directional selection in the lineage, selection did not appear to affect the observed morphological changes, suggesting that this method is biased against finding evidence for directional change (*18*). For fossils, the number of generations between successive samples is generally so high that the amount of morphological change would have to be unrealistically high for directional selection to be detected with current methods.

Our research compared trends for the same taxon in different geographical locations and identified a possible environmental influence on evolutionary modes and rates. We found that the two current quantitative methods used for delineating evolutionary mode provide equivalent results but that the likelihood-based test can be more useful when comparing sequences with fewer time (stratigraphic) intervals. Our analysis demonstrates that, for the same suite of characters over similar time periods, all three modes of evolution (directional, random, and stasis) are found within the genus *Buchia* throughout its geographic range, indicating that there is an environmental component to mode. More specifically, we found that there may be a relation between mode and paleolatitude, because random evolutionary trajectories were all found at the highest paleolatitudes (Table 1), but this must be confirmed by studying buchiids at a wider range of paleolatitudes. Our results also suggest that, in *Buchia*, stasis occurs more frequently in deep-water marine environments.

References and Notes

1. P. D. Roopnarine, *Paleobiology* **27**, 446 (2001).
2. G. Hunt, *Paleobiology* **32**, 578 (2006).
3. M. Lynch, *Am. Nat.* **136**, 727 (1990).
4. B. Hannisdal, *Paleobiology* **33**, 98 (2007).
5. J. A. Jeletzky, *Geol. Surv. Can. Bull.* **103** (1965).
6. J. Sha *et al.*, *Cretac. Res.* **24**, 715 (2003).
7. F. Surlyk, V. A. Zakharov, *Palaeontology* **25**, 727 (1982).
8. I. I. Sey, E. D. Kalacheva, *Palaeogeogr. Palaeoclimatol. Palaeoecol.* **150**, 49 (1999).
9. J. B. C. Jackson, A. H. Cheetham, *Trends Ecol. Evol.* **14**, 72 (1999).
10. H. D. Sheets, C. E. Mitchell, *Genetica* **112-113**, 105 (2001).
11. G. Hunt, *Proc. Natl. Acad. Sci. U.S.A.* **104**, 18404 (2007).
12. Materials and methods are available as supporting material on *Science* Online.
13. M. Grey, J. W. Haggart, P. L. Smith, *Palaeontology* **51**, 583 (2008).
14. Canonical variate analysis (CVA) determines which variables discriminate between groups of previously identified specimens and is also used for predictive classification. A priori grouping for the CVA was based on the stratigraphy (a proxy for time) of each specimen; this allowed changes in morphology to be tracked through relative time (e.g., a specimen found in a higher part of the geologic section is geologically "younger" than one found in the lower part of the section).
15. P. D. Roopnarine, *Annu. Rev. Ecol. Evol. Syst.* **34**, 605 (2003).
16. P. D. Gingerich, *Science* **222**, 159 (1983).
17. D. N. Reznick, F. H. Shaw, H. Rodd, R. G. Shaw, *Science* **275**, 1934 (1997).
18. M. A. Bell, M. P. Travis, D. M. Blouw, *Paleobiology* **58**, 814 (2006).

19. Supported by a Natural Sciences and Engineering Research of Canada (NSERC) grant (to P.L.S.) and an NSERC PGS-A scholarship (to M.G.). We are grateful to P. Alsen, T. Bogdanova, A. Crame, J. S. Crampton, J. Dougherty, J. Grant-Mackie, D. A. T. Harper, D. Hikuroa, N. Hudson, E. Kalacheva, J. Rassmussen, J. Sha, I. Sey, and F. Surlyk for use of their collections and hospitality; to P.G. Lelièvre for creating the MatLab morphometrics program (MorphLab 1.0); and to G. Hunt and P. Roopnarine for help and program codes for analyzing mode. This manuscript was substantially improved by the comments and suggestions of three reviewers.

Supporting Online Material

www.sciencemag.org/cgi/content/full/1162046/DC1
Materials and Methods
SOM Text
Figs. S1 to S5
Tables S1 and S2
References

19 June 2008; accepted 14 October 2008
Published online 23 October 2008;
10.1126/science.1162046
Include this information when citing this paper.

Selfish Genetic Elements Promote Polyandry in a Fly

T. A. R. Price,[1] D. J. Hodgson,[1] Z. Lewis,[2] G. D. D. Hurst,[3] N. Wedell[1]*

It is unknown why females mate with multiple males when mating is frequently costly and a single copulation often provides enough sperm to fertilize all a female's eggs. One possibility is that remating increases the fitness of offspring, because fertilization success is biased toward the sperm of high-fitness males. We show that female *Drosophila pseudoobscura* evolved increased remating rates when exposed to the risk of mating with males carrying a deleterious sex ratio–distorting gene that also reduces sperm competitive ability. Because selfish genetic elements that reduce sperm competitive ability are generally associated with low genetic fitness, they may represent a common driver of the evolution of polyandry.

Why females of nearly all animals tend to mate with more than one male (polyandry) remains a pressing question for evolutionary biology (*1*, *2*). However, the driving forces behind the origin and maintenance of polyandry are uncertain, because for many species, the costs of mating multiply often appear to outweigh the benefits (*3*–*5*). Polyandry may benefit females by increasing the fitness of their offspring (*6*, *7*). For example, in polyandrous systems, females could remate with more attractive males, increasing their likelihood of producing sons with higher mating success (*8*). Polyandry also allows females control over paternity through sperm competition [the competition for fertilization of ova that occurs between sperm from more than one male (*2*)]. If paternity is biased toward the sperm of males of higher viability, then a multiply mating female will on average have higher-fitness offspring than a monogamous female (*7*, *9*). In crickets, for example, polyandrous females have a higher hatching success when they bias paternity against more closely related males (*10*). It is also possible that selfish genetic elements promote polyandry by creating a correlation between male fitness and sperm competitive ability (*11*, *12*).

Selfish genetic elements spread through populations because they subvert normal patterns of inheritance in ways that increase their representation in the next generation, often at a cost to the bearer. They are ubiquitous in living organisms, where they can make up a large part of the genome (*11*). Several selfish genetic elements, such as meiotic drive elements, B chromosomes, and endosymbionts, are associated with reduced male fertility, often due to direct manipulation of spermatogenesis (*13*), and female fitness is frequently reduced when females mate with males that carry these elements (*11*). Where males harboring such selfish genes suffer reduced paternity in sperm competition, theory predicts that this will favor increased polyandry in females, because remating will reduce exposure through biased fertilization against gametes carrying these elements (*12*).

To test the hypothesis that the presence of selfish genetic elements may select for increased rates of female remating, we examined the evolutionary response of remating rate to the presence of the selfish gene SR (sex ratio), an X-chromosome meiotic driver in the fruit fly *Drosophila pseudoobscura* (*14*, *15*). This gene is common in populations of *D. pseudoobscura*. It has little consistent effect in females, but in males it causes the developmental failure of all sperm bearing a Y chromosome. Consequently, male carriers produce fewer sperm and sire only daughters (*14*), frequently resulting in female-biased population sex ratios (*16*). There is no genetic resistance to sex ratio drive in *D. pseudoobscura* (*17*). Females have higher fitness if they avoid mating with SR males due to higher sperm numbers in non-SR males, which sire offspring of both sexes and whose male offspring have a full complement of sperm. Yet, females cannot distinguish between SR and non-SR males before

[1]School of Biosciences, University of Exeter, Cornwall Campus, Penryn TR10 9EZ, UK. [2]Graduate School of Environmental Science, Okayama University, Okayama 700-8530, Japan. [3]School of Biological Sciences, University of Liverpool, Liverpool L69 7ZB, UK.

*To whom correspondence should be addressed. E-mail: n.wedell@exeter.ac.uk

mating (*15*). However, the loss of half of the developing sperm (Y-chromosome that do not carry SR) reduces the number of sperm that SR males transfer to females (*18*), which makes them poor sperm competitors relative to unaffected males (*18*). Given the poor performance of SR males in sperm competition, we predict that in populations containing SR sperm, females should evolve high rates of remating to promote sperm competition and thus reduce the average paternity of SR males.

Female *D. pseudoobscura* flies collected from Arizona showed significant variation in remating rate between isofemale lines (descendants of a single wild-caught female) (see supporting online methods; median test for remating day differences between F_2 daughters of isofemale lines: $N = 100$, $\chi^2_{20} = 31.8$, $P = 0.045$; range of mean day of remating: 2 to 5 days), demonstrating that this population contained variation in remating rates that selection could act upon. Four selection lines carried SR at an initial frequency of 30% and were maintained at a 2:1 female-to-male sex ratio, as observed in many natural SR-carrying populations (*16*). Four lines lacked a SR element and had a 1:1 sex ratio, representing natural non-SR populations. An additional four lines lacked SR but were experimentally maintained at a 2:1 sex ratio, to control for any potential effect of sex ratio on the evolution of female remating rate. Before setting up the populations, we examined the remating propensities of the SR and non-SR isolines and found that they did not differ significantly ($N = 28$, $\chi^2_1 = 0.015$, $P = 0.902$), thus ensuring there was no initial difference in female remating rate between the SR and non-SR flies from which the selection lines were formed. We also used simulations to confirm that the experimental setup produced an unbiased sample of female mating rates between treatments in the first experimental generation [supporting online material (SOM)].

After 10 generations of experimental evolution, we assayed remating rates within the selection lines by counting the number of females that mated on their second exposure to males (SOM) (Fig. 1). In a second assay at generation 11, we individually mated virgin females from each line to standard, stock non-SR males and then recorded the time until remating occurred (SOM) (Figs. 2 and 3). Both methods of assessment showed that females from SR-carrying lines were more likely to remate at the first opportunity than females from the other treatments (first assay: $\chi^2_2 = 15.279$ $P = 0.0005$; second assay: $\chi^2_2 = 6.524$, $P = 0.0383$), and that there was a shorter overall time to remating (second assay $\chi^2_2 = 17.731$, $P = 0.0001$). Thus, evolution of increased remating rates in the presence of SR was both rapid and dramatic, with females doubling their likelihood of remating at the first opportunity compared to females from non-SR lines, and reducing the mean number of days to remating from 3.25 to 2.75 in only 10 generations of experimental evolution.

The presence of SR chromosomes was associated with an increased willingness of females to remate, as predicted by theory (*12*). Three alternative explanations for the increase in remating—a male effect, phenotypic effect, and inbreeding—were rejected. First, the increase in female remating rates could not be due to differences in the ability of males from different lines to acquire copulations and prevent female remating (either randomly or due to the presence of the SR chromosome) because the effect was still observed when females were exposed to tester males from an unselected non-SR stock population. Second, it is unlikely that increased female remating rates were caused by a phenotypic effect of the SR chromosome when expressed in females, because the frequency of SR was less than 5% in each line when the assays were conducted. Ad-

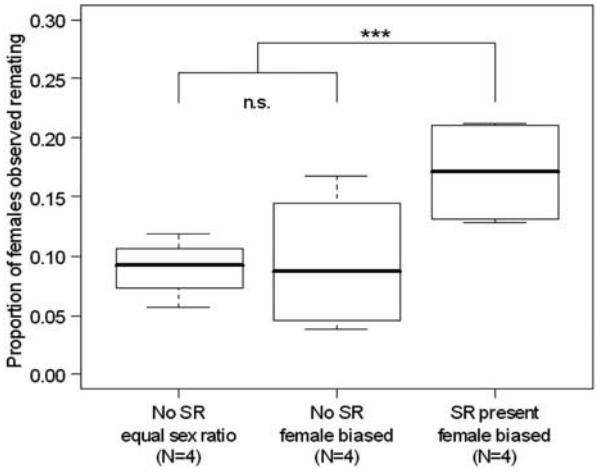

Fig. 1. The proportion of females observed remating at generation 10, for each selection regime (N = 12 lines), showing median, interquartile range, and range. ***P, 0.0005; n.s., not significant.

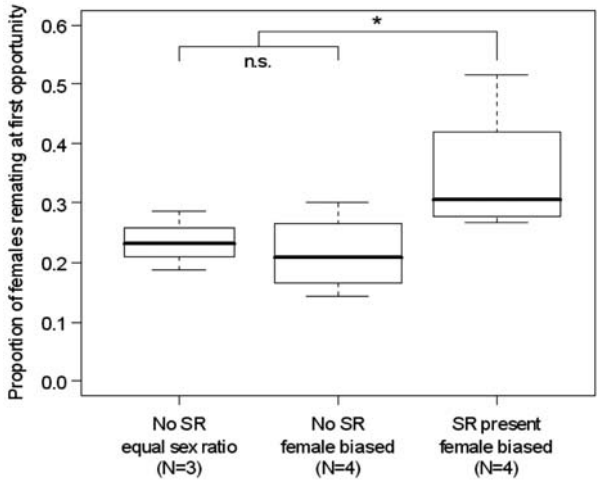

Fig. 2. The proportion of selection line females remating at their first opportunity, for each selection regime (N = 11 lines), when offered standardized stock males, showing median, interquartile range, and range. *P, 0.0383; n.s., not significant.

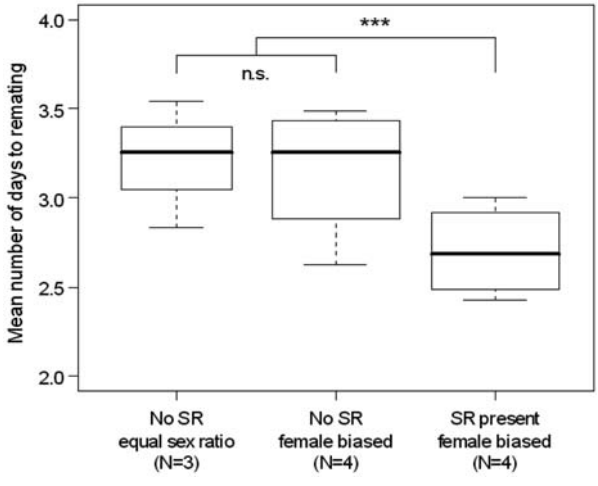

Fig. 3. The mean number of days to remating, for each selection regime (N = 11 lines), when females mated to standardized stock males, showing median, interquartile range, and range. ***P, 0.0001; n.s., not significant.

ditionally, no influence of SR alleles on female remating behavior has been shown (*15*). Third, we doubt that the remating rate increase was driven by SR populations being more inbred and females avoiding inbreeding depression (*10*), because our experimental populations of 120 adults per generation were large enough to avoid appreciable inbreeding (*19*). There also was no a priori reason to expect a greater inbreeding effect in the female-biased SR populations compared to the non-SR female–biased populations.

The most parsimonious explanation for the evolution of increased female remating rates in the presence of SR is therefore that the direct benefits of decreased risk of sperm limitation due to mating with SR males, combined with the benefit of reduced exposure of the progeny of polyandrous females to SR, has driven the spread of alleles for increased remating rates through the populations. The observed increases in female remating rates (2.75 versus 3.25 days) are well within the natural variation of the source population (2 to 5 days). Thus, we conclude that increased female remating rates evolved through selection for alleles that promote polyandry in SR populations.

Avoidance of selfish genetic elements is likely to promote the evolution of polyandry, wherever a selfish genetic element reduces both the sperm competitive ability of carrier males and the fitness of progeny that inherit the gene (*7*, *12*). Sex chromosome meiotic drive elements are likely to considerably reduce male fertility because their transmission mode involves a substantial failure of spermatogenesis (*13*, *14*). They also impose serious costs because they destroy sperm and distort brood sex ratios (*14*). Sex chromosome drive is widespread (*20*) and may be common (*21*, *22*). Most segregation distorters are active in males (*23*), and reduced sperm production associated with both X- and Y-chromosome drive has been observed (*13*). Similarly, males may suffer reduced fertility from other selfish genes, such as autosomal drive genes, B chromosomes (*24*), intracellular parasites (*25*), and possibly some transposons (*26*). Thus, a wide range of selfish genetic elements potentially provide the critical combination of low sperm competitive ability and low fitness that could favor polyandry. Because such selfish elements are ubiquitous in living organisms and frequently compromise male fertility, they may provide a generally overlooked explanation for why polyandry is very widespread.

References and Notes

1. T. R. Birkhead, A. P. Møller, *Sperm Competition and Sexual Selection* (Academic Press, London, 1998).
2. L. W. Simmons, *Sperm Competition and Its Evolutionary Consequences in the Insects* (Princeton Univ. Press, Princeton, NJ, 2001).
3. G. Arnqvist, L. Rowe, *Sexual Conflict* (Princeton Univ. Press, Princeton, NJ, 2005).
4. T. Chapman, L. F. Liddle, J. M. Kalb, M. F. Wolfner, L. Partridge, *Nature* **373**, 241 (1995).
5. H. Crudgington, M. Siva-Jothy, *Nature* **407**, 855 (2000).
6. M. D. Jennions, M. Petri, *Biol. Rev. Camb. Philos. Soc.* **75**, 21 (2000).
7. J. A. Zeh, D. W. Zeh, *Proc. R. Soc. London Ser. B* **264**, 69 (1997).
8. M. Olsson, R. Shine, T. Madsen, A. Gullberg, H. Tegelström, *Nature* **383**, 585 (1996).
9. D. J. Hosken, T. W. J. Garner, T. Tregenza, N. Wedell, P. I. Ward, *Proc. R. Soc. London Ser. B* **270**, 1933 (2003).
10. T. Tregenza, N. Wedell, *Nature* **415**, 71 (2002).
11. A. Burt, R. Trivers, *Genes in Conflict: The Biology of Selfish Genetic Elements* (Harvard Univ. Press, Cambridge, MA, 2006).
12. D. Haig, C. T. Bergstrom, *J. Evol. Biol.* **8**, 265 (1995).
13. T. A. R. Price, N. Wedell, *Genetica* **132**, 295 (2008).
14. J. Jaenike, *Annu. Rev. Ecol. Syst.* **32**, 25 (2001).
15. J. Powell, *Progress and Prospects in Evolutionary Biology: The Drosophila Model* (Oxford Univ. Press, Oxford, 1997).
16. S. H. Bryant, A. T. Beckenbach, G. Cobbs, *Evolution* **36**, 27 (1982).
17. D. Policansky, B. Dempsey, *Evolution* **32**, 922 (1978).
18. T. A. R. Price *et al.*, *Evolution* **62**, 1644 (2008).
19. W. Rice, B. Holland, *Evolution* **59**, 682 (2005).
20. L. D. Hurst, A. Pomiankowski, *Genetics* **128**, 841 (1991).
21. J. Jaenike, *Am. Nat.* **148**, 237 (1996).
22. F. Jiggins, G. Hurst, M. Majerus, *Am. Nat.* **154**, 481 (1999).
23. D. R. Taylor, P. K. Ingvarsson, *Genetica* **117**, 27 (2003).
24. L. W. Beukeboom, *Evol. Ecol.* **8**, 1 (1994).
25. F. E. Champion de Crespigny, N. Wedell, *Proc. R. Soc. London Ser. B* **273**, 1455 (2006).
26. T. K. Rajendra, K. V. Prasanth, S. C. Lakhotia, *J. Genet.* **80**, 97 (2001).

27. Isofemale lines were collected with assistance from T. Markow. We thank J. Blount, F. E. Champion de Crespigny, D. J. Hosken, and T. Tregenza for discussion. Funded by a Natural Environment Research Council grant (N.W. and G.H.).

Supporting Online Material

www.sciencemag.org/cgi/content/full/322/5905/1241/DC1
Materials and Methods
References

25 July 2008; accepted 15 October 2008
10.1126/science.1163766

Regulation of Microtubule Dynamics by Reaction Cascades Around Chromosomes

Chaitanya A. Athale, Ana Dinarina, Maria Mora-Coral, Céline Pugieux, Francois Nedelec, Eric Karsenti*

During spindle assembly, chromosomes generate gradients of microtubule stabilization through a reaction-diffusion process, but how this is achieved is not well understood. We measured the spatial distribution of microtubule aster asymmetry around chromosomes by incubating centrosomes and micropatterned chromatin patches in frog egg extracts. We then screened for microtubule stabilization gradient shapes that would generate such spatial distributions with the use of computer simulations. Only a long-range, sharply decaying microtubule stabilization gradient could generate aster asymmetries fitting the experimental data. We propose a reaction-diffusion model that combines the chromosome generated Ran–guanosine triphosphate–Importin reaction network to a secondary phosphorylation network as a potential mechanism for the generation of such gradients.

In eukaryotic cells, chromosomes regulate spindle assembly by generating a gradient of Ran–guanosine triphosphate (RanGTP) in their vicinity (*1*–*5*). In frog eggs and egg extracts, it has been shown that this gradient triggers the nucleation of spindle microtubules (MTs) by activating the protein TPX2 (*6*) and stabilizes the plus ends of centrosomal MTs by activating the kinase CDK11 (*7*). When centrosomes are incubated together with chromatin stripes or beads in those extracts, the centrosomal asters are asymmetric, sending longer microtubules preferentially toward chromatin, presumably because of their increased stability in this region (*8*, *9*). However, the exact distribution of the CDK11-dependent MT stabilization activity and how this could translate into a defined asymmetry of centrosomal asters in the vicinity of chromosomes has remained unclear.

To visualize the shape of the stabilization gradient, we designed an experimental system allowing the precise measurement of centrosomal MT asymmetry as a function of centrosome distance from chromatin (*10*). We immobilized chromatin beads on patches of defined sizes and distributions and incubated them in *Xenopus* egg extracts containing purified human centrosomes (Fig. 1, A and B). The chromatin patches nucleated MTs actively, and spindles assembled robustly on practically all patches. In the experiments designed to assay aster asymmetry, we added anti-TPX2 antibodies to the extract to prevent chromatin mediated MT nucleation around the beads and their interaction with centrosome-nucleated MTs (*7*). In this assay, centrosomal MTs displayed a radial symmetric distribution when far away from chromatin patches and became asymmetric when closer, whereas no obvious interactions between astral microtubules and the beads could be detected (Fig. 1B).

In parallel to this experimental setup, we developed a simple generic model to carry out com-

Cell Biology and Biophysics Unit, European Molecular Biology Laboratory (EMBL), Meyerhofstrasse 1, Heidelberg, Germany.

*To whom correspondence should be addressed. E-mail: karsenti@embl.de