergent genetic optima, but supporting data from wild populations are still scarce[13–15]. Here we present evidence for sexually antagonistic fitness variation in a natural population, using data from a long-term study of red deer (*Cervus elaphus*). We show that male red deer with relatively high fitness fathered, on average, daughters with relatively low fitness. This was due to a negative genetic correlation between estimates of fitness in males and females. In particular, we show that selection favours males that carry low breeding values for female fitness. Our results demonstrate that sexually antagonistic selection can lead to a trade-off between the optimal genotypes for males and females; this mechanism will have profound effects on the operation of selection and the maintenance of genetic variation in natural populations.

Polygynous ungulates such as red deer are particularly likely candidates for taxa in which sexually antagonistic genetic variation may occur, because their pronounced size dimorphism and male weaponry require substantial genotype–sex interactions to enable different developmental schemata for males and females. Furthermore, reproductive roles differ greatly between the sexes. Males compete intensely for matings during the short annual rut but do not invest in offspring care, whereas female maternal investment extends over a long period during each reproductive event. Consequently, male and female life histories are likely to be under divergent selective pressures, and a particular genotype may have very different effects on fitness in males than in females. Last, the polygynous mating system, with short periods of tenure by dominant males, probably reduces the correlation for fitness between mating pairs, enhancing the chance that sexually antagonistic genetic variation may evolve[16]. We investigated the heritable genetic basis of variation in fitness in a wild population of red deer living in the North Block of the Isle of Rum, Scotland. We used individual contributions to population growth, estimated through a method known as 'de-lifing'[17], as a measure of fitness. This measure, $p_{t(i)}$, estimates an individual's annual contribution to changes in population size through both reproduction and survival, and it approximates the expected future representation of an individual's alleles in the population gene pool[17]. The de-lifing method measures performance at each potential reproductive event, rather than on the basis of a per-generation time scale. This allows the incorporation of additional information about annual environmental variation, as well as data from incomplete life histories. Where complete life histories were available, we also calculated two lifetime measures of fitness, the lifetime sum of $p_{t(i)}$, and lifetime reproductive success (LRS) as the number of surviving offspring produced over the entire lifespan. Methods and results for the traditional fitness measure LRS are given in Supplementary Information. The correlation coefficient between LRS and the lifetime sum of $p_{t(i)}$ was 0.79 and 0.77 in males and females, respectively ($n = 284$ males and 301 females, both $P < 0.001$).

First, we conducted classical parent–offspring regressions to investigate the sex-specific heritability of fitness. We regressed average son and average daughter values of the lifetime sum of $p_{t(i)}$ (only for individuals with complete life history information) on the values for their fathers and mothers (Fig. 1). Male red deer with a relatively high lifetime sum of $p_{t(i)}$ sired, on average, daughters with a relatively low lifetime sum of $p_{t(i)}$ (Fig. 1; $P = 0.003$). However, there was no significant relationship between the lifetime sum of $p_{t(i)}$ of fathers and sons, or of mothers and their offspring (Fig. 1). Although the slope of the mother–daughter regression was not significantly positive, it differed from the slope of the father–daughter regression (test for the equality of regression slopes[18], $P < 0.01$). This may indicate an underlying genetic antagonistic effect, which is masked by compensating maternal effects on daughters of females with low breeding values for fitness. The slopes of the mother–daughter and the mother–son regressions were not significantly different ($P > 0.05$). If female red deer can compensate for their offspring's disadvantages from sexually antagonistic genes through direct maternal effects, we would indeed not expect such a difference in a phenotypic analysis. Under the assumption that the patterns observed in the parent–offspring regressions are partly due to heritable genetic variance for fitness, we predicted that $p_{t(i)}$ would have a heritable component and that it would show a negative genetic covariance between the sexes.

We estimated heritable genetic variance by using the 'animal model', a mixed-model approach that uses the relatedness between all pairs of individuals in a pedigree to estimate the genetic variance

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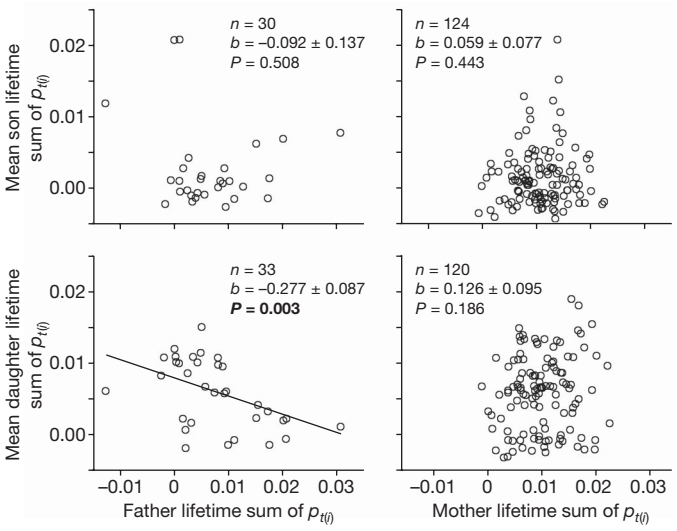

Figure 1 | Sex-specific parent–offspring regressions of observed fitness in red deer. Fitness was estimated as the lifetime sum of $p_{t(i)}$, a measure of an individual's annual contribution to changes in population size through reproduction and survival. Here, only deer with complete life history information are included. The very different sample sizes for fathers and mothers are expected under this highly polygynous mating system. The strong skew in male reproductive success produces a female-biased sex ratio among reproductively active red deer. Many males never reproduce and appear as sons, although not as fathers, in this analysis. *b* is the slope of a linear regression of mean offspring fitness on parental fitness.

component of a trait[19]. In contrast with the parent–offspring regressions, this approach allowed us to use all available data in one analysis, to account for direct maternal effects, and to include repeated annual measures of $p_{t(i)}$, data from males and females that never reproduced, and data from incomplete life histories. As expected for a fitness-related trait[20,21], the annual measure of $p_{t(i)}$ showed low narrow-sense heritability h^2 (significant only in females; Table 1), but high coefficients of additive genetic variation. The lower heritability in males may be due to high stochasticity of male mating success (increasing the residual variance) and the lack of information on some paternities in and around the study area (reducing the genetic variance). However, we detected a significant negative genetic covariance for male and female $p_{t(i)}$ in a bivariate animal model, which results in a significant negative genetic correlation between male and female $p_{t(i)}$ (Table 1, $P = 0.002$). Hence, male red deer that contribute most to the annual population growth have female relatives that contribute less, whereas males that are less successful have relatively more successful female relatives. Fitting separate animal models for the two components of $p_{t(i)}$, namely annual reproduction and survival, we found a negative genetic correlation between male and female reproduction but not between male and female survival (Table 1). This is consistent with expectation, as sexual antagonism is likely to be more pronounced with respect to reproduction (where the sexes diverge most) as opposed to survival. We found no significant heritability or genetic covariance for a more crude measure of fitness, LRS (see Supplementary Information). However, the genetic correlation of male and female LRS was also negative and significantly less than +1 (Supplementary Information, $P < 0.001$). When pleiotropic effects are perfectly correlated across the sexes, the expected genetic correlation between male and female traits is of +1 (ref. 4). A significant deviation from this may be caused by antagonistic genetic variation, by sex-biased gene expression, or by both. In either case, it suggests the potential for constraints on the evolution of a single genetic optimum[4].

We detected a significant positive covariance for maternal effects on male and female $p_{t(i)}$ (Table 1), indicating that a mother's maternal effects on her offspring (that is, those acting in addition to the additive genetic effects of the genes that the offspring has inherited from her) benefit her sons and daughters in similar ways. This positive covariance may possibly have masked any sexually antagonistic genetic effects in our mother–offspring phenotype regressions. In the presence of sexually antagonistic genetic variation for fitness, maternal investment that enhances the fitness of both sons and daughters equally may be particularly adaptive, if mothers are not capable of choosing specific mates or manipulating offspring sex to optimize the genetic merit (or breeding value) for fitness of their sons and daughters. Correlated maternal effects on male and female offspring may therefore be a common reason why sexually antagonistic variation for fitness-related traits is difficult to detect in the field. Laboratory experiments on the fruitfly *Drosophila melanogaster*, however, demonstrated the negative consequences of an antagonistic effect on both male and female phenotypes[6], suggesting small or uncorrelated maternal effects on offspring fitness in this system.

Our findings show that optimal genotypes differ between male and female red deer, because a genotype that produces a male phenotype with relatively high fitness will, on average, produce a phenotype with lower fitness when expressed in a female. The sexually antagonistic genetic variation for fitness should counteract selection on the optimal male and female genotype, respectively. To test this, we calculated the prediction of each individual's additive genetic merit for the annual measure of $p_{t(i)}$ in the opposite sex, known as its breeding value[4], from the animal model. Note that we were able to predict a breeding value for $p_{t(i)}$ in the opposite sex for all animals in which we observed $p_{t(i)}$ in opposite-sex relatives. If optimal genotypes differ between the sexes, we expect that males with a high breeding value for female $p_{t(i)}$ will show low fitness, and vice versa. In support of this expectation, we found that a male's breeding value for female $p_{t(i)}$ was negatively related to his own phenotypic value of male $p_{t(i)}$, and hence fitness (Fig. 2a; $P = 0.017$). This confirms our results from the parent–offspring regression: successful males sired, on average, daughters that were less successful, because their genotype had a lower breeding value for female $p_{t(i)}$. In contrast, a female's breeding value for male $p_{t(i)}$ was not significantly related to her own observed $p_{t(i)}$ (Fig. 2b). Again, this confirms the finding that successful females did not, on average, produce sons that were less successful. Our study

Table 1 | Variance components, heritability h^2, and the inter-sexual genetic correlation of fitness in red deer

Variance components No. of observations (individuals)	Mean ± s.d.	Additive genetic	Maternal	Permanent environment	Residual	h^2	P	CV_A
Female $p_{t(i)}$ 2852 (387)	0.00080 ± 0.0013	**12.53** (3.46)	0.30 (2.11)	0	**132.74** (3.76)	**0.086** (0.023)	**0.002**	44.25
Male $p_{t(i)}$ 2116 (342)	0.00018 ± 0.0016	7.93 (7.06)	1.03 (4.24)	**12.76** (7.17)	**205.02** (6.82)	0.035 (0.031)	0.128	156.45

Covariances	Additive genetic	Maternal	Genetic correlation	P
Female $p_{t(i)}$ − male $p_{t(i)}$	**−10.26** (4.18)	**5.08** (2.01)	**−0.95** (0.42)	**0.014**
Female reproduction R − male reproduction R	**−8.42** (1.97)	**3.61** (0.98)	**−1.38** (0.42)	<**0.001**
Female survival S − male survival S	−1.35 (2.59)	2.02 (1.51)	−0.45 (0.89)	0.527

Univariate animal models were used to partition the variance of male and female $p_{t(i)}$, a measure of an individual's annual contribution to changes in population size through reproduction (R) and survival (S). Covariances and correlations of female and male $p_{t(i)}$ and its components R and S were obtained from bivariate animal models. Values in parentheses are standard errors as estimated by the software ASReml. Statistically significant components ($P < 0.05$) are indicated in bold; the probabilities shown are for additive genetic effects. All presented variance components and covariances are based on transformed values of $p_{t(i)}$, R and S (×10,000). The coefficient of variation for the additive genetic component (CV_A) is a standardized measure of genetic variance and was calculated as 100 × √(additive genetic variance)/transformed mean $p_{t(i)}$.

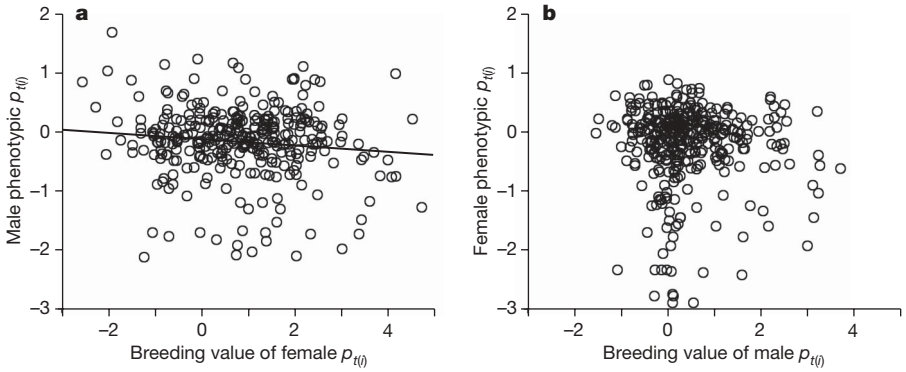

Figure 2 | Selection on opposite-sex breeding values of annual fitness in males and females. The plotted data and regression line are based on average phenotypic values of residual annual $p_{t(i)}$ (accounting for effects of age and, in females, recent reproductive history); the statistics refer to linear mixed models with male (**a**) or female (**b**) identity as a random factor. **a**, Relationship between a male's mean phenotypic value of $p_{t(i)}$ and his breeding value for female $p_{t(i)}$ ($n = 332$ males, $b = (-8.62 \pm 3.62) \times 10^{-5}$, Wald statistic = 5.67, d.f. = 1, $P = 0.017$). **b**, Relationship between a female's mean phenotypic value of $p_{t(i)}$ and her breeding value for male $p_{t(i)}$ ($n = 371$ females, $b = (-5.41 \pm 3.60) \times 10^{-5}$, Wald statistic = 2.27, d.f. = 1, $P = 0.132$).

therefore provides evidence for sexually antagonistic genetic variation for $p_{t(i)}$, a measure of annual survival and reproductive success. Our findings suggest that selection for successful males causes a correlated selective pressure against high female breeding values.

In species with male heterogamety, such as mammals, sexually antagonistic genetic variation is expected to accumulate on the X chromosome[7,11]. X-linked alleles with new antagonistic mutations should spread rapidly within a population's gene pool if they are recessive and beneficial to males, because such alleles are directly accessible to positive selection in all males that inherit them (because males possess only one X chromosome) but are protected from negative selection in all heterozygous females. In concordance with the predictions for an X-linked effect, we observed a negative covariance for fitness between fathers and daughters but no covariance between fathers and sons (Fig. 1). We did not detect the expected covariances between mothers and sons (negative), and mothers and daughters (positive), but these are likely to be masked by direct maternal effects on offspring fitness, which are apparent in our animal model analyses (Table 1). Finally, antagonistic alleles seem to be selected for in males (Fig. 2a), but on average not selected against in females (Fig. 2b). Our results are therefore broadly consistent with the predictions for an antagonistic effect of recessive alleles at one or several X-linked loci, as observed in *Drosophila*[11].

Our findings imply a limit to the adaptive evolution of male and female phenotypes in red deer. Sexually antagonistic selection may thus maintain heritable genetic variance in reproductive traits[20], but it may also have a role in a reduced response to directional sexual selection, as observed in male red deer[22]. Sexually antagonistic effects have largely been neglected in models of sexual selection that relate individual mating decisions to the indirect benefit of 'good genes'[23]. Our study provides evidence from a natural population that such 'good genes' may be gender-specific and would not provide equal benefits to sons and to daughters[14,24]. Sexually antagonistic genetic variation could therefore reduce or even reverse indirect benefits from sexual selection. Females that mate with successful males bear the costs of producing daughters with breeding values for low reproductive output. If the antagonistic effect is X-linked, they will also not gain any indirect benefits through their sons. Choosy females should gain fitness advantages through successful grandsons, but this benefit is likely to be reduced through a generation delay and recombination, and may further be attenuated by the likely reduced reproductive output of granddaughters[24]. The data from red deer on Rum reveal that sexually antagonistic heritable genetic variance is contributing to trait variance in the wild, and that its consequences for sex-specific fitness are not confined to experimental or laboratory populations, which typically show reduced environmental variance for fitness and weak effects of natural selection. As a consequence, the potential of sexually antagonistic fitness variation needs careful consideration when investigating natural and particularly sexual selection, and more studies focusing on such effects in natural populations are warranted.

METHODS SUMMARY

We used life history data on individual red deer (*Cervus elaphus*) living in the North Block of the Isle of Rum, Scotland, between 1971 and 2005. All animals in the study population are individually recognizable, and their survival and reproductive success have been monitored[20,22,25–27]. We obtained a population pedigree including 3,559 animals from eight generations, using microsatellite paternity data[28] and rut observations[20]. For all animals that lived for at least three years, we measured fitness using the de-lifing approach[17], a jack-knifing procedure to calculate an individual's contribution to changes in population size, $p_{t(i)}$, through both reproduction and survival. $p_{t(i)}$ estimates the relative performance of an individual in each year, and this procedure therefore allowed us to include incomplete life histories. We also present analyses of the two separate components of $p_{t(i)}$: annual survival *S*, and annual reproduction *R*. For a single lifetime measure of fitness (only for individuals with complete life history information, excluding shot deer or deer that are still alive), we used the sum of all annual $p_{t(i)}$ values, referred to as the lifetime sum of $p_{t(i)}$. We present *P* values from two-tailed tests with $\alpha = 0.05$.

We partitioned phenotypic trait variance into additive genetic variance, maternal variance, permanent environment variance, and residual variance by using the animal model as implemented by the software ASReml[29]. The animal model uses pedigree information to extract the additive genetic component in a mixed-model framework based on restricted maximum-likelihood (REML) estimation[19]. We applied univariate models to estimate narrow-sense heritabilities (h^2) and bivariate models for the calculation of inter-sexual covariances and genetic correlations. We used predictions of the additive genetic effect from the bivariate model to investigate selection on opposite-sex breeding values.

Full Methods and any associated references are available in the online version of the paper at www.nature.com/nature.

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1. Merila, J., Sheldon, B. C. & Kruuk, L. E. B. Explaining stasis: microevolutionary studies in natural populations. *Genetica* **112**, 199–222 (2001).
2. Barton, N. H. & Keightley, P. D. Understanding quantitative genetic variation. *Nature Rev. Genet.* **3**, 11–21 (2002).
3. Turelli, M. & Barton, N. H. Polygenic variation maintained by balancing selection: Pleiotropy, sex-dependent allelic effects and G × E interactions. *Genetics* **166**, 1053–1079 (2004).
4. Lynch, M. & Walsh, B. *Genetics and Analysis of Quantitative Traits* (Sinauer, Sunderland, Massachusetts, 1998).
5. Hedrick, P. W. Antagonistic pleiotropy and genetic polymorphism: a perspective. *Heredity* **82**, 126–133 (1999).
6. Rice, W. R. & Chippindale, A. K. Intersexual ontogenetic conflict. *J. Evol. Biol.* **14**, 685–693 (2001).
7. Rice, W. R. Sex chromosomes and the evolution of sexual dimorphism. *Evolution Int. J. Org. Evolution* **38**, 735–742 (1984).
8. Chippindale, A. K., Gibson, J. R. & Rice, W. R. Negative genetic correlation for adult fitness between sexes reveals ontogenetic conflict in *Drosophila*. *Proc. Natl Acad. Sci. USA* **98**, 1671–1675 (2001).

9. Fedorka, K. M. & Mousseau, T. A. Female mating bias results in conflicting sex-specific offspring fitness. *Nature* **429,** 65–67 (2004).
10. Rice, W. R. Sexually antagonistic genes: experimental evidence. *Science* **256,** 1436–1439 (1992).
11. Gibson, J. R., Chippindale, A. K. & Rice, W. R. The X chromosome is a hot spot for sexually antagonistic fitness variation. *Proc. R. Soc. Lond. B* **269,** 499–505 (2002).
12. Meagher, T. R. The quantitative genetics of sexual dimorphism in *Silene latifolia* (Caryophyllaceae). 1. Genetic variation. *Evolution Int. J. Org. Evolution* **46,** 445–457 (1992).
13. Forsman, A. Opposing fitness consequences of colour pattern in male and female snakes. *J. Evol. Biol.* **8,** 53–70 (1995).
14. Calsbeek, R. & Sinervo, B. Within-clutch variation in offspring sex determined by differences in sire body size: cryptic mate choice in the wild. *J. Evol. Biol.* **17,** 464–470 (2004).
15. Robinson, M. R., Pilkington, J. G., Clutton-Brock, T. H., Pemberton, J. M. & Kruuk, L. E. B. Live fast, die young: Trade-offs between fitness components and sexually antagonistic selection on weaponry in Soay sheep. *Evolution Int. J. Org. Evolution* **60,** 2168–2181 (2006).
16. Rice, W. R. Dangerous liaisons. *Proc. Natl Acad. Sci. USA* **97,** 12953–12955 (2000).
17. Coulson, T. *et al.* Estimating individual contributions to population growth: evolutionary fitness in ecological time. *Proc. R. Soc. Lond. B* **273,** 547–555 (2006).
18. Sokal, R. R. & Rohlf, F. J. *Biometry* 3rd edn (Freeman, New York, 1995).
19. Kruuk, L. E. B. Estimating genetic parameters in natural populations using the 'animal model'. *Phil. Trans. R. Soc. Lond. B* **359,** 873–890 (2004).
20. Kruuk, L. E. B. *et al.* Heritability of fitness in a wild mammal population. *Proc. Natl Acad. Sci. USA* **97,** 698–703 (2000).
21. Houle, D. Comparing evolvability and variability of quantitative traits. *Genetics* **130,** 195–204 (1992).
22. Kruuk, L. E. B. *et al.* Antler size in red deer: heritability and selection but no evolution. *Evolution Int. J. Org. Evolution* **56,** 1683–1695 (2002).
23. Kokko, H. Fisherian and 'good genes' benefits of mate choice: how (not) to distinguish between them. *Ecol. Lett.* **4,** 322–326 (2001).
24. Pischedda, A. & Chippindale, A. K. Intralocus sexual conflict diminishes the benefits of sexual selection. *PLoS Biol.* **4,** e356 (2006).
25. Clutton-Brock, T. H., Guinness, F. E. & Albon, S. D. *Red Deer: Behavior and Ecology of Two Sexes* (Univ. of Chicago Press, Chicago, 1982).
26. Kruuk, L. E. B., Clutton-Brock, T. H., Rose, K. E. & Guinness, F. E. Early determinants of lifetime reproductive success differ between the sexes in red deer. *Proc. R. Soc. Lond. B* **266,** 1655–1661 (1999).
27. Coulson, T., Albon, S., Guinness, F., Pemberton, J. & Clutton-Brock, T. Population substructure, local density, and calf winter survival in red deer (*Cervus elaphus*). *Ecology* **78,** 852–863 (1997).
28. Marshall, T. C. *et al.* Estimating the prevalence of inbreeding from incomplete pedigrees. *Proc. R. Soc. Lond. B* **269,** 1533–1539 (2002).
29. Gilmour, A. R., Gogel, B. J., Cullis, B. R., Welham, S. J. & Thompson, R. *ASReml User Guide 1.0.* (VSN International, Hemel Hempstead, 2002).

Supplementary Information is linked to the online version of the paper at www.nature.com/nature.

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Author Contributions The idea for this study originated from discussions between L.E.B.K., B.C.S. and K.F. The data used stem from a long-term study run by T.H.B.C., with involvement from L.E.B.K. and J.M.P.; J.M.P. was also responsible for the molecular paternity analysis. K.F. conducted all data analyses and drafted the manuscript. L.K. helped with the quantitative genetics analyses, and T.C. helped in applying the de-lifing approach. All authors discussed the results and commented on the manuscript.

Author Information Reprints and permissions information is available at npg.nature.com/reprintsandpermissions. The authors declare no competing financial interests. Correspondence and requests for materials should be addressed to K.F. (foerster@orn.mpg.de) or L.E.B.K. (loeske.kruuk@ed.ac.uk).

METHODS

De-lifing. To measure fitness we used the de-lifing approach[17], a jack-knifing procedure to calculate an individual's contribution to changes in population size, $p_{t(i)}$. $p_{t(i)}$ was suggested as an appropriate fitness measure for stochastic environments[17,30] such as those encountered by red deer. In Supplementary Information we discuss some relevant properties of $p_{t(i)}$ in comparison with lifetime reproductive success, a more traditional fitness estimate. Here we calculated

$$p_{t(i)} = \frac{s_{t(i)} - \overline{s}_t}{N_t - 1} + \frac{f_{t(i)} - \overline{f_t}}{N_t - 1}$$

for individual i in year t, where $s_{t(i)}$ is a binary variable representing whether individual i survives from year t to $t+1$, and $f_{t(i)}$ is 0.5 times the number of offspring produced by individual i in year t that survive to year $t+1$. $\overline{s_t}$ and $\overline{f_t}$ are the means of $s_{t(i)}$ and $f_{t(i)}$, and N_t is the adult population size (males and females aged at least 3 years) in year t. Individuals with negative values of $p_{t(i)}$ did worse than the population mean, whereas individuals with positive $p_{t(i)}$ performed above average. $p_{t(i)}$ estimates the relative contribution of an individual to population growth in each year and thus allowed us to include incomplete life histories. We also present results based on analyses of the two separate components of the above equation: an individual's annual survival

$$S = \frac{s_{t(i)} - \overline{s}_t}{N_t - 1}$$

and its annual reproduction

$$R = \frac{f_{t(i)} - \overline{f_t}}{N_t - 1}$$

For a single lifetime measure of fitness (only for individuals with complete life history information, excluding shot deer or deer that are still alive), we used the sum of all annual $p_{t(i)}$ values, referred to as the lifetime sum of $p_{t(i)}$. We present P values from two-tailed tests with $\alpha = 0.05$.

Animal model analyses. We partitioned phenotypic trait variance into additive genetic variance, maternal variance, permanent environment variance, and residual variance by using the animal model as implemented by the software ASReml[29]. The animal model uses pedigree information to extract the additive genetic component in a mixed-model framework based on restricted maximum-likelihood (REML) estimation[19]. The pedigree of the red deer on Rum included 3,559 animals from eight generations. We modelled $p_{t(i)}$, R and S as repeated annual measures and chose fixed effects that have been shown to influence reproductive success in this population. For both sexes we included the year of birth to account for cohort effects[26], and the individual's age as a quadratic function[22]. For females only, we added population subdivision (to account for variation in habitat quality)[27] and recent reproductive history (whether or not the female had reared a calf the previous year and whether or not that calf survived for six months)[31].

We ran univariate animal models separately for males and females, to calculate narrow-sense heritability (h^2) and to obtain starting values for the bivariate analysis. We estimated the significance of h^2 as the probability that the additive genetic variance component was greater than zero by using a log-likelihood ratio test. We then applied bivariate animal models with the male and the female trait as two dependent variables and estimated the significance of genetic and maternal covariances by comparing the residual deviance of the final model with that of a model with a fixed covariance of 0 in a log-likelihood ratio test. We multiplied $p_{t(i)}$, R and S by 10,000 to enable model convergence, and all reported model parameters are based on transformed values.

Breeding values. An individual's breeding value for a given phenotypic trait is the total additive effect of its genes on that trait[4]. In an animal model, the breeding value is estimated as an individual's best linear unbiased predictor (BLUP) for the additive genetic effect. It is determined by the deviation of the individual's own phenotype and those of all its relatives, scaled by their relatedness to the given individual, from the population mean[19]. For all animals in which we observed $p_{t(i)}$ in opposite-sex relatives, we were able to predict a breeding value for $p_{t(i)}$ in the opposite sex. To investigate selection on opposite-sex breeding values of $p_{t(i)}$, we reran the univariate analyses as linear mixed models without the additive genetic effect and included an individual's breeding value for $p_{t(i)}$ in the opposite sex as a fixed effect.

30. Metcalf, C. J. E. & Pavard, S. Why evolutionary biologists should be demographers. *Trends Ecol. Evol.* **22**, 205–212 (2007).
31. Clutton-Brock, T. H., Guinness, F. E. & Albon, S. D. The costs of reproduction to red deer hinds. *J. Anim. Ecol.* **52**, 367–383 (1983).