Evolutionary Ecology Research, 2003, **5**: 1–18

Assortative mating and plant phenology: evolutionary and practical consequences

Gordon A. Fox*

Department of Biology (SCA110) and Department of Environmental Science and Policy, University of South Florida, 4202 E. Fowler Avenue, Tampa, FL 33620, USA

ABSTRACT

Variation in mating phenology causes assortative mating for phenological traits. Here I show that this assortative mating can be strong, as it is caused not only by periods of non-overlap in flowering, but also by variation in the composition of the mating pool during periods of overlap. Using a one-locus, two-allele model, I show that this temporal assortative mating can: (1) lead to declines in mean fitness under balancing selection, including fixation of one allele; (2) strongly affect the rate of response to directional selection; and (3) determine the boundaries of the basins of attraction under disruptive selection. These results suggest that the evolution of phenological traits – and traits functionally coupled with them – may be more complex than we have generally thought. They also suggest caution in interpreting the results of studies (e.g. quantitative genetic studies) that assume randomly mating populations.

Keywords: flowering, life-history evolution, population genetics, timing.

INTRODUCTION

Plant reproductive timing and its evolution have been the focus of study for evolutionary ecology, quantitative genetics, pollination biology and plant breeding. Many studies have shown that reproductive timing often has strong fitness consequences (e.g. Fox, 1989; Kelly, 1992; Fox and Kelly, 1993). Seasonal variation occurs in environmental factors with major effects on plant survival and fecundity (Fox, 1989), as well as pollinator availability (Schmitt, 1983a; Bawa *et al.*, 1985), seed survival (Augspurger, 1983a) and herbivory (Pilson, 2000).

There is also a rapidly growing body of evidence on the genetics of flowering phenology. Quantitative genetic studies have repeatedly found heritable variation within populations (Campbell and Kondra, 1978; Fox, 1990; Kelly, 1992; Conner and Via, 1993; Mazer and LeBuhn, 1999). Studies of quantitative trait loci (QTLs) in several species – mainly Brassicaceae, maize and tomatoes – have identified genomic regions responsible for variation between strains (e.g. Ferreira *et al.*, 1995; Camargo and Osborn, 1996; Kuittinen *et al.*, 1997; Reba *et al.*, 1997). Typically, the number of inferred major QTLs has been fairly small

---

* e-mail: gfox@chuma1.cas.usf.edu

(often 3 or 4), raising the hope that we may soon achieve an understanding of the genetic basis of much of the variation in flowering time. Taken together, these ecological and genetic studies appear to point to an optimistic conclusion: we may soon have the essential information necessary for understanding the evolution of this key aspect of plant life histories.

Unfortunately, there are reasons for caution in drawing such optimistic conclusions. The complexity of real flowering phenologies, and the assortative mating induced by genetic variation in these phenologies, present a number of ecological and genetic problems. Here I discuss these problems, focusing in particular on the ecological ones.

Individual plants, as well as plant populations, often have complicated flowering phenologies. Individuals within a population sometimes cannot mate with one another at all, because of phenological differences (e.g. Schmitt, 1983a,b). In several species, flower production is bimodally distributed over time either within or between individuals, or both. For example, Fox (1989, 1990) studied *Eriogonum abertianum*, a desert annual in which some individuals flower in April–May, some in August–September and some in both seasons. Onset of flowering can vary by as much as 6 months within a population; occasionally, a third flowering season has been observed in this species (P. Chesson, personal communication). In experimental studies, onset of flowering appears most often to be gamma-distributed (Fox, 1990, 2001). Some 6% of annual plants in the Tucson Mountains of Arizona have bimodal flowering patterns (Rondeau *et al.*, 1996). Schedules of flower production can be multimodal for another reason: flowering in some species is on a branch-by-branch basis, in which branches begin flowering at separate times (Owens, 1995).

I consider the effects of this phenological complexity on populations. I focus on the way phenological variation leads to assortative mating for life-history traits (and other traits that advance or delay flowering; Lyons and Mully, 1992) and on how this assortative mating interacts with selection on phenological traits. I use the term ‘phenology’ to refer to flowering phenology.

Genetic variation for phenological traits leads to assortative mating for those traits. Thus, phenological traits evolve under the influence of both assortative mating and selection. By ‘assortative mating’ in this context, I mean that the phenotypes of mating individuals are correlated. Here I show that, when the mating pool has a temporal structure, we can usefully think of assortative mating as having two components. One component has received previous comment: reproductive activity of some individuals has limited temporal overlap. The other component is more subtle: even if individuals reproduce over the same period, differences in their schedules of reproductive activity always reduce their chances of mating. The net result is that assortative mating for phenological traits can be much stronger than one might expect based only on the amount of temporal overlap. A novel aspect of flowering time variation is that the degree of assortative mating depends on the genotypic composition of the population. I examine some of the joint consequences of strong assortative mating for, and selection on, a phenological trait. Finally, I consider practical consequences that strong assortative mating may have for the study of the evolution of phenological traits.

TWO FORMS OF TEMPORAL ASSORTATIVE MATING

Consider a simple model of assortative mating for phenology in an annual plant. Each individual of genotype i makes N_{it} flowers on day t. Each flower lasts 1 day, all flowers are pollinated and each flower produces a single seed with no seed or fruit abortion. On each day, there is random mating among all flowers that are open. Flowering time is controlled by one locus with two alleles. Now focus on the case where there are two genotypes in a population (e.g. a population fixed for the A allele has just come into contact with one fixed for the a allele). For notational simplicity, let x, y and z refer to the frequencies of AA, Aa and aa, respectively, and the genotypes themselves can be referred to as X, Y and Z, respectively. Definitions of all mathematical symbols are given in Table 1.

Temporal assortative mating can occur in either or both of two forms. I will call these 'standard' and 'cryptic' assortative mating. Examples of standard and cryptic assortative mating are shown in Fig. 1.

Standard assortative mating

Standard assortative mating occurs because two phenologies are offset by time: during the period when only X are flowering, all matings must be $X \times X$, and the reverse must be true when only Z are flowering. The net result is an excess of $X \times X$ and $Z \times Z$ matings, and a deficit of $X \times Z$ matings, compared with a randomly mating population.

As an example, assume that there are no differences in fecundity between the two types and they are of equal frequency. Each genotype produces, say, 100 flowers per day for 10 days, giving a total of 1000 flowers (and, by assumption, 1000 seeds). Under random mating, there would be 500 $X \times X$ matings, 1000 $X \times Z$ matings and 500 $Z \times Z$ matings. But if the two genotypes' flowering periods are offset from one another, there can be sizeable departures from these proportions, as shown in Table 2. I call this component 'standard' because this is what most people have in mind when they discuss assortative mating for phenological traits (e.g. Gale *et al.*, 1974; Augsperger, 1983b; Hartl and Clark, 1997).

Table 1. Definitions of symbols

Symbol	Definition
X, Y, Z; x, y, z	genotypes AA, Aa and aa, respectively; their relative frequencies
t	index for time
N_{it}	number of flowers made by individuals of type i on day t
θ_i	frequency of type-i flowers in the mating pool
$p_i(t)$; $\bar{p}_i$	frequency of genotype (or allele) i in mating pool on day t; its mean over the season
F, $\hat{F}$	Wright's inbreeding coefficient and its value at equilibrium
τ; d	unit of phenotypic value; degree of dominance
W_i; $\bar{W}$; s	fitness of genotype i; mean fitness; selection coefficient
H; A	equilibrial values: under Hardy-Weinberg assumptions and under assortative mating, respectively
$\bar{t}_0$	mean flowering time
σ^2, $\hat{\sigma}^2$	phenotypic variance and its equilibrial value
σ_A^2, $\sigma_A^2(0)$	additive genetic variance measured, respectively, in an assortatively mating and in a randomly mating population
h^2	heritability measured in a randomly mating population
ρ_p	phenotypic correlation among mates
n; γ	effective number of loci; shorthand for $1 - 1/(2n)$
θ^*, θ'	single parameters describing phenologies (see Figs 3 and 4)

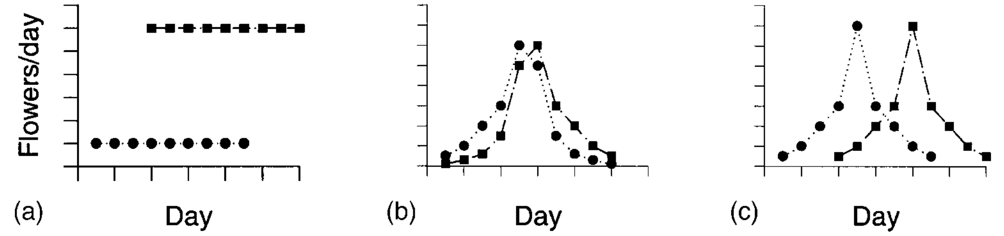


Fig. 1. Standard assortative mating (a) occurs when genotypes do not overlap in flowering. Cryptic assortative mating (b) occurs when there is overlap in flowering but there is day-to-day variation in the frequencies of flowers of each genotype. When two time-varying phenologies are offset from one another (c), both types of assortative mating occur.

Table 2. Examples of 'standard' assortative mating

	$X\times X$	$X\times Z$	$Z\times Z$
Offset = 1 day			
Total matings	550	900	550
E (matings | random)	500	1000	500
(Total – expected)/expected	0.10	−0.10	0.10
Offset = 3 days			
Total matings	650	700	650
E (matings | random)	500	1000	500
(Total – expected)/expected	0.30	−0.30	0.30

Note: Each genotype produces 100 flowers per day for 10 days, but the two flowering intervals are offset by either 1 or 3 days.

Cryptic assortative mating

Cryptic assortative mating occurs when there is variation in the frequencies of two genotypes during the flowering interval – even if all plants begin and end flowering simultaneously. Consider a simple example. Two genotypes flower over the same period and make the same numbers of flowers; X produces 20, 50 and 80 flowers over 3 days, and Z produces 80, 50 and 20 flowers. Naively one would expect that the frequency of $X\times Z$ matings should be $2\times 0.5^2 = 0.5$; after flowering ends, there would be 150 developing $X\times Z$ seeds. Table 3 shows that, at the end of the season, however, we have only 114 $X\times Z$ progeny.

The reason assortative mating occurs when flowering schedules differ is simple. Random mating in every day's mating pool means that every day t, the frequency of the three possible matings is $\{[x\theta_x(t)]^2, 2x\theta_x(t)z\theta_z(t), [z\theta_z(t)]^2\}$, where θ is the fraction of each genotype's flowers that is in the mating pool that day, and x and z are the frequencies of AA and aa, respectively. The total number of each type of mating (over the entire season) is given by the sum of these frequencies times the total number of flowers each day.

The total number of heterozygotes in this sum is guaranteed to be less than or equal to the total number of heterozygotes that would be obtained if the mating pool had no

Table 3. An example of cryptic assortative mating

						Number of matings		
Day (t)	N_x	N_z	$x\theta_x(t)$	$z\theta_z(t)$	$2x\theta_x(t)z\theta_z(t)$	$X \times X$	$X \times Z$	$Z \times Z$
1	20	80	0.2	0.8	0.32	4	32	64
2	50	50	0.5	0.5	0.50	25	50	25
3	80	20	0.8	0.2	0.32	64	32	4
Total						93	114	93
E(matings | random)						75	150	75
(Total – expected)/expected						0.24	−0.24	0.24

temporal structure. This is true as a general case, not just in unusual circumstances like those in Table 3. For example, in a population with two alleles, assume for the moment that every day the mating pool is in Hardy-Weinberg equilibrium, with $p_{\cdot}(t)$ the frequency of each allele in the mating pool on day t and $\bar{p}$ the average over the entire mating season of the daily frequencies. Then

$$\frac{1}{T}\sum_{t=1}^{T} p_a(t)p_A(t) < \bar{p}_a\bar{p}_A \tag{1}$$

with equality only if the p's are constant through the mating season. This guarantee is due to Jensen's inequality (Ruel and Ayres, 1999), and can be seen easily from the fact that the mean of a product of two varying quantities is the product of their means plus their covariance; since genotype frequencies sum to one, the covariance is negative. If there are more than two alleles in the population, equation (1) does not necessarily hold for any particular pair of alleles, but it remains true that the total number of heterozygotes will be smaller than in a random mating population. In other words,

$$\frac{1}{T}\sum_{t=1}^{T}\sum_{i\neq j} p_i(t)p_j(t) \leq \sum_{i\neq j} \bar{p}_i\bar{p}_j \tag{2}$$

Jensen's inequality also underlies the so-called Wahlund effect in population genetics.

Because previous discussion of temporal assortative mating has concerned standard assortative mating, it has generally underestimated the strength of assortative mating. Phenological schedules vary substantially within populations, so assortative mating may often be fairly strong. This can have important consequences.

In this paper, I am concerned with exploring the general consequences of temporal assortative mating and, therefore, treat all assortative mating as though it were standard, which permits enormous simplifications in the notation and mathematics. The importance of cryptic assortative mating for this paper is that, by recognizing its prevalence, we can appreciate the potential for strong assortative mating.

EVOLUTIONARY CONSEQUENCES OF ASSORTATIVE MATING

What are the evolutionary consequences of strong assortative mating, especially when combined with selection? I discuss the consequences of selection and assortative mating when each acts alone, and then the consequences of their joint action.

Under constant selection in a one-locus system, as is well known, the population evolves to a point at which mean fitness $\bar{W}$ is a local maximum. Under balancing selection, all genotypes remain in the population, whereas under directional or disruptive selection, fixation occurs. Aside from the Hardy-Weinberg equilibrium, these are probably the most widely known results in population genetics.

Assortative mating by itself causes evolutionary change

Under the present model, assortative mating acting by itself does not change allele frequencies, but it does generate an excess of homozygotes. The relative difference between heterozygote frequencies in the two cases is equal to Wright's inbreeding coefficient

$$F = \frac{2p(1-p) - y}{2p(1-p)}$$

where p is the allele frequency of A and y, as defined above, is the frequency of heterozygotes. In this context, F describes inbreeding at the A locus, not an average throughout the genome.

An excess of homozygotes is produced because only Z offspring are produced during the first part of the season, when only Z individuals are flowering, and only X offspring are produced at the end of the season, when only X plants are flowering. What may not be so obvious is that this change in genotype frequencies generally changes the phenotypic mean of the population as well as both the genetic and phenotypic variances of the population.

To examine changes in phenotype caused by this inbreeding, assume that Z individuals have a phenotypic value of 0, the X plants a phenotypic value of 2τ and the heterozygotes a phenotypic value of $(1 + d)\tau$. Thus τ is half the difference between the two homozygotes and d describes the degree of dominance. Let H denote values at Hardy-Weinberg equilibrium and A denote those at the assortative mating equilibrium. Let t_0 be the date of onset of

Table 4. Equilibrial results under assortative mating only (A) and at the Hardy-Weinberg equilibrium (H)

Quantity	Formula
Mean flowering time (A)	$2\tau(1-p)\,[1 + dp(1-\hat{F})]$
Mean flowering time (H)	$2\tau(1-p)(1+dp)$
Difference between means	$-2\hat{F}dp(1-p)\tau$
Phenotypic variance (A)	$2\tau^2\{p(1-p)(1-\hat{F})(1+d)^2$ $+ 2(1-p)^2\,[dp(\hat{F}-1)-1]$ $+ 2(1+p[\hat{F}-2-p(\hat{F}-1)])\}$
Phenotypic variance (H)	$2\tau^2 p(1-p)\,\{1 + d[d-2+4p-2dp(1-p)]\}$
Inflation of additive genetic variance (H)	$\dfrac{2 + \left[\sqrt{1-4\gamma\rho_p h^2(1-h^2)} - 1\right]/h^2}{2(1-\gamma\rho_p)}$

Note: See Table 1 for symbol definitions.

flowering. The results are shown in Table 4. The mean flowering date changes unless heterozygotes are intermediate ($d = 0$). The phenotypic variance is greater under assortative than random mating. This is best seen if $d = 0$: the mean is unchanged but the variance is increased by $2\hat{F}p(1 - p)\tau^2$.

As first pointed out by Wright (1921), assortative mating inflates the additive genetic variance of a population (Lynch and Walsh, 1998). Several studies – both traditional genetic studies and more recent QTL studies – suggest that modest numbers of loci (generally 2–4 major loci and a few minor ones) are involved in date of anthesis (Wehrhahn and Allard, 1965; Murfet and Reid, 1974; Murfet, 1975; McIntyre and Best, 1978; Baggett and Kean, 1989; Conner, 1993; Ferreira *et al.*, 1995; Camargo and Osborn, 1996; Kuittinen *et al.*, 1997; Reba *et al.*, 1997).

The number of effective loci is crucial for small n. Figure 2 shows the variance inflation as a function of n for intermediate values of h^2 and ρ_p; variance estimates can be inflated by 10% or so with n smaller than 5, modest assortative mating and weak heritability, or by 250% for 15 or 20 loci, strong assortative mating and large heritability. Variance inflation may be a reason populations often show considerable genetic variance for phenological traits, though closely tied to fitness. Thus, estimates of heritability of phenological traits should be interpreted with caution.

Effects of assortative mating and selection together

Assortative mating and selection can have important interactive effects. In the Appendix, I describe a model useful in examining this interaction. The central idea in the model is to partition the change in genotype frequencies into components caused by the time-structuring of the mating pool. Thus, if there is a period in which, say, Z plants alone are in the mating pool, this period contributes all $Z \times Z$ matings, and its contribution to the total change in frequency of Z plants is weighted by the fraction of Z flowers produced in this period.

Here I consider some simple cases of this model. There are two alleles at a single locus and constant selection. Plant phenologies are determined entirely by their genotype, but

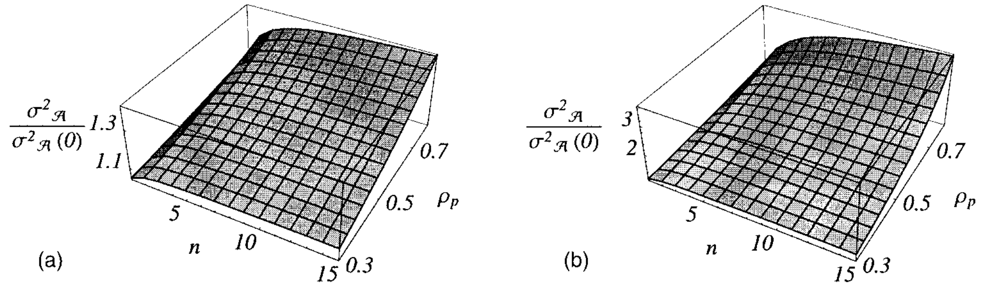


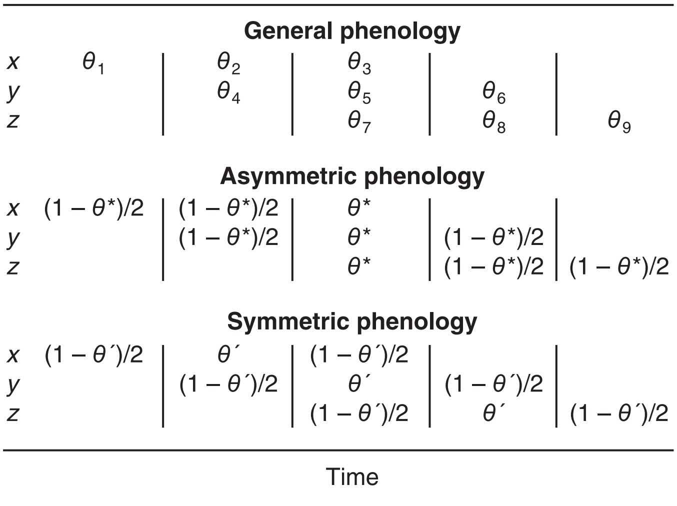
Fig. 2. Inflation of genetic variance as a function of effective number of loci n, phenotypic correlation between mates ρ_p, and heritability in a reference (randomly mating) population h^2. (a) $h^2 = 0.3$; (b) $h^2 = 0.8$.

since plants are assumed here not to be pollen-limited, the amount of assortative mating is frequency-dependent. I consider two types of phenologies, each characterized by a single parameter.

In the ‘asymmetric phenology’, θ^* is the fraction of flowers produced by each genotype during the period when all three genotypes are flowering (Fig. 3). Thus, $\theta^* = 1$ means no assortative mating and $\theta^* = 0$ means that the two homozygotes cannot mate with one another. This is ‘asymmetric’ because it means that the genotypes differ not only in their date of first flowering, but in their schedule of flower production as well.

By contrast, under the ‘symmetric phenology’, θ' is the fraction of flowers produced by each genotype during the middle period for each genotype (Fig. 3); $\theta' = 1$ means complete assortative mating. All genotypes have the same basic schedule of flower production, but they are offset from one another. As a simple example, consider genotypes that differ only in the threshold of a stimulus (such as daylength or number of degree-days) needed to flower. There is, thus, no value of θ' that corresponds to no assortative mating. When $\theta' = 1/3$, there is only standard assortative mating; when $\theta' = 0$, each genotype has a bimodal phenology (Fox, 1989; G. LeBuhn, personal communication), with half the flowers in the first period and half in the third. Consequently, there are x and z flowers being produced simultaneously.

These two models are simple, but nonetheless capture a wide range of possible ways temporal assortative mating can occur. The forms of selection and phenologies are summarized in Table 5 and Fig. 3. I studied the equilibrial properties of these models. To keep graphics manageable, I show the results only for $s \leq 0.1$. Larger values of s always produced qualitatively similar results.



General phenology

x	θ_1	θ_2	θ_3		
y		θ_4	θ_5	θ_6	
z			θ_7	θ_8	θ_9

Asymmetric phenology

x	$(1-\theta^*)/2$	$(1-\theta^*)/2$	θ^*		
y		$(1-\theta^*)/2$	θ^*	$(1-\theta^*)/2$	
z			θ^*	$(1-\theta^*)/2$	$(1-\theta^*)/2$

Symmetric phenology

x	$(1-\theta')/2$	θ'	$(1-\theta')/2$		
y		$(1-\theta')/2$	θ'	$(1-\theta')/2$	
z			$(1-\theta')/2$	θ'	$(1-\theta')/2$

Time

Fig. 3. Simple forms of assortative mating. Horizontal lines show each genotype’s flowering period, and mathematical expressions give the fraction of the genotype’s flowers produced in that period. (Top) General model; θ’s can take any value between 0 and 1, but θ’s for each genotype must sum to 1. (Middle) Asymmetric phenologies: θ^* is the fraction of each genotype’s flowers produced when all three genotypes are in flower. (Bottom) Symmetric phenologies: θ' is the fraction produced during each genotype’s middle period.

Table 5. Forms of selection and their parameterizations

	Genotypic fitness		
Selection	W_X	W_Y	W_Z
Balancing	$1 - s$	1	$1 - 2s$
Directional	$1 + s$	1	$1 - s$
Disruptive	$1 + 2s$	$1 - s$	$1 + s$

Balancing selection

Equilibrium values for $\hat{p}$ are shown in Fig. 4. Perhaps the most interesting result is that fixations can occur, though this is balancing selection. Fixations here require that the selection be asymmetric, but this is generally the case. More generally, $\hat{p}$ is always much greater than in the absence of assortative mating. In the symmetric phenology, there is a trough in the graph, corresponding to $\theta' = 1/3$, the case of no cryptic assortative mating. The peak on the right (for $\theta' = 0$) is the case of bimodal flowering schedule (Fox, 1990; G. LeBuhn, personal communication).

These results reflect a profound contrast with the random-mating case: the population no longer evolves to a point at which mean fitness is maximized. In fact, mean fitness can decline (Fig. 5) and fixation can occur. This result is surprising to many ecologists. Hayman (1953) showed that the interaction of selection and the mating system can lead to qualitatively different outcomes than would occur under either by itself. In particular, he studied a system related to the present one – models with heterozygote superiority and partial selfing – and showed that the outcomes included fixations as well as polymorphic equilibria with either more or fewer heterozygotes than under random mating.

Why do fixations occur, both in my model and in that of Hayman? The answer is that non-random mating reduces the frequency of heterozygotes, and selection reduces the

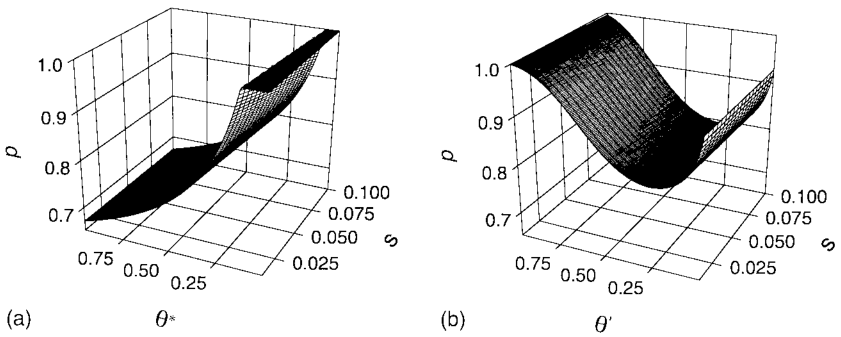


Fig. 4. Equilibrial gene frequencies $\hat{p}$ for the A allele under balancing selection. (a) Asymmetric phenologies: assortative mating increases monotonically with θ^*. (b) Symmetric phenologies: $\theta' = 0$ gives bimodal phenologies, while $\theta^* = 1$ gives complete assortative mating between the two homozygotes. See also Fig. 3.

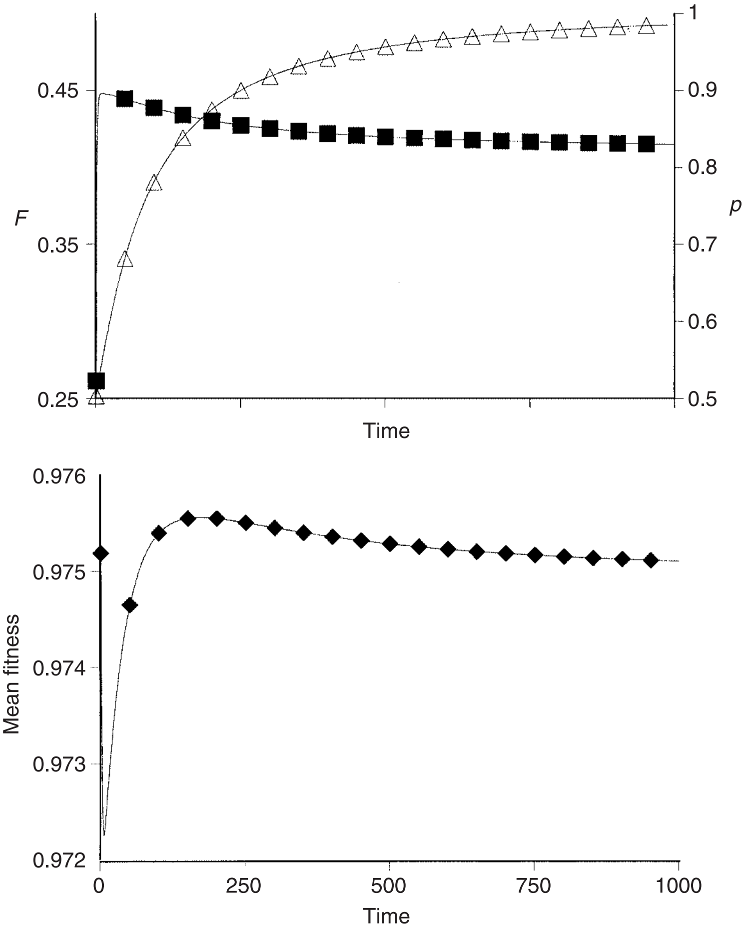


Fig. 5. Change in gene frequency p (△), in local inbreeding F (■) and in mean fitness $\overline{W}$ (◆) over time under balancing selection, with asymmetric phenologies. This example is one leading to fixation; those leading to polymorphic equilibria are similar except, of course, for the ending point. Examples for symmetric phenologies are qualitatively similar. Initial conditions were $p = 0.5$ at Hardy-Weinberg equilibrium.

frequency of the less fit homozygote. The net effect is to change the allele frequencies. If there are sufficiently few matings between heterozygotes, this can cause fixation. Fixations do not occur if non-random mating is weaker (smaller θ^* or more intermediate θ'). In the present model, there are always far fewer Y's and Z's than in a randomly mating population. Mean fitness declines because the most fit individuals are, by definition, Y's, whose numbers decline.

The evolutionary outcome – the final values of $\hat{p}$ and $\hat{F}$ – clearly depends more strongly on assortative mating (θ) than selection (s). This remains true even when s is as large as 0.25 (not shown). Finally, the time to equilibrium depends much more strongly on assortative mating than selection (Fig. 6).

Directional selection

Under directional selection, the effect of assortative mating is to reduce the time to fixation (Fig. 7). Because assortative mating inflates the heritability, the evolutionary response is faster. This result has been extensively used by breeders, although not, to my knowledge, in selecting on phenological traits.

Disruptive selection

Under disruptive selection, the outcome is still a fixation that depends on initial conditions, but the basins of attraction for $p = 1$ and $p = 0$ also depend on the strength of assortative mating. As with the other cases, assortative mating has a strong effect on the time to

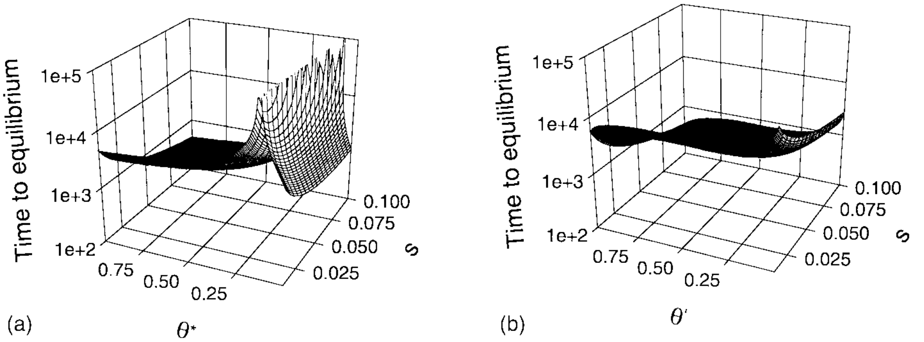


Fig. 6. Time to equilibrium under balancing selection and assortative mating. (a) Asymmetric phenology. The discontinuity is between the fixation (small θ^*) and polymorphic (larger θ^*) equilibria. (b) Symmetric phenology. In both cases, assortative mating has strong effects on the time to equilibrium, and they are not monotonic. See Figs 3 and 4 for meanings of θ^* and θ'.

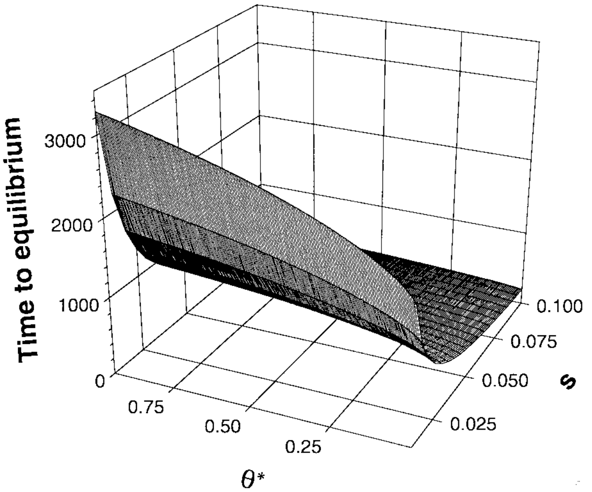


Fig. 7. Time to equilibrium under directional selection, with asymmetric phenology. Fixations occur more quickly under stronger assortative mating. The figure for symmetric phenology is similar. See Figs 3 and 4 for meaning of θ^*.

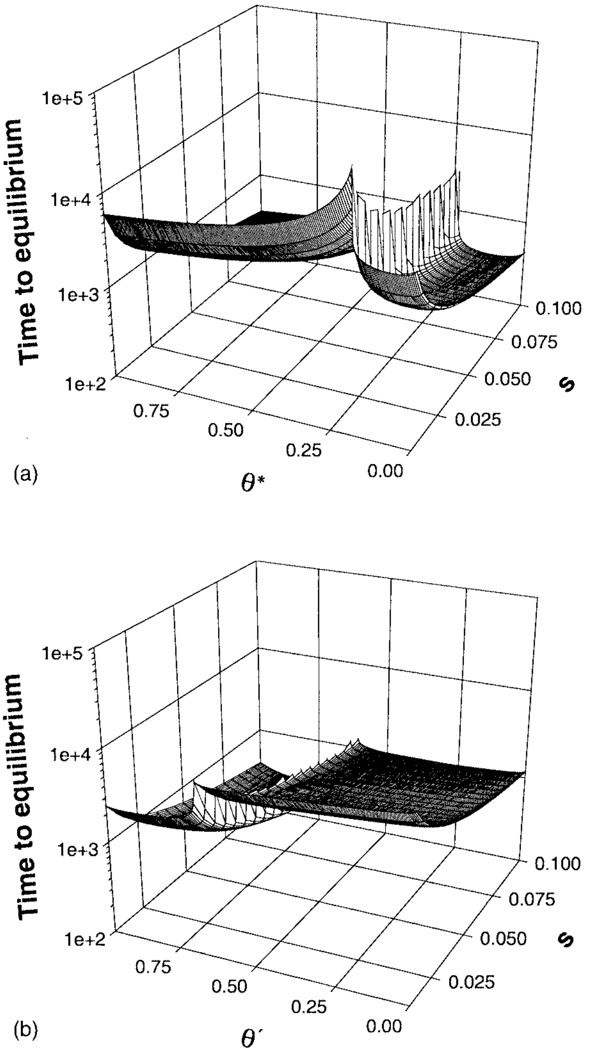


Fig. 8. Time to fixation under disruptive selection and both (a) asymmetric and (b) symmetric phenologies. The discontinuities are caused by the boundaries of the basins of attraction of the two fixation equilibria. See Figs 3 and 4 for meanings of θ^* and θ'.

equilibrium. Figure 8 shows the time to equilibrium with both asymmetric and symmetric phenologies, for the initial condition $(x, y, z) = (0.025, 0.0725, 0.9025)$. The discontinuity corresponds to the boundaries of the two basins of attraction: points to the right are attracted to $p = 0$, while points to the left are attracted to $p = 1$.

DISCUSSION

The evolution of phenological traits depends on selection – the ecological consequences of a phenology – and on the strength of assortative mating (i.e. on how different the alleles in a population are from one another). This can cause rapid evolution, since assortative mating accelerates the response to directional selection. However, assortative mating also imposes limits to the fine-tuning of such adaptations through balancing selection: mean fitness is not maximized under assortative mating and balancing selection. Assortative mating also has large effects on directional and disruptive selection, affecting the rate of evolutionary response and, for disruptive selection, defining the boundaries of basins of attraction.

Assortative mating may have an additional important consequence. By restricting gene flow between phenologically distinct groups in a population, assortative mating may make it easier for correlated adaptations to evolve. For example, early- and late-flowering individuals confront different herbivores, pathogens and weather conditions. Evolution of, say, both early-flowering, frost-hardy individuals and of late-flowering, heat-resistant individuals would be difficult in a random-mating population, but assortative mating is likely to make it easier. This speculation is supported by multilocus simulations (G.A. Fox, unpublished).

Temporal assortative mating is different from other forms of assortative mating. The strength of temporal assortative mating depends on the genotypic composition of the population. Karlin (1969; see also Gregorius, 1984) studied systems in which all mating occurs simultaneously. He concluded that, unless two phenotypes assort at the same frequency, one of them (the 'choosier') ultimately becomes fixed. This is because individuals are assumed to assort with fixed probability. By contrast, the temporal structure of the mating pool actually protects the less choosy type. For example, under standard assortative mating, the less choosy type – one that flowers for a shorter period and, therefore, has a greater proportional overlap with the other type – is guaranteed a period of non-overlapping flowering in which all matings have to be with its type. Moreover, during the period of overlap, there would still be an excess of like × like matings because (assuming no selection) the less choosy type would produce more flowers during this period than the more choosy one. Thus, temporal assortative mating generally increases variation (see 'Effects of assortative mating by itself'); by contrast, the type of assortative mating studied by Karlin and Gregorius generally destroys it. This result may depend on the assumption that all flowers have the same probability of mating.

Consequences for study

I suggested above that assortative mating for phenological traits is ubiquitous. Changing any phenological trait is almost certain to induce assortative mating for that trait and possibly for others as well. This implies that traits like 'date of anthesis' and 'date of last flower' generally cannot evolve independently from one another, or from the schedule of flower production. Note that I am not claiming that there is some sort of necessary functional constraint that generates correlations between the beginning, ending and intervening events in a reproductive phenology. We do not yet know enough about the underlying biological mechanisms to speculate about this. But, if changing one trait – say, date of anthesis – almost always generates assortative mating not only for that trait but also in some of the others – say, the rate at which flowers are produced – then we are making a mistake to think of these as single traits evolving in isolation from one another. We need to begin to describe and compare entire phenologies quantitatively.

Quantifying assortative mating requires data throughout the season, not just data on the timing of first and last flowers. At the very least, we need to know the dates of first and last flower and to have a description of the flowering schedule between these dates. If we were lucky, we might be able to achieve such a description with a function and a parameter – in other words, we might be able to quantify assortative mating with as few as four pieces of information.

There is no reason to suppose that we will generally be so lucky. The data in Meagher and Delph (2001) or in G. Fox and C. Pickering (in prep.) certainly do not suggest we should be optimistic, but at this point we can only speculate. Almost all empirical studies of which I am aware, including my own (Fox, 1989, 1990), have concentrated on only one or two pieces of this minimum description. Most authors have studied either the date of anthesis or the date of peak flower production; a few have also studied the date of last flower.

Empirical studies of temporal assortative mating face some daunting problems. A naive approach might be to study the genotype frequencies of seeds at the end of the season. But this would also be affected by several forms of selection. For example, if granivores are most active just when genotypes X and Y have matured their fruits, we might erroneously attribute the dearth of these genotypes and their crosses to assortative mating, rather than granivory. There is no way out of this kind of problem at present. We cannot count genotypes right after mating because there are no non-invasive ways to be certain whether fertilization has occurred, much less to identify the pollen donor. Among the ecologically important phenomena that can easily confound empirical studies of assortative mating are variations during the course of the flowering season in fertility, pollinator activity, seedset, seed viability, seed and/or fruit abortion, and granivory.

Thus, it may be most practical at present to study the opportunity for assortative mating (variation in phenologies themselves), rather than the frequencies of different mating types. With estimates of complete phenologies, it is straightforward (G. Fox and C. Pickering, in prep.) to quantify the opportunity for assortative mating.

Care must be taken when designing and interpreting quantitative genetic studies of phenological traits or traits closely correlated with them, such as growth rates. Standard quantitative genetic theory (Lynch and Walsh, 1998) assumes a random-mating parental population. Mitchell-Olds and Rutledge (1986) pointed to important biases when this is violated in experimental studies, and researchers generally take great pains to minimize these biases. For phenological traits, this presents a problem: if one can ensure random mating among parents (which may be impossible in a population with sufficiently divergent flowering times), how does one interpret the ensuing estimates of heritabilities? These may have little to do with the transmission of genes in natural populations, precisely because natural mating is assortative. Nor would such an experiment necessarily estimate the same quantities as an experiment in which one permitted phenological assortative mating. Thus, an important challenge is to develop quantitative genetic theory that can accommodate assortative mating.

I have argued that we cannot understand the evolution of phenological traits without thinking in terms of assortative mating. Since phenological traits are major components of fitness, by itself this would justify an effort to understand and quantify temporal assortative mating. But there is a larger reason for doing so. E. Lyons (personal communication) has pointed out that because ‘stressors’ like drought, herbivory, disease and competition tend to affect flowering time, plant responses to these stressors necessarily evolve under the influence of assortative mating. This suggests that understanding temporal assortative mating may be an important part of understanding several aspects of plant population biology.

ACKNOWLEDGEMENTS

G. LeBuhn, C. Pickering and P. Chesson provided useful criticism. I thank E. Lyons for many enlightening discussions and B. Kendall and S. Schwinning for comments on an early draft of the manuscript.

REFERENCES

Augsperger, C.K. 1983a. Offspring recruitment around tropical trees: changes in cohort distance with time. *Oikos*, **40**: 189–196.

Augsperger, C.K. 1983b. Phenology, flowering synchrony, and fruit set of six neotropical shrubs. *Biotropica*, **15**: 257–267.

Baggett, J.R. and Kean, D. 1989. Inheritance of annual flowering in *Brassica oleracea*. *Hort. Sci.*, **24**: 662–664.

Bawa, K.S., Perry, D.R. and Beach, J.H. 1985. Reproductive biology of tropical lowland rainforest trees. I. Sexual systems and incompatibility mechanisms. *Am. J. Bot.*, **72**: 331–345.

Camargo, L.E.A. and Osborn, T.C. 1996. Mapping loci controlling flowering time in *Brassica oleracea. Theor. Appl. Genet.*, **92**: 610–616.

Campbell, D.C. and Kondra, Z.P. 1978. A genetic study of growth characters and yield characters of oilseed rape. *Euphytica*, **27**: 177–183.

Conner, J. 1993. Tests for major genes affecting quantitative traits in wild radish, *Raphanus raphanistrum. Genetica*, **90**: 41–45.

Conner, J. and Via, S. 1993. Patterns of phenotypic and genetic correlations among morphological and life-history traits in wild radish, *Raphanus raphanistrum. Evolution*, **47**: 704–711.

Ferreira, M.E., Satagopan, J., Yandell, B.S., Williams, P.H. and Osborn, T.C. 1995. Mapping loci controlling vernalization requirement and flowering time in *Brassica napus. Theor. Appl. Genet.*, **90**: 727–732.

Fox, G.A. 1989. Consequences of flowering time variation in a desert annual: adaptation and history. *Ecology*, **70**: 1294–1306.

Fox, G.A. 1990. Components of flowering time variation in a desert annual. *Evolution*, **44**: 1404–1423.

Fox, G.A. 2001. Failure time analysis: studying times-to-events and rates at which events occur. In *Design and Analysis of Ecological Experiments*, 2nd edn (S.M. Scheiner and J. Gurevitch, eds), pp. 253–289. Oxford: Oxford University Press.

Fox, G.A. and Kelly, C.K. 1993. Plant phenology: neutrality and selection. *Trends Ecol. Evol.*, **8**: 34–35.

Gale, J.S., Rana, M.S. and Lawrence, M.J. 1974. Variation in wild populations of *Papaver dubium*. IX. Limited possibilities for assortative mating. *Heredity*, **32**: 389–396.

Gregorius, H.-R. 1984. Convergence of genotypic frequencies for differential selfing and positive assortative mating at a biallelic locus. *J. Math. Biol.*, **20**: 159–169.

Hartl, D.L. and Clark, A.G. 1997. *Principles of Population Genetics*, 3rd edn. Sunderland, MA: Sinauer Associates.

Hayman, B.I. 1953. Mixed selfing and random mating when homozygotes are at a disadvantage. *Heredity*, **7**: 185–192.

Karlin, S. 1969. *Equilibrium Behavior of Population Genetic Models with Non-random Mating*. New York: Gordon & Breach.

Kelly, C.A. 1992. Spatial and temporal variation in selection on correlated life-history traits and plant size in *Chamaecrista fasciculata. Evolution*, **46**: 1658–1673.

Kuittinen, H., Sillanp, M.J. and Savolainen, O. 1997. Genetic basis of adaptation: flowering time in *Arabidopsis thaliana. Theor. Appl. Genet.*, **95**: 573–583.

Lynch, M. and Walsh, J.B. 1998. *Evolutionary Quantitative Genetics*. Sunderland, MA: Sinauer Associates.

Lyons, E.E. and Mully, T.W. 1992. Density effects on flowering phenology and mating potential in *Nicotiana alata. Oecologia*, **91**: 93–100.

Mazer, S.J. and LeBuhn, G. 1999. Genetic variation in life history traits: evidence within and among populations, and lessons learned. In *Life History Evolution in Plants* (T. Vuorisalo and P. Mutikainen, eds), pp. 85–135. Dordrecht: Kluwer Academic.

McIntyre, G.I. and Best, K.F. 1978. Studies on the flowering of *Thlaspi arvense* L. IV. Genetic and ecological differences between early- and late-flowering strains. *Bot. Gaz.*, **139**: 190–195.

Meagher, T.R. and Delph, L.F. 2001. Individual flower demography, floral phenology, and life history in *Silene latifola*. *Evol. Ecol. Res.*, **3**: 845–860.

Mitchell-Olds, T. and Rutledge, J.J. 1986. Quantitative genetics in natural plant populations: a review of the theory. *Am. Nat.*, **127**: 379–402.

Murfet, I.C. 1975. Flowering in *Pisum*: multiple alleles at the *lf* locus. *Heredity*, **35**: 85–98.

Murfet, I.C. and Reid, J.B. 1974. Flowering in *Pisum*: the influence of photoperiod and vernalising temperatures on the expression of genes *Lf* and *Sn. Z. pflanzenphysiol.*, **71**: 323–331.

Owens, J.N. 1995. Constraints to seed production: temperate and tropical forest trees. *Tree Phys.*, **15**: 477–484.

Pilson, D. 2000. Herbivory and natural selection on flowering phenology in wild sunflower, *Helianthus annuus. Oecologia*, **122**: 72–82.

Reba, A., Blanchard, P., Perret, D. and Vincourt, P. 1997. Mapping quantitative trait loci controlling silking date in a diallele cross among four lines of maize. *Theor. Appl. Genet.*, **95**: 451–459.

Rondeau, R., Van Devender, T., Bertelsen, C., Jenkins, P., Wilson, R. and Dimmitt, M. 1996. Annotated flora and vegetation of the Tucson Mountains, Pima County, Arizona. *Desert Plants*, **12**: 1–46.

Ruel, J.J. and Ayres, M.P. 1999. Jensen's inequality predicts effects of environmental variation. *Trends. Ecol Evol.*, **14**: 361–366.

Schmitt, J. 1983a. Density dependent pollinator foraging, flowering phenology, and temporal pollen dispersal patterns in *Linanthus bicolor. Evolution*, **37**: 124–157.

Schmitt, J. 1983b. Individual flowering phenology, plant size, and reproductive success in *Linanthus androsaceaus*, a California annual. *Oecologia*, **59**: 135–140.

Wehrhahn, C. and Allard, R.W. 1965. The detection and measurement of the effects of individual genes involved in the inheritance of a quantitative character. *Genetics*, **51**: 109–119.

Wright, S. 1921. Systems of mating. II. The effects of inbreeding on the genetic composition of a population. *Genetics*, **6**: 124–143.

APPENDIX: MODELLING TEMPORAL ASSORTATIVE MATING AND SELECTION

To model selection and assortative mating in a mating pool structured by time, we need to describe, for each day of the mating season, the frequencies of flowers of each genotype in the mating pool. As in the main text, assume that flowers last 1 day, have one ovule and are all pollinated.

For a general model, let P_i be the frequency of genotype-i individuals in the population. Let $\theta_i(t)$ be the fraction of all flowers produced in the entire mating season by genotype-i plants that are open on day t. Then each genotype i is represented in the mating pool by $P_i\theta_i(t)$ flowers on day t. Since mating is assumed to be random with respect to the composition of the mating pool each day, the daily contribution to the total frequency of each mating type is given by the expansion of

$$\frac{[\Sigma_k P_k \theta_k(t)]^2}{\Sigma_k P_k \theta_k(t)} \tag{A1}$$

For example, assume that the three genotypes X, Y and Z have the frequencies $(x, y, z) = (0.2, 0.4, 0.4)$ and, on a particular day t, plants of these genotypes produce 10%, 5% and 20%, respectively, of the total number of their flowers. With temporal assortative mating but no selection, the contribution of day t to the total frequency of $x \times x$ matings is

$$\frac{(0.2 \times 0.1)^2}{(0.2 \times 0.1) + (0.4 \times 0.05) + (0.4 \times 0.2)} = \frac{0.0004}{0.12} = 0.003$$

The total frequency of $x \times x$ matings is the sum of all of these daily contributions; by contrast, in a random-mating population, the total frequency is simply $0.2^2 = 0.04$. The numerator in this expression is analogous to the usual expansion of $(p + q)^2$ to derive random-mating probabilities, but each gene frequency is weighted by a θ to allow for temporal assortative mating. The denominators are simply the relative sizes of the mating pool on each day; using them guarantees that the total of all mating types over the entire season is 1.

To gain insight on the general consequences of temporal assortative mating, selection and their interaction, this approach can be greatly simplified. Assume for the moment that, regardless of when they occur, matings have the same chance of leading to a viable seed at the end of the season. Then, standard and cryptic assortative mating have the same evolutionary consequences. Modelling standard assortative mating greatly simplifies the mathematics (and especially the notation) without any loss of generality. Under standard assortative mating, rather than modelling each day's mating pool as in equation (A1), we can model the mating pool for each period defined by a distinct set of genotypes in the mating pool. If the phenologies are not offset from one another too much, there are five discrete mating periods (Fig. 3).

Since the flower frequencies are constant under standard assortative mating, one can think of θ as defining the fraction of flowers produced in an entire mating period. The fractions of each genotype's flower production in each mating period are given in the top part of Fig. 3.

The entries in Table A1 give the frequencies of each mating type during the five mating periods. By adding the rules of Mendelian segregation to the frequency of each mating type, we get the following recursions for change in the genotype frequencies under pure assortative mating:

$$x' = \theta_1 x + \frac{(\theta_2 x + \frac{1}{2}\theta_4 y)^2}{\theta_2 x + \theta_4 y} + \frac{(\frac{1}{2}\theta_6 y)^2}{\theta_6 y + \theta_8 z} + \frac{(\theta_3 x + \frac{1}{2}\theta_5 y)^2}{\theta_3 x + \theta_5 y + \theta_7 z} \tag{A2}$$

$$y' = \frac{\frac{1}{2}\theta_4 y(2\theta_2 x + \theta_4 y)}{\theta_2 x + \theta_4 y} + \frac{\frac{1}{2}(\theta_5 y + 2\theta_7 z)\,(2\theta_3 x + \theta_5 y)}{\theta_3 x + \theta_5 y + \theta_7 z} + \frac{\frac{1}{2}\theta_6 y(2\theta_8 z + \theta_6 y)}{\theta_6 y + \theta_8 z} \tag{A3}$$

$$z' = \frac{(\frac{1}{2}\theta_4 y)^2}{\theta_2 x + \theta_4 y} + \theta_9 z + \frac{(\frac{1}{2}\theta_5 y + \theta_7 z)^2}{\theta_3 x + \theta_5 y + \theta_7 z} + \frac{(\frac{1}{2}\theta_6 y + \theta_8 z)^2}{\theta_6 y + \theta_8 z} \tag{A4}$$

The fractions in equations (A2–A4) exist if, and only if, their denominators are > 0. Note that the θ's for each genotype sum to 1 and, if $\theta_3 = \theta_5 = \theta_7 = 1$, there is no assortative mating and the population will go to Hardy-Weinberg equilibrium in a generation.

There are many ways to add selection to this model. In principle, selection could vary among the mating periods for each genotype, yielding an additional nine parameters to an already parameter-rich model. There could also be frequency-dependent or fertility selection. In this paper, I consider only constant selection fixed over the entire lifespan (i.e. there is a single selection coefficient for each genotype). This can most easily be interpreted biologically as corresponding to genotype-specific differences in survival or growth before the breeding season begins. To write this model in full, use equations (A2–A4) and substitute for each genotype frequency i the product of the frequency and its fitness, iW_i; this yields recursions for the frequencies multipled by the mean fitness, $\bar{W}$.

Table A1. Frequencies of mating types during each mating period

	Genotypes in pool				
Matings	x	x, y	x, y, z	y, z	z
$x \times x$	$\frac{(\theta_1 x)^2}{\theta_1 x}$	$\frac{(\theta_2 x)^2}{\theta_2 x + \theta_4 y}$	$\frac{(\theta_3 x)^2}{\theta_3 x + \theta_5 y + \theta_7 z}$	0	0
$x \times y$	0	$\frac{2\theta_2\theta_4 xy}{\theta_2 x + \theta_4 y}$	$\frac{2\theta_3 x\theta_5 y}{\theta_3 x + \theta_5 y + \theta_7 z}$	0	0
$x \times z$	0	0	$\frac{2\theta_3 x\theta_7 z}{\theta_3 x + \theta_5 y + \theta_7 z}$	0	0
$y \times y$	0	$\frac{(\theta_4 y)^2}{\theta_2 x + \theta_4 y}$	$\frac{(\theta_5 y)^2}{\theta_3 x + \theta_5 y + \theta_7 z}$	$\frac{(\theta_6 y)^2}{\theta_6 y + \theta_8 z}$	0
$y \times z$	0	0	$\frac{2\theta_5 y\theta_7 z}{\theta_3 x + \theta_5 y + \theta_7 z}$	$\frac{2\theta_6 y\theta_8 z}{\theta_6 y + \theta_8 z}$	0
$z \times z$	0	0	$\frac{(\theta_7 z)^2}{\theta_3 x + \theta_5 y + \theta_7 z}$	$\frac{(\theta_8 z)^2}{\theta_6 y + \theta_8 z}$	$\frac{(\theta_9 z)^2}{\theta_9 z}$