

## 1 Supplement – numerical example

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3 Consider a simple numerical example. A deleterious allele  $a$  is segregating in two populations  
4 of a diploid organism – one (P1) composed entirely of asexual females, the other (P2) sexual  
5 with 1:1 sex ratio. Homozygotes  $aa$  have fitness = 0, heterozygotes ( $Aa$ ) are 50% less fit than  
6 wild-type homozygotes  $AA$  (*i.e.*  $w_{AA}=1$ ,  $w_{Aa}=0.5$ ,  $w_{aa}=0$ ). Selection against  $a$  takes place at  
7 the stage of reproduction. In the sexual population, the selection acts via differential fecundity  
8 in females and differential sexual competitiveness (e.g. courtship or combat success) in males,  
9 but it has equal strength in both sexes.

10 Let's say that at generation  $x$ , both populations are composed of 80% wild-type homozygotes  
11 and 20% heterozygotes ( $p_{AA_x} = 0.8$ ,  $p_{Aa_x} = 0.2$ ; in the sexual population, genotype  
12 frequencies are identical in both sexes). Thus, the frequency of  $a$ ,  $p_{a_x} = 0.5 * p_{Aa_x} = 0.1$ , and  
13 mean relative fitness  $w_x = w_{AA} * p_{AA_x} + w_{Aa} * p_{Aa_x} = 0.9$  (*i.e.* an average female is 10%  
14 less fit than would be the case in the absence of  $a$ ) in both populations. Assuming no new  
15 mutations, in generation  $x+1$  genotype frequencies in the asexual population are given by

16 
$$p_{AA_{x+1}} = p_{AA_x} * w_{AA} / w_x = 0.89$$

17 
$$p_{Aa_{x+1}} = p_{Aa_x} * w_{Aa} / w_x = 0.11.$$

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19 In the sexual population, assuming no correlation in genotype between pairs of fusing  
20 gametes, the genotype frequencies are

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$$p_{AA_{x+1}} = g_{A_f} * g_{A_m},$$

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$$p_{Aa_{x+1}} = g_{A_f} * g_{a_m} + g_{a_f} * g_{A_m}$$

23 
$$p_{aa_{x+1}} = g_{a_f} * g_{a_m}$$

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25 where  $g_A$  and  $g_a$  are frequencies of gametes with respective genotypes at generation  $x$ , and  
26 subscripts  $f$  and  $m$  denote female and male gametes. Gamete frequencies are determined by  
27 genotype frequencies and selection, which are the same for females and males, so

28 
$$g_{A_f} = g_{A_m} = (p_{AA_x} * w_{AA} + 0.5 * p_{Aa_x} * w_{Aa}) / w_x = 0.94$$

29 
$$g_{a_f} = g_{a_m} = (0.5 * p_{Aa_x} * w_{Aa}) / w_x = 0.06$$

30 Hence,  $p_{AA_{x+1}} = 0.892$ ,  $p_{Aa_{x+1}} = 0.105$  and  $p_{aa_{x+1}} = 0.003$ .

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32 The frequency of deleterious allele in generation  $x+1$  is  $p_{a_{x+1}} = 0.5 * p_{Aa_{x+1}} + p_{aa_{x+1}}$  and  
33 mean relative fitness  $w_{x+1} = w_{AA} * p_{AA_{x+1}} + w_{Aa} * p_{Aa_{x+1}} + w_{aa} * p_{aa_{x+1}}$ . Substituting the  
34 proportions calculated above,  $p_{a_{x+1}} = 0.06$  and  $w_{x+1} = 0.94$  in both populations. Thus, despite

35 the alignment of fecundity selection and sexual selection (*i.e.* allele *a* being harmful both for  
36 female fecundity and male competitiveness) in the sexual population, deleterious allele is  
37 eliminated with exactly the same efficiency as in the asexual population. In other words,  
38 sexual selection does not contribute to paying the costs of sex.

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40 Now consider what could happen if we manipulated the intensity of sexual selection in two  
41 experimental populations derived from P2. In one treatment (E1) we introduce enforced  
42 monogamy by randomly pairing males and females and only allowing reproduction within  
43 these pairs. This treatment eliminates sexual selection, and hence – turns off selection against  
44 *a* in males. Thus, male gamete frequencies in E1 are determined solely by male genotype  
45 frequencies:

$$46 \quad gA_m = pAA_x + 0.5 * pAa_x$$

$$47 \quad ga_m = 0.5 * pAa_x$$

48 Female gamete frequencies are the same as in P2, and the resulting genotype frequencies in  
49 the next generation are  $pAA_{x+1} = 0.850$ ,  $pAa_{x+1} = 0.144$  and  $paa_{x+1} = 0.006$ , frequency of  
50 deleterious allele  $pa_{x+1} = 0.08$  (higher than in P1 and P2) and relative mean fitness  $w_{x+1} =$   
51  $0.92$  (lower than in P1 and P2).

52

53 In the other treatment (E2) we increase the intensity of sexual competition between males, for  
54 example by increasing the male:female sex ratio. Let us assume that the intensified  
55 competition completely prevents *Aa* males from breeding, such that frequency of *a* is 0  
56 among successful sperm. In the next generation,  $pAA_{x+1} = 0.944$ ,  $pAa_{x+1} = 0.056$  and  $paa_{x+1} =$   
57  $0$ ,  $pa_{x+1} = 0.03$  (lower than in P1 and P2) and relative mean fitness  $w_{x+1} = 0.97$  (higher than in  
58 P1 and P2).

59 Thus, comparing these two treatments, we could conclude that sexual selection helps clearing  
60 the deleterious mutation load – which indeed it does. However, if we went further to conclude  
61 that it contributes to offsetting the cost of sex, we would be wrong: there is no difference in  
62 mutation load between P1 and P2.

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