# One Function of Sex — An Empirical Study of Genetic and Ecological Variation

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Forum[1]

# One function of sex — an empirical study of genetic and ecological variation

**N. Gilbert, D.A. Raworth, G.R. Allen**

**Abstract**—The question ''why sex?'' is a longstanding fundamental puzzle in biology. Although there may be several answers, no satisfactory theory has emerged. We present an argument in favour of one function of sex, derived from a study of the population genetics and ecology of the cabbage butterfly, *Pieris rapae* (L.) (Lepidoptera: Pieridae), on three continents between 1984 and 2009, and from previously published studies of other organisms. We provide evidence that responsiveness to directional selection (RDS), a measure related to ''narrow-sense heritability'', can be dramatically reduced by truncation selection in a single generation and rapidly restored within a few generations. Viewing a population as a collection of sexual families, we show that rapid restoration of RDS after truncation selection is essential to maintain population variance. The only known mechanism that will rapidly restore RDS is sexual recombination. We therefore conclude that in *P. rapae*, sex restores the genetic variation that a population needs to match unpredictable environmental variation, despite selection tending to reduce that genetic variation.

**Résumé**—La question de savoir à quoi sert la sexualité demeure depuis longtemps une énigme fondamentale de la biologie. Bien qu'il puisse y avoir plusieurs réponses, il n'est apparu aucune théorie satisfaisante. Nous présentons un argument qui appuie une des fonctions de la sexualité tiré d'une étude de la génétique de population et de l'écologie de la piéride du chou, *Pieris rapae* (L.) (Lepidoptera : Pieridae), sur trois continents, de 1984 à 2009, ainsi que d'études antérieures sur d'autres organismes. Nous apportons des données qui indiquent que capacité de réagir à une sélection directionnelle (RDS), une mesure reliée à l'« héritabilité dans le sens strict », peut être réduite de façon spectaculaire par une sélection par troncature en une seule génération et restaurée en quelques générations. Considérant la population comme un ensemble de familles sexuelles, nous montrons que la restauration rapide de la RDS après une sélection par troncature est

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[1]Papers published in the Forum of *The Canadian Entomologist* are intended to stimulate debate about general principles in insect biology. The Forum provides an opportunity to suggest new hypotheses, challenge current thinking on issues, discuss new ideas or ways of interpreting existing information, or respond to Forum topics previously presented in *The Canadian Entomologist*.
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essentielle pour maintenir la variance de la population. Le seul mécanisme connu qui peut restaurer rapidement la RDS est la recombinaison sexuelle. Nous concluons donc que, chez *P. rapae*, la sexualité restaure la variation génétique dont la population a besoin pour contrer la variation environnementale imprévisible, malgré que la sélection tende à réduire la variation génétique.

[Traduit par la Rédaction]

## Introduction

The question "why sex?" dates back to at least the days of Darwin, and there are many theories that address it (Bell 1982; Barton and Charlesworth 1998). Although there may be several answers to the question, no satisfactory theory has emerged (Ghiselin 1974; Stearns 1990; Kondrashov 1993; Barton and Charlesworth 1998; Bell 2008).

It is well understood (*e.g.*, Fisher 1930) that in the absence of selection, Mendelian or particulate (as opposed to blending) inheritance will preserve genetic variance — but selection, whether truncation or centripetal, is expected to reduce it. Natural selection occurs in field populations (Endler 1986; Kingsolver *et al.* 2001), although the mechanism is often not known in detail. Here we present empirical genetic and ecological evidence that sex restores genetic variation in the face of environmental fluctuations that tend to reduce that variation — an idea suggested by others as early as 1860 (Daubeny 1861). We do not claim that this is *the* function of sex, but is, rather, *one* function, in the organism studied and within the specific ecological context.

Our approach stems largely from Morris (1971), who presented convincing evidence for climate-driven genetic changes in populations of fall webworm, *Hyphantria cunea* (Drury) (Lepidoptera: Arctiidae), over a large area in Eastern Canada. Our study focuses on the cabbage butterfly, *Pieris rapae* (L.) (Lepidoptera: Pieridae), which is amenable to ecological field studies (Jones and Ives 1979) and genetic studies of biologically important quantitative traits (Gilbert 1984*b*).

The paper is divided into six sections, each critical to the development of our argument for a function of sex. Section 1 develops the idea of responsiveness to directional selection (RDS), a measure of heritable quantitative (polygenic) variation that makes no assumption about individual gene effects. Section 2 uses pupal development times and pupal masses collected in selection experiments during several years to develop a simple graphical model that can explain changes in RDS in terms of changes in genotype. Section 3 reanalyses selection experiments from the literature to show that within-family variances resist centripetal selection, and given this phenomenon, the observed phenotypic responses can be explained in terms of RDS and Normal theory. Section 4 examines our empirical evidence for rapid changes in RDS in natural field populations. Section 5 describes field-cage experiments that provide empirical evidence for the proximal cause of decreases in RDS in the field. Finally, section 6 describes a stochastic simulation model that brings all aspects of the study together and shows the role of sexual recombination in restoring genetic variation.

## 1. Galton's rule and RDS

The standard approach to quantitative genetics is based on Fisher's (1918) model of additive gene effects (Falconer and Mackay 1996). Our research, however, is based on Galton's rule (Galton 1889), which is much simpler and requires no genetic assumptions. Here we justify our approach, define RDS, examine our assumptions, and provide biological details of the measurement.

Fisher (1918) settled a dispute between the biometricians and geneticists of that time by showing that observed correlations between relatives could be interpreted in terms of Mendelian genetics. For that purpose he introduced his model of additive gene effects, with limited interactions. Later students have applied and extended this model in ways that Fisher himself regarded as "rather optimistic" (R.A. Fisher, personal communication). As he noted, "the possibilities of Epistacy have only been touched upon" (Fisher 1918). For if all possible interactions are included, the additive model can reproduce any conceivable behaviour, because

the number of available parameters always exceeds the number of data. Analyses of population bottlenecks endorse his scepticism, for they show that so-called additive components of genetic variance must include important contributions from interactions (Bryant and Meffert 1996).

Consider a (fictitious) random-mating population with perfectly additive gene effects — no dominance, no epistacy. In that case, Galton's (1889) midparental rule — that the mean genetic value of all progeny of two parents will approximate the average of the genetic values of the two parents — is exact. Then Fisher's additive model, and Galton's rule, predict the same genetic correlations — 1/2, 1/4, etc. — between relatives of various degrees (Gilbert 1989). But analyses of quantitative genetics are based on the empirical values of those correlations. So the reason why additive models absorb a large fraction of genetic variance is, simply, Galton's rule. This rule is a mass-action model that makes no genetic assumption beyond equal parental contributions; it tells us nothing about effects of individual genes. There is no reason to suppose that so-called additive genetic variance measures the additive effects of genes.

All we reliably know about quantitative variation is the measured values of individual phenotypes, from which means, correlations, etc. may be estimated. Arguably, all we need to know for practical breeding work is the responsiveness to directional selection or RDS, defined as the mean difference between two progeny cohorts produced by two selected groups of parents, divided by the mean difference between those two parental groups themselves. Where mothers and fathers within one group differ, we invoke Galton's rule to justify using the midparental value to calculate the group mean. (Galton's rule strictly applies to genotypic values, but in practice works well — but not perfectly — for phenotypic values too (Gilbert 1967).) Then RDS is closely related to the offspring/midparent regression of conventional quantitative genetics.

Any linear regression is most accurately estimated, for a given total number of observations, from data close to each end of the regression line, thus firmly anchoring each end. In genetics this requires "positive assortative mating" (Reeve 1953) of the chosen parents, which means, in the laboratory, selecting males and females with the largest values of a quantitative trait and mating them together, and the smallest to the smallest. Then RDS coincides numerically with "narrow-sense heritability" ($Va/Vp$) (Falconer and Mackay 1996), because both are the offspring/midparent regression coefficient. But here we shall use RDS as a purely statistical measure of responsiveness to selection, unconcerned with problems of genetic interpretation: the measured regression need not coincide with the heritability ascribable solely to additive gene effects, when interactions occur (Reeve 1953). It is still advisable to ensure that the offspring/midparent regression is close to linear, since otherwise the value of RDS will vary according to the values of the chosen parents. Provided that the parental and progeny generations experience the same environmental conditions, estimates of RDS are as accurate as any other regressions, as indicated by their standard errors (SE).

We measured RDS of pupal development time (*i.e.*, days from pupation to adult emergence) at 25 °C, which varies between 5.5 and 8 days; 85% of the variance in this trait must be genetic (Appendix A). Development times of larvae and pupae are genetically correlated — selection of either stage produces corresponding responses in both (Gilbert 1986) — but the pupal stage is least affected by host-plant quality. Laboratory experiments with caged plants have shown that differences in plant quality account for 9% of phenotypic variance in larval development time and only 4% of that in pupal development time (21 df).

In the field, it is not feasible to measure development times of an adequate sample of butterfly pupae at defined temperatures. We therefore measured RDS in the laboratory, not of field populations as such, but of their immediate descendents at Cambridge, United Kingdom (52°12′N, 0°08′E); Hobart, Tasmania, Australia (42°53′S, 147°20′E); and Vancouver, British Columbia, Canada (49°15′N, 123°07′W). This requires special consideration of sample size, to ensure that the progeny of $n$ mated females caught in the field represent nearly all the quantitative genetic variance of the field population. The sample size of mated,

field-collected females was between 4 and 10 at Cambridge — but in 2006 only 2 were caught — and between 16 and 33 elsewhere (Appendix B). However, based on Galton's rule, a sample as small as 4 mated females will be adequate because it is expected to transmit 94% of the population genetic variance (Appendix C).

The following description provides a single measure of RDS and represents one "trial". Field-collected females, provided with honey solution in cotton-wool feeders, laid eggs on 8-week-old kale (*Brassica oleracea* L. var. *acephala* DC. 'Maris Kestrel'; Brassicaceae) plants. It has been established previously that all females caught in the field can lay fertile eggs, so the families of different females were not kept separate. From the eggs, a first-generation stock of 200 or more individuals was reared at 25 °C and 16L:8D. New pupae, collected every 6 h, were synchronized by storage at 4 °C for a maximum of 3 days. They were then reared at 25 °C, in constant light to nullify the circadian rhythm — adults normally emerge in early morning — and were checked at 6-h intervals for emergence. As parents for the second generation, the first-emerging 8–10 females were mass-mated with the first 6–8 males. Similarly, the last-emerging 8–10 females and 6–8 males were mated together. These extreme parents may have poor fitness, so when possible, the next earliest or latest emerging adults were mated as a backup. The mated females produced second-generation cohorts of 103.6 (range 41–401) progeny pupae per pooled mating, from which pupal development time and sex were determined as described above. RDS was then estimated by dividing the difference in pupal development times between the early and late progeny cohort means by the corresponding parental difference. This is the same as the offspring/midparent regression calculated from the same data. On average, female pupae develop faster than males, but all figures are balanced for sex assuming a 1:1 ratio; SEs for RDS include the separate variances for males and females in each cohort. On 11 occasions, individuals with intermediate pupal development times were selected and mated to check for linearity. RDS may then be estimated by (weighted) offspring/midparent regression, but the gain in accuracy is slight, unless one or other of the extreme progenies has failed badly.

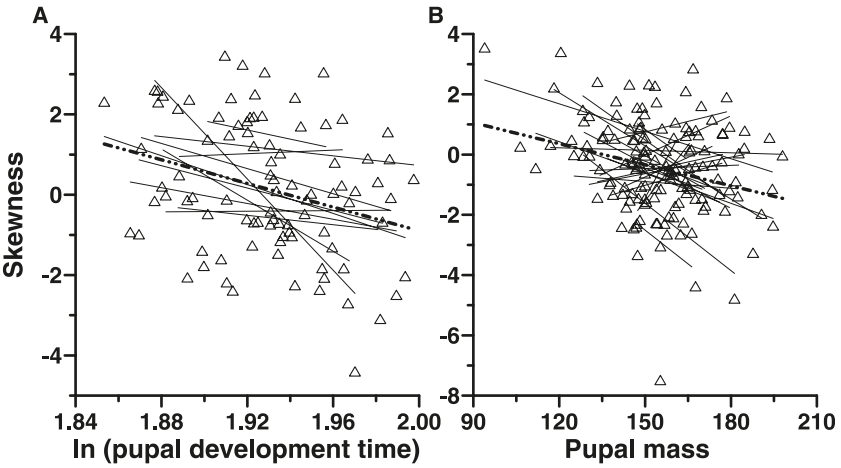
This paper explores aspects of quantitative variation, relying on the twin empiricisms of Galton's rule and RDS — but free of assumptions about gene effects. Before we examine the field and laboratory estimates of RDS we first describe two studies: one shows the expected response of RDS to truncation selection, and one demonstrates that RDS and Normal theory are sufficient to interpret observed changes in means and variances.

## 2. Selective limits and RDS

In natural populations the range of genetic variation of a quantitative trait is bounded by selective limits that block responses to selection beyond those limits. Individuals close to those limits often experience severe loss of fitness (Haldane 1954) (*i.e.*, there is centripetal or stabilizing selection). We expect that the selective limits will push the phenotypic expression $\overline{p}$ of extreme genotypes, $g$, inwards towards the centre of the range of variation, so that the distribution is compressed near each end of the range and skewed towards the centre. This is now verified in *P. rapae* for the trait of interest in this paper, viz. ln(pupal development time in days at 25 °C), and for pupal mass (mg), taken from a previous study, to corroborate the pattern.

We have analysed 11 independent "trials" consisting of 92 single-sex cohorts ($n > 100$ pupae in each of 43 cohorts and $n > 50$ pupae in each of 88 cohorts) produced by groups of parents variously selected for faster or slower pupal development (RDS measurement; section 1), and 23 independent "trials" consisting of 144 cohorts ($n > 100$ pupae in each of 34 cohorts and $n > 50$ pupae in each of 129 cohorts) from parents selected on the basis of pupal mass (Gilbert 1984*b*). Between 2 and 8 cohorts were selected over the range of variation per "trial", and males and females were treated separately so that each cohort produced two values of skewness. In every case the grandparents were freshly field-caught. No pupa was used for both measurements; total numbers were 9500 pupae for development time and 12 000 for mass. For each

**Fig. 1.** Overall within-"trial" regression (thick broken-dotted line) and individual within-"trial" regressions (thin lines) of Fisher's index of skewness in *Pieris rapae* cohort ln(pupal development time) (A) and pupal mass (B) *versus* the cohort mean of the respective variable.




cohort we calculated Fisher's index of skewness (Kendall and Stuart 1963):

$$[1] \quad \gamma = \left(\sum (\mathrm{X}-\mu)^3\right) \Big/ n\sigma^3$$

which gives reliable answers only for samples of 50 or more individuals, and preferably more than 100. Although females are, on average, lighter than males and develop faster, analysis of covariance (within "trials") shows no sex difference for skewness when corrected for cohort average development time or pupal mass, so the same selection limits apply to males and females. Then the within-"trial" regressions of skewness on cohort average development time and pupal mass are as follows:

$$[2] \quad \text{Skewness} = -14.73\,[\ln(\text{pupal development time}) - 1.93]$$
$$(\pm 4.87 \text{ SE}, 80 \text{ df}; \text{Fig. 1A})$$

$$[3] \quad \text{Skewness} = -0.0233(\text{pupal mass} - 155)$$
$$(\pm 0.00656 \text{ SE}, 120 \text{ df}; \text{Fig. 1B})$$

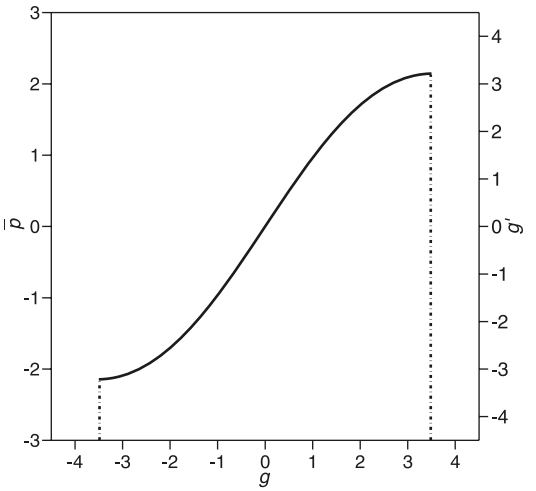
The SEs are estimated appropriately from variances between cohorts within trials. The values 1.93 and 155 (eqs. 2, 3) are close to the population means, so the regressions confirm that the distribution of phenotypes is positively skewed at the lower end of the range and negatively skewed at the high end. Therefore, the phenotypic variation is compressed at each end. But there is so much "noise" that it has taken 21 500 pupae to demonstrate the effect. Jinks *et al.* (1973) observed a similar pattern: phenotypic means for inbred lines were compressed from either end towards the centre of the range of their parental means. This pattern is independent of the detailed genetic interpretation that the authors advance.

The compression of phenotypic variation suggests that the relation of the phenotypic value $\overline{p}$ to the genotypic value, $g$, must look something like Figure 2: as selection for extreme genotypes continues, a reduced phenotypic response is expected at either end. The (arbitrary) curve shown is

$$[4] \quad \overline{p} = g \exp\left(-0.04g^2\right)$$

where $g$ is assumed, without loss of generality, to have a standard Normal (0,1) distribution. The exponent 0.04 is chosen so that $\overline{p}$ has maximum and minimum values — *i.e.*, selective limits — at $g = \pm 3.5$ standard deviations (SD). The correlation between $g$ and $\overline{p}$, integrated over the whole distribution, is 0.996, so it would be practically impossible to detect the

**Fig. 2.** Mean phenotypic value, $\overline{p}$, for individuals of *Pieris rapae* with the genotypic value, $g$, averaged over all possible environments; $\overline{p} = g\exp(-0.04g^2)$, where $g$ is the underlying genetic value, which is Normally distributed and satisfies Galton's rule; $g'$ is the expressed — and compressed — genetic value such that $p = g' + e$, and for each individual, $p$ is a measured phenotypic value and $e$ is an environmental or nongenetic term.

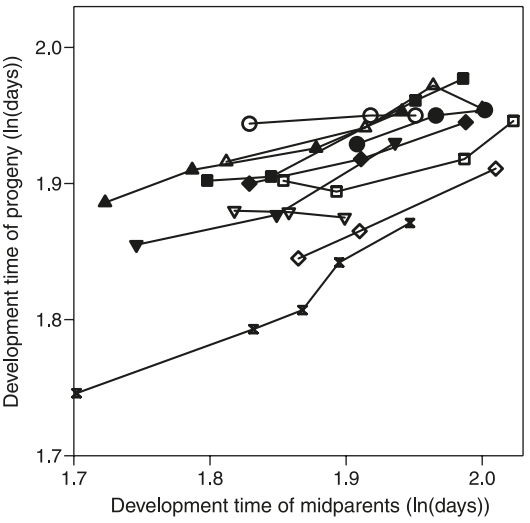



curvature of Figure 2 by direct measurement. In confirmation, Figure 3 shows several checks on linearity of the offspring/midparent relationship, where parents have been selected to produce intermediate as well as extreme cohorts (section 1). Offspring/midparent regressions appear more or less linear, with no direct hint of the compression at each end of the range.

Figure 2 is only a simplified illustration. After regressions [2] and [3] have been fitted, the remainder mean squares of the skewness indices are 2.42 (80 df) and 2.04 (120 df), respectively. If the remainder distributions were Normal, both remainder mean squares would be close to 1.0. The discrepancy indicates further departures from strict Normality, but not large enough to discredit ordinary statistical analyses.

In Figure 2 the slope of the curve decreases towards either end of the range. The regression of $\overline{p}$ on $g$, calculated by integration over the whole range of $g$, is 0.891. Over either 5% tail of the distribution it is halved to 0.453, reflecting the flattening at each end of the curve. Hence, RDS may reasonably be expected to

**Fig. 3.** Mean pupal development time of *Pieris rapae* progeny *versus* midparental pupal development time in laboratory ''trials''. Average differences between trials are ascribable to differences in temperature and host-plant qualities. Differences in slopes are differences in responsiveness to directional selection (RDS).




decrease as selection proceeds towards either end of the range of variation.

## 3. Reanalysis of selection experiments using RDS

Here we utilize RDS and Normal theory to reanalyse a number of selection experiments in the literature without making detailed genetic assumptions. We show that there is strong within-family resistance to centripetal selection, and if we accept that phenomenon, RDS and Normal theory are sufficient to interpret the observed changes of means and variances during selection.

In several published experiments (Table 1), RDS and phenotypic variance were measured at the start and end of several generations' artificial selection — which may be ''directional'' (parents chosen from one tail of the distribution) or ''stabilizing'', *i.e.*, centripetal (parents chosen from the centre of the range); many similar published experiments could not be used because essential information was not available. All authors used *Drosophila melanogaster* Meigen (Diptera: Drosophilidae), except

**Table 1.** Responses of traits of various insect taxa to laboratory selection.

| Source | Trait | No. of generations of selection | Fraction selected (%) | Initial RDS* | *Factg* † | *Facte*† |
|---|---|---|---|---|---|---|
| **Directional selection** | | | | | | |
| Scharloo *et al.* (1967) | Ratio of wing veins | 4 | 10 up | 0.65 ± 0.06 | 0.89 ± 0.043 | 0 |
| | | 5 | 10 down | 0.65 ± 0.06 | 0.90 ± 0.048 | 0 |
| Bos and Scharloo (1973) | Thorax length | 10 | 20 up | 0.63 ± 0.17 | — | — |
| | | 10 | 20 down | 0.63 ± 0.17 | See text | |
| **Centripetal selection** | | | | | | |
| Scharloo *et al.* (1967) | Ratio of wing veins | 5 | 10 | 0.71 ± 0.05 | 0.55 ± 0.09 | 0.45 |
| | | 36 | 10 | 0.65 ± 0.06 | 0.19 ± 0.02 | 0.09 |
| Bos and Scharloo (1973) | Thorax length | 19 | 20 | 0.53 ± 0.11 | 0.03 ± 0.08 | 0 |
| Kaufman *et al.* (1977) | Pupal mass | 94 | 15 | 0.22 ± 0.014 | 0.007 ± 0.0066 | 0 |
| Tantawy and Tayel (1970) | Wing length | 5 | 15 | 0.36 ± 0.03 | 0.30 ± 0.16 | 0 |

*Responsiveness to directional selection (RDS) was determined from experiments in the literature as $V_g/V_p$ (Appendix D).
†*Facte* and *factg* measure average responsiveness to the different types of selection on $V_e$ and $V_g$, respectively, averaged over several or many generations, and expressed as a fraction of the theoretical response predicted by Normal theory.

Kaufman *et al.* (1977), who used *Tribolium castaneum* (Herbst) (Coleoptera: Tenebrionidae). In every case except Tantawy and Tayel (1970), selection was made within replicate double families of about 40 progeny produced by two females and either one or two males, so selection was made almost entirely within families. In directional cases, selection was continued after the period shown in Table 1, and demonstrated that the limits to selection had already been approached. Most experiments began with stocks previously maintained in the laboratory for many generations, but Tantawy and Tayel (1970) and Bos and Scharloo (1973) used freshly caught field stocks. The traits concerned are measures of body size, which in *D. melanogaster* is correlated with fecundity, so in the absence of counterselection, there is steady natural selection for ever-increasing size. In *P. rapae*, the upper limit (section 2; Haldane 1954) blocks that selection (Gilbert 1984*a*). It may therefore be expected that average body size would already be fairly close to the upper selective limit, at least in field stocks. Exceptionally, Scharloo *et al.* (1967) measured an artificial trait, the ratio of the lengths of two adjacent wing veins in heterozygotes of a recessive lethal Mendelian mutant that interrupts one of those veins; so selection is not on that mutant itself, but on its expression in different genetic backgrounds. Those authors chose this ratio as a "selectively naïve" trait that had not previously experienced natural selection in the field; and as will appear, it responded quite differently to artificial selection.

In the experiments listed in Table 1, initial RDS and number of generations varied. To compare the various responses, we used two computer programs (Appendix D), one for each type of selection, that calculate generation by generation the expected effects of truncation selection, using Normal theory. It is assumed, that the phenotypic measurement, $p$, for each individual is the sum of a "genetic" term, $g$, with variance $V_g$ and an (uncorrelated) "environmental" term, $e$, with variance $V_e$, so $V_p = V_g + V_e$, and that values of $g$ and $e$ are Normally distributed. These are the standard simplifications of conventional statistical practice: genetic compression of $g$' (Fig. 2) is ignored. We postulated initially that $V_g$ would respond to selection and $V_e$ would not, in which case Normal theory predicts that RDS will equal $V_g/V_p$; but Scharloo *et al.*'s (1967) selectively naïve trait requires (Table 1) that both $V_g$ and $V_e$ respond, but at different rates. Therefore, in the programs the changing value of RDS in successive generations is multiplied

by a factor *factg* when selecting on $V_g$ and by *facte* when selecting on $V_e$. These two factors are adjusted iteratively until the calculation matches the observed final changes in $V_p$ and RDS (or, for directional selection, in $V_p$ and the mean value of $p$). The values of *factg* and *facte* that provide the closest match (Table 1) provide an index of the actual responses to selection, averaged over several generations, expressed as a fraction of the theoretical responses predicted by Normal theory. They convert the very heterogeneous data in the original publications to factors that permit a comparison of selective responses in different experiments.

Although the values of *factg* and *facte* in Table 1 are very variable, they conform to a pattern. We start with the selectively naïve trait "ratio of wing veins" (Table 1). For directional selection, *factg* is close to 1.0, as expected because initial RDS is measured by the same selection but in a single generation. If *factg* is actually less than 1.0, it reflects the approach to the upper and lower selective limits encountered shortly afterwards. *Facte* is zero, confirming that $V_e$ includes no variation capable of responding to directional selection that is not already accounted for by RDS. In this naïve trait, both $V_g$ and $V_e$ are reduced by centripetal selection, but (per generation) less than predicted by RDS in the first experiment with 5 generations, and much less in the second with 36. So $V_e$ included some genetic variation that did not respond to directional selection, but decreased under centripetal selection — Scharloo *et al.* (1967) call this "canalization". Perhaps it is the same selective process that produces the skewed compression already demonstrated in *P. rapae* (section 2).

The other traits listed in Table 1 are all measures of body size, and therefore correlated with fecundity. They can be expected to have previously experienced natural selection, possibly relaxed in captivity. It is impossible to estimate *factg* and *facte* for the directional selection of Bos and Scharloo (1973) (Table 1) because upwards selection produced little response, but downwards selection produced a large response in the mean and an increase in $V_p$. So thorax length in the original stock was already close to the upper limit, and the observed increase in $V_p$ after downwards selection may be ascribed either to a scaling problem or to compression of phenotypic variance close to the limit, as shown in Figure 2 but not recognised in this analysis.

In the remaining experiments, *facte* turns out to be zero, so $V_e$ contains no variation capable of responding to centripetal selection: this justifies, in these cases at least, the division of $p$ into $g + e$. *Factg* is very much less than 1.0, indicating that the actual response to centripetal selection is much less than that predicted by Normal theory. Therefore, $V_g$ is normally very resistant to centripetal selection within families; also, the longer that centripetal selection continues, the smaller *factg* becomes. This resistance to within-family selection is supported by selection experiments on the difference between male and female *P. rapae* (Appendix E); furthermore, intensive study of development rate in *D. melanogaster* could not explain why "selection does not expunge genetic variation in sexual populations" (Burke *et al.* 2010).

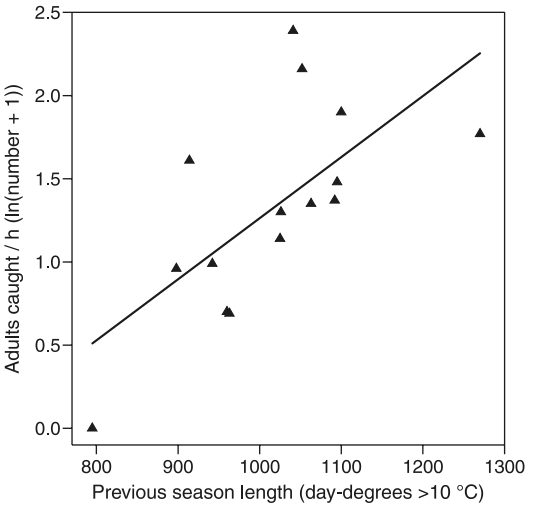
The outcome of these reanalyses is that RDS and Normal theory suffice to interpret observed changes in means and variances, provided we recognize that within-family variances resist centripetal selection. If we think in terms of RDS (which is directly measurable, but which itself changes as selection proceeds) rather than heritability, the assumptions required are reduced to those of ordinary parametric statistics. We now examine our measurements of RDS in the field and laboratory.

## 4. RDS in the field

In optimal conditions, *P. rapae* takes 350 day-degrees (dd) above 10 °C to develop from egg to adult (Jones and Ives 1979), but longer on tough field plants. Allowing extra time for mating, egg laying, and bad weather, two field generations require about 1000 dd. Individual development times vary by $\pm 10\%$, so we expect that in cool seasons of 1000 dd or less, only fast-developing individuals will complete two generations and enter diapause before the onset of winter. In that case, both population density and RDS (Fig. 2) should decrease.

Our studies were conducted between 1984 and 2009 at three locations: Cambridge, Hobart, and Vancouver. Season lengths are calculated in day-degrees above the development threshold

**Fig. 4.** Numbers of adult *Pieris rapae* caught per hour of sunshine *versus* length of the previous season; at Cambridge, May 1992–2009; $y = -2.4 + 0.0037x$, $r = 0.66$ (13 df).

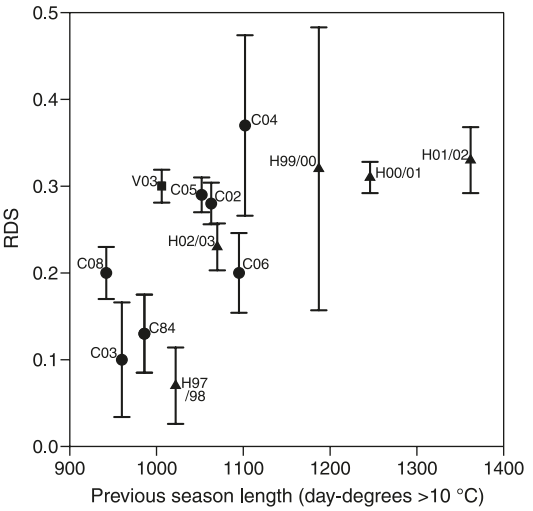



of 10 °C (Jones and Ives 1979) by the sine method (Raworth 1994) for the months when adults are active and monthly average temperatures exceed 10 °C: May–October inclusive at Cambridge and Vancouver and October–April inclusive at Hobart — the actual dates of first emergence and last entry into diapause are unknown.

A very crude estimate of the population density of overwintered adults in spring shows some correlation with the length of the previous year's season (Fig. 4). We take this as evidence that *P. rapae* suffers extra mortality during, or shortly after, short seasons. It does not prove selection against slow developers.

Figure 5 shows values of RDS measured not on the overwintered adults themselves, but on their immediate laboratory progeny (section 1). There is a significant correlation between RDS and the length of the previous season — with a critical season length of 1000 dd as expected. The ecological mechanism underlying this correlation, and the apparently anomalous value of RDS for Vancouver, will be considered in section 5. The most important feature of Figure 5 is that RDS is fully restored a few generations after it has been reduced in a short season (from C03 to C04 and from H97/98 to H99/00, though the latter may overestimate restoration time, as there was no RDS measurement for H98/99). Because RDS cannot be measured directly on first-generation field-collected adults, but only on their progeny, the rapid restoration of RDS means that the effect of truncation selection on RDS is presumably more severe than is indicated in Figure 5.

**Fig. 5.** RDS of the immediate progeny of overwintered *Pieris rapae* caught in the field (mean ± SE) *versus* length of the previous season. Comparisons are accurate between years within sites, but not between sites (C, Cambridge; H, Hobart; V, Vancouver; *e.g.*, "C08" refers to RDS for Cambridge in May 2008, with previous season length determined using 2007 temperatures. C84 parents were caught at Ascot, 100 km south of Cambridge but with a similar climate. For sites C and H, the within-sites regression of RDS on day-degrees, weighted inversely by the variance of RDS, is 0.000634 ± 0.0001706, $r = 0.78$ (9 df); this ignores the apparent curvature at site H.




The rapid restoration of RDS in the field is confirmed by laboratory measurements (Table 2A) that exclude the possible influence of immigration. In Table 2A, the parents of each successive laboratory generation were chosen to represent the whole phenotypic distribution of the previous generation, including both tails. In particular, both the mean and the variance were, as closely as possible, unchanged. When the parents for two successive laboratory generations were chosen ±1 SD from the mean (none central), so that the current mean and

**Table 2.** Responsiveness to directional selection of ln(pupal development time) at 25 °C in the first (spring) and second (summer) field generations of *Pieris rapae* and two laboratory generations following from the previous field stock.

| Location and year | First field generation | Second field generation | First laboratory generation | Second laboratory generation |
|---|---|---|---|---|
| (A) Parents for each successive laboratory generation chosen from full range of variation | | | | |
| Hobart | | | | |
| 1999−2000 | 0.32 ± 0.163 (30) | 0.48 ± 0.040 (694) | — | — |
| 2002−2003 | 0.23 ± 0.027 (768) | 0.43 ± 0.023 (666) | — | — |
| Cambridge | | | | |
| 2002 | 0.28 ± 0.024 (824) | — | 0.42 ± 0.037 (439) | 0.46 ± 0.036 (467) |
| 2003 | 0.10 ± 0.066 (597) | 0.28 ± 0.038 (1414) | 0.36 ± 0.055 (925) | 0.64 ± 0.046 (1264) |
| 2007 | — | 0.19 ± 0.032 (529) | 0.56 ± 0.053 (569) | 0.30 ± 0.033 (402) |
| (B) Parents for each successive laboratory generation chosen from ±1 SD of current mean | | | | |
| Cambridge, 2007 | 0.20 ± 0.046 (399) | — | 0.16 ± 0.042 (555) | 0.20 ± 0.031 (483) |

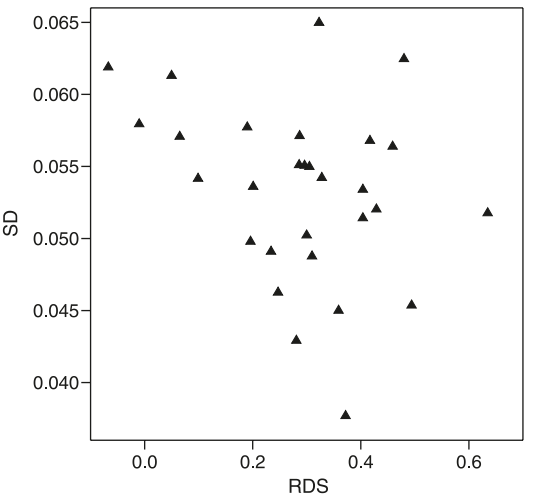
**Note**: Values are given as the mean ± SE, with degrees of freedom in parentheses.

variance were preserved but the tails of the distribution were excluded, there was no tendency for RDS to increase (Table 2B).

Following Normal theory, and given the equation $p = g + e$ with corresponding components of variance $V_g$ and $V_e$ (section 3), $V_g$ is estimated as RDS × $V_p$. Although measured values of RDS vary greatly, they never approach 85%, our estimate of total genetic variance in pupal development time (Appendix A); Yang *et al.* (2010) examined in detail a similar discrepancy in humans. Furthermore, there is no correlation between RDS and total phenotypic variance, $V_p$ (Fig. 6). This suggests that $V_g$ and $V_e$ are to some extent interchangeable, and that $V_g$ is contained within a relatively invariant $V_p$. There is no reason to suppose that $V_g$ and $V_e$ correspond to any discrete biological entities; therefore, the equation $p = g + e$ is a statistical model only. The canalization of Scharloo *et al.*'s (1967) "environmental" variance, $V_e$ (Table 1), shows that $V_e$ is not purely environmental, but is some kind of genetic−environmental interaction. Only a small part of the genetic variance responds to directional selection, and that part varies greatly within a relatively invariant whole.

We conjecture that the increases in RDS (Fig. 5, Table 2A) represent a shift, by genetic recombination, from the flat but compressed extreme of Figure 2 towards the centre. This is by no means certain; but whatever the true genetic explanation, the changes in RDS require interconversion of so-called "additive" and "nonadditive" genetic variance, as proposed by Carson (1990).

**Fig. 6.** Within-sex phenotypic SD of ln(pupal development time in days) (118−1414 df) *versus* RDS estimated from the same *Pieris rapae* stock. The correlation between RDS and ln(variance) weighted by df is −0.29 (27 df); Bartlett's test of homogeneity of variances, $\chi^2 = 264.7$ (28 df).




The conclusion here is that RDS is indeed reduced by truncation in the field, but is rapidly restored within a few generations. We next consider experiments designed to determine the proximal cause of truncation selection — and reductions in RDS — in the field.

**Table 3.** Mean numbers of late-V-instar larvae arising from introductions of adult *Pieris rapae* into two unique cages on sequential dates, with associated day-degrees (dd) since the start of the season, mean temperatures during oviposition, and larval development time during the first field-cage experiment, 2006.

| | Date of introduction | | | | | |
|---|---|---|---|---|---|---|
| | 7 Aug. | 19 Aug. | 31 Aug. | 10 Sept. | 17 Sept. | |
| Dd >10 °C since 1 May | 736 | 820 | 894 | 975 | 1036 | |
| Mean temp. (0930–1530) for 5 days after introduction (°C) | 20.9 | 19.6 | 22.2 | 22.8 | 20.7 | |
| No. of V-instar larvae per female | 26.8 | 38.4 | 27.2 | 9.4 | 0.5 | (±1.78, 5)* |
| Mean dd >10 °C from introduction to late-V instar | 297 | 258 | 256 | 232 | 152 | (±6.2, 5)* |

**Note:** Mean additional time from collection of late-V-instar larva to pupation is 60 dd.
*Mean ± SE, with degrees of freedom in parentheses.

## 5. Truncation selection in field conditions

Gilbert and Raworth (1996) assumed that in short seasons, late developers would be killed by early frosts. The timing is correct but the ecological mechanism is not. To examine events at the end of the season, three experiments were conducted in field cages, each 1.2 $m^2$ × 1.9 m high, at Cambridge from 2006 to 2008. In each experiment, about 10 kale plants were grown per cage and eight young mated female *P. rapae* — the progeny of field-collected adults — were introduced into each cage, with additional males, and left to lay eggs for the term of their natural lives. When the experiment involved sequential releases, adults for each release were derived from a single parental stock, and physiological age was synchronized among releases by reducing the temperature of the developing larvae or the adults. Newly-emerged adults were provided honey solution in cotton-wool feeders and mated in small cages in a greenhouse for 2−3 days before release. During the egg-laying period, honey solution in cotton-wool feeders was added to the field cages early each morning before flight occurred. At the time of introduction, the female *P. rapae* would have been approaching the maximum rate of egg production, so most eggs would be laid during the first 5 days in the field cages. Adult survival was gauged while opening the cages in the morning, and by observing adults flying in the cages during the day; survival – about 10 days – was good in all trials. Sunny, warm conditions (Tables 3–5), required for egg laying, occurred during the oviposition period in all trials. Reproductive success was determined by counting and removing late-V-instar larvae every 2−9 days depending on temperature. In the first experiment these larvae were then fed separately on detached leaves to ensure that they pupated. Mean development time was calculated for the late-V-instar larvae from each cage in dd >10 °C between the date when adults were introduced and the time when larvae were removed. Table 3 compares reproductive success and mean development time on five successive introduction dates during the exceptionally warm summer of 2006; wild *P. rapae* can be observed ovipositing on kale in gardens on these dates in any given year, depending on weather. Reproductive success began to decline by the end of August and was negligible by mid-September, when only larvae with the shortest development time — nearly half that of earlier introductions (Table 3) — survived to become diapausing pupae. Judging from feeding damage, late-V-instar larvae were produced only on the youngest plants, with relatively tender leaves, in the final trial, whereas plants of all ages were utilized in the earlier trials. These results confirm the occurrence of truncation selection against slow-developing larvae towards the end of the season. Table 4 compares three dates of introduction on young, middle-aged, and old plants during August and September 2007. Reproduction in the generally cool season of 2007 (Table 4) was much lower than in 2006

**Table 4.** Mean numbers of late-V-instar larvae arising from introductions of *Pieris rapae* into three unique cages on sequential dates, with associated day-degrees (dd) since the start of the season, mean temperatures during oviposition, and larval development times, during the second field-cage experiment, 2007.

| | Date of introduction | | | | | | | | |
|---|---|---|---|---|---|---|---|---|---|
| | 8 Aug. | | | 25 Aug. | | | 10 Sept. | | |
| Dd >10 °C since 1 May | 559 | | | 661 | | | 768 | | |
| Mean temp. (0930–1530) for 5 days after introduction (°C) | 23.3 | | | 21.2 | | | 21.3 | | |
| Plant age | O | M | Y | O | M | Y | O | M | Y |
| No. of V-instar larvae per female | 6.6 | 13.0 | 11.4 | 2.1 | 1.4 | 2.4 | 1.5 | 4.7 | 4.7 |
| Mean dd >10 °C from introduction to late-V instar | 248 | 264 | 243 | 250 | 230 | 222 | 184 | 184 | 182 |

**Note:** A cage contained either 8 old (O), 10 middle-aged (M), or 14 young (Y) plants sown on 30 April, 18 May, or 15 June, respectively.

(Table 3), and reproductive decline began earlier; the oldest plants grew to only 60 cm height compared with 90 cm in 2006 and 2008. During 20 years, the latest oviposition date permitting any reasonable chance of survival to pupa has varied (when calculated on the basis of temperature alone) from early August to early September. Table 4 shows the same decrease as Table 3 in the development times of surviving larvae in successive introductions. Table 5 compares, at midseason 2008, reproductive success on plants of two different ages, representative of *Brassica* plants that wild *P. rapae* encounter in the field during the second half of the season. The observed higher recruitment on young plants than on older plants explains the Vancouver value of RDS in Figure 5, which, unlike the other values of RDS, refers to a population feeding on "organic" commercial plants sown every 6 weeks through the summer, so that young plants were always available and all larvae would have been able to develop through to diapause.

In the first two cage experiments, most survivors had pupated before the first frosts, but the few latest larvae survived air frosts of −3 °C: 97 late-V-instar larvae were collected from all cages between 5 November and 30 December 2006 after a subzero period from 2 to 4 November; and 30 late-V-instar larvae were collected between 27 October and 8 November 2007 after a subzero period from 19 to 23 October. These survivors were a small fraction of all V-instar larvae collected. Frost may have killed some larvae, but far from all. So the end of the season is not determined proximally by frosts but, with relative importance varying among years, by (*i*) entry into diapause and (*ii*) age of host plants. The timing nevertheless anticipates the onset of winter.

## 6. One function of sex

"Despite many years of theoretical and experimental work, the explanation for why sex is so common as a reproductive strategy continues to resist understanding" (de Visser and Elena 2007). Here we show that one explanation for sex can be found by analysing a natural population as a set of sexual families, and we demonstrate the necessity of sexual reproduction for maintaining RDS and hence genetic variation in the population.

Galton's rule refers to family means. The family is the smallest unit of population that contains its own variance. The experiments summarized in Table 1 show that directional selection (unlike centripetal selection) can slowly increase or decrease within-family phenotypic variances. We have written a computer program (Appendix F) that represents a sexual population composed of individual families, each with its own mean and phenotypic SD. It mimics, but does not closely simulate, the results of Morris (1971), who compared the predicted and observed responses of average $K_p$, the heat requirement for pupal eclosion of univoltine fall webworm to natural selection

**Table 5.** Mean numbers of late-V-instar larvae arising from introductions of *Pieris rapae* on 20 July into each of four cages with 8 kale plants sown on 23 February and four cages each with 10 plants sown on 26 April, with associated larval development times, in the third field-cage experiment, 2008.

| | Plant age | | |
|---|---|---|---|
| | 21 weeks | 12 weeks | |
| No. of V-instar larvae per female | 14.4 | 38.6 | ($\pm$6.34, 6)* |
| Mean dd >10 °C from introduction to late-V instar | 273 | 262 | ($\pm$8.0, 6)* |

**Note:** The mean temperature (0930–1530) for 5 days after introduction was 24.2 °C; 448 dd >10 °C from the start of the season, 1 May, to 20 July.

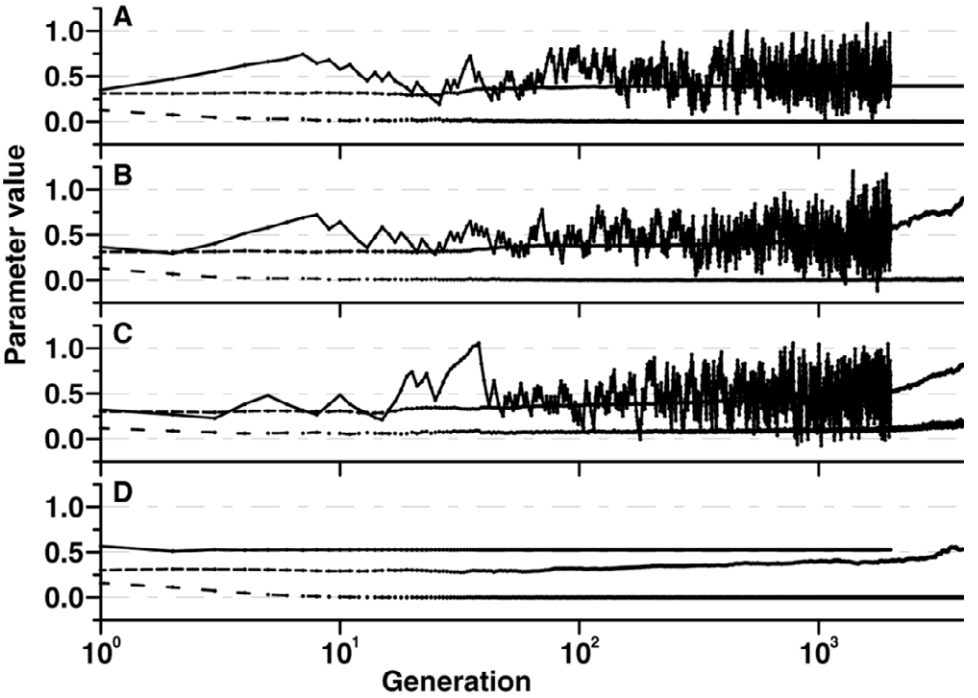
*Means, with overall SE and degrees of freedom in parentheses.

during 12 successive seasons. Morris' is perhaps the most successful attempt so far to measure natural selection in the field; however, as he said, it is impossible to reproduce his results solely by selection against high $K_p$ in cool seasons; they also require selection against low $K_p$ in warm seasons. The latter type of selection is probably due to metabolic exhaustion of individuals that pupate too early in the season (Morris 1971). In our program, different generations experience truncation selection against individuals with high $K_p$ in cool years, or its mirror image against individuals with low $K_p$ in warm years. Instead of the intensely associative mating (for the trait $K_p$) of fall webworm, the program provides for random mating among sexual families. It provides for complete ecological density-dependence because the number of families in the population remains constant over generations.

Following Morris (1971), for each generation the program generates a random Normal-value, "cut", such that in warm years, all individuals with values of $K_p$ above the cut survive, whereas all those below it die, and conversely in cool years. This selection of individuals then translates into two types of selection on families. First, it determines the number of survivors in each family, which determines that family's chance of contributing to the next generation; second, it shifts the mean of $K_p$ among the survivors by an amount calculated using Normal theory, but modified by RDS. Because of their strong resistance to centripetal selection (Table 1), family SDs remain unaltered. So family means are subject to both types of selection but family SDs to the first type only. Using Normal theory, the program applies the truncation selection, above or below the cut, to each family (Appendix G). Selective limits, and the associated skewness and reduced fitness (section 2), are ignored. All surviving individuals have an equal chance of parenthood; so in practice, each family has a chance of being chosen for reproduction that is proportional to the number of survivors in that family. Some families may be chosen more than once, others not at all. A mating between parents from two sexual families produces a new sexual family with a mean calculated using Galton's rule. Because nothing is known about the inheritance of family SDs, the new SD is set equal to the SD of either parental family, chosen at random (Appendix H). All new families have the same size before selection is applied.

In the computer program, families with means of $K_p$ close to the average value of cut survive better, in the long run, than do all others. So the population as a whole experiences long-term centripetal selection, yet it maintains genetic variation, as follows. In any one generation, different families experience selection in different directions, according to their position relative to the cut. In warm years, when all individuals above the cut survive, families with larger SDs have an advantage if their mean is below the cut, because a larger SD then increases the number of survivors; but they are disadvantaged if their mean is above the cut, because a larger SD then decreases survival. And the converse occurs in cool years. At the same time, the family mean is not transmitted unchanged from generation to generation; instead, new families are formed with new means.

**Fig. 7.** Results from a stochastic model that simulates randomly fluctuating truncation selection in a population of sexual families (nsex = n = 200; Appendix F) over thousands of generations. Output parameters in each subplot are as follows: the upper (solid) line shows the weighted family mean $K_p$, the heat requirement for eclosion of pupae of *Hyphantria cunea* on an arbitrary scale initially set uniformly between 0.2 and 0.8 (plotted only for 2000 generations); the middle (broken) line shows the weighted average within-family SD; and the lower (broken-dotted) line shows the weighted between-family SD, where the weight is the proportion of survivors in each family after truncation selection. (A) Within-family SDs (fsd(i)) initially set uniformly between 0.2 and 0.4 and not reduced by truncation selection, additional within-family variation (dev) zero, RDS = 0.3, within-family variation not transferred to differences between family means. (B) As in A, but dev = 0.01. (C) As in B, but within-family variation is transferred to differences between family means by adding one random Normal within-family deviate, multiplied by RDS (0.3), to the family mean. (D) As in C, but RDS = 0.




So there is reversible selection of both family means and family SDs, which maintains a quasi-equilibrium, persisting for thousands of generations (Fig. 7A). It is a quasi-equilibrium because the sexual mechanism initially maintains whatever amount of genetic variance is present in the population at the start of the calculation.

This mechanism works only because within-family variance resists selection tending to reduce it (Table 1). Otherwise, truncation selection would quickly reduce within-family variance to zero, and the population would implode to a set of individuals all with genetic values equal to the average value of cut. We do not know genetically how within-family variances resist centripetal selection, but given that they do, no further theory (*e.g.*, heterozygote advantage, frequency-dependent selection, or geographic heterogeneity; cf. Bell 1982) is needed to explain how sexual populations maintain genetic variation in the face of centripetal selection.

According to the model, as the generation-to-generation variance of cut increases, so does the population variance. This offers a possible mechanism to match the amount of genetic variance to environmental unpredictability.

In the model there is very slow selection for increasing within-family variance because families that spread over the whole range of values of cut survive and reproduce, in the long term,

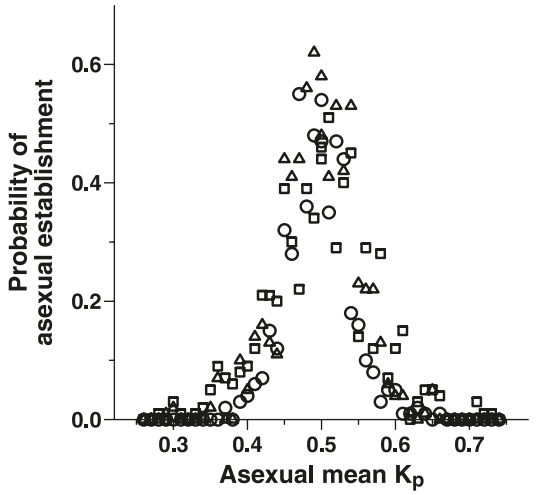
better than families with less variance. When the model uses only the original family SDs, all family SDs eventually converge on the maximum initial value in the population (Fig. 7A, middle (broken) line); but when an additional term, "dev" (Appendix F), is added to prevent convergence by supplying additional random variation to the family SDs, there is selection for ever-increasing within-family variance (Fig. 7B). This suggests that selective limits (Haldane 1954) block not only selection for ever-increasing individual size and fecundity (Gilbert 1984*a*), but also selection for ever-increasing within-family variance in other traits subject to fluctuating selection.

When the population is viewed in this way as a collection of sexual families, we see that genetic variation is maintained by the resistance of within-family variance to centripetal selection. To maintain population variance, that variation must be transferred to the differences between family means. Otherwise, Galton's rule approximately halves the between-family variance in each generation, and those family means implode to a common value (Fig. 7A, lower (broken-dotted) line). The program achieves the transfer by calculating genetic values, *g*, for individual parents as follows: to the parental family mean it adds a random Normal deviate with an SD equal to the within-family SD. This is equivalent to within-family directional selection of a single individual. But the chosen new parent will contribute only its genetic value (*g*) to a new progeny family; and since the within-family SD is phenotypic, Normal theory requires that the deviation must be multiplied by RDS to calculate the new parent's value of *g*. So RDS appears twice in the computer program: to shift family means according to truncation selection, and to calculate individual genetic values for subsequent breeding parents (Fig. 7C, Appendix I). If RDS were zero, then although the members of any family would still be genetically variable, they would not transmit that variation to their own progenies in the next generation, and the family means would converge towards a common value (Fig. 7D, upper (solid) line). It is therefore essential that when RDS is reduced, it shall be rapidly restored — as it is (Fig. 5, Table 2A). This restoration of RDS can be achieved only by sexual recombination, since mutation is far too slow (Barton and Keightley 2002). Our observations illustrated in Figure 5 and Table 2A therefore identify one function of sex: it rapidly restores RDS and so maintains the genetic variation that a population needs to match unpredictable environmental variation. This point is here presented as a long-term, whole-population advantage, but it arises by continuous, reversible selection of individuals and families within a single population. Overall survival remains unchanged as RDS increases, so this population advantage entails no disadvantage to individuals. Spieth (1985) and Sauer and Grüner (1988) confirmed that genetic variance in different populations corresponds to the environmental unpredictability that those populations experience. And Morris (1971) described a natural population that, if asexual, would risk complete extermination in a series of extremely warm and cool seasons.

According to Weismann (1904), an asexual female has a twofold reproductive advantage over her sexual counterparts because she produces only female progeny — no males. Weismann's argument is usually known as "the cost of sex". Our computer program allows for asexual immigrants to compete with an established sexual population. The program shows that an immigrant asexual female has some stochastic chance of establishing an asexual subpopulation that will eventually replace the sexual population — but only if her value of $K_p$ is close to the optimal $K_p$ prevailing when she arrives (Fig. 8). Considering that her genotype similarly needs to be near a transient optimum for each of a whole range of other traits, we conclude that an asexual invasion is unlikely to succeed unless it involves the arrival of a large number of individuals with many combinations of adaptive genotypes. We speculate that sexual immigrants have a much better chance of success, either by establishing a new population or by contributing genetically to an existing one.

Nevertheless, any explanation of sex must consider Weismann's (1904) argument. A sexual female who switched to asexuality, without any increase in fecundity, would have twice as many daughters, four times as many granddaughters, and so on; so her asexual descendents would

**Fig. 8.** Probability of an asexual clone overtaking a sexual population as a function of the clone's mean $K_p$ with respect to the optimal $K_p$. Because "cut", the point at which truncation selection occurs, has a Normal (0.5, 0.08) distribution restricted within (0.1, 0.9), the optimal $K_p$ is 0.5. Sexual family means are initially distributed randomly between $K_p$ 0.2 and 0.8, whereas each asexual clone was given a specific mean $K_p$. Each point represents a probability determined from 100 runs, each for as many generations as needed to obtain asexual, or sexual, displacement. Initial parameter values are as follows: n =100, nsex = 99, dev = 0.01; ○, RDS = 0.1; Δ, RDS = 0.3; □, RDS = 0.5 (Appendix F).




quickly swamp the sexual population. This seems to mean that sex must confer on individual parents some advantage that is more than twofold, to overcome the intrinsic theoretical advantage of asexuality; but it ignores ecological density-dependence. Our counterargument is that a switch to asexuality is numerically equivalent to maintaining the sexuality of the original female while doubling the fecundity, not of that female herself but of all her female sexual descendents in all succeeding generations (Appendix J). Assuming that her original fecundity was close to optimal in the face of whatever density-dependent considerations restrict it, doubling that fecundity must reduce subsequent survival by more than half, so that the postulated reproductive advantage is more than cancelled. Many aphid, monogonont rotifer, and cladoceran species support this counterargument: they regularly switch to asexuality — so obtaining the twofold advantage that Weismann envisaged — when, and only when, density-dependence is temporarily in abeyance for a generation or more (Bell 1982; Appendix J). If this counterargument is correct, the computer program confirms that an asexual clone would not survive in a sexual population.

The conclusion is that sex restores the value of RDS, and thereby maintains the genetic variation that a population needs in order to match unpredictable environmental variation, despite selection tending to reduce that genetic variation. We do not suppose that this is the sole function of sex; its original function may have been quite different (Hickey 2000), and it may have other functions (Bell 1982). The mechanism envisioned here involves continuous selection, not of whole populations but of individuals and families within one population. We speculate that the reductions in RDS, observed in Figure 5, involve a shift onto the flat extremes of Figure 2; but in any case, the rapid restoration of RDS certainly requires sexual recombination. Of long-term evolution, Bell (2008) wrote, "selection is generally rather strong and fluctuates on all time-scales such that abrupt changes can occur over short periods of time and gradual directional change occurs over long periods of time". Here we suggest that sex provides a necessary mechanism for chasing an ever-fluctuating optimum. Until we begin to understand the underlying genetics, studies of other species or traits would be useful to determine the generality of the mechanism discovered here.

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## Appendix A

The greater part of the phenotypic variation in pupal development time must be genetic. Development time is affected by temperature, held constant at 25 °C, and by the quality of the host plants on which the larvae fed. Otherwise, pupae are self-contained. In the laboratory conditions adopted here, differences in plant quality are estimated to account for 4% (21 df) of variance and the 6-h time interval between successive pupal collections for 11%: the latter variance is nongenetic, and adds on to the rest. We conclude, by elimination, that the remaining 85% of variance must be genetic, affecting the developmental process within pupae.

## Appendix B

At Hobart and Vancouver, with high population densities, several dozen females could be caught as they visited host plants to lay eggs. But at Cambridge, individual adults were caught in free flight in open fields; the sex ratio of such catches is about 10 males to 1 female. The conditions required for flight (full sun, and temperature more than 15 °C) occur infrequently during the first (spring) generation at Cambridge. Consequently, the number of females caught during that generation at Cambridge was lower than at Hobart and Vancouver. RDS is then estimated from the immediate progeny of those females.

## Appendix C

It follows statistically from Galton's rule that in a stable, random-mating population, the genetic variance between family means is about half the total genetic variance in the whole population. Fisher (1930) derived the same conclusion empirically from correlations between human relatives (Fisher 1918). Then the progeny of a single mated female will exclude about half the total genetic variance, so the combined progenies of a random $n$ mated females exclude a fraction $2^{-n}$, and therefore include a fraction $1 - 2^{-n}$ of the total population variance. Thus, if $n = 4$, the first-generation progenies of four mated females (*i.e.*, of eight parents) are expected to contain 15/16 of the population genetic variance. In empirical confirmation, Bryant *et al.* (1986) found that the combined progenies of four mated females gave full estimates of narrow-sense heritabilities of eight quantitative traits in *Musca domestica* L. (Diptera: Muscidae) two generations removed from the wild.

## Appendix D

Programs written in FORTRAN 77 to reanalyse selection experiments in the literature (section 3).

**Directional selection**

```
      program seln
      p=.1
      theta=1.2816
      coft=.398942*exp(-.5*theta**2)/p
      nct=0
      ve=.35
      vg=.65
      v=vg+ve
      pmean=0.
      write(*,2)
    2 format(' values of factg, facte?')
      read(*,*)factg,facte
    1 rds=factg*vg/v
      nct=nct+1
      pm=coft*rds*sqrt(v)
      vg=vg*(1.+coft*theta*rds)-pm**2
      pmean=pmean-pm
      rds=facte*ve/v
      pm=coft*rds*sqrt(v)
      ve=ve*(1.+coft*theta*rds)-pm**2
      pmean=pmean-pm
      v=vg+ve
      write(*,*)nct,115+11.619*pmean,vg/v,135*v
      if(nct.gt.5)then
        pause
        stop
      endif
      goto1
      end
```

**Description of program seln:**

The internal parameters are altered to suit each experiment and the program is recompiled. Then appropriate values of *factg* and *facte* are found by trial and error. The variable $p$ is the selection rate 4/40 used in the selection experiment by Scharloo *et al.* (1967). Theta is the standard Normal deviate corresponding to the tail probability $p$, *i.e.*,

$$\frac{1}{\sqrt{2\pi}} \int_{-\infty}^{-\vartheta} e^{-\frac{1}{2}x^2} dx = p$$

0.398942 is $\frac{1}{\sqrt{2\pi}}$

115 is the initial observed mean

11.619 is the initial observed phenotypic SD, so the program works on the standard Normal scale and converts the result to the original scale of measurement.

**Worked example (**based on Figure 2 from Scharloo *et al.* (1967) except where noted):

Two initial estimates of RDS, 0.62 $\pm$ 0.11 and 0.68 $\pm$ 0.08 (Table 2 in Scharloo *et al.* 1967), with average 0.65 $\pm$ 0.068. Initial phenotypic variance ($V_p$) is 135, omitting within-fly variance ($V_{WF}$), which is irrelevant (*i.e.*, $V_p - V_{WF} = 160 - 25$). The selection regime is 1/10 truncation with random mating for five generations. Observed responses were upwards to 155 after five generations, and downwards to 76, so responses are symmetric. Phenotypic variance is unchanged in the upwards line, decreases to 75 ($V_p - V_{WF} = 90 - 15$) in low after five generations. Heterozygotes were misclassified in the upwards line because as selection proceeds one cannot with certainty distinguish between heterozygotes and homozygotes.

The appropriate values for reproducing the final mean and variance are *factg* = 0.88, *facte* = 0. With these values, the predicted mean and phenotypic variance at *nct* = 5 are 74.1 and 74 and the observed values 76 and 75, respectively.

**Centripetal selection**

```
      program selns
      p=.1
      theta=.125661
      coft=.797885*theta*exp(-.5*theta**2)/p
      nct=0
      ve=.35
      vg=.65
      v=vg+ve
      write(*,2)
    2 format(' values of factg, facte?')
      read(*,*)factg,facte
    1 rds=factg*vg/v
      nct=nct+1
      vg=vg*(1.-coft*rds)
      rds=facte*ve/v
      ve=ve*(1.-coft*rds)
      v=vg+ve
      if((nct.eq.7).or.(nct.eq.19).or.
     * (nct.eq.36))write(*,*)nct,vg/v,191*v
      if(nct.gt.36)then
        pause
        stop
      endif
      goto1
      end
```

**Description of program selns:**

Since selection is centripetal, the mean does not change and the object is to find values of *factg*, *facte* to match observed values of RDS and phenotypic variance. Also, see the description of program seln above.

## Appendix E

In *P. rapae*, female pupae develop, on average, faster than males. About 27% of all within-family genetic variance for pupal development rate is directly due to the average difference between male and female pupae. By selecting on that sex difference itself, we can check the conclusion (section 3) that within-family variation resists centripetal selection. The selection procedures are identical with those described

in section 1, except that to select for sex difference we mate early females with late males and conversely, whereas to estimate RDS we mate early females with early males and late with late. The results are as follows: at Cambridge in 2004, RDS was 0.37 $\pm$ 0.104 (334 df) and responsiveness to selection on sex difference in the same generation was 0.11 $\pm$ 0.092 (334 df). At Cambridge in 2008, RDS in the previous generation was 0.20 $\pm$ 0.030 (681 df) and responsiveness to selection on sex difference was $-0.05 \pm 0.027$ (468 df).

There is little or no response to selection on sex difference, which tends to confirm that within-family variance resists centripetal selection.

## Appendix F

Simulation model of sexual and asexual population means, and within- and between-family variances, under conditions of random truncation selection, and variable levels of responsiveness to directional selection (RDS) in sexual populations (section 6).

```
      program morris
      dimension fm(1024),fsd(1024),fmold(1024),fsdold(1024),
     *e(33),freq(1024),nadist(30),nsdist(30),p(33)
      data e/0.,.004,.008,.013,.023,.036,.055,.082,.117,.163,
     *.219,.288,.367,.459,.562,.675,.798,.929,1.069,1.215,
     *1.368,1.525,1.687,1.854,2.024,2.199,2.373,2.552,
     *2.731,2.915,3.097,3.283,3.5/
      data p/1.,.99865,.99744,.99534,.9918,.9861,.97725,.96407,
     *.9452,.91924,.88493,.84132,.78815,.72573,.6554,.57927,.5,
     *.42073,.3446,.27427,.21185,.15868,.11507,.08076,.0548,
     *.03593,.02275,.0139,.0082,.00466,.00256,.00135,0./
c             set random generator seed. functions srand, rand
c             and time are said to be standard for FORTRAN77 and later
      call srand(time())
      write(*,1)
    1 format(' values of n, nsex, RDS, dev?')
      read(*,*)n,nsex,rds,dev
      nct=0
      nasex=n-nsex
c             initialize fm(i), fsd(i)
      do3i=1,n
      x=rand(0)
      fm(i)=.2+.6*x
      x=rand(0)
    3 fsd(i)=.2+.2*x
c             start period of 100 generations
    4 do5i=1,30
      nadist(i)=0
    5 nsdist(i)=0
      totsd1=0.
      totsd2=0.
c             start each generation
    6 tafr=0
      tsfr=0
c             calculate cut as N(.5,.08) within range (.1,.9).
c             cut is the cutoff for individual survival, so for
```

```
c               each family calculate no. survivors and shifted mean
      x=.25
      do10j=1,12
c               rand(0) gives uniformly distributed random (0,1)
      z=rand(0)
   10 x=x+z
      cut=.08*x
      if(cut.lt.0.1)cut=.1
      if(cut.gt.0.9)cut=.9
c               with probability 0.5, in the current generation all
c               individuals above 'cut' survive; sexual means increase
      if(z.gt.0.5)then
        do20i=1,n
        x=(cut-fm(i))/fsd(i)
        nc=5.*x+17.5
        if(nc.lt.1)nc=1
        if(nc.gt.33)nc=33
        freq(i)=p(nc)
        if(i.le.nsex)then
          fm(i)=fm(i)+e(nc)*rds*fsd(i)
          tsfr=tsfr+freq(i)
        else
          tafr=tafr+freq(i)
        endif
   20   continue
      else
c               all below the cut survive, and sexual means decrease
        do30i=1,n
        x=(fm(i)-cut)/fsd(i)
        nc=5.*x+17.5
        if(nc.lt.1)nc=1
        if(nc.gt.33)nc=33
        freq(i)=p(nc)
c               sexuals
        if(i.le.nsex)then
          tsfr=tsfr+freq(i)
          fm(i)=fm(i)-e(nc)*rds*fsd(i)
        else
c               asexuals
          tafr=tafr+freq(i)
        endif
   30   continue
      endif
      taf=0.
      tsf=0.
      tam=0.
      tsm=0.
      tasd=0.
      tssd=0.
      tam2=0.
      tsm2=0.
```

```
c               for sexuals and asexuals separately, convert freq(i)
c               to sum to 1 and then to cumulative values.
c               calculate weighted population means and
c               between-family variance
      do50i=1,n
c               sexuals
      if(i.le.nsex)then
        freq(i)=freq(i)/tsfr
        tsm=tsm+freq(i)*fm(i)
        tsm2=tsm2+freq(i)*fm(i)**2
        tssd=tssd+freq(i)*fsd(i)**2
        tsf=tsf+freq(i)
        freq(i)=tsf
      else
c               asexuals
        freq(i)=freq(i)/tafr
        tam=tam+freq(i)*fm(i)
        tam2=tam2+freq(i)*fm(i)**2
        tasd=tasd+freq(i)*fsd(i)**2
        taf=taf+freq(i)
        freq(i)=taf
      endif
      fmold(i)=fm(i)
   50 fsdold(i)=fsd(i)
      if(nasex.eq.0)goto51
c               tam2 is weighted SD between asexual family means
      tam2=tam2-tam**2
      if(tam2.lt.0.)tam2=0.
      tam2=sqrt(tam2)
c               tasd is weighted av. SD within asexual families
      tasd=sqrt(tasd)
   51 if(nsex.eq.0)goto52
c               tsm2 is weighted SD between sexual family means
      tsm2=tsm2-tsm**2
      if(tsm2.lt.0.)tsm2=0.
      tsm2=sqrt(tsm2)
c               tssd is weighted av SD within sexual families
      tssd=sqrt(tssd)
c               choose parents randomly by family frequencies
   52 do100i=1,n
      x=rand(0)
c               sexuals
      if(i.le.nsex)then
        j=1
   81   if(x.le.freq(j))then
          nend=j
          goto82
        else
          j=j+1
          goto81
        endif
```

```
   82    x=rand(0)
         j=1
   83    if(x.le.freq(j))then
           mend=j
           goto86
         else
           j=j+1
           goto83
         endif
       else
c                asexuals
         j=nsex+1
   84    if(x.le.freq(j))then
           nend=j
           goto86
         else
           j=j+1
           goto84
         endif
       endif
   86  continue
c              calculate fm(i), fsd(i) for mating of parents
c              from parental families i=nend, i=mend or for selfing
c              of parent from asexual family i=nend
       x=-6.
       do87j=1,12
       z=rand(0)
   87  x=x+z
c              sexuals
       if(i.le.nsex)then
         y=-6.
         do88j=1,12
         z=rand(0)
   88    y=y+z
         fm(i)=.5*(fmold(mend)+fmold(nend)
     *  +rds*(x*fsdold(mend)+y*fsdold(nend)))
c              set fsd(i) equal to fsd of either parent
         x=rand(0)
         if(x.lt.0.5)then
           fsd(i)=fsdold(nend)
         else
           fsd(i)=fsdold(mend)
         endif
         fsd(i)=fsd(i)*(1.+dev*(z-.5))
         j=10.*fm(i)+11.
         if((j.lt.1).or.(j.gt.30))goto100
         nsdist(j)=nsdist(j)+1
       else
c              asexuals
         fm(i)=fmold(nend)
         fsd(i)=fsdold(nend)*(1.+dev*(z-.5))
```

```
            j=10.*fm(i)+11.
            if((j.lt.1).or.(j.gt.30))goto100
            nadist(j)=nadist(j)+1
          endif
  100     continue
          x=n
c                 calculate new no. of sexual families = newsex.
c                 if numbers increase, replace old asexual families in i=
c                (nsex+1, newsex) by new sexual families from N(tsm,tssd)
          newsex=x*tsfr/(tsfr+tafr)+.5
          if(newsex.gt.n)newsex=n
          if(newsex.eq.nsex)goto140
          if(newsex.gt.nsex)then
c                 overwrite asexuals(i=nsex+1,newsex) with new sexuals
            i=nsex
  110       i=i+1
            x=-6.
            do120j=1,12
            z=rand(0)
  120       x=x+z
            fm(i)=tsm+x*tssd
            fsd(i)=tssd*(1.+dev*(z-.5))
            if(i.lt.newsex)goto110
            goto140
          else
c                 overwrite sexuals(i=newsex+1,nsex) with old asexuals
c                 no need to choose randomly because already randomized
            i=newsex
            j=nsex
  130       i=i+1
            j=j+1
            if(j.gt.n)j=nsex+1
            fm(i)=fm(j)
            fsd(i)=fsd(j)
            if(i.lt.nsex)goto130
          endif
  140     nsex=newsex
          nasex=n-nsex
          totsd1=totsd1+tssd
          totsd2=totsd2+tsm2
          nct=nct+1
c                 if nct is integer multiple of 100, print
          if(nct.ne.100*(nct/100))goto6
          write(*,190)nct,nsex,tsm,.01*totsd1,
         *.01*totsd2,nsdist,
         *nasex,tam,tasd,tam2,nadist
  190     format(i5/(i5,3f10.4/3(10i6/)))
          pause
          goto4
          end
```

**Description of program morris** (based on Morris 1971):

A population consists of n (<1025) families. Starting at i = 1, the first, nsex, families are sexual and the remaining, nasex (nsex + nasex = n), families are asexual. The i-th family has mean fm(i), within-family SD fsd(i), and frequency freq(i), which represents in relative terms the number of surviving individuals in that family. The population size, n, remains unchanged, so there is strict density-dependence in that sense. Values of nsex, nasex change with the ratio of sexual:asexual individuals in the population, *i.e.*, there is competition between sexual and asexual individuals according to their survival rates. Instead of values of $K_p$, the heat requirement of fall webworm pupae for eclosion, the program uses an arbitrary scale of measurement. The values of fm(i) are initially set uniformly between 0.2 and 0.8, but calculated values thereafter can spread outside that range. Following Morris (1971), there is a "cut", which is the cutoff in any one generation. The values of cut in successive generations have a Normal (0.5, 0.08) distribution restricted within (0.1, 0.9). In one half of all generations, all individuals above the cut survive; otherwise, all individuals below the cut survive. The calculation is therefore symmetric about the value 0.5 and the population mean fluctuates about that value.

The values of e are means of a standard Normal distribution after truncation at different abscissae. They come from tables of integrals of the standard Normal distribution and when multiplied by RDS are used to calculate responses of sexual family means to truncation selection above or below the cut. Asexual families are truncated, which reduces the number of their surviving members, but they do not respond to directional selection. The values of p are corresponding tail probabilities of the same Normal distribution above the same abscissae. They come from tables of probits and supply calculated survival rates in truncated families, both sexual and asexual. The next generation is from parents chosen randomly with equal probability from all surviving sexual individuals and with equal probability from all surviving asexual individuals. Consequently, the probability of choosing the i-th family as a parent is proportional to freq(i), which is initially the proportion of survivors in that family after selection by cut. Later in the program, the values of freq(i) are divided, for sexual and asexual families separately, by their total so that they now sum to 1.0, and then accumulated from i = 1 to nsex (*i.e.*, all the sexual families) and again from i = nsex + 1 to n (*i.e.*, all the asexual families). This is done to permit choice of families to provide parents, in proportion to the frequency of survivors in each family — so ensuring that all individuals, whether sexual or asexual, have the same chance of parenthood. Sexual matings use Galton's rule that the progeny family mean equals the arithmetic mean of the "genetic values" of the two parents. The genetic value of each parent is calculated by adding to the family mean fm(i) (as adjusted by truncation selection) a random Normal deviate (0, fsd(i)), multiplied by RDS. Inbreeding, *i.e.*, both parents chosen from the same family, is permitted but can easily be forbidden by rewriting the program — it makes little difference. The within-family SD of the progeny of a sexual mating is set equal to the SD of either parental family, chosen at random with equal probability. Initially, fsd(i) varies between 0.2 and 0.4 (mean 0.3), which is rather high compared with the variation in cut; it provides plenty of variation for the selection to work on. In asexual progenies the family mean and SD are the same as in the parental family. But in all families the SD is slightly increased or decreased according to the parameter "dev". This may be regarded as a form of mutation, introduced to permit continuing natural selection, according to family survival rates, of both family means and family SDs. Unlike the family mean fm(i), the within-family SD fsd(i) is *not* reduced, according to standard Normal theory, by truncation, even in sexual families — if the program is altered to do this, the family means rapidly implode. So the within-family SDs, fsd(i), experience selection only because different families experience different survival rates freq(i). All families have the same fecundity, *i.e.*, the same size before selection by cut is applied.

Every 100 generations, the program prints:

The generation count:

For sexuals: (1) nsex, (2) the mean of all surviving individuals in the current generation,

(3) the average within-family SD (averaged over all sexual families and over the previous 100 generations, (4) the SD between-family means (averaged over the previous 100 generations), and (5) a 30-category statistical distribution of the family means, accumulated over the previous 100 generations.

And for asexuals: nasex and the same statistics, except that items 3 and 4 are not averaged over 100 generations, but refer to the current generation only.

Suitable trial values of n, nsex, RDS, and dev are 200, 200, 0.3, and 0.01, respectively, for an all-sexual population (nsex = n), or 200, 190, 0.3, 0.01, respectively, for an initial mixture of 190 sexuals and 10 asexuals, which usually win out because they include families with values of fm(i) close to the optimal value, 0.5. To show that asexuals otherwise do not displace the sexuals, it is necessary to rewrite the initialization at the DO-loop beginning with do3i = 1,n accordingly, with separate initialization of sexuals and asexuals.

A run that begins all asexual (nsex = 0) rapidly implodes to a single central family. A run that is all sexual (nsex = n) maintains variation for tens of thousands of generations. There is slow selection for increasing values of fsd(i), owing to greater survival of families with larger fsd(i), and increasing fsd(i) entails increasing within- and between-family variation. At label 10 + 6, and similarly 20 + 3, a value of x is found by division by fsd(i), so that the larger fsd(i) is, the smaller is the absolute value of x and (on average) the greater the family survival — depending on the values of fm(i) and cut. As an extreme example, if cut − fm(i) = 0.3 and fsd(i) = 0.5, family survival is 0.27; but if fsd(i) increases to 1.0, survival increases to 0.34. But if cut − fm(i) = 0, there is no increase in survival.

## Appendix G

This paper uses Galton's rule, RDS, and Normal theory, without any assumption about individual gene effects. It might be objected that the use of Normal theory implies a hidden assumption of additive gene effects. This is not so. Provided that all possible interactions are included, Fisher's additive model can embrace any genetic pattern whatsoever, because the number of available parameters always exceeds the number of data. (But unless the main effects predominate, the additive model is not a helpful way of visualizing the situation.) The Normal distribution arises when the number of genes tends to infinity and each gene effect tends to zero, while the mean and variance remain finite. In that case, interactions vanish (Kendall and Stuart 1963) and the situation becomes perfectly additive. But as long as the number of genes remains finite, gene effects can still interact, and Normality does not entail strict additivity. As usual, Normality is merely a statistical convention that the data approximate.

## Appendix H

The computer program assumes that within-family genetic variance itself varies from family to family and can be transmitted from parent to offspring. We know of no direct evidence for this, but Tantawy and Tayel (1970) selected for increased range (and therefore variance) within experimental cohorts comprising a mixture of three sexual families each, and obtained an increase in variance with no change in the mean; and Thoday (1959) selected on the within-family range of single families and obtained an increase in within-family variance with no change in the mean. The results of these experiments show that it is possible to select for increasing within-family variance, and because selection continued for several generations, the change in variance must be transmissible from parents to progeny.

## Appendix I

The second value of RDS predicts the genetic value of one randomly chosen (surviving) progeny individual, which in the next generation becomes a parent of a new family. The first value of RDS predicts the genetic mean value of all (surviving) progeny individuals, and is

therefore some kind of average of all possible second values of RDS. If the distribution is truly Normal, these two values of RDS must be identical. But the distribution is not Normal, since it is compressed at each end (section 2). Then the two values of RDS may still be identical, or not, depending on the unknown underlying genetics. But when natural selection reduces all possible second values of RDS to zero (Fig. 5), their average — and consequently the first value of RDS — must be zero too. That is all that is needed to sustain the argument for sex.

## Appendix J

The following table compares the hypothetical family sizes produced by a sexual female with fecundity 2n (n males + n females), but whose female descendents all have doubled fecundity 4n (2n males + 2n females), and an asexual female with fecundity 2n, whose descendents are all female with the same, unchanged, fecundity.

| | Sexual | | | Asexual |
|---|---|---|---|---|
| Generation | Males | Females | Total | Females |
| 1 | n | n | 2 n | 2 n |
| 2 | $2n^2$ | $2n^2$ | $4n^2$ | $4n^2$ |
| 3 | $4n^3$ | $4n^3$ | $8n^3$ | $8n^3$ |

These two families maintain the same totals in every succeeding generation, and therefore experience the same density-dependent resistance, assuming that males and females are equally vulnerable.

This counterargument assumes that the deleterious effects of excessive fecundity rebound on the guilty female or on her own progeny: she cannot shift them partly onto her competitors. We know of no direct evidence on that point — but if she could so shift them, natural selection would increase individual fecundity above the population optimum. The numerous field experiments on clutch size in birds show only that average individual fecundity rarely exceeds the individual optimum; but in *Masonaphis maxima* (Mason) (Homoptera: Aphididae), fecundity equals the population optimum (Gilbert 1980).

That many aphids, monogonont rotifers, and cladocerans abandon sexuality whenever the environment regularly permits unbridled population increase is the best available evidence for Weismann's cost-of-sex argument. That many aphids, monogonont rotifers, and cladocerans revert to sexuality when the environment deteriorates shows that sex must confer some continuing advantage — it is not just an evolutionary hangover. It might be objected that aphids must revert to sexuality because only sexual females can lay the overwintering eggs, but that objection does not extend to rotifers, where some species revert to sexuality and others do not (Butlin 2002), nor does it extend to cladocerans, where increased density, not seasonality, is the driving factor (Bell 1982).

In the past, several theories of sex have been rejected because they apparently could not overcome Weismann's argument. But this counterargument applies generally, so any of those theories might after all be correct. We cannot assess the relative importance of these and other proposed functions of sex.

During evolution, some mechanism that originally fulfilled one function is commonly adapted to perform quite another. Fisher (1930) proposed that sex maintains the genetic variation needed to accelerate continuous directional evolution. That function is evidently related to the one proposed here, but (*i*) assumes competition between sexual and a sexual populations, (*ii*) has limited theoretical validity (Cavalli-Sforza and Bodmer 1971), and (*iii*) has no proven case history in support. However, both Fisher's proposal and ours depend on the maintenance of RDS, so the same sexual mechanism will serve both purposes.