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Mutation-selection balance in mixed mating populations

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Abstract

An approximation to the average number of deleterious mutations per gamete, Q , is derived from a model allowing selection on both zygotes and male gametes. Progeny are produced by either outcrossing or self-fertilization with fixed probabilities. The genetic model is a standard in evolutionary biology: mutations occur at unlinked loci, have equivalent effects, and combine multiplicatively to determined fitness. The approximation developed here treats individual mutation counts with a generalized Poisson model conditioned on the distribution of selfing histories in the population. The approximation is accurate across the range of parameter sets considered and provides both analytical insights and greatly increased computational speed. Model predictions are discussed in relation to several outstanding problems, including the estimation of the genomic deleterious mutation rates (U), the generality of ‘selective interference’ among loci, and the consequences of gametic selection for the joint distribution of inbreeding depression and mating system across species. Finally, conflicting results from previous analytical treatments of mutation-selection balance are resolved to assumptions about the life-cycle and the initial fate of mutations.

Keywords

gametic selection; genetic load; mixed mating; mutation; self-fertilization; selective interference

1. Introduction

The continuous input of deleterious mutations has a diversity of consequences for the evolution of natural populations (Crow, 1993; Crow, 2000). The simplest multi-locus models treating this process assume that all deleterious mutations have equivalent effects, that loci are unlinked, and that the fitness effects of mutations combine multiplicatively (Heller and Maynard Smith, 1978; Kondrashov, 1985; Charlesworth et al., 1990b). In this situation, mutation-selection balance is dependent on three parameters: the genomic deleterious mutation rate (U), the dominance coefficient (h), and the selection coefficient (s). Selection is assumed to act on diploid genotypes and s is the proportional fitness reduction caused by a mutation in homozygous form, while h characterizes mutational effects in heterozygotes (see Hartl and Clark, 1989, pp 199–200).

The equilibrium solution for mutation-selection balance can be characterized simply when all mutations have equivalent effects. Let Q denote the average number of deleterious mutations per gamete. Q is the product of the number of loci (L) and the frequency of the deleterious allele at each locus (q). While Q is not measured directly in most experimental studies, estimable measures of genetic load are simple functions of this variable. For example,

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numerous studies have estimated the ‘inbreeding load’, B , which is the slope of the regression of $-\ln[\text{fitness}]$ onto f , the inbreeding coefficient of individuals (Morton et al., 1956; Charlesworth and Charlesworth, 1987). The average $\ln[\text{fitness}]$ of outbred individuals is $-2Qh$ while the corresponding value for fully inbred (homozygous) individuals is $-Qs$. This implies that $B = Qs(1 - 2h)$, i.e. the inbreeding load is a linear function of Q .

A closed-form equation for Q can be obtained if the population is random mating (fully outcrossing) or reproduces exclusively by either self-fertilization or asexual means (this theory is reviewed in section 3 below). However, many hermaphroditic plants and animals have mixed mating systems (Vogler and Kalisz, 2001; Jarne et al., 2000). The basic mixed-mating model stipulates that progeny are produced by outcrossing with probability t , and by self-fertilization with probability $1 - t$. Kondrashov (1985), abbreviated K85, introduced an explicit multi-locus model for mutation-selection balance in mixed mating populations. This deterministic model retains the assumption that loci are unlinked, but still allows associations among loci which can be substantial with mixed mating (Lande et al., 1994).

Charlesworth and Charlesworth (1992) generalized K85 to allow selection on male gametes. In plants, there is substantial overlap between genes expressed in pollen grains and those expressed in the sporophyte (Willing and Mascarenhas, 1984; Honys and Twell, 2004). While selection might also act on female gametes, pollen tube competition provides clear opportunity for selection on male gametes and this process has garnered most empirical attention (Mulcahy et al., 1996). Gametic selection reduces genetic load, and because dominance is irrelevant for haploid pollen genotypes, it alters the relationship between equilibrium load and mating system.

In this paper, I develop an analytical approximation to K85 as generalized to allow pollen selection, essentially Model 1 of Charlesworth and Charlesworth (1992). This approximation is useful for two reasons. First, the dynamical variables of K85 are proportions (of individuals) distributed across an infinite series of categories (defined by mutation counts in heterozygous and homozygous condition, respectively). Because the model must be solved iteratively, infinite arrays must be approximated by finite arrays and, as the mean number of mutations per individual increases, so does the necessary size of these finite arrays. This greatly reduces speed and the model becomes numerically intractable when U/s becomes sufficiently large. The Inbreeding History Model, an approximation abbreviated IHM, must also be solved iteratively. However, it converges very rapidly and predicts Q for parameter sets that are intractable for K85. Second, the IHM predicts equilibria for a number of variables in addition to Q . These quantities yield novel insights about mutation-selection balance, particularly the dynamical consequences of inter-locus associations in mixed mating populations.

2. The Inbreeding History Model

The life cycle is a simple but critical component of the model. Each generation starts with an infinite population of zygotes. Selection is imposed on this population, followed by mutation and then gamete production (as in K85 and Charlesworth et al., 1990b). Finally, gametes are randomly combined to form outcrossed progeny or matched within individuals to produce selfed progeny. A second round of selection may occur on male gametes during this final stage. I measure Q and other statistics at the zygote stage. This differs from some previous studies (e.g. Heller and Maynard Smith, 1978) where accounting is done after selection.

Let X denote the number of heterozygous loci per zygote and Y denote the number of loci that are homozygous for the deleterious allele. The equivalence of mutations in this deterministic model implies that allele frequencies are the same across loci (deleterious alleles are equally rare). If we then stipulate that loci are independent, it follows that X and Y should be Poisson

distributed. This stipulation is correct if the population is either fully outcrossing ($t = 1$) or fully selfing ($t = 0$) and allows direct calculation of Q (Crow, 1970; Heller and Maynard Smith, 1978, 1979; Charlesworth et al., 1990a; **Section 3**). As noted by Charlesworth et al. (1991) however, the number of deleterious mutations per gamete will not be Poisson with mixed mating. The population of gametes that merge to form outbred zygotes is a mixture of contributions from outbred and inbred adults. The mean number of mutations per gamete will differ between these groups owing to differences in the effectiveness of selection on outbred vs. inbred genotypes. The consequence is a subtle form of linkage disequilibrium within the outbred zygote population and the variance in mutation count will slightly exceed the mean.

The other major complication associated with mixed mating is that individuals will vary in the extent to which they are inbred. A fraction t of zygotes are outbred, while the remainder are inbred to varying extents ($f = 1/2, 3/4, 7/8$, etc.). This variation causes identity disequilibria, genotypic associations among loci even when these loci are unlinked (Haldane, 1949; Bennett and Binet, 1956). However, if we conceptually subdivide a population into ‘cohorts’ of individuals that share the same inbreeding coefficient (e.g. Campbell, 1986; Kelly, 1999a,b), variation in inbreeding history can be accommodated (see Figure 1). Identity disequilibrium is absent within cohorts, at least as long as the relevant loci are unlinked.

The Inbreeding History Model (IHM) is a set of recursions predicting changes in the joint distribution of X and Y across an infinite array of cohorts. Let $M_{X(i)}$ and $M_{Y(i)}$ denote the means of X and Y among zygotes in cohort i . Q is an average across cohorts:

$$Q = \sum_{i=0}^{\infty} K_i (M_{Y(i)} + \frac{1}{2} M_{X(i)}) \quad (1)$$

where K_i is the fraction of zygotes in cohort i . The cohort proportions are determined by both mating system and selection. The outbred fraction is fixed ($K_0 = t$) while the inbred cohort fractions satisfy the following simple recursion:

$$K_{i+1} = K_i (1 - t) \frac{W_i}{W} \quad (2)$$

where W_i is the mean fitness within cohort i and $W = \sum_{i=0}^{\infty} K_i W_i$ is the overall mean fitness.

We need to specify the joint distribution of X and Y within each cohort to determine how selection changes $M_{X(i)}$ and $M_{Y(i)}$. As noted above, linkage disequilibria will make mutation counts non-Poisson within the outbred cohort, and these associations persist in selfed progeny. However, the deviation from the Poisson (within cohorts) must usually be quite modest, and for this reason, we assume that the intra-cohort distribution of X and Y is nearly-Poisson:

$$P[X = i, Y = j] = \frac{\exp(-\lambda_x - \lambda_y) \lambda_x^i \lambda_y^j}{\phi i! j!} (1 + \epsilon_1 i^2 + \epsilon_2 j^2 + \epsilon_3 ij) \quad (3)$$

where $\phi = [1 + \epsilon_1 \lambda_x (1 + \lambda_x) + \epsilon_2 \lambda_y (1 + \lambda_y) + \epsilon_3 \lambda_x \lambda_y]$ is a normalization factor. The quantities λ_x , λ_y , ϵ_1 , ϵ_2 , and ϵ_3 are cohort specific parameters. Equation 3 converges on the bivariate Poisson as ϵ_1 , ϵ_2 , and ϵ_3 vanish and I will assume these quantities are small (and thus that the distribution of X and Y remains nearly Poisson within cohorts). Higgs (1994) used the univariate version of equation 3 to study the distribution of deleterious mutations per gamete with synergistic selection.

In principle, one could develop recursions for λ_x , λ_y , ϵ_1 , ϵ_2 , and ϵ_3 under the joint action of selection, mutation, and outcrossing/selfing. However, because the effects of mutation, outcrossing, and selfing can be described directly in terms of the distribution moments without regard to the underlying distribution, it is natural to seek recursions for the moments. To first order in ϵ_1 , ϵ_2 , and ϵ_3 , the first and second central moments of this distribution are:

$$M_X \approx \lambda_x(1 + \epsilon_1(1 + 2\lambda_x) + \epsilon_3\lambda_y) \quad (4a)$$

$$M_Y \approx \lambda_y(1 + \epsilon_2(1 + 2\lambda_y) + \epsilon_3\lambda_x) \quad (4b)$$

$$V_X \approx \lambda_x(1 + \epsilon_1(1 + 4\lambda_x) + \epsilon_3\lambda_y) \quad (4c)$$

$$V_Y \approx \lambda_y(1 + \epsilon_2(1 + 4\lambda_y) + \epsilon_3\lambda_x) \quad (4d)$$

$$C_{XY} \approx \lambda_x\lambda_y\epsilon_3 \quad (4e)$$

where V_X is the variance in X , V_Y is the variance in Y , and C_{XY} is the covariance of X and Y . I have suppressed the subscripts here, but these moments and the recursions outlined below apply distinctly to each cohort.

The fitness of a zygote with X heterozygous mutations and Y homozygotes is $(1 - hs)^X (1 - s)^Y$, where h is the dominance coefficient and s is the selection coefficient (Haldane 1927). K85 requires that mutations are not completely recessive ($h > 0$) and thus remain sufficiently rare so as to invariably appear as heterozygotes in outbred zygotes. I retain this assumption here. To first order in ϵ_1 , ϵ_2 , and ϵ_3 , the predicted moments within cohorts after selection are

$$M'_X \approx (1 - hs)(M_X - sh(V_X - M_X) - sC_{XY}) \quad (5a)$$

$$M'_Y \approx (1 - s)(M_Y - s(V_Y - M_Y) - shC_{XY}) \quad (5b)$$

$$V'_X \approx (1 - hs)(V_X - 2sh(V_X - M_X) - sC_{XY}) \quad (5c)$$

$$V'_Y \approx (1 - s)(V_Y - 2s(V_Y - M_Y) - shC_{XY}) \quad (5d)$$

$$C'_{XY} \approx (1 - hs)(1 - s)C_{XY} \quad (5e)$$

After mutation within each cohort, $M''_X = M'_X + U$ and $V''_X = V'_X + U$, while the other moments remain unaltered: $M''_Y = M'_Y$, $V''_Y = V'_Y$, and $C''_{XY} = C'_{XY}$. Here, U is the genomic deleterious mutation rate, a sum of locus specific (diploid) rates across all loci subject to mutation.

Pollen-level selection can occur during both outcrossing and selfing. The critical difference: with selfing, grains are competing against siblings. Let s_p denote the selective effect of a deleterious mutation in pollen grains. Allowing haploid selection (with the two alleles initially equally frequent) on male but not female gametes, a heterozygous locus in the parent will segregate to a homozygous mutant with probability $(1 - s_p)/(4 - 2s_p)$, to a homozygous wild-type with probability $1/(4 - 2s_p)$, and to a heterozygote with probability $1/2$. Noting that homozygous genotypes are directly transmitted to progeny and that the selfed progeny of cohort i constitute cohort $i + 1$ in the next generation (Figure 1), we find that

$$M_{X(i+1)} = \frac{1}{2}M''_{X(i)} \quad (6a)$$

$$M_{Y(i+1)} = M''_{Y(i)} + \left(\frac{1-s_p}{4-2s_p}\right)M''_{X(i)} \quad (6b)$$

$$V_{X(i+1)} = \frac{1}{4}V''_{X(i)} + \frac{1}{4}M''_{X(i)} \quad (6c)$$

$$V_{Y(i+1)} = V''_{Y(i)} + \left(\frac{1-s_p}{4-2s_p}\right)^2 V''_{X(i)} + \left(\frac{1-s_p}{4-2s_p}\right)\left(\frac{3-s_p}{4-2s_p}\right)M''_{X(i)} + \left(\frac{2-2s_p}{4-2s_p}\right)C''_{XY(i)} \quad (6d)$$

$$C_{XY(i+1)} = \frac{1}{2}C''_{XY(i)} + \frac{1}{2}\left(\frac{1-s_p}{4-2s_p}\right)(V''_{X(i)} - M''_{X(i)}) \quad (6e)$$

The outbred cohort in the next generation is formed by random union of gametes recruited from adults across the entire population. Let Z denote the number of mutations per gamete prior to any gametic selection. Taking appropriate averages over cohorts, the mean (M_Z) and variance (V_Z) of Z are

$$M_Z = \sum_{i=0}^{\infty} K_i \frac{W_i}{W} [M''_{Y(i)} + \frac{1}{2}M''_{X(i)}] \quad (7a)$$

$$V_Z = \sum_{i=0}^{\infty} K_i \frac{W_i}{W} \left[\frac{1}{4}M''_{X(i)} + \frac{1}{4}V''_{X(i)} + C''_{XY(i)} + V''_{Y(i)} + \left(\frac{1}{2}M''_{X(i)} + M''_{Y(i)} - M_Z\right)^2 \right] \quad (7b)$$

Applying selection to the population of pollen grains and combining pollen grains with (unselected) female gametes:

$$M_{X(0)} = M_Z + (1-s_p)(M_Z - s_p(V_Z - M_Z)) \quad (8a)$$

$$V_{X(0)} = V_Z + (1-s_p)(V_Z - 2s_p(V_Z - M_Z)) \quad (8a)$$

And by assumption, $M_{Y(0)} = V_{Y(0)} = C_{XY(0)} = 0$. The rightmost term in equations 8 is based on the univariate version of equation 3.

Finally, we need to specify the mean fitness of each cohort. To first order in ε_1 , ε_2 , and ε_3 ,

$$\ln[W_i] = -hsM_{X(i)} - sM_{Y(i)} + (hs)^2 \frac{V_{X(i)} - M_{X(i)}}{2} + s^2 \frac{V_{Y(i)} - M_{Y(i)}}{2} + hs^2 C_{XY(i)} \quad (9)$$

Equations 1–2, 5–9 fully specify a discrete recursive system that can be solved iteratively. The infinite series of cohorts must be approximated by a finite series, but the number of cohorts can be made large without great numerical cost. With a few exceptions, this system rapidly converges to equilibrium yielding Q (via equation 1), as well as other quantities (M_Z , V_Z , the cohort proportions, the population mean fitness, etc.). As expected, V_Z is typically greater than M_Z when $0 < t < 1$. An executable program that yields the solution for any given U , h , s , s_p , and t is available upon request.

Results

The Inbreeding History Model (IHM) prediction for $U = 1$, $h = 0.05$, $s = 0.2$, and $s_p = 0$ is given for the full range of outcrossing rates in Figure 2. It accurately predicts the numerical results

from K85 as calculated using the recursions in the Appendix. The other trajectory is an extrapolation of the single locus prediction of Ohta and Cockerham (1974), abbreviated OC74. These authors solved for q , the equilibrium frequency of the deleterious allele, as a function of mutational parameters (without gametic selection) and the outcrossing rate, t . The OC74 solution for a single locus can be extrapolated to predict mutational load for the full genome if associations among loci are inconsequential. Charlesworth et al. (1990) compared OC74 to K85 over a range of values for U , h , s , and t and concluded that agreement between the models is generally good. However, Figure 2 shows that there are parameter combinations for which the predictions do not match closely at all.

For intermediate t , the Q predicted by K85 typically exceeds the single locus prediction. The difference is largest when deleterious alleles are substantially recessive ($h < 0.2$) and the mutation rate is high. This deviation is a consequence of ‘selective interference’ (Lande et al., 1994), wherein identity disequilibria among loci reduces the efficiency of selection. For low outcrossing rates (high selfing), the mutational load predicted by OC74 consistently overestimates the Q predicted by K85 (Figure 3). With $t = 0$, the overestimate is typically only 5–10% if the selection coefficient is small ($s = 0.1$), but increases to 30–40% when $s > 0.8$. This discrepancy is not due to associations among loci. Instead, it is a consequence of different assumptions about when new mutations are introduced in the life-cycle. OC74 assumes that new mutations are introduced individually to gametes, while K85 adds mutations to diploid individuals following selection. As shown algebraically in the next section, this translates to a difference in the immediate effects of selection. This is responsible for the differing predictions for Q .

As expected, selection at the pollen stage reduces load: Q declines as s_p increases if other parameters are held constant. Less obviously, the importance of selection at the pollen stage is inversely related to the rate of self-fertilization (Charlesworth and Charlesworth, 1992; Figure 4). If deleterious mutations are partially recessive in their effects on the sporophyte and $s_p = 0$, then Q declines dramatically as the outcrossing rate changes from 1 to 0. With gametic selection the magnitude of this change is much smaller. This is a simple consequence of the fact that selection against deleterious alleles is more effective through the competition of outcross pollen grains than among self-grains (at least in so far as the process is accurately modeled by equations 4 and 6). In the absence of zygotic selection ($s = 0$), Q actually increases with the rate of self-fertilization (Charlesworth and Charlesworth, 1992, pg 708).

To evaluate the IHM, I have iterated the generalized K85 for almost 35,000 distinct parameter sets with U ranging from 0.01 to 1.0, h ranging from 0.01 to 0.4, s ranging from 0.01 to 0.99, s_p ranging from 0 up to s , and t ranging from 0 to 1.0. Due to limits on array sizes (a computing constraint), this dataset includes only cases where $U/(hs) \leq 50$ and many parameter combinations are excluded. For cases with $s_p = 0$, the average deviation between OC74 and K85 (measured as a proportion of the Q predicted by K85) is about 10%, although it can be much larger (e.g. Figure 2). The average error of the IHM is less than 1% in parameter sets with $U = 1.0$. The error is uniformly negligible when the $U \leq 0.1$. In the full collection of runs with $U/(hs) \leq 50$ (available upon request), the largest absolute deviation between the IHM and the generalized K85 is thirteen hundredths of a mutation.

The close correspondence of IHM and K85 suggests that the former may serve as an adequate proxy for the latter in most circumstances. However, caution is necessary when extrapolating outside the tested parameter region. I have run a more limited collection of parameter sets with $U/(hs) = 100$ (e.g. Figure 2). While agreement between IHM and K85 remains very good, there is one interesting discrepancy. With $U = 1$, $h = 0.05$, $s = 0.2$, $s_p = 0$, and $t = 0.5$, K85 predicts an oscillatory approach to the equilibrium $Q = 28.72$. In contrast, the IHM predicts sustained

oscillations around this value (Figure 5). The mean Q over the predicted limit cycle (29.16) is close to the K85 stable point, but the fluctuations persist.

The IHM predicts fluctuations with increasing frequency as $U/(hs)$ increases. Holding hs constant, increasing U expands the range of intermediate outcrossing rates over which fluctuations are observed. However, this range of t is always within the ‘transition zone’ as Q rapidly falls from the high value of a fully outcrossing population and the low value of a fully selfing population. For parameter combinations where K85 can be run, I have only observed sustained cycles in IHM when K85 predicts an oscillatory approach to equilibrium.

3. Contrasting results for completely selfing or outcrossing populations

If selection is zygotc (acting only on the sporophyte), then $Q = U/(2hs)$ in a completely outcrossing population. This result can be obtained in a number of ways, most simply by multiplying the classic result $q = u/(hs)$ by the number of loci and then converting between the per locus mutation rate (u) and the genomic rate U (Haldane, 1927; see also Crow, 1970, pg 144). Alternatively, noting that X is Poisson in zygotes, we can then solve for Q by considering the various events of the life cycle in sequence. The mean for X after selection is $M'_X = (1 - hs)M_X$. After mutation, $M''_X = M'_X + U = (1 - hs)M_X + U$. Let g_f denote the mean number of mutations per female gamete and g_m the corresponding value for pollen. Because I am limiting selection to males, $g_f = M''_X/2$ while $g_m = (1 - s_p)M''_X/2$. Equating M_X to $(g_f + g_m)$ yields

$$Q = \frac{U(2 - s_p)}{4hs + 2s_p(1 - hs)} \quad (10)$$

which exactly matches iterative results from the generalized K85 with $t = 1.0$ (see also the Appendix of Charlesworth and Charlesworth, 1992).

Now consider a completely selfing population ($t = 0$). Heller and Maynard Smith (1978) showed that, in the absence of gametic selection, X and Y are independent Poisson random variables (see also Hopf et al., 1988). Thus, the respective means after zygotic selection are $M'_X = (1 - hs)M_X$ and $M'_Y = (1 - s)M_Y$. Mutation adds a Poisson distributed value (mean U) to X . Finally, segregation of alleles in heterozygotes will occur with the production of selfed progeny. If there is no gametic selection, the predicted means in the next generation of zygotes are

$$M'''_X = [(1 - hs)M_X + U]/2 \quad (11a)$$

and

$$M'''_Y = (1 - s)M_Y + [(1 - hs)M_X + U]/4 \quad (11b)$$

Solving ($M'''_X = M_X$ and $M'''_Y = M_Y$) yields the following equilibria:

$$M_X = \frac{U}{1 + hs} \quad (12a)$$

$$M_Y = \frac{U}{2s(1 + hs)} \quad (12b)$$

and $Q = M_Y + \frac{1}{2} M_X$. These predictions are also confirmed by iteration of K85 with $s_p = 0$ and $t = 0$.

Why do OC74 and K85 differ for complete selfing (Figure 3)? The OC74 recursions for a fully selfing population can be re-written in notation of this paper:

$$M_X''' = (1 - hs)M_X / 2 + U \quad (13a)$$

and

$$M_Y''' = (1 - s)M_Y + (1 - hs)M_X / 4 \quad (13b)$$

Solving, $M_X = 2U/(1 + hs)$ and $M_Y = U(1 - hs)/(2s(1 + hs))$. The predicted number of heterozygotes is greater while homozygotes are fewer than predicted by equations 12.

Equations 13 imply that all new mutations appear first as heterozygotes. In contrast, equations 11 imply that a fraction of new mutations will appear in their first zygote as homozygotes. If deleterious mutations are at least partially recessive ($h < 0.5$), OC74 predicts a higher equilibrium load because new mutations are partially shielded from selection during their first full generation. This rather subtle effect can be quantitatively important when the average lifespan of mutations is short. For the purpose of comparisons, it is useful to re-derive the single locus model using the K85 life cycle. For arbitrary t , the equilibria are:

$$M_X = \frac{U\Omega}{s(1 + \Omega + t)} \quad (14a)$$

and

$$M_Y = \frac{U(1 - t)}{2s(1 - h\Omega + t)} \quad (14b)$$

where $\Omega = s(1 - t) + 2t$. When Q from equations 14 is plotted onto Figure 2, the trajectory is consistently lower than OC74, matching K85 at both extremes ($t = 0$ and 1).

Interestingly, gametic selection changes the mean of Y , but not of X . However, their joint distribution remains Poisson when $t = 0$. Applying the same methods, when $s_p > 0$, equation 11b becomes

$$M_Y''' = (1 - s)M_Y + [(1 - hs)M_X + U](\frac{1 - s_p}{4 - 2s_p}) \quad (15)$$

Noting that $M_X = \frac{U}{1 + hs}$ and solving,

$$M_Y = \frac{U}{s(1 + hs)}(\frac{1 - s_p}{2 - s_p}) \quad (16)$$

as long as $s > 0$. Numerical iterations of K85 confirm this result. An interesting special case is when there is no zygotic selection ($s = 0$). Unless mutations are lethal to pollen grains ($s_p = 1$), equation 15 implies that deleterious mutations will accumulate indefinitely without selection on the sporophyte ($M_Y \rightarrow \infty$).

4. Discussion

The Inbreeding History Model (IHM) provides an accurate approximation of mutation selection balance in mixed mating populations, at least over the range of parameter sets considered. Because both the generalized K85 (Charlesworth and Charlesworth, 1992; Appendix) and the IHM must be solved iteratively in most cases, it is fair to ask why this approximation is useful. Speed and numerical tractability are practical justifications for the IHM. Most IHM runs converge on a solution several hundred times faster than K85. The IHM can thus be used to rapidly provide a 'start point' for K85 that is very close to the true equilibrium. Second, K85 becomes numerically intractable for large U or small values of h and s . This is a serious difficulty if most deleterious mutations have very slight effects, as suggested by recent surveys of molecular data (Eyre-Walker et al., 2006; Loewe et al., 2006). Large U/s values are effectively accommodated by the IHM, although if s is sufficiently small (on the order of the reciprocal of the effective population size), then genetic drift must also be considered.

The IHM also provides insight about the process of multi-locus, mutation-selection balance. Consider selective interference, which is responsible for the large difference between single and multi-locus predictions in Figure 2. This effect is often associated with deleterious mutations of large effect because Lande et al. (1994) focused on embryonic lethals in their study of interference. Their parameter sets were clearly motivated by empirical studies, but it is worth noting that selective interference is not a consequence of high selection coefficients. Instead, the key property of mutations is their recessivity. Consider a population with $U = 0.5$, $h = 0.02$, and $t = 0.7$. If mutations are lethal ($s = 1$), the IHM predicts $Q = 11.98$. This is about seven times greater than the single locus prediction (OC74). However, with the same parameters except $s = 0.1$, the IHM predicts $Q = 121.13$ which is nine times greater than predicted by OC74. Thus, selective interference becomes (proportionally) more important as s declines.

Scofield and Schultz (2006) hypothesize that selective interference is responsible for the rarity of highly selfing, long-lived plants. It is thus important to determine the region of parameter space over which this process has substantial effects. Lethals are known to be highly recessive ($h = 0.01$ – 0.02), but it is unclear that they occur at a sufficient rate to generate the mutational load necessary for interference. The estimated lethal mutation rate is only a few percent per generation in *Drosophila* (Simmons and Crow, 1977). While the rate could be somewhat higher in long-lived plants (Klekowski and Godfrey, 1989), it seems likely that most inbreeding depression is due to the aggregate effects of mutations with smaller effects. While it is commonly stated that mildly deleterious mutations are likely to be 'nearly additive', estimates for the average dominance coefficient of such alleles are typically in the range of 0.05 to 0.3 (Johnston and Schoen, 1995; Willis, 1999; Charlesworth and Hughes, 2000). The IHM predicts substantial interference, at least in the lower part of this range ($h = 0.05$ – 0.15), if $U \geq 1$.

Additional information is provided by other dynamical variables of the IHM, specifically the cohort proportions (K_i) and cohort specific mutation counts. Figure 6 illustrates cohort proportions before and after selection for a case in which selective interference is important. Selection dramatically alters the proportions, and as a consequence, the outbred cohort produces the great majority of individuals in the next generation. The increased efficiency of selection against deleterious mutations within inbred cohorts becomes inconsequential because these cohorts make a very limited contribution to the next generation (Ganders, 1972; Lande et al., 1994). In this case, it is the aggregate effect of many mutations and not the exposure of a single recessive lethal that limits the contribution of inbred cohorts.

Now consider the oscillatory approach to equilibrium of Figure 5. These fluctuations are not a simple consequence of the large immediate change in outcrossing rate (from $t = 1$ to $t = 0.5$). If t makes a more dramatic change, say from 1 to 0.2, K85 predicts a rapid approach to the new equilibrium Q . The sustained oscillation result from the complex evolution of the inter-locus associations that are most pronounced at intermediate outcrossing rates. The cohort proportions and the cohort specific moments of X and Y evolve in predictable ways over the course of a cycle. At the ebb of a cycle (when Q is below the equilibrium), M_X and M_Y are relatively low within the first few cohorts. Because the strength of selection is roughly proportional to these means (equations 4), Q increases. As the means increase, selection becomes more efficient which should subsequently reduce the rate of increase in Q . However, over most of the upward part of the cycle, V_X/M_X and V_Y/M_Y actually decline. This limits selection and allows Q to climb substantially above the equilibrium. At the crest of the cycle, V_X/M_X and V_Y/M_Y start to increase, but M_X and M_Y are sufficiently high that selection overtakes mutation and these means begin to decrease. However, V_X and V_Y continue to increase (or decline only slightly) over this first stage of downward cycle. V_X/M_X and V_Y/M_Y (within the first few cohorts) reach their maxima in the midst of the down cycle, and because selection becomes most efficient substantially after the crest, this drives Q below the eventual equilibrium. The cycling is essentially caused by a time-lag in adjustments of cohort specific means and variances.

The difference between means and variances ultimately stems from the mixing of gametes derived from different cohorts (Charlesworth et al. 1991; equation 7). Thus, changes in V_X/M_X and V_Y/M_Y over a cycle can also be interpreted in terms of changing cohort proportions and their respective contributions to the outbred gamete pool. Over the course of the upward cycle, K_0' gradually increases. This limits the contribution of the more inbred cohorts and thus the variance in the number of mutations per gamete (V_Z in equation 7). K_0' begins to decline at the crest of the cycle and V_Z increases relative M_Z , which translates to higher values for V_X/M_X and V_Y/M_Y . Of course, the fact that K85 predicts damped oscillations while they are persistent for IHM (at least in Figure 5) indicates a subtle but nonetheless significant difference between equation 3 and the actual joint distribution of X and Y within cohorts.

Relationship to previous theory

The IHM is similar to the analytic approximations of Charlesworth et al. (1991) who developed recursions for the moments of X and Y in a population subdivided into two categories, inbred vs outbred. In the IHM, the inbred category is parsed into an infinite series of ‘cohorts’, each of which is uniformly inbred (Figure 1). Campbell (1986) used a similar partitioning to study the dynamics of mating system modifiers and I applied it to predict response to selection on quantitative traits in a partially self-fertilizing population (Kelly, 1999a,b). Parsing the population this way simplifies the treatment of dynamics because identity disequilibrium is absorbed into the cohort structure of the model (all of this assuming that loci are unlinked).

The second way that the IHM differs from the approximations of Charlesworth et al. (1991) concerns the joint distribution of heterozygote and homozygote counts, X and Y respectively. These authors assumed that the X and Y were either bi-variate normal within each category or independent Poisson variables. The latter was used when the bi-variate normal was found to be inaccurate for high selfing rates. The IHM distribution (equation 3) combines several essential features of the Poisson and Bi-Normal distributions. Like the Poisson, it predicts counts from a large series of trials in which a ‘success’ (the locus harbors one or two mutant alleles) is a rare event. Like the Bi-Normal, equation 16 allows means to differ from variances, and for X to be correlated with Y . This combination of properties provides accurate predictions for mutational load over the entire range of outcrossing rates, even when loci combine in a non-multiplicative way (Higgs, 1994; Kelly, unpublished results). The IHM can also be generalized to consider evolutionary changes in the outcrossing rate. The dynamics of ‘mating

system modifiers' under both constant and variable inbreeding depression will be treated in a separate paper (Porcher et al, *in prep*).

Closed-form solutions for Q , or other quantities that characterize mutation-selection balance, can be obtained with either complete outcrossing or complete selfing (Section 3). Each situation has been treated by numerous authors, but published results differ in appearance. There are three major reasons for discrepancies. First, different treatments census the population at different points in the life cycle. Here, I count mutations at the zygote stage, while Hopf et al. (1988) census the population following selection. Second, there are subtle differences in the nature of approximations (see below). Third, different treatments assume that new mutations are introduced at different points in the life cycle.

The life cycle assumption is responsible for the differing predictions of OC74 and K85 for Q in highly selfing populations (Figure 3). Mutations introduced prior to gamete formation (as in K85) can be segregated to homozygous form in their first zygote. This allows selection to remove them more rapidly than when mutations are introduced individually to gametes (as in OC74) and thus invariably appear in their first zygote as heterozygotes. While the OC74 life cycle is more consistent with the classical view of mutations in population genetics, somatic mutations are more accurately described by the K85 life cycle, at least as long as these mutations do not effect the fitness of the maternal plant.

An important application of genetic load predictions is the estimation of U , the genomic deleterious mutation rate. Charlesworth et al. (1990a) develop an estimator for U based on the amount of inbreeding depression, δ , in a fully selfing population:

$$U \approx \frac{-2\text{Ln}(1-\delta)}{1-2h} \quad (17)$$

The value for h must be established from other data. Johnston and Schoen (1995) applied this technique to four highly selfing populations within the genus *Amsinckia*, estimating δ and h from fitness measurements on both outcrossed and selfed progeny. Equation (24) yielded U estimates that ranged from 0.24 to 0.87 across populations.

This estimator for U can be related to the equations in Section 3 by noting that, if $t = 0$, the inbreeding load, B , is equal to $-\text{Ln}(1-\delta)$. Since $B = Qs(1-2h)$, we can combine equations 12 and solve for U to obtain

$$U = \frac{-2\text{Ln}(1-\delta)}{1-2h} \left(\frac{1+hs}{1+s} \right) \quad (18)$$

If mutations are introduced individually to gametes (as in OC74), then

$$U = \frac{-2\text{Ln}(1-\delta)}{1-2h} \left(\frac{1}{1+s(2-h)} \right) \quad (19)$$

Equations 18 and 19 converge on equation 17 as s gets small. This makes sense: if s is small, segregation will cause mutations to be lost or fixed within a lineage before selection can have any appreciable effect. Thus, selection acts strictly on alleles in homozygous form, which is the argument that produces equation 17 (Charlesworth et al., 1990a).

Gametic selection

Selection on the haploid pollen genotype reduces Q and thus the magnitude of inbreeding depression. If gametic selection is important, it will downwardly bias estimates of U obtained via equation 17 because the observed inbreeding depression is less than predicted with purely

zygotic selection. However, the quantitative effect of gametic selection is severely limited with complete selfing (Figure 4), so this bias should be small. With $t = 0$, the mean number of loci that are heterozygous for deleterious mutations is unaffected by s_p , while the mean for homozygous loci is reduced only by a factor of $(1-s_p)/(1-s_p/2)$. This should be minor unless s_p is large.

In fact, pollen selection is generally insufficient to constrain the accumulation of deleterious mutations in fully selfing populations. Unless mutations are lethal to pollen grains ($s_p = 1$), equation 15 implies that deleterious mutations will accumulate indefinitely if there is no selection on the sporophyte ($s = 0$). A fully selfing population is essentially a collection of distinct, self-perpetuating lineages. The homozygous mutation count (Y) for a particular lineage never declines and will continuously increase when $s_p < 1$. Zygotic selection allows lineages with low Y to displace lineages with high Y , thus preventing the population mean from increasing indefinitely. Without zygotic selection however, mutations accumulate continuously in a process analogous to, but much faster than, Muller's ratchet.

The pollen selection model, essentially Model 1 of Charlesworth and Charlesworth (1992), has a number of features that merit comment. First, the dependency on mating system (Figure 4) is due to differing selection regimes on outcross and self pollen. Grains are competing against the full population of genotypes to fertilize ovules through outcrossing, but against full siblings in self-fertilization. The model stipulates a 'non-competing' mode of self-fertilization (see Lloyd, 1979), wherein a fixed proportion of eggs/ovules are fertilized by self pollen. Selfing may occur prior to, or after, fertilization of the remaining ovules by cross pollen. However, if self and cross pollen are simultaneously contending, what Lloyd (1979) termed 'competing selfing', then the fraction of selfed ovules will not be fixed. Instead, it will depend on the genotypic composition of the self and cross pollen pools. A lower fraction of ovules will be selfed if the mean pollen fitness of the maternal plant is below average, a higher fraction for maternal plants with low mutation counts in their pollen grains. Competing selfing would thus increase the efficiency of selection.

A second assumption the present model is that plants produce many pollen grains for each ovule. Equation 6 assumes sampling of pollen grains with replacement in the fertilization of ovules, which is valid if the pollen/ovule ratio is high. However, many habitually self-fertilizing species exhibit reduced pollen/ovule ratios. A hypergeometric form for equation 6 (sampling without replacement) would reduce the efficiency of selection against deleterious mutations. A third notable feature of the model: pollen fitness is determined exclusively by mutation count and is independent of the maternal genotype. Much of the data on pollen competition suggests a dependency of pollen fitness on maternal genotype (Jones, 1924; Pfahler, 1967; Cruzan, 1990). Finally, the model limits selection to male gametes. Selection might also occur on female gametes, although it would likely be different in both magnitude and character. Butruille and Boiteux (2000) present a single locus model allowing selection on both male and female gametes (different selection coefficients for each) in a tetraploid organism.

The simple prediction of the model is that gametic selection can substantially reduce genetic load *if* mutations affecting sporophyte fitness are also detrimental to pollen performance. The high fraction of genes expressed in both pollen and sporophyte indicate a potential for this kind of selection (Mulcahy et al., 1996; Honys and Twell, 2004), although evidence regarding the effects of specific mutations is more limited. In principle, this could be addressed experimentally by measuring the correlation between pollen and sporophyte fitness across a collection of mutation accumulation lines. The importance of pollen-level selection is contingent on the mating system and is much stronger in outcrossing than self-fertilizing species. As a corollary, this process tends to diminish the extent that load declines with increased inbreeding (Figure 4). This is directly relevant to the rather complicated relationship

between mating system and the amount of inbreeding depression within species (Husband and Schemske, 1996; Armbruster and Gobeille Rogers, 2004). Outcrossers are expected to have higher inbreeding depression than selfers (Crow, 1970; Lande and Schemske, 1985), but selection on pollen grains should reduce the magnitude of this difference.

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Appendix

The recursions below provide the K85 predictions for Q . I use the form of the model developed by Charlesworth et al., 1990b. $F[i, j]$ is the frequency of zygotes carrying i mutations in heterozygous form and j mutations as homozygotes. Thus, the frequencies after zygotic selection, $F' [i, j]$, are:

$$F' [i, j] = F[i, j] \frac{(1 - hs)^i (1 - s)^j}{W}$$

Because the population is infinite in size and deleterious mutations remain rare, all new mutations occur at loci previously homozygous for the wild type allele. This implies that the frequencies after mutation, $F'' [i, j]$, can be predicted from the following formula:

$$F'' [i, j] = \sum_{k=0}^i F' [i - k, j] \frac{e^{-U} U^k}{k!}$$

Gametes are produced by sampling from the distribution $F'' [i, j]$. To predict the composition of outcrossed zygotes, it is necessary to distinguish the distributions of deleterious mutations per female gamete, $G_F[k]$, from the comparable distribution for male gametes, $G_M[k]$. For female gametes,

$$G_F[k] = \sum_{i,j} F'' [i, j] Z[k | i, j]$$

where $Z[k | i, j]$ is the probability that segregation of diploid genotype $\{i, j\}$ yields a gamete bearing k deleterious mutations. Because loci are unlinked, $k = j + x$, where x is a binomial random variable with $n=i$ and $p=0.5$. The equation for male gametes allows selection in which pollen fitness is a multiplicative function of mutation count:

$$G_M[k] = \sum_{i,j} F'' [i, j] Z[k | i, j] \frac{(1 - s_p)^k}{\omega}$$

where ω is the mean fitness of pollen grains: $\omega = \sum_{i,j} F'' [i, j] Z[k | i, j] (1 - s_p)^k$. Let $H[i, j]$ denote the frequency of zygotes produced by self-fertilization that have i mutations as heterozygotes and j as homozygotes:

$$H[i, j] = \sum_{x \geq i} \sum_{y \leq j} F'' [x, y] M[x - i, j - y | x]$$

where $M[*]$ is the multinomial probability:

$$M[a, b | x] = \binom{x}{a \quad b} \left(\frac{1}{2}\right)^a \left(\frac{1 - s_p}{4 - 2s_p}\right)^b \left(\frac{1}{4 - 2s_p}\right)^{x-a-b}$$

Here, x is the number of heterozygotes in the parent, a is the number among these loci that segregate to heterozygotes and b is the number that segregate to mutant homozygotes.

The zygotic distribution of the next generation is a simple combination of the last three equations:

$$F[i, 0] = (1 - t)H[i, 0] + t \sum_{k \leq i} G_M[k] G_F[i - k]$$

and

$$F[i, j] = (1 - t)H[i, j] \text{ for } j > 0.$$

Figure 1.

The inbreeding cohort structure of a partially self-fertilizing population is depicted. Each circle denotes a cohort (a collection of individuals of the same inbreeding coefficient) and the arrows denote transmission of gametes.

Figure 2.

The value for Q (measured in zygotes prior to selection) is predicted as a function of the outcrossing rate, t , for three different models (K85=diamonds, OC74=squares, IHM=triangles). The parameters: $U = 1$, $h = 0.05$, $s = 0.2$, and $s_p = 0.0$. See text for an explanation of the three models.

Figure 3. The predicted values of Q from K85 (diamonds) and OC74 (squares) within fully selfing populations are given as a function of the selection coefficient.

Figure 4.

The equilibrium Q is given as a function of the outcrossing rate (t) with $U = 0.1$, $h = 0.1$, $s = 0.1$ and for three different values of s_p : $s_p = 0$ (diamonds), $s_p = 0.01$ (squares), and $s_p = 0.05$ (triangles). The IHM and K85 predictions are indistinguishable for all cases.

Figure 5.

The predicted trajectory of Q when t changes from 1.0 (at generation 0) immediately to $t = 0.5$. The solid line is the IHM while the squares are K85. Here, $U = 1.0$, $h = 0.05$, $s = 0.2$, and $s_p = 0$.

Figure 6.

The IHM cohort proportions before and after selection (hatched bars for K_i and unfilled bars for K_i') are given for cohorts 0 – 4 at equilibrium with $t = 0.6$, $U = 1$, $h = 0.05$, and $s = 0.2$. The neutral proportions (solid bars) are shown for comparison.