

ASSORTATIVE MATING BY FITNESS AND SEXUALLY ANTAGONISTIC GENETIC VARIATION

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Recent documentations of sexually antagonistic genetic variation in fitness have spurred an interest in the mechanisms that may act to maintain such variation in natural populations. Using individual-based simulations, I show that positive assortative mating by fitness increases the amount of sexually antagonistic genetic variance in fitness, primarily by elevating the equilibrium frequency of heterozygotes, over most of the range of sex-specific selection and dominance. Further, although the effects of assortative mating by fitness on the protection conditions of polymorphism in sexually antagonistic loci were relatively minor, it widens the protection conditions under most reasonable scenarios (e.g., under heterozygote superiority when fitness is averaged across the sexes) but can also somewhat narrow the protection conditions under other circumstances. The near-ubiquity of assortative mating in nature suggests that it may contribute to upholding standing sexually antagonistic genetic variation in fitness.

KEY WORDS: Heterozygote excess, homogamy, mate choice, polymorphism, population genetics, sexual conflict, sexual selection.

A series of recent empirical studies have revealed that a sizeable proportion of the standing genetic variation in fitness is sexually antagonistic (henceforth, SA) (Chippindale et al. 2001; Fedorka and Mousseau 2004; Pischedda and Chippindale 2006; Brommer et al. 2007; Foerster et al. 2007; Prasad et al. 2007; Bilde et al. 2009; Delcourt et al. 2009; Innocenti and Morrow 2010a). Although this observation is very consequential (Bonduriansky and Chenoweth 2009), it is somewhat of a puzzle because the conditions under which SA polymorphism should be maintained have been thought to be fairly restrictive, because the allele for which sex-specific selection is strongest should be driven to fixation (Prout 2000): polymorphism requires either strong SA selection of similar magnitude in the two sexes (Kidwell et al. 1977), X-chromosome linkage of SA loci (Rice 1984; Patten and Haig 2009; but see Hedrick and Parker 1997) or unequal dominance of alleles in the two sexes (Kidwell et al. 1977; Fry 2010). However, selection in nature is typically weak (Kingsolver et al. 2001)

and is often very different in magnitude in males and females (Cox and Calsbeek 2009; Innocenti and Morrow 2010b). Moreover, it is currently unclear whether SA alleles are commonly X-linked (Fry 2010; Innocenti and Morrow 2010a). Kidwell et al. (1977) showed that heterozygote superiority when fitness is averaged across the sexes (Fig. 1C) is particularly favorable to the maintenance of SA polymorphism and Fry (2010) suggested this form of sex-specific dominance of SA alleles may be common for fitness-related traits. Further, Patten et al. (2010) showed that the conditions under which SA polymorphism is maintained are expanded when SA variation is due to multiple linked loci. However, previous work in this field is all based on the assumption of random mating, which greatly simplifies theoretical analyses but may rarely be valid. Recent theoretical explorations of related domains have revealed a role for assortative mating by fitness in the purging of deleterious mutations (Rice 1998) and for the evolution of recombination (Blachford and Agrawal 2006). Here,

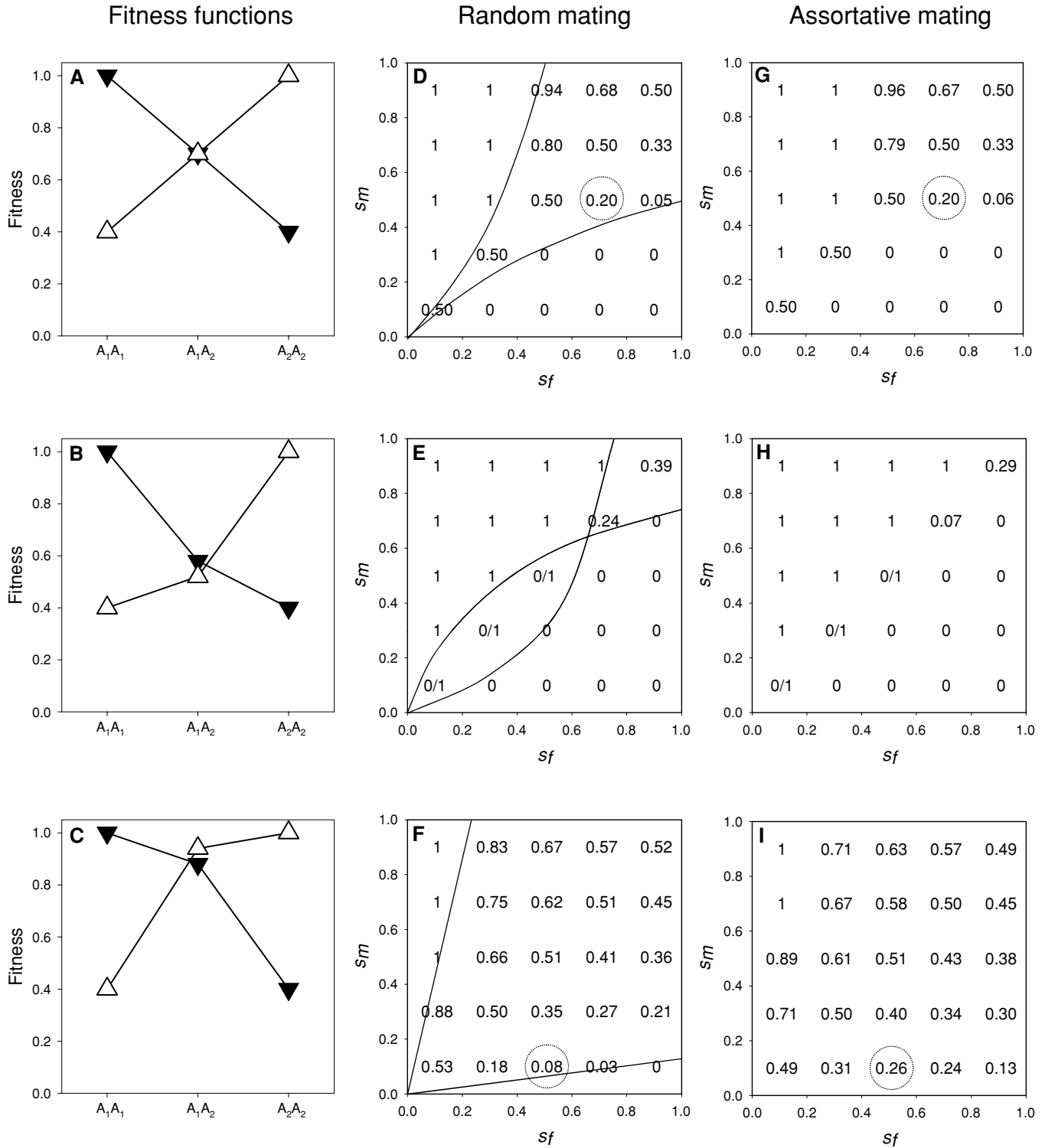


Figure 1. The maintenance of genetic variation at an autosomal locus with two sexually antagonistic alleles, one of which (A_1) is favored in males and another (A_2) which is favored in females. Previous theory identified three equilibrium regimes under random mating (Kidwell et al. 1977) that differ by the dominance relationships in males and females. Representative scenarios of these three regimes are illustrated here. First, the top panel row (A, D, and G) exemplifies equal dominance for a particular allele in the two sexes (i.e., $h_m + h_f = 1$) (here, $h_f = 0.5$; $h_m = 0.5$). Second, the middle row (B, E, and H) describes heterozygote disadvantage on average across the sexes (i.e., $h_m + h_f > 1$) (here, $h_f = 0.8$; $h_m = 0.7$) and, third, the bottom row (C, F, and I) illustrates a case where there is heterozygote advantage on average across the sexes (i.e., $h_m + h_f < 1$) (here, $h_f = 0.1$; $h_m = 0.2$). The leftmost panel column illustrates the shape of the fitness functions for females (open triangles) and males (closed triangles) that result from these particular dominance relationships

I use individual-based simulations to explore the effects of positive assortative mating by fitness on SA genetic variation. Because assortative mating under SA genetic variation leads to an overrepresentation of matings between different types of homozygotes, there are good reasons to believe that assortative mating may affect SA genetic variation.

Model

The conditions for the maintenance of SA genetic variation under random mating were originally delineated by Kidwell et al. (1977) for autosomal loci and by Rice (1984) and, more recently, Patten and Haig (2009) for sex-linked loci. I employed individual-based simulations to examine the role of assortative mating, because of the difficulties involved with assessing the effects of non-random mating in this context analytically (Caballero and Hill 1992). I used the simulation program simuPOP (version 1.0.5svn) (Peng and Kimmel 2005) which offers a versatile individual-based forward-time modeling environment for advanced evolutionary simulations (Peng et al. 2007), including patterns of nonrandom mating (Peng and Amos 2008).

I explored a scenario in a sexually reproducing diploid species with separate sexes where, at a single autosomal locus with standard Mendelian inheritance and zero mutation rate, one allele (A_1) is favored by selection in males and another (A_2) is favored in females. Following Kidwell et al. (1977), the most fit genotype of each sex was given the relative fitness of 1 and s_f and s_m represent the selection coefficients against the less-fit homozygote in females and males, respectively. The sex-specific dominance parameters, h_f and h_m , represent the dominance of the less-fit allele in females and males, respectively, and thus refer to the dominance of different alleles in the two sexes (ranging from $h = 0$ [recessive] over $h = 0.5$ [additive] to $h = 1$ [dominant]). The resulting sex-specific relative fitness values are given in Table 1.

The parameter space simulated covered all permutations of the values 0, 0.1, 0.3, 0.5, 0.7, 0.9, and 1 for the four parameters h_f , s_f , h_m , and s_m . For h_f and h_m , this range captures standard sexual antagonism and it mirrors previous theory (Kidwell et al. 1977; Rice 1984; Patten and Haig 2009; Fry 2010; Patten et al. 2010).

(shown here for $s_m = s_f = 0.6$). The central column shows the portion of parameter space (for varying selection intensities in females [s_f] and males [s_m]) with protected allelic polymorphism under random mating. The lines in the middle column delineate the upper and lower boundary regions for equilibria under random mating, given by the analytical expressions of Kidwell et al. (1977). Numbers represent the results of the individual-based simulations: "1" denotes the fixation of A_1 , "0" the loss of A_1 , "0/1" the conditional loss/fixation of A_1 depending upon the starting frequency of A_1 , and any decimal number represents the average (across replicate runs) frequency of the male benefit allele A_1 (i.e., P) after 1000 generations at a single stable polymorphic equilibrium. As illustrated in the middle column, the simulations validated the three equilibrium regimes predicted by Kidwell et al. (1977). The rightmost panel column shows the portion of the same parameter space with allelic polymorphism under assortative mating by fitness, using the same denotations. The simulations showed that assortative mating by fitness always expands upon the region with protected polymorphism whenever $h_m + h_f < 1$ and that it more generally elevates the frequency of heterozygotes in the population (Fig. 2). Dotted circles denote the points of parameter space illustrated in Figure 2.

Table 1. Sex-specific relative fitness set.

| | Genotype | | |
|---------|----------|-------------|----------|
| | A_1A_1 | A_1A_2 | A_2A_2 |
| Females | $1-s_f$ | $1-h_f s_f$ | 1 |
| Males | 1 | $1-h_m s_m$ | $1-s_m$ |

All simulation runs involved 10 independent replicate populations with a stable population size of 5000 individuals each, to enable verification of uniformity across replicate runs and minimize the impact of genetic drift, and were run for 1000 generations to ensure that allele frequencies equilibrated. Points of parameter space in which both alleles remained at some nonzero frequency in all replicate populations after 1000 generations were deemed to show protected polymorphism.

All points of the explored parameter space were simulated with starting frequencies of the A_1 allele of $P = 0.5$, $P = 0.1$, and $P = 0.9$, to assess internal stability of equilibria. I also seeded a large number of simulations of selected points of parameter space with a very low or high frequency of A_1 ($P = 0.005$ or $P = 0.995$) and these analyses confirmed that alleles invaded when rare (under conditions with stable equilibria) unless lost by genetic drift.

Kidwell et al. (1977) delineated three main equilibrium regimes under random mating, differing by the pattern of sex-specific dominance (i.e., $h_m + h_f < 1$, $h_m + h_f = 1$ and $h_m + h_f > 1$). To validate these regimes and to verify the performance of the simulations, I first ran all simulations under random mating. Here, adult males and females were paired randomly (with replacement) and each mating produced a single offspring. I then introduced assortative mating by fitness by first sorting all adults into quartiles of the fitness distribution (i.e., 1–25, 26–50, 51–75, and 76–100%), separately for the two sexes, at every generation. Adult males selected at random were then paired with an adult female drawn at random from within the same female fitness quartile (i.e., 1st, 2nd, 3rd, or 4th), such that co-reproducing males and females derived from the same sex-specific fitness quartiles. Thus,

mating was assortative across fitness quartiles but random within fitness quartiles and no fitness quartile had a deterministically higher sex-specific per capita probability of mating than the others. I note that McNamara and Collins (1990) predicted that mutual mate choice would result in precisely such a mating pattern. As for the random mating case, each mating produced a single offspring. Under both random and assortative mating, natural selection was imposed by making survival of offspring to adulthood proportional to their relative fitness. Here, the relative fitness of each offspring individual (Table 1) was considered a probability to survive, such that an individual offspring was discarded if its relative fitness value was smaller than a random number drawn for each individual offspring from a standard uniform distribution. Mating continued until full recruitment of offspring to the adult mating pool of the next generation was reached (i.e., $N = 5000$ adults). Principal examples of simuPOP scripts used are provided as Supporting information.

Results

The analyses validated the protection conditions delineated by Kidwell et al. (1977) for polymorphism at a single autosomal locus and under random mating (e.g., Fig. 1D–F). Assortative mating by fitness had two general effects on SA genetic variation. First, assortative mating affected the area of parameter space with protected SA polymorphism, but the effect was contingent upon the pattern of sex-specific dominance. Under equal dominance in the two sexes (i.e., $h_m + h_f = 1$), assortative mating did not noticeably alter the protection conditions (e.g., Fig. 1A, D and G). Under sex-specific dominance where $h_m + h_f < 1$, including but not restricted to scenarios with heterozygote superiority when fitness is averaged across the sexes (Fry 2010), assortative mating marginally expanded upon the region showing protected polymorphism under random mating (e.g., Fig. 1C, F and I). In contrast, in those regions defined by $h_m + h_f > 1$, assortative mating instead somewhat reduced the region with protected polymorphism (e.g., Fig. 1B, E and H).

Second, assortative mating by fitness generally increased the equilibrium frequency of heterozygotes for loci under SA selection over that observed under random mating (excluding cases where $h_m + h_f > 1$). This effect occurred both under additivity (Fig. 2A) as well as other forms of equal dominance in the two sexes, but was especially sizeable under protected polymorphism where $h_m + h_f < 1$ (Fig. 2B).

Discussion

The diversifying effects of assortative mating by fitness on SA genetic variation documented here are, in fact, quite intuitive, because assortative mating by fitness means that mating is dis-

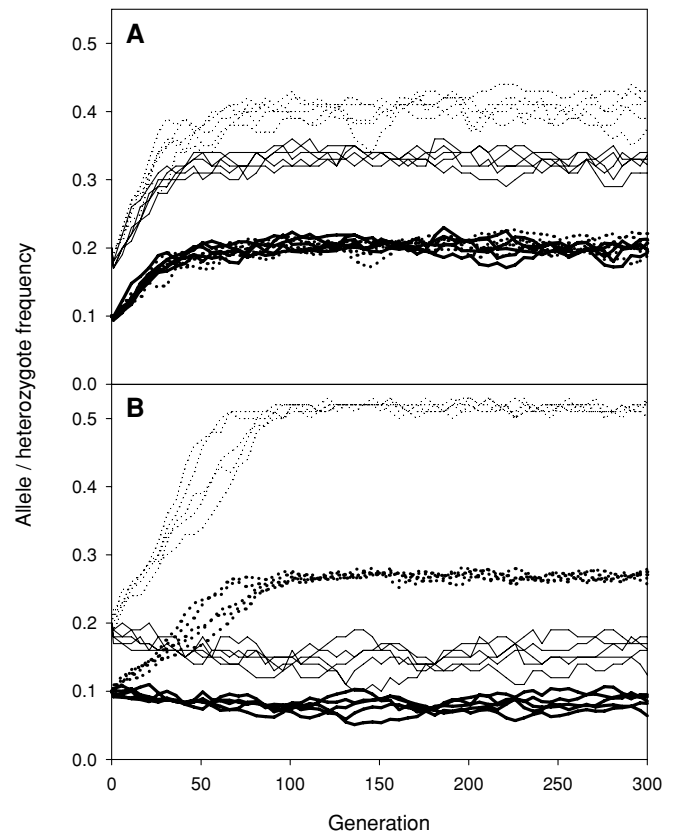


Figure 2. The frequency of the male benefit allele A_1 (thick lines) and of heterozygotes (thin lines) under random mating (solid lines) and assortative mating by fitness (dotted lines). The top panel (A) illustrates additivity ($h_f = 0.5$; $h_m = 0.5$; $s_f = 0.7$; $s_m = 0.5$) and the bottom panel (B) a case where $h_m + h_f < 1$ ($h_f = 0.1$; $h_m = 0.2$; $s_f = 0.5$; $s_m = 0.1$) (see dotted circles in Fig. 1). Even in cases of equal dominance in the two sexes where assortative mating by fitness does not alter the protection conditions or the equilibrium allele frequency, it markedly elevates the frequency of heterozygotes (A). This effect is even more dramatic in cases where $h_m + h_f < 1$ (B). The figure shows five arbitrary replicate runs of each scenario; all have a starting frequency of $P = 0.1$, population size = 5000 and frequencies were sampled every five generations.

sortative at the genotypic level for these loci. An overrepresentation of matings between males and females with similar fitness (i.e., matings between distinct homozygotes) will thus provide an increased input of heterozygotes in the population and matings between heterozygotes with intermediate fitness will keep generating both types of homozygotes. However, it is clear that the effects of assortative mating depend on the pattern of dominance of SA alleles in males and females.

I am unaware of any direct empirical data on the pattern of sex-specific dominance of SA alleles. Because most known major mutations causing discrete phenotypes (e.g., diseases, phenotypic markers) are equally dominant/recessive in both sexes, it might seem reasonable to assume that most SA mutations should show

equal dominance. Yet, there are many examples of quantitative traits showing sex-specific dominance in humans (e.g., Weiss et al. 2005; Pan et al. 2007) and Fry (2010) suggested that cases where $h_m + h_f < 1$ may be the norm for SA genetic variation because fitness functions should tend to be concave around sex-specific trait optima: whichever allele is beneficial in a given sex, this allele should be partly dominant in that sex (see Fig. 2 in Fry 2010). If this is true, assortative mating should commonly expand the protection conditions of SA polymorphism in natural populations.

The most general and sizeable effect of assortative mating by fitness is an increase in the amount of sex-specific genetic variation in fitness, occurring in most regions of the explored parameter space. This effect comes about as a result of assortative mating increasing the equilibrium frequency of heterozygotes in the population relative to random mating (Fig. 2). In addition, assortative mating interacts with selection to increase the equilibrium frequency of the rarer allele in cases where $h_m + h_f < 1$ (Fig. 1F vs. I). It is well known that negative assortative mating by genotype generates heterozygote excess (Hedrick 2009), along with other mechanisms such as overall heterozygote superiority (Dobzhansky 1955) and a low effective population size (Pudovkin et al. 1996). However, the fact that heterozygote excess should be a hallmark of SA genetic variation under positive assortative mating by fitness has previously been unappreciated. Assortative mating for loci showing SA genetic variation will thus impede genetic differentiation and speciation, in contrast to other types of loci where assortative mating instead decreases the frequency of heterozygotes and increases the probability of speciation (e.g., Gavrillets 2003).

Positive assortative mating by phenotypic traits is virtually ubiquitous in natural populations of most animals (Cézilly 2004), ranging from arthropods (Crespi 1989) and fish (McKaye 1986) through birds (Jawor et al. 2003) and reptiles (Olsson 1993) to mammals (Little et al. 2003), and is frequently strong. A large number of different processes can lead to mating assortment. These include mate choice by either or both sexes, intrasexual competition, variation in mate availability, resource competition, mating constraints and/or various combinations of these (Crespi 1989; McNamara and Collins 1990; Arnqvist et al. 1996; Fawcett and Johnstone 2003). Importantly, assortment normally occurs on the basis of traits that reflect phenotypic condition, such as body size, coloration, ornamentation, general vigor or persistence/resistance. Because such traits tend to reflect variation in underlying genetic quality (Rowe and Houle 1996; Hunt et al. 2004; Tomkins et al. 2004) and because mate choice itself may often be condition-dependent in both sexes (Fawcett and Johnstone 2003; Härdling et al. 2008), I suggest that some degree of assortative mating by fitness should be near ubiquitous in natural populations (Fawcett and Johnstone 2003; Blachford and Agrawal 2006; Sharp and Agrawal 2009).

Because fitness itself is always under directional selection, evolutionary theory predicts that selection should exhaust genetic variation for fitness (Fisher 1918) and reconciling this fact with the frequent empirical observation of sizeable levels of additive genetic variation in fitness components is a long-standing challenge in evolutionary biology (Ellegren and Sheldon 2008). In light of the recent empirical studies mentioned at the outset of the introduction, it seems likely that SA selection may be an important promoter of genetic variation in fitness (Kidwell et al. 1977). Although the conditions under which opposing selection between the sexes will protect autosomal SA polymorphism were originally thought to be quite restricted (Prout 2000), sex-specific dominance for fitness (Fry 2010) and polygenic sexual antagonism involving several linked loci (Patten et al. 2010) markedly expand upon these conditions. In addition, I have shown here that assortative mating by fitness can both elevate SA genetic variance and expand the protection conditions of SA polymorphism. Further, although the current analyses are restricted to the single-locus case, it seems likely that assortative mating will have even stronger effects on SA genetic variation when SA involves multiple genes. This is chiefly because the linkage that is predicted to evolve across loci under SA selection (Úbeda et al. 2011) should increase both total selection on particular alleles (Patten et al. 2010) and the degree to which positive assortative mating by fitness is negatively assortative at the genotypic level.

In conclusion, theory now suggests that SA selection is capable of maintaining SA autosomal genetic variation for fitness under a fairly wide range of conditions. This rests on the arguably reasonable assumptions that dominance for fitness is often sex-specific (Fry 2010), SA antagonistic variation is polygenic (Patten et al. 2010; Úbeda et al. 2011) and/or mating is assortative by fitness. Although recent work in *Drosophila* has shown that SA genetic variation is highly polygenic (e.g., Innocenti and Morrow 2010a), I am unaware of any direct data on the pattern of dominance for fitness or assortative mating by fitness for SA alleles. There is clearly a need for more empirical studies addressing these issues.

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